

**NTP TECHNICAL REPORT
ON THE
TOXICOLOGY AND CARCINOGENESIS
STUDIES IN Hsd:SPRAGUE DAWLEY SD RATS
EXPOSED TO WHOLE-BODY RADIO FREQUENCY
RADIATION AT A FREQUENCY (900 MHz) AND
MODULATIONS (GSM AND CDMA) USED BY
CELL PHONES**

Scheduled Peer Review Date: March 26 to 28, 2018

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NTP TR 595



National Toxicology Program

**National Institutes of Health
Public Health Service
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES**

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(WHOLE-BODY EXPOSURE)**

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ABSTRACT

GSM- AND CDMA-MODULATED CELL PHONE RADIO FREQUENCY RADIATION

Synonyms: Cell phone radio frequency radiation; mobile phone radio frequency radiation

The predominant source of human exposure to radio frequency radiation (RFR) occurs through the use of cellular phone handsets. The Food and Drug Administration nominated cell phone RFR emission for toxicology and carcinogenicity testing in 1999. At that time, animal experiments were deemed crucial because meaningful human exposure data from epidemiological studies were not available. Male and female Hsd:Sprague Dawley SD rats were exposed to time-averaged whole-body specific absorption rates of Global System for Mobile Communications (GSM)- or Code Division Multiple Access (CDMA)-modulated cell phone RFR at frequencies of 900 MHz (herein referred to as “cell phone RFR”) *in utero*, during lactation, and after weaning for 28 days or 2 years. Genetic toxicology studies were conducted in rat peripheral blood erythrocytes and leukocytes, brain cells, and liver cells.

STUDY DESIGN

28-Day Studies

Beginning on gestation day (GD) 6, groups of 20 time-mated F₀ female rats were housed in specially-designed reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 3, 6 or 9 W/kg for 5 to 7 days per week, continuing throughout gestation and lactation. The daily exposure duration was 9 hours and 10 minutes over an 18-hour and 20-minute period, as exposures cycled between modulations every 10 minutes. There were seven exposure groups per sex, including a shared sham control and three exposure groups for each modulation. At weaning, 10 males and 10 females per group were selected across four litters for continuation. Weaning occurred on the day the last litter reached postnatal day (PND) 21, marking the beginning of the 28-day study. Male and female F₁ offspring continued to receive whole-body exposures to GSM- or CDMA-modulated cell phone RFR at the same power levels and under the same exposure paradigm, 5 to 7 days per week for up to 28 days.

2-Year Studies

Beginning on GD 5, groups of 56 time-mated F₀ female rats were housed in reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 1.5, 3, or 6 W/kg for 7 days per week, continuing throughout gestation and lactation. The daily exposure duration was 9 hours and 10 minutes over an 18-hour and 20-minute period, as exposures cycled between modulations every 10 minutes. There were seven exposure groups per sex, including a shared sham control and three exposure groups for each modulation. At weaning, three males and three females per litter from 35 litters were randomly selected per exposure group for continuation. Weaning occurred on the day the last litter reached PND 21, marking the beginning of the 2-year studies. Groups of 105 male and 105 female F₁ offspring continued to receive whole-body exposures to GSM- or CDMA-modulated cell phone RFR at the same power levels and under the same exposure paradigm, 7 days per week for up to 104 weeks. After 14 weeks of exposure, 10 rats per group were randomly selected for interim histopathologic evaluation and five were designated for genetic toxicity evaluation.

PERINATAL FINDINGS AND THERMAL EFFECTS

Consistent perinatal effects were observed between modulations, and in both the 28-day and 2-year studies, including lower dam body weights in late gestation and lactation, lower pup body weights and lower pup survival rates. Whole-body exposure to GSM- or CDMA-modulated cell phone RFR had no effect on survival of dams during gestation or lactation and no effect on littering, litter size or live litter pup numbers on PND 1. Lower body weight gains were observed during gestation in dams exposed to GSM during the 28-day and 2-year studies and the CDMA 28-day studies, with body weight effects becoming more pronounced and persisting throughout lactation for both modulations and studies. Lower pup survival was observed for GSM exposure at 9 W/kg in early lactation (before PND 4) and at 6 and 9 W/kg in CDMA-exposed animals, in early and late (after PND 4) lactation. Lower male and female pup body weights were observed beginning in early lactation following exposure to \geq 6 W/kg of either GSM- or CDMA-modulated cell phone RFR.

Body weight decreases in RFR exposed groups persisted throughout the post-weaning period in the 28-day studies, were observed at the 14-week interim evaluation in the 2-year studies, but eventually resolved and were not

observed at later time points in the 2-year studies. There were no clinical observations associated with exposures to either modulation.

In the 28-day studies, subcutaneously implanted microchips were used to record body temperatures of animals within 3 to 5 minutes of exposure pauses. Body temperatures were recorded in F₀ females during gestation and lactation and in F₁ offspring during the post-weaning phase. Higher body temperatures were observed during gestation in 9 W/kg GSM dams and during lactation in ≥ 6 W/kg GSM dams and 9 W/kg CDMA dams. At power levels selected for the 2-year studies (up to 6 W/kg), body temperature elevations did not exceed 1° C in the 28-day study measurements. No exposure-related temperature effects were observed in F₁ offspring.

2-YEAR STUDIES

In the 2-year studies, there was significantly lower survival in the shared male sham control group compared to almost all exposed groups, for both modulations. Survival began to decline at a faster rate than in exposed groups after week 75. In the sham control group, 28% of animals survived to study termination, compared to 48% to 68% for exposed groups across both modulations. Lower survival in sham control male rats was largely attributed to higher severity of chronic progressive nephropathy and there was a spectrum of lesions in other organs considered secondary to chronic progressive nephropathy that occurred at higher incidences in male sham controls. Survival in the shared female sham control group was significantly lower than the 6 W/kg CDMA-exposed group; however, it was similar to all other exposure groups, across modulations. At study termination, there was no effect on body weight in male or female rats, and there were no exposure-related clinical observations.

At the 14-week interim evaluation, there were increased incidences of right ventricular cardiomyopathy in the heart of male rats following exposure to GSM- and CDMA-modulated cell phone RFR compared to sham controls.

At 14 weeks, sperm motility and counts were evaluated in male rats exposed to GSM or CDMA. Exposure to whole-body GSM- or CDMA-modulated cell phone RFR, up to 6 W/kg, did not result in significant changes/differences in reproductive organ histopathology or sperm parameters in male rats compared to the sham controls.

At 2 years, there were similarities in neoplastic and nonneoplastic responses between modulations. Following exposure to GSM- or CDMA-modulated cell phone RFR, there were increases in the incidences of malignant schwannoma in the heart of male rats, with a significant positive trend in the incidences in GSM- and CDMA-exposed males and a significant pairwise increased incidence in CDMA 6 W/kg males. Also observed in the heart were significantly increased incidences of right ventricular cardiomyopathy in 3 and 6 W/kg GSM male and female rats and 6 W/kg CDMA male rats.

Several other, weaker, responses were observed in both modulations including malignant glioma in the brain, adenomas in the pituitary gland (pars distalis), and pheochromocytomas of the adrenal medulla. Additionally, in GSM male rats there were marginal responses in the prostate gland, granular cell tumors of the brain, and in pancreatic islets that were not observed in CDMA-exposed rats, and in CDMA-exposed male rats, there was a response in the liver. The relationship between these responses and exposure to GSM or CDMA RFR was uncertain.

In the brain, there were incidences (not statistically significant) of malignant glioma in all groups of GSM male rats, in 6 W/kg CDMA male rats, and in 1.5 W/kg CDMA females, compared to no incidences in either the male or female sham control groups. There were also occurrences of glial cell hyperplasia in the brain of GSM and CDMA male rats and CDMA female rats that were not observed in sham control animals.

In the pituitary gland (pars distalis) of male rats, there were increased (not statistically significant) incidences of adenoma in all GSM-exposed groups and significantly increased incidences in 3 W/kg CDMA males compared to the sham controls.

There were significantly increased incidences of benign, malignant or complex pheochromocytoma (combined) in the adrenal medulla of the 1.5 and 3 W/kg GSM male rats and 1.5 W/kg CDMA female rats. In GSM female rats, there were increased incidences of hyperplasia in the adrenal medulla at 6 W/kg.

There were increased incidences (not statistically significant) of prostate gland adenoma in 3 W/kg rats, and a single incidence of prostate gland carcinoma in the same group. The incidence and severity of prostate epithelial hyperplasia was slightly higher in all exposed groups of GSM male rats. An exposure-related increase in the incidence of prostate gland epithelial hyperplasia was also observed in CDMA male rats.

There were increased incidences (not statistically significant) of benign granular cell tumor in the brain of all exposed groups of GSM male rats compared to the sham controls, and a single incidence of malignant granular cell tumor in the 3 W/kg GSM group.

There was a significantly increased incidence of adenoma or carcinoma (combined) in pancreatic islets in 1.5 W/kg GSM male rats.

In CDMA male rats, there were incidences of hepatocellular adenoma in all exposed groups, and one incidence of carcinoma each in the 3 and 6 W/kg groups. These neoplasms were not statistically significant, but were not observed in the sham control group.

A few nonneoplastic lesions that were not associated with any of the neoplastic responses were also observed.

There were increased incidences of thyroid gland C-cell hyperplasia in all groups of GSM-exposed female rats.

GENETIC TOXICOLOGY

As part of the 14-week interim evaluation, samples of frontal cortex, hippocampus, cerebellum, liver, and blood leukocytes were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, and five tissues per animal). Samples of peripheral blood were also evaluated for chromosome damage in the micronucleus assay. Results are based on the 100-cell scoring approach that was standard at the time of the studies; data obtained using a second, 150-cell scoring approach recommended in a recently adopted international guideline for the *in vivo* comet assay, are noted for the few instances where results differed between the two methods. A significant increase in DNA damage (% tail DNA) was observed in hippocampus cells of male rats exposed to the CDMA modulation. Although the levels of DNA damage in hippocampus cells were also increased in an exposure-

related fashion using the 150-cell scoring approach, the increases were not statistically significant. An exposure-related increase in DNA damage seen in the cells of the frontal cortex of male rats exposed to the CDMA modulation was judged to be equivocal based on a significant trend test. Although results from scoring 100 cells were negative for male rat blood leukocytes exposed to either CDMA or GSM modulations, the results (both CDMA and GSM) were judged to be equivocal when evaluated using the 150-cell scoring method. No statistically significant increases in DNA damage were observed in any of the female rat samples scored with the 100-cell approach; with the 150-cell approach, results in peripheral blood leukocytes of female rats (CDMA) were judged to be equivocal.

No significant increases in micronucleated red blood cells or changes in the percentage of immature erythrocytes among total erythrocytes were observed in peripheral blood of rats of either sex exposed to either modulation of cell phone RFR.

CONCLUSIONS

Under the conditions of this 2-year whole-body exposure study, there was *some evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley SD rats based on the incidences of malignant schwannoma in the heart. The incidences of adenoma or carcinoma (combined) in the prostate gland, malignant glioma and benign or malignant granular cell tumors in the brain, adenoma of the pars distalis in the pituitary gland, pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla, and pancreatic islet cell adenoma or carcinoma (combined) may have been related to cell phone RFR exposure. There was *no evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley SD rats administered 1.5, 3, or 6 W/kg. There was *some evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley SD rats based on the incidences of malignant schwannoma in the heart. The incidences of malignant glioma in the brain, adenoma of the pars distalis in the pituitary gland, and adenoma or carcinoma (combined) of the liver may have been related to cell phone RFR exposure. There was *equivocal evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley SD rats based on the incidences of malignant glioma in the brain and pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla.

Increases in nonneoplastic lesions in the heart, brain, and prostate gland of male rats, and of the heart, thyroid gland, and adrenal gland in female rats occurred with exposures to GSM cell phone RFR at 900 MHz. Increases in nonneoplastic lesions of the heart, brain, and prostate gland occurred in males, and of the brain in females exposed to CDMA cell phone RFR at 900 MHz.

* Explanation of Levels of Evidence of Carcinogenic Activity is on page 15.

**Summary of the 2-Year Carcinogenesis and Genetic Toxicology Studies
of GSM- and CDMA-Modulated Cell Phone RFR Exposure in Rats**

	GSM-Modulated Cell Phone RFR Male Rats	GSM-Modulated Cell Phone RFR Female Rats	CDMA-Modulated Cell Phone RFR Male Rats	CDMA-Modulated Cell Phone RFR Female Rats
Whole-body GSM- or CDMA-modulated cell phone RFR exposure	0, 1.5, 3, or 6 W/kg	0, 1.5, 3, or 6 W/kg	0, 1.5, 3, or 6 W/kg	0, 1.5, 3, or 6 W/kg
Survival rates	25/90, 45/90, 50/90, 60/90	48/90, 55/90, 48/90, 57/90	25/90, 43/90, 56/90, 43/90	48/90, 46/90, 50/90, 61/90
Body weights	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group
Nonneoplastic effects	<u>Heart</u> : ventricle right, cardiomyopathy (54/90, 62/90, 72/90, 74/90); Schwann cell hyperplasia (0/90, 1/90, 0/90, 2/90) <u>Prostate gland</u> : epithelium, hyperplasia (5/90, 13/90, 11/90, 11/90) <u>Brain</u> : glial cell, hyperplasia (0/90, 2/90, 3/90, 1/90)	<u>Heart</u> : ventricle right, cardiomyopathy (4/90, 9/90, 14/90, 15/90) <u>Thyroid gland</u> : C-cell, hyperplasia (28/90, 49/88, 45/90, 43/88) <u>Adrenal medulla</u> : hyperplasia (13/86, 19/90, 14/90, 25/86)	<u>Heart</u> : ventricle right, cardiomyopathy (54/90, 45/90, 62/90, 74/90); Schwann cell hyperplasia (0/90, 0/90, 0/90, 3/90) <u>Brain</u> : glial cell, hyperplasia (0/90, 2/90, 0/90, 2/90)	<u>Brain</u> : glial cell, hyperplasia (0/90, 0/90, 1/90, 1/90)
Neoplastic effects	<u>Heart</u> : schwannoma malignant (0/90, 2/90, 1/90, 5/90)	None	<u>Heart</u> : schwannoma malignant (0/90, 2/90, 3/90, 6/90)	None
Equivocal findings	<u>Prostate gland</u> : adenoma or carcinoma (2/90, 2/90, 7/90, 3/90) <u>Brain</u> : glioma malignant (0/90, 3/90, 3/90, 2/90); meninges, granular cell tumor benign or malignant (1/90, 3/90, 4/90, 3/90) <u>Pituitary gland</u> : pars distalis, adenoma (17/89, 28/90, 26/90, 26/90)	None	<u>Brain</u> : glioma malignant (0/90, 0/90, 0/90, 3/90) <u>Pituitary gland</u> : pars distalis, adenoma (17/89, 25/90, 34/90, 13/90) <u>Liver</u> : hepatocellular adenoma or carcinoma (combined) (0/90, 2/90, 4/89, 1/88)	<u>Brain</u> : glioma malignant (0/90, 3/90, 0/90, 0/90) <u>Adrenal medulla</u> : benign, malignant, or complex pheochromocytoma (1/86, 9/89, 5/87, 4/88)

**Summary of the 2-Year Carcinogenesis and Genetic Toxicology Studies
of GSM- and CDMA-Modulated Cell Phone RFR Exposure in Rats**

	GSM-Modulated Cell Phone RFR Male Rats	GSM-Modulated Cell Phone RFR Female Rats	CDMA-Modulated Cell Phone RFR Male Rats	CDMA-Modulated Cell Phone RFR Female Rats
Equivocal findings (continued)	<u>Adrenal medulla:</u> benign, malignant, or complex <u>pheochromocytoma</u> (11/88, 24/90, 28/89, 14/87)			
	<u>Islets, pancreatic:</u> adenoma or carcinoma (13/90, 27/89, 19/86, 16/85)			
Level of evidence of carcinogenic activity	Some evidence	No evidence	Some evidence	Equivocal evidence
Genetic toxicology				
DNA damage: GSM-modulated		Negative in frontal cortex, hippocampus, cerebellum, liver, and leukocytes (males and females)		
CDMA-modulated		Positive in hippocampus (males); negative in hippocampus and frontal cortex (females), cerebellum, liver, and leukocytes (males and females); equivocal in frontal cortex (males)		
Micronucleated erythrocytes in peripheral blood <i>in vivo</i> :				
GSM-modulated		Negative in males and females		
CDMA-modulated		Negative in males and females		

EXPLANATION OF LEVELS OF EVIDENCE OF CARCINOGENIC ACTIVITY

The National Toxicology Program describes the results of individual experiments on a test agent and notes the strength of the evidence for conclusions regarding each study. Negative results, in which the study animals do not have a greater incidence of neoplasia than control animals, do not necessarily mean that a test agent is not a carcinogen, inasmuch as the experiments are conducted under a limited set of conditions. Positive results demonstrate that a test agent is carcinogenic for laboratory animals under the conditions of the study and indicate that exposure to the test agent has the potential for hazard to humans. Other organizations, such as the International Agency for Research on Cancer, assign a strength of evidence for conclusions based on an examination of all available evidence, including animal studies such as those conducted by the NTP, epidemiologic studies, and estimates of exposure. Thus, the actual determination of risk to humans from test agents found to be carcinogenic in laboratory animals requires a wider analysis that extends beyond the purview of these studies.

Five categories of evidence of carcinogenic activity are used in the Technical Report series to summarize the strength of evidence observed in each experiment: two categories for positive results (**clear evidence and some evidence**); one category for uncertain findings (**equivocal evidence**); one category for no observable effects (**no evidence**); and one category for experiments that cannot be evaluated because of major flaws (**inadequate study**). These categories of interpretative conclusions were first adopted in June 1983 and then revised on March 1986 for use in the Technical Report series to incorporate more specifically the concept of actual weight of evidence of carcinogenic activity. For each separate experiment (male rats, female rats, male mice, female mice), one of the following five categories is selected to describe the findings. These categories refer to the strength of the experimental evidence and not to potency or mechanism.

- **Clear evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a dose-related (i) increase of malignant neoplasms, (ii) increase of a combination of malignant and benign neoplasms, or (iii) marked increase of benign neoplasms if there is an indication from this or other studies of the ability of such tumors to progress to malignancy.
- **Some evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a test agent-related increased incidence of neoplasms (malignant, benign, or combined) in which the strength of the response is less than that required for clear evidence.
- **Equivocal evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a marginal increase of neoplasms that may be test agent related.
- **No evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing no test agent-related increases in malignant or benign neoplasms
- **Inadequate study** of carcinogenic activity is demonstrated by studies that, because of major qualitative or quantitative limitations, cannot be interpreted as valid for showing either the presence or absence of carcinogenic activity.

For studies showing multiple test agent-related neoplastic effects that if considered individually would be assigned to different levels of evidence categories, the following convention has been adopted to convey completely the study results. In a study with clear evidence of carcinogenic activity at some tissue sites, other responses that alone might be deemed some evidence are indicated as "were also related" to test agent exposure. In studies with clear or some evidence of carcinogenic activity, other responses that alone might be termed equivocal evidence are indicated as "may have been" related to test agent exposure.

When a conclusion statement for a particular experiment is selected, consideration must be given to key factors that would extend the actual boundary of an individual category of evidence. Such consideration should allow for incorporation of scientific experience and current understanding of long-term carcinogenesis studies in laboratory animals, especially for those evaluations that may be on the borderline between two adjacent levels. These considerations should include:

- adequacy of the experimental design and conduct;
- occurrence of common versus uncommon neoplasia;
- progression (or lack thereof) from benign to malignant neoplasia as well as from preneoplastic to neoplastic lesions;
- some benign neoplasms have the capacity to regress but others (of the same morphologic type) progress. At present, it is impossible to identify the difference. Therefore, where progression is known to be a possibility, the most prudent course is to assume that benign neoplasms of those types have the potential to become malignant;
- combining benign and malignant tumor incidence known or thought to represent stages of progression in the same organ or tissue;
- latency in tumor induction;
- multiplicity in site-specific neoplasia;
- metastases;
- supporting information from proliferative lesions (hyperplasia) in the same site of neoplasia or other experiments (same lesion in another sex or species);
- presence or absence of dose relationships;
- statistical significance of the observed tumor increase;
- concurrent control tumor incidence as well as the historical control rate and variability for a specific neoplasm;
- survival-adjusted analyses and false positive or false negative concerns;
- structure-activity correlations; and
- in some cases, genetic toxicology.

**NATIONAL TOXICOLOGY PROGRAM TECHNICAL REPORTS
PEER REVIEW PANEL**

The members of the Peer Review Panel who evaluated the draft NTP Technical Report on GSM- and CDMA-Modulated Cell Phone RFR on March 26-28, 2018, are listed below. Panel members serve as independent scientists, not as representatives of any institution, company, or governmental agency. In this capacity, panel members have five major responsibilities in reviewing the NTP studies:

- to ascertain that all relevant literature data have been adequately cited and interpreted,
- to determine if the design and conditions of the NTP studies were appropriate,
- to ensure that the Technical Report presents the experimental results and conclusions fully and clearly,
- to judge the significance of the experimental results by scientific criteria, and
- to assess the evaluation of the evidence of carcinogenic activity and other observed toxic responses.

SUMMARY OF PEER REVIEW PANEL COMMENTS

NOTE: A summary of the Peer Review Panel's remarks will appear in a future draft of this report.

INTRODUCTION

GSM- AND CDMA-MODULATED CELL PHONE RADIO FREQUENCY RADIATION

Synonyms: Cell phone radio frequency radiation; mobile phone radio frequency radiation

OVERVIEW

All consumer cell phone devices function through the transmission of radio waves on a cellular network. The cellular network itself is composed of a collection of individual “cells” that include a fixed-location transceiver (a device that transmits and receives radio signals), also referred to as a cell tower. The collection of adjacent smaller “cells” in the cellular network enables cell phones and towers to use low-power transmitters, thereby allowing for the same frequencies to be reused in non-adjacent cells without interference. Together the individual “cells” comprise the cellular network that provides coverage over a large geographical area. In the United States, there are two major nation-wide cellular networks: CDMA (Code Division Multiple Access) and GSM (Global System for Mobile Communications). With technologies rapidly evolving to meet consumers’ increased demand for better coverage, increased call quality, faster data transfer rates, and increased accessibility, the terms CDMA and GSM tend to group together multiple, sometimes successive, technologies that are implemented by the service providers that maintain the two networks. In the United States, Sprint® and Verizon® use and maintain the CDMA network; AT&T® and T-Mobile® use and maintain the GSM network.

For both the GSM and CDMA networks, transmissions occur at specific radio wave frequencies, which are allocated and regulated by the Federal Communications Commission (FCC). While the transmission of radio signals occurs at the same frequencies for both networks, the networks differ in the method by which their signal is modulated. In telecommunications, modulation is a process of conveying a signal, like a cell phone user’s voice during a call,

inside another signal that can be physically transmitted. This process involves modulation of the signal prior to transmission at one end, and then demodulation at the other end. Because this process requires different technologies for CDMA and GSM, many cell phones are not interchangeable between the two networks and will only function on one or the other of the networks, not both.

The constantly evolving cellular technologies are commonly referred to by their successive generations (G). The first generation (1G) devices were analogue phones, as opposed to the digital phones of today. Digital voice systems of the second generation (2G) replaced the analogue system of 1G. At the time that these studies were being designed, 2G technology was the primary technology in use and 3G technologies were emerging. Therefore, the current studies were conducted using modulated signals that replicated the 2G and 3G technology in use at the time. Over the course of the studies, however, more advanced 4G technologies were developed. Currently, all of these technologies (2G, 3G, and 4G) are still actively in use for mobile communication applications. 2G and 3G are still the basis for voice calling applications, while 3G and 4G technologies were primarily developed to offer faster access to the internet. Some of the 3G technology is based on 2G technology. While 2G technology is being phased out in the United States, this technology will remain in use in other places throughout the world. More advanced and efficient technologies that are currently in development, such as 5G, will utilize higher frequencies than existing technologies.

RADIO FREQUENCY RADIATION (RFR)

In the context of this report, radio frequency (RF) radiation refers to the broad range of electromagnetic fields from 3 kilohertz (3 kHz) to 300 gigahertz (300 GHz). Different applications utilize different frequency bands within the RF portion of the electromagnetic spectrum. The range of frequencies for radio and television are in the 145 kHz to 850 MHz range. These include long, medium, shortwave, and very high frequency (VHF) radio transmissions and VHF and ultra-high frequency (UHF) over-the-air television transmissions. Wireless communications and networking typically utilize frequencies between 800 MHz and 6 GHz. Cell phone networks (2G, 3G, and 4G-LTE) utilize frequencies in the range of 600 MHz to 5.7 GHz. In the United States, wireless telecommunications networks and devices operate in bands at frequencies of nominally 800 MHz, 850 MHz, or 1,900 MHz for 2G; 850 MHz, 1,700 MHz, 1,900 MHz, or 2,100 MHz for 3G; and 600 MHz, 700 MHz, 800 MHz, 850 MHz, 1,700 MHz,

1,900 MHz, 2,100 MHz, 2,300 MHz, 2,500 MHz, 5,200 MHz, or 5,700 MHz for 4G. The next generation, i.e., the 5th generation of wireless communications, will also utilize the RFR spectrum above 6 GHz. Other terms are also used in the literature for part of the RFR spectrum, e.g., microwaves for frequencies above 1 GHz, millimeter waves for frequencies above 30 GHz.

CELL PHONES AND RFR

Cell phones and other commonly used wireless communication devices transmit their signals via RFR to enable voice calls and data transfer, including communication through the internet. Wireless phones are two-way radios that contain both a receiver and a transmitter. When a user makes a call, voice sound is converted into digital information. The information is imposed on to RFR and transmitted to the nearest base station. Base stations, commonly referred to as cell towers, have antennas placed on towers that are free standing or mounted on existing structures such as trees, water tanks, or tall buildings and contain electronic equipment and antennas that receive and transmit RF signals and form a bridge to the rest of the communications infrastructure. The base station receives and transmits radio signals in its area or “cell.” As the user moves around, the radio signal can be relayed within the communications network from one “cell” of coverage to another, maintaining call connection. The call is routed through the communications network either through a land line phone or another wireless phone again using radio signals. To conserve energy and minimize interference, mobile phones automatically regulate the RFR signal strength, and hence the emitted field, to the lowest power level possible for a connection to be made. However, in a poor transmission environment (caused by, e.g., a distant base station, presence of obstacles between the base station and the mobile phone, or interferences from adjacent calls), there is a higher output power and emission from the mobile phone in order to make a connection. Therefore, the better the connection, the lower the power output of the wireless device.

PROPERTIES OF CELL PHONE RFR

Cell phone RFR is a form of nonionizing electromagnetic energy that consists of propagating electromagnetic waves of oscillating electric (E-) and magnetic (H-) fields that move together through space at the speed of light. As opposed to ionizing radiation, which contains enough energy when passing through matter to break chemical bonds

or remove an electron from an atom or molecule to produce charged ions, nonionizing radiation refers to electromagnetic energy that at most only has sufficient energy for excitation of an electron to a higher energy state. Nonionizing radiation includes a broad range of the electromagnetic spectrum from extremely low frequency (ELF) radiation to radio and microwaves, infrared, visible light, and near ultraviolet radiation. It has a lower frequency and longer wavelength than ionizing radiation (Figure 1).

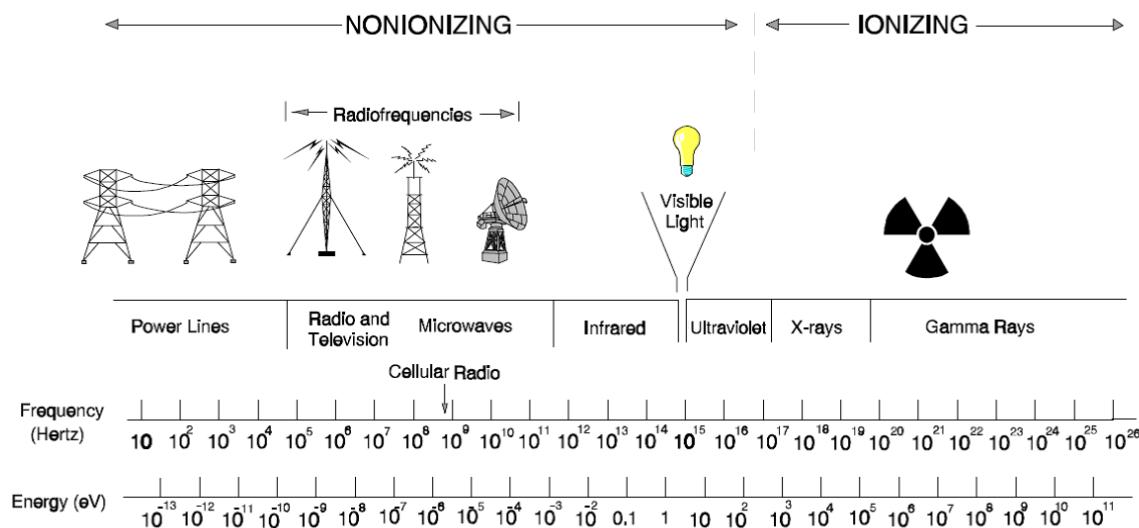


FIGURE 1
Electromagnetic Spectrum (OET, 1999)

Cell phone RFR fields transport large amounts of data at a very fast rate over long distances. RF waves are characterized by their wavelength (the distance covered by one complete cycle of the electromagnetic wave) and their frequency [the number of electromagnetic waves passing a given point in 1 second (sec)]. The frequency of an RF signal is expressed in terms of Hertz (Hz), where one Hz is equivalent to one cycle per second. The RF segment of the electromagnetic spectrum is generally defined as the frequencies between approximately 3 kHz to 300 GHz. The intensity of an RF field can be expressed by its electric and magnetic components and is measured in volts per meter (V/m) for electric fields and amperes per meter (A/m) for magnetic fields. Another measure of RFR is the power density, which is defined as the power per unit area and is expressed in watts per square meter (W/m^2) in the

far-field of sources. The quantity used to describe the amount of RFR energy absorbed by the body is referred to as the specific absorption rate (SAR), which is expressed in watts per kilogram (W/kg). SAR is a function of the geometry and the dielectric properties of biological tissues absorbing the energy (which result from the interaction of electromagnetic radiation with constituents at the cellular and molecular level), the square of the strength of the induced E-field, and the mass density of the exposed tissue. The SAR value is derived by averaging the absorbed energy over a specific volume (typically 1 gram, 10 grams, or the whole body for regulatory purposes).

Cell Phone RFR Signal Modulation

In wireless telecommunications, modulation is the process of conveying digital or analog signals or information (the message) by varying one or more parameters of another signal (the carrier), typically at a much higher frequency, that can be transmitted over a distance. The modulated carrier contains complete information about the message signal and the original message can be recovered by suitable signal processing of the signal when received at a remote location (base station). One of the main goals of the modulation used in mass wireless communications systems is to transfer as much data as possible in the least amount of spectrum. Over the years, multiple modulation techniques have emerged to achieve and improve spectral efficiency, either when considering a single user in isolation or multiple users simultaneously using the same spectrum.

Cell phone technology is typically referred to in “generations.” The first generation (1G) of wireless technology was an analog system that used analog frequency modulation for voice calls. The 1G networks were introduced in the 1980s and continued until they were replaced by networks of the second-generation (2G) networks. These networks differed from the 1G networks in that they were digital, provided encryption, were significantly more efficient, and introduced data services [i.e., text messages, picture messages, and Multimedia Message Service (MMS)] in addition to voice calls. The 2G networks became commercially available in 1992 and used three common multiple access technologies for accommodating multiple simultaneous users:

- Frequency Division Multiple Access (FDMA): the available spectrum is split into a number of distinct parts (channels) each large enough to accommodate a single user or call without overlap, all users utilize their channel 100% of the time for the duration of the call or message. The channels are normally of equal bandwidth;
- Time Division Multiple Access (TDMA): the available spectrum is allocated to a single channel, each user or call assigned a certain portion of time;
- Code Division Multiple Access (CDMA): the available spectrum is allocated to a single channel, each user or call is assigned a unique sequence code to spread the message over the available spectrum. All users use the whole of the spectrum all of the time. At the receiver, the same unique sequence code is used to recover the desired signal from the sum of all the user calls.

2G systems used a combination of FDMA/TDMA or CDMA, for example, GSM and cdmaOne (IS-95), respectively. While the 2G technology continues to operate, subsequent third and fourth generations of network technologies were introduced in 1998 (3G), 2006 (4G), and 2011 (4G-LTE). These technologies were developed to support increased data needs for multimedia access with increased bandwidth and transfer rates to accommodate internet-based broadband applications, including video conferencing, streaming video, sending and receiving faxes, and instantly downloading e-mail messages with attachments. With the introduction of 3G technology, “smartphones” were developed. With these devices, the newer technologies were overlaid with 2G to support multiple access modes (2G, 3G, and 4G) (Buddhikot *et al.*, 2009). Although the 2G technologies will be phased out over time and replaced by newer technologies, the current wireless communication networks continue to utilize 2G for voice and text.

All 3G systems utilize CDMA/WCDMA technology and fall into two groups complying with the 3rd Generation Partnership Project (3GPP) or 3GPP2 family of standards. Universal Mobile Telecommunications Service (UMTS), Wideband Code Division Multiple Access (WCDMA), and Time Division-Synchronous Code Division Multiple Access (TD-SCDMA) are 3GPP variants, CDMA2000 (which is based on 2G cdmaOne) is 3GPP2. 4G systems use Orthogonal Frequency Division Multiplexing (OFDM) within the E-UTRAS (LTE-Advanced) or Worldwide Interoperability for Microwave Access (WiMAX) standards.

Modulation Schemes (GSM and CDMA)

The Global System for Mobile Communications (originally *Groupe Spécial Mobile*; GSM) was developed to establish a digital standard for compatibility throughout Europe. GSM is a circuit-switched system that uses both FDMA and TDMA technologies. The frequency division mechanism divides the GSM band into 200 kHz-wide channels. The time division mechanism enables up to eight time slots (voice channels) per frequency channel wherein a single cell phone transmits in only one out of eight available time slots during a voice communication. This introduces a pulsed signal shape with a pulse repetition rate of 217 Hz. Such a TDMA frame has a length of 4.6 milliseconds (ms), and 26 TDMA frames make up a multiframe with a 120 ms duration. During a multiframe, a mobile phone transmits in 25 out of 26 possible time slots. This TDMA frame structure causes significant low frequency amplitude modulation components to be superimposed on the RF carrier at 8.3 and 217 Hz.

With GSM, the duplexing between uplink (when the handset transmits to the base station) and downlink (when the base station transmits to the handset) is implemented in the frequency and time domain. Constant frequency spacing is maintained between up and downlink frequencies: in the United States the uplink is 1,850 to 1,910 MHz, and the downlink 1,930 to 1,990 MHz. The uplink and downlink frequencies are chosen according to the cell (area that is covered by a base station) into which the mobile is registered. In order to minimize interference between neighboring cells, a frequency reuse policy is applied. In this approach, when a mobile phone moves from one cell into an adjacent cell, frequencies used for data uplink and downlink change in association with this movement (i.e., transmission frequencies change at handover from one cell to another).

GSM technology implements a power control in order to increase the battery life of mobile handsets. The power control has a dynamic range of 30 decibel (dB) subdivided into 2 dB power-level steps. The power control is typically implemented using the Slow Associated Control Channel (SACCH), which facilitates a power control update rate no faster than every four multiframes (480 ms). Once a target power level is received, the mobile station is able to regulate its power in 2 dB steps every 60 ms. This means that a power regulation over 15 steps (full dynamic range) takes 900 ms. GSM base stations typically average the received signal strength from a mobile phone over 1 second, such that the actual power regulation usually takes place after multiples of 480 ms.

The GSM supports data transfer speeds up to 9.6 kilobits/second, allowing the transmission of basic data services such as Short Message Service (SMS), but not large packets of data such as internet access and streaming video.

CDMA technology uses a form of coded transmission known as Direct Sequence Spread Spectrum (DSSS) in which data multiplies by a much faster pseudo random code before being modulated on to the carrier. The effect of the multiplication is to spread the message across all frequency bands available for use at any time but with very specific characteristics. CDMA signal access technology is based on code division separation of mobile stations as well as base stations. This implies strong differences of the signal structure compared to GSM. For example, in IS-95 in the forwardlink (downlink), a set of 64 Walsh codes (which are deterministic and orthogonal) are applied to spread/separate the individual channels in the downlink of a cell. After the orthogonal spreading, a short (16-bit) Pseudo Noise (PN) code is applied to further spread the signal and identify the cell. Hence, a separation of neighboring cells in the frequency domain is no longer necessary. Eventually, there is no need for the mobile station to change its transmission frequency during the transition from one cell into another. As with GSM systems, the duplexing between the forward and reverselinks is implemented in the frequency domain. In CDMA systems, an efficient power control is crucial. Because all mobile stations transmit and interfere in the same frequency channel, each mobile device decreases the signal to noise ratio of all the other mobile devices. Hence, the output power of a mobile phone should be kept at a minimum that guarantees good transmission quality. On the other hand, when moving around, the mobile device is subject to slow and fast fading, shadowing, external interference, etc. In order to keep the signal received at the base station constant and compensate for effects on the communication channel, a fast power control is necessary. Therefore, when a CDMA mobile station is active (communicating), a closed-loop power control is applied. The base station monitors the signal quality in the reverse link and inserts power-control bits in the communication channel. For example, in Interim Standard 95 (IS-95), the power control over a dynamic range of 48 dB in 1 dB steps with an update rate of 800 Hz is implemented. The power control is implemented by sending a binary value of “1” to regulate the transmit power 1 dB down, and “0” to regulate the transmit power 1 dB up. A quasistatic power level is therefore implemented by an alternating 0101 power-control pattern.

IS-95, also known as cdmaOne, was developed by Qualcomm (San Diego, CA) as the first 2G CDMA-based digital cellular technology. The term IS-95 generally applies to a protocol revision (P_REV=1) that was adopted as a

standard (TIA-EIA-95) by the Telecommunications Industry Association (TIA) in 1995. Over time, subsequent iterations of the IS-95 protocol such as IS-95A, TSB-74, and IS-95B were developed, each with incremental improvements over the previous protocols. Later, more advanced versions of the CDMA technology have evolved to include IS-2000, which incorporated much higher transfer rates than the previous 2G versions.

SOURCES, USE, AND HUMAN EXPOSURE

The predominant source of cell phone RFR for the majority of the population is in telecommunications and mobile internet access applications for wireless devices. Aside from telecommunications, there are other man-made applications of RFR, which include microwave ovens, radar, industrial heating and sealing, medical diagnostics [Magnetic Resonance Imaging (MRI)] and therapy (surgical diathermy and ablation), and remote tracking or detection of objects [anti-theft, Radio frequency Identification (RFID)]. However, there are also natural sources of RFR such as atmospheric electrical discharges (lightning) and solar and cosmic radiation. RFR exposures from natural sources are much smaller and tend to be spread over a much wider range of frequencies compared to man-made fields (IARC, 2013).

Highest human exposure to cell phone RFR primarily occurs through the use of cellular phone handsets and other wireless devices held in closest proximity of the human body such as tablets and laptop computers. The use of cell phones has become widespread over the last two decades amongst adults and children, thereby increasing the level of RFR the population is exposed to. Concern has been expressed regarding the potential health risks associated with use of cell phones. Particularly, there has been a great deal of focus on the possibility of increased risk of brain cancer because traditionally these devices were used in close proximity (0 to 2 cm) to the head, yet the advent of smart phones has altered dramatically the usage scenarios for such devices away from a simple phone call. The RFR exposure of a person is defined in terms of SAR, the power absorbed in the body, because the body has complex geometry and tissue distributions, and even exposure to uniform RFR electromagnetic fields (EMF) will result in nonuniform SAR distributions. In general (apart from the case when very close to the antenna), the level of RFR exposure by a cell phone is inversely proportional to the square of the distance of the body from the device's antenna, and the highest SAR levels occur in the parts of the body nearest to the antenna. Accordingly, there is a very nonuniform exposure to cell phone RFR across the whole body of cell phone users and even of bystanders.

Accurate and detailed estimation of cell phone RFR exposure in humans is difficult to obtain because the output power of wireless devices constantly varies depending on several factors. Overall, the network carrier adjusts the output power of each connected device to the lowest level that is still compatible with a good quality signal. This adaptive power control occurs continuously and is achieved by a logarithmic downscaling of the time-averaged power from the maximum of 0.125 and 0.25 W to a level as low as 1 mW. When a device is in use, the output power (and subsequent exposure to cell phone RFR) from the device is increased compared to the output from that same device in “standby” mode. Therefore, levels of exposures are related to the amount of active time a user spends on the device. The output power of a device changes based on the signal received at the base station. Decreases in signal strength result in higher output powers. Therefore, there are increases in the output power as the distance between the device and the base station increases, if there are physical obstacles between the device and the base station, multiple reflections, and during handovers in the case of GSM (handover is the passing of a call from one base station to another when the user moves across the borders of cells or by network request to optimize communication traffic). The proximity of the device to the body and the type, number, and position of antennas in the device are other important factors affecting the amount of exposure to cell phone RFR.

Potential exposure to cell phone RFR also occurs from the cell phone towers (or base stations) that form the network. While modern towers emit substantially more power than devices, exposures from base station antennas are considerably lower in users than from the handheld device. Typically, base station antennas are placed at heights of 50 to 200 feet, in order to adequately cover an area (or cell). The antennas direct RF energy toward the horizon, with some downward tilt. As with all forms of radiation (ionizing and nonionizing), the RF energy level decreases rapidly as the distance from the antenna increases. As a result, the level of exposure to cell phone RFR at ground level is very low compared to the level close to the antenna. Overall, the exposure level from base stations is very small compared to exposure from the handheld devices.

Some base station antennas are installed on rooftops and at the top of lamp poles that are in close proximity or adjacent to office space and residential buildings. Levels of exposure from these sources can approach or exceed Federal Communications Commission (FCC) safety guidelines. Occupational exposure occurs during maintenance

on base stations. As a result, the FCC established guidelines for occupational exposures. Safety guidelines and regulatory compliance are discussed below.

The levels of cell phone RFR inside buildings with base station antennas mounted on the roof or on the side of the building are typically much lower than the level outside, depending on the construction materials of the building. Wood or cement block reduces the exposure to cell phone RFR by a factor of about 10. Due to the directional nature of the signals, the energy level behind an antenna is orders of magnitude lower than in front of the antenna.

According to a Pew Research poll (Pew, 2017), approximately 95% of adult Americans own a cell phone. As of December 2015, the number of active wireless subscriber connections was 377.9 million, which exceeded the population of the United States (CTIA, 2017). According to the same survey, 49.3% of households in the United States utilize only a wireless phone, and not a landline.

Safety Guidelines for Exposure

The Federal Communications Commission (FCC) and U.S. Food and Drug Administration (FDA) are jointly responsible for the regulation of wireless communication devices.

Federal Communications Commission

The FCC is required by its responsibilities under the National Environmental Policy Act of 1969 to evaluate the impact of emissions from FCC-regulated transmitters on the quality of the human environment (42 USC §4321 *et seq.*). As a result, the FCC regulates both the wireless devices as well as the base stations that form the cells of the network. Since 1996, the FCC has required that all wireless communications devices (transmitting in the 100 kHz to 6 GHz frequency range) sold in the United States comply with its minimum guidelines for safety and maximum RFR absorption standards based on SAR. The FCC requires a formal approval process for all devices sold in the United States. FCC approval is contingent on the demonstration that the device does not exceed the maximum allowable SAR level when the device is operating at its maximum power. The SAR limit adopted by the FCC for exposure in the general population is 0.08 W/kg, as averaged over the whole body, and a peak spatial-average SAR of 1.6 W/kg, averaged over any 1 gram of tissue (47 CFR §1.1310) when averaged over 6 minutes.

Exceptions are made for the extremities (hands, wrists, feet, ankles, and pinnae), where the peak spatial-average SAR limit is 4 W/kg, averaged over any 10 grams of tissue for an exposure period of no longer than 30 minutes. For occupational exposures, the whole-body SAR limit is 0.4 W/kg, and the limit for the peak spatial-average SAR is 8 W/kg, averaged over any 1 gram of tissue. For the hands, wrists, feet, ankles, and pinnae, the peak spatial-average SAR limit for occupational exposure is 20 W/kg, averaged over any 10 grams of tissue for an exposure period not to exceed 6 minutes.

The FCC rules and guidelines for cell phone RFR exposure are based upon standards initially developed by the Institute of Electrical and Electronics Engineers (IEEE) and the National Council on Radiation Protection and Measurements (NCRP). These standards for RF exposure in workers and the general population are based on protection against adverse effects that might occur due to increases in tissue or body temperature in excess of 1° C (wbSAR, approximately 4 W/kg) or less (after applying safety factors). Because RF-energy absorption and any induced effects are dependent on the frequency of incident-field parameters and the composition of exposed tissues, it has been suggested that quantifying SARs in small averaging regions is more relevant for evaluations of human health effects.

Food and Drug Administration

The FDA does not currently regulate the use of wireless communications devices or the devices themselves. The FDA also does not require safety evaluations for radiation-emitting wireless communication devices. It does maintain the authority to take regulatory action if it is demonstrated that exposure to the emitted cell phone RFR from these devices is hazardous to the user.

ABSORPTION OF CELL PHONE RFR

RFR interacts with the human body via inductive or capacitive coupling or a combination of both. The absorption of the coupled RFR is dependent on the frequency of the signal and the dielectric properties of the exposed tissue. It generates oscillating currents in the tissue, which in turn give rise to induced E-fields. The energy is transferred into molecular motion of polar molecules like water, a strongly dipolar molecule and major component of biological tissues. Resonant oscillations in polar subgroups of cellular macromolecules are damped by collisions with

surrounding water molecules that disperse the energy of the RF signal into random molecular motion. Tissue heating occurs as the energy is transferred to the surrounding aqueous environment as heat (IARC, 2013).

The SAR (W/kg) is a measure of the absorption of RF energy by biological tissues. It is a function of several main factors: the electrical conductivity (Siemens/meter) of the tissue, the square of the strength (Volts/meter) of the induced E-field, and the geometry and mass density (kg/meter³) of the tissue absorbing the energy. The SAR is calculated as the average of the absorbed power over a specific volume of tissue (typically 1 or 10 gram volume of tissue or the whole body).

TOXICITY

A comprehensive review of the toxicity of cell phone RFR in *in vitro* models, laboratory animals, and humans was recently conducted and published in the International Agency for Research on Cancer (IARC) Monograph series (IARC, 2013).

Thermal Effects

Given the ability of cell phone RFR to heat tissues, the toxic effects of cell phone RFR are often classified as thermal or nonthermal effects, based on whether the observed effect was a result of a significant temperature change (thermal effects) or independent of any change in temperature considered in excess of thermal noise (nonthermal effects). The most well-established and biologically plausible mechanism for cell phone RFR-induced effects in biological systems is through tissue heating resulting in damage. It has been well established that excessive heating causes significant damage to cells, tissues, and organs. At high enough levels of cell phone RFR exposure, the absorption of energy could lead to increased heating to the point that it overwhelms an organism's ability to thermoregulate and maintain an acceptable body temperature. Because human exposures to cell phone RFR occur at intensities that are not expected to cause thermal effects, the nonthermal effects are more appropriate to the evaluation of effects in humans.

Nonthermal effects refer to biological changes that occur with body temperature increases that are below 1° C.

Changes of temperature up to 1° C are considered in the range of thermal noise (IARC, 2013). There is an ongoing debate regarding whether nonthermal biological effects can occur as a result of exposures to low-intensity cell phone RFR. It has been suggested that there is no plausible nonthermal mechanism by which exposure to low-intensity RFR could induce significant biological effects (Adair, 2003; Prohofsky, 2004; Sheppard *et al.*, 2008). However, there are numerous reports of specific biological effects associated with cell phone RFR exposures at levels considered below those expected to result in a measurable amount of tissue heating. Other than tissue heating, the mechanisms of interaction between cell phone RFR and biological systems have not been well characterized, but several mechanisms have been proposed for these nonthermal effects in biological systems, including the generation of reactive oxygen species, induction of ferromagnetic resonance, demodulation of pulsed RF signals, and the alteration of ligand binding to hydrophobic sites in receptor proteins (IARC, 2013). Additionally, low levels of exposure to cell phone RFR may result in small temperature changes in localized areas of exposed tissues that cause conformational changes in temperature-sensitive proteins and induce the expression of heat-shock proteins.

Experimental Animals

Toxic effects have been reported in various types of studies in cell phone RFR-exposed laboratory animals and *in vitro* systems. Most studies investigating the potential toxicity of cell phone RFR have focused primarily on genotoxicity and related effects. These findings are summarized in the Genetic Toxicity section. However, several studies have been conducted to evaluate other aspects of toxicity, including specific studies on gene and protein expression, immunotoxicity, and permeability of the blood-brain barrier. The results of these studies have been mixed. It is important to note that these studies were conducted with cell phone RFR of differing parameters (frequency, power density, continuous wave versus amplitude-modulated signals, etc.). Because there may be differences in cell phone RFR-induced responses depending on the frequency, modulation, and power density, it is not surprising that the results reported in the literature can be somewhat inconsistent.

Several effects on the humoral and cell-mediated responses of the immune system have been reported at various frequencies of cell phone RFR in rats and mice. These include effects on the activity of NK cells, plaque-forming cell response to sheep erythrocytes, production of tumor necrosis factor (TNF) in peritoneal macrophages and

splenic T-cells, mitogenic response in T lymphocytes, phagocytic activity of neutrophils, leukocyte profile, and thymic and splenic cellularity (Smialowicz *et al.*, 1983; Guy *et al.*, 1985; Veyret *et al.*, 1991; Novoselova *et al.*, 1999; Lushnikov *et al.*, 2001; Kolomytseva *et al.*, 2002). However, many of these effects were observed in studies conducted with cell phone RFR at frequencies greater than 10 GHz. Other studies have demonstrated no exposure-related effects on the immune system (Elekes *et al.*, 1996; Chagnaud and Veyret, 1999; Lushnikov *et al.*, 2001; Gatta *et al.*, 2003; Nasta *et al.*, 2006).

A few studies have investigated the impact of cell phone RFR at frequencies between 800 and 1,900 MHz on gene and protein expression. Several studies have demonstrated that cell phone RFR can alter the expression of certain genes in the brain (Fritze *et al.*, 1997; Belyaev *et al.*, 2006; Nittby *et al.*, 2008), while others have failed to associate cell phone RFR exposure with changes in gene expression (Stagg *et al.*, 2001; Paparini *et al.*, 2008). The expression of various proteins has also been investigated in rats and mice. These studies have primarily yielded negative results for the specific proteins being evaluated in the rat brain (Fritze *et al.*, 1997; Belyaev *et al.*, 2006; Ammari *et al.*, 2008, 2010; Dasdag *et al.*, 2009). Similarly, no effects of cell phone RFR on protein expression have been reported in the testis (Lee *et al.*, 2010) or in the skin (Masuda *et al.*, 2006; Sanchez *et al.*, 2006, 2008). Changes in the expression of bone morphogenic protein and bone morphogenic protein receptors have been reported in the kidney of newborn rats (Pyrgasopoulou *et al.*, 2004). A study by Eşmekaya *et al.* (2010) also demonstrated increased expression and activity for caspase 3 and caspase 9 in the thyroid gland of Wistar rats.

Exposure to cell phone RFR induces changes in markers for oxidative stress in multiple tissues, including the brain (Ilhan *et al.*, 2004; Meral *et al.*, 2007; Ammari *et al.*, 2008; Sokolovic *et al.*, 2008; Imge *et al.*, 2010), heart (Ozguner *et al.*, 2005a), kidney (Oktem *et al.*, 2005; Ozguner *et al.*, 2005b), eye (Ozguner *et al.*, 2006), liver (Ozgur *et al.*, 2010; Tomruk *et al.*, 2010), endometrium (Oral *et al.*, 2006; Guney *et al.*, 2007), and testis and epididymis (Mailankot *et al.*, 2009). A few studies have also demonstrated cell phone RFR-mediated effects on differentiation and apoptosis in the endometrium (Oral *et al.*, 2006; Guney *et al.*, 2007) and brain (Dasdag *et al.*, 2009; Sonmez *et al.*, 2010). Changes have also been noted in the permeability of the blood-brain barrier in some studies (Eberhardt *et al.*, 2008; Nittby *et al.*, 2009, 2011). However, other studies conducted under similar experimental conditions

failed to demonstrate any effect of cell phone RFR exposure on the permeability of the blood-brain barrier (Grafström *et al.*, 2008; de Gannes *et al.*, 2009; McQuade *et al.*, 2009; Masuda *et al.*, 2009).

Humans

Numerous epidemiology studies have been conducted to investigate the association between exposure to cell phone RFR and health effects in humans. However, many of these studies were conducted in small groups exposed to cell phone RFR signals with different characteristics (frequencies, modulations, intensities, etc.) than the specific frequency bands and modulated cell phone RFR signals used in wireless communication. Many of these studies evaluate microwaves, extremely low frequency (ELF) fields, and radar, which are all different forms of RFR. While these studies may provide additional data for the evaluation of the toxicity of RFR in general, a smaller subset of these studies, which specifically evaluated cell phone RFR at the frequencies and modulations used in wireless communications, is more critical to evaluating the potential toxicity of cell phone RFR from mobile communication devices.

There is a very limited set of research investigating the general toxicity of cell phone RFR in humans because most of the focus for research has been on the potential for carcinogenic effects. Studies in humans have failed to demonstrate any consistent adverse health effects in cell phone RFR-exposed populations. There are reports of some exposed individuals that complain of acute, subjective effects following exposure to cell phone RFR, including headaches, fatigue, skin itching, and sensations of heat (Frey, 1998; Chia *et al.*, 2000; Hocking and Westerman, 2000; Sandström *et al.*, 2001; Santini *et al.*, 2002a,b). However, these have primarily been reported in people that consider themselves electrosensitive, and not in the general population. It has been suggested that there are likely other causes, not cell phone RFR, for these subjective symptoms (Kwon and Hääläinen, 2011). In fact, the validity of electrosensitivity as an actual phenomenon has been questioned and debated. Variable results have been observed in the electroencephalogram (EEG) of volunteers exposed to RFR during sleep. Some studies indicate that exposure to cell phone RFR induces changes in sleep latency and sleep EEG (Mann and Röschke, 1996; Wagner *et al.*, 1998, 2000; Borbély *et al.*, 1999; Huber *et al.*, 2000, 2002, 2003; Loughran *et al.*, 2005; Hung *et al.*, 2007; Regel *et al.*, 2007; Lowden *et al.*, 2011). Glucose metabolism in the brain, a marker for brain activity, is increased

in the region of the brain closest to the antenna (Volkow *et al.*, 2011). While these results demonstrate exposure-related effects, the toxicologic significance of these findings is unclear.

No effects of cell phone RFR on the neuroendocrine system, auditory and vestibular systems, or consistent effects on cognitive performance have been reported in humans. There is also no clear evidence of effects on heart rate or blood pressure.

CARCINOGENICITY

The carcinogenic potential of cell phone RFR in animals and humans is controversial. A comprehensive review of the carcinogenicity of cell phone RFR in laboratory animals and humans was recently conducted and published in the IARC Monograph series (IARC, 2013).

Experimental Animals

Studies published to date have not demonstrated consistently increased incidences of tumors at any site associated with exposure to cell phone RFR in rodents (Lin, 2017). No increases in tumor incidences were observed in B6C3F1 mice exposed to GSM-modulated cell phone RFR for 24 months (Tillmann *et al.*, 2007), F344 rats exposed to CDMA-modulated cell phone RFR for 24 months (La Regina *et al.*, 2003), or Wistar rats exposed to GSM-modulated cell phone RFR for 24 months (Smith *et al.*, 2007). In studies conducted in transgenic and tumor-prone mouse strains, exposure to cell phone RFR has not been consistently associated with an increased incidence of tumors at any site (Utteridge *et al.*, 2002; Sommer *et al.*, 2004, 2007; Oberto *et al.*, 2007; Lee *et al.*, 2011). While these studies have advanced the knowledge of the potential toxicity of cell phone RFR, critical limitations in the design of many of these studies severely limit the utility of the information to adequately evaluate the carcinogenicity of cell phone RFR. These limitations include studies with very short daily exposure durations (≤ 2 hours per day) in heavily restrained animals or with levels of cell phone RFR exposures too low to adequately assess carcinogenic potential. The focus of many of these studies conducted in genetically altered and tumor-susceptible mice was not to evaluate the overall carcinogenicity of cell phone RFR, but to investigate the effects in the specific predisposed tissues in that model.

Based on the constraints in the designs of the existing studies, it is difficult to definitively conclude that these negative results clearly indicate that cell phone RFR is not carcinogenic. To adequately evaluate the potential chronic toxicity and carcinogenicity of cell phone RFR, further studies with enhanced study designs and improved exposure paradigms were needed.

Humans

As a result of the IARC review conducted in 2011, RF electromagnetic fields were classified as possibly carcinogenic to humans (Group 2B). This classification was based on limited evidence of carcinogenicity in humans based on positive associations between exposure to RFR from wireless phones and increased risk for gliomas and acoustic neuromas, specifically in users with the greatest amount of cell phone usage. The IARC Working Group acknowledged that the findings were affected by potential selection and information bias, weakness of associations, and inconsistencies between study results (IARC, 2011).

While several other studies were considered, the IARC evaluation was based primarily on reports from the INTERPHONE Study, the largest research effort conducted to date examining the potential association between exposure to cell phone RFR and cancer in humans. INTERPHONE was an IARC-coordinated research effort that included a series of studies conducted with a common core protocol at 16 study centers in 13 countries: Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, and the United Kingdom (Cardis *et al.*, 2007). The studies were specifically designed to investigate the association between cell phone RFR and tumors of the brain (glioma and meningioma), acoustic nerve (schwannoma), and parotid gland. The final report for the INTERPHONE studies was published in 2011 (IARC, 2011).

The results of these studies seemingly demonstrated an elevated risk of glioma and acoustic neuroma in the group in the highest decile for exposure (cumulative phone call time). However, the INTERPHONE study group concluded that recall and selection biases and implausible values for usage reported by the participants in the study may explain the increased risk (INTERPHONE Study Group, 2010, 2011). Further, the INTERPHONE studies and other published epidemiological studies may have been concluded prior to the potential lag time (the interval between the

time of the onset of exposure and the subsequent development of a tumor) for the development of slow-growing brain tumors. Overall, the authors of these studies concluded that there was no significant increase in risk of glioma, meningioma, or acoustic neuroma associated with the use of cell phones.

Other studies have compared time trends in cell phone usage and the incidences of different types of cancers to investigate indirect evidence of an association between cell phone RFR and cancer. These studies were conducted across several different countries (Saika and Katanoda, 2011), and in a group of European countries (Lönn *et al.*, 2004a; Nelson *et al.*, 2006; Röösli *et al.*, 2007; Deltour *et al.*, 2009; de Vocht *et al.*, 2011), the United States (Muscat *et al.*, 2006; Propp *et al.*, 2006; Inskip *et al.*, 2010), Japan (Nomura *et al.*, 2011), New Zealand (Cook *et al.*, 2003), and Israel (Czerninski *et al.*, 2011). Overall, the evaluations suggest that there was no significant change in the trends of cancer incidences. Any minor increases in cancer rates that were observed in these studies were attributed to enhanced detection capabilities for cancer that were the result of advances in diagnostic medical equipment, like computerized tomography (CT) scans and MRI.

Several cohort studies have been conducted, but also failed to establish a clear association between cell phone RFR and the development of any of the investigated cancer types (Johansen *et al.*, 2001; Schüz *et al.*, 2006, 2011).

Additional studies have demonstrated that there was no association between cell phone usage and pituitary gland tumors (Takebayashi *et al.*, 2008; Schoemaker and Swerdlow, 2009), testicular tumors (Schüz *et al.*, 2006; Hardell *et al.*, 2007a), parotid gland tumors (Hardell *et al.*, 2004; Lönn *et al.*, 2006), uveal melanoma in the eye (Schüz *et al.*, 2006; Stang *et al.*, 2009), and cutaneous melanoma (Hardell *et al.*, 2011). Some studies have demonstrated that there was no association between cell phone usage and leukemia (Johansen *et al.*, 2001; Schüz *et al.*, 2006) and non-Hodgkin's lymphoma (Hardell *et al.*, 2005), whereas others have reported increased risk of non-Hodgkin's lymphoma (Linet *et al.*, 2006) and leukemia (Kaufman *et al.*, 2009).

Many of the epidemiological studies that have been published are limited in their ability to definitively establish a causal association between cell phone usage and increased cancer incidences due to recall and selection bias, confounding factors, and low study participation.

As mentioned previously, the utility of human studies with regard to evaluation of the carcinogenic potential of cell phone RFR is dependent upon the length of time the subjects in the studies were exposed to cell phone RFR. Given the long latency period between the initiation of exposures and the development of tumors, a sufficient duration of exposure must be reached in order to evaluate the association between exposure and cancer outcome. Because widespread usage did not occur until the 1990s in some countries, these populations may not have been exposed long enough to expect any changes in cancer incidences compared to studies in populations where widespread use occurred five or more years earlier in the late 1980s.

GENETIC TOXICITY

Extensive reviews of the literature on the genotoxicity of various frequencies and modulations of cell phone RFR, covering experimental systems ranging broadly from cell-free DNA preparations to cells of exposed animals and humans, have concluded that evidence for cell phone RFR-associated genotoxicity is inconsistent and weak (Brusick *et al.*, 1998; Verschaeve *et al.*, 2010; Repacholi *et al.*, 2012; Vijayalaxmi and Prihoda, 2012). Interpretations of the genotoxicity studies and the ability to draw definitive conclusions based on weight-of-evidence from the large number of studies that have been reported have been hampered by inadequacies in experimental design, especially related to exposure standards and radiation-measuring procedures (Brusick *et al.*, 1998). Although the majority of studies report a lack of effect, the several reports of a positive response are concentrated among experiments assessing chromosomal or DNA damage in mammalian cell systems *in vitro* and *in vivo*. Some key studies reporting cell phone RFR-associated genotoxicity in human cell lines, including DNA damage and chromosomal effects, could not be replicated (Speit *et al.*, 2007, 2013). A critical complicating factor in the study of the genotoxic effects of cell phone RFR is that under certain conditions, cell phone RFR is sufficiently energetic to heat cells and tissues, and not all studies have considered this factor in their design. Heating of cells *in vivo* and *in vitro* has produced positive results in tests for genotoxicity, such as the comet assay and micronucleus assay (Asanami and Shimono, 1997; Komae *et al.*, 1999; Speit and Schütz, 2013). The mode of action whereby heat induces these effects may be through induction of protein denaturation and aggregation, which can interfere with chromatin structure and slow the kinetics of DNA repair or interfere with mitosis by disrupting microtubule function (Kampinga and Dikomey, 2001; Hunt *et al.*, 2007). Thus, heat-induced increases in DNA migration seen in the comet assay may reflect slowed repair of endogenous lesions, and similarly, activity in the micronucleus assay may

be due to aneugenic rather than clastogenic events (Asanami and Shimono, 1997; Komae *et al.*, 1999; Speit and Schütz, 2013). Therefore, it is important to distinguish between nonthermal and thermal conditions when studying measures of genotoxicity following exposure to cell phone RFR.

STUDY RATIONALE

The FDA nominated cell phone RFR emissions of wireless communication devices for toxicology and carcinogenicity testing. Current exposure guidelines are based on protection from acute injury from thermal effects and little is known about the potential for health effects from long-term exposure to RFR below the thermal hazard threshold. Epidemiology studies that have been conducted to date have not demonstrated a causal link between cell phone RFR and any health problems in humans, however the results of these studies are complicated by confounding factors and potential biases. Additionally, exposures in the general population may not have occurred for a long enough period to account for the long latency period of some types of cancers in humans. Similar to the challenges faced in epidemiological studies, studies in laboratory animals have been complicated by limitations that researchers have faced in conducting robust studies designed to characterize the toxicity and carcinogenicity of cell phone RFR.

For years, the primary concern regarding the potential health risk of chronic exposure to cell phone RFR was brain cancer based on the proximity of wireless devices near the head during use. While the brain is an organ of concern, understanding the potential toxicity and carcinogenicity of whole-body exposure is critical. Cell phone RFR is constantly emitted from wireless devices to communicate with base stations, regardless of whether the user is on a call or not. As the public has become more aware of the uncertainty regarding the potential effects of cell phone RFR on the brain, more emphasis has been placed on the use of wired or wireless headsets (like Bluetooth), which minimize cell phone RFR exposure to the head. In recent years, the density of cell towers has increased to cope with the increasing demand for capacity, resulting in installations closer to residential neighborhoods and schools. Additional cell phone RFR technologies, like SmartMeters used by power companies, transmit data in real time using cell phone-type RFR. These existing and emerging technologies may potentially increase the level of exposures in human populations. These and other additional sources also expose different parts of the body, not only the head.

In 2011, cell phone RFR was classified by the IARC as possibly carcinogenic to humans based on limited evidence of an association between exposure to cell phone RFR from heavy wireless phone use and glioma and vestibular schwannoma (acoustic neuroma) in human epidemiology studies and limited evidence for the carcinogenicity of cell phone RFR in experimental animals (IARC, 2013). While ionizing radiation is a well-accepted human carcinogen, theoretical arguments have been raised against the possibility that nonionizing radiation could induce tumors (discussed in IARC, 2013). Given the extremely large number of people who use wireless communication devices, even a very small increase in the incidence of disease resulting from exposure to the cell phone RFR generated by those devices would translate to a large number of affected individuals, which would have broad implications for public health. Due to the exposure and use pattern of cell phones by pregnant women and women of childbearing age, perinatal exposure was selected for use in these studies.

MATERIALS AND METHODS

OVERVIEW

The establishment of the National Toxicology Program (NTP) research program on radio frequency radiation (RFR) has required the coordination of expertise from multiple scientific and engineering disciplines. At the initiation of the RFR research program, a collaboration was established with technical experts from the Radio-Frequency Fields Group in the Radio Frequency (RF) Technology Division, which is part of the Communications Technology Laboratory (CTL) at the National Institute of Standards and Technology (NIST, Boulder, CO). NIST evaluated the existing exposure systems and identified the types of improvements that would be required to provide a system of sufficient size and power to conduct robust toxicology and carcinogenicity studies with uniform RFR exposures in unrestrained, individually housed animals for a minimum of 6 hours a day at frequencies and modulations that reflected those in use at the time. The design of the chambers and toxicology studies required special consideration of logistical, financial, and engineering limitations.

NIST tested the feasibility of a reverberation chamber-type exposure system by conducting a series of studies on field strengths, field uniformity, and power requirements under various conditions of RFR exposure in such chambers. These studies provided critical information for the design of experimental studies with respect to the number of cages that could be placed in specific size chambers, the arrangement of cages within each chamber, and the input power requirements.

NTP also worked with the Foundation for Research on Information Technologies in Society (IT'IS, Zurich, Switzerland), which conducted studies using computational models that simulated RFR dosimetry to provide estimates of whole-body and organ-specific internal field strengths and specific absorption rates (SARs) during exposure. Based on information and parameters obtained during the NIST feasibility studies, IT'IS built a prototype reverberation chamber as the basis for an exposure system to study health effects of long-term exposure of

laboratory animals. Following completion, NIST evaluated the prototype exposure chamber to determine if it met the requirements specified by the NTP.

After prototype-testing by IT'IS Foundation and NIST, the IT'IS Foundation built the reverberation chambers required for the NTP RFR exposure facility. Chambers were installed at the Illinois Institute of Technology (IIT) Research Institute (IITRI, Chicago, IL). Following the installation and initial testing of the exposure system by IT'IS and IITRI, technical experts from NIST conducted an independent validation of the system. NIST confirmed that the probe readings in the system were consistent, that field uniformity was within expected specifications, and that the signal quality was acceptable. NIST performed additional evaluations prior to initiation of the 2-year studies and after completion of the studies to determine if any changes occurred in the signal quality, field uniformity, or consistency of in-chamber field measurements. All studies were conducted at IITRI with real-time monitoring of the system performance at IT'IS Foundation.

Institution	Role
National Institute of Standards and Technology (NIST) (Boulder, CO)	Suggested reverberation chamber exposure system Conducted feasibility studies for reverberation chambers Established various technical parameters for chambers Evaluated the prototype chamber built by IT'IS Foundation Validated the system prior to the conduct of studies at IITRI Reevaluated RFR exposures prior to and after 2-year studies
IT'IS Foundation (Zurich, Switzerland)	Constructed and tested prototype chamber Refined technical parameters Built the chambers for the NTP exposure facility Installed chambers at IITRI Monitored system performance throughout all phases of the studies Conducted maintenance on exposure system hardware and software
IIT Research Institute (IITRI) (Chicago, IL)	Tested exposure system after installation Conducted maintenance of exposure system hardware Conducted all toxicology and carcinogenicity studies Conducted day-to-day operations

REVERBERATION CHAMBER METHOD OF EXPOSURE

The use of the reverberation exposure chamber as a method for exposing rats and mice to cell phone RFR was conceptualized by the NIST and further designed and tested by NIST and the IT'IS Foundation. A reverberation chamber is a resonant box where the resonances and field structure is continuously modified under the influence of metallic stirrers, introduced to change the effective geometry, such that when averaged over time, the field strength is uniform over the entire exposure volume. A reverberation chamber exposure system was selected for the NTP for the primary benefit that controlled exposures can be achieved in unrestrained animals (rats and mice) with extended daily RFR exposure periods compared to other methods of exposure for up to 2 years.

Preliminary studies were first conducted at the NIST to test the concept of reverberation chambers. In these studies, field strengths and field uniformity were measured under various conditions of cell phone RFR exposure, including an empty chamber and a chamber loaded with water bottles (simulating animals) at different locations in the chamber. Power requirements were evaluated to achieve desired SAR levels. The effects of proximity between water bottles were also investigated to avoid electromagnetic coupling. These studies provided critical information for the design of experimental studies with respect to the number of cages that could be placed in specific size chambers, the arrangement of cages within each chamber, and the input power requirements. The results of these investigations demonstrated that while variations occurred over time and space the average cell phone RFR field was uniform over the large volume of the chamber. These studies also demonstrated that cell phone RFR field exposure occurred from all directions and all polarizations, and that there was uniformity of SAR in reverberation chambers. Based on the information and parameters obtained during the NIST feasibility studies, a custom-built prototype reverberation chamber was constructed and tested by the IT'IS Foundation. The development of the prototype chamber involved the design of amplifiers and antennas for signal generation, the design of vertical and horizontal stirrers to improve the homogeneity of experimentally generated RF fields, the development of both hardware and software for the control and monitoring of experimentally generated RF signals, and testing of chamber performance. During the design of the prototype exposure chamber, engineering studies were performed to optimize the following prior to construction:

- The uniform field volume within each chamber to minimize spatial variability in the characteristics of generated RF fields within a chamber such that all animals housed within the chamber space were exposed to comparable RF field strengths
- The design and placement of stirrers in each chamber in order to maximize homogeneity of experimentally generated RF fields
- The design and location of RF antennas in each chamber
- The location of cage racks within the exposure chamber in order to provide appropriate separation of individual animal cages and cage racks from all reflective surfaces (chamber walls, chamber floor and ceiling, antennas, and stirrers) in the reverberation chamber
- Chamber volume to provide adequate space for staff to observe animals, collect data, and perform routine animal husbandry operations, while minimizing overall chamber volume to minimize the chamber size/footprint and the RF power required to maintain target SARs

The final reverberation chamber design for use in these studies was a fully shielded room constructed of stainless steel, equipped with a shielded room door to eliminate leakage of RFR signals, two rotating stirrers (one horizontal and one vertical), ventilation structures, and RFR excitation antennas. A detailed rationale for the selection of reverberation chambers for exposure to RFR and a full description of the exposure system are provided in Capstick *et al.* (2017) and Gong *et al.* (2017).

As part of the validation of the reverberation chamber exposure system design, a team of engineers from NIST conducted an independent evaluation of chamber design and exposure system operation in order to evaluate the suitability of the reverberation chamber model for use in the program. NIST engineers evaluated the design and operation of the prototype chamber and performed an extensive series of RF measurements to support an evaluation of system performance.

CELL PHONE RFR EXPOSURE FACILITY

The exposure facility was specifically designed to expose rats in reverberation chambers to three different power levels of modulated cell phone RFR [Global System for Mobile Communications (GSM) or Code Division Multiple

Access (CDMA)] at 900 MHz for up to 2 years to evaluate toxicity and carcinogenicity. The completed exposure facility consisted of a total of 21 reverberation exposure chambers (14 designated for rats); the RFR signal generation, amplification, and monitoring systems; software for chamber operation; and hardware and software for monitoring of environmental and exposure conditions within each chamber. All system hardware and software were installed by the IT'IS Foundation.

During exposures, modulated (GSM or CDMA) cell phone RFR signals were generated by a signal generator, amplifiers amplified the signals, and the signals were delivered by antennas in the reverberation chambers. RFR field strengths were monitored in real time and were adjusted throughout the studies to achieve specific exposure levels [based on SARs quantitated in watts (W) per kg body weight]. Environmental conditions were also monitored and controlled in real time throughout the study. RFR exposures and environmental conditions were monitored and controlled by a computer in a control room at the study laboratory at IITRI; the IT'IS Foundation was also capable of remote system monitoring and control.

Facility Design and Reverberation Chambers

Each reverberation chamber was permanently programmed for a specified modulation (GSM or CDMA) of the 900 MHz cell phone RFR specified for the rat studies. SARs for each chamber were adjustable and selected prior to exposures. The field strength required to achieve a given target SAR (W/kg) exposure level is a function of animal body weight (kg); separate chambers were required for male and female rats because their body weights differ by almost a factor of two after a few weeks of development. To conduct robust toxicology studies with three exposure groups (low, medium, and high), six chambers were required for different levels of exposures for GSM modulation and six for CDMA modulation. Two sham exposure chambers without any cell phone RFR signal provided shared control groups for the parallel studies of the two modulations. As per these requirements, the cell phone RFR exposure facility consisted of 14 reverberation chambers for exposures in rats including:

- Three power levels for F₀ females and F₁ males exposed to GSM-modulated cell phone RFR at 900 MHz
- Three power levels for F₁ females exposed to GSM-modulated cell phone RFR at 900 MHz
- Three power levels for F₀ females and F₁ males exposed to CDMA-modulated cell phone RFR at 900 MHz
- Three power levels for F₁ females exposed to CDMA-modulated cell phone RFR at 900 MHz
- One sham control chamber for F₀ females and F₁ males with no RFR exposure
- One sham control chamber for F₁ females with no RFR exposure

The chamber size was designed to accommodate the RF field stirring paddles (described below), approximately 110 individually housed rats, and a minimum distance (3/4 of a wavelength) between the cages and the walls, floor, ceiling and stirrers, respectively. The interior of the chamber was suitable for cleaning using high-pressure water (after the RF antennas were protected). The internal dimensions of the chambers were 2.2 m (width) × 3.7 m (length) × 2.6 m (height); the exterior dimensions were 2.3 m (width) × 3.8 m (length) × 2.85 m (height). A floorplan for the exposure facility and images of the interior and exterior of the chambers are presented in Figures 2 and 3.

Each chamber contained two motor-controlled stirring paddles (one vertical and one horizontal) with adjustable speed control (1 to 50 rpm) and large asymmetrical reflecting surfaces. Stirring paddles were placed off center in the chamber for maximum scattering of the RFR fields to generate a statistically homogeneous field distribution when averaged over time. The horizontal stirrer was mounted on the ceiling of the chamber. The vertical stirrer was at the rear of the chamber, and was protected by rack guides that prevented contact with the animal cage racks.

Cage Racks and Watering System

Cages, cage racks, and watering systems for standard laboratory use contain elements that have the ability to alter the exposure of the animals or introduce potential confounding factors. Because cage racks and the drinking water delivery system were contained inside the chambers during exposure periods, it was required that these components be constructed of durable materials that had essentially no impact on the RF fields generated in the chamber.

Metallic cage rack components, cage lids, feed dispensers, and cage grommets all needed to be eliminated. Hence,

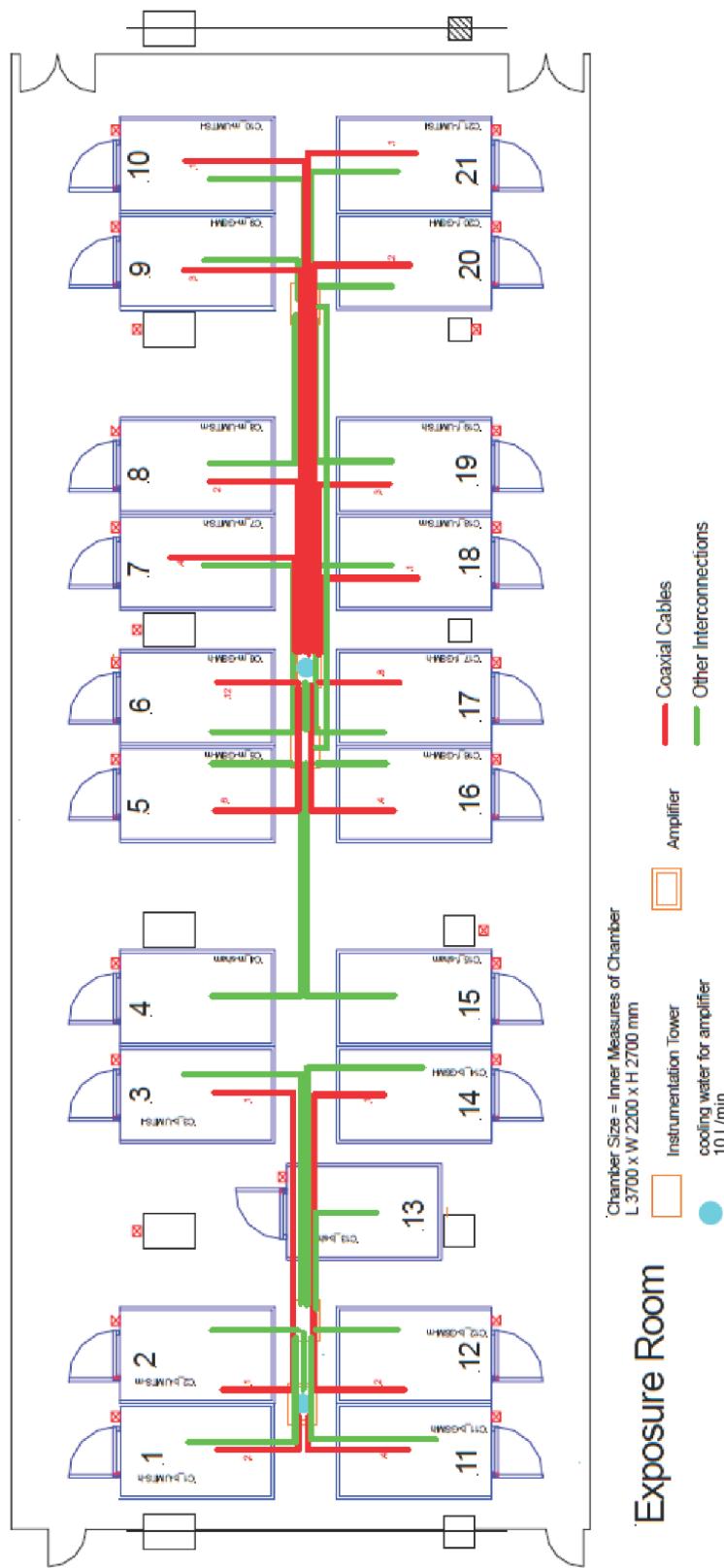


FIGURE 2
Exposure Facility Floor Plan for the Cell Phone RFR Studies (Not shown are the Ethernet connections to computers in the control room.)
 Rat chamber designations: F₀ females and F₁ males - low GSM=9; medium GSM=5; high GSM=5; low CDMA=10; medium CDMA=8; high CDMA=7; sham=4; F₁ females - low GSM=20; medium GSM=16; high GSM=17; low CDMA=21; medium CDMA=18; high CDMA=19; sham=15. The seven other chambers (including six for cell phone RFR exposure and one for sham control) were designated for concurrent mouse studies.



FIGURE 3
Exterior View of Chambers, Empty Chamber Showing the Vertical and Horizontal Stirrers, and Chamber with Cage Racks in Place

custom engineering was required to overcome the challenges regarding potential RFR exposure-altering aspects of the caging and cage racks used to house the animals during the studies. The safe provision of drinking water provided the largest challenge for the studies.

The absorption of RFR energy by water if supplied by nonmetallic sipper tubes and distribution systems or bottles, could lead to dose-dependent elevated water temperatures. At the same time, the potential for enhanced exposure fields by metallic sipper tubes or lixits precluded the use of water bottles or a standard automatic watering system in the reverberation chambers. The absorption of RFR energy by water could result in significant heating of the drinking water, thereby decreasing water palatability, and increasing the required RFR power to achieve the desired exposure field strength, potentially to the extent that the exposure levels could not be met. To overcome these challenges, adaptations were made to an automatic watering system so that the delivery of drinking water to the animals would not interfere with cell phone RFR dosimetry. The water system was constructed from stainless steel ensuring no dose-dependent energy absorption in the water (avoiding exposure-dependent water temperature) and in structures around the lixits to ensure no enhanced fields that could lead to excessive SAR in the animals while drinking.

Customized, nonmetallic animal cage racks for the reverberation chambers were designed by IITRI to minimize any absorption of RFR or disruption of RF field homogeneity. Cage racks were constructed primarily of box beam fiberglass (with some angle beam fiberglass used in nonweight-bearing areas of the rack). The shelves/cage lids were constructed of a clear polycarbonate sheet with slots for increased airflow. The potential impact of the racks on RF fields was evaluated in the prototype reverberation chamber by the IT'IS Foundation. Cage racks were designed to accommodate the automatic watering system and position the perimeter of each animal cage at least one-half wavelength from any reflecting surface. The specific considerations for design and further details of the custom-designed cage racks and adapted automated watering system are provided in Capstick *et al.* (2017).

Cell Phone RFR Exposure System Control

The hardware and chambers designated for rats (using an exposure frequency of 900 MHz) were connected to a dedicated computer control system using an Ethernet protocol. The computerized control system managed and

monitored the cell phone RFR exposures and environmental conditions in the chambers. A more detailed description of the computer control of cell phone RFR exposure is provided in Capstick *et al.* (2017).

The control computer managed the exposure schedule, stirrer rotation speeds, and exposure signal and level and monitored air flow, temperature, humidity, light, and the electric and magnetic fields (E- and H-fields, respectively) in each chamber. The hardware for the exposure system consisted of the control computer and a rack containing communications interfaces and instrumentation for signal generation, data acquisition, and signal monitoring, signal amplifiers, and the chamber hardware (which included the stirrer motors and environmental and RFR sensors). The instrumentation rack contained the equipment that generated the cell phone RFR signal, acquired cell phone RFR field strengths and environmental data, and provided an interface between the components and the control computer.

On the main rack, the rat system hardware included an Ethernet to general purpose interface bus, a cell phone RFR signal generator, four data acquisition units, four RF field measurement units, a power supply unit, an Ethernet hub, and a 1W medium power amplifier. A second rack contained an Ethernet to general purpose interface bus, two data acquisition units, three RF field measurement units, a power supply unit, and an Ethernet hub. The amplifier array housed signal amplifiers, an amplifier cooling system, and three real-time digital control units that directly controlled the 12 amplifiers in the rat system. Each amplifier produced 400 W peak power and in excess of 200 W average power. A closed-circuit cooling system ran cool water through the amplifiers to keep them from overheating. The real-time digital control units controlled which chamber the amplifier output was routed to and the level of amplifier output power while it was routed to that particular chamber.

CELL PHONE RFR SIGNAL GENERATION

GSM-modulated and CDMA-modulated cell phone RFR signals were generated experimentally via a SMIQ02B vector signal generator with options SMIQB11 and SMIQB20 and software options 100421 – 100423 (Rohde and Schwarz, Munich, Germany). Signals were amplified using 12 LSE™ amplifiers (LSE, Spanga, Sweden) in the exposure system. The outputs of each individual amplifier were set by real-time controllers on a slot-by-slot basis for GSM or CDMA modulation to control the E-field strength in each chamber. Each chamber contained at least one standard gain antenna (two half-wave dipoles) that was mounted a quarter of a wavelength in front of a reflector

plate. Antennas were directed towards one of the two stirrers to maximize scattering and obtain acceptable E-field homogeneity within the chamber space. The computerized control system managed the exposure schedule, stirrer rotation speeds, and exposure signal type and level.

The RFR power introduced into a given chamber was adjusted to achieve target field strengths; to maintain constant exposure levels (W/kg) in a given chamber, the field strengths [measured in volts (V) per meter] were regularly adjusted to reflect changes in the average mass of the exposed animals. The relationship between animal mass, field strength, and SAR was determined from numerical dosimetry and programmed into the control software, hence the required exposure field strength was computed from the average animal weights entered for each exposure group. The interval at which animal weights were updated was determined on how rapidly the animals were growing, at the start of the exposure period this was once per week, and as long as up to every 4 weeks later in the studies.

VERIFICATION OF CELL PHONE RFR EXPOSURE

Prior to initiation of the animal studies, the RF Fields Group in the Communications Technology Laboratory at the NIST performed an independent, detailed evaluation of 18 of the reverberation chambers (excluding the three sham control chambers; Figure 2) to verify the cell phone RFR exposure fields, chamber characteristics (field uniformity), and signal quality to determine the accuracy of field values reported by the developers of the exposure system (IT'IS Foundation). Full reports detailing the procedures for measurements and calculations are available from the NTP. NIST performed two additional detailed evaluations: (1) in the interim period between completion of the 28-day studies and prior to initiation of the 2-year studies, and (2) following completion of the 2-year studies.

All E-field measurements were within the estimated uncertainty bounds, indicating that the chamber fields measured by the NIST agreed with the measurements provided by the IT'IS Foundation probes. During validation, it was determined that the H-field probes at higher signal levels in the mid- and high-power GSM chambers reported higher fields than indicated by other measurements, potentially leading to a modest overestimation of chamber field strengths. In these chambers, H-field probes were replaced with E-field probes, which provided more accurate measurements of the RF fields. The magnitude of field variation throughout the volume of a fully loaded chamber was consistent with earlier values reported for the prototype chamber. However, it was determined that there may

have been up to ± 2.5 dB of variation in the exposure field depending on location in the cage racks. To mitigate this positional variation, cages were routinely rotated to various locations within and between the cage racks. The quality of the modulated signals was found to be acceptable with regard to distortion and harmonic content.

Overall, NIST confirmed that the cell phone RFR reverberation chamber exposure system was operating correctly and cell phone RFR exposures were within specifications.

CELL PHONE RFR EXPOSURE MONITORING

During all exposure periods, experimentally generated cell phone RFR was continuously monitored by the control system via two RF sensors (E- and/or H-field probes) in each exposure chamber that measured real-time signal strengths. The use of two probes provided two independent measurements of RF field strengths and ensured that appropriate quantitation of experimentally generated RF fields continued even in the unlikely event that one probe failed. The E-field sensor measured electric field strength (V/m). The H-field sensor measured magnetic field strength [measured in amperes (A) per meter]. All chambers were instrumented either with one E-field sensor (ER3DV6) and one H-field sensor (H3DV6) [both from Schmid and Partner Engineering AG (SPEAG), Zurich, Switzerland], except for the medium and high power GSM chambers. These chambers were instrumented with two E-field probes because H-field probes saturated at high field strengths. This change in hardware did not result in the loss of monitoring capability. The measured E- and H-fields were communicated to the control computer in order to maintain exposure to selected levels of RFR. During daily shutdown periods when RFR exposures were not active, RF sensors monitored ambient RF fields in the exposure chambers. RF sensors were calibrated twice by the manufacturer (SPEAG); once prior to initiation of any of the animal studies and once prior to initiation of the 2-year studies. All E-field probes were calibrated in air from 100 MHz to 3.0 GHz, and had an absolute accuracy of $\pm 6.0\%$ ($k=2$) with a spherical isotropy of better than ± 0.4 dB. All H-field probes were calibrated in air from 200 MHz to 3.0 GHz and had an absolute accuracy of $\pm 6.0\%$ ($k=2$) with a spherical isotropy of better than ± 0.2 dB.

Data collected by the RF sensors were transmitted to the exposure and monitoring system on a real-time basis and were recorded throughout the studies. Chamber field strengths are reported as V/m and animal exposure levels (SAR values) are reported as W/kg. The chamber field strength is the average effective E-field strength from both

probes. E- and H-field strengths are related by the impedance of free space which is ~377 Ohms. Where an H-field probe was used, the value in A/m was multiplied by 377 to calculate the equivalent E-field strength in V/m; it is this effective E-field value that was used to report the chamber field strength. Field strength data reported for each day of exposure included mean \pm standard deviation, minimum field strength, maximum field strength, total number of readings in range/total number of readings for the period, and percentage of readings in range. After each exposure day, cell phone RFR exposure data were downloaded onto DVDs for long-term archival. Summaries of the 2-year cell phone RFR exposure data from the studies are presented in Appendix I. The SAR and chamber-fields in the exposure chambers were within the target ranges (defined as ± 2 dB) for >99.97% of recorded measurements over the course of the 2-year study; $\geq 99.25\%$ of recorded E-field and H-field measurements were within the target ranges. All recorded SAR and field measurements were within the target ranges for the sham chamber. In the 28-day studies, the performance of the sham and exposure chambers was similar for SAR and field measurements as in the 2-year studies (data not shown).

As previously stated, the performance of the cell phone RFR exposure and monitoring system was independently validated by engineers from the NIST prior to the initiation of the animal studies.

MONITORING AND MAINTENANCE OF ENVIRONMENTAL CONDITIONS

Environmental conditions including temperature, humidity, and airflow in all exposure chambers, as well as in other areas of the IITRI cell phone RFR exposure facility, were maintained by a computer-controlled environmental management system (Siemens Industries, Inc.). Monitoring instrumentation for each chamber was located in the air exhaust duct. Each chamber was fitted by the IT'IS Foundation with a sensor box that contained sensors for temperature and humidity (Type EE06; E + E Elektronik GmbH, Engerwitzdorf, Austria), oxygen level (Pewatron Type FCX-MC25; Zurich, Switzerland), air speed (model EE65A; E + E Elektronik GmbH), light (light-dependent resistor), noise (design based on WL-93 microphone; Shure Brothers, Inc., Evanston, IL), and RFR. Outputs from the sensor box were monitored using Agilent data acquisition units, with the exception of the RF sensor. The RF sensor was directly wired to a warning light as a safety precaution to indicate active RFR exposures and not intended to quantitatively measure RFR field strengths.

Exposure chambers were equipped with incandescent lights located on light bars in each corner of the chamber. All connections were RF-filtered. Chamber lighting was controlled using an adjustable daily cycle of 12 hours on, 12 hours off. In order to minimize the heat load generated by the incandescent lights, low wattage bulbs were used that maintained chamber lighting within a range that was sufficient to support normal *in vivo* operations, while minimally affecting chamber temperature.

Differences in noise levels in the exposure chambers that resulted from the heating, ventilation, and air conditioning system were equalized by the installation of sound baffles in various ducts within the system. An audible signal generated by the high intensity GSM signal was detected and equalized in all chambers by the introduction of a “pink noise” masking sound; this masking noise equalized sound levels in all chambers. As a result of the combination of these efforts, noise levels in all chambers were essentially equivalent and met the NC-35 noise specification [the noise criterion (NC) is a widely accepted numerical index commonly used to define the maximum allowable noise. It primarily applies to the noise produced by ventilation systems, but is applied to other noise sources, as well. Standards organizations, such as the American National Standards Institute (ANSI), Acoustical Society of American (ASA), and International Standards Organization, provide definitions of various NCs for ambient noise in enclosed spaces. The ANSI/ASA standard (S12.2-2008) recommends NCs for various types of rooms, including private residences (NC 25-40), schools (NC 25-35), offices (NC 25-40), libraries (NC 30-35), and restaurants (NC 40-45)].

ANIMAL SOURCE

Time-mated (F_0) female Hsd:Sprague Dawley SD rats were obtained from Harlan Laboratories, Inc. (now Envigo, Indianapolis, IN), for use in the 28-day and 2-year studies.

ANIMAL WELFARE

Animal care and use are in accordance with the Public Health Service Policy on Humane Care and Use of Animals. All animal studies were conducted in an animal facility accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care International. Studies were approved by the IITRI Animal Care and Use

Committee and conducted in accordance with all relevant NIH and NTP animal care and use policies and applicable federal, state, and local regulations and guidelines.

28-DAY STUDIES

The 28-day studies were conducted to evaluate the cumulative effects of repeated GSM- or CDMA-modulated cell phone RFR exposure and to determine the appropriate cell phone RFR power levels to be used in the 2-year studies.

Beginning gestation day (GD) 6, groups of F₀ female rats were housed in reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 3, 6, or 9 W/kg, for 9 hours and 10 minutes per day for 5 or 7 days per week with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups, but were not exposed to cell phone RFR; shared groups of unexposed rats served as sham controls for both cell phone RFR modulations.

In order to evaluate potential toxicity that arises from *in utero* and early postnatal exposure, these developmental windows were included in the cell phone RFR studies in rats. F₀ female rats were approximately 11 to 14 weeks old upon receipt. GD 1 was defined as day with evidence of mating, and F₀ females were received on GD 2 and held in quarantine until GD 5. Animals were randomly assigned to GSM or CDMA exposure groups (20 F₀ females/cell phone RFR power level per modulation) with a single group of 20 F₀ females serving as the sham control group for both the GSM and CDMA modulations. Randomization was based on body weights that produced a similar group mean value (ToxData, version 2.1.E.11, PDS Pathology Data Systems, Inc., Basel, Switzerland).

In 10 F₀ females per group, subcutaneously implanted temperature microchips and monitoring equipment (Bio Medic Data Systems, Seaford, DE) were used to monitor individual animal body temperatures. Body temperature measurements were taken prior to initial exposure (GD 6) and on GDs 7, 11, and 16 and postnatal days (PND) 1, 4, 7, and 14 within 3.5 (GDs) or 2 (PNDs) minutes of exposure pauses at the end of the second to the last “on” cycle.

F₀ females were housed individually during gestation and with their respective litters during lactation. During gestation, F₀ females were weighed on GDs 6, 9, 12, 15, 18, and 21. During lactation, F₀ females were weighed on PNDs 1, 4, 7, 14, 17, and 21 and individual F₁ pup weights were recorded on PNDs 4, 7, 14, 17, and 21. The day of parturition was considered PND 0. From GD 20 to 25, F₀ females were observed twice daily for parturition. All F₀ females that did not deliver within 3 to 4 days of the anticipated delivery date were euthanized and the uteruses were examined for uterine implantations/resorptions. On the day after parturition (PND 1), the number of live and dead F₁ pups, sex ratio, whole litter weights, and litter weights/sex were recorded.

F₁ litters were standardized on PND 4 to eight pups/litter, preferably with four males and four females each to equalize lactational pressure on F₀ females. Litters that did not meet a minimum of eight pups were removed from the study. For continuation of exposure after weaning, three males and three females per litter from 10 F₁ litters were randomly selected per exposure group. Weaning occurred on PND 21. Pups not selected and all F₀ females were euthanized with 100% carbon dioxide without necropsy. Weaning marked the beginning of the 28-day prechronic phase of the study.

Groups of 10 male and 10 female F₁ rats were housed in the same reverberation chambers and continued and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at the same power levels for 9 hours and 10 minutes per day for 5 or 7 (last week of study) days per week for at least 28 days, with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day.

The health of the animals was monitored during the studies according to the protocols of the NTP Sentinel Animal Program (Appendix K). All test results were negative.

Animals were observed twice daily and weighed weekly. Clinical findings were recorded weekly. Subcutaneously implanted temperature microchips and monitoring equipment (Bio Medic Data Systems, Seaford, DE) were used to monitor individual animal body temperatures. Body temperature measurements were taken on day 8 after microchip implantation and on days 16, 20, and 27 within 5 minutes of exposure pauses at the end of the second to the last “on” cycle.

Rats were housed individually. Feed and water were available *ad libitum*. To avoid interference with cell phone RFR dosimetry, feed was provided in glass (nonmetallic) jars and water was delivered in an adapted automatic watering system. Cages were changed weekly and rotated within the racks weekly; racks were changed biweekly. Further details of animal maintenance are given in Table 1. Information on feed composition and contaminants is provided in Appendix J.

Necropsies were performed on all F₁ rats on PND 29 or 30. Organs weighed were the right adrenal gland, brain, heart, right kidney, liver, lung, right testis, and thymus. Tissues for microscopic examination were fixed and preserved in 10% neutral buffered formalin (except eyes, testis with epididymis, and vaginal tunics were first fixed in Davidson's solution or modified Davidson's solution), processed and trimmed, embedded in paraffin, sectioned to a thickness of 4 to 6 µm, and stained with hematoxylin and eosin. Complete histopathologic examinations were performed by the study laboratory pathologist on all 0 (sham control) and 9 W/kg GSM- and 9 W/kg CDMA-modulated cell phone RFR core study F₁ rats. Table 1 lists the tissues and organs examined.

The laboratory reports and selected histopathology slides were reviewed by a quality assessment pathologist (QAP). Any inconsistencies in the diagnoses made by the study laboratory and QA pathologists were resolved by the NTP pathology peer review (PPR) process (see description of PPR process on page 60 of this report). A PPR was conducted to confirm treatment-related findings and resolved inconsistencies in diagnoses. Final diagnoses for reviewed lesions represent a consensus of the PPR or a consensus between the study laboratory pathologist (SP), NTP pathologist, and QAP. Details of these review procedures have been described, in part, by Maronpot and Boorman (1982) and Boorman *et al.* (1985).

2-YEAR STUDIES

Study Design

Beginning on GD 5, groups of F₀ female rats were housed in reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 1.5, 3, or 6 W/kg, for 9 hours and 10 minutes per day for 7 days per week with continuous cycling of 10 minutes on and 10 minutes off

during a period of 18 hours and 20 minutes each day. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups, but were not exposed to cell phone RFR; shared groups of unexposed rats served as sham controls for both RFR modulations.

F₀ female rats were approximately 11 to 14 weeks old upon receipt. GD 1 was defined as day with evidence of mating, and F₀ females were received on GD 2 and held in quarantine until GD 4. Animals were randomly assigned to GSM or CDMA exposure groups (56 F₀ females/cell phone RFR power level per modulation) with a single group of 56 F₀ females serving as the sham control group for both the GSM and CDMA modulations. Randomization was stratified by body weight that produced similar group mean weights (ToxData, version 3.0, PDS Pathology Data Systems, Inc., Basel, Switzerland).

F₀ females were housed individually during gestation and with their respective litters during lactation. During gestation, F₀ females were weighed on GDs 6, 9, 12, 15, 18, and 21. During lactation, F₀ females were weighed on PNDs 1, 4, 7, 14, 17, and 21 and individual F₁ pup weights were recorded on PNDs 4, 7, 14, 17, and 21. The day of parturition was considered PND 0. All time-mated females that did not deliver within 3 to 4 days of the anticipated delivery date were euthanized and the uteruses were stained for uterine implantations/resorptions. On the day after parturition (PND 1), the number of live and dead F₁ pups, sex ratio, whole litter weights, and litter weights/sex were recorded.

F₁ litters were standardized on PND 4 to eight pups/litter, preferably with four males and four females each to equalize lactational pressure on F₀ females. Litters that did not meet a minimum of eight pups were removed from the study. For continuation of exposure after weaning, three males and three females per litter from 35 F₁ litters were randomly selected per exposure group. Weaning occurred over PND 21 and 22 and F₁ rats were housed individually. Pups not selected and the F₀ females were euthanized with 100% carbon dioxide without necropsy. Weaning marked the beginning of the 2-year chronic phase of the study.

Groups of 105 male and 105 female F₁ rats were housed in reverberation chambers and continued to receive whole-body exposures to GSM- or CDMA-modulated cell phone RFR at the same power levels for 9 hours and 10 minutes

per day for 7 days per week for 104 weeks, with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. At 14 weeks, 10 rats per group were randomly selected for interim evaluation and five were designated for genetic toxicity evaluation.

The health of the animals was monitored during the studies according to the protocols of the NTP Sentinel Animal Program (Appendix K). All test results were negative.

Feed and water were available *ad libitum*. To avoid interference with RFR dosimetry, feed was provided in ceramic (nonmetallic) bowls and water was delivered in an adapted automatic watering system (Capstick *et al.*, 2017). Cages were changed weekly and rotated within the racks biweekly; racks were changed biweekly. Further details of animal maintenance are given in Table 1. Information on feed composition and contaminants is provided in Appendix J.

Clinical Examinations and Pathology

Animals were observed twice daily and were weighed initially, twice a week for the first 13 weeks, and at 4-week intervals from weeks 14 to 86, and then every 2 weeks from week 90 until the end of the studies. Clinical observations were recorded once during quarantine and at least every 4 weeks during the studies.

Hematology evaluations were performed on 10 male and 10 female interim evaluation rats from each group at 14 weeks. Rats were anesthetized with 70% CO₂/30% O₂ and blood was collected from the retroorbital sinus and placed into tubes containing EDTA as an anticoagulant. Hematology parameters were determined on an ADVIA™ 120 automated hematology analyzer (Bayer Diagnostic Division, Tarrytown, NY). The parameters measured are listed in Table 1. Wright Giemsa stained peripheral blood smears were prepared and evaluated for any blood cell abnormalities. Blood was collected from the remaining five male and five female interim evaluation rats per exposure group at 14 weeks for use in the comet and micronucleus assays; methods for these assays are presented in Appendix E.

At 14 weeks, samples were collected for sperm motility and count and vaginal cytology evaluations on 10 male and 10 female interim evaluation rats from each group. The parameters evaluated are listed in Table 1. For 16 consecutive days prior to scheduled euthanasia, the vaginal vaults of the females were moistened with saline, if necessary, and samples of vaginal fluid and cells were stained. However, due to inconsistent sample collection and slide staining, an assessment of estrous cyclicity could not be made. Male animals were evaluated for sperm count and motility. The left testis and left epididymis were isolated and weighed. The tail of the epididymis (cauda epididymis) was then removed from the epididymal body (corpus epididymis) and weighed. Modified Tyrode's buffer was applied to slides and a small incision was made at the distal border of the cauda epididymis. The sperm effluxing from the incision were dispersed in the buffer on the slides, and the numbers of motile and nonmotile spermatozoa were counted for five fields per slide by two observers. Following completion of sperm motility estimates, each left cauda epididymis was placed in buffered saline solution. Caudae were finely minced, and the tissue was incubated in the saline solution and then heat fixed at 65° C. Sperm density was then determined microscopically with the aid of a hemacytometer. To quantify spermatogenesis, the testicular spermatid head count was determined by removing the tunica albuginea and homogenizing the left testis in phosphate-buffered saline containing 10% dimethyl sulfoxide. Homogenization-resistant spermatid nuclei were counted with a hemacytometer.

A complete necropsy was conducted on every animal at study termination. For the 14-week interim evaluation rats, the cerebellum, frontal cortex, hippocampus, and liver were collected from five male and five female rats per exposure group for use in the comet assay; methods for this assay are presented in Appendix E. Microscopic examinations were performed on 10 male and 10 female interim evaluation rats in each group at 14 weeks and all core study rats, including those found dead or euthanized moribund. At the interim evaluation, the brain, right and left epididymides, heart, right and left kidneys, liver, lung, right and left ovaries, right and left testes, and thymus were weighed. At necropsy, all organs and tissues were examined for grossly visible lesions, and all major tissues were fixed and preserved in 10% neutral buffered formalin (except eyes, testes, vaginal tunics, and epididymides were first fixed in Davidson's solution or a modified Davidson's solution), processed and trimmed, embedded in paraffin, sectioned to a thickness of 4 to 6 μm , and stained with hematoxylin and eosin for microscopic examination.

For all paired organs (e.g., adrenal gland, kidney, ovary), samples from each organ were examined. Tissues examined microscopically are listed in Table 1.

Microscopic evaluations were completed by the study laboratory pathologist, and the pathology data were entered into the Toxicology Data Management System Enterprise. The report, slides, paraffin blocks, residual wet tissues, and pathology data were sent to the NTP Archives for inventory and NTP PPR. All data and materials are available for review upon request from the NTP Archives.

NTP Pathology Peer Review Process

The primary goals of the NTP pathology review are to reach consensus agreement on the diagnoses of all potentially treatment-related findings, confirm the diagnoses of all neoplasms, confirm that consistent and acceptable nomenclature is being used, and confirm the diagnosis of any unusual lesions. There are several elements in this process:

Pathology Data Review (PDR) is a complete review of the pathology data generated by the study laboratory to identify potential target organs and discrepant data and to harmonize terminology. The review involves a multidisciplinary meeting by the NTP staff and pathology-support-contract pathologists to determine the organs and lesions to be reviewed by the QAP, including all neoplasms.

Audit of Pathology Specimens (APS) is a review of the physical data and residual wet tissues (typically from 10% of the animals) to ensure all gross lesions were evaluated microscopically; of the slides and blocks (typically from 10% of the animals) to ensure correct labeling and quality of sections; and of the submitted reports to ensure accuracy. Also evaluated is whether or not the study laboratory adhered to NTP pathology specifications.

Quality Assessment (QA) is a review of the slides of target organs and lesions identified in the PDR by a pathologist from one of the NTP's pathology support contract laboratories not involved with the initial pathology evaluation of the study. All differences in diagnoses between the SP and QAP are identified in the Differences Report prepared

by the QAP. The NTP pathologist attempts to resolve the discrepant diagnoses between the SP and QAP; those that are not resolved are reviewed by the pathology working group (PWG).

Pathology Working Group (PWG) is a review of selected slides by a panel of pathologists in order to confirm the diagnoses of all treatment-related neoplastic and nonneoplastic lesions and unusual lesions, resolve discrepancies between the SP, QAP, and NTP pathologist, harmonize nomenclature, propose further characterization of the lesions, and address possible mechanisms. The QAP, with oversight from the NTP pathologist, selects slides for the PWG and conducts the PWG. Typically, experts in a particular organ of interest are invited to participate.

Pathology Peer Review (PPR) is a peer review meeting that convenes to resolve minor issues or issues limited in scope (such as review of short-term studies with limited findings), or review findings of post-PWG actions. Reports are prepared for all these activities. Once the PWG is complete, all written documentation of data changes is reviewed for accuracy. Once the entire PPR process and review of data changes are completed, the study data are updated. The pathology data and all written documentation of data changes are then submitted to an outside independent auditor to ensure the accuracy of the updated data. Once all issues identified by the independent auditor have been addressed, the final pathology data tables are generated.

Pathology Review of Cell Phone RFR Studies in Rats

The pathology data presented in this report on cell phone RFR were subjected to a rigorous PPR process. All elements of the NTP PPR process were performed, but the sequence of events was altered. Identification of increased incidences of lesions in the brain and heart of male rats in the original study laboratory report warranted a more immediate review than would occur in the standard NTP PPR process. Malignant glioma of the brain and schwannomas have been observed in human studies, so the observation of an apparent increase in these same lesions in the rat study prompted the need for an expedited review given the magnitude of human exposure to cell phone RFR and therefore the need to communicate this information to our regulatory partners and the public as soon as possible. Data for the brain and heart were reported to the U.S. Food and Drug Administration and Federal Communications Commission and published in a partial report (Wyde *et al.*, 2016).

For this expedited review, an APS (APS 1) was performed on the hearts and brains. This entailed reviewing the residual wet tissues to ensure all gross lesions were trimmed and processed to slide, reviewing the slides and blocks to ensure quality, and reviewing the data tables. For the expedited review, a QA review was done on all lesions in the central and peripheral nervous systems, all proliferative lesions from the heart, and all schwannomas in other organs. The QAPs, with oversight from the NTP pathologist, selected lesions for PWG review. These lesions were reviewed in several PWGs. Four separate PWGs were held for the brains and hearts. The first PWG (PWG 1), held on January 29, 2016, evaluated the proliferative glial lesions and some reactive glial lesions in the brain, some unusual brain and pineal gland lesions, and schwannomas and Schwann cell hyperplasias in the heart. The second PWG (PWG 2) evaluated schwannomas in the head and neck region and nonglial proliferative lesions in the brain (e.g., granular cell tumors and meningiomas). Due to the volume of slides to be reviewed, PWG 2 was conducted over four sessions (February 11, February 12, March 23, and April 11, 2016). Due to the need for definitive criteria for glial cell and Schwann cell hyperplasia, two additional PWGs composed of experts in neuropathology and cardiovascular pathology, respectively, from around the country were convened. The first (PWG 3) reviewed glial lesions in the brain and was held on February 25, 2016. The second (PWG 4), held on March 3, 2016, reviewed cardiac lesions and schwannomas in organs other than the heart and head and neck region. Subsequent to the release of NTP's Report of Partial Findings (Wyde *et al.*, 2016), the remaining tissues were reviewed. After the Report of Partial Findings, the remaining tissues, and the nonproliferative lesions in the heart, were reviewed (including PWGs 5 and 6). These tissues were subjected to the standard NTP PPR process as described earlier.

TABLE 1
**Experimental Design and Materials and Methods in the Whole-Body Exposure Studies
of GSM- and CDMA-Modulated Cell Phone RFR**

28-Day Studies	2-Year Studies
Study Laboratory IIT Research Institute (Chicago, IL)	IIT Research Institute (Chicago, IL)
Strain and Species Sprague Dawley (Hsd:Sprague Dawley SD) rats	Sprague Dawley (Hsd:Sprague Dawley SD) rats
Animal Source Harlan Laboratories, Inc. (Indianapolis, IN)	Harlan Laboratories, Inc. (Indianapolis, IN)
Time Held Before Studies 4 days (F ₀ females)	3 days (F ₀ females)
Average Age When Studies Began 13 to 16 weeks (F ₀ females)	12 to 15 weeks (F ₀ females)
Date of First Exposure November 1, 2010 (F ₀ females) December 9, 2010 (F ₁ rats)	August 8, 2012 (F ₀ females) September 16, 2012 (F ₁ rats)
Duration of Exposure 9 hours and 10 minutes per day, over an 18-hour and 20-minute period as exposures cycled between modulations every 10 minutes, 7 days per week for perinatal phase and last week of prechronic phase, and 5 days per week otherwise	9 hours and 10 minutes per day, over an 18-hour and 20-minute period as exposures cycled between modulations every 10 minutes, 7 days per week for 14 weeks (interim evaluation) or 106 (males) or 107 (females) weeks (2-year studies)
Date of Last Exposure December 9, 2010 (F ₀ females) January 6-7, 2011 (F ₁ groups)	September 16, 2012 (F ₀ females) December 18 and 20 (males) or 17 and 20 (females), 2012 (interim evaluation) September 15-22 (males) or 22-30 (females), 2014 (chronic study)
Necropsy Dates January 6-7, 2011 (F ₁ groups)	December 18 and 20 (males) or 17 and 20 (females), 2012 (interim evaluation) September 15-22 (males) or 22-30 (females), 2014 (chronic study)
Age at Necropsy 7 to 8 weeks	Interim: 17 weeks Study termination: 108 to 109 weeks (males) or 109 to 110 weeks (females)
Size of Study Groups F ₀ females: 20 per exposure group F ₁ rats: 10 males and 10 females	F ₀ females: 56 per exposure group F ₁ core study: 90 males and 90 females F ₁ interim evaluation: 10 males and 10 females F ₁ genetic toxicity: five males and five females
Method of Distribution Animals were distributed randomly into groups of approximately equal initial mean body weights.	Same as 28-day studies
Animals per Cage 1 except during lactation when pups were housed with nursing dams	Same as 28-day studies
Method of Animal Identification F ₀ females: Tail marking with permanent pen F ₁ rats: Tail tattoo	F ₀ females: Tail marking with permanent pen F ₁ rats: Tail tattoo

TABLE 1
**Experimental Design and Materials and Methods in the Whole-Body Exposure Studies
of GSM- and CDMA-Modulated Cell Phone RFR**

28-Day Studies	2-Year Studies
Diet Irradiated NIH-07 rodent wafer diet (perinatal phase) or irradiated NTP-2000 rodent wafer diet (prechronic phase) (Zeigler Brothers, Inc., Gardners, PA), available <i>ad libitum</i> , glass jars changed weekly	Same as 28-day studies, except ceramic bowls
Water Tap water (Chicago municipal supply) via an adapted automatic watering system (SE Lab Group, Cincinnati, OH), available <i>ad libitum</i>	Same as 28-day studies
Cages Solid polycarbonate (Allentown Caging, Allentown, NJ), changed and rotated weekly, except rotated every 2 weeks during parturition	Same as 28-day studies
Bedding Certified, irradiated hardwood bedding (P.J. Murphy Forest Products Corp., Montville, NJ), changed weekly	Same as 28-day studies
Racks Custom-designed fiberglass cage racks (Ultra, Inc., Milwaukee, WI), changed every 2 weeks	Same as 28-day studies
Reverberation Chambers Fully-shielded, stainless steel room equipped with a stainless steel door to eliminate leakage of RFR signals, RFR excitation antennas, and two rotating stirrers; chambers were cleaned at least once weekly.	Same as 28-day studies
Reverberation Chamber Environment Temperature: $72^{\circ} \pm 3^{\circ}$ F Relative humidity: $50\% \pm 15\%$ Room incandescent light: 12 hours/day Chamber air changes: at least 10/hour	Temperature: $72^{\circ} \pm 3^{\circ}$ F Relative humidity: $50\% \pm 15\%$ Room incandescent light: 12 hours/day Chamber air changes: at least 10/hour
Exposure Concentrations Time-averaged whole-body SARs of 0 (sham control), 3, 6, and 9 W/kg GSM- or CDMA-modulated cell phone RFR	Time-averaged whole-body SARs of 0 (sham control), 1.5, 3, and 6 W/kg GSM- or CDMA-modulated cell phone RFR
Type and Frequency of Observation F_0 females: Observed twice daily. Body temperature was measured on GD 6 and within 3.5 minutes of exposure pauses at the end of the second to last “on” cycle on GDs 7, 11, and 16. Body temperature during lactation was measured within 2 minutes of exposure pauses at the end of the second to last “on” cycle on PNDs 1, 4, 7, and 14. Animals were weighed on GDs 6, 9, 12, 15, 18, and 21, and PNDs 1, 4, 7, 14, and 21. Clinical findings were recorded weekly. F_1 rats: Observed twice daily. Body temperature was measured on day 8 and within 5 minutes of exposure pauses at the end of the second to last “on” cycle on study days 16, 20, and 27. Animals were weighed during the perinatal phase on PND 1 (litter weights by sex), 4, 7, 14, and 21 and weekly during the prechronic phase. Clinical findings were recorded weekly.	F_0 females: Observed twice daily; animals were weighed on GDs 6, 9, 12, 15, 18, and 21, and on PNDs 1, 4, 7, 14, and 21. Clinical findings were recorded on GD 6 through PND 21. F_1 rats: Observed twice daily; during perinatal phase, number, sex, and viability status were determined on PND 1. Animals were weighed on PNDs 1 (litter weights by sex), 4, 7, 14, 17, and 21. During the chronic phase, animals were weighed on day 1, twice a week through week 13, at 4-week intervals during weeks 14 to 86, and then every 2 weeks from week 90 until the end of the studies. Clinical findings were recorded at 4-week intervals.

TABLE 1
Experimental Design and Materials and Methods in the Whole-Body Exposure Studies
of GSM- and CDMA-Modulated Cell Phone RFR

28-Day Studies	2-Year Studies
Method of Euthanasia Carbon dioxide asphyxiation	Same as 28-day studies
Necropsy Necropsies were performed on all rats. Organs weighed were the right adrenal gland, brain, heart, right kidney, liver, lung, right testis, and thymus.	Necropsies were performed on all rats. Organs weighed in 10 rats per exposure group at 14 weeks were the brain, heart, kidney (left and right), liver, lung, ovary (left and right), testis (left and right) with epididymis (left and right), and thymus
Clinical Pathology None	Blood was collected from the retroorbital sinus of 10 rats per group at 14 weeks for hematology and clinical chemistry. Hematology: hematocrit (auto and manual); hemoglobin concentration; erythrocyte, reticulocyte, nucleated erythrocyte, and platelet counts; mean cell volume; mean cell hemoglobin; mean cell hemoglobin concentration; and leukocyte count and differentials. Clinical chemistry: urea nitrogen, creatinine, glucose, total protein, albumin, cholesterol, triglycerides, alanine aminotransferase, alkaline phosphatase, creatine kinase, sorbitol dehydrogenase, and bile acid.
Histopathology Complete histopathology was performed on all 0 (sham control) and 9 W/kg groups. In addition to gross lesions and tissue masses, the following tissues were examined: adrenal gland, aorta, bone with marrow, brain, clitoral gland, epididymis, esophagus, eyes, Harderian gland, heart, large intestine (cecum, colon, rectum), small intestine (duodenum, jejunum, ileum), kidney, liver, lung, lymph nodes (mandibular and mesenteric), mammary gland, nose, ovary, pancreas, parathyroid gland, pituitary gland, preputial gland, prostate gland, salivary gland, seminal vesicle, skin, spleen, stomach (forestomach and glandular), testis, thymus, thyroid gland, trachea, urinary bladder, and uterus.	Complete histopathology was performed on 10 F ₁ rats from each exposure group at 14 weeks, on all rats that died early, and on all rats surviving to the end of the studies. In addition to gross lesions and tissue masses, the following tissues were examined: adrenal gland, aorta, bone with marrow, brain, clitoral gland, esophagus, eyes, Harderian gland, heart, large intestine (cecum, colon, rectum), small intestine (duodenum, jejunum, ileum), kidney, liver, lung with bronchi, lymph nodes (mandibular and mesenteric), mammary gland, muscle, nerve (sciatic, trigeminal, and peripheral), nose, ovary, pancreas, parathyroid gland, pituitary gland, preputial gland, prostate gland, salivary gland, seminal vesicle, skin, spinal cord, spleen, stomach (forestomach and glandular), testis with epididymis, thymus, thyroid gland, trachea, urinary bladder, and uterus.
Sperm Motility and Count and Vaginal Cytology None	Spermatid and sperm samples were collected from 10 male rats in each group at 14 weeks. The following parameters were evaluated: spermatid heads per testis and per gram testis, sperm motility, and sperm per cauda epididymis and per gram cauda epididymis. The left cauda, left epididymis, and left testis were weighed. Vaginal samples were collected from 10 females in each group for 16 days prior to the 14-week interim evaluation.

STATISTICAL METHODS

P values less than 0.05 were considered statistically significant.

Survival Analyses

The probability of survival was estimated by the product-limit procedure of Kaplan and Meier (1958) and is presented in the form of graphs. Animals found dead of other than natural causes or missing were censored; animals dying from natural causes were not censored. Statistical analyses for possible dose-related effects on survival used Cox's (1972) method for testing two groups for equality and Tarone's (1975) life table test to identify dose-related trends. All reported P values for the survival analyses are two sided.

Calculation of Incidence

The incidences of neoplasms or nonneoplastic lesions are presented in Tables A1, A4, B1, B4, C1, C4, D1, and D4 as the numbers of animals bearing such lesions at a specific anatomic site and the numbers of animals with that site examined microscopically. For calculation of statistical significance, the incidences of most neoplasms (Tables A2, B2, C2, and D2) and all nonneoplastic lesions are given as the numbers of animals affected at each site examined microscopically. However, when macroscopic examination was required to detect neoplasms in certain tissues (e.g., mesentery, pleura, peripheral nerve, skeletal muscle, tongue, tooth, and Zymbal's gland) before microscopic evaluation, the denominators consist of the number of animals that had a gross abnormality. When neoplasms had multiple potential sites of occurrence (e.g., leukemia or lymphoma), the denominators consist of the number of animals on which a necropsy was performed. Tables A2, B2, C2, and D2 also give the survival-adjusted neoplasm rate for each group and each site-specific neoplasm. This survival-adjusted rate (based on the Poly-3 method described below) accounts for differential mortality by assigning a reduced risk of neoplasm, proportional to the third power of the fraction of time on study that it survived, only to site-specific, lesion-free animals that do not reach terminal euthanasia.

Analysis of Neoplasm and Nonneoplastic Lesion Incidences

Statistical analyses of neoplasm and nonneoplastic lesion incidences took into account two features of the data.

Some animals did not survive the entire 2 years of the study, so survival differences between groups had to be taken into account. Also, up to three animals per sex were randomly selected from each litter to participate in the study. The statistical analysis of lesion incidences used the Poly-3 test to account for survival differences, with a Rao-Scott adjustment for litter effects, as described below.

The Poly-k test (Bailer and Portier, 1988; Portier and Bailer, 1989; Piegorsch and Bailer, 1997) was used to assess neoplasm and nonneoplastic lesion prevalence. This test is a survival-adjusted quantal-response procedure that modifies the Cochran-Armitage linear trend test to take survival differences into account. More specifically, this method modifies the denominator in the quantal estimate of lesion incidence to approximate more closely the total number of animal years at risk. For analysis of a given site, each animal is assigned a risk weight. This value is one if the animal had a lesion at that site or if it survived until terminal euthanasia; if the animal died prior to terminal euthanasia and did not have a lesion at that site, its risk weight is the fraction of the entire study time that it survived, raised to the kth power.

This method yields a lesion prevalence rate that depends only upon the choice of a shape parameter for a Weibull hazard function describing cumulative lesion incidence over time (Bailer and Portier, 1988). Unless otherwise specified, a value of k=3 was used in the analysis of site-specific lesions. This value was recommended by Bailer and Portier (1988) following an evaluation of neoplasm onset time distributions for a variety of site-specific neoplasms in control F344 rats and B6C3F₁ mice (Portier *et al.*, 1986). Bailer and Portier (1988) showed that the Poly-3 test gave valid results if the true value of k was anywhere in the range from 1 to 5. A further advantage of the Poly-3 method is that it does not require lesion lethality assumptions. Variation introduced by the use of risk weights, which reflect differential mortality, was accommodated by adjusting the variance of the Poly-3 statistic as recommended by Bieler and Williams (1993). Poly-3 tests used the continuity correction described by Nam (1987).

Because up to three pups per sex per litter were in the core study and in the 28-day study, the Poly-3 test was modified to accommodate litter effects using the Rao-Scott approach (Rao and Scott, 1992). Litter effects arise

when littermates are more similar to each other than they are to animals from other litters. If intra-litter correlations are present but ignored in the statistical analysis, the variance of the data will be underestimated, leading to P values that are too small. The Rao-Scott approach accounts for litter effects by estimating the ratio of the variance in the presence of litter effects to the variance in the absence of litter effects. This ratio is then used to adjust the sample size downward to yield the estimated variance in the presence of litter effects. The Rao-Scott approach was implemented in the Poly-3 test as recommended by Fung *et al.* (1994), formula \bar{T}_{RS2} .

Tests of significance included pairwise comparisons of each dosed group with controls and a test for an overall dose-related trend. Continuity-corrected Rao-Scott-adjusted Poly-3 tests were used in the analysis of lesion incidence, and reported P values are one sided. The significance of lower incidences or decreasing trends in lesions is represented as 1-P with the letter N added (e.g., P=0.99 is presented as P=0.01N). For neoplasms and nonneoplastic lesions observed without litter structure (eg, at the interim evaluation), Poly-3 tests that included the continuity correction, but without adjustment for potential litter effects, was used for trend and pairwise comparisons to the control group.

To evaluate litter incidences, the proportions of litters affected by each lesion type were tested among groups. Cochran-Armitage exact trend tests and Fisher exact tests (Gart *et al.*, 1979) were used to test for trends and pairwise differences from the control group, respectively.

The statistical analysis of brain gliomas and heart schwannomas reported in the NTP's Report of Partial Findings (Wyde *et al.*, 2016) differed from those presented here. In the previously reported analyses, only gliomas of the brain and schwannomas of the heart were analyzed. Because these are rare tumors and did not occur in more than one animal per litter and because effective statistical methods for litter effect adjustments had not been programmed at that point, Poly-3 and Poly-6 tests were used without adjustment for potential litter effects. The rarity of the tumors and the fact that no litter had more than one animal with the tumors indicated that the adjustment for litter effects would be negligible.

Analysis of Continuous Variables

Two approaches were employed to assess the significance of pairwise comparisons between dosed and control groups in the analysis of continuous variables. In the 28-day and 2-year studies, pup organ and body weight data, and body temperatures, which historically have approximately normal distributions, were analyzed with mixed effects linear models, for trend and pairwise tests with a Dunnett (1955)-Hsu (1992) adjustment, where litters were the random effect. Body temperatures for dams in all studies were analyzed using the parametric multiple comparison procedures of Dunnett (1955) and Williams (1971, 1972). At the 14-week interim evaluations in the 2-year studies, hematology, clinical chemistry, spermatid, and epididymal spermatozoal data, which have typically skewed distributions, were analyzed using the nonparametric multiple comparison methods of Shirley (1977) (as modified by Williams, 1986) and Dunn (1964). Litter sizes, pup survival, implantations, number of resorptions, and proportions of male pups per litter for all studies were also analyzed using these nonparametric methods. For all quantitative endpoints unaffected by litter structure, Jonckheere's test (Jonckheere, 1954) was used to assess the significance of the dose-related trends and to determine at the 0.01 level of significance, whether a trend-sensitive test (Williams' or Shirley's test) was more appropriate for pairwise comparisons than a test that does not assume a monotonic dose-related trend (Dunnett's or Dunn's test). Prior to statistical analysis, extreme values identified by the outlier test of Dixon and Massey (1957), for small samples ($n \leq 20$), and Tukey's outer fences method (Tukey, 1977), for large samples ($n > 20$), were examined by NTP personnel, and implausible values were eliminated from the analysis.

Post-weaning body weights were measured on three pups per sex per litter in the 2-year study and up to three pups per sex per litter in the 28-day study (with a total of 10 animals per dose group). More than three pups per sex per litter were possible in pre-weaning body weight measurements. The analyses of pup body weights and body weights adjusted for litter size (described below) of these animals took litter effects into account by use of mixed effects regression, where litters were the random effects. Dam body weights, dam body weights adjusted for litter size during gestation, as well as dam body weights during lactation were analyzed with the parametric multiple comparison procedures of Dunnett (1955) and Williams (1971, 1972), depending on whether Jonckheere's test indicated the use of a trend-sensitive test.

P values for these analyses are two sided.

Analysis of Gestational and Fertility Indices

Significances of trends in gestational and fertility indices across dose groups was tested using Cochran-Armitage trend tests. Pairwise comparisons of each dosed group with the control group were conducted using the Fisher exact test. P values for these analyses are two sided.

Body Weight Adjustments

Adjusted dam body weights and adjusted pup body weights were calculated to account for litter size. Dam weights measured during gestation were adjusted for litter size using gestational-day-specific analyses of variance on dam weight as a function of litter size and dose. Dam body weights were adjusted to the overall mean PND 1 total litter size of all groups under analysis, combined, and the residuals from the analyses of covariance were added back in to retain the original variances. Pre-weaning pup body weights were adjusted for PND 1 live litter size using the same analysis of covariance approach, with the additional random effect of litter added to the models to account for litter effects. Although the same sham control group was used to analyze GSM and CDMA exposed groups, adjusted body weights for the sham control group differ between GSM and CDMA because the overall mean PND 1 live litter size differs between the GMS and CDMA analyses. Post-weaning pup body weights were adjusted using a random effect of litter to account for litter effects without accounting for overall mean PND 1 litter size.

Historical Control Data

The historical control Hsd:Sprague Dawley SD rat data used in the current studies are limited to data obtained from three recent finalized studies and differ from the historical control data provided in NTP's Report of Partial Findings (Wyde *et al.*, 2016). When the NTP's Report of Partial Findings was released very limited data were available describing the prevalence of glial and Schwann cell lesions in Harlan Sprague Dawley rats in NTP studies. Consequently, an effort was made to review control groups from as many comparable studies as possible regardless of whether they had been subjected to peer review.

In NTP's Report of Partial Findings, control groups of male Harlan Sprague Dawley rats from the cell phone RFR studies and nine (for brain) and 12 (for heart) other recently completed NTP studies were tabulated to increase the sample size of rats from which control rates of malignant gliomas of the brain or schwannoma of the heart could be determined. For evaluation of the heart lesions, the 12 studies included black cohosh, resveratrol, sodium tungstate dehydrate, tris(chloropropyl) phosphate, indole-3-carbinol, perfluorooctanoic acid, dietary zinc, *p*-chloro- α,α,α -trifluorotoluene, dibutyl phthalate, 2-hydroxy-4-methoxy-benzophenone, diethylhexyl phthalate (2 studies). Three fewer studies were available for evaluation of brain lesions than for the evaluation of heart lesions because of a recent change by the NTP to increase the standard number of examined sections from three to seven. Because the sectioning in these three studies differed, the studies with three brain sections (indole-3-carbinol, perfluorooctanoic acid, and dietary zinc) were excluded from evaluation of brain lesions. For studies in which the in-life portion was completed, but the final pathology data were not yet available, special reviews of the control rat brains for malignant gliomas and hearts for schwannomas were performed.

QUALITY ASSURANCE METHODS

The 28-day and 2-year studies were conducted in compliance with Food and Drug Administration Good Laboratory Practice Regulations (21 CFR, Part 58). In addition, the 28-day and 2-year study reports were audited retrospectively by an outside independent QA contractor against study records submitted to the NTP Archives. Separate audits covered completeness and accuracy of the pathology data, pathology specimens, final pathology tables, and a draft of this NTP Technical Report. Audit procedures and findings are presented in the reports and are on file at NIEHS. The audit findings were reviewed and assessed by NTP staff, and all comments were resolved or otherwise addressed during the preparation of this Technical Report.

GENETIC TOXICOLOGY

The genetic toxicity of GSM- and CDMA-modulated cell phone RFR was assessed by measuring the frequency of micronucleated erythrocytes in peripheral blood and DNA damage in five different tissues of male and female rats following 14 weeks of exposure. Micronuclei (literally "small nuclei" or Howell-Jolly bodies) are biomarkers of induced structural or numerical chromosomal alterations and are formed when acentric fragments or whole

chromosomes fail to incorporate into either of two daughter nuclei during cell division (Schmid, 1975; Heddle *et al.*, 1983). The alkaline (pH>13) comet assay (OECD, 2014) (also known as the single cell gel electrophoresis assay) detects DNA damage in any of a variety of eukaryotic cell types (Tice *et al.*, 2000; Collins, 2004; Brendler-Schwaab *et al.*, 2005; Burlinson *et al.*, 2007); cell division is not required. The type of DNA damage detected includes nicks, adducts, strand breaks, and abasic sites that are converted to DNA strand breaks after treatment of cells in an alkaline (pH>13) solution. Transient DNA strand breaks generated by the process of DNA excision repair may also be detected. DNA damage caused by crosslinking agents has been detected as a reduction of DNA migration (Pfuhler and Wolf, 1996; Hartmann *et al.*, 2003). The fate of the DNA damage detected by the comet assay is varied; most of the damage is rapidly repaired resulting in no sustained impact on the tissue but some may result in cell death or may be incorrectly processed by repair proteins and result in a fixed mutation or chromosomal alteration. The detailed protocols for these studies and the results are given in Appendix E.

The genetic toxicity studies have grown out of an earlier effort by the NTP to develop a comprehensive database permitting a critical anticipation of a test article's carcinogenicity in experimental animals based on the results from a number of *in vitro* and *in vivo* short-term tests measuring functionally distinct genotoxicity endpoints. The short-term tests were originally developed to clarify proposed mechanisms of chemical-induced DNA damage based on the relationship between electrophilicity and mutagenicity (Miller and Miller, 1977) and the somatic mutation theory of cancer (Straus, 1981; Crawford, 1985). However, it should be noted that not all cancers arise through genotoxic mechanisms, and in these studies, the test article is not a chemical. Many studies have established the genotoxicity of some forms of radiation including, for example, UV light radiation and X-ray radiation, which are both forms of ionizing radiation.

Clearly positive results in long-term peripheral blood micronucleus tests have high predictivity for rodent carcinogenicity; a weak response in one sex only or negative results in both sexes in this assay do not correlate well with either negative or positive results in rodent carcinogenicity studies (Witt *et al.*, 2000). The relationship between comet assay results and rodent carcinogenicity was investigated previously and a close association was observed (Sasaki *et al.*, 2000); however, this assay is best employed as a hazard identification assay. Because of the theoretical and observed associations between induced genetic damage and adverse effects in somatic and germ

cells, the determination of *in vivo* genetic effects is important to the overall understanding of the risks associated with exposure to a particular test article.

RESULTS

GSM

28-DAY STUDY

Perinatal Exposure

No exposure-related effects were observed on survival or littering rates (littering/pregnant ratio) (Table 2).

Gestation body weights were unaffected by exposure to GSM-modulated cell phone RFR by pairwise comparison (Table 3). A significant negative trend was observed in gestation day (GD) 21 body weights that was likely due to reduced body weight gain in late gestation in the 9 W/kg group (Table 3). There was an overall (GD 6 to GD 21) lower body weight gain of 9% in the 9 W/kg group compared to that of the sham controls.

TABLE 2
Summary of Disposition During Perinatal Exposure and F₁ Allocation
in the 28-Day Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Time-mated Females	20	20	20	20
Pregnant Females	20	19	18	20
Non-Pregnant Females	0	1	2	0
Pregnant Dams not Delivering	0	0	0	0
Died	0	0	0	0
Littered	20	19	18	20
Pregnant/Mated Percentage ^a	100.0%	95.0%	90.0%	100.0%
Littered/Pregnant Percentage ^a	100.0%	100.0%	100.0%	100.0%
Litters Removed (Insufficient Size) (PND 4)	0	0	0	0
Litters Post Standardization (PND 4)	20	19	18	20
Weaned/Sex (PND 21) ^b	30	30	30	30

^a Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparisons against sham control group

^b Total number of weaned animals per sex from 10 litters

TABLE 3

Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Gestation in the 28-Day Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Gestation Day				
6	238.3 ± 2.2 (20) ^b	236.7 ± 2.4 (19)	238.8 ± 2.6 (18)	238.3 ± 2.3 (20)
9	250.7 ± 2.4 (20)	249.8 ± 2.2 (19)	251.4 ± 2.5 (18)	249.8 ± 2.5 (20)
12	266.2 ± 2.5 (20)	263.0 ± 2.3 (19)	265.9 ± 2.6 (18)	264.2 ± 2.6 (20)
15	282.5 ± 2.9 (20)	280.7 ± 2.8 (19)	283.0 ± 2.9 (18)	281.3 ± 2.5 (20)
18	319.3 ± 3.0 (20)	316.9 ± 3.2 (19)	319.4 ± 3.6 (18)	313.6 ± 2.6 (20)
21	366.4 ± 4.3 (20) ^{▲▲}	360.2 ± 4.8 (19)	363.6 ± 4.5 (18)	354.8 ± 3.3 (20)
Gestation Day Interval				
6 to 9	12.5 ± 1.2 (20)	13.1 ± 0.9 (19)	12.6 ± 1.1 (18)	11.5 ± 0.5 (20)
9 to 12	15.5 ± 1.1 (20)	13.3 ± 0.8 (19)	14.5 ± 0.6 (18)	14.4 ± 0.7 (20)
12 to 15	16.3 ± 1.0 (20)	17.7 ± 1.0 (19)	17.1 ± 0.5 (18)	17.1 ± 0.6 (20)
15 to 18	36.7 ± 0.9 (20) ^{▲▲}	36.2 ± 1.3 (19)	36.4 ± 1.1 (18)	32.3 ± 0.8 (20)**
18 to 21	47.1 ± 1.8 (20) ^{▲▲}	43.3 ± 2.0 (19)	44.2 ± 1.3 (18)	41.2 ± 1.5 (20)*
6 to 21	128.1 ± 3.3 (20) ^{▲▲}	123.5 ± 4.3 (19)	124.8 ± 3.0 (18)	116.5 ± 2.6 (20)*

^{▲▲}Significant trend ($P \leq 0.01$) by Jonckheere's test

* Significantly different ($P \leq 0.05$) from the sham control group by Williams' test

** $P \leq 0.01$

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

^b Number of dams

Total and live litter size on postnatal day (PND) 1 was unaffected by exposure and there was no statistically significant effect on live litter size throughout lactation (Table 4). However, there were higher numbers of dead pups in the exposed groups from PND 1 to 4 and single incidences in the 6 and 9 W/kg groups from PND 5 to 21 (Table 5). The number of dead pups per litter was significantly increased from PND 1 to 4 in addition to a decreased survival ratio in the 9 W/kg group.

TABLE 4

**Mean Number of Surviving F₁ Male and Female Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Total Pups per Litter				
PND 1	11.95 ± 0.38 (20) ^b	11.74 ± 0.55 (19)	12.78 ± 0.37 (18)	12.40 ± 0.48 (20)
Live Pups per Litter				
PND 1	11.90 ± 0.39 (20)	11.63 ± 0.54 (19)	12.78 ± 0.37 (18)	12.20 ± 0.51 (20)
PND 4 (Preculling)	11.85 ± 0.39 (20)	11.53 ± 0.54 (19)	12.44 ± 0.37 (18)	11.45 ± 0.51 (20)
PND 4 (Postculling)	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.94 ± 0.06 (18)	7.90 ± 0.07 (20)
PND 7	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.90 ± 0.07 (20)
PND 10	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.90 ± 0.07 (20)
PND 14	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.85 ± 0.08 (20)
PND 17	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.85 ± 0.08 (20)
PND 21	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.85 ± 0.08 (20)
Live Males per Litter				
PND 1	6.10 ± 0.42 (20)	5.95 ± 0.39 (19)	6.72 ± 0.52 (18)	5.80 ± 0.55 (20)
PND 4 (Preculling)	6.05 ± 0.41 (20)	5.74 ± 0.38 (19)	6.50 ± 0.52 (18)	5.45 ± 0.54 (20)
PND 4 (Postculling)	4.00 ± 0.15 (20)	3.95 ± 0.18 (19)	3.94 ± 0.13 (18)	3.75 ± 0.24 (20)
Live Females per Litter				
PND 1	5.80 ± 0.42 (20)	5.68 ± 0.36 (19)	6.06 ± 0.45 (18)	6.40 ± 0.60 (20)
PND 4 (Preculling)	5.80 ± 0.43 (20)	5.79 ± 0.37 (19)	5.94 ± 0.40 (18)	6.00 ± 0.50 (20)
PND 4 (Postculling)	3.95 ± 0.14 (20)	3.89 ± 0.11 (19)	4.00 ± 0.14 (18)	4.15 ± 0.24 (20)

^a All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.

^b Number of dams

TABLE 5

**Offspring Mortality and Survival Ratio of Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Pup Survival per Litter				
Total Dead PND 1 to 4 ^b	2 (238/20)	4 (221/19)	6 (230/18)	19 (244/20)
Total Dead PND 5 to 21	0 (159/20)	0 (149/19)	1 (143/18)	1 (158/20)
Dead/Litter PND 1 to 4	0.100 ± 0.069 (20)**	0.211 ± 0.123 (19)	0.333 ± 0.229 (18)	0.950 ± 0.223 (20)**
Dead/Litter PND 4 to 21	0.000 ± 0.000 (20)	0.000 ± 0.000 (19)	0.056 ± 0.056 (18)	0.050 ± 0.050 (20)
Survival Ratio PND 1 to 4 ^c	0.996 ± 0.004 (20)**	0.991 ± 0.006 (19)	0.976 ± 0.016 (18)	0.940 ± 0.014 (20)**
Survival Ratio PND 4 to 21 ^d	1.000 ± 0.000 (20)	1.000 ± 0.000 (19)	0.993 ± 0.007 (18)	0.994 ± 0.006 (20)

** Significantly different ($P \leq 0.01$) from the sham control by Shirley's or Dunn's test

^a All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.

^b Number of pups/number of dams

^c Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1)

^d Number of pups alive on PND 21/number of pups at postculling on PND 4

Exposed dams had decreased weight gain during lactation (PND 1-21), and maternal body weights of the 9 W/kg group were up to 1% lower than those of the sham controls (Table 6). Combined F₁ body weights were 8% lower starting on PND 1 in the 9 W/kg group when adjusted for litter size (Table 7). As lactation progressed, the adjusted pup weights (combined) were up to 17% lower in the 9 W/kg group and up to 8% lower in the 6 W/kg group compared to sham controls. The magnitude of the effect was consistent between males and females.

TABLE 6
Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Postnatal Day				
1	272.7 ± 2.7 (20) ^b	267.8 ± 2.3 (19)	271.6 ± 2.7 (18)	263.5 ± 2.5 (20)*
4	263.8 ± 3.6 (20)	265.1 ± 2.9 (19)	269.9 ± 4.2 (18)	264.1 ± 2.2 (20)
7	284.1 ± 2.7 (20)**	280.3 ± 1.9 (19)	281.8 ± 2.7 (18)	270.0 ± 2.3 (20)**
14	292.4 ± 2.6 (20)**	289.9 ± 2.9 (19)	286.4 ± 2.8 (18)	266.1 ± 2.6 (20)**
21	279.7 ± 3.5 (20)**	267.3 ± 3.7 (19)**	265.8 ± 3.6 (11)**	248.5 ± 2.3 (20)**
Postnatal Day Interval				
1 to 4	-8.9 ± 3.2 (20)*	-2.7 ± 2.8 (19)	-1.7 ± 2.8 (18)	0.6 ± 1.5 (20)*
4 to 7	20.2 ± 2.3 (20)**	15.2 ± 2.0 (19)	12.0 ± 2.6 (18)**	5.9 ± 1.1 (20)**
7 to 14	8.3 ± 1.4 (20)**	9.6 ± 2.1 (19)	4.6 ± 2.0 (18)	-4.0 ± 1.5 (20)**
14 to 21	-12.7 ± 3.5 (20)	-22.7 ± 3.2 (19)*	-16.9 ± 3.1 (11)	-17.6 ± 1.7 (20)
1 to 21	7.0 ± 3.8 (20)**	-0.5 ± 2.6 (19)	-1.9 ± 2.5 (11)	-15.0 ± 1.8 (20)**

* Significantly different ($P \leq 0.05$) from the sham control by Williams' or Dunnett's test

** Significantly different ($P \leq 0.01$) from the sham control by Williams' or Dunnett's test

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

^b Number of dams

TABLE 7
**Adjusted Mean Body Weights of F₁ Male and Female Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Adjusted Male Live Pup Weight				
PND 1 ^b	6.70 ± 0.10 (20) ^{c▲▲}	6.71 ± 0.09 (19)	6.46 ± 0.10 (18)*	5.99 ± 0.09 (20) ^{▲▲}
PND 4 (Preculling)	9.68 ± 0.14 (121/20) ^{d***}	9.59 ± 0.13 (109/19)	9.08 ± 0.13 (117/18)*	8.09 ± 0.19 (108/20)**
PND 4 (Postculling)	9.75 ± 0.13 (81/20)**	9.67 ± 0.14 (74/19)	9.18 ± 0.13 (71/18)*	8.20 ± 0.16 (75/20)**
PND 7	16.22 ± 0.19 (81/20)**	15.56 ± 0.21 (74/19)	14.84 ± 0.28 (71/18)**	13.06 ± 0.31 (75/20)**
PND 14	31.52 ± 0.42 (81/20)**	31.29 ± 0.31 (74/19)	30.05 ± 0.42 (71/18)*	26.63 ± 0.47 (75/20)**
PND 21	53.19 ± 0.72 (81/20)**	52.86 ± 0.61 (74/19)	51.20 ± 0.71 (71/18)	45.52 ± 0.94 (75/20)**
Adjusted Female Live Pup Weight				
PND 1	6.28 ± 0.10 (20) ^{▲▲}	6.43 ± 0.07 (19)	6.17 ± 0.11 (18)	5.89 ± 0.09 (20) ^{▲▲}
PND 4 (Preculling)	9.09 ± 0.15 (116/20)**	9.16 ± 0.14 (110/19)	8.76 ± 0.16 (107/18)	7.95 ± 0.18 (121/20)**
PND 4 (Postculling)	9.13 ± 0.16 (79/20)**	9.23 ± 0.14 (73/19)	8.74 ± 0.16 (73/18)	8.11 ± 0.17 (83/20)**
PND 7	15.17 ± 0.24 (79/20)**	14.91 ± 0.23 (73/19)	13.96 ± 0.30 (72/18)**	12.95 ± 0.31 (83/20)**
PND 14	29.76 ± 0.40 (79/20)**	30.03 ± 0.36 (73/19)	28.67 ± 0.48 (72/18)	26.41 ± 0.42 (82/20)**
PND 21	49.78 ± 0.73 (79/20)**	49.45 ± 0.58 (73/19)	48.47 ± 0.76 (72/18)	44.25 ± 0.87 (82/20)**
Adjusted Combined Live Pup Weight				
PND 1	6.48 ± 0.10 (20) ^{▲▲}	6.57 ± 0.08 (19)	6.33 ± 0.10 (18)	5.95 ± 0.08 (20) ^{▲▲}
PND 4 (Preculling)	9.38 ± 0.13 (237/20)**	9.38 ± 0.14 (219/19)	8.94 ± 0.13 (224/18)	8.05 ± 0.17 (229/20)**
PND 4 (Postculling)	9.44 ± 0.13 (160/20)**	9.45 ± 0.14 (147/19)	8.96 ± 0.14 (144/18)	8.16 ± 0.16 (158/20)**
PND 7	15.69 ± 0.19 (160/20)**	15.24 ± 0.21 (147/19)	14.40 ± 0.27 (143/18)**	13.00 ± 0.30 (158/20)**
PND 14	30.62 ± 0.39 (160/20)**	30.66 ± 0.32 (147/19)	29.35 ± 0.43 (143/18)	26.51 ± 0.43 (157/20)**
PND 21	51.46 ± 0.67 (160/20)**	51.17 ± 0.55 (147/19)	49.84 ± 0.70 (143/18)	44.87 ± 0.88 (157/20)**

▲▲Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

* Significantly different ($P \leq 0.05$) for PNDs after PND 1 endpoints (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

^a Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND1 refer to the total pup weight divided by the number of pups in litter at PND1, and the statistical analysis performed is by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. Individual pup body weights first adjusted for live PND1 litter size via the analysis of covariance.

** Significantly different ($P \leq 0.01$)

^b Values listed as PND1 refer to the total pup weight divided by the number of pups in litter at PND1

^c Number of dams

^d Number of pups/number of dams

Postnatal Exposure

All rats survived to the end of the study (Table 8). There were lower mean body weights in the male 6 (6% to 9%) and 9 (16% to 19%) W/kg groups compared to sham controls at all time points including terminal sacrifice (Table 8 and Figure 4). Mean body weight gains were also lower in these groups (10% to 16%) (Data not presented). In 3 W/kg males, mean body weights were lower on day 22 (5%) and at terminal sacrifice (7%), but body weight gains were comparable to that of the sham controls. In females, mean body weights were lower on days 1, 8, 15, and 22 in the 9 W/kg group (8% to 11%) and on days 8 and 15 in the 6 W/kg group (5%). However, mean body weights at terminal sacrifice and mean body weight gains were similar to those of the sham controls in all exposed female groups. There were no notable clinical observations in either sex during the study.

TABLE 8
Mean Body Weights and Survival of Rats Exposed to GSM-Modulated Cell Phone RFR for 28 Days

Day	Sham Control		3 W/kg			6 W/kg			9 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
Male											
1	60.8	10	60.3	99.1	10	57.4	94.3	11	50.8	83.5	10
8	94.9	10	91.3	96.3	10	87.1	91.9	11	77.3	81.5	10
15	144.5	10	137.2	94.9	10	132.4	91.6	11	117.7	81.4	10
22	195.3	10	184.7	94.5	10	178.0	91.1	11	158.6	81.2	10
29	248.7	10	231.3	93.0	10	227.0	91.3	10	204.6	82.3	10
Female											
1	55.9	10	54.4	97.3	10	53.7	96.1	10	49.6	88.8	10
8	83.1	10	80.0	96.2	10	78.9	94.9	10	73.6	88.5	10
15	119.8	10	114.8	95.8	10	113.9	95.1	10	107.5	89.7	10
22	146.5	10	142.9	97.6	10	143.1	97.7	10	134.6	91.9	10
30	166.5	10	163.1	98.0	10	168.0	100.9	10	155.7	93.5	10

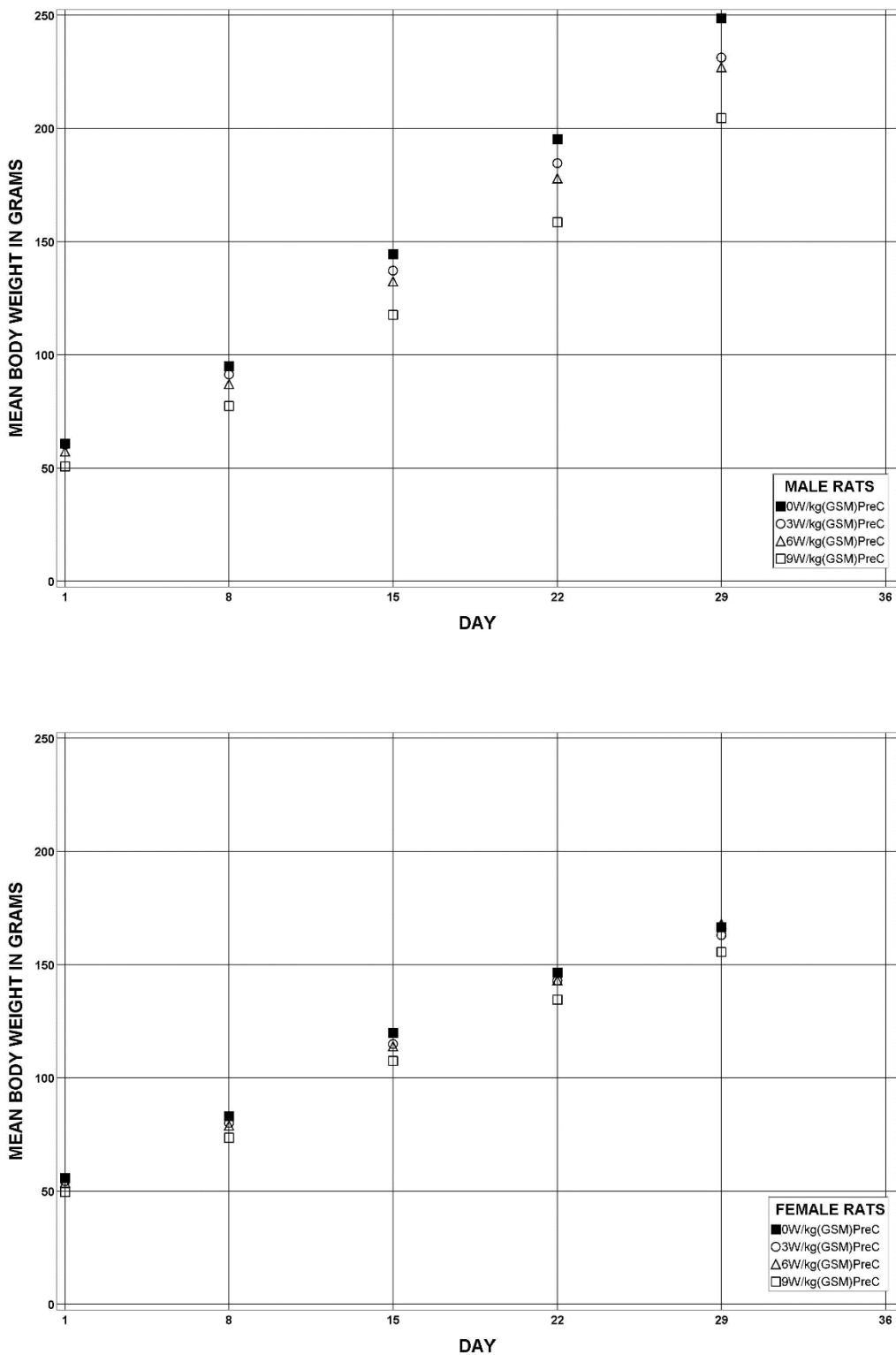


FIGURE 4
Growth Curves for Rats Exposed to GSM-Modulated Cell Phone RFR for 28 Days

The average temperature over gestation (GD 7-16) was increased compared to controls in the 6 and 9 W/kg groups by 0.4 and 0.5 degrees respectively. During lactation, the average (LD 1 -14) temperature was also increased in the 6 and 9 W/kg groups by 0.7 and 1.0 degrees respectively (Table 9). In the F1 offspring, average (GD 16-27; ~PND 37-48) body temperatures were decreased by 0.5 degrees in the 3 W/kg male group and decreased in the 6 W/kg female group by 0.9 degrees.

TABLE 9
Mean Body Temperatures of Rats Exposed to GSM-Modulated Cell Phone RFR for 28 Days^a

Day	Sham Control		3 W/kg		6 W/kg		9 W/kg	
	Temperature (° C)	No. Measured						
F₀ Female^b								
GD 6	36.7 ± 0.1	10 ^c	37.4 ± 0.2**	9	36.5 ± 0.1	9	36.8 ± 0.2	10
GD 7	36.6 ± 0.1**	10	36.7 ± 0.1	9	37.1 ± 0.1*	9	37.2 ± 0.1**	10
GD 11	36.7 ± 0.2**	10	36.5 ± 0.1	9	37.1 ± 0.1	9	37.2 ± 0.1*	10
GD 16	36.5 ± 0.1**	10	36.5 ± 0.1	9	36.8 ± 0.1	9	37.0 ± 0.1**	10
GD 7-16 ^d	36.6 ± 0.1**	10	36.6 ± 0.1	9	37.0 ± 0.1**	9	37.1 ± 0.0**	10
LD 1	37.7 ± 0.1**	10	37.8 ± 0.1	9	38.1 ± 0.2	9	38.4 ± 0.2**	10
LD 4	36.7 ± 0.1**	10	37.1 ± 0.2	9	37.5 ± 0.2**	9	37.9 ± 0.1**	10
LD 7	36.8 ± 0.2	10	37.1 ± 0.2	9	37.2 ± 0.2	9	37.2 ± 0.2	10
LD 14	36.9 ± 0.2	10	37.1 ± 0.1	9	37.8 ± 0.2**	9	38.3 ± 0.2**	8
LD 1-14 ^d	37.0 ± 0.1**	10	37.3 ± 0.1	9	37.7 ± 0.1**	9	38.0 ± 0.1**	10
F₁ Male^c								
16	37.3 ± 0.1	4	37.1 ± 0.1	4	37.3 ± 0.1	4	37.3 ± 0.1	4
20	37.6 ± 0.1	4	37.0 ± 0.1**	4	37.3 ± 0.1	4	37.4 ± 0.1	4
27	37.2 ± 0.1	4	37.0 ± 0.1	4	37.2 ± 0.1	3	37.4 ± 0.1	4
16-27 ^d	37.4 ± 0.1	4	37.0 ± 0.1*	4	37.3 ± 0.1	4	37.4 ± 0.1	4
F₁ Female^c								
16	37.9 ± 0.2	4	37.0 ± 0.2**	4	37.1 ± 0.1*	4	37.4 ± 0.1	4
20	38.0 ± 0.2	4	37.5 ± 0.2	4	37.1 ± 0.1**	4	37.6 ± 0.2	4
27	37.9 ± 0.2	4	38.0 ± 0.2	4	37.4 ± 0.3	4	37.6 ± 0.2	4
16-27 ^d	37.9 ± 0.1*	4	37.5 ± 0.1	4	37.2 ± 0.1**	4	37.5 ± 0.1	4

* Significantly different ($P \leq 0.05$)

** Significantly different ($P \leq 0.01$)

^a Temperatures are given as mean ± standard error. GD=gestation day; LD=lactation day.

^b Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

^c Statistical analysis for linear trends was performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.

^d Average of days

Male body weights were 8%, 10%, and 20% lower in the 3 W/kg, 6 W/kg, and 9 W/kg groups respectively compare to the sham controls at necropsy. Relative brain and testis weights were increased in males, but this was considered

to be due to the lower body weights and not an exposure related effect on the organ. Significant decreases in absolute heart weight were observed in the 6 W/kg and 9 W/kg (14 and 22% lower compared to the controls respectively), but relative heart weight was not affected. Similarly, right kidney and liver weights were also decreased in the 9 W/kg group (24 and 19% lower), but without a corresponding effect in relative weights. These decreases in organ weights could be related to the observed lower body weights (Table G2). No effects were observed in female rats (Table G2).

In females, there were higher incidences of chronic progressive nephropathy in the kidney of 3 and 9 W/kg groups (sham controls, 0/10; 3 W/kg, 4/10; 6 W/kg, 3/10; 9 W/kg, 4/10) compared to controls. The severity of these lesions was minimal (1.0). Chronic progressive nephropathy was characterized by scattered tubular segments with basophilic epithelial cells with crowded nuclei, slightly thickened basement membranes, and occasional mononuclear inflammatory cells. There were no exposure-related renal lesions in male rats.

Exposure Level Selection Rationale: Based on pup mortality, reduced maternal and pup body weights, and increased body temperature measurements at 9 W/kg in the 28-day studies and increased body temperature in adult rats at ≥ 8 W/kg in the thermal pilot studies (Wyde *et al.*, 2018), the highest exposure level selected for the 2-year studies was 6 W/kg. In the thermal pilot studies and in the 28-day study, exposure to 6 W/kg resulted in some increases in core body temperature, but these increases were less than 1° C. Therefore, 6 W/kg would provide an exposure adequate to challenge the animals without causing excessive heating or disruption of the thermoregulatory process. The lowest exposure level selected for the 2-year studies was 1.5 W/kg, which is close to the 1.6 W/kg maximum output limit for cell phone devices in the United States.

2-YEAR STUDY

Perinatal Exposure

No exposure-related effects were observed on pregnancy status, maternal survival, or the percent of pregnant animals that littered (Table 10). Maternal body weights during gestation were similar to those of the sham control group (Table 11). Body weight gains were generally unaffected across time intervals except in the 6 W/kg group at the GD 15 through 18 interval where body weight gain was 10% lower than that of the sham control group and the GD 6 through 21 interval where body weight gain was 7% lower than that of the sham control group.

TABLE 10
Summary of Disposition During Perinatal Exposure and F₁ Allocation
in the 2-Year Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Time-mated Females	56	56	56	56
Pregnant Females	52	50	50	52
Non-Pregnant Females	4	6	6	4
Pregnant Dams not Delivering	2	3	3	4
Died ^a	1	0	0	0
Littered	50	47	47	48
Pregnant/Mated Percentage ^b	92.9%	89.3%	89.3%	92.9%
Littered/Pregnant Percentage ^b	96.2%	94.0%	94.0%	92.3%
Litters Removed (Insufficient Size)	2	4	5	2
Litters Post Standardization	48	43	42	46
Weaned/Sex ^c	105	105	105	105

^a One pregnant female died on GD 25 with pups in uterus

^b Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparison against control group

^c Total number of weaned animals per sex from 35 litters

TABLE 11
**Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Gestation
in the 2-Year Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Gestation Day				
6	238.4 ± 1.4 (51) ^b	239.9 ± 1.4 (47)	239.0 ± 1.4 (47)	238.9 ± 1.3 (48)
9	256.2 ± 1.6 (51)	256.3 ± 1.6 (47)	254.9 ± 1.6 (47)	254.7 ± 1.3 (48)
12	270.5 ± 1.6 (51)	270.4 ± 1.7 (47)	269.2 ± 1.6 (47)	268.1 ± 1.4 (48)
15	290.0 ± 1.9 (51)	289.7 ± 2.0 (47)	288.8 ± 1.8 (47)	287.6 ± 1.6 (48)
18	332.7 ± 2.3 (51) [▲]	329.8 ± 2.6 (47)	328.9 ± 2.2 (47)	326.1 ± 2.1 (48)
21	380.2 ± 2.8 (51) [▲]	376.9 ± 3.6 (47)	374.6 ± 3.5 (47)	371.2 ± 3.0 (48)
Gestation Day Interval				
6 to 9	17.7 ± 0.8 (51)	16.4 ± 0.6 (47)	15.9 ± 0.6 (47)	15.8 ± 0.5 (48)
9 to 12	14.3 ± 0.6 (51)	14.1 ± 0.5 (47)	14.2 ± 0.5 (47)	13.4 ± 0.5 (48)
12 to 15	19.6 ± 0.6 (51)	19.3 ± 0.8 (47)	19.7 ± 0.6 (47)	19.5 ± 0.6 (48)
15 to 18	42.7 ± 1.0 (51) ^{▲▲}	40.2 ± 1.1 (47)	40.1 ± 0.9 (47)	38.5 ± 0.9 (48)**
18 to 21	47.5 ± 1.0 (51)	47.1 ± 1.5 (47)	45.6 ± 1.5 (47)	45.1 ± 1.3 (48)
6 to 21	141.7 ± 2.2 (51) ^{▲▲}	137.0 ± 3.3 (47)	135.5 ± 2.9 (47)	132.3 ± 2.6 (48)*

[▲] Significant trend ($P \leq 0.05$) by Jonckheere's test

^{▲▲} Significant trend ($P \leq 0.01$) by Jonckheere's test

* Significantly different ($P \leq 0.05$) from the sham control by Williams' or Dunnett's test

** $P \leq 0.01$

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests

^b Number of dams

Total litter size on PND 1 and live litter size at all time points were unaffected by exposure with no effects observed in pup mortality or survival ratio in early postnatal development (PND 1 through PND 4) or thereafter (PND 4 through PND 21) (Tables 12 and 13).

TABLE 12
Mean Number of Surviving F₁ Male and Female Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total Pups per Litter				
PND 1	12.76 ± 0.32 (50) ^b	12.06 ± 0.43 (47)	12.26 ± 0.51 (47)	12.31 ± 0.39 (48)
Live Pups per Litter				
PND 1	12.56 ± 0.40 (50)	12.04 ± 0.43 (47)	12.23 ± 0.51 (47)	12.29 ± 0.39 (48)
PND 4 (Preculling)	12.73 ± 0.30 (48)	12.65 ± 0.26 (43)	12.98 ± 0.27 (42)	12.41 ± 0.32 (46)
PND 4 (Postculling)	8.00 ± 0.00 (48)	8.00 ± 0.00 (43)	8.00 ± 0.00 (42)	8.00 ± 0.00 (46)
PND 7	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.95 ± 0.03 (42)	7.98 ± 0.02 (46)
PND 10	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
PND 14	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
PND 17	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
PND 21	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
Live Males per Litter				
PND 1	6.20 ± 0.30 (50)	6.00 ± 0.34 (47)	6.11 ± 0.38 (47)	6.02 ± 0.32 (48)
PND 4 (Preculling)	6.33 ± 0.28 (48)	6.35 ± 0.30 (43)	6.62 ± 0.32 (42)	6.15 ± 0.30 (46)
PND 4 (Postculling)	3.96 ± 0.05 (48)	3.98 ± 0.07 (43)	4.00 ± 0.07 (42)	4.02 ± 0.10 (46)
Live Females per Litter				
PND 1	6.36 ± 0.28 (50)	6.04 ± 0.32 (47)	6.13 ± 0.33 (47)	6.27 ± 0.33 (48)
PND 4 (Preculling)	6.40 ± 0.25 (48)	6.30 ± 0.26 (43)	6.36 ± 0.30 (42)	6.26 ± 0.34 (46)
PND 4 (Postculling)	4.04 ± 0.05 (48)	4.02 ± 0.07 (43)	4.00 ± 0.07 (42)	3.98 ± 0.10 (46)

^a All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere (trend) and Shirley's or Dunn's (pairwise) tests

^b Number of dams

TABLE 13
Offspring Mortality and Survival Ratio of Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pup Survival per Litter				
Total Dead PND 1 to 4 ^b	19 (628/49) ^c	4 (566/43)	12 (575/42)	10 (590/46)
Total Dead PND 5 to 21	0 (348/48)	1 (344/43)	3 (336/42)	1 (368/46)
Dead/Litter PND 1 to 4	0.388 ± 0.193 (49)	0.093 ± 0.045 (43)	0.286 ± 0.104 (42)	0.217 ± 0.076 (46)
Dead/Litter PND 4 to 21	0.000 ± 0.000 (48)	0.023 ± 0.023 (43)	0.071 ± 0.040 (42)	0.022 ± 0.022 (46)
Survival Ratio PND 1 to 4 ^d	0.986 ± 0.005 (48)	0.994 ± 0.003 (43)	0.981 ± 0.007 (42)	0.985 ± 0.005 (46)
Survival Ratio PND 4 to 21 ^e	1.000 ± 0.000 (48)	0.997 ± 0.003 (43)	0.991 ± 0.005 (42)	0.997 ± 0.003 (46)

^a All values shown as mean ± standard error; PND = postnatal day. Differences in the number of pups per litter from the sham control group are not significant by Dunn's test. Statistical analysis performed by Jonckheere (trend) and Shirley's or Dunn's (pairwise) tests.

^b Includes dead on PND 1. Survival information on PND 4 was not available for some non-acceptable litters, so these were excluded from the analysis.

^c Number of pups/number of dams

^d Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1)

^e Number of pups alive on PND 21/number of pups at postculling on PND 4

During the lactation period, maternal body weights of the 3 and 6 W/kg groups were significantly decreased (up to 5% and 9%, respectively) compared to those of sham controls from PND 4 through 21 (Table 14). At PND 1, male and female pup weights in the 6 W/kg groups were 4% to 5% less than those of the sham controls (Table 15). Male and female pup weights were also significantly decreased compared to the sham controls with a 4% to 8% decrease across most time points in the 3 W/kg groups and a 6% to 8% decrease across all time points in the 6 W/kg groups.

TABLE 14
Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Postnatal Day				
1	280.9 ± 2.0 (50) ^{b▲▲}	280.6 ± 2.0 (47)	277.0 ± 1.7 (47)	275.9 ± 1.7 (48)
4	289.7 ± 2.1 (48) ^{▲▲}	288.7 ± 2.0 (43)	284.3 ± 1.9 (42)	280.6 ± 1.8 (46)**
7	297.1 ± 2.2 (48) ^{▲▲}	293.7 ± 2.1 (43)	290.4 ± 1.9 (42)*	286.4 ± 1.9 (46)**
14	314.4 ± 2.0 (48) ^{▲▲}	310.1 ± 2.3 (43)	302.3 ± 1.8 (42)**	290.7 ± 2.2 (46)**
17	313.9 ± 2.2 (48) ^{▲▲}	309.2 ± 2.4 (43)	299.2 ± 1.8 (42)**	285.3 ± 2.5 (46)**
21	299.7 ± 2.2 (48) ^{▲▲}	295.9 ± 2.4 (43)	287.6 ± 1.8 (42)**	278.1 ± 2.4 (45)**
Postnatal Day Interval				
1 to 4	9.2 ± 0.9 (48) ^{▲▲}	8.1 ± 1.0 (43)	7.3 ± 1.1 (42)	4.8 ± 1.1 (46)**
4 to 7	7.4 ± 1.5 (48)	5.0 ± 1.2 (43)	6.1 ± 1.3 (42)	5.8 ± 0.9 (46)
7 to 14	17.4 ± 1.4 (48) ^{▲▲}	16.4 ± 1.4 (43)	11.9 ± 1.1 (42)**	4.3 ± 1.2 (46)**
14 to 17	-0.6 ± 1.0 (48) ^{▲▲}	-0.9 ± 1.4 (43)	-3.1 ± 1.0 (42)	-5.5 ± 1.1 (46)**
17 to 21	-14.1 ± 1.3 (48) ^{▲▲}	-13.3 ± 1.7 (43)	-11.6 ± 1.2 (42)	-7.1 ± 1.8 (45)**
1 to 21	19.3 ± 1.5 (48) ^{▲▲}	15.3 ± 1.3 (43)	10.6 ± 1.8 (42)**	2.4 ± 2.0 (45)**

* Significantly different ($P \leq 0.05$) for pairwise

** Significantly different ($P \leq 0.01$) for pairwise

▲ Significantly different ($P \leq 0.05$) for trend

▲▲ Significantly different ($P \leq 0.01$) for trend

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

^b Number of dams

TABLE 15
Adjusted Mean Body Weights of F₁ Male and Female Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of GSM-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted Male Live Pup Weight				
PND 1 ^d	7.22 ± 0.07 (49) ^{b▲▲}	7.18 ± 0.06 (46)	7.06 ± 0.07 (46)	6.84 ± 0.08 (48) ^{▲▲}
PND 4	10.90 ± 0.12 (190/48) ^{c**}	10.70 ± 0.12 (171/43)	10.24 ± 0.14 (168/42)**	9.90 ± 0.12 (185/46)**
PND 7	17.22 ± 0.19 (189/48)**	17.21 ± 0.19 (170/43)	15.97 ± 0.26 (166/42)**	15.64 ± 0.21 (185/46)**
PND 14	35.22 ± 0.43 (181/46)**	34.56 ± 0.35 (166/42)	33.04 ± 0.53 (162/41)**	32.37 ± 0.37 (185/46)**
PND 17	42.46 ± 0.48 (190/48)**	41.97 ± 0.43 (165/42)	40.75 ± 0.59 (162/41)*	39.79 ± 0.45 (185/46)**
PND 21	58.50 ± 0.62 (190/48)**	58.40 ± 0.61 (170/43)	56.36 ± 0.78 (166/42)	54.99 ± 0.67 (185/46)**
Adjusted Female Live Pup Weight				
PND 1 ^d	6.83 ± 0.06 (49) ^{▲▲}	6.84 ± 0.08 (47)	6.68 ± 0.09 (46)	6.54 ± 0.07 (48) ^{▲▲}
PND 4	10.46 ± 0.11 (194/48)**	10.23 ± 0.12 (172/43)	9.78 ± 0.14 (168/42)**	9.57 ± 0.15 (183/46)**
PND 7	16.49 ± 0.18 (194/48)**	16.53 ± 0.18 (172/43)	15.35 ± 0.22 (168/42)**	15.05 ± 0.23 (182/46)**
PND 14	33.89 ± 0.38 (192/48)**	33.47 ± 0.31 (165/41)	32.42 ± 0.37 (164/41)*	31.34 ± 0.38 (181/46)**
PND 17	40.82 ± 0.44 (194/48)**	40.42 ± 0.36 (168/42)	39.65 ± 0.42 (167/42)	38.30 ± 0.45 (182/46)**
PND 21	55.42 ± 0.53 (194/48)**	55.28 ± 0.45 (169/42)	54.17 ± 0.53 (167/42)	52.24 ± 0.64 (182/46)**
Adjusted Combined Live Pup Weight				
PND 1 ^d	7.03 ± 0.06 (49) ^{▲▲}	7.00 ± 0.08 (47)	6.92 ± 0.08 (47)	6.71 ± 0.07 (48) ^{▲▲}
PND 4	10.68 ± 0.11 (384/48)**	10.47 ± 0.11 (343/43)	10.01 ± 0.13 (336/42)**	9.74 ± 0.13 (368/46)**
PND 7	16.84 ± 0.18 (383/48)**	16.87 ± 0.18 (342/43)	15.66 ± 0.22 (334/42)**	15.35 ± 0.21 (367/46)**
PND 14	34.48 ± 0.40 (373/48)**	34.01 ± 0.31 (331/42)	32.70 ± 0.41 (326/42)**	31.86 ± 0.36 (366/46)**
PND 17	41.62 ± 0.46 (384/48)**	41.19 ± 0.38 (333/42)	40.20 ± 0.46 (329/42)	39.06 ± 0.43 (367/46)**
PND 21	56.93 ± 0.56 (384/48)**	56.88 ± 0.50 (339/43)	55.23 ± 0.60 (333/42)	53.65 ± 0.63 (367/46)**

▲▲ Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

* Significantly different ($P \leq 0.05$) for PNDs after PND 1 endpoints (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

** Significantly different ($P \leq 0.01$)

^a Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND1 refer to the total pup weight divided by the number of pups in litter at PND1, and the statistical analysis performed is by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. Individual pup body weights first adjusted for live PND1 litter size via the analysis of covariance.

^b Number of dams

^c Number of pups/number of dams

^d Values listed as PND1 refer to the total pup weight divided by the number of pups in litter at PND1

Postnatal Exposure

Survival

Estimates of 2-year survival probabilities for male and female rats are shown in Table 16 and in the Kaplan-Meier survival curves (Figure 5). Survival of all male exposed groups was significantly greater than that of the sham controls. Decreased survival in the sham control group was largely attributed to the higher severity of chronic progressive nephropathy in the kidney. Survival of exposed females was similar to that of the sham controls.

TABLE 16
Survival of Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Animals initially in study	105	105	105	105
14-week interim evaluation ^a	15	15	15	15
Accidental deaths ^b	1	0	0	1
Moribund	44	24	19	13
Natural deaths	20	21	21	16
Animals surviving to study termination	25	45	50	60
Percent probability of survival at end of study ^c	28	50	56	68
Mean survival (days) ^d	642	675	690	684
Survival analysis ^e	<0.001N	P=0.002N	P=0.001N	P=0.001N
Female				
Animals initially in study	105	105	105	105
14-week interim evaluation	15	15	15	15
Accidental death	1	0	0	0
Moribund	30	25	31	22
Natural deaths	11	10	11	11
Animals surviving to study termination	48 ^f	55 ^g	48 ^f	57
Percent probability of survival at end of study	54	59	53	63
Mean survival (days)	659	682	662	676
Survival analysis	P=0.300N	P=0.412N	P=0.100	P=0.226N

^a Excluded from survival analysis

^b Censored in the survival analysis

^c Kaplan-Meier determinations

^d Mean of all deaths (uncensored, censored, and terminal euthanasia)

^e The result of the life table trend test (Tarone, 1975) is in the sham control column, and the results of the life table pairwise comparisons (Cox, 1972) with the sham controls are in the exposed group columns. A negative trend or lower mortality in an exposure group is indicated by N.

^f Includes one animal that died during the last week of the study

^g Includes three animals that died during the last week of the study

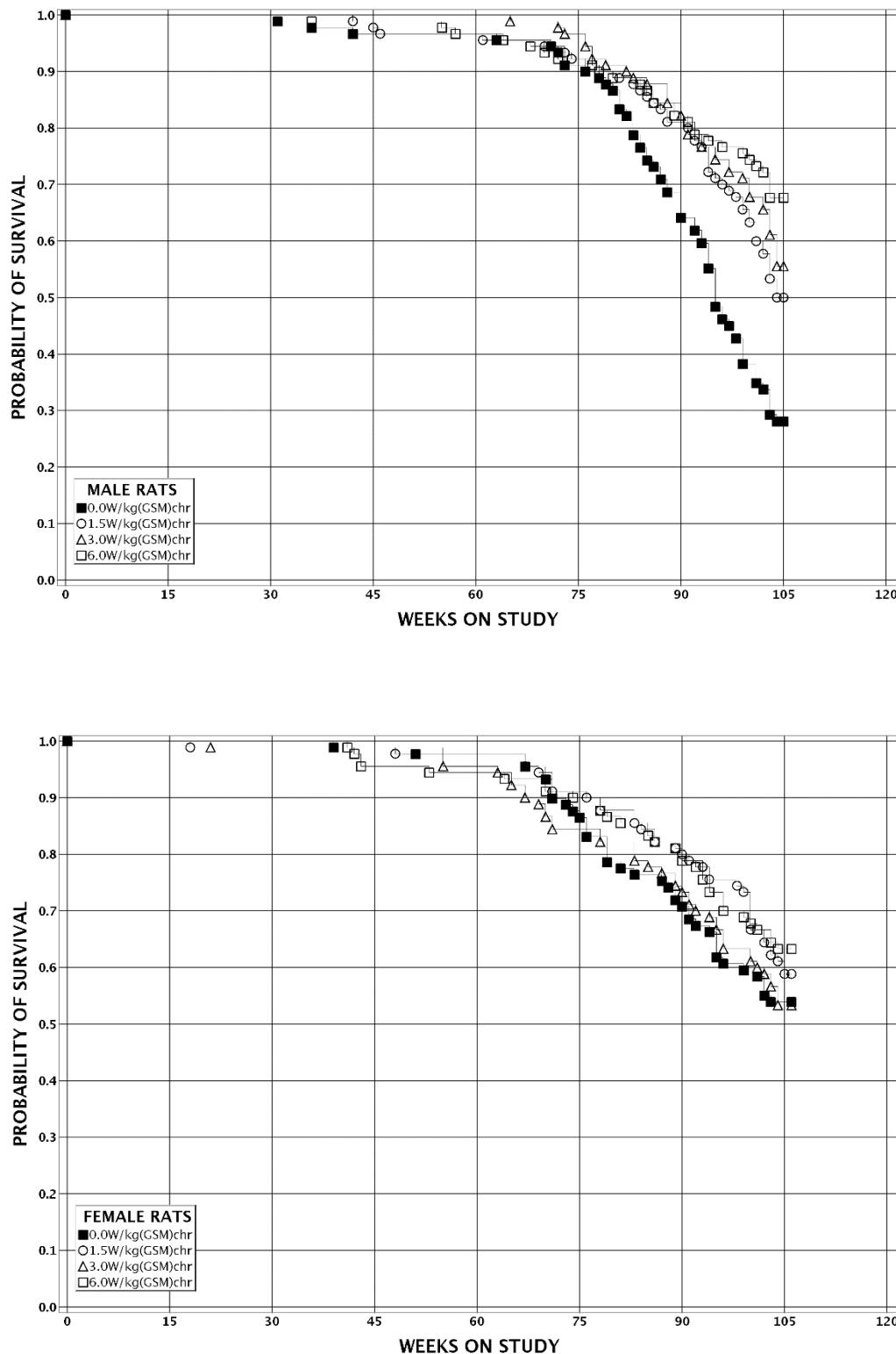


FIGURE 5
Kaplan-Meier Survival Curves for Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

Body Weights and Clinical Observations

In 6 W/kg males, mean body weights were lower (3% to 6%) at all time points through day 401 (Table 17 and Figure 6); however, from day 541 to terminal sacrifice the mean body weights were greater than those of the sham controls (up to 7.2% greater on day 681). In the 1.5 and 3 W/kg male groups, mean body weights were 5% to 7% greater than the sham controls at some time points, but the increases were sporadic. However, at the end of the study, the mean body weights of these male groups were similar to those of the sham controls. In exposed female groups, the mean body weights and mean body weight gains were similar to those of the sham controls throughout the study (Table 18 and Figure 6). There were no exposure-related clinical observations in males or females.

Table 17
Mean Body Weights and Survival of Male Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

Day	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
1	64.3	105	63.9	99.4	105	62.0	96.5	105	60.9	94.8	105
5	78.7	105	77.6	98.7	105	75.1	95.4	105	74.3	94.4	105
9	99.0	105	98.1	99.1	105	94.9	95.8	105	93.9	94.9	105
12	117.6	105	115.7	98.4	105	112.9	96.0	105 ^a	111.6	94.9	105
16	145.5	105	143.1	98.4	105	139.5	95.9	105	137.9	94.8	105
19	167.0	105	165.4	99.1	105	161.1	96.5	105	159.2	95.3	105
23	197.8	105	194.9	98.5	105	189.9	96.0	105	187.1	94.6	105
26	217.1	105	213.7	98.4	105	209.6	96.5	105	205.0	94.4	105
30	246.5	105	241.3	97.9	105	236.6	96.0	105	231.7	94.0	105
33	264.3	105	260.4	98.5	105	254.7	96.4	105	249.2	94.3	105
37	290.4	105	285.9	98.4	105	280.2	96.5	105	274.1	94.4	105
40	304.5	105	298.7	98.1	105	294.9	96.8	105	287.3	94.4	105
44	322.9	105	318.0	98.5	105	313.7	97.1	105	306.4	94.9	105
47	333.8	105	329.8	98.8	105	324.3	97.1	105	313.9	94.0	105
51	349.0	105	343.7	98.5	105	338.9	97.1	105	331.4	95.0	105
54	357.6	105	353.5	98.8	105	348.5	97.5	105	341.0	95.3	105
58	370.2	105	366.4	99.0	105	361.3	97.6	105	350.7	94.7	105
61	379.1	105	370.9	97.8	105	370.2	97.6	105	357.2	94.2	105
65	389.6	105	383.0	98.3	105	380.3	97.6	105	370.2	95.0	105
68	395.2	105	389.9	98.6	105	387.0	97.9	105	377.4	95.5	105
72	404.0	105	395.9	98.0	105	395.2	97.8	105	384.1	95.1	105
75	409.0	105	402.5	98.4	105	400.0	97.8	105	389.2	95.2	105
79	417.9	105	408.3	97.7	105	408.0	97.6	105	396.5	94.9	105
82	422.7	105	412.2	97.5	105	413.1	97.7	105	401.4	95.0	105
86	427.7	105	420.4	98.3	105	418.5	97.8	105	409.1	95.7	105
89	432.4	105	422.6	97.7	105	422.7	97.8	105	410.2	94.9	105
93	439.3	105	432.3	98.4	105	429.8	97.8	105	419.3	95.4	105
121 ^b	470.9	90	469.8	99.8	90	464.1	98.6	90	458.6	97.4	90
149	501.8	90	496.2	98.9	90	493.7	98.4	90	482.5	96.2	90
177	522.1	90	517.8	99.2	90	513.0	98.2	90	503.9	96.5	90
205	540.7	90	538.7	99.6	90	531.8	98.4	90	523.3	96.8	90
233	561.3	89	557.8	99.4	90	550.9	98.1	90	539.7	96.2	90
261	576.1	88	574.0	99.6	90	566.9	98.4	90	556.0	96.5	89
289	591.0	88	589.4	99.7	90	582.1	98.5	90	571.0	96.6	89
317	607.5	87	607.9	100.1	88	596.0	98.1	90	585.8	96.4	89
345	617.9	87	619.4	100.2	87	606.8	98.2	90	597.1	96.6	89
373	628.1	87	633.9	100.9	87	617.7	98.4	90	607.0	96.6	89
401	636.2	87	642.8	101.0	87	628.2	98.7	90	615.0	96.7	87
429	640.8	87	648.2	101.1	86	635.6	99.2	90	625.1	97.5	87
457	639.1	86	653.2	102.2	86	641.1	100.3	89	631.2	98.8	86
485	644.7	86	662.0	102.7	86	651.7	101.1	89	641.5	99.5	85
513	654.1	82	676.6	103.4	84	668.4	102.2	87	650.2	99.4	83
541	651.9	80	688.8	105.7	81	679.5	104.2	83	658.2	101.0	82
569	649.3	73	687.5	105.9	80	676.3	104.2	82	662.2	102.0	80
597	658.1	66	691.2	105.0	77	679.2	103.2	79	667.5	101.4	76
625	646.6	59	689.7	106.7	73	673.6	104.2	75	670.7	103.7	74
639	638.4	57	682.1	106.9	70	676.1	105.9	71	668.5	104.7	71
653	627.9	53	672.8	107.2	69	670.5	106.8	69	667.3	106.3	71
667	638.2	42	671.7	105.2	64	663.2	103.9	67	668.9	104.8	70
681	625.4	39	659.5	105.5	62	659.4	105.4	65	670.3	107.2	68
695	624.8	34	660.1	105.6	58	648.9	103.9	64	666.0	106.6	66
709	620.7	31	653.6	105.3	53	644.3	103.8	59	655.0	105.5	65
723	632.6	25	636.6	100.6	46	636.4	100.6	53	650.2	102.8	60
Mean for Weeks											
1-14	297.9		292.9	98.3		289.7	97.2		282.6	94.9	
15-52	554.4		552.3	99.6		545.0	98.3		535.3	96.6	
53-104	638.6		665.3	104.2		655.9	102.7		651.5	102.0	

^a The number of animals weighed on this day is less than the number of animals surviving.

^b Interim evaluation occurred during week 14.

TABLE 18
Mean Body Weights and Survival of Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

Day	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
1	60.4	105	60.0	99.3	105 ^a	58.7	97.2	105	58.0	96.1	105
5	72.1	105	71.1	98.6	105 ^a	70.4	97.7	105	69.6	96.5	105
9	88.7	105	87.2	98.3	105	85.8	96.7	105	85.0	95.8	105
12	103.3	105	100.7	97.5	105	99.6	96.5	105	98.6	95.5	105
16	123.2	105	120.1	97.5	105	118.8	96.4	105	117.8	95.6	105
19	136.3	105	134.4	98.6	105	133.0	97.6	105	131.2	96.3	105
23	151.5	105	150.4	99.3	105	148.6	98.0	105	147.7	97.5	105
26	158.5	105	160.3	101.1	105	156.7	98.9	105	155.8	98.3	105
30	169.1	105	170.2	100.7	105	166.4	98.4	105	167.2	98.9	105
33	176.4	105	178.3	101.1	105	175.0	99.2	105	174.6	99.0	105
37	188.7	105	190.8	101.1	105	185.1	98.1	105	186.4	98.8	105
40	195.8	105	196.8	100.5	105	193.2	98.7	105	192.3	98.2	105
44	203.7	105	205.4	100.9	105	201.8	99.1	105	202.2	99.3	105
47	209.9	105	210.9	100.5	105	208.5	99.3	105	207.1	98.7	105
51	218.1	105	220.7	101.2	105	214.5	98.3	105	216.4	99.2	105
54	220.1	105	225.2	102.3	105	220.6	100.2	105	220.7	100.3	105
58	228.6	105	230.6	100.9	105	225.8	98.8	105	226.7	99.2	105
61	233.9	105	235.2	100.6	105	230.2	98.4	105	229.9	98.3	105
65	239.0	105	238.7	99.9	105	236.2	98.8	105	235.2	98.4	105
68	241.7	105	242.5	100.3	105	240.5	99.5	105	240.6	99.5	105
72	244.9	105	243.9	99.6	105	242.0	98.8	105	240.7	98.3	105
75	247.6	105	246.6	99.6	105	246.7	99.7	105	246.3	99.5	105
79	251.6	105	251.4	99.9	105	249.7	99.3	105	249.2	99.1	105
82	253.5	105	252.9	99.8	105	252.2	99.5	105	250.9	99.0	105
86	254.4	105	255.8	100.6	105	253.8	99.8	105	253.5	99.6	105
89	256.2	105	255.1	99.5	105	255.5	99.7	105	254.1	99.2	105
93 ^b	257.4	95	261.6	101.6	95	257.1	99.9	95	259.5	100.8	95
121 ^b	275.7	90	279.0	101.2	90	273.1	99.0	90	275.8	100.0	90
149	285.0	90	287.8	101.0	89	282.0	99.0	89	286.0	100.4	90
177	295.0	90	297.0	100.7	89	292.9	99.3	89	296.4	100.5	90
205	302.9	89	307.9	101.6	89	300.8	99.3	89	305.2	100.7	90
233	311.9	89	316.0	101.3	89	309.8	99.3	89	314.8	100.9	90
261	318.1	89	324.2	101.9	89	316.3	99.4	89	322.9	101.5	90
289	331.0	88	331.1	100.0	89	327.3	98.9	89	326.4	98.6	89
317	341.8	88	341.9	100.0	89	334.5	97.9	89	338.2	98.9	86
345	349.2	88	348.4	99.8	88	343.0	98.2	89	344.5	98.7	86
373	356.1	87	355.8	99.9	88	352.4	99.0	89	349.8	98.2	85
401	367.6	87	366.5	99.7	88	361.2	98.3	86	360.2	98.0	85
429	377.0	87	375.8	99.7	88	371.8	98.6	86	368.2	97.7	85
457	387.5	87	383.8	99.0	88	380.7	98.2	83	376.2	97.1	84
485	400.0	85	394.6	98.6	85	392.7	98.2	80	390.8	97.7	84
513	410.8	78	410.5	99.9	82	399.4	97.2	76	401.7	97.8	82
541	421.5	74	420.7	99.8	81	408.6	96.9	76	410.4	97.4	81
570	426.9	69	431.1	101.0	79	418.1	97.9	74	422.9	99.1	77
598	435.9	68	437.8	100.4	75	429.3	98.5	70	435.5	99.9	75
626	446.9	64	446.4	99.9	72	435.4	97.4	67	450.0	100.7	71
640	456.8	61	452.4	99.0	71	440.7	96.5	63	453.7	99.3	71
654	462.4	60	451.7	97.7	69	443.4	95.9	63	458.1	99.1	68
668	450.5	54	460.8	102.3	68	447.0	99.2	60	462.0	102.6	66
682	452.6	54	467.3	103.3	67	447.0	98.8	57	465.3	102.8	63
696	454.3	53	476.9	105.0	64	452.6	99.6	56	464.7	102.3	62
710	451.7	51	461.2	102.1	60	453.0	100.3	54	473.8	104.9	60
724	442.6	48	463.6	104.7	55	455.3	102.9	50	471.2	106.5	58
Mean for Weeks											
1-14	192.0		192.5	100.3		189.9	98.9		189.5	98.7	
15-52	312.3		314.8	100.8		308.9	98.9		312.2	100.0	
53-104	423.6		426.9	100.8		417.0	98.4		424.4	100.2	

^a The number of animals weighed on this day is less than the number of animals surviving.

^b Interim evaluation occurred during week 14

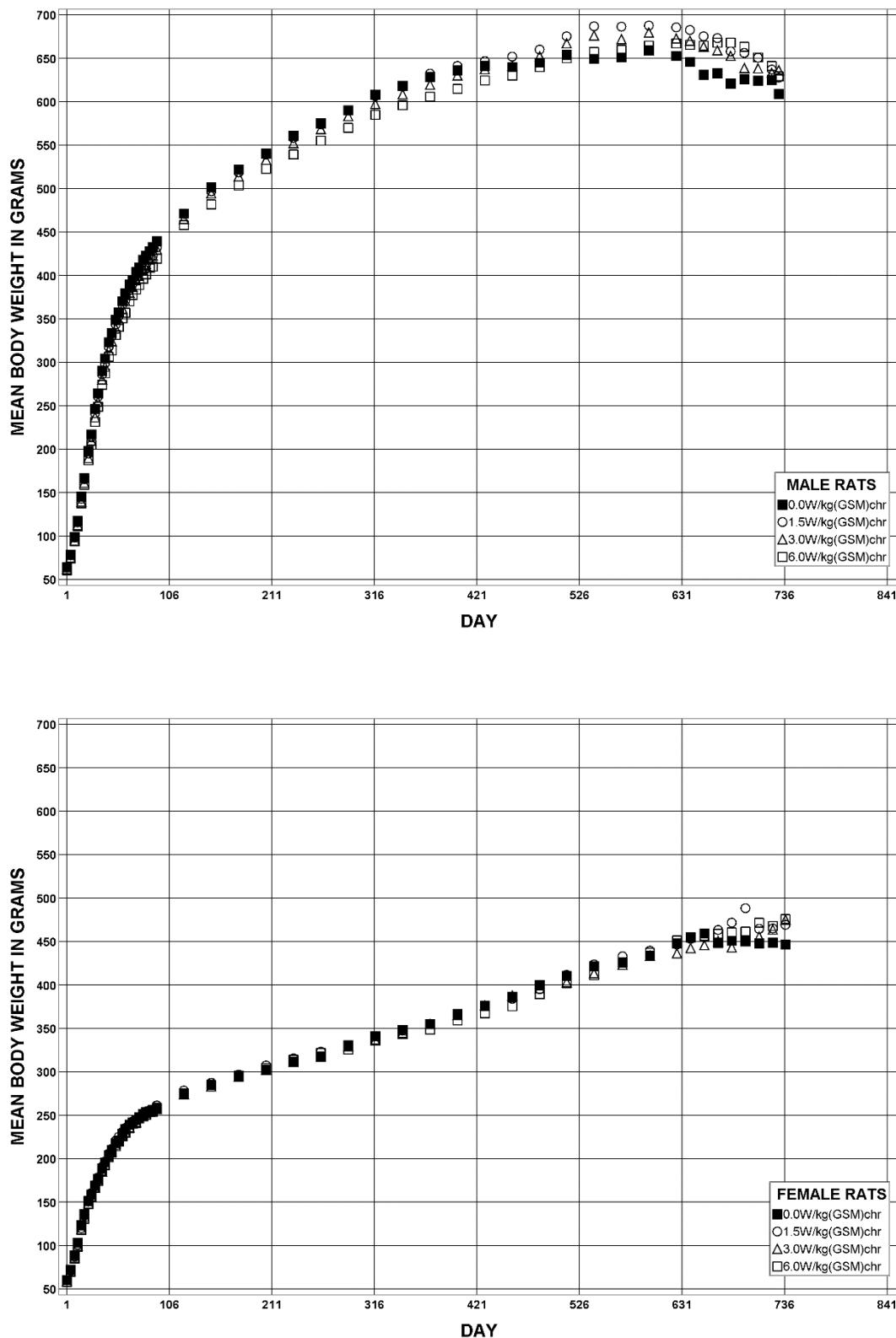


FIGURE 6
Growth Curves for Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

14-Week Interim Evaluation

There were significantly decreased leukocyte and lymphocyte counts (22%) in 3 W/kg females (Table F1). While these decreases may have been stress related, they only occurred in the 3 W/kg group and only in females, thus the biological relevance is uncertain. In 6 W/kg females, there were significant decreases in cholesterol (19%) and triglyceride (38%) concentrations. These decreases may have been due to changes in lipid metabolism.

In males, the absolute left and right kidney weights were significantly decreased in the 1.5 (9% and 10%, respectively) and 6 W/kg (15% and 11%, respectively) groups (Table G4). The absolute liver weight of 6 W/kg males was also significantly decreased (10%). The relative weights of these organs were not significantly decreased, which was likely due to the lower terminal mean body weights of the 1.5 (4%) and 6 W/kg (8%) groups. In females, the relative brain weights of the 1.5 and 6 W/kg groups were significantly increased, which was likely due to lower mean body weights (Table G4). The absolute left kidney weights were significantly decreased in all exposed female groups (9% to 14%). Similarly, the absolute right kidney weights were significantly decreased in the 3 (8%) and 6 W/kg (12%) females. There were also significant decreases in the absolute lung weight in 6 W/kg females (16%) and the absolute thymus weights in 1.5 (18%) and 6 W/kg (22%) females. The absolute liver weights were significantly decreased in 1.5 (17%) and 6 W/kg (20%) females, with corresponding decreased relative weights. None of these changes were associated with histopathologic findings.

There were no GSM exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility in males (Table H1). Due to the poor diagnostic quality of the cytology slides, the estrous cycle in females was not evaluated.

In the heart, there was a significant positive trend ($P=0.013$) in the incidences of cardiomyopathy in the right ventricle in males (Table 19). The average severity was comparable to that of the sham control group. Cardiomyopathy was also seen in the left ventricular free wall and interventricular septum. Cardiomyopathy in the right ventricle was initially diagnosed separately and a higher incidence was found in the right ventricle in the 6 W/kg group compared to the sham controls.

TABLE 19
Incidences of Selected Nonneoplastic Lesions at the 14-Week Interim Evaluation
in Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years^a

	Sham Control	1.5 W/kg	3 W/kg	6 w/kg
Male				
Heart ^b	10	10	10	10
Cardiomyopathy (Excluding Right Ventricle) ^c	2 (1.5) ^d	4 (1.0)	3 (1.0)	4 (1.0)
Ventricle Right, Cardiomyopathy	1 (1.0)	1 (2.0)	5 (1.2)	5 (1.0)
Cardiomyopathy, All Sites	3 (1.3)	5 (1.2)	8* (1.1)	7 (1.0)
Lymph Node, Mandibular	10	10	10	10
Hyperplasia, Lymphocyte	0	0	0	4* (1.0)
Proliferation, Plasma Cell	0	0	0	2 (1.0)
Female				
Heart	10	10	10	10
Cardiomyopathy (Excluding Right Ventricle)	0	2 (1.0)	1 (1.0)	2 (1.0)
Ventricle Right, Cardiomyopathy	0	0	1 (1.0)	0
Cardiomyopathy, All Sites	0	2 (1.0)	2 (1.0)	2 (1.0)
Lymph Node, Mandibular	10	10	10	10
Hyperplasia, Lymphocyte	0	0	0	2 (1.0)
Proliferation, Plasma Cell	0	0	0	3 (1.0)

* Significantly different ($P \leq 0.05$)

^a Lesions were examined for trend by the Cochran-Armitage exact test. Pairwise testing against the sham control group was done by the Fisher exact test.

^b Number of animals with tissue examined microscopically

^c Number of animals with lesion

^d Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

In the left ventricle and interventricular septum, the incidence of cardiomyopathy was similar in all groups. In females, no effect was observed on cardiomyopathy.

Cardiomyopathy was characterized by degeneration and necrosis of myofibers with a mild inflammatory response of macrophages and lymphocytes with occasional neutrophils. In the right ventricle, the cardiomyopathy was most prominent in the subepicardial region in the lower half (toward the apex) of the heart. In more severe cases, the lesions in the right ventricle extended deeper into the myocardium. In some areas, there appeared to be areas of cardiomyocyte loss, characterized by linear, clear areas containing a few scattered cell nuclei and variable amounts of linear, eosinophilic material (likely collagen bundles).

In the mandibular lymph node of males, there was a significant positive trend ($P<0.001$) in the incidences of lymphocyte hyperplasia and the incidence in the 6 W/kg group was significantly increased (Table 19). There was also a significant positive trend ($P=0.038$) in the incidences of plasma cell proliferation. In females, there were significant positive trends in the incidences of lymphocyte hyperplasia ($P=0.038$) and plasma cell proliferation ($P=0.005$), but none of the exposed group incidences were statistically significant when compared to the sham control incidences. In all cases, the severities of these lesions were minimal. There were no similar findings in other lymph nodes in either modulation.

Pathology and Statistical Analyses

This section describes the statistically significant or biologically noteworthy changes in the incidences of neoplasms and nonneoplastic lesions of the heart, prostate gland, brain, pituitary gland (pars distalis), adrenal medulla, pancreatic islets, thyroid gland, adrenal cortex, kidney and other organs, mammary gland, pancreas, and seminal vesicle. Summaries of the incidences of neoplasms and nonneoplastic lesions, statistical analyses of primary neoplasms that occurred with an incidence of at least 5% in at least one animal group, and historical incidences for the neoplasms mentioned in this section are presented in Appendix A for male rats and Appendix B for female rats.

Heart: Malignant schwannomas were observed in all exposed male groups. No schwannomas were observed in the sham controls. The incidences in the exposed groups occurred with a significant positive trend (Tables 20, A1, and A2). In females, schwannomas occurred in two 3 W/kg animals (Tables 20 and B1). Endocardial Schwann cell hyperplasia, a putative preneoplastic Schwann cell lesion, was diagnosed in one 1.5 W/kg male and two 6 W/kg males (Tables 20 and A4); there were none in females. Schwannomas were seen in other organs, including the pituitary gland, trigeminal nerve, salivary glands, Harderian gland, eye, thymus gland, uterus, ovary, and vagina. When the incidences of schwannoma in all organs (including the heart) were combined, they were generally higher in exposed males, but not significantly different from the sham controls.

Schwannomas in the heart may be endocardial or myocardial. They were not recorded separately in the final data because their biological behavior and morphology is similar. Endocardial schwannomas typically arise in the subendocardial region of the left ventricle, though not exclusively. Larger neoplasms may extend into the right ventricle and atria. They are composed of two morphologically distinct cell populations, both of which have indistinct cell boundaries. One population, immediately adjacent to the endocardium, is elongated and has ovoid nuclei, prominent nucleoli, and scant, pale cytoplasm. The other population is more spindloid, with fusiform, hyperchromatic nuclei oriented in parallel (palisading nuclei), which, when abundant, may exhibit a wavy pattern.

TABLE 20
Incidences of Malignant Schwannoma and Neoplasms and Nonneoplastic Lesions of the Heart in Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Heart ^a	90	90	90	90
Cardiomyopathy ^b	79 (1.9) ^c	82 (1.8)	78 (2.1)	79 (1.6)
Ventricle Right, Cardiomyopathy	54 (1.1)	62 (1.5)	72* (1.9)	74** (1.8)
Endocardium, Hyperplasia, Schwann Cell	0	1 (1.0)	0	2 (2.0)
Endocardium, Schwannoma Malignant	0	1	1	2
Myocardium, Schwannoma Malignant	0	1	0	3
Malignant Schwannoma ^d				
Overall rate ^e	0/90 (0%)	2/90 (2%)	1/90 (1%)	5/90 (6%)
Litters rate ^f	0/35 (0%)	2/35 (6%)	1/35 (3%)	5/35 (14%)
Adjusted rate ^g	0.0%	2.7%	1.3%	6.4%
Terminal rate ^h	0/25 (0%)	2/45 (4%)	1/50 (2%)	3/60 (5%)
First incidence (days)	— ^j	730 (T)	730 (T)	582
Rao-Scott adjusted poly-3 test ⁱ	P=0.041	P=0.297	P=0.540	P=0.080
All Organs: Malignant Schwannoma ^k				
Overall rate	3/90 (3%)	3/90 (3%)	5/90 (6%)	7/90 (8%)
Litters rate	3/35 (9%)	3/35 (9%)	5/35 (14%)	7/35 (20%)
Adjusted rate	4.5%	4.0%	6.4%	8.9%
Terminal rate	1/25 (4%)	2/45 (4%)	3/50 (6%)	4/60 (7%)
First incidence (days)	555	720	661	582
Rao-Scott adjusted poly-3 test	P=0.133	P=0.577N	P=0.435	P=0.238
Female				
Heart	90	90	90	90
Cardiomyopathy	40 (1.1)	30* (1.2)	39 (1.1)	27* (1.1)
Ventricle Right, Cardiomyopathy	4 (1.0)	9 (1.1)	14* (1.1)	15* (1.2)
Endocardium, Schwannoma Malignant	0	0	1	0
Myocardium, Schwannoma Malignant	0	0	1	0
Malignant Schwannoma ^l				
Overall rate	0/90 (0%)	0/90 (0%)	2/90 (2%)	0/90 (0%)
Litters rate	0/35 (0%)	0/35 (0%)	2/35 (6%)	0/35 (0%)
Adjusted rate	0.0%	0.0%	2.8%	0.0%
Terminal rate	0/48 (0%)	0/53 (0%)	1/48 (2%)	0/57 (0%)
First incidence (days)	—	—	578	—
Rao-Scott adjusted poly-3 test	P=0.640	— ^m	P=0.365	—
All Organs: Malignant Schwannoma ⁿ				
Overall rate	4/90 (4%)	1/90 (1%)	5/90 (6%)	2/90 (2%)
Litters rate	3/35 (9%)	1/35 (3%)	5/35 (14%)	2/35 (6%)
Adjusted rate	5.7%	1.3%	7.0%	2.7%
Terminal rate	2/48 (4%)	0/53 (0%)	2/48 (4%)	1/57 (2%)
First incidence (days)	489	480	578	622
Rao-Scott adjusted poly-3 test	P=0.428N	P=0.212N	P=0.519	P=0.354N

TABLE 20
Incidences of Malignant Schwannoma and Neoplasms and Nonneoplastic Lesions of the Heart in Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

- * Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test
- ** $P \leq 0.01$
- (T) Terminal euthanasia
- a Number of animals with tissue examined microscopically
- b Number of animals with lesion
- c Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked
- d Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 2/240 (1.0% \pm 1.2%), range 0%-2%
- e Number of animals with neoplasm per number of animals necropsied
- f Number of litters with animals with neoplasm per number of litters necropsied
- g Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
- h Observed incidence at terminal euthanasia
- i Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
- j Not applicable; no neoplasms in animal group
- k Historical incidence for all routes of 2-year studies: 6/240 (2.3% \pm 1.8%), range 0%-4%
- l Historical incidence for all routes of 2-year studies: 0/239
- m Value of statistic cannot be computed
- n Historical incidence for all routes of 2-year studies: 8/240 (3.1% \pm 2.1%), range 0%-4%

The cells may have mild atypia and scattered mitotic figures. Schwannomas are invasive and invade into adjacent myocardial tissue. In larger neoplasms, the spindle-shaped cells are more abundant and may display Antoni type A and B patterns, giant nuclei, Verocay bodies, and multinucleated cells. Myocardial schwannomas have a similar appearance, but are often less cellular than endocardial schwannomas. They are composed of the spindle-shaped cells, in a loose arrangement. Endocardial Schwann cell hyperplasia has a similar appearance, but is less extensive, does not exhibit cellular atypia, has no or few mitotic figures, and does not invade into the adjacent myocardium.

Cardiomyopathy of the right ventricular free wall was seen in all male and female groups (including sham controls) (Tables 20, A4, and B4). In males and females, the incidences were higher in all exposed groups compared to sham controls; the increases were statistically significant in the 3 and 6 W/kg groups. The increasing trends were also significant. There was also a slight elevation in the severity in 3 and 6 W/kg males, but there was no such elevation in females. Cardiomyopathy in the right ventricle was initially diagnosed separately, and a higher incidence was found in the right ventricle in the 6 W/kg group(s) compared to the sham controls. The incidence of right ventricle cardiomyopathy was then quantified during pathology peer review. Cardiomyopathy is a very common spontaneous disease in rats and was also seen in other sites in the heart (most notably the left ventricular free wall, but also the interventricular septum) where it is more frequently seen. In the current study, it was more prominent in the apex

and lower half (toward the apex) of the heart. When cardiomyopathy was evaluated in the entire heart (including the right ventricle), there was no significant difference in the incidences or average severities in exposed male groups when compared to sham controls. In females, there were significantly decreased incidences of cardiomyopathy of the whole heart in the 1.5 and 6 W/kg groups. Cardiomyopathy was characterized by degeneration and necrosis of myofibers with a mild inflammatory response of macrophages and lymphocytes with occasional neutrophils. In later stages of the disease, fibrosis may be prominent. In the right ventricle, the cardiomyopathy was most prominent in the subepicardial region in the lower half (toward the apex) of the heart. In more severe cases, the lesions extended deeper into the right ventricular wall. The lesions were similar to those described above. In some areas, there appeared to be areas of cardiomyocyte loss, characterized by linear, clear areas containing a few scattered cell nuclei and variable amounts of linear, eosinophilic material (likely collagen bundles). Fibrosis was sometimes fairly prominent. The effect of GSM-modulated cell phone RFR on the incidence of cardiomyopathy appears to be specific to the right ventricular free wall.

Prostate Gland: There were increased incidences of adenoma and adenoma or carcinoma (combined) in the 3 W/kg groups compared to sham controls, but the increase was not statistically significant (Tables 21, A1, and A2). A single carcinoma occurred in the 3 W/kg group. Prostate gland carcinomas are rare neoplasms in rats, with a mean historical control incidence of 0/240 in Hsd:Sprague Dawley SD rats (Table A3b), 0.57% in Wistar Han rats (range 0%–2%), and 0.43% in F344/N rats (range 0%–4%); the combined incidence in the 3 W/kg group exceeded the historical control ranges for all rat strains that have been used by the National Toxicology Program (NTP, 2018a). The incidences and severities of epithelial hyperplasia were slightly increased in all exposed groups (Tables 21 and A4). Prostate gland adenomas were expansile lesions that filled the lumen of at least one acinus and compressed the adjacent tissue. The cuboidal to columnar cells formed papillary or cribriform patterns, and there was little cellular pleomorphism.

TABLE 21
**Incidences of Neoplasms and Nonneoplastic Lesions of the Prostate Gland in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	90	90
Epithelium, Hyperplasia ^a	5 (1.2) ^b	13 (1.6)	11 (1.9)	11 (2.4)
Adenoma ^c				
Overall rate ^d	2/90 (2%)	2/90 (2%)	6/90 (7%)	3/90 (3%)
Litters rate ^e	2/35 (6%)	2/35 (6%)	5/35 (14%)	3/35 (9%)
Adjusted rate ^f	3.0%	2.7%	7.7%	3.9%
Terminal rate ^g	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	730 (T)	730 (T)
Rao-Scott adjusted poly-3 test ^h	P=0.419	P=0.625N	P=0.224	P=0.566
Carcinoma ⁱ	0	0	1	0
Adenoma or Carcinoma ^a				
Overall rate	2/90 (2%)	2/90 (2%)	7/90 (8%)	3/90 (3%)
Litters rate	2/35 (6%)	2/35 (6%)	6/35 (17%)	3/35 (9%)
Adjusted rate	3.0%	2.7%	9.0%	3.9%
Terminal rate	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	717	730 (T)
Rao-Scott adjusted poly-3 test	P=0.412	P=0.626N	P=0.161	P=0.566

(T) Terminal euthanasia

^a Number of animals with lesion

^b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

^c Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 2/240 (0.6% \pm 1.1%), range 0%-2%

^d Number of animals with neoplasm per number of animals with prostate gland examined microscopically

^e Number of litters with animals with neoplasm per number of litters with prostate gland examined microscopically

^f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

^g Observed incidence at terminal euthanasia

^h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott statistic performs the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A lower incidence in an exposure group is indicated by N.

ⁱ Historical incidence for all routes of 2-year studies: 0/240

Brain: In males, malignant glioma and glial cell hyperplasia occurred in all exposed groups (Tables 22, A1, and A4) and neither lesion occurred in the sham control group; however, the incidences were not significant compared to those in the sham controls. In females, malignant glioma occurred in one 6 W/kg animal and glial cell hyperplasia occurred one 3 W/kg animal (Tables 22, B1, and B4).

Malignant gliomas are relatively large neoplasms with indistinct borders. The cells are usually densely packed and may be seen clustered around blood vessels (perivascular cuffing) or aggregated around neurons (satellitosis). The

TABLE 22
**Incidences of Neoplasms and Nonneoplastic Lesions of the Brain in Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia ^a	0	2 (2.0) ^b	3 (3.0)	1 (4.0)
Meninges, Hyperplasia, Granular Cell	1 (1.0)	0	1 (1.0)	0
Glioma Malignant ^c				
Overall rate ^d	0/90 (0%)	3/90 (3%)	3/90 (3%)	2/90 (2%)
Litters rate ^e	0/35 (0%)	3/35 (9%)	3/35 (9%)	2/35 (6%)
Adjusted rate ^f	0.0%	4.0%	3.8%	2.6%
Terminal rate ^g	0/25 (0%)	2/45 (4%)	1/50 (2%)	1/60 (2%)
First incidence (days)	— ⁱ	715	649	716
Rao-Scott adjusted poly-3 test ^h	P=0.382	P=0.155	P=0.166	P=0.277
Meninges, Granular Cell Tumor Benign ^j				
Overall rate	1/90 (1%)	3/90 (3%)	3/90 (3%)	3/90 (3%)
Litters rate	1/35 (3%)	3/35 (9%)	3/35 (9%)	3/35 (9%)
Adjusted rate	1.5%	4.0%	3.9%	3.9%
Terminal rate	1/25 (4%)	1/45 (2%)	3/50 (6%)	3/60 (5%)
First incidence (days)	730 (T)	655	730 (T)	730 (T)
Rao-Scott adjusted poly-3 test	P=0.350	P=0.328	P=0.342	P=0.341
Meninges, Granular Cell Tumor Malignant	0	0	1	0
Meninges, Granular Cell Tumor Benign or Malignant ^j				
Overall rate	1/90 (1%)	3/90 (3%)	4/90 (4%)	3/90 (3%)
Litters rate	1/35 (3%)	3/35 (9%)	4/35 (11%)	3/35 (9%)
Adjusted rate	1.5%	4.0%	5.1%	3.9%
Terminal rate	1/25 (4%)	1/45 (2%)	3/50 (6%)	3/60 (5%)
First incidence (days)	730 (T)	655	699	730 (T)
Rao-Scott adjusted poly-3 test	P=0.343	P=0.327	P=0.220	P=0.340
Female				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia	0	0	1 (4.0)	0
Meninges, Hyperplasia, Granular Cell	1 (3.0)	0	1 (3.0)	0
Glioma Malignant ^k				
Overall rate	0/90 (0%)	0/90 (0%)	0/90 (0%)	1/90 (1%)
Litters rate	0/35 (0%)	0/35 (0%)	0/35 (0%)	1/35 (3%)
Adjusted rate	0.0%	0.0%	0.0%	1.3%
Terminal rate	0/48 (0%)	0/53 (0%)	0/48 (0%)	0/57 (0%)
First incidence (days)	— ⁱ	—	—	669
Rao-Scott adjusted poly-3 test	— ⁱ	—	—	—
Meninges, Granular Cell Tumor Benign ^m				
Overall rate	1/90 (1%)	1/90 (1%)	2/90 (2%)	0/90 (0%)
Litters rate	1/35 (3%)	1/35 (3%)	2/35 (6%)	0/35 (0%)
Adjusted rate	1.4%	1.3%	2.8%	0.0%
Terminal rate	1/48 (2%)	1/53 (2%)	1/48 (2%)	0/57 (0%)
First incidence (days)	730 (T)	737 (T)	669	—
Rao-Scott adjusted poly-3 test	P=0.375N	P=0.722N	P=0.503	P=0.489N
Meninges, Granular Cell Tumor Malignant	0	0	0	1

TABLE 22
**Incidences of Neoplasms and Nonneoplastic Lesions of the Brain in Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Female (continued)				
Meninges, Granular Cell Tumor Benign or Malignant ^m				
Overall rate	1/90 (1%)	1/90 (1%)	2/90 (2%)	1/90 (1%)
Litters rate	1/35 (3%)	1/35 (3%)	2/35 (6%)	1/35 (3%)
Adjusted rate	1.4%	1.3%	2.8%	1.3%
Terminal rate	1/48 (2%)	1/53 (2%)	1/48 (2%)	1/57 (2%)
First incidence (days)	737 (T)	737 (T)	669	737 (T)
Rao-Scott adjusted poly-3 test	P=0.594	P=0.712N	P=0.485	P=0.713N

(T) Terminal euthanasia

a Number of animals with lesion

b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

c Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 2/190 (1.3% \pm 2.3%), range 0%-4%

d Number of animals with neoplasm per number of animals with brain examined microscopically

e Number of litters with animals with neoplasm per number of litters with brain examined microscopically

f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

g Observed incidence at terminal euthanasia

h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott statistic performs the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.

i Not applicable; no neoplasms in animal group

j Historical incidence for all routes of 2-year studies: 3/190 (1.7% \pm 2.1%), range 0%-4%

k Historical incidence for all routes of 2-year studies: 1/190 (0.7% \pm 1.2%), range 0%-2%

l Value of statistic cannot be computed

m Historical incidence for all routes of 2-year studies: 2/190 (1.0% \pm 1.0%), range 0%-2%

cells may also invade the meninges. The cells have small to moderate amounts of eosinophilic cytoplasm and indistinct margins. They have small, round to elongated, hyperchromatic nuclei. A few mitotic figures may be present. Glial cell hyperplasia has a similar appearance, but is smaller with less densely packed cells (all cells are separated by neuropil). There may be satellitosis, but perivascular cuffing is minimal and there is no meningeal invasion and generally no mitotic figures. In glial cell hyperplasia, there are no reactive, degenerative, or necrotic elements within the associated parenchyma or other evidence of damage (e.g., hemorrhage, edema). The hyperplastic cells may be hypertrophied, but there are no gitter cells or gemistocytes.

In males, there were three benign granular cell tumors of the meninges in each exposed group and one in the sham control group (Tables 22 and A1). There was also a single malignant granular cell tumor in the 3 W/kg group. Granular cell hyperplasia, which is thought to be on a continuum with benign and malignant granular cell tumors, occurred in one sham control male and one 3 W/kg male (Tables 22 and A4). In females, the incidences of granular

cell tumors were 1%, 1%, 2%, 0% in the sham control, 1.5, 3, and 6 W/kg groups, respectively (Tables 22 and B1). There was also a single malignant granular cell tumor in a 6 W/kg female. Granular cell hyperplasia occurred in one female each from the sham control and 3 W/kg groups.

Granular cell tumors were observed in the meninges or choroid plexus and were composed of sheets of large, densely packed polygonal cells. The cells had abundant, eosinophilic, granular cytoplasm with indistinct cell borders and small, uniform, round to oval nuclei. A few smaller cells with dark basophilic nuclei and sparser, less granular cytoplasm were also present. No mitotic figures were observed. Benign granular cell tumors were discrete, non-invasive masses that caused variable compression of the adjacent brain parenchyma. Granular cell hyperplasia was similar in appearance, but were smaller, non-invasive, and non-compressive. Malignant granular cell tumors were invasive, extending into the underlying neuropil, and had some nuclear pleomorphism.

Pituitary Gland (Pars Distalis): There were increased incidences of adenoma in all exposed male groups compared to the sham controls, but none were statistically significant (Tables 23, A1, and A2). In females, the incidences of adenoma in the 1.5 and 6 W/kg groups were significantly decreased (Tables 23, B1, and B2). The incidences and severities of hyperplasia in exposed groups of males and females were similar to those of the sham controls (Tables 23, A4, and B4). Adenomas were characterized by a well-delineated mass composed of solid sheets of cells that compressed the adjacent tissue. Cells were often hypertrophied and cellular atypia and pleomorphism were not uncommon. Vascular patterns were often altered. The incidences of cysts in males also increased with increasing exposure SAR; the incidences were statistically significant by the Rao-Scott adjusted poly-3 test in the 3 and 6 W/kg exposure groups (Tables 23 and B4). Cysts are generally considered developmental abnormalities and the toxicologic significance of these is uncertain.

Adrenal Medulla: In males, there were significantly increased incidences of benign pheochromocytoma and benign, malignant, or complex pheochromocytoma (combined) in the 1.5 and 3 W/kg groups (Tables 24, A1, and A2). The incidence of malignant pheochromocytoma was slightly increased in 3 W/kg males, but none were recorded in 6 W/kg males. The upper range of benign pheochromocytoma in the available historical control data for male Hsd:Sprague Dawley SD rats is 24% (mean historical incidence is 16%) (Table A3e). There were decreased

incidences of hyperplasia in exposed male groups, and the severity was similar to that in the sham controls (Tables 24 and A4). In females, there were slightly increased incidences of benign pheochromocytoma in exposed groups (Tables 24 and B1). There was a significant positive trend in the incidences of hyperplasia and the incidence in 6 W/kg females was significant; however, as in males, the average

TABLE 23
Incidences of Neoplasms and Nonneoplastic Lesions of the Pituitary Gland (Pars Distalis) in Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	89	90	90	90
Hyperplasia ^a	32 (2.4) ^b	34 (2.4)	35 (2.4)	32 (2.2)
Cyst	5	9	15*	16*
Adenoma, Multiple	0	0	1	0
Adenoma (includes multiple) ^c				
Overall rate ^d	17/89 (19%)	28/90 (31%)	26/90 (29%)	26/90 (29%)
Litters rate ^e	13/35 (37%)	23/35 (66%)	19/35 (54%)	22/35 (63%)
Adjusted rate ^f	24.9%	35.2%	32.2%	32.4%
Terminal rate ^g	5/25 (20%)	15/45 (33%)	17/50 (34%)	19/60 (32%)
First incidence (days)	527	309	537	384
Rao-Scott adjusted poly-3 test ^h	P=0.301	P=0.126	P=0.216	P=0.210
Female				
Number Examined Microscopically	90	90	90	90
Hyperplasia	20 (2.5)	26 (2.0)	22 (1.9)	22 (2.0)
Adenoma, Multiple	1	3	3	0
Adenoma (includes multiple) ⁱ				
Overall rate	43/90 (48%)	33/90 (37%)	38/90 (42%)	32/90 (36%)
Litters rate	28/35 (80%)	24/35 (69%)	26/35 (74%)	23/35 (66%)
Adjusted rate	57.1%	42.5%	51.2%	41.6%
Terminal rate	28/48 (58%)	23/53 (43%)	24/48 (50%)	24/57 (42%)
First incidence (days)	464	578	545	565
Rao-Scott adjusted poly-3 test	P=0.077N	P=0.049N	P=0.283N	P=0.038N

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

a Number of animals with lesion

b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

c Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 47/239 (19.8% \pm 7.5%), range 10%-28%

d Number of animals with neoplasm per number of animals with pituitary gland examined microscopically

e Number of litters with animals with neoplasm per number of litters with pituitary gland examined microscopically

f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

g Observed incidence at terminal euthanasia

h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.

i Historical incidence for all routes of 2-year studies: 98/240 (39.4% \pm 5.6%), range 36%-48%

TABLE 24
Incidences of Neoplasms and Nonneoplastic Lesions of the Adrenal Medulla in Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	88	90	89	87
Hyperplasia ^a	42 (2.0) ^b	24** (2.1)	26** (1.9)	35 (2.0)
Benign Pheochromocytoma, Bilateral	1	2	4	0
Benign Pheochromocytoma, Multiple	1	0	2	0
Benign Pheochromocytoma (includes bilateral and multiple) ^c				
Overall rate ^d	10/88 (11%)	23/90 (26%)	25/89 (28%)	14/87 (16%)
Litters rate ^e	8/35 (23%)	19/35 (54%)	21/35 (60%)	12/35 (34%)
Adjusted rate ^f	15.2%	29.9%	31.7%	18.3%
Terminal rate ^g	3/23 (13%)	13/45 (29%)	14/49 (29%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted poly-3 test ^h	P=0.472N	P=0.030	P=0.017	P=0.384
Malignant Pheochromocytoma, Bilateral	0	1	0	0
Malignant Pheochromocytoma	1	1	4	0
Complex Pheochromocytoma	1	0	0	0
Benign, Malignant, or Complex Pheochromocytoma ⁱ				
Overall rate	11/88 (13%)	24/90 (27%)	28/89 (31%)	14/87 (16%)
Litters rate	9/35 (26%)	19/35 (54%)	23/35 (66%)	12/35 (34%)
Adjusted rate	16.7%	31.1%	35.3%	18.3%
Terminal rate	3/23 (13%)	13/45 (29%)	15/49 (31%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted poly-3 test	P=0.409N	P=0.035	P=0.010	P=0.472
Female				
Number Examined Microscopically	86	90	90	86
Hyperplasia	13 (1.5)	19 (1.2)	14 (1.4)	25* (1.8)
Benign Pheochromocytoma, Bilateral	0	0	0	1
Benign Pheochromocytoma (includes bilateral) ^j				
Overall rate	1/86 (1%)	3/90 (3%)	3/90 (3%)	2/86 (2%)
Litters rate	1/35 (3%)	3/35 (9%)	3/35 (9%)	2/35 (6%)
Adjusted rate	1.5%	4.0%	4.2%	2.8%
Terminal rate	1/45 (2%)	3/53 (6%)	3/48 (6%)	2/53 (4%)
First incidence (days)	737 (T)	737 (T)	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.486	P=0.347	P=0.319	P=0.500

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

** $P \leq 0.01$

(T) Terminal euthanasia

^a Number of animals with lesion

^b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

^c Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 36/238 (15.8% \pm 6.5%), range 10%-24%

^d Number of animals with neoplasm per number of animals with adrenal medulla examined microscopically

^e Number of litters with animals with neoplasm per number of litters with adrenal medulla examined microscopically

^f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

^g Observed incidence at terminal euthanasia

^h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend is indicated by N.

ⁱ Historical incidence for all routes of 2-year studies: 45/238 (20.1% \pm 7.1%), range 13%-28%

^j Historical incidence for all routes of 2-year studies: 4/235 (1.8% \pm 2.9%), range 0%-6%

severities in the exposed groups were similar to that in the sham controls (Tables 24 and B4). Benign pheochromocytomas were characterized by a well-delineated mass that compressed the adjacent tissue and was composed of cells arranged in large solid clusters or thick trabeculae. Cells exhibited mild to marked alteration in size, shape, and/or staining qualities. Cellular atypia and pleomorphism were common. A diagnosis of malignant pheochromocytoma was made when there was evidence of invasion of the capsule or metastasis.

Pancreatic Islets: In males, there were increased incidences of adenoma or carcinoma (combined) in all exposed groups, but only the incidence in the 1.5 W/kg group was significant (Tables 25, A1, and A2). There were also increased incidences of adenoma in all exposed groups and of carcinoma in the 1.5 and 3 W/kg groups, but they were not statistically significant. In females, the incidences of adenoma and carcinoma were similar to those in the sham controls (Tables B1 and B2). There were decreased incidences of hyperplasia in all exposed male and female groups, and the decreases were statistically significant in 1.5 and 3 W/kg males and 1.5 W/kg females (Tables 25, A4, and B4).

The adenomas were well circumscribed, occasionally encapsulated nodules that typically compressed the surrounding pancreatic tissue. The polygonal cells were arranged in cords or small nests and there was minimal to mild cellular pleomorphism in some of the larger masses.

TABLE 25
**Incidences of Neoplasms and Nonneoplastic Lesions of the Pancreatic Islets in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Number Examined Microscopically	90	89	86	85
Hyperplasia ^a	12 (1.5) ^b	5* (2.8)	5* (2.0)	7 (1.7)
Adenoma, Multiple	0	2	1	1
Adenoma (includes multiple) ^c				
Overall rate ^d	5/90 (6%)	14/89 (16%)	10/86 (12%)	11/85 (13%)
Litters rate ^e	5/35 (14%)	12/35 (34%)	9/34 (26%)	11/35 (31%)
Adjusted rate ^f	7.6%	18.5%	13.2%	14.8%
Terminal rate ^g	2/25 (8%)	10/45 (22%)	9/50 (18%)	11/60 (18%)
First incidence (days)	624	531	677	730 (T)
Rao-Scott adjusted poly-3 test ^h	P=0.282	P=0.051	P=0.204	P=0.140
Carcinoma, Multiple	0	2	0	0
Carcinoma (includes multiple) ⁱ				
Overall rate	8/90 (9%)	15/89 (17%)	10/86 (12%)	5/85 (6%)
Litters rate	8/35 (23%)	12/35 (34%)	10/34 (29%)	4/35 (11%)
Adjusted rate	12.0%	19.7%	13.1%	6.7%
Terminal rate	3/25 (12%)	7/45 (16%)	8/50 (16%)	4/60 (7%)
First incidence (days)	663	531	537	544
Rao-Scott adjusted poly -3 test	P=0.088N	P=0.173	P=0.517	P=0.220N
Adenoma or Carcinoma ^j				
Overall rate	13/90 (14%)	27/89 (30%)	19/86 (22%)	16/85 (19%)
Litters rate	12/35 (34%)	19/35 (54%)	17/34 (50%)	14/35 (40%)
Adjusted rate	19.4%	35.2%	24.8%	21.3%
Terminal rate	5/25 (20%)	16/45 (36%)	16/50 (32%)	15/60 (25%)
First incidence (days)	624	531	537	544
Rao-Scott adjusted poly test	P=0.344N	P=0.032	P=0.282	P=0.462

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

(T) Terminal euthanasia

^a Number of animals with lesion

^b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

^c Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 18/240 (7.9% \pm 5.5%), range 4%-16%

^d Number of animals with neoplasm per number of animals with pancreatic islets examined microscopically

^e Number of litters with animals with neoplasm per number of litters with pancreatic islets examined microscopically

^f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

^g Observed incidence at terminal euthanasia

^h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.

ⁱ Historical incidence for all routes of 2-year studies: 8/240 (2.2% \pm 4.4%), range 0%-9%

^j Historical incidence for all routes of 2-year studies: 26/240 (10.1% \pm 6.0%), range 4%-16%

[Table 26 was omitted]

Thyroid Gland: In females, the incidences of C-cell hyperplasia were significantly increased in all exposed groups compared to the sham controls (Tables 27 and B4). In males, there was a slightly increased incidence of C-cell hyperplasia in the 1.5 W/kg group, but the incidence was not significant (Tables 27 and A4; NTP, 2018a).

Adrenal Cortex: The incidences of hypertrophy increased with a significant positive trend, and the incidence was significantly increased in 6.0 W/kg males compared to the sham controls (Tables 28 and A4). However, the average severity of the lesion did not increase with increasing exposure concentration. There was a similar finding in the CDMA-exposed males, but there was no such effect in females exposed to either modulation. In females, there were significantly increased incidences of adrenal cortical hyperplasia in the 3 and 6 W/kg groups, but the average severity was similar between exposed and sham control groups (Tables 29 and B4). There was a similar response in the CDMA-exposed females, but not in the males exposed to either modulation. In 6 W/kg females, there was a significantly decreased incidence of cytoplasmic vacuolization and the average severity was also slightly decreased for this lesion. The incidences of this lesion in exposed males were similar to those in the sham controls.

TABLE 27
Incidences of C-Cell Hyperplasia of the Thyroid Gland in Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically C-Cell, Hyperplasia ^a	89 16 (1.8) ^b	89 24 (1.9)	89 18 (1.9)	87 14 (1.6)
Female				
Number Examined Microscopically C-Cell, Hyperplasia	90 28 (2.3)	88 49** (1.6)	90 45** (1.8)	88 43* (1.7)

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

** $P \leq 0.01$

a Number of animals with lesion

b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

TABLE 28
Incidences of Nonneoplastic Lesions of the Adrenal Cortex in Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically Hyperplasia ^a	90 47 (1.7) ^b	90 46 (1.8)	90 46 (1.8)	88 45 (1.9)
Hypertrophy	35 (1.5)	43 (1.6)	50 (1.4)	54* (1.3)
Vacuolation, Cytoplasmic	20 (1.5)	32 (1.4)	25 (1.6)	22 (1.3)
Female				
Number Examined Microscopically Hyperplasia	90 14 (1.9)	90 26 (1.8)	89 40** (1.9)	90 26* (1.6)
Hypertrophy	52 (1.5)	54 (1.8)	51 (1.8)	56 (1.5)
Vacuolation, Cytoplasmic	18 (1.5)	21 (1.4)	11 (1.6)	8* (1.1)

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott test

** $P \leq 0.01$

a Number of animals with lesion

b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

Kidney and Other Organs: The severity of chronic progressive nephropathy was lower in all exposed groups compared to the sham controls (Table 29). There were decreased incidences in a number of lesions in other organs in exposed groups, some statistically significant, that were thought to be secondary to the chronic progressive nephropathy, either directly or indirectly (Tables 29 and A4). These lesions included hyperplasia of the parathyroid gland; mineral in the blood vessels in the cecum, colon, liver, mesentery, pancreas, salivary glands, brain, heart, kidney, skeletal muscle, glandular stomach, and aorta; fibrous osteodystrophy of bone; polyarteritis nodosa (chronic active inflammation of the blood vessels) of the epididymis, testis, cecum, liver, pancreas, salivary glands, and thymus; germ cell degeneration of the testis; edema, erosion, epithelial regeneration, acute inflammation, chronic active inflammation, and ulcer of the cecum; epithelial regeneration of the colon; red pulp atrophy and white pulp atrophy of the spleen; and exfoliated germ cell and hypospermia of the epididymis.

Other Lesions: In females, there was a significant negative trend ($P=0.038$) in the incidences of mammary gland adenoma, and the incidences in the 3 and 6 W/kg groups were decreased but not significantly (Tables B1 and B2). The incidence in the sham controls (9%) was at the high end of the historical control range for this neoplasm in Hsd:Sprague Dawley SD rats, and the incidences in the 3 and 6 W/kg groups (2%) were within the historical control range (15/240 [5.7% \pm 4.0%], range 0%-9%). The biological significance of these findings is unclear.

TABLE 29
Incidences of Nonneoplastic Lesions Associated with the Decreased Severity of Chronic Progressive Nephropathy of the Kidney in Male Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Kidney ^a	90	90	90	90
Nephropathy, Chronic Progressive ^b	88 (3.7) ^c	89 (3.2)	90 (2.9)	89 (2.6)
Aorta	90	90	90	90
Mineral	30 (2.1)	7** (2.3)	12** (1.6)	6** (1.5)
Bone	90	90	90	90
Fibrous Osteodystrophy	46 (1.4)	18** (1.1)	14** (1.0)	6** (1.5)
Brain	90	90	90	90
Mineral	5 (1.0)	4 (1.0)	6 (1.2)	2 (1.0)
Epididymis	90	90	90	90
Artery, Inflammation, Chronic Active	2 (2.5)	2 (2.0)	1 (4.0)	2 (2.5)
Exfoliated Germ Cell	51 (1.9)	26** (1.5)	29** (1.4)	15** (1.5)
Hypospermia	28 (3.4)	20 (3.2)	23 (3.0)	8** (3.3)
Heart	90	90	90	90
Artery, Mineral	20 (2.5)	7** (1.9)	3** (2.0)	2** (1.5)
Intestine Large, Cecum	75	75	79	80
Artery, Inflammation, Chronic Active	20 (2.1)	9* (2.0)	5** (1.8)	6** (1.8)
Artery, Mineral	1 (2.0)	0	0	0
Edema	11 (2.0)	1** (2.0)	0**	4 (1.8)
Epithelium, Regeneration	14 (2.4)	0**	0**	2** (2.5)
Erosion	10 (2.5)	0**	0**	3 (2.0)
Inflammation, Acute	10 (2.8)	1* (2.0)	0**	2* (1.5)
Inflammation, Chronic Active	1 (3.0)	0	0	0
Ulcer	6 (2.3)	0	0*	0*
Intestine Large, Colon	81	83	81	82
Artery, Mineral	2 (2.0)	0	0	0
Epithelium, Regeneration	5 (2.6)	0	0	2 (1.0)
Intestine Large, Rectum	83	81	85	87
Epithelium, Regeneration	3 (2.3)	0	0	0
Kidney	90	90	90	90
Artery, Mineral	2 (2.0)	1 (1.0)	0	0
Liver	90	90	90	90
Artery, Inflammation, Chronic Active	2 (3.5)	5 (2.4)	1 (1.0)	0
Artery, Mineral	1 (1.0)	0	0	0
Mesentery	39	19	17	7
Artery, Mineral	21 (2.1)	4* (2.3)	5 (2.6)	2 (1.5)
Pancreas	90	89	88	86
Artery, Inflammation, Chronic Active	48 (2.3)	28** (2.1)	26** (2.4)	14** (2.0)
Artery, Mineral	11 (1.8)	3* (1.7)	3* (2.0)	1** (3.0)
Parathyroid Gland	83	87	87	81
Hyperplasia	51 (2.5)	35** (2.0)	46 (2.0)	28** (1.6)
Salivary Glands	90	90	90	90
Artery, Inflammation, Chronic Active	11 (2.5)	7 (2.6)	3* (2.7)	1** (3.0)
Artery, Mineral	2 (2.5)	0	0	0

TABLE 29

Incidences of Nonneoplastic Lesions Associated with the Decreased Severity of Chronic Progressive Nephropathy of the Kidney in Male Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Skeletal Muscle Mineral	90 2 (1.0)	90 0	90 0	90 0
Spleen	90	90	89	90
Red Pulp, Atrophy	26 (2.2)	10** (2.0)	10** (2.4)	3** (2.3)
White Pulp, Atrophy	30 (2.1)	16** (1.8)	13** (1.8)	11** (2.1)
Stomach, Glandular Mineral	86 31 (2.5)	88 7** (2.9)	87 8** (2.4)	86 4** (2.5)
Testis	90	90	90	90
Artery, Inflammation, Chronic Active	52 (2.9)	40* (2.9)	37** (2.9)	20** (2.7)
Germ Cell, Degeneration	51 (2.3)	35* (2.2)	42 (2.1)	20** (2.0)
Thymus	88	86	88	86
Artery, Inflammation, Chronic Active	6 (2.7)	3 (3.0)	2 (2.5)	1 (2.0)

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

** $P \leq 0.01$

a Number of animals with tissue examined microscopically

b Number of animals with lesion

c Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

In males, there was a significantly decreased trend ($P < 0.001$) for acinar hyperplasia of the pancreas, and the incidences in the 3 and 6 W/kg groups were significantly decreased compared to sham controls (Table A4). There was a significant decreased trend ($P < 0.001$) of secretory fluid in the seminal vesicle and significantly decreased incidences in all exposed groups (Table A4). The incidences of both lesions decreased in an exposure-related fashion; however, the biological significance of these decreases is unclear.

CDMA

28-DAY STUDY

Perinatal Exposure

No exposure-related effects were observed on survival or littering rates (littering/pregnant ratio) (Table 30). A single incidence of whole litter resorption was observed in the 9 W/kg group, and it was unclear if this was related to exposure due to the low incidence. Gestation body weights were unaffected by exposure to CDMA (Table 31). An overall (GD 6-21) lower body weight gain of 11% compared to sham controls was observed.

TABLE 30
Summary of Disposition During Perinatal Exposure and F₁ Allocation
in the 28-Day Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Time-mated Females	20	20	20	20
Pregnant Females	20	17	17	17
Non-Pregnant Females	0	3	3	3
Pregnant Dams not Delivering	0	0	0	1
Died	0	0	0	0
Littered	20	17	17	16
Pregnant/Mated Percentage ^b	100.0%	85.0%	85.0%	85.0%
Littered/Pregnant Percentage ^a	100.0%	100.0%	100.0%	94.1%
Litters Removed (Insufficient Size) (PND 4)	0	0	0	0
Litters Post Standardization (PND 4)	20	17	17	16
Weaned/Sex (PND 21) ^b	30	30	30	30

^a Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparisons against sham control group

^b Total number of weaned animals per sex from 10 litters

TABLE 31

Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Gestation in the 28-Day Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Gestation Day				
6	238.3 ± 2.2 (20) ^b	237.2 ± 1.8 (17)	236.9 ± 2.6 (17)	237.8 ± 2.2 (16)
9	250.7 ± 2.4 (20)	250.4 ± 2.4 (17)	252.3 ± 2.8 (17)	250.4 ± 2.1 (16)
12	266.2 ± 2.5 (20)	265.1 ± 2.3 (17)	266.6 ± 3.4 (17)	263.8 ± 2.5 (16)
15	282.5 ± 2.9 (20)	281.9 ± 2.7 (17)	283.5 ± 3.6 (17)	279.8 ± 2.7 (16)
18	319.3 ± 3.0 (20)	316.9 ± 2.3 (17)	318.0 ± 4.8 (17)	312.2 ± 3.1 (16)
21	366.4 ± 4.3 (20) [▲]	359.7 ± 3.5 (17)	360.0 ± 6.9 (17)	352.0 ± 3.6 (16)
Gestation Day Interval				
6 to 9	12.5 ± 1.2 (20)	13.2 ± 0.9 (17)	15.4 ± 0.9 (17)	12.6 ± 0.8 (16)
9 to 12	15.5 ± 1.1 (20)	14.7 ± 0.9 (17)	14.3 ± 1.3 (17)	13.4 ± 0.8 (16)
12 to 15	16.3 ± 1.0 (20)	16.8 ± 0.9 (17)	16.9 ± 1.3 (17)	16.0 ± 0.7 (16)
15 to 18	36.7 ± 0.9 (20) [▲]	35.0 ± 1.1 (17)	34.5 ± 1.9 (17)	32.4 ± 0.9 (16)*
18 to 21	47.1 ± 1.8 (20) ^{▲▲}	42.8 ± 1.6 (17)	42.0 ± 3.0 (17)	39.7 ± 1.4 (16)*
6 to 21	128.1 ± 3.3 (20) [▲]	122.5 ± 2.7 (17)	123.1 ± 6.2 (17)	114.2 ± 2.3 (16)*

[▲] Significant trend ($P \leq 0.05$) by Jonckheere's test

^{▲▲} Significant trend ($P \leq 0.01$) by Jonckheere's test

* Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests

^b Number of dams

Total and live litter size on PND 1 was unaffected by exposure and there was no statistically significant effect on live litter size throughout lactation (Table 32). However, there were higher numbers of dead pups in the exposed groups from PND 1 to 4 and in the 6 and 9 W/kg groups from PND 5 to 21 (Table 33).

TABLE 32

**Mean Number of Surviving F₁ Male and Female Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Total Pups per Litter				
PND 1	11.95 ± 0.38 (20) ^b	11.76 ± 0.30 (17)	11.18 ± 1.03 (17)	11.88 ± 0.48 (16)
Live Pups per Litter				
PND 1	11.90 ± 0.39 (20)	11.65 ± 0.32 (17)	10.82 ± 1.04 (17)	11.63 ± 0.50 (16)
PND 4 (Preculling)	11.85 ± 0.39 (20)	11.47 ± 0.33 (17)	10.47 ± 1.09 (17)	11.38 ± 0.56 (16)
PND 4 (Postculling)	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	7.06 ± 0.57 (17)	8.13 ± 0.09 (16)
PND 7	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.94 ± 0.57 (17)	7.75 ± 0.39 (16)
PND 10	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.88 ± 0.61 (17)	7.63 ± 0.52 (16)
PND 14	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.82 ± 0.64 (17)	7.63 ± 0.52 (16)
PND 17	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.82 ± 0.64 (17)	7.63 ± 0.52 (16)
PND 21	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.82 ± 0.64 (17)	7.63 ± 0.52 (16)
Live Males per Litter				
PND 1	6.10 ± 0.42 (20)	5.71 ± 0.43 (17)	5.00 ± 0.67 (17)	6.44 ± 0.47 (16)
PND 4 (Preculling)	6.05 ± 0.41 (20)	5.82 ± 0.50 (17)	5.12 ± 0.63 (17)	6.25 ± 0.52 (16)
PND 4 (Postculling)	4.00 ± 0.15 (20)	4.18 ± 0.23 (17)	3.53 ± 0.27 (17)	4.50 ± 0.37 (16)
Live Females per Litter				
PND 1	5.80 ± 0.42 (20)	5.94 ± 0.52 (17)	5.82 ± 0.69 (17)	5.19 ± 0.56 (16)
PND 4 (Preculling)	5.80 ± 0.43 (20)	5.65 ± 0.58 (17)	5.35 ± 0.65 (17)	5.13 ± 0.59 (16)
PND 4 (Postculling)	3.95 ± 0.14 (20)	3.82 ± 0.23 (17)	3.53 ± 0.33 (17)	3.63 ± 0.36 (16)

^a All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.

^b Number of dams

TABLE 33

**Offspring Mortality and Survival Ratio of Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Pup Survival per Litter				
Total Dead PND 1 to 4	2 (238/20) ^b	5 (198/17)	12 (184/17)	8 (186/16)
Total Dead PND 5 to 21	0 (159/20)	0 (136/17)	4 (120/17)	8 (130/16)
Dead/Litter PND 1 to 4	0.100 ± 0.069 (20)	0.294 ± 0.143 (17)	0.706 ± 0.318 (17)	0.500 ± 0.204 (16)
Dead/Litter PND 4 to 21	0.000 ± 0.000 (20)	0.000 ± 0.000 (17)	0.235 ± 0.136 (17)	0.500 ± 0.500 (16)
Survival Ratio PND 1 to 4 ^c	0.996 ± 0.004 (20)	0.985 ± 0.008 (17)	0.942 ± 0.031 (17)	0.975 ± 0.011 (16)
Survival Ratio PND 4 to 21 ^d	1.000 ± 0.000 (20)	1.000 ± 0.000 (17)	0.868 ± 0.081 (17)	0.938 ± 0.063 (16)

^a All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.

^c Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1)

^d Number of pups alive on PND 21/number of pups at postculling on PND 4

During the lactation period 9 W/kg dams had decreased weight gain, and their body weights were 5% to 12% lower than those in the sham controls from PND 7 through 21 (Table 34). F₁ body weights were 8% lower starting on PND 1 in the 9 W/kg group whether male, female, or combined when not adjusted or adjusted for litter size (Table 35). As lactation progressed, the adjusted pup weights (combined) were up to 23% lower in the 9 W/kg group and up to 16% lower in the 6 W/kg group compared to sham controls. The magnitude of the effect was consistent between males and females, with a recovery in the 6 W/kg group on PND 14 and 21.

TABLE 34
Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Postnatal Day				
1	272.7 ± 2.7 (20) ^b	268.9 ± 2.4 (17)	271.7 ± 4.1 (17)	263.8 ± 2.6 (16)
4	263.8 ± 3.6 (20)	264.1 ± 2.9 (16)	269.3 ± 4.1 (17)	264.6 ± 2.1 (16)
7	284.1 ± 2.7 (20) ^{▲▲}	282.5 ± 2.7 (17)	282.1 ± 4.0 (17)	269.6 ± 1.9 (16)**
14	292.4 ± 2.6 (20) ^{▲▲}	293.3 ± 2.7 (17)	284.9 ± 4.1 (17)	264.1 ± 3.1 (16)**
21	279.7 ± 3.5 (20) ^{▲▲}	282.6 ± 3.0 (17)	272.9 ± 3.7 (17)	245.9 ± 3.1 (16)**
Postnatal Day Interval				
1 to 4	-8.9 ± 3.2 (20) [▲]	-3.8 ± 3.3 (16)	-2.4 ± 3.1 (17)	0.8 ± 1.4 (16)
4 to 7	20.2 ± 2.3 (20) ^{▲▲}	18.1 ± 3.2 (16)	12.8 ± 2.2 (17)*	5.1 ± 1.2 (16)**
7 to 14	8.3 ± 1.4 (20) ^{▲▲}	10.8 ± 1.8 (17)	2.9 ± 2.3 (17)*	-5.5 ± 2.0 (16)**
14 to 21	-12.7 ± 3.5 (20)	-10.7 ± 2.8 (17)	-12.1 ± 2.7 (17)	-18.2 ± 2.4 (16)
1 to 21	7.0 ± 3.8 (20) ^{▲▲}	13.7 ± 2.7 (17)	1.1 ± 2.8 (17)	-17.9 ± 3.3 (16)**

▲ Significant trend ($P \leq 0.05$) by Jonckheere's test

▲▲ Significant trend ($P \leq 0.01$) by Jonckheere's test

* Significantly different ($P \leq 0.05$) by Shirley's or Dunn's test

** $P \leq 0.01$

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

^b Number of dams

TABLE 35
Adjusted Mean Body Weights of F₁ Male and Female Rats During Lactation
in the 28-Day Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Adjusted Male Live Pup Weight				
PND 1 ^d	6.73 ± 0.10 (20) ^{b▲▲}	6.76 ± 0.11 (17)	6.52 ± 0.10 (17)	6.16 ± 0.11 (16) ^{▲▲}
PND 4 (Preculling)	9.78 ± 0.14 (121/20) ^c	9.43 ± 0.16 (99/17)	8.78 ± 0.30 (87/17)**	8.14 ± 0.28 (100/16)**
PND 4 (Postculling)	9.84 ± 0.14 (81/20)**	9.57 ± 0.15 (69/17)	8.82 ± 0.30 (61/17)**	8.19 ± 0.27 (71/16)**
PND 7	16.33 ± 0.19 (81/20)**	15.30 ± 0.27 (69/17)	13.64 ± 0.67 (61/17)**	12.52 ± 0.61 (64/16)**
PND 14	31.61 ± 0.42 (81/20)**	30.91 ± 0.41 (69/17)	29.73 ± 0.47 (59/15)**	26.75 ± 0.47 (63/15)**
PND 21	53.33 ± 0.72 (81/20)**	52.57 ± 0.71 (69/17)	51.42 ± 0.75 (59/15)	45.11 ± 0.84 (63/15)**
Adjusted Female Live Pup Weight				
PND 1 ^d	6.30 ± 0.10 (20) ^{▲▲}	6.43 ± 0.11 (17)	6.14 ± 0.12 (16)	5.77 ± 0.13 (14) ^{▲▲}
PND 4 (Preculling)	9.11 ± 0.16 (116/20)**	8.85 ± 0.15 (96/17)	8.75 ± 0.17 (91/15)	7.86 ± 0.18 (82/14)**
PND 4 (Postculling)	9.19 ± 0.16 (79/20)**	9.00 ± 0.15 (67/17)	8.78 ± 0.17 (59/15)	7.97 ± 0.19 (57/14)**
PND 7	15.26 ± 0.24 (79/20)**	14.63 ± 0.24 (67/17)	13.90 ± 0.29 (57/15)**	12.29 ± 0.36 (57/14)**
PND 14	29.82 ± 0.40 (79/20)**	29.78 ± 0.38 (67/17)	28.57 ± 0.41 (57/15)	25.66 ± 0.51 (57/14)**
PND 21	49.87 ± 0.73 (79/20)**	49.50 ± 0.54 (67/17)	49.03 ± 0.64 (57/15)	43.24 ± 0.87 (57/14)**
Adjusted Combined Live Pup Weight				
PND 1 ^d	6.51 ± 0.10 (20) ^{▲▲}	6.60 ± 0.11 (17)	6.33 ± 0.11 (17)	5.99 ± 0.10 (16) ^{▲▲}
PND 4 (Preculling)	9.45 ± 0.13 (237/20)**	9.17 ± 0.15 (195/17)	8.51 ± 0.34 (178/17)*	7.90 ± 0.25 (182/16)**
PND 4 (Postculling)	9.52 ± 0.13 (160/20)**	9.30 ± 0.14 (136/17)	8.57 ± 0.33 (120/17)*	8.00 ± 0.25 (128/16)**
PND 7	15.79 ± 0.19 (160/20)**	14.97 ± 0.23 (136/17)	13.29 ± 0.69 (118/17)**	12.21 ± 0.58 (121/16)**
PND 14	30.70 ± 0.39 (160/20)**	30.33 ± 0.37 (136/17)	29.17 ± 0.43 (116/15)*	26.29 ± 0.46 (120/15)**
PND 21	51.57 ± 0.67 (160/20)**	51.07 ± 0.59 (136/17)	50.29 ± 0.68 (116/15)	44.31 ± 0.82 (120/15)**

▲▲ Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

* Significantly different ($P \leq 0.05$) for PNDs after PND 1 endpoints (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

** $P \leq 0.01$

^a Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND1, and the statistical analysis performed is by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. Individual pup body weights first adjusted for live PND 1 litter size via the analysis of covariance.

^b Number of dams

^c Number of pups/number of dams

^d Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1

Postnatal Exposure

All rats survived to the end of the study (Table 36). In males, there were lower mean body weights in the 3 (5% to 7%) and 9 W/kg groups (14% to 18%) compared to the sham controls at all time points including terminal sacrifice (except day 1 for the 3 W/kg group) (Table 36 and Figure 7). In 6 W/kg males, mean body weights were lower (6% to 8%) at all time points except terminal sacrifice. Mean body weight gains were lower (6% to 14%) in all three male exposed groups compared to sham controls (data not presented). In females, mean body weights were lower on days 1, 8, 15, and 22 in the 9 W/kg group (7% to 15%), on days 1, 8, and 15 in the 6 W/kg group (4% to 5%), and on day 8 in the 3 W/kg group (4%). However, at terminal sacrifice, the mean body weights of all exposed female groups were similar to those of the sham controls. Mean body weight gains of all exposed female groups were similar to that of the sham controls. There were no notable clinical observations in any groups of either sex during the study.

TABLE 36
Mean Body Weights and Survival of Rats Exposed to CDMA-Modulated Cell Phone RFR for 28 Days

Day	Sham Control		3 W/kg			6 W/kg			9 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
Male											
1	60.8	10	58.4	95.9	10	58.0	95.3	10	52.9	87.0	10
8	94.9	10	89.8	94.6	10	88.8	93.6	10	80.5	84.9	10
15	144.5	10	136.1	94.1	10	134.2	92.9	10	123.6	85.5	10
22	195.3	10	182.8	93.6	10	184.6	94.5	10	169.2	86.6	10
29	248.7	10	231.1	92.9	10	236.4	95.0	10	215.0	86.4	10
Female											
1	55.9	10	53.5	95.8	10	55.7	99.7	10	49.5	88.6	10
8	83.1	10	79.5	95.6	10	81.8	98.3	10	73.4	88.3	10
15	119.8	10	114.4	95.5	10	118.5	98.9	10	107.9	90.1	10
22	146.5	10	144.6	98.7	10	148.7	101.5	10	136.9	93.4	10
30	166.5	10	162.7	97.7	10	171.7	103.1	10	159.0	95.5	10

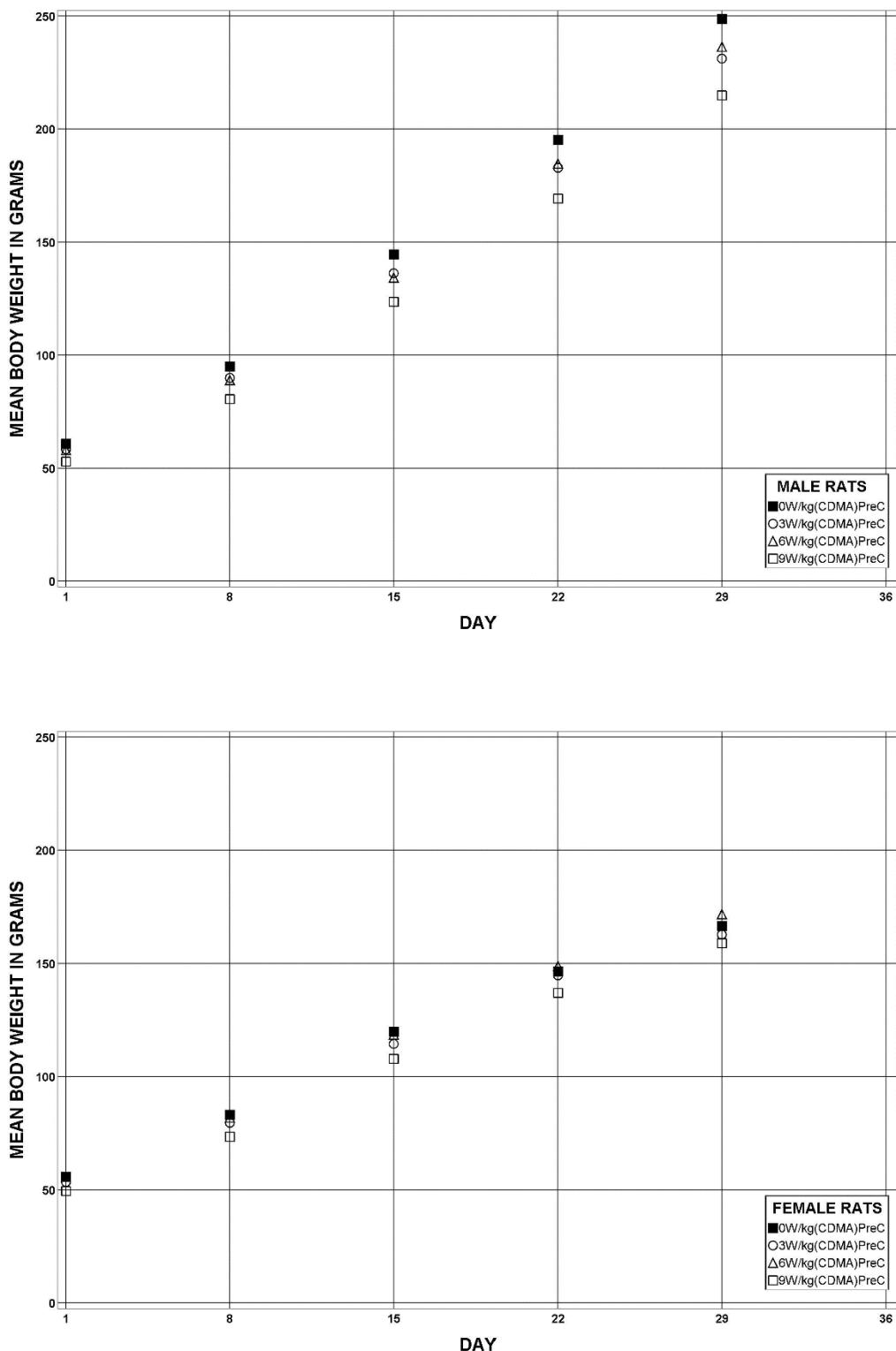


FIGURE 7
Growth Curves for Rats Exposed to CDMA-Modulated Cell Phone RFR for 28 Days

Body temperatures were significantly, but sporadically increased in F₀ females at several time points (Table 37; Figure 9). Body temperature in the F₁ groups were similar to those of the sham controls throughout the study (Tables 37 and G5).

TABLE 37**Mean Body Temperatures of Rats Exposed to CDMA-Modulated Cell Phone RFR for 28 Days^a**

Day	Sham Control		3 W/kg		6 W/kg		9 W/kg	
	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured
F₀ Female^b								
GD 6	36.7 ± 0.1	10 ^c	36.8 ± 0.1	9	36.6 ± 0.2	9	36.4 ± 0.2	8
GD 7	36.6 ± 0.1*	10	36.3 ± 0.2	9	36.6 ± 0.1	9	37.2 ± 0.1*	8
GD 11	36.7 ± 0.2	10	36.4 ± 0.1	9	36.2 ± 0.2*	9	36.8 ± 0.2	8
GD 16	36.5 ± 0.1*	10	36.5 ± 0.2	9	36.6 ± 0.1	9	37.2 ± 0.2*	8
GD 7-16 ^d	36.6 ± 0.1	10	36.4 ± 0.1	9	36.4 ± 0.1	9	37.1 ± 0.1*	8
LD 1	37.7 ± 0.1	10	37.3 ± 0.2	9	37.7 ± 0.1	9	37.9 ± 0.2	8
LD 4	36.7 ± 0.1**	10	37.0 ± 0.2	9	37.3 ± 0.3*	9	38.1 ± 0.2**	8
LD 7	36.8 ± 0.2*	10	37.0 ± 0.1	9	37.4 ± 0.2	9	37.5 ± 0.3	8
LD 14	36.9 ± 0.2**	10	37.0 ± 0.3	9	37.6 ± 0.1*	9	38.3 ± 0.3**	7
LD 7-14 ^d	37.0 ± 0.1**	10	37.1 ± 0.1	9	37.5 ± 0.1*	9	37.9 ± 0.2**	8
F₁ Male^e								
16	37.3 ± 0.1	4	37.0 ± 0.1	4	37.1 ± 0.1	4	37.3 ± 0.1	4
20	37.6 ± 0.1	4	37.2 ± 0.1	4	37.1 ± 0.1	4	37.5 ± 0.2	4
27	37.2 ± 0.1	4	36.8 ± 0.1	4	37.0 ± 0.1	4	37.4 ± 0.2	4
16-27 ^d	37.4 ± 0.1	4	37.0 ± 0.1	4	37.1 ± 0.1	4	37.5 ± 0.2	4
F₁ Female^e								
16	37.9 ± 0.2	4	37.2 ± 0.2*	3	37.5 ± 0.1	4	37.5 ± 0.1	4
20	38.0 ± 0.2*	4	38.0 ± 0.1	4	37.6 ± 0.2	4	37.6 ± 0.2	4
27	37.9 ± 0.2	4	37.1 ± 0.2	4	38.0 ± 0.2	4	38.0 ± 0.2	4
16-27 ^d	37.9 ± 0.1	4	37.4 ± 0.1	4	37.7 ± 0.1	4	37.7 ± 0.1	4

* Significantly different ($P \leq 0.05$)

** $P \leq 0.01$

a Temperatures are given as mean ± standard error. GD=gestation day; LD=lactation day.

b Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

c For F₀ females, number measured refers to individual animals, for F₁ pups numbers measured refers to litters.

d Average of days

e Statistical analysis for linear trends was performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.

Male body weights were lower at 9W/kg at the end of the 28-day studies. There were a few statistically significant changes in organ to body weight ratios at this exposure level (Table G6) that were considered secondary to the reduced body weights. There were no biologically significant changes in organ to body weights in females.

Kidney: There was a higher incidence of chronic progressive nephropathy in the 6 W/kg females compared to controls (0/10, 2/10, 4/10*, 3/10; *significantly different ($P \leq 0.05$) from the sham control group by the Fisher exact test). The positive trend was also statistically significant ($P=0.045$). The severity in all cases was minimal. This lesion was characterized by scattered tubular segments with basophilic epithelial cells with crowded nuclei, slightly thickened basement membranes, and occasional mononuclear inflammatory cells. Chronic progressive nephropathy is a very common background lesion in rats. Therefore, chronic progressive nephropathy was not considered to be related to treatment with CDMA-modulated cell phone RFR. There were no exposure-related renal lesions in male rats.

[Table 38 omitted]

Exposure Level Selection Rationale: Based on reduced maternal and pup weights and increased body temperature measurements at 9 W/kg in the 28-day studies and increased body temperature in adult rats at ≥ 8 W/kg in the thermal pilot studies (Wyde *et al.*, 2018), the highest exposure level selected for the 2-year studies was 6 W/kg. In the thermal pilot studies and 28-day studies, exposure to 6 W/kg resulted in some increases in core body temperature, but these increases were less than 1° C. Therefore, 6 W/kg would provide an exposure adequate to challenge the animals without causing excessive heating or disruption of the thermoregulatory process. The lowest exposure level selected for the 2-year studies was 1.5 W/kg, which is close to the 1.6 W/kg maximum output limit for cell phone devices in the United States.

2-YEAR STUDY

Perinatal Exposure

No exposure-related effects were observed on pregnancy status, maternal survival, or the percent of pregnant animals that littered (Table 39). Maternal body weights during gestation were similar to those of the sham control group (Table 40). Body weight gains were generally unaffected across time intervals except in the 3 W/kg group at the GD 6 through 9 interval where weight gains were lower than that of the sham control group, but this was not considered to be exposure related.

TABLE 39
Summary of Disposition During Perinatal Exposure and F₁ Allocation
in the 2-Year Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Time-mated Females	56	56	56	56
Pregnant Females	52	50	48	49
Non-Pregnant Females	4	6	8	7
Pregnant Dams not Delivering	2	2	2	1
Died ^a	1	0	0	0
Littered	50	48	46	48
Pregnant/Mated Percentage ^b	92.9%	89.3%	85.7%	87.5%
Littered/Pregnant Percentage ^b	96.2%	96.0%	95.8%	98.0%
Litters Removed (Insufficient Size)	2	3	3	2
Litters Post Standardization	48	45	43	46
Weaned/Sex ^c	105	105	105	105

^a One pregnant female died on GD 25 with pups in uterus

^b Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparisons against sham control group.

^c Total number of weaned animals per sex from 35 litters

TABLE 40
**Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Gestation
in the 2-Year Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Gestation Day				
6	238.4 ± 1.4 (51) ^b	239.0 ± 1.3 (48)	239.9 ± 1.6 (46)	237.8 ± 1.6 (48)
9	256.2 ± 1.6 (51)	255.2 ± 1.4 (48)	254.5 ± 1.8 (46)	253.7 ± 1.7 (48)
12	270.5 ± 1.6 (51)	268.7 ± 1.4 (48)	268.8 ± 1.6 (46)	267.0 ± 1.7 (48)
15	290.0 ± 1.9 (51)	288.5 ± 1.6 (48)	289.3 ± 1.8 (46)	287.5 ± 2.0 (48)
18	332.7 ± 2.3 (51)	329.7 ± 2.1 (48)	329.1 ± 2.3 (46)	329.0 ± 2.5 (48)
21	380.2 ± 2.8 (51)	375.9 ± 3.0 (48)	375.1 ± 3.2 (46)	375.0 ± 3.1 (48)
Gestation Day Interval				
6 to 9	17.7 ± 0.8 (51)	16.1 ± 0.7 (48)	14.6 ± 1.0 (46)*	15.9 ± 0.7 (48)
9 to 12	14.3 ± 0.6 (51)	13.6 ± 0.5 (48)	14.3 ± 1.0 (46)	13.3 ± 0.6 (48)
12 to 15	19.6 ± 0.6 (51)	19.8 ± 0.5 (48)	20.5 ± 0.7 (46)	20.5 ± 0.5 (48)
15 to 18	42.7 ± 1.0 (51)	41.3 ± 1.1 (48)	39.8 ± 0.8 (46)	41.5 ± 0.8 (48)
18 to 21	47.5 ± 1.0 (51)	46.2 ± 1.2 (48)	46.0 ± 1.2 (46)	46.0 ± 1.0 (48)
6 to 21	141.7 ± 2.2 (51)	136.9 ± 2.8 (48)	135.2 ± 2.5 (46)	137.1 ± 2.3 (48)

* Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

^b Number of dams

On PND 1, there were no effects on total litter size or live litter size (Table 41). However, beginning on PND 7, live litter size was decreased in the 6 W/kg groups compared to the sham control group. There was a higher incidence of pup mortality (found dead or missing and presumed cannibalized) between PNDs 4 and 21 in the 6 W/kg group that corresponded to an increase in average number of dead pups per litter and a reduced survival ratio (Table 42).

TABLE 41
Mean Number of Surviving F₁ Male and Female Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total Pups per Litter				
PND 1	12.76 ± 0.32 (50) ^b	12.42 ± 0.41 (48)	12.43 ± 0.39 (46)	12.94 ± 0.35 (48)
Live Pups per Litter				
PND 1	12.56 ± 0.40 (50)	12.33 ± 0.42 (48)	12.39 ± 0.41 (46)	12.94 ± 0.35 (48)
PND 4 (Preculling)	12.73 ± 0.30 (48)	12.72 ± 0.26 (46)	12.77 ± 0.31 (43)	12.87 ± 0.30 (46)
PND 4 (Postculling)	8.00 ± 0.00 (48)	8.00 ± 0.00 (45)	8.00 ± 0.00 (43)	8.00 ± 0.00 (46)
PND 7	8.00 ± 0.00 (48)**	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.85 ± 0.06 (46)**
PND 10	8.00 ± 0.00 (48)**	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.76 ± 0.08 (46)**
PND 14	8.00 ± 0.00 (48)**	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.52 ± 0.11 (46)**
PND 17	8.00 ± 0.00 (48)**	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.52 ± 0.11 (46)**
PND 21	8.00 ± 0.00 (48)**	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.52 ± 0.11 (46)**
Live Males per Litter				
PND 1	6.20 ± 0.30 (50)	6.33 ± 0.30 (48)	6.07 ± 0.31 (46)	6.69 ± 0.30 (48)
PND 4 (Preculling)	6.33 ± 0.28 (48)	6.78 ± 0.24 (46)	6.26 ± 0.33 (43)	6.76 ± 0.29 (46)
PND 4 (Postculling)	3.96 ± 0.05 (48)	4.00 ± 0.06 (45)	3.95 ± 0.09 (43)	4.04 ± 0.05 (46)
Live Females per Litter				
PND 1	6.36 ± 0.28 (50)	6.00 ± 0.32 (48)	6.33 ± 0.36 (46)	6.25 ± 0.26 (48)
PND 4 (Preculling)	6.40 ± 0.25 (48)	5.93 ± 0.28 (46)	6.51 ± 0.35 (43)	6.11 ± 0.24 (46)
PND 4 (Postculling)	4.04 ± 0.05 (48)	4.00 ± 0.06 (45)	4.05 ± 0.09 (43)	3.96 ± 0.05 (46)

** Significantly different ($P \leq 0.01$)

a All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

b Number of dams

TABLE 42
Offspring Mortality and Survival Ratio of Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pup Survival per Litter				
Total Dead PND 1 to 4 ^b	19 (628/49) ^c	9 (592/46)	6 (570/43)	16 (621/46)
Total Dead PND 5 to 21	0 (348/48)	1 (360/45)	1 (344/43)	22 (368/46)
Dead/Litter PND 1 to 4	0.388 ± 0.193 (49)	0.196 ± 0.074 (46)	0.140 ± 0.063 (43)	0.348 ± 0.099 (46)
Dead/Litter PND 4 to 21	0.000 ± 0.000 (48)**	0.022 ± 0.022 (45)	0.023 ± 0.023 (43)	0.478 ± 0.106 (46)**
Survival Ratio PND 1 to 4 ^d	0.986 ± 0.005 (48)	0.991 ± 0.004 (46)	0.989 ± 0.005 (43)	0.975 ± 0.007 (46)
Survival Ratio PND 4 to 21 ^e	1.000 ± 0.000 (48)**	0.997 ± 0.003 (45)	0.997 ± 0.003 (43)	0.940 ± 0.013 (46)**

** Significantly different ($P \leq 0.01$)

a All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.

b Includes dead on PND 1. Survival information on PND 4 was not available for some non-acceptable litters, so these were excluded from te analysis.

c Number of pups/number of dams

d Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1)

e Number of pups alive on PND 21/number of pups at postculling on PND 4

During the lactation period, maternal body weights and body weight gains (PND 1-21) in the 3 and 6 W/kg groups were significantly decreased (up to 3% and 7%, respectively) compared to sham controls (Table 43). At PND 1, male and female pup weights in the 6 W/kg groups were 5% to 6% less than those of the sham controls (Table 44). Male and female pup weights were also significantly decreased compared to the sham controls in the 3 W/kg groups at PND 4 and in the 6 W/kg groups at all time points. The lower weights occurred in similar magnitudes between the sexes with 5% to 6% decreases at PND 4 in the 3 W/kg group and up to 15% decreases in the 6 W/kg group at PND 7.

TABLE 43
Mean Body Weights and Mean Body Weight Gains of F₀ Female Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Postnatal Day				
1	280.9 ± 2.0 (50) ^b	278.3 ± 1.7 (48)	278.2 ± 1.8 (45)	275.8 ± 2.2 (48)
4	289.7 ± 2.1 (48) ^{▲▲}	289.1 ± 2.0 (45)	286.4 ± 1.8 (43)	282.3 ± 2.0 (46)**
7	297.1 ± 2.2 (48) ^{▲▲}	297.4 ± 1.9 (45)	293.5 ± 1.8 (43)	285.3 ± 1.9 (45)**
14	314.4 ± 2.0 (48) ^{▲▲}	309.8 ± 1.9 (45)	306.6 ± 2.1 (43)*	293.5 ± 2.5 (46)**
17	313.9 ± 2.2 (48) ^{▲▲}	307.8 ± 1.9 (45)*	303.6 ± 2.1 (42)**	294.9 ± 2.4 (46)**
21	299.7 ± 2.2 (48) ^{▲▲}	293.8 ± 2.0 (45)*	291.2 ± 1.8 (43)**	277.6 ± 2.2 (46)**
Postnatal Day Interval				
1 to 4	9.2 ± 0.9 (48) ^{▲▲}	11.2 ± 1.0 (45)	7.5 ± 0.8 (42)	5.7 ± 1.0 (46)**
4 to 7	7.4 ± 1.5 (48) ^{▲▲}	8.2 ± 1.0 (45)	7.1 ± 1.0 (43)	3.4 ± 1.3 (45)*
7 to 14	17.4 ± 1.4 (48) ^{▲▲}	12.5 ± 1.0 (45)*	13.1 ± 1.5 (43)*	7.7 ± 1.4 (45)**
14 to 17	-0.6 ± 1.0 (48)	-2.0 ± 1.2 (45)	-2.6 ± 1.3 (42)	1.3 ± 1.0 (46)
17 to 21	-14.1 ± 1.3 (48)	-14.0 ± 1.4 (45)	-12.7 ± 1.5 (42)	-17.3 ± 1.5 (46)
1 to 21	19.3 ± 1.5 (48) ^{▲▲}	15.9 ± 1.7 (45)	12.2 ± 1.6 (42)**	1.0 ± 1.4 (46)**

^{▲▲}Significant trend ($P \leq 0.01$) by Jonckheere's test

* Significantly different ($P \leq 0.05$) by Williams' or Dunnett's test

** $P \leq 0.01$

^a Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.

^b Number of dams

TABLE 44
Adjusted Mean Body Weights of F₁ Male and Female Rats During Lactation
in the 2-Year Perinatal and Postnatal Study of CDMA-Modulated Cell Phone RFR^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted Male Live Pup Weight				
PND 1 ^b	7.19 ± 0.07 (49) ^{c▲▲}	7.02 ± 0.07 (47)	7.09 ± 0.06 (46)	6.78 ± 0.06 (48) ^{▲▲}
PND 4	10.87 ± 0.12 (190/48) ^{d**}	10.54 ± 0.15 (166/42)	10.35 ± 0.11 (169/43)**	9.58 ± 0.11 (178/44)**
PND 7	17.18 ± 0.19 (189/48)**	16.83 ± 0.22 (179/45)	16.82 ± 0.18 (166/42)	14.75 ± 0.25 (183/46)**
PND 14	35.20 ± 0.43 (181/46)**	33.89 ± 0.42 (179/45)	34.48 ± 0.32 (170/43)	30.81 ± 0.52 (176/46)**
PND 17	42.44 ± 0.49 (190/48)**	41.55 ± 0.50 (179/45)	41.88 ± 0.40 (170/43)	38.08 ± 0.60 (175/46)**
PND 21	58.46 ± 0.62 (190/48)**	57.30 ± 0.72 (179/45)	57.85 ± 0.55 (170/43)	52.63 ± 0.83 (176/46)**
Adjusted Female Live Pup Weight				
PND 1 ^b	6.79 ± 0.06 (49) ^{▲▲}	6.65 ± 0.09 (47)	6.73 ± 0.06 (45)	6.44 ± 0.05 (48) ^{▲▲}
PND 4	10.43 ± 0.11 (194/48)**	10.16 ± 0.13 (172/43)	9.80 ± 0.12 (171/43)**	9.21 ± 0.11 (182/46)**
PND 7	16.45 ± 0.18 (194/48)**	16.16 ± 0.21 (180/45)	15.90 ± 0.20 (173/43)	14.12 ± 0.22 (174/45)**
PND 14	33.87 ± 0.39 (192/48)**	32.71 ± 0.38 (180/45)	32.91 ± 0.37 (169/42)	29.59 ± 0.50 (170/46)**
PND 17	40.80 ± 0.45 (194/48)**	39.99 ± 0.47 (180/45)	39.90 ± 0.42 (173/43)	36.58 ± 0.55 (170/46)**
PND 21	55.38 ± 0.53 (194/48)**	54.23 ± 0.64 (180/45)	54.37 ± 0.57 (173/43)	50.29 ± 0.70 (170/46)**
Adjusted Combined Live Pup Weight				
PND 1 ^b	6.99 ± 0.06 (49) ^{▲▲}	6.83 ± 0.09 (48)	6.90 ± 0.06 (46)	6.62 ± 0.05 (48) ^{▲▲}
PND 4	10.65 ± 0.11 (384/48)**	10.36 ± 0.13 (338/44)	10.07 ± 0.11 (340/43)**	9.39 ± 0.10 (360/46)**
PND 7	16.80 ± 0.18 (383/48)**	16.50 ± 0.20 (359/45)	16.34 ± 0.18 (339/43)	14.45 ± 0.22 (357/46)**
PND 14	34.46 ± 0.40 (373/48)**	33.31 ± 0.38 (359/45)	33.69 ± 0.32 (339/43)	30.23 ± 0.48 (346/46)**
PND 17	41.60 ± 0.46 (384/48)**	40.78 ± 0.47 (359/45)	40.88 ± 0.39 (343/43)	37.35 ± 0.54 (345/46)**
PND 21	56.89 ± 0.56 (384/48)**	55.77 ± 0.65 (359/45)	56.10 ± 0.53 (343/43)	51.51 ± 0.71 (346/46)**

▲▲Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

** Significantly different ($P \leq 0.01$) for PNDs after PND 1 endpoints (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test)

^a Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND1 refer to the total pup weight divided by the number of pups in litter at PND 1, and the statistical analysis was performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. Individual pup body weights first adjusted for live PND 1 litter size via the analysis of covariance.

^b Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1.

^c Number of dams

^d Number of pups/number of dams

Postnatal Exposure

Survival

Estimates of 2-year survival probabilities for male and female rats are shown in Table 45 and in the Kaplan-Meier survival curves (Figure 8). In males, survival was greater in all exposed groups compared to sham controls, though it was statistically significant only in the 1.5 and 3 W/kg groups. Survival in the sham control group was 28% compared to 48%, 62%, and 48% in the 1.5, 3, and 6 W/kg groups, respectively. Decreased survival in the sham control group was largely attributed to the higher severity of chronic progressive nephropathy in the kidney. In females, there was a small, but statistically significant increase in survival in the 6 W/kg group. Survival in the sham control females was similar to that in the 1.5 and 3 W/kg groups.

TABLE 45
Survival of Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Animals initially in study	105	105	105	105
14-week interim evaluation ^a	15	15	15	15
Accidental death ^b	1	0	0	0
Moribund	44	24	13	6
Natural deaths	20	23	21	41
Animals surviving to study termination	25	43	56 ^f	43
Percent probability of survival at end of study ^c	28	48	62	48
Mean survival (days) ^d	642	675	687	637
Survival analysis ^e	P=0.070N	P=0.005N	P<0.001N	P=0.072N
Female				
Animals initially in study	105	105	105	105
14-week interim evaluation ^a	15	15	15	15
Accidental death	1	0	0	0
Moribund	30	29	28	16
Natural deaths	11	15	12	13
Animals surviving to study termination	48 ^f	46 ^g	50	61
Percent probability of survival at end of study	54	50	56	68
Mean survival (days)	659	673	665	701
Survival analysis	P=0.020N	P=1.000	P=0.841N	P=0.037N

^a Excluded from survival analysis

^b Censored in the survival analysis

^c Kaplan-Meier determinations

^d Mean of all deaths (uncensored, censored, and terminal euthanasia)

^e The result of the life table trend test (Tarone, 1975) is in the sham control column, and the results of the life table pairwise comparisons (Cox, 1972) with the sham controls are in the exposed group columns. A negative trend or lower mortality in an exposure group is indicated by N.

^f Includes one animal that died during the last week of the study

^g Includes two animals that died during the last week of the study

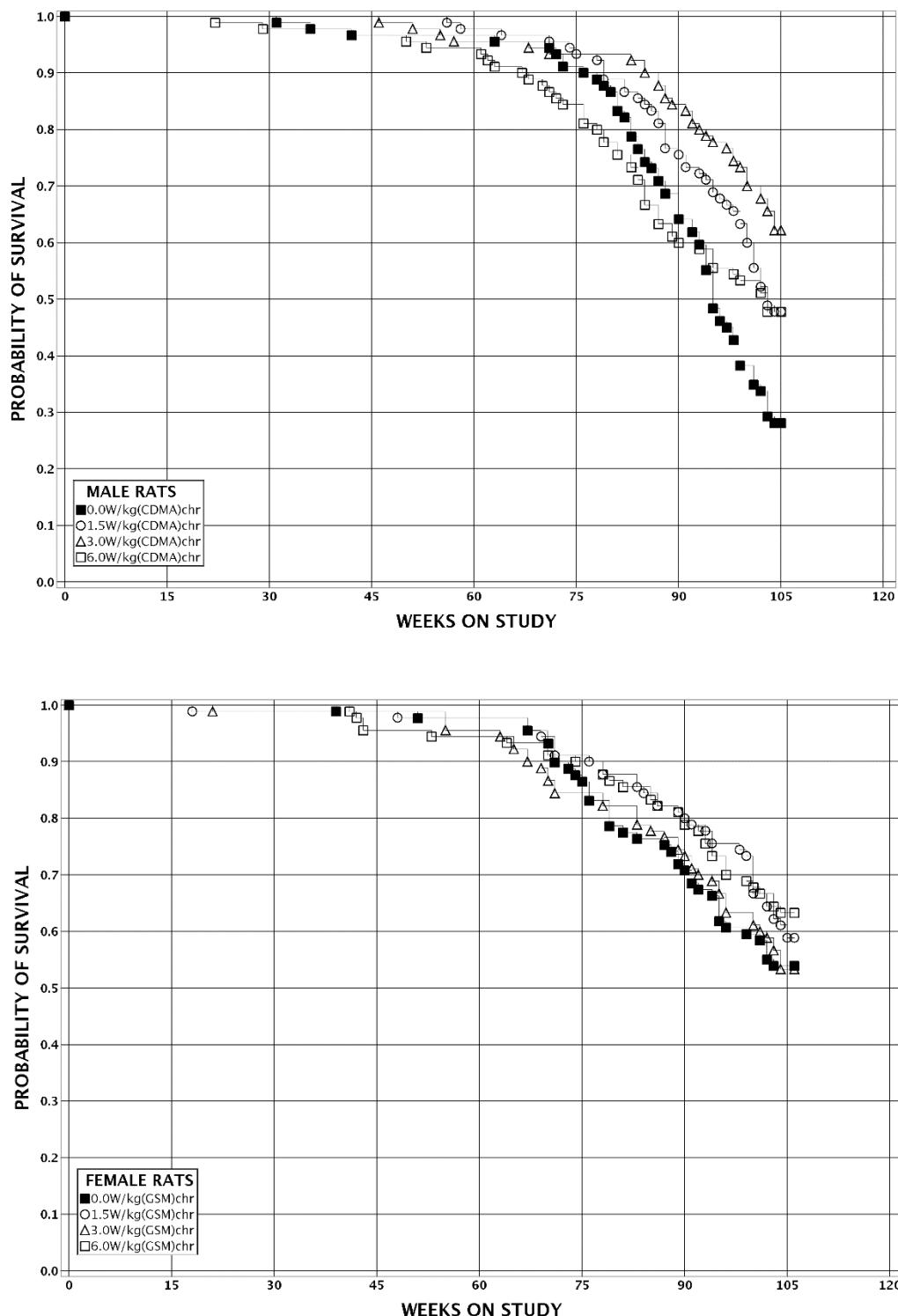


FIGURE 8
Kaplan-Meier Survival Curves for Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

Body Weights and Clinical Observations

In 6 W/kg males, body weights were lower (4% to 9%) than those of the sham controls at all time points through day 457 (Figure 9 and Table 46); however, at the end of the study, the mean body weight was similar to that of the sham controls. In 1.5 and 3 W/kg males, mean body weights were significantly higher (compared to sham controls) at several time points, but at the end of the study, though the mean body weights were higher than in sham controls, the difference was not statistically significant. Mean body weights of exposed females were similar to those of the sham controls throughout the study (Figure 9 and Table 47). There were no clinical observations in males or females related to CDMA-modulated cell phone RFR exposure.

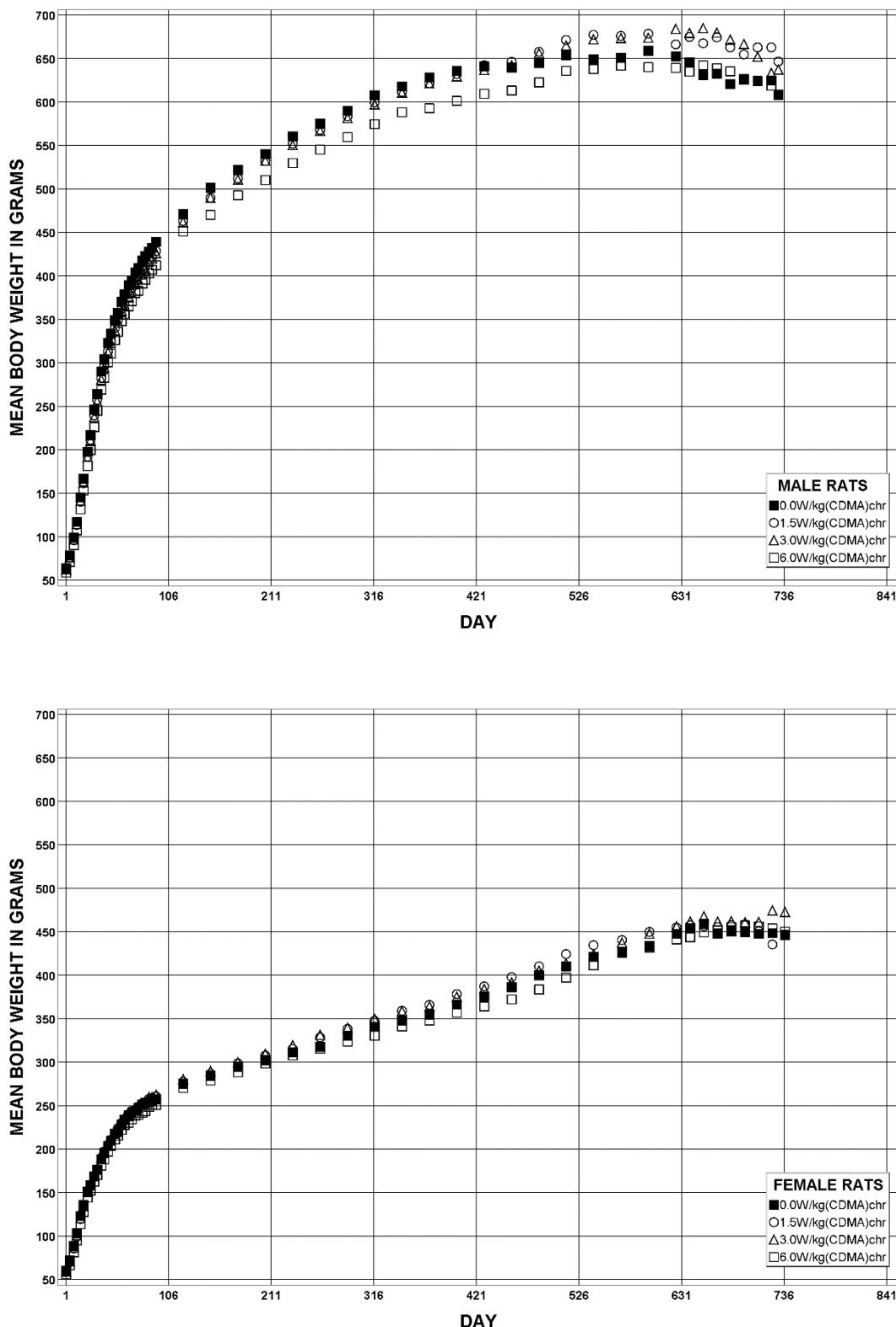


FIGURE 9
Growth Curves for Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

TABLE 46
Mean Body Weights and Survival of Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

Day	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
1	64.3	105	62.6	97.4	105 ^a	63.0	98.1	105	59.2	92.1	105
5	78.7	105	76.0	96.6	105	76.5	97.3	105	71.4	90.8	105
9	99.0	105	96.2	97.2	105	96.8	97.7	105	90.5	91.5	105
12	117.6	105	113.7	96.7	105	114.9	97.7	105	107.5	91.4	105 ^a
16	145.5	105	140.4	96.5	105	141.5	97.3	105	132.0	90.7	105
19	167.0	105	161.7	96.8	105	162.4	97.3	105	153.5	92.0	105
23	197.8	105	192.2	97.2	105	192.0	97.1	105	181.5	91.8	105
26	217.1	105	211.6	97.4	105	211.0	97.2	105	200.1	92.2	105
30	246.5	105	239.5	97.2	105	237.8	96.5	105	226.5	91.9	105
33	264.3	105	257.4	97.4	105	255.7	96.7	105	245.2	92.8	105
37	290.4	105	282.6	97.3	105	280.9	96.7	105	269.5	92.8	105
40	304.5	105	296.4	97.3	105	294.9	96.9	105	283.2	93.0	105
44	322.9	105	313.4	97.0	105	312.6	96.8	105	301.0	93.2	105
47	333.8	105	324.6	97.2	105	323.1	96.8	105	310.9	93.1	105
51	349.0	105	340.3	97.5	105	337.7	96.8	105	326.6	93.6	105
54	357.6	105	349.4	97.7	105	347.6	97.2	105	335.7	93.9	105
58	370.2	105	361.8	97.8	105	360.2	97.3	105	348.3	94.1	105
61	379.1	105	368.0	97.1	105	367.5	96.9	105	356.0	93.9	105
65	389.6	105	379.1	97.3	105	376.3	96.6	105	365.1	93.7	105
68	395.2	105	386.3	97.7	105	383.4	97.0	105	371.3	94.0	105
72	404.0	105	392.9	97.2	105	391.9	97.0	105	380.8	94.3	105
75	409.0	105	399.7	97.7	105	396.1	96.8	105	383.3	93.7	105
79	417.9	105	406.8	97.3	105	404.1	96.7	105	391.5	93.7	105
82	422.7	105	411.3	97.3	105	408.6	96.7	105	395.8	93.6	105
86	427.7	105	417.1	97.5	105	416.3	97.3	105	403.0	94.2	105
89	432.4	105	422.0	97.6	105	420.4	97.2	105	407.3	94.2	105
93	439.3	105	428.6	97.6	105	426.2	97.0	105	412.5	93.9	105
121 ^b	470.9	90	462.1	98.1	90	463.0	98.3	90	452.2	96.0	90
149	501.8	90	488.9	97.4	90	491.2	97.9	90	472.2	94.1	90
177	522.1	90	510.6	97.8	90	512.3	98.1	90	495.2	94.8	89
205	540.7	90	530.6	98.1	90	534.0	98.8	90	511.4	94.6	88
233	561.3	89	550.3	98.0	90	552.1	98.4	90	530.9	94.6	88
261	576.1	88	566.3	98.3	90	569.1	98.8	90	547.2	95.0	88
289	591.0	88	582.2	98.5	90	584.1	98.8	90	561.5	95.0	87
317	607.5	87	597.9	98.4	90	599.6	98.7	89	577.0	95.0	87
345	617.9	87	610.1	98.7	90	613.4	99.3	89	590.3	95.5	87
373	628.1	87	620.5	98.8	90	623.8	99.3	88	594.1	94.6	85
401	636.2	87	631.5	99.3	89	632.5	99.4	86	603.9	94.9	85
429	640.8	87	641.9	100.2	88	640.5	99.9	86	611.0	95.3	84
457	639.1	86	645.9	101.1	87	646.3	101.1	86	614.6	96.2	82
485	644.7	86	658.9	102.2	87	661.2	102.6	85	625.5	97.0	80
513	654.1	82	672.1	102.8	86	671.2	102.6	84	637.7	97.5	76
541	651.9	80	678.5	104.1	84	679.3	104.2	84	640.9	98.3	73
569	649.3	73	674.4	103.9	79	681.0	104.9	84	644.4	99.2	68
597	658.1	66	679.2	103.2	75	682.7	103.8	81	640.5	97.3	60
625	646.6	59	674.2	104.3	68	691.1	106.9	76	638.2	98.7	55
639	638.4	57	676.0	105.9	66	687.3	107.7	75	632.7	99.1	54
653	627.9	53	669.2	106.6	65	689.3	109.8	71	638.9	101.8	53
667	638.2	42	671.6	105.2	61	683.9	107.2	70	634.7	99.4	50
681	625.4	39	661.6	105.8	60	675.8	108.1	69	633.3	101.3	49
695	624.8	34	655.1	104.9	55	671.5	107.5	65	624.7	100.0	48
709	620.7	31	661.1	106.5	50	659.4	106.2	63	622.8	100.3	47
723	632.6	25	665.3	105.2	43	648.3	102.5	59	620.1	98.0	43
Mean for Weeks											
1-14	297.9		290.1	97.4		288.9	96.7		278.1	93.4	
15-52	554.4		544.3	98.2		546.5	98.6		526.4	94.9	
53-104	638.6		661.0	103.5		666.2	104.3		626.9	98.2	

^a The number of animals weighed on this day is less than the number of animals surviving.

^b Interim evaluation occurred during week 14

TABLE 47
Mean Body Weights and Survival of Female Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

Day	Sham Control		2.5 W/kg			5 W/kg			10 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
1	60.4	105	59.3	98.2	105	59.6	98.7	105	55.9	92.6	105
5	72.1	105	70.2	97.3	105 ^a	71.4	99.0	105	66.6	92.5	105
9	88.7	105	85.8	96.7	105	87.1	98.2	105	81.7	92.1	105
12	103.3	105	99.6	96.5	105	101.1	97.9	105	95.2	92.1	105
16	123.2	105	119.7	97.1	105	120.6	97.9	104	113.9	92.4	105
19	136.3	105	133.7	98.1	105	134.8	98.9	104	128.1	94.0	105
23	151.5	105	150.7	99.4	105	150.8	99.5	104	144.8	95.6	105
26	158.5	105	159.3	100.5	105	160.0	100.9	104	152.8	96.4	105
30	169.1	105	168.4	99.6	105	169.8	100.5	104	162.8	96.3	105
33	176.4	105	177.2	100.4	105	177.6	100.7	104	170.6	96.7	105
37	188.7	105	190.2	100.8	105	190.2	100.8	104	181.4	96.1	105
40	195.8	105	197.3	100.8	105	198.0	101.1	104	188.6	96.3	105
44	203.7	105	205.3	100.8	105	206.2	101.2	104	197.6	97.0	105
47	209.9	105	210.9	100.5	105	211.8	100.9	104	204.3	97.4	105
51	218.1	105	218.4	100.1	105	221.1	101.4	104	211.5	97.0	105
54	220.1	105	224.4	102.0	105	225.7	102.6	104	215.8	98.0	105
58	228.6	105	230.4	100.8	105	232.8	101.9	104	222.6	97.4	105
61	233.9	105	234.6	100.3	105	237.3	101.5	104	228.0	97.5	105
65	239.0	105	238.7	99.9	105	238.0	99.6	104	230.5	96.5	105
68	241.7	105	244.0	100.9	105	242.2	100.2	104	234.6	97.1	105
72	244.9	105	244.9	100.0	105	246.0	100.4	104	239.2	97.7	105
75	247.6	105	249.2	100.7	105	248.9	100.5	104	239.5	96.7	105
79	251.6	105	252.4	100.3	105	251.1	99.8	104	242.1	96.2	105
82	253.5	105	253.3	99.9	105	253.3	99.9	104	243.7	96.1	105
86	254.4	105	257.8	101.4	105	259.9	102.2	104	249.0	97.9	105
89	256.2	105	258.9	101.0	105	259.5	101.3	104	251.0	97.9	105
93 ^b	257.4	95	261.3	101.5	95	262.3	101.9	94	250.9	97.5	95
121 ^b	275.7	90	277.7	100.7	90	280.6	101.8	89	270.7	98.2	90
149	285.0	90	287.3	100.8	90	290.1	101.8	89	279.9	98.2	90
177	295.0	90	299.3	101.5	90	300.4	101.8	89	288.7	97.9	90
205	302.9	89	308.0	101.7	90	310.6	102.5	89	299.5	98.9	90
233	311.9	89	315.8	101.3	90	320.6	102.8	89	308.8	99.0	90
261	318.1	89	328.3	103.2	90	332.1	104.4	89	316.4	99.5	90
289	331.0	88	338.8	102.3	90	340.5	102.9	88	324.5	98.0	90
317	341.8	88	346.1	101.3	87	351.4	102.8	88	332.3	97.2	89
345	349.2	88	356.6	102.1	85	360.3	103.2	88	342.8	98.2	89
373	356.1	87	363.7	102.1	85	366.3	102.9	88	349.6	98.2	89
401	367.6	87	375.4	102.1	85	377.2	102.6	88	358.7	97.6	89
429	377.0	87	384.9	102.1	84	385.8	102.3	86	366.5	97.2	89
457	387.5	87	395.7	102.1	84	393.5	101.5	85	374.5	96.6	89
485	400.0	85	408.4	102.1	83	405.6	101.4	83	386.3	96.6	89
513	410.8	78	423.0	103.0	82	417.2	101.6	79	401.1	97.6	88
541	421.5	74	433.7	102.9	81	426.8	101.3	76	415.9	98.7	86
570	426.9	69	437.7	102.5	79	440.1	103.1	71	430.6	100.9	83
598	435.9	68	448.6	102.9	77	449.9	103.2	70	435.9	100.0	79
626	446.9	64	454.0	101.6	72	458.7	102.6	70	444.4	99.4	78
640	456.8	61	454.5	99.5	70	462.8	101.3	68	447.6	98.0	76
654	462.4	60	454.0	98.2	67	472.1	102.1	65	450.3	97.4	75
668	450.5	54	455.8	101.2	64	466.7	103.6	62	452.7	100.5	73
682	452.6	54	456.9	101.0	61	473.2	104.6	59	455.1	100.6	70
696	454.3	53	456.5	100.5	58	472.0	103.9	59	457.3	100.6	67
710	451.7	51	451.0	99.9	54	470.8	104.2	55	455.7	100.9	65
724	442.6	48	444.6	100.5	48	476.6	107.7	51	454.2	102.6	61
Mean for Weeks											
1-14	192.0		192.4	100.2		193.2	100.6		185.3	96.5	
15-52	312.3		317.5	101.7		320.7	102.7		307.1	98.3	
53-104	423.6		429.3	101.3		436.2	103.0		419.8	99.1	

^a The number of animals weighed on this day is less than the number of animals surviving.

^b Interim evaluation occurred during week 14.

14-Week Interim Evaluation

There were no changes to the hematology or clinical chemistry variables attributable to CDMA exposure (Table F2).

In 6 W/kg males, there were significantly lower absolute left and right kidney weights (13% to 15%) and absolute (14%) and relative liver weights compared to the sham controls (Table G8). In females, the absolute left and right kidney weights were significantly lower (13% to 46%) in the 6 W/kg group, and the left absolute kidney weights were significantly lower in the 1.5 (10%) and 3 (8%) W/kg groups. The mean relative kidney weights were not similarly decreased, which was likely due to the decrease in mean body weight of the 6 W/kg group compared to the sham controls. These changes did not correlate with any histopathologic findings.

There were no CDMA exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility in males (Table H2). Due to the frequency of poor slide quality, the estrous cycle in females was not evaluated.

In males, there was a significant positive trend ($P=0.006$) in the incidences of cardiomyopathy in the heart, but the severities were similar in all groups (Table 48). Although the incidences of cardiomyopathy of the right ventricle in all exposed groups were increased compared to that in sham controls, they were not statistically significant, and the severities were minimal in all cases. There were marginally increased incidences of cardiomyopathy of the heart in 3 and 6 W/kg females, but they were not statistically significant (Table 48). Cardiomyopathy is a common spontaneous disease in rats that typically has no clinical manifestations. It is characterized by degeneration and necrosis of myofibers with a mild inflammatory response of macrophages and lymphocytes with occasional neutrophils. In later stages of the disease, fibrosis may be prominent.

TABLE 48
**Incidences of Nonneoplastic Lesions of the Heart at the 14-Week Interim Evaluation
in Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 w/kg
Male				
Number Examined Microscopically	10	10	10	10
Cardiomyopathy (excluding right ventricle) ^a	2 (1.5) ^b	0	3 (1.0)	6 (1.0)
Ventricle Right, Cardiomyopathy	1 (1.0)	5 (1.0)	4 (1.0)	4 (1.0)
Cardiomyopathy, All Sites	3 (1.3)	5 (1.0)	6 (1.0)	6 (1.0)
Female				
Number Examined Microscopically	10	10	10	10
Cardiomyopathy (excluding right ventricle)	0	0	2 (1.0)	2 (1.0)
Ventricle Right, Cardiomyopathy	0	0	1 (1.0)	1 (1.0)
Cardiomyopathy, All Sites	0	0	3 (1.0)	3 (1.0)

^a Number of animals with lesion

^b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

Pathology and Statistical Analyses

This section describes the statistically significant or biologically noteworthy changes in the incidences of neoplasms and nonneoplastic lesions of the heart, brain, pituitary gland (pars distalis), adrenal medulla, liver, prostate gland, kidney and other organs, pancreas, mammary gland, adrenal cortex, and thymus. Summaries of the incidences of neoplasms and nonneoplastic lesions, statistical analyses of primary neoplasms that occurred with an incidence of at least 5% in at least one animal group, and historical incidences for the neoplasms mentioned in this section are presented in Appendix C for male rats and Appendix D for female rats.

Heart: Malignant schwannomas were observed in all exposed male groups (Tables 49, C1 and C2). No schwannomas were observed in sham controls. The incidence in the 6 W/kg group was significant, as was the positive trend. The 6 W/kg incidence slightly exceeded the historical control range for all exposure routes (Table C3a). Endocardial Schwann cell hyperplasia, a putative preneoplastic Schwann cell lesion, was seen in three 6 W/kg males, resulting in a significant positive trend ($P=0.044$; Tables 49 and C4).

In females, there were two malignant schwannomas each in the 1.5 and 6 W/kg groups (Tables 49 and D1). Neither of these incidences nor the positive trend were statistically significant. These incidences were also within the historical control range for all routes of exposure. A single occurrence of endocardial Schwann cell hyperplasia was diagnosed in each of the three exposure groups, but there were none in the sham control group (Tables 49 and D4).

The malignant schwannomas and Schwann cell hyperplasias in males and females were morphologically similar to those seen in the rats exposed to GSM-modulated cell phone RFR.

Cardiomyopathy of the right ventricular free wall was seen in all male and female groups, including the sham controls (Tables 49, C4, and D4). In males and females, the incidences in exposed groups were increased compared to the sham controls; the increased incidence in 6 W/kg males was statistically significant. The positive trend ($P<0.001$) was also significant in males. There was also a slight elevation in the severity of this nonneoplastic lesion

TABLE 49
Incidences of Malignant Schwannoma and Neoplasms and Nonneoplastic Lesions of the Heart in Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Heart ^a	90	90	90	90
Cardiomyopathy ^b	79 (1.9) ^c	84 (1.9)	83 (1.8)	85 (1.3)
Ventricle Right, Cardiomyopathy	54 (1.1)	45 (1.2)	62 (1.3)	74* (1.7)
Endocardium, Hyperplasia, Schwann Cell	0	0	0	3 (2.0)
Malignant Schwannoma ^d				
Overall rate ^e	0/90 (0%)	2/90 (2%)	3/90 (3%)	6/90 (7%)
Litters rate ^f	0/35 (0%)	2/35 (6%)	3/35 (9%)	6/35 (17%)
Adjusted rate ^g	0.0%	2.7%	3.8%	8.8%
Terminal rate ^h	0/25 (0%)	2/43 (5%)	2/56 (4%)	3/43 (7%)
First incidence (days)	— ^j	730 (T)	642	488
Rao-Scott adjusted poly-3 test ⁱ	P=0.011	P=0.273	P=0.175	P=0.030
All Organs: Malignant Schwannoma ^k				
Overall rate	3/90 (3%)	4/90 (4%)	4/90 (4%)	8/90 (9%)
Litters rate	3/35 (9%)	4/35 (11%)	4/35 (11%)	7/35 (20%)
Adjusted rate	4.5%	5.4%	5.1%	11.6%
Terminal rate	1/25 (4%)	2/43 (5%)	2/56 (4%)	4/43 (9%)
First incidence (days)	555	573	619	153
Rao-Scott adjusted poly-3 test	P=0.075	P=0.551	P=0.582	P=0.136
Female				
Heart	90	90	90	90
Cardiomyopathy	40 (1.1)	43 (1.1)	33 (1.2)	45 (1.1)
Ventricle Right, Cardiomyopathy	4 (1.0)	7 (1.0)	9 (1.0)	9 (1.0)
Endocardium, Hyperplasia, Schwann Cell	0	1 (3.0)	1	1 (1.0)
Malignant Schwannoma ^l				
Overall rate	0/90 (0%)	2/90 (2%)	0/90 (0%)	2/90 (2%)
Litters rate	0/35 (0%)	2/34 (6%)	0/35 (0%)	2/35 (6%)
Adjusted rate	0.0%	2.7%	0.0%	2.5%
Terminal rate	0/48 (0%)	1/45 (2%)	0/50 (0%)	2/61 (3%)
First incidence (days)	—	649	—	737 (T)
Rao-Scott adjusted poly-3 test	P=0.343	P=0.317	— ^m	P=0.342
All Organs: Malignant Schwannoma ⁿ				
Overall rate	4/90 (4%)	2/90 (2%)	2/90 (2%)	4/90 (4%)
Litters rate	3/35 (9%)	2/34 (6%)	2/35 (6%)	4/35 (11%)
Adjusted rate	5.7%	2.7%	2.8%	5.0%
Terminal rate	2/48 (4%)	1/45 (2%)	2/50 (4%)	4/61 (7%)
First incidence (days)	489	649	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.561	P=0.346N	P=0.354N	P=0.577N

TABLE 49
Incidences of Malignant Schwannoma and Neoplasms and Nonneoplastic Lesions of the Heart in Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

- * Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test
- (T) Terminal euthanasia
- a Number of animals with tissue examined microscopically
- b Number of animals with lesion
- c Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked
- d Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 2/240 (1.0% \pm 1.2%), range 0%-2%
- e Number of animals with neoplasm per number of animals necropsied
- f Number of litters with animals with neoplasm per number of litters necropsied
- g Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
- h Observed incidence at terminal euthanasia
- i Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A lower incidence in an exposure group is indicated by N.
- j Not applicable; no neoplasms in animal group
- k Historical incidence for all routes of 2-year studies: 6/240 (2.3% \pm 1.8%), range 0%-4%
- l Historical incidence for all routes of 2-year studies: 0/239
- m Value of statistic cannot be computed
- n Historical incidence for all routes of 2-year studies: 8/240 (3.1% \pm 2.1%), range 0%-4%

in 6 W/kg males, but there was no similar elevation in severity in females. Cardiomyopathy of the right ventricle was initially diagnosed separately and an increased incidence was found in the 6 W/kg males compared to the sham controls. Cardiomyopathy in the CDMA-exposed rats was morphologically identical to that described previously for the GSM-exposed rats (page 100).

Brain: In males, there were three malignant gliomas in the 6 W/kg group, resulting in a significant positive trend (Tables 50 and C1). In females, malignant glioma occurred in three 1.5 W/kg animals; no malignant gliomas were observed in the other exposed groups or in the sham controls (Tables 50 and D1). There was no significant positive trend for this neoplasm in females, and the increased incidence was not significant. Glial cell hyperplasia occurred in most groups of males and females, but none of the incidences were significantly increased and there were no positive trends (Tables 50, C4, and D4). The proliferative glial cell lesions were morphologically similar to those seen in the rats exposed to GSM-modulated RFR.

TABLE 50
**Incidences of Neoplasms and Nonneoplastic Lesions of the Brain in Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia ^a	0	2 (1.5) ^b	0	2 (2.5)
Glioma Malignant ^c				
Overall rate ^d	0/90 (0%)	0/90 (0%)	0/90 (0%)	3/90 (3%)
Litters rate ^e	0/35 (0%)	0/35 (0%)	0/35 (0%)	3/35 (9%)
Adjusted rate ^f	0.0%	0.0%	0.0%	4.5%
Terminal rate ^g	0/25 (0%)	0/43 (0%)	0/56 (0%)	3/43 (7%)
First incidence (days)	— ⁱ	—	—	730 (T)
Rao-Scott adjusted poly-3 test ^h	P=0.044	— ^j	—	P=0.221
Female				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia	0	0	1 (2.0)	1 (2.0)
Glioma Malignant ^k				
Overall rate	0/90 (0%)	3/90 (3%)	0/90 (0%)	0/90 (0%)
Litters rate	0/35 (0%)	3/34 (9%)	0/35 (0%)	0/35 (0%)
Adjusted rate	0.0%	4.1%	0.0%	0.0%
Terminal rate	0/48 (0%)	2/45 (4%)	0/50 (0%)	0/61 (0%)
First incidence (days)	—	550	—	—
Rao-Scott adjusted poly-3 test	P=0.384N	P=0.236	—	—

(T) Terminal euthanasia

^a Number of animals with lesion

^b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

^c Historical incidence for all routes of 2-year studies (mean ± standard deviation): 2/190 (1.3% ± 2.3%), range 0%-4%

^d Number of animals with neoplasm per number of animals with brain examined microscopically

^e Number of litters with animals with neoplasm per number of litters with brain examined microscopically

^f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

^g Observed incidence at terminal euthanasia

^h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test performs the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend is indicated by N.

ⁱ Not applicable; no neoplasms in animal group

^j Value of statistic cannot be computed

^k Historical incidence for all routes of 2-year studies: 1/190 (0.7% ± 1.2%), range 0%-2%

[Table 51 omitted]

Pituitary Gland (Pars Distalis): In males, there were increased incidences of adenoma in the 1.5 and 3 W/kg groups, but only the 3 W/kg incidence was significant (Tables 52, C1, and C2). No carcinomas occurred in males and there was no significant increase in the incidence or severity of hyperplasia (Tables 52, C1, and C4). In females exposed to 3 W/kg, there were significantly decreased incidences of adenoma and adenoma or carcinoma

(combined) (Tables 52, D1, and D2). There were no increased incidences of carcinoma or hyperplasia in females (Tables 52, D1, and D4). In the males, there was a significantly increased incidence of cyst in the 1.5 W/kg group (Tables 52 and C4). In the females, there was a significantly decreased incidence of cyst in the 6 W/kg group and the decreasing trend was also significant ($P=0.032N$; Table 52 and C4). Pituitary gland cysts are considered developmental anomalies and are fairly common, so the toxicologic significance of these changes is unclear.

TABLE 52
Incidences of Neoplasms and Nonneoplastic Lesions of the Pituitary Gland (Pars Distalis) in Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	89	90	90	90
Cyst ^a	5	15*	7	6
Hyperplasia	32 (2.4) ^b	32 (2.4)	34 (2.5)	27 (2.2)
Adenoma, Multiple	0	0	1	2
Adenoma (includes multiple) ^c				
Overall rate ^d	17/89 (19%)	25/90 (28%)	34/90 (38%)	13/90 (14%)
Litters rate ^e	13/35 (37%)	18/35 (51%)	24/35 (69%)	12/35 (34%)
Adjusted rate ^f	24.9%	32.7%	41.8%	19.0%
Terminal rate ^g	5/25 (20%)	16/43 (37%)	22/56 (39%)	6/43 (14%)
First incidence (days)	527	605	471	567
Rao-Scott adjusted poly-3 test ^h	P=0.226N	P=0.208	P=0.030	P=0.273N
Female				
Number Examined Microscopically	90	89	89	90
Cyst	7	5	3	1*
Hyperplasia	20 (2.5)	22 (2.2)	26 (1.9)	22 (2.8)
Adenoma, Multiple	1	1	0	0
Adenoma (includes multiple) ⁱ				
Overall rate	43/90 (48%)	41/89 (46%)	30/89 (34%)	40/90 (44%)
Litters rate	28/35 (80%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.1%	52.9%	39.5%	49.0%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)
First incidence (days)	464	578	493	626
Rao-Scott adjusted poly-3 test	P=0.156N	P=0.360N	P=0.026N	P=0.204N
Carcinoma ^j	1	1	1	0
Adenoma or Carcinoma ^k				
Overall rate	44/90 (49%)	42/89 (47%)	31/89 (35%)	40/90 (44%)
Litters rate	29/35 (83%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.9%	54.1%	40.7%	49.0%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)
First incidence (days)	464	578	493	626
Rao-Scott adjusted poly-3 test	P=0.131N	P=0.381N	P=0.030N	P=0.180N

▲ Significant trend ($P \leq 0.05$) by the Rao-Scott adjusted poly-3 test

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

^a Number of animals with lesion

^b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

^c Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 47/239 (19.8% \pm 7.5%), range 10%-28%

^d Number of animals with neoplasm per number of animals with pituitary gland examined microscopically

^e Number of litters with animals with neoplasm per number of litters with pituitary gland examined microscopically

^f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

^g Observed incidence at terminal euthanasia

^h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott adjusted poly-3 test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.

ⁱ Historical incidence for all routes of 2-year studies: 98/240 (39.4% \pm 5.6%), range 36%-48%

^j Historical incidence for all routes of 2-year studies: 1/240 (0.3% \pm 0.6%), range 0%-1%

^k Historical incidence for all routes of 2-year studies: 99/240 (39.7% \pm 6.2%), range 36%-49%

Adrenal Medulla: In females, there were increased incidences of benign pheochromocytoma and benign, malignant, or complex pheochromocytoma (combined) in all exposed groups, but only the incidence of benign, malignant, or complex pheochromocytoma (combined) at 1.5 W/kg was significant (Tables 53, D1, and D2). There were higher incidences of malignant pheochromocytoma in the 1.5 and 3 W/kg groups compared to controls. There were higher incidences of hyperplasia in all exposed female groups, but the incidences were not significant (Tables 53 and D4).

Liver: In males, there were higher incidences of hepatocellular adenoma in the 1.5 and 3 W/kg groups compared to controls (Tables 54 and C1). There were also hepatocellular carcinomas, one each in the 3 and 6 W/kg groups. The incidences of hepatocellular adenoma or carcinoma (combined) were increased in all exposed groups, but none of the incidences were significant. In 6 W/kg females, there was a decreased incidence of hepatocellular adenoma with a significant negative trend (Tables 54, D1, and D2). The NTP historical control incidence of hepatocellular adenoma in male Hsd:Sprague Dawley SD rats by all routes of exposure is 1/240, and no hepatocellular carcinomas have been seen in this strain of rat in the NTP historical control data. In females, the historical control incidence of hepatocellular adenoma in NTP studies by all routes of exposure is 11/240, and as with males, no hepatocellular carcinomas have been seen. In males, the incidences of mixed cell focus were increased in all exposed groups, but only the incidence in the 1.5 W/kg group was significant (Tables 54 and C4).

Prostate Gland: The incidences of epithelial hyperplasia were increased in all exposed groups compared to the sham controls, and the severity increased slightly. There was a significant positive trend ($P=0.025$), but only the incidence in the 6 W/kg group was significant (sham control, 5/90; 1.5 W/kg, 11/90; 3 W/kg, 9/90; 6 W/kg, 15/85; Table C4). Epithelial hyperplasia of the prostatic epithelium was characterized as infoldings or papillary projections of epithelial cells into the lumen of a prostatic gland. The cells did not fill or distend the gland, and there was no atypia and no mitotic figures.

TABLE 53
**Incidences of Neoplasms and Nonneoplastic Lesions of the Adrenal Medulla in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Number Examined Microscopically	86	89	87	88
Hyperplasia ^a	13 (1.5) ^b	20 (1.7)	20 (1.3)	18 (1.9)
Benign Pheochromocytoma ^c				
Overall rate ^d	1/86 (1%)	7/89 (8%)	3/87 (3%)	4/88 (5%)
Litters rate ^e	1/35 (3%)	7/34 (21%)	3/35 (9%)	4/35 (11%)
Adjusted rate ^f	1.5%	9.6%	4.4%	5.2%
Terminal rate ^g	1/45 (2%)	5/44 (11%)	3/48 (6%)	4/60 (7%)
First incidence (days)	737 (T)	464	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test ^h	P=0.466	P=0.059	P=0.322	P=0.248
Malignant Pheochromocytoma ⁱ	0	2	1	0
Complex Pheochromocytoma	0	0	1	0
Benign, Malignant, or Complex Pheochromocytoma ^j				
Overall rate	1/86 (1%)	9/89 (10%)	5/87 (6%)	4/88 (5%)
Litters rate	1/35 (3%)	9/34 (26%)	5/35 (14%)	4/35 (11%)
Adjusted rate	1.5%	12.3%	7.2%	5.2%
Terminal rate	1/45 (2%)	7/44 (16%)	4/48 (8%)	4/60 (7%)
First incidence (days)	737 (T)	464	652	737 (T)
Rao-Scott adjusted poly-3 test	P=0.546	P=0.022	P=0.126	P=0.242

(T) Terminal euthanasia

^a Number of animals with lesion

^b Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

^c Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 4/235 (1.8% \pm 2.9%), range 0%-6%

^d Number of animals with neoplasm per number of animals with adrenal medulla examined microscopically

^e Number of litters with animals with neoplasm per number of litters with adrenal medulla examined microscopically

^f Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

^g Observed incidence at terminal euthanasia

^h Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott adjusted poly-3 test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation.

ⁱ Historical incidence for all routes of 2-year studies: 2/235 (1.0% \pm 2.0%), range 0%-4%

^j Historical incidence for all routes of 2-year studies: 6/235 (2.8% \pm 4.8%), range 0%-10%

TABLE 54
**Incidences of Neoplasms and Nonneoplastic Lesions of the Liver in Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	89	88
Mixed Cell Focus ^a	32	51*	47	37
Hepatocellular Adenoma ^b				
Overall rate ^c	0/90 (0%)	2/90 (2%)	4/89 (4%)	0/88 (0%)
Litters rate ^d	0/35 (0%)	2/35 (6%)	4/35 (11%)	0/35 (0%)
Adjusted rate ^e	0.0%	2.7%	5.1%	0.0%
Terminal rate ^f	0/25 (0%)	2/43 (5%)	4/56 (7%)	0/43 (0%)
First incidence (days)	— ^h	730 (T)	730 (T)	— ⁱ
Rao-Scott adjusted poly-3 test ^g	P=0.556N	P=0.310	P=0.132	— ⁱ
Hepatocellular Carcinoma ^j	0	0	1	1
Hepatocellular Adenoma or Carcinoma ^b				
Overall rate	0/90 (0%)	2/90 (2%)	4/89 (4%)	1/88 (1%)
Litters rate	0/35 (0%)	2/35 (6%)	4/35 (11%)	1/35 (3%)
Adjusted rate	0.0%	2.7%	5.1%	1.5%
Terminal rate	0/25 (0%)	2/43 (5%)	4/56 (7%)	0/43 (0%)
First incidence (days)	— ^h	730 (T)	730 (T)	594
Rao-Scott adjusted poly-3 test	P=0.416	P=0.281	P=0.113	P=0.475
Female				
Hepatocellular Adenoma ^k				
Overall rate	7/90 (8%)	2/90 (2%)	2/90 (2%)	1/90 (1%)
Litters rate	6/35 (17%)	2/34 (6%)	2/35 (6%)	1/35 (3%)
Adjusted rate	10.1%	2.7%	2.8%	1.3%
Terminal rate	6/48 (13%)	1/45 (2%)	2/50 (4%)	1/61 (2%)
First incidence (days)	707	493	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.042N	P=0.118N	P=0.125N	P=0.052N
Hepatocellular Carcinoma ^j	0	0	0	1

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

(T) Terminal euthanasia

^a Number of animals with lesion

^b Historical incidence for all routes of 2-year studies (mean \pm standard deviation): 1/240 (0.5% \pm 1.0%), range 0%-2%

^c Number of animals with neoplasm per number of animals with liver examined microscopically

^d Number of litters with animals with neoplasm per number of litters with liver examined microscopically

^e Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

^f Observed incidence at terminal euthanasia

^g Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott adjusted poly-3 test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) with an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.

^h Not applicable; no neoplasms in animal group

ⁱ Value of statistic cannot be computed

^j Historical incidence for all routes of 2-year studies: 0/240

^k Historical incidence for all routes of 2-year studies: 11/240 (3.9% \pm 3.2%), range 0%-8%

Kidney and Other Organs: The severity of chronic progressive nephropathy was lower in all exposed groups compared to the sham controls (Table 55). There were decreased incidences in a number of lesions in other organs in exposed groups, some statistically significant, that were thought to be secondary to the chronic progressive nephropathy, either directly or indirectly (Tables 55 and C4). These lesions included hyperplasia of the parathyroid gland; mineral in the blood vessels in the colon, liver, mesentery, pancreas, salivary glands, brain, heart, kidney, skeletal muscle, glandular stomach, spleen, and aorta; mineral in the heart, salivary gland, and stomach; fibrous osteodystrophy of bone; polyarteritis nodosa (chronic active inflammation of the blood vessels) of the epididymis, testis, cecum, liver, pancreas, glandular stomach, and thymus; germ cell degeneration of the testis; edema, erosion, epithelial regeneration, acute inflammation, and ulcer of the cecum; epithelial regeneration of the colon; epithelial regeneration and acute inflammation of the rectum; red pulp atrophy and white pulp atrophy of the spleen; and exfoliated germ cell and hypospermia of the epididymis.

Other Organs: In the pancreas of males, there was a significantly decreased incidence of adenoma in the 6 W/kg group (Tables C1 and C2). There was a significant negative trend ($P=0.034$) in the incidences of this neoplasm although the incidences in 1.5 and 3 W/kg male groups were slightly increased compared to those in the sham controls. Only one male in the 3 W/kg group had a carcinoma but no adenoma, so there was also a significant negative trend ($P=0.037$) in the incidences of adenoma or carcinoma (combined) as well as a significantly decreased incidence in the 6 W/kg males.

In females, there were significant negative trends in the incidences of adenoma (sham control, 8/90; 1.5 W/kg, 4/90; 3 W/kg, 1/90; 6 W/kg, 2/90; $P=0.035$) and adenoma or carcinoma (combined) (16/90, 12/90, 7/90, 6/90; $P=0.009$) in the mammary gland and the incidences in the 3 and 6 W/kg groups were significantly decreased (Tables D1 and D2). There was also a significant negative trend in the incidences of mammary gland carcinoma ($P=0.042$).

In males, the incidences of adrenal cortex hypertrophy were increased in all exposed groups compared to the sham controls, and the incidence in the 3 W/kg group was significant (35/90, 42/90, 55/90, 44/89; $P=0.013$) (Table C4). There was also a significant increasing trend for this nonneoplastic lesion ($P=0.028$). In 6 W/kg males, there were significantly increased incidences of lung congestion (13/90, 13/90, 11/90, 33/90) and thymic hemorrhage

TABLE 55
Incidences of Nonneoplastic Lesions Associated with the Decreased Severity of Chronic Progressive Nephropathy of the Kidney in Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Kidney ^a	90	90	90	87
Nephropathy, Chronic Progressive ^b	88 (3.7) ^c	90 (3.3)	90 (3.0)	86 (2.3)
Aorta	90	90	90	90
Mineral	30 (2.1)	8** (2.8)	6** (2.2)	2** (1.5)
Bone	90	90	90	90
Fibrous Osteodystrophy	46 (1.4)	20** (1.7)	15** (1.6)	5** (1.6)
Brain	90	90	90	90
Mineral	5 (1.0)	3 (1.0)	4 (1.0)	4 (1.0)
Epididymis	90	90	90	90
Artery, Inflammation, Chronic Active	2 (2.5)	3 (3.0)	3 (2.3)	3 (2.7)
Exfoliated Germ Cell	51 (1.9)	33** (1.7)	33** (1.7)	17** (1.5)
Hypospermia	28 (3.4)	24 (3.1)	13** (3.7)	13* (3.0)
Heart	90	90	90	90
Artery, Mineral	20 (2.5)	7* (2.1)	2** (2.0)	1** (2.0)
Intestine Large, Cecum	75	76	74	68
Artery, Inflammation, Chronic Active	20 (2.1)	8* (1.9)	7* (1.9)	2** (2.5)
Edema	11 (2.0)	0**	0**	0**
Epithelium, Regeneration	14 (2.4)	1** (2.0)	0**	1** (2.0)
Erosion	10 (2.5)	1* (3.0)	1* (4.0)	1* (2.0)
Inflammation, Acute	10 (2.8)	1** (2.0)	0**	1* (2.0)
Ulcer	6 (2.3)	0*	0*	0
Intestine Large, Colon	81	83	82	76
Artery, Mineral	2 (2.0)	0	0	0
Epithelium, Regeneration	5 (2.6)	0	0	0
Intestine Large, Rectum	83	81	80	76
Epithelium, Regeneration	3 (2.3)	0	0	0
Inflammation, Acute	2 (2.5)	0	0	0
Kidney	90	90	90	87
Artery, Mineral	2 (2.0)	0	0	0
Liver	90	90	89	88
Artery, Inflammation, Chronic Active	2 (3.5)	1 (2.0)	0	0
Artery, Mineral	1 (1.0)	1 (1.0)	0	0
Mesentery	39	19	17	6
Artery, Mineral	21 (2.1)	5* (2.0)	2** (2.5)	0*
Pancreas	90	88	87	78
Artery, Inflammation, Chronic Active	48 (2.3)	28** (2.0)	23** (2.0)	5** (2.2)
Artery, Mineral	11 (1.8)	2* (2.5)	0**	0**
Parathyroid Gland	83	83	83	82
Hyperplasia	51 (2.5)	35* (2.5)	32** (2.0)	17** (1.8)
Salivary Glands	90	90	90	86
Artery, Mineral	2 (2.5)	1 (2.0)	1 (2.0)	0

TABLE 55

Incidences of Nonneoplastic Lesions Associated with the Decreased Severity of Chronic Progressive Nephropathy of the Kidney in Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Skeletal Muscle Mineral	90 2 (1.0)	90 0	90 1 (1.0)	90 0
Spleen	90	90	90	85
Arteriole, Mineral	1 (2.0)	0	0	0
Red Pulp, Atrophy	26 (2.2)	14* (1.9)	12** (2.1)	13* (2.0)
White Pulp, Atrophy	30 (2.1)	11** (2.3)	10** (2.4)	24 (1.9)
Stomach, Glandular	86	86	85	78
Artery, Inflammation, Chronic Active	3 (2.3)	0	0	0
Mineral	31 (2.5)	9** (3.1)	6** (2.7)	1** (2.0)
Testis	90	89	90	90
Artery, Inflammation, Chronic Active	52 (2.9)	37** (2.8)	30** (2.5)	12** (3.1)
Germ Cell, Degeneration	51 (2.3)	37* (2.6)	31** (2.2)	24** (2.1)
Thymus	88	85	87	82
Artery, Inflammation, Chronic Active	6 (2.7)	3 (2.3)	2 (1.5)	1 (2.0)

* Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted poly-3 test

** $P \leq 0.01$

a Number of animals with tissue examined microscopically

b Number of animals with lesion

c Average severity grade of lesions in affected animals: 1=minimal, 2=mild, 3=moderate, 4=marked

(2/88, 2/85, 2/87, 20/82) (Table C4). The positive trends were also significant ($P < 0.001$). There was also a significant positive trend in the incidences of thymic cysts in male rats ($P = 0.042$).

GENETIC TOXICOLOGY

Twenty tissue samples obtained from animals at the 14-week interim evaluation in the 2-year study were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, five tissues). Results are based on the standard 100-cell scoring approach in use at the time these data were collected; data obtained using a 150-cell scoring approach, recommended in a recently adopted international guideline for the *in vivo* comet assay, are noted for the few instances where results differed between the two methods. The complete 100-cell and 150-cell data are presented in Appendix E data tables. A significant increase in DNA damage (% comet tail DNA) was observed in hippocampus cells of male rats exposed to the CDMA modulation (Table E1). Although the levels of DNA damage in hippocampus cells were also increased in an exposure-related fashion using the 150-cell scoring approach, the increases were not statistically significant (Table E3). An exposure-related increase (trend test

P=0.004) in DNA damage was seen in the cells of the frontal cortex of male rats exposed to the CDMA modulation (Table E1); however, no individual exposure groups were significantly elevated over the sham control group and the result was therefore judged to be equivocal. For male rat blood leukocytes exposed to either the CDMA or GSM modulation (Tables E1 and E2), results from scoring 100 cells were negative; however, these leukocyte samples showed equivocal responses with the 150-cell method due to a significant trend test (P=0.012) or pairwise test (P=0.021) for CDMA- and GSM-exposed rats, respectively (Tables E3 and E4). No statistically significant increases in the percent comet tail DNA were observed in any of the female rat samples scored with the 100-cell approach (Tables E5 and E6). The 150-cell scoring approach yielded a significant trend test (P=0.013) in peripheral blood leukocytes of female rats exposed to the CDMA modulation, but these results were driven by data from a single animal (Table E7).

In contrast to what was seen in the mice (NTP, 2018b), a high degree of interanimal variability was observed in the percent comet tail DNA values in rats within a treatment group, and this level of variability reduced the statistical power to detect increases in DNA migration, although the magnitudes of the increases observed in some rats suggested these were treatment-related effects. To rule out any influence from technical artifacts or protocol features, percent comet tail DNA values and percent hedgehogs were correlated to the position of slides in the electrophoresis chambers, the interval from exposure cessation to tissue collection, and the date of slide preparation; no patterns emerged for any of these variables and the level of DNA damage observed.

Similar to what was seen in the mice, no significant increases in micronucleated red blood cells or in the percentage of reticulocytes were observed in rats of either sex exposed to either modulation of cell phone RFR (Table E9).

DISCUSSION AND CONCLUSIONS

While epidemiology studies have not definitively established an association between cell phone radio frequency radiation (RFR) exposure and any specific health problems in humans, the results from some studies are suggestive of potential effects (Lönn *et al.*, 2004b; Hardell *et al.*, 2006, 2007b; Hardell and Carlberg, 2009; INTERPHONE 2010, 2011; Benson *et al.*, 2013). Based on available studies, a working group of the International Agency for Research on Cancer (IARC, 2011) classified radiofrequency electromagnetic fields as possibly carcinogenic to humans. Of particular concern were possible associations (limited evidence) with brain glioma and acoustic neuroma (vestibular schwannoma) in the region of the head that is most exposed to RFR when a wireless phone is used at the ear. However, interpretation of these results is complicated by potential misclassification of exposures and by selection and recall biases. It is also possible that exposures to RFR in the general population, such as those from cellular communication, have not occurred for a long enough period of time to ascertain an effect due to the apparent long latency period for some types of adult-onset cancers in humans. Studies in laboratory animals have also been complicated by design and logistical limitations that researchers have faced in conducting toxicity and carcinogenicity studies.

To improve on the existing methods of exposing laboratory animals to cell phone RFR, NTP worked in collaboration with technical experts from the National Institute of Standards and Technology (NIST, Boulder, CO) and the Foundation for Research on Information Technologies in Society (IT'IS Foundation) (Zurich, Switzerland) to design, construct, and validate a novel system of delivering cell phone RFR exposure. Together with the NIST and IT'IS Foundation, the NTP constructed an exposure system designed to expose unrestrained, individually housed animals to a statistically uniform field of cell phone RFR at frequencies (900 MHz in rats and 1900 MHz in mice) and with modulations (GSM and CDMA) used in cellular communication devices. The exposure facility was installed at IIT Research Institute (Chicago, IL), where all animal studies were conducted following system testing and RFR exposure validation. The design and performance characteristics for the exposure system are reported fully

in Capstick *et al.* (2017), and detailed tissue specific RFR exposure modeling at these frequencies is presented in Gong *et al.* (2017).

Studies assessing the effects of GSM- and CDMA-modulated RFR on body temperatures of rats, including exposure in dams, are reported in Wyde *et al.* (2018). Exposures beginning *in utero* were selected for use in these studies, due to the exposure and use pattern of cell phones by pregnant women and women of childbearing age. NTP conducted 28-day and 2-year studies to characterize the potential toxicity and carcinogenicity of whole-body exposure to cell phone RFR in mice beginning at 5 weeks of age and in rats beginning *in utero* with exposures continuing throughout gestation and lactation, and for an additional 28 days or 2 years.

Hsd:Sprague Dawley SD rats received whole-body exposure to GSM- or CDMA-modulated cell phone RFR (900 MHz) *in utero* for 9 hours and 10 minutes a day during an 18-hour exposure period starting during gestation and continuing throughout lactation and for an additional 28 days, 14 weeks, or 2 years. Exposures for the GSM- and CDMA-modulated cell phone RFR studies were conducted in parallel with a common nonexposed (sham) control group that was housed in an identical chamber that differed from the RFR exposure chambers only in the absence of cell phone RFR emission during the exposure periods.

Nonionizing RFR transfers energy to biological tissues through a process that results in some degree of heating of the exposed tissue. While prolonged exposures to high levels of RFR are known to cause damage to tissues and biological systems through excessive heating, the safety of chronic exposures to power levels permitted for use in mobile communications has not been established. Because exposure to excessive levels of cell phone RFR results in overt thermal changes and is not reflective of the human exposure scenario, a series of short-term pilot studies was conducted to characterize the impact of body size and pregnancy status on body temperature following exposure to cell phone RFR and to identify adequately challenging exposure levels (specific absorption rates; SARs) of cell phone RFR below those that raised body temperature by more than 1° C (Wyde *et al.*, 2018). In general, these studies demonstrated a significant SAR-dependent increase in body temperature that was greater in larger, older rats than in smaller, young rats. Exposures to 10 W/kg or greater cell phone RFR (both modulations) induced excessively high body temperatures compared to sham controls, leading to mortality in many cases, and increased

resorptions in pregnant rats at 12 W/kg. These data suggest that exposure at these levels resulted in the potential breakdown of the thermoregulatory capacity in rats. In these studies, body temperatures were higher with increasing SAR compared to sham controls at exposures of 6 W/kg or greater for both modulations. Male rats were more sensitive than females to cell phone RFR-induced rises in body temperature compared to sham controls. In the 28-day studies, body temperature was higher in the F₀ dams compared to sham controls during perinatal exposure at 6 and 9 W/kg. These findings were consistent with the effects observed in the thermal pilot studies in pregnant dams at similar SARs (Wyde *et al.*, 2018). No increases in body temperature were observed in the F₁ offspring at 6 and 9 W/kg.

Based on temperature changes (> 1° C) in adult rats at 8 W/kg or greater in the thermal pilot studies (Wyde *et al.*, 2018), increased body temperature in F₀ dams at 9 W/kg, and decreased F₁ pup survival at 9 W/kg in the 28-day studies, the highest exposure concentration selected for the 2-year studies was 6 W/kg. This exposure level was selected to provide an exposure considered adequate to challenge the animals without causing disruption of the thermoregulatory process. The lowest exposure concentration selected for the 2-year studies in rats, 1.5 W/kg, is close to the current Federal Communications Commission guidelines for localized exposure of 1.6 W/kg for cell phone devices in the United States. The localized and whole-body exposure limits (0.08 W/kg) are based on protection from acute injury induced by thermal effects of cell phone RFR (ICNIRP, 1998). While core body temperature is a good general surrogate for the heating effects of cell phone RFR, it must be noted that core body temperature does not address the potential for localized heating in some tissues at exposures that do not induce higher core temperature compared to control animals. This concept is further illustrated in the relative tissue SAR modeling studies of Gong *et al.* (2017). Due to logistical constraints, body temperature measurements were not collected in the 2-year studies.

Significant effects occurred during the perinatal exposure in dams (F₀) and their offspring (F₁) that were associated with exposure to cell phone RFR in both the 28-day and 2-year studies, regardless of modulation. In the F₀ cell phone RFR-exposed dams, lower body weights and body weight gains compared to sham controls were observed during the gestation and lactation periods. In both the 28-day and 2-year studies, significantly lower body weight gains late in gestation (GD 15 to 21) were observed at 9 W/kg and 6 W/kg, respectively, which may be related to the

lower pup weights on PND 1. During the lactation period, there were decreased maternal body weight gains, body weights in the 6 and 9 W/kg groups were lower in both the 28-day and 2-year studies compared to sham controls, and body weights were generally lower with increasing SAR.

In the F₁ offspring, body weights were significantly lower than sham controls on PND 1 and throughout the lactation period. Males appeared to be slightly more susceptible to the effects of cell phone RFR on body weight, because the magnitude of the effect was marginally higher in males than in females. While there was no effect on live litter size throughout lactation, there was a lower survival ratio at 9 W/kg compared to sham controls in the GSM study, early in the lactation period (PND 1 to 4), prior to culling litters to a standard size. Additionally, there was greater pup mortality during the lactation period (PND 4 to 21) in the 2-year CDMA study at 6 W/kg than in sham controls, which was not observed in the 28-day CDMA study at the same exposure level.

The results indicate the possibility that RFR at these exposures could impact normal development. However, the occurrence of early pup deaths and slowed pup weight gain with cell phone RFR exposure compared to sham controls could also be secondary to effects on the dams. For example, changes in maternal behavior or capacity to properly nourish their pups may have contributed to these effects as the magnitude of the lower pup body weights appeared to increase during early lactation and then decrease as the pups aged and required less maternal care. Unfortunately, behavioral abnormalities could not be directly observed in the current study because the design of the chambers prohibited observation during the 18-hour daily cell phone RFR exposure periods. Further research would be required to elucidate the mechanism by which cell phone RFR induces these effects in pups. The lack of further decreases in body weight over time suggests that the cell phone RFR-mediated effects of exposure on body weight in the F₁ offspring may be specific to the perinatal period.

At the end of the 2-year studies, survival was significantly greater in all groups of exposed male rats in the GSM study (50% to 68%) and at 3 and 6 W/kg in the CDMA study (48% to 62%) compared to the male sham control group (28%). In the male sham control group, survival declined more rapidly after week 75 than in all exposed groups reflecting a higher rate of moribund sacrifices. The resulting 28% survival rate in sham control males was lower than the rate observed in the historical controls (40% to 60%); however, survival of the GSM- and

CDMA-exposed groups (48% to 68%) was similar to the historical control range. When including NTP Harlan Sprague Dawley (HSD) rat studies on other test articles studied under various conditions that are currently under review and have not yet been reported, survival in the male control groups is highly variable, ranging from 24% to 72% (NTP, 2018a). The differences in survival in male rats may reflect the inherent variability in survival observed among the control groups in the HSD male rat. However, in female rats, survival in the CDMA 6 W/kg group (68%) was greater than in the sham controls (54%) and exceeded the survival rate in historical controls (42% to 60%).

The higher mortality in the sham control group of males compared to the exposed males was largely attributed to a high severity of chronic progressive nephropathy in the kidneys that resulted in moribund removal of a large number of male rats from the study. Chronic progressive nephropathy is a common cause of death in Sprague Dawley rats as well as other rat strains, and typically occurs to some degree in many aged rats, although with greater severity in males. Of note, the severity of chronic progressive nephropathy in males decreased with increasing SAR exposure to either modulation.

Advanced chronic progressive nephropathy can result in markedly impaired renal function and a host of secondary lesions in other organs. In males, a broad spectrum of lesions, considered to be secondary to chronic progressive nephropathy, was seen in all groups (sham control and exposed) with the highest incidences in the sham control group and decreasing with increasing SAR exposure, including parathyroid gland hyperplasia, mineralization in multiple tissues, and fibrous osteodystrophy of bone. Chronic progressive nephropathy can also cause an increased incidence of polyarteritis nodosa, a spontaneous vascular disease that most commonly affects medium-sized arteries in the mesentery, pancreas, kidney, pancreaticoduodenal artery, and testis, although arteries in most other organs can also be affected (Barthold *et al.*, 2016). In the current study, there were a number of organs with arterial inflammation consistent with polyarteritis nodosa. The incidence of these vascular lesions was greater in the sham control group than in exposed groups, correlating to the high severity of chronic progressive nephropathy in the sham control group. The apparent SAR-dependent effects in males and the slight effects (decreased incidence of nephropathy) observed in females suggest that the decrease in chronic progressive nephropathy may have been related to cell phone RFR exposure. Whether this reflects a direct effect of cell phone RFR on possible suppression

of inflammatory processes through stimulation of stress response pathways (Hauet-Broere *et al.*, 2006), or is secondary to a possible reduction in feed intake, leading to a reduction in the severity of nephropathy (Rao, 2002), remains to be established.

There were higher incidences of neoplasms and nonneoplastic lesions of the heart in male rats exposed to cell phone RFR than in sham controls. For both modulations, exposure to cell phone RFR resulted in a statistically significant positive trend in the incidences of cardiac schwannoma. The incidence at 6 W/kg for CDMA was significantly higher compared to sham controls. Cardiac Schwann cell hyperplasia also occurred at this SAR of CDMA-modulated cell phone RFR. These lesions are relevant to the evaluation of neoplasms because Schwann cell hyperplasia in the heart may progress to cardiac schwannomas (MacKenzie and Alison, 1990; Berridge *et al.*, 2016). No Schwann cell hyperplasia or schwannomas of the heart were observed in the sham control group of male rats. The 5.5% and 6.6% incidences of schwannoma observed in the 6 W/kg GSM- and CDMA-modulated cell phone RFR groups, respectively, exceeded the mean historical incidence (0.8%), and exceed the highest rate observed in a single historical control group (2%) of completed peer reviewed studies. [Note: the historical control rate reported in Wyde *et al.* (2016) was 9/699 (range 0-6%) because control groups from NTP studies using the Hsd:Sprague Dawley SD rat model were included that had not yet been peer reviewed.] Because the 28% survival rate of the sham control group of male rats in the current studies was relatively low compared to other recent NTP studies in Hsd:Sprague Dawley SD rats (NTP, 2018a) the absence of these lesions in sham control males in the current studies could conceivably be related to their shorter longevity. While most (13/19) of these lesions were observed at terminal necropsy, some (6/19) were observed at 70 to 94 weeks, a time when survival in the sham control male rats was greater than 65%. Based on the rarity of this lesion, the presence of nonneoplastic hyperplasia, and the higher incidence in the highest exposure group, the higher incidences of schwannoma in the heart was considered a result of cell phone RFR exposure and was the basis for the conclusion of *some evidence for carcinogenic activity* for both GSM- and CDMA-modulated cell phone RFR in male rats.

Schwannomas are tumors of Schwann cell origin occurring in the peripheral nervous system. Schwann cells are similar to glial cells in the brain in that they are specialized supportive cells whose functions include maintaining homeostasis, forming myelin, and providing support and protection for neurons of the peripheral nervous system

(PNS). In the PNS, Schwann cells produce myelin and are analogous to oligodendrocytes of the central nervous system. Schwannomas occurred in these studies in organs other than the heart, and the incidences of these extra-cardiac schwannomas were not higher with cell phone RFR exposures in the current studies.

In the heart, cell phone RFR exposure also induced higher incidences and severity of cardiomyopathy in the right ventricular free wall of male rats exposed to either GSM- or CDMA-modulated cell phone RFR and in female rats exposed to GSM-modulated cell phone RFR. The effect of cell phone RFR on cardiomyopathy appears to be specific to the right ventricle in these groups because incidences of cardiomyopathy in the whole heart were unchanged in males and CDMA females and lower in the GSM females compared to sham controls. Higher incidences of cardiomyopathy in the right ventricle of male rats exposed to GSM-modulated cell phone RFR compared to sham controls were also observed at the 14-week evaluation. For males exposed to CDMA-modulated cell phone RFR for 14 weeks, higher incidences of cardiomyopathy were observed relative to sham controls across the whole heart, not specifically in the right ventricle. The sporadic occurrences of schwannomas in the heart in females exposed to GSM or CDMA cell phone radiation were not considered to be related to RFR exposure.

Cardiomyopathy is a common spontaneous disease in rats that typically has no clinical manifestations. The early observation of this lesion at 14 weeks in the 3 and 6 W/Kg GSM males and the relatively higher modeled organ-specific SAR for heart for both males and females compared to other organs (Gong *et al.* 2017) suggest that the heart is a specific target organ for cell phone RFR.

In many cases isolated nonneoplastic or neoplastic lesion increases occurred in single or lower exposure groups, lacked a clear exposure response, or incidences were similar to incidences seen in control groups in past NTP studies. This reduced the confidence that these lesion increases were attributable to the cell phone RFR exposure. These were considered uncertain findings. In NTP conclusions, such uncertain responses in the absence of other clearer effects on carcinogenicity would be referred to as *equivocal evidence of carcinogenicity* (i.e., may have been related to exposure).

Malignant glioma of the brain was of specific interest given the epidemiology findings suggesting a possible link between this neoplasm type and cell phone use. Malignant gliomas are neoplasms of glial cells in the central nervous system (CNS). CNS glial cells include astrocytes, oligodendroglial cells, microglial cells, and ependymal cells, and collectively are termed glioma when cancerous.

In the current studies, there were occurrences of malignant glioma and/or glial cell hyperplasia in males at all SARs in the GSM study, and at the 1.5 and 6 W/kg SARs in the CDMA study. Neither of these lesions was observed in any of the sham control males. The occurrence of these lesions in RFR-exposed males and not in sham control males may reflect differences in survival, because nearly all (10/11) of the malignant gliomas in males were observed at 101 weeks or later and more than half (6/10) of the glial cell hyperplasias were observed at terminal necropsy. The incidences of malignant glioma at all SARs in the GSM study (2.2% to 3.3%) and at 6 W/kg in the CDMA study (3.3%) exceeded the historical control range for incidences of malignant gliomas in the brain of male rats (0%-2%; NTP, 2018a). However, as mentioned previously, the historical control data are currently limited to studies that have been conducted and peer reviewed since the NTP began using the Hsd:Sprague Dawley SD rat model (the data from the indol-3-carbinol were excluded because those studies were conducted prior to NTP's implementation of the enhanced sectioning of the brain performed in the current studies). To increase confidence in assessing the rarity of malignant gliomas in the Hsd:Sprague Dawley SD rat, a survey of malignant gliomas in control groups from nine other NTP studies that are currently under review (NTP, 2018a) confirmed this mean background rate of 2%, although one study had an incidence of 8%. It should be noted that none of these other studies were conducted under the same housing and exposure conditions as the current studies. In females, there were occurrences of malignant glioma at 1.5 W/kg, and a single instance of glial cell hyperplasia at 3 and 6 W/kg in the CDMA study. In their SAR modeling estimates for cerebral hemisphere, Gong *et al.* (2017) indicated a relatively low absorption rate in relation to other tissues. Taken together, these findings in GSM and CDMA males, and CDMA females are judged as *equivocal evidence of carcinogenic activity*.

In addition to the malignant gliomas, the combined occurrence of benign or malignant granular cell tumors in the male GSM groups may have been related to exposure. Although the higher occurrences were not statistically significant, granular cell tumors occurred in all GSM-exposed male groups, and the combined incidences of these

tumors (3% to 4%) were higher than the concurrent sham control (1%) and the historical control range (0%-2%; NTP, 2018a). The granular cell tumors and malignant gliomas in GSM males were not considered to be biologically related because they arise from different tissues.

In the adrenal medulla of GSM-exposed males, there were higher incidences of benign pheochromocytoma in the 1.5 and 3 W/kg groups compared to sham controls and greater incidences of benign, malignant, or complex pheochromocytoma (combined) in the same exposure groups compared to sham controls due mostly to the rise in the benign neoplasms. At these same exposure groups, the incidences of hyperplasia were lower. Compared to sham controls, the higher benign, malignant, or complex pheochromocytoma (combined) incidences in the low- and mid-exposure groups (27% and 31%, respectively), were higher than the historical control range of 13%-28%. Taken together, the higher incidences of benign, malignant, or complex pheochromocytoma (combined) may have been related to exposure. In the female adrenal medulla, there was a higher incidence of hyperplasia at 6 W/kg in the GSM study, but no increase in pheochromocytoma, compared to sham controls.

In CDMA-exposed males, there was no exposure-related effect on the incidence of pheochromocytoma. In females, there was a higher incidence in the 1.5 W/kg group of benign, malignant, or complex pheochromocytoma (combined) compared to sham controls mostly due to the higher incidence of benign pheochromocytoma and no effect on the hyperplasia incidences in any of the exposed groups. This higher incidence (8% vs. 1% in sham controls) of benign pheochromocytoma occurred only at the lowest exposure and was outside the historical control range (0%-6%). This low-dose effect may have been related to CDMA exposure in females. The isolated increased incidence in the low-exposure group reduces the confidence that this effect is attributable to the cell phone RFR exposure and it was considered an equivocal finding.

Incidences of adenoma or carcinoma (combined) in the liver were observed in all groups of male rats exposed to CDMA-modulated RFR; none were observed in the sham control group. Hepatocellular adenomas and carcinomas appear to be rare in the Hsd:Sprague Dawley SD rat (1/240 and 0/240, respectively) and the combined incidence at 3 W/kg (4%) in CDMA RFR-exposed male rats exceeded the highest incidence seen in single studies in the

historical controls (2%). However, the increased incidence in the mid-exposure group reduces the confidence that this is attributable to the cell phone RFR exposure, so this was considered an equivocal finding.

The incidences of adenoma or carcinoma (combined) in pancreatic islets were higher in all groups of male rats exposed to GSM-modulated cell phone RFR compared to the sham controls; however, only the incidence at 1.5 W/kg was significantly increased compared to sham controls. The incidences in all groups of GSM-exposed rats (19% to 30%) exceeded the range in the historical controls (4%-16%). The lack of an exposure-response gradient reduced the confidence that this is attributable to cell phone RFR exposure, so this was considered an equivocal finding.

The incidences of adenoma of the pituitary gland (pars distalis) were increased (not significant) in most groups of male rats exposed to either GSM- or CDMA-modulated cell phone RFR, compared to the sham controls. In the GSM study, the incidences in all groups of exposed male rats exceeded the incidence in the historical controls. However, the incidences of adenoma were similar between all exposed groups regardless of SAR. In the CDMA study, there was a significantly higher incidence of adenoma at the 3 W/kg male group compared to sham controls, with a similar (nonsignificant) response at 1.5 W/kg. The incidence in the 6 W/kg group was lower than in sham controls. The lack of an exposure-response gradient reduces the confidence that the increased incidences are attributable to cell phone RFR exposure, so this was considered an equivocal finding. There were no differences in the incidences of hyperplasia in any of the groups of exposed male rats compared to sham controls in either of the studies (GSM or CDMA).

In GSM-exposed males, there were higher incidences of prostate adenoma (7%) and adenoma or carcinoma (combined) (8%) (due mostly to the adenomas) in the 3 W/kg group versus the sham control (2%). Although not statistically significant, the background rate of prostate gland adenoma is low (2/240) for Hsd:Sprague Dawley SD rats with the two adenomas observed in the limited historical control coming from the current study. A single occurrence of prostate gland carcinoma was also observed in the mid-exposure group; this neoplasm has not been observed in historical controls (0/240) and is considered rare in rats. Preneoplastic lesions of epithelial hyperplasia were higher in the exposure groups, but these were not statistically higher compared to sham controls. The higher

incidence in prostate gland adenoma or carcinoma (combined) in the mid-exposure group may have been related to GSM-modulated cell phone RFR exposure, although the lack of an exposure response reduces the confidence that the increased incidence is attributable to exposure, therefore, this was considered an equivocal finding. No effect in prostate gland neoplasms was observed in the CDMA study; however, prostate gland epithelial hyperplasia was higher compared to sham controls in all CDMA groups, with a significant increase in the 6 W/kg group versus sham controls and a statistically significant positive trend across exposure groups.

Subsets of male and female rats from the 2-year studies were examined at 14 weeks to evaluate biomarkers of genotoxicity. Chromosomal damage was evaluated using the peripheral blood erythrocyte micronucleus (MN) assay, and DNA damage was evaluated in the frontal cortex, hippocampus, cerebellum, liver, and peripheral blood using the comet assay. Results of the MN assays were negative, but higher levels of DNA damage were observed in some tissues of male rats (hippocampus and frontal cortex in the CDMA modulation). In general, results of the comet assay suggested that CDMA induced more effects than GSM, and male rats showed greater sensitivity than female rats. However, the difference between the response in males and females may have been related to the longer interval between cessation of exposure and tissue sampling that occurred for the females, potentially allowing for an increased amount of DNA repair to take place.

There were several instances in which one or two animals within a group of cell phone RFR-exposed rats showed high levels of DNA damage compared to the rest of the animals within the exposure group, while levels of DNA damage in sham control animals tended to be more tightly clustered. Interanimal variation in response to cell phone RFR might be due to the genetic heterogeneity of these Hsd:Sprague Dawley SD rats, which are maintained as an outbred stock. However, tissues from a single rat rarely tracked together with regard to the extent of DNA damage (i.e., there was never a case in which all of the tissues from one rat had the highest mean percent comet tail DNA). This observation suggests intertissue variability in response, as has also been seen with chemicals (Sasaki *et al.*, 2000). Although the markedly higher levels of DNA damage observed in some rats were suggestive of an exposure-related effect, the high degree of interanimal variation within a treatment group resulted in nonsignificant statistical tests in most instances (for example, male rat cerebellum exposed to CDMA and female rat peripheral blood exposed to CDMA).

No histopathologic assessments of cytotoxicity (apoptosis and necrosis) were conducted in the brain or liver tissues that were examined for DNA damage, which leaves open the possibility that apoptosis or necrosis may have confounded the comet assay results. However, this seems unlikely as brain sections from other groups of rats at the 14-week interim evaluation and in the 2-year study did undergo histopathologic assessment and no significant findings were reported. Unlike ionizing radiation or ultraviolet light, cell phone RFR is not sufficiently energetic, by several orders of magnitude, to directly damage macromolecules (IARC, 2013), and little is known about the mechanism by which RFR could induce DNA damage in the absence of a thermal effect. Proposed mechanisms include, for example, induction of oxygen radicals and interference with DNA repair mechanisms (Ruediger, 2009; Yakymenko *et al.*, 2016).

The primary effects of cell phone RFR in rats included perinatal effects on body weights and body weight gains in the F₀ dams and the F₁ offspring, higher body temperatures in the F₀ dams, higher incidences of neoplasms (schwannomas) and nonneoplastic lesions (Schwann cell hyperplasia and right ventricular cardiomyopathy) of the heart, and higher incidences of nonneoplastic lesions of the adrenal gland, prostate gland, and thyroid gland. The greater survival in cell phone RFR-exposed males compared to sham controls was attributed to decreased chronic progressive nephropathy, which may be a result of cell phone RFR exposure. These findings occurred in both modulations suggesting the modulations did not impact the response to the 900 MHz cell phone RFR exposure.

With a few exceptions, male rats seemed more sensitive to effects of RFR than females. In pilot studies of effects of RFR on body temperature in young and aged rats, temperatures were generally higher in aged males than females at SARs of 10 W/kg and above with both GSM and CDMA modulation (Wyde *et al.*, 2018). The higher incidences of right ventricular cardiomyopathy compared to sham controls observed at 14 weeks and 2 years also occurred to a greater extent in males than in females. Heart schwannomas were exposure related in cell phone RFR-exposed males and not females with both modulations. More instances of marginally higher incidences in lesions compared to sham controls that may have been related to RFR exposure were observed in males (brain, pituitary gland, prostate gland, adrenal gland, and pancreas) than in females (brain and adrenal gland). Finally, reduced chronic progressive nephropathy in RFR-exposed male rats compared to sham controls in the 2-year studies was the likely

basis for the higher survival rate of RFR-exposed males (in particular in the GSM exposure groups). Reasons for these sex differences remain to be explored.

CONCLUSIONS

Under the conditions of this 2-year whole-body exposure study, there was *some evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley SD rats based on the incidences of malignant schwannoma in the heart. The incidences of adenoma or carcinoma (combined) in the prostate gland, malignant glioma and benign or malignant granular cell tumors in the brain, adenoma of the pars distalis in the pituitary gland, pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla, and pancreatic islet cell adenoma or carcinoma (combined) may have been related to cell phone RFR exposure. There was *no evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley SD rats administered 1.5, 3, or 6 W/kg. There was *some evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley SD rats based on the incidences of malignant schwannoma in the heart. The incidences of malignant glioma in the brain, adenoma of the pars distalis in the pituitary gland, and adenoma or carcinoma (combined) of the liver may have been related to cell phone RFR exposure. There was *equivocal evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley SD rats based on the incidences of malignant glioma in the brain and pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla.

Increases in nonneoplastic lesions in the heart, brain, and prostate gland of male rats, and of the heart, thyroid gland, and adrenal gland in female rats occurred with exposures to GSM cell phone RFR at 900 MHz. Increases in nonneoplastic lesions of the heart, brain, and prostate gland occurred in males, and of the brain in females exposed to CDMA cell phone RFR at 900 MHz.

* Explanation of Levels of Evidence of Carcinogenic Activity is on page 15.

REFERENCES

- Adair, R.K. (2003). Biophysical limits on athermal effects of RF and microwave radiation. *Bioelectromagnetics* **24**, 39-48.
- Ammari, M., Lecomte, A., Sakly, M., Abdelmelek, H., and de Seze, R. (2008). Exposure to GSM 900 MHz electromagnetic fields affects cerebral cytochrome c oxidase activity. *Toxicology* **250**, 70-74.
- Ammari, M., Gamez, C., Lecomte, A., Sakly, M., Abdelmelek, H., and de Seze, R. (2010). GFAP expression in the rat brain following sub-chronic exposure to a 900 MHz electromagnetic field signal. *Int. J. Radiat. Biol.* **86**, 367-375.
- Asanami, S., and Shimono, K. (1997). High body temperature induces micronuclei in mouse bone marrow. *Mutat. Res.* **390**, 79-83.
- Bailer, A.J., and Portier, C.J. (1988). Effects of treatment-induced mortality and tumor-induced mortality on tests for carcinogenicity in small samples. *Biometrics* **44**, 417-431.
- Barthold, S.W., Griffey, S.M., and Percy, D.H. (2016). *Pathology of Laboratory Rodents and Rabbits*, 4th ed., p. 156. John Wiley & Sons, Inc., Hoboken, NJ.
- Belyaev, I.Y., Koch, C.B., Terenius, O., Roxström-Lindquist, K., Malmgren, L.O.G., Sommer, W.H., Salford, L.G., and Persson, B.R.R. (2006). Exposure of rat brain to 915 MHz GSM microwaves induces changes in gene expression but not double stranded DNA breaks or effects on chromatin conformation. *Bioelectromagnetics* **27**, 295-306.
- Benson, V.S., Pirie, K., Schüz, J., Reeves, G.K., Beral, V., and Green, J. (2013). Mobile phone use and risk of brain neoplasms and other cancers: Prospective study. *Int. J. Epidemiol.* **42**, 792-802.
- Berridge, B.R., Mowat, V., Nagai, H., Nyska, A., Okazaki, Y., Clements, P.J., Rinke, M., Snyder, P.W., Boyle, M.C., and Wells, M.Y. (2016). Non-proliferative and proliferative lesions of the cardiovascular system of the rat and mouse. *J. Toxicol. Pathol.* **29** (3 Suppl.), 1S-47S.
- Bieler, G.S., and Williams, R.L. (1993). Ratio estimates, the delta method, and quantal response tests for increased carcinogenicity. *Biometrics* **49**, 793-801.
- Boorman, G.A., Montgomery, C.A., Jr., Eustis, S.L., Wolfe, M.J., McConnell, E.E., and Hardisty, J.F. (1985). Quality assurance in pathology for rodent carcinogenicity studies. In *Handbook of Carcinogen Testing* (H.A. Milman and E.K. Weisburger, Eds.), pp. 345-357. Noyes Publications, Park Ridge, NJ.
- Borbély, A.A., Huber, R., Graf, T., Fuchs, B., Gallmann, E., and Achermann, P. (1999). Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram. *Neurosci. Lett.* **275**, 207-210.
- Brendler-Schwaab, S., Hartmann, A., Pfuhler, S., and Speit, G. (2005). The *in vivo* comet assay: Use and status in genotoxicity testing. *Mutagenesis* **20**, 245-254.
- Brusick, D., Albertini, R., McRee, D., Peterson, D., Williams, G., Hanawalt, P., and Preston, J. (1998). Genotoxicity of radiofrequency radiation. DNA/Genetox Expert Panel. *Environ. Mol. Mutagen.* **32**, 1-16.
- Buddhikot, M.M., Kennedy, I., Mullany, F., and Viswanathan, H. (2009). Ultra-broadband femtocells via opportunistic reuse of multi-operator and multi-service spectrum. *Bell Labs Tech. J.* **13**, 129-143.

Burlinson, B., Tice, R.R., Speit, G., Agurell, E., Brendler-Schwaab, S.Y., Collins, A.R., Escobar, P., Honma, M., Kumaravel, T.S., Nakajima, M., Sasaki, Y.F., Thybaud, V., Uno, Y., Vasquez, M., and Hartmann, A. (2007). Fourth International Work Group on Genotoxicity Testing: Results of the *in vivo* comet assay workgroup. *Mutat. Res.* **627**, 31-35.

Capstick, M., Kuster, N., Kuehn, S., Gong, Y., Wilson, P., Ladbury, J., Koepke, G., McCormick, D.L., Gauger, J., and Melnick, R.L. (2017). A radio frequency radiation exposure system for rodents based on reverberation chambers. *IEEE Trans. Electromagn. Compatibil.* **59**, 1041-1052.

Cardis, E., Richardson, L., Deltour, I., Armstrong, B., Feychtig, M., Johansen, C., Kilkenny, M., McKinney, P., Modan, B., Sadetzki, S., Schüz, J., Swerdlow, A., Vrijheid, M., Auvinen, A., Berg, G., Blettner, M., Bowman, J., Brown, J., Chetrit, A., Christensen, H.C., Cook, A., Hepworth, S., Giles, G., Hours, M., Iavarone, I., Jarus-Hakak, A., Klaeboe, L., Krewski, D., Lagorio, S., Lönn, S., Mann, S., McBride, M., Muir, K., Nadon, L., Parent, M.E., Pearce, N., Salminen, T., Schoemaker, M., Schlehofer, B., Siemiatycki, J., Taki, M., Takebayashi, T., Tynes, T., van Tongeren, M., Vecchia, P., Wiart, J., Woodward, A., and Yamaguchi, N. (2007). The INTERPHONE study: Design, epidemiological methods, and description of the study population. *Eur. J. Epidemiol.* **22**, 647-664.

Cellular Telecommunications Industry Association (CTIA) (2017). Annual wireless industry survey.
<<https://www.ctia.org/industry-data/ctia-annual-wireless-industry-survey>>

Chagnaud, J.L., and Veyret, B. (1999). *In vivo* exposure of rats to GSM-modulated microwaves: Flow cytometry analysis of lymphocyte subpopulations and of mitogen stimulation. *Int. J. Radiat. Biol.* **75**, 111-113.

Chia, S.E., Chia, H.P., and Tan, J.S. (2000). Prevalence of headache among handheld cellular telephone users in Singapore: A community study. *Environ. Health Perspect.* **108**, 1059-1062.

Code of Federal Regulations (CFR) **21**, Part 58.

Code of Federal Regulations (CFR) **47** §1.1310.

Collins, A.R. (2004). The comet assay for DNA damage and repair. Principles, applications and limitations. *Mol. Biotechnol.* **26**, 246-261.

Cook, A., Woodward, A., Pearce, N., and Marshall, C. (2003). Cellular telephone use and time trends for brain, head and neck tumours. *N. Z. Med. J.* **116**, U457.

Cox, D.R. (1972). Regression models and life-tables. *J. R. Stat. Soc. B* **34**, 187-220.

Crawford, B.D. (1985). Perspectives on the somatic mutation model of carcinogenesis. In *Advances in Modern Environmental Toxicology. Mechanisms and Toxicity of Chemical Carcinogens and Mutagens* (M.A. Mehlman, W.G. Flamm, and R.J. Lorentzen, Eds.), pp. 13-59. Princeton Scientific Publishing Co., Inc., Princeton, NJ.

Czerninski, R., Zini, A., and Sgan-Cohen, H.D. (2011). Risk of parotid malignant tumors in Israel (1970–2006). *Epidemiology* **22**, 130-131.

Dasdag, S., Akdag, M.Z., Ulukaya, E., Uzunlar, A.K., and Ocak, A.R. (2009). Effect of mobile phone exposure on apoptotic glial cells and status of oxidative stress in rat brain. *Electromagn. Biol. Med.* **28**, 342-354.

de Gannes, F.P., Billaudel, B., Taxile, M., Haro, E., Ruffié, G., Lévéque, P., Veyret, B., and Lagroye, I. (2009). Effects of head-only exposure of rats to GSM-900 on blood-brain barrier permeability and neuronal degeneration. *Radiat. Res.* **172**, 359-367.

Deltour, I., Johansen, C., Auvinen, A., Feychtig, M., Klaeboe, L., and Schüz, J. (2009). Time trends in brain tumor incidence rates in Denmark, Finland, Norway, and Sweden, 1974–2003. *J. Natl. Cancer Inst.* **101**, 1721-1724.

de Vocht, F., Burstyn, I., and Cherrie, J.W. (2011). Time trends (1998–2007) in brain cancer incidence rates in relation to mobile phone use in England. *Bioelectromagnetics* **32**, 334-339.

Dixon, W.J., and Massey, F.J., Jr. (1957). *Introduction to Statistical Analysis*, 2nd ed., pp. 276-278, 412. McGraw-Hill Book Company, Inc., New York.

Dunn, O.J. (1964). Multiple comparisons using rank sums. *Technometrics* **6**, 241-252.

Dunnett, C.W. (1955). A multiple comparison procedure for comparing several treatments with a control. *J. Am. Stat. Assoc.* **50**, 1096-1121.

Eberhardt, J.L., Persson, B.R.R., Brun, A.E., Salford, L.G., and Malmgren, L.O.G. (2008). Blood-brain barrier permeability and nerve cell damage in rat brain 14 and 28 days after exposure to microwaves from GSM mobile phones. *Electromagn. Biol. Med.* **27**, 215-229.

Elekes, E., Thuróczy, G., and Szabó, L.D. (1996). Effect on the immune system of mice exposed chronically to 50 Hz amplitude-modulated 2.45 GHz microwaves. *Bioelectromagnetics* **17**, 246-248.

Eşmekaya, M.A., Seyhan, N., and Ömeroğlu, S. (2010). Pulse modulated 900 MHz radiation induces hypothyroidism and apoptosis in thyroid cells: A light, electron microscopy and immunohistochemical study. *Int. J. Radiat. Biol.* **86**, 1106-1116.

Frey, A.H. (1998). Headaches from cellular telephones: Are they real and what are the implications? *Environ. Health Perspect.* **106**, 101-103.

Fritze, K., Wiessner, C., Kuster, N., Sommer, C., Gass, P., Hermann, D.M., Kiessling, M., and Hossmann, K.A. (1997). Effect of global system for mobile communication microwave exposure on the genomic response of the rat brain. *Neuroscience* **81**, 627-639.

Fung, K.Y., Krewski, D., Rao, J.N.K., and Scott, A.J. (1994). Tests for trend in developmental toxicity experiments with correlated binary data. *Risk Anal.* **14**, 639-648.

Gart, J.J., Chu, K.C., and Tarone, R.E. (1979). Statistical issues in interpretation of chronic bioassay tests for carcinogenicity. *JNCI* **62**, 957-974.

Gatta, L., Pinto, R., Ubaldi, V., Pace, L., Galloni, P., Lovisolo, G.A., Marino, C., and Pioli, C. (2003). Effects of *in vivo* exposure to GSM-modulated 900 MHz radiation on mouse peripheral lymphocytes. *Radiat. Res.* **160**, 600-605.

Gong, Y., Capstick, M., Kuehn, S., Wilson, P., Ladbury, J., Koepke, G., McCormick, D.L., Melnick, R.L., and Kuster, N. (2017). Life-time dosimetric assessment for mice and rats exposed in reverberation chambers of the 2-year NTP cancer bioassay study on cell phone radiation. *IEEE Trans. Electromagn. Compatibil.* **59**, 1798-1808.

Grafström, G., Nittby, H., Brun, A., Malmgren, L., Persson, B.R.R., Salford, L.G., and Eberhardt, J. (2008). Histopathological examinations of rat brains after long-term exposure to GSM-900 mobile phone radiation. *Brain Res. Bull.* **77**, 257-263.

Guney, M., Ozguner, F., Oral, B., Karahan, N., and Mungan, T. (2007). 900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium: Protection by vitamins E and C. *Toxicol. Ind. Health* **23**, 411-420.

Guy, A.W., Chou, C.-K., Kunz, L.L., Crowley, J., and Krupp, J. (1985). Effects of long-term low-level radiofrequency radiation exposure on rats. Volume 9. Summary. Bioelectromagnetics Research Laboratory, Department of Rehabilitation Medicine, School of Medicine, University of Washington, Seattle, WA.
[<http://www.dtic.mil/docs/citations/ADA159512>](http://www.dtic.mil/docs/citations/ADA159512)

- Hardell, L., and Carlberg, M. (2009). Mobile phones, cordless phones and the risk for brain tumours. *Int. J. Oncol.* **35**, 5-17.
- Hardell, L., Hallquist, A., Hansson Mild, K., Carlberg, M., Gertzén, H., Schildt, E.B., and Dahlqvist, A. (2004). No association between the use of cellular or cordless telephones and salivary gland tumours. *Occup. Environ. Med.* **61**, 675-679.
- Hardell, L., Eriksson, M., Carlberg, M., Sundström, C., and Mild, K.H. (2005). Use of cellular or cordless telephones and the risk for non-Hodgkin's lymphoma. *Int. Arch. Occup. Environ. Health* **78**, 625-632.
- Hardell, L., Mild, K.H., Carlberg, M., and Söderqvist, F. (2006). Tumour risk associated with use of cellular telephones or cordless desktop telephones. *World J. Surg. Oncol.* **4**, 74.
- Hardell, L., Carlberg, M., Ohlson, C.G., Westberg, H., Eriksson, M., and Hansson Mild, K. (2007a). Use of cellular and cordless telephones and risk of testicular cancer. *Int. J. Androl.* **30**, 115-122.
- Hardell, L., Carlberg, M., Söderqvist, F., Mild, K.H., and Morgan, L.L. (2007b). Long-term use of cellular phones and brain tumours: Increased risk associated with use for ≥ 10 years. *Occup. Environ. Med.* **64**, 626-632.
- Hardell, L., Carlberg, M., Hansson Mild, K., and Eriksson, M. (2011). Case-control study on the use of mobile and cordless phones and the risk for malignant melanoma in the head and neck region. *Pathophysiology* **18**, 325-333.
- Hartmann, A., Agurell, E., Beevers, C., Brendler-Schwaab, S., Burlinson, B., Clay, P., Collins, A., Smith, A., Speit, G., Thybaud, V., and Tice, R.R. (2003). Recommendations for conducting the *in vivo* alkaline Comet assay. *Mutagenesis* **18**, 45-51.
- Hauet-Broere, F., Wieten, L., Guichelaar, T., Berlo, S., van der Zee, R., and Van Eden, W. (2006). Heat shock proteins induce T cell regulation of chronic inflammation. *Ann. Rheum. Dis.* **65**(Suppl. III), 65-68.
- Heddle, J.A., Hite, M., Kirkhart, B., Mavournin, K., MacGregor, J.T., Newell, G.W., and Salamone, M.F. (1983). The induction of micronuclei as a measure of genotoxicity. A report of the U.S. Environmental Protection Agency Gene-Tox Program. *Mutat. Res.* **123**, 61-118.
- Hocking, B., and Westerman, R. (2000). Neurological abnormalities associated with mobile phone use. *Occup. Med. (London)* **50**, 366-368.
- Hsu, J.C. (1992). The factor analytic approach to simultaneous inference in the general linear model. *Journal of Computational and Graphical Statistics*. **1**, 151-168.
- Huber, R., Graf, T., Cote, K.A., Wittmann, L., Gallmann, E., Matter, D., Schuderer, J., Kuster, N., Borbély, A.A., and Achermann, P. (2000). Exposure to pulsed high-frequency electromagnetic field during waking affects human sleep EEG. *Neuroreport* **11**, 3321-3325.
- Huber, R., Treyer, V., Borbély, A.A., Schuderer, J., Gottselig, J.M., Landolt, H.P., Werth, E., Berthold, T., Kuster, N., Buck, A., and Achermann, P. (2002). Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG. *J. Sleep Res.* **11**, 289-295.
- Huber, R., Schuderer, J., Graf, T., Jütz, K., Borbély, A.A., Kuster, N., and Achermann, P. (2003). Radio frequency electromagnetic field exposure in humans: Estimation of SAR distribution in the brain, effects on sleep and heart rate. *Bioelectromagnetics* **24**, 262-276.
- Hung, C.S., Anderson, C., Horne, J.A., and McEvoy, P. (2007). Mobile phone 'talk-mode' signal delays EEG-determined sleep onset. *Neurosci. Lett.* **421**, 82-86.

- Hunt, C.R., Pandita, R.K., Laszlo, A., Higashikubo, R., Agarwal, M., Kitamura, T., Gupta, A., Rief, N., Horikoshi, N., Baskaran, R., Lee, J.-H., Löbrich, M., Paull, T.T., Roti Roti, J.L., and Pandita, T.K. (2007). Hyperthermia activates a subset of ataxia-telangiectasia mutated effectors independent of DNA strand breaks and heat shock protein 70 status. *Cancer Res.* **67**, 3010-3017.
- Ilhan, A., Gurel, A., Armutcu, F., Kamisli, S., Iraz, M., Akyol, O., and Ozen, S. (2004). Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain. *Clin. Chim. Acta.* **340**, 153-162.
- Imge, E.B., Kılıçoglu, B., Devrim, E., Cetin, R., and Durak, I. (2010). Effects of mobile phone use on brain tissue from the rat and a possible protective role of vitamin C – a preliminary study. *Int. J. Radiat. Biol.* **86**, 1044-1049.
- Inskip, P.D., Hoover, R.N., and Devesa, S.S. (2010). Brain cancer incidence trends in relation to cellular telephone use in the United States. *Neuro-oncology* **12**, 1147-1151.
- International Agency for Research on Cancer (IARC) (2011). IARC Report to the Union for International Cancer Control (UICC) on the Interphone Study. IARC, Lyon, France.
<http://interphone.iarc.fr/UICC_Report_Final_03102011.pdf>
- International Agency for Research on Cancer (IARC) (2013). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Non-ionizing Radiation, Part 2: Radiofrequency Electromagnetic Fields, Vol. 102. IARC, Lyon, France.
- International Commission on Non-Ionizing Radiation Protection (ICNIRP) (1998). Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). *Health Phys.* **74**, 494-522.
- INTERPHONE Study Group (2010). Brain tumour risk in relation to mobile telephone use: Results of the INTERPHONE international case-control study. *Int. J. Epidemiol.* **39**, 675-694.
- INTERPHONE Study Group (2011). Acoustic neuroma risk in relation to mobile telephone use: Results of the INTERPHONE international case-control study. *Cancer Epidemiol.* **35**, 453-464.
- Johansen, C., Boice, J.D., Jr., McLaughlin, J.K., and Olsen, J.H. (2001). Cellular telephones and cancer – a nationwide cohort study in Denmark. *J. Natl. Cancer Inst.* **93**, 203-207.
- Jonckheere, A.R. (1954). A distribution-free k -sample test against ordered alternatives. *Biometrika* **41**, 133-145.
- Kampinga, H.H., and Dikomey, E. (2001). Hyperthermic radiosensitization: Mode of action and clinical relevance. *Int. J. Radiat. Biol.* **77**, 399-408.
- Kaplan, E.L., and Meier, P. (1958). Nonparametric estimation from incomplete observations. *J. Am. Stat. Assoc.* **53**, 457-481.
- Kaufman, D.W., Anderson, T.E., and Issaragrisil, S. (2009). Risk factors for leukemia in Thailand. *Ann. Hematol.* **88**, 1079-1088.
- Kissling, G.E., Dertinger, S.D., Hayashi, M., and MacGregor, J.T. (2007). Sensitivity of the erythrocyte micronucleus assay: Dependence on number of cells scored and inter-animal variability. *Mutat. Res.* **634**, 235-240.
- Kolomytseva, M.P., Gapeev, A.B., Sadovnikov, V.B., and Chemeris, N.K. (2002). Suppression of nonspecific resistance of the body under the effect of extremely high frequency electromagnetic radiation of low intensity [in Russian]. *Biofizika* **47**, 71-77.
- Komae, N., Hibino, Y., and Sugano, N. (1999). Analysis of micronuclei induced under hyperthermic conditions in human lymphocyte culture by fluorescence *in situ* hybridization (FISH) and spectral karyotyping (SKY) methods [in Japanese]. *Yakugaku Zasshi* **119**, 763-772.

Kwon, M.S., and Hämäläinen, H. (2011). Effects of mobile phone electromagnetic fields: Critical evaluation of behavioral and neurophysiological studies. *Bioelectromagnetics* **32**, 253-272.

La Regina, M., Moros, E.G., Pickard, W.F., Straube, W.L., Baty, J., and Roti Roti, J.L. (2003). The effect of chronic exposure to 835.62 MHz FDMA or 847.74 MHz CDMA radiofrequency radiation on the incidence of spontaneous tumors in rats. *Radiat. Res.* **160**, 143-151.

Lee, H.J., Pack, J.K., Kim, T.H., Kim, N., Choi, S.Y., Lee, J.S., Kim, S.H., and Lee, Y.S. (2010). The lack of histological changes of CDMA cellular phone-based radio frequency on rat testis. *Bioelectromagnetics* **31**, 528-534.

Lee, H.J., Jin, Y.B., Lee, J.S., Choi, S.Y., Kim, T.H., Pack, J.K., Choi, H.D., Kim, N., and Lee, Y.S. (2011). Lymphoma development of simultaneously combined exposure to two radiofrequency signals in AKR/J mice. *Bioelectromagnetics* **32**, 485-492.

Lin, J.C. (2017). Cancer occurrences in laboratory rats from exposure to RF and microwave radiation. *IEEE REM* **1**, 2-13.

Linet, M.S., Taggart, T., Severson, R.K., Cerhan, J.R., Cozen, W., Hartge, P., and Colt, J. (2006). Cellular telephones and non-Hodgkin lymphoma. *Int. J. Cancer* **119**, 2382-2388.

Lönn, S., Klaeboe, L., Hall, P., Mathiesen, T., Auvinen, A., Christensen, H.C., Johansen, C., Salminen, T., Tynes, T., and Feychting, M. (2004a). Incidence trends of adult primary intracerebral tumors in four Nordic countries. *Int. J. Cancer* **108**, 450-455.

Lönn, S., Ahlbom, A., Hall, P., and Feychting, M. (2004b). Mobile phone use and the risk of acoustic neuroma. *Epidemiology* **15**, 653-659.

Lönn, S., Ahlbom, A., Christensen, H.C., Johansen, C., Schüz, J., Edström, S., Henriksson, G., Lundgren, J., Wennerberg, J., and Feychting, M. (2006). Mobile phone use and risk of parotid gland tumor. *Am. J. Epidemiol.* **164**, 637-643.

Lorenzo, Y., Costa, S., Collins, A.R., and Azqueta, A. (2013). The comet assay, DNA damage, DNA repair and cytotoxicity: Hedgehogs are not always dead. *Mutagenesis* **28**, 427-432.

Loughran, S.P., Wood, A.W., Barton, J.M., Croft, R.J., Thompson, B., and Stough, C. (2005). The effect of electromagnetic fields emitted by mobile phones on human sleep. *Neuroreport* **16**, 1973-1976.

Lowden, A., Åkerstedt, T., Ingre, M., Wiholm, C., Hillert, L., Kuster, N., Nilsson, J.P., and Arnetz, B. (2011). Sleep after mobile phone exposure in subjects with mobile phone-related symptoms. *Bioelectromagnetics* **32**, 4-14.

Lushnikov, K.V., Gapeev, A.B., Sadovnikov, V.B., and Cheremis, N.K. (2001). Effect of extremely high frequency electromagnetic radiation of low intensity on parameters of humoral immunity in healthy mice [in Russian]. *Biofizika* **46**, 753-760.

MacGregor, J.T., Bishop, M.E., McNamee, J.P., Hayashi, M., Asano, N., Wakata, A., Nakajima, M., Saito, J., Aidoo, A., Moore, M.M., and Dertinger, D.D. (2006). Flow cytometric analysis of micronuclei in peripheral blood reticulocytes: II. An efficient method for monitoring chromosomal damage in the rat. *Toxicol. Sci.* **94**, 92-107.

MacKenzie, W.F., and Alison, R. (1990). Heart. In *Pathology of the Fischer Rat. Reference and Atlas* (G.A. Boorman, S.L. Eustis, M.R. Elwell, C.A. Montgomery, Jr., and W.F. MacKenzie, Eds.), p. 461. Academic Press, Inc., San Diego, CA.

McQuade, J.M.S., Merritt, J.H., Miller, S.A., Scholin, T., Cook, M.C., Salazar, A., Rahimi, O.B., Murphy, M.R., and Mason, P.A. (2009). Radiofrequency-radiation exposure does not induce detectable leakage of albumin across the blood-brain barrier. *Radiat. Res.* **171**, 615-621.

- Mailankot, M., Kunnath, A.P., Jayalekshmi, H., Koduru, B., and Valsalan, R. (2009). Radio frequency electromagnetic radiation (RF-EMR) from GSM (0.9/1.8GHz) mobile phones induces oxidative stress and reduces sperm motility in rats. *Clinics (Sao Paulo)* **64**, 561-565.
- Mann, K., and Röschke, J. (1996). Effects of pulsed high-frequency electromagnetic fields on human sleep. *Neuropsychobiology* **33**, 41-47.
- Maronpot, R.R., and Boorman, G.A. (1982). Interpretation of rodent hepatocellular proliferative alterations and hepatocellular tumors in chemical safety assessment. *Toxicol. Pathol.* **10**, 71-80.
- Masuda, H., Sanchez, S., Dulou, P.E., Haro, E., Anane, R., Billaudel, B., Lévéque, P., and Veyret, B. (2006). Effect of GSM-900 and -1800 signals on the skin of hairless rats. I: 2-Hour acute exposures. *Int. J. Radiat. Biol.* **82**, 669-674.
- Masuda, H., Ushiyama, A., Takahashi, M., Wang, J., Fujiwara, O., Hikage, T., Nojima, T., Fujita, K., Kudo, M., and Ohkubo, C. (2009). Effects of 915 MHz electromagnetic-field radiation in TEM cell on the blood-brain barrier and neurons in the rat brain. *Radiat. Res.* **172**, 66-73.
- Meral, I., Mert, H., Mert, N., Deger, Y., Yoruk, I., Yetkin, A., and Keskin, S. (2007). Effects of 900-MHz electromagnetic field emitted from cellular phone on brain oxidative stress and some vitamin levels of guinea pigs. *Brain Res.* **1169**, 120-124.
- Miller, J.A., and Miller, E.C. (1977). Ultimate chemical carcinogens as reactive mutagenic electrophiles. In *Origins of Human Cancer* (H.H. Hiatt, J.D. Watson, and J.A. Winsten, Eds.), pp. 605-627. Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.
- Muscat, J.E., Hinsvark, M., and Malkin, M. (2006). Mobile telephones and rates of brain cancer. *Neuroepidemiology* **27**, 55-56.
- Nam, J.M. (1987). A simple approximation for calculating sample sizes for detecting linear trend in proportions. *Biometrics* **43**, 701-705.
- Nasta, F., Prisco, M.G., Pinto, R., Lovisolo, G.A., Marino, C., and Pioli, C. (2006). Effects of GSM-modulated radiofrequency electromagnetic fields on B-cell peripheral differentiation and antibody production. *Radiat. Res.* **165**, 664-670.
- National Toxicology Program (NTP) (2018a). Historical controls. <<https://ntp.niehs.nih.gov/results/dbsearch/historical/index.html>> Accessed January 24, 2018.
- National Toxicology Program (NTP) (2018b). Toxicology and Carcinogenesis Studies of GSM- and CDMA-Modulated Cell Phone Radio Frequency Radiation at 1,900 MHz in B6C3F1/N Mice (Whole-Body Exposure). Technical Report Series No. 596. National Institutes of Health, Public Health Service, U.S. Department of Health and Human Services, Research Triangle Park, NC (in preparation).
- Nelson, P.D., Toledano, M.B., McConville, J., Quinn, M.J., Cooper, N., and Elliott, P. (2006). Trends in acoustic neuroma and cellular phones: Is there a link? *Neurology* **66**, 284-285.
- Nittby, H., Widegren, B., Krogh, M., Grafström, G., Berlin, H., Rehn, G., Eberhardt, J.L., Malmgren, L., Persson, B.R.R., and Salford, L.G. (2008). Exposure to radiation from global system for mobile communications at 1,800 MHz significantly changes gene expression in rat hippocampus and cortex. *Environmentalist* **28**, 458-465.
- Nittby, H., Brun, A., Eberhardt, J., Malmgren, L., Persson, B.R.R., and Salford, L.G. (2009). Increased blood-brain barrier permeability in mammalian brain 7 days after exposure to the radiation from a GSM-900 mobile phone. *Pathophysiology* **16**, 103-112.
- Nittby, H., Brun, A., Strömlad, S., Moghadam, M.K., Sun, W., Malmgren, L., Eberhardt, J., Persson, B.R., and Salford, L.G. (2011). Nonthermal GSM RF and ELF EMF effects upon rat BBB permeability. *Environmentalist* **31**, 140-148.

- Nomura, E., Ioka, A., and Tsukuma, H. (2011). Trends in the incidence of primary intracranial tumors in Osaka, Japan. *Jpn. J. Clin. Oncol.* **41**, 291-294.
- Novoselova, E.G., Fesenko, E.E., Makar, V.R., and Sadovnikov, V.B. (1999). Microwaves and cellular immunity. II. Immunostimulating effects of microwaves and naturally occurring antioxidant nutrients. *Bioelectrochem. Bioenerg.* **49**, 37-41.
- Oberto, G., Rolfo, K., Yu, P., Carbonatto, M., Peano, S., Kuster, N., Ebert, S., and Tofani, S. (2007). Carcinogenicity study of 217 Hz pulsed 900 MHz electromagnetic fields in *Pim1* transgenic mice. *Radiat. Res.* **168**, 316-326.
- Office of Engineering and Technology (OET) (1999). Questions and answers about biological effects and potential hazards of radiofrequency electromagnetic fields. OET Bulletin 56, 4th ed. Federal Communication Commission, Washington, DC. <https://transition.fcc.gov/Bureaus/Engineering_Technology/Documents/bulletins/oet56/oet56e4.pdf>
- Oktem, F., Ozguner, F., Mollaoglu, H., Koyu, A., and Uz, E. (2005). Oxidative damage in the kidney induced by 900-MHz-emitted mobile phone: Protection by melatonin. *Arch. Med. Res.* **36**, 350-355.
- Oral, B., Guney, M., Ozguner, F., Karahan, N., Mungan, T., Comlekci, S., and Cesur, G. (2006). Endometrial apoptosis induced by a 900-MHz mobile phone: Preventive effects of vitamins E and C. *Adv. Ther.* **23**, 957-973.
- Organisation for Economic Cooperation and Development (OECD) (2014). OECD Guideline for the Testing of Chemicals. In vivo Mammalian Alkaline Comet Assay. Testing Guideline 489. OECD Publishing, Paris.
- Ozguner, F., Altinbas, A., Ozaydin, M., Dogan, A., Vural, H., Kisio glu, A.N., Cesur, G., and Yildirim, N.G. (2005a). Mobile phone-induced myocardial oxidative stress: Protection by a novel antioxidant agent caffeic acid phenethyl ester. *Toxicol. Ind. Health* **21**, 223-230.
- Ozguner, F., Oktem, F., Arman, A., Yilmaz, R., Koyu, A., Demirel, R., Vural, H., and Uz, E. (2005b). Comparative analysis of the protective effects of melatonin and caffeic acid phenethyl ester (CAPE) on mobile phone-induced renal impairment in rat. *Mol. Cell. Biochem.* **276**, 31-37.
- Ozguner, F., Bardak, Y., and Comlekci, S. (2006). Protective effects of melatonin and caffeic acid phenethyl ester against retinal oxidative stress in long-term use of mobile phone: A comparative study. *Mol. Cell. Biochem.* **282**, 83-88.
- Ozgur, E., Güler, G., and Seyhan, N. (2010). Mobile phone radiation-induced free radical damage in the liver is inhibited by the antioxidants N-acetyl cysteine and epigallocatechin-gallate. *Int. J. Radiat. Biol.* **86**, 935-945.
- Paparini, A., Rossi, P., Gianfranceschi, G., Brugaletta, V., Falsaperla, R., De Luca, P., and Romano Spica, V. (2008). No evidence of major transcriptional changes in the brain of mice exposed to 1800 MHz GSM signal. *Bioelectromagnetics* **29**, 312-323.
- Pew Research Center Survey (2017). Mobile fact sheet. Internet and Technology, Pew Research Center. Washington, D.C. <<http://www.pewinternet.org/fact-sheet/mobile>>
- Pfuhler, S., and Wolf, H.U. (1996). Detection of DNA-crosslinking agents with the alkaline comet assay. *Environ. Mol. Mutagen.* **27**, 196-201.
- Piegorsch, W.W., and Bailer, A.J. (1997). *Statistics for Environmental Biology and Toxicology*, Section 6.3.2. Chapman and Hall, London.
- Portier, C.J., and Bailer, A.J. (1989). Testing for increased carcinogenicity using a survival-adjusted quantal response test. *Fundam. Appl. Toxicol.* **12**, 731-737.

- Portier, C.J., Hedges, J.C., and Hoel, D.G. (1986). Age-specific models of mortality and tumor onset for historical control animals in the National Toxicology Program's carcinogenicity experiments. *Cancer Res.* **46**, 4372-4378.
- Prohofsky, E.W. (2004). RF absorption involving biological macromolecules. *Bioelectromagnetics* **25**, 441-451.
- Propp, J.M., McCarthy, B.J., Davis, F.G., and Preston-Martin, S. (2006). Descriptive epidemiology of vestibular schwannomas. *Neurooncology* **8**, 1-11.
- Pyrpasopoulou, A., Kotoula, V., Cheva, A., Hytioglou, P., Nikolakaki, E., Magras, I.N., Xenos, T.D., Tsiboukis, T.D., and Karkavelas, G. (2004). Bone morphogenetic protein expression in newborn rat kidneys after prenatal exposure to radiofrequency radiation. *Bioelectromagnetics* **25**, 216-227.
- Rao, G.N. (2002). Diet and kidney disease in rats. *Toxicol. Pathol.* **30**, 651-656.
- Rao, J.N.K., and Scott, A.J. (1992). A simple method for the analysis of clustered binary data. *Biometrics* **48**, 577-585.
- Recio, L., Hobbs, C., Caspary, W., and Witt, K.L. (2010). Dose-response assessment of four genotoxic chemicals in a combined mouse and rat micronucleus (MN) and Comet assay protocol. *J. Toxicol. Sci.* **35**, 149-162.
- Regel, S.J., Tinguely, G., Schuderer, J., Adam, M., Kuster, N., Landolt, H.P., and Achermann, P. (2007). Pulsed radio-frequency electromagnetic fields: Dose-dependent effects on sleep, the sleep EEG and cognitive performance. *J. Sleep Res.* **16**, 253-258.
- Repacholi, M.H., Lerchl, A., Röösli, M., Sienkiewicz, Z., Auvinen, A., Breckenkamp, J., d'Inzeo, G., Elliott, P., Frei, P., Heinrich, S., Lagroye, I., Lahkola, A., McCormick, D.L., Thomas, S., and Vecchia, P. (2012). Systematic review of wireless phone use and brain cancer and other head tumors. *Bioelectromagnetics* **33**, 187-206.
- Röösli, M., Michel, G., Kuehni, C.E., and Spoerri, A. (2007). Cellular telephone use and time trends in brain tumour mortality in Switzerland from 1969 to 2002. *Eur. J. Cancer Prev.* **16**, 77-82.
- Ruediger, H.W. (2009). Genotoxic effects of radiofrequency electromagnetic fields. *Pathophysiology* **16**, 89-102.
- Rundell, M.S., Wagner, E.D., and Plewa, M.J. (2003). The comet assay: Genotoxic damage or nuclear fragmentation? *Environ. Mol. Mutagen.* **42**, 61-67.
- Saika, K., and Katanoda, K. (2011). Comparison of time trends in brain and central nervous system cancer mortality (1990–2006) between countries based on the WHO mortality database. *Jpn. J. Clin. Oncol.* **41**, 304-305.
- Sanchez, S., Masuda, H., Billaudel, B., Haro, E., Anane, R., Lévéque, P., Ruffie, G., Lagroye, I., and Veyret, B. (2006). Effect of GSM-900 and -1800 signals on the skin of hairless rats. II: 12-Week chronic exposures. *Int. J. Radiat. Biol.* **82**, 675-680.
- Sanchez, S., Masuda, H., Ruffie, G., De Gannes, F.P., Billaudel, B., Haro, E., Lévéque, P., Lagroye, I., and Veyret, B. (2008). Effect of GSM-900 and -1800 signals on the skin of hairless rats. III: Expression of heat shock proteins. *Int. J. Radiat. Biol.* **84**, 61-68.
- Sandström, M., Wilen, J., Oftedal, G., and Hansson Mild, K. (2001). Mobile phone use and subjective symptoms. Comparison of symptoms experienced by users of analogue and digital mobile phones. *Occup. Med. (London)* **51**, 25-35.
- Santini, R., Santini, P., Danze, J.M., Le Ruz, P., and Seigne, M. (2002a). Investigation on the health of people living near mobile telephone relay stations: I/Incidence according to distance and sex [in French]. *Pathol. Biol. (Paris)* **50**, 369-373.

- Santini, R., Seigne, M., Bonhomme-Faivre, L., Bouffet, S., Defrasne, E., and Sage, M. (2002b). Symptoms experienced by users of digital cellular phones: A study of a French engineering school. *Electromagn. Biol. Med.* **21**, 81-88.
- Sasaki, Y.F., Sekihashi, K., Izumiya, F., Nishidate, E., Saga, A., Ishida, K., and Tsuda, S. (2000). The comet assay with multiple mouse organs: Comparison of comet assay results and carcinogenicity with 208 chemicals selected from the IARC monographs and U.S. NTP carcinogenicity database. *Crit. Rev. Toxicol.* **30**, 629-799.
- Schmid, W. (1975). The micronucleus test. *Mutat. Res.* **31**, 9-15.
- Schoemaker, M.J., and Swerdlow, A.J. (2009). Risk of pituitary tumors in cellular phone users: A case-control study. *Epidemiology* **20**, 348-354.
- Schüz, J., Jacobsen, R., Olsen, J.H., Boice, J.D., Jr., McLaughlin, J.K., and Johansen, C. (2006). Cellular telephone use and cancer risk: Update of a nationwide Danish cohort. *J. Natl. Cancer. Inst.* **98**, 1707-1713.
- Schüz, J., Elliott, P., Auvinen, A., Kromhout, H., Poulsen, A.H., Johansen, C., Olsen, J.H., Hillert, L., Feychtig, M., Fremling, K., Toledano, M., Heinävaara, S., Slottje, P., Vermeulen, R., and Ahlbom, A. (2011). An international prospective cohort study of mobile phone users and health (Cosmos): Design considerations and enrolment. *Cancer Epidemiol.* **35**, 37-43.
- Sheppard, A.R., Swicord, M.L., and Balzano, Q. (2008). Quantitative evaluations of mechanisms of radiofrequency interactions with biological molecules and processes. *Health Phys.* **95**, 365-396.
- Shirley, E. (1977). A non-parametric equivalent of Williams' test for contrasting increasing dose levels of a treatment. *Biometrics* **33**, 386-389.
- Smialowicz, R.J., Rogers, R.R., Garner, R.J., Riddle, M.M., Luebke, R.W., and Rowe, D.G. (1983). Microwaves (2,450 MHz) suppress murine natural killer cell activity. *Bioelectromagnetics* **4**, 371-381.
- Smith, P., Kuster, N., Ebert, S., and Chevalier, H.J. (2007). GSM and DCS wireless communication signals: Combined chronic toxicity/carcinogenicity study in the Wistar rat. *Radiat. Res.* **168**, 480-492.
- Sokolovic, D., Djindjic, B., Nikolic, J., Bjelakovic, G., Pavlovic, D., Kocic, G., Krstic, D., Cvetkovic, T., and Pavlovic, V. (2008). Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain. *J. Radiat. Res.* **49**, 579-586.
- Sommer, A.M., Streckert, J., Bitz, A.K., Hansen, V.W., and Lerchl, A. (2004). No effects of GSM-modulated 900 MHz electromagnetic fields on survival rate and spontaneous development of lymphoma in female AKR/J mice. *BMC Cancer* **4**, 77.
- Sommer, A.M., Bitz, A.K., Streckert, J., Hansen, V.W., and Lerchl, A. (2007). Lymphoma development in mice chronically exposed to UMTS-modulated radiofrequency electromagnetic fields. *Radiat. Res.* **168**, 72-80.
- Sonmez, O.F., Odaci, E., Bas, O., and Kaplan, S. (2010). Purkinje cell number decreases in the adult female rat cerebellum following exposure to 900 MHz electromagnetic field. *Brain. Res.* **1356**, 95-101.
- Speit, G., and Schütz, P. (2013). Hyperthermia-induced genotoxic effects in human A549 cells. *Mutat. Res.* **747-748**, 1-5.
- Speit, G., Schütz, P., and Hoffmann, H. (2007). Genotoxic effects of exposure to radiofrequency electromagnetic fields (RF-EMF) in cultured mammalian cells are not independently reproducible. *Mutat. Res.* **626**, 42-47.
- Speit, G., Gminski, R., and Tauber, R. (2013). Genotoxic effects of exposure to radiofrequency electromagnetic fields (RF-EMF) in HL-60 cells are not reproducible. *Mutat. Res.* **755**, 163-166.

- Stagg, R.B., Hawel, L.H., III, Pastorian, K., Cain, C., Adey, W.R., and Byus, C.V. (2001). Effect of immobilization and concurrent exposure to a pulse-modulated microwave field on core body temperature, plasma ACTH and corticosteroid, and brain ornithine decarboxylase, Fos and Jun mRNA. *Radiat. Res.* **155**, 584-592.
- Stang, A., Schmidt-Pokrzywniak, A., Lash, T.L., Lommatzsch, P.K., Taubert, G., Bornfeld, N., and Jöckel, K.H. (2009). Mobile phone use and risk of uveal melanoma: Results of the risk factors for uveal melanoma case-control study. *J. Natl. Cancer Inst.* **101**, 120-123.
- Straus, D.S. (1981). Somatic mutation, cellular differentiation, and cancer causation. *JNCI* **67**, 233-241.
- Takebayashi, T., Varsier, N., Kikuchi, Y., Wake, K., Taki, M., Watanabe, S., Akiba, S., and Yamaguchi, N. (2008). Mobile phone use, exposure to radiofrequency electromagnetic field, and brain tumour: A case-control study. *Br. J. Cancer* **12**, 652-659.
- Tarone, R.E. (1975). Tests for trend in life table analysis. *Biometrika* **62**, 679-682.
- Tice, R.R., Agurell, E., Anderson, D., Burlinson, B., Hartmann, A., Kobayashi, H., Miyamae, Y., Rojas, E., Ryu, J.-C., and Sasaki, Y.F. (2000). Single cell gel/Comet assay: Guidelines for *in vitro* and *in vivo* genetic toxicology testing. *Environ. Mol. Mutagen.* **35**, 206-221.
- Tillmann, T., Ernst, H., Ebert, S., Kuster, N., Behnke, W., Rittinghausen, S., and Dasenbrock, C. (2007). Carcinogenicity study of GSM and DCS wireless communication signals in B6C3F1 mice. *Bioelectromagnetics* **28**, 173-187.
- Tomruk, A., Güler, G., and Dincel, A.S. (2010). The influence of 1800 MHz GSM-like signals on hepatic oxidative DNA and lipid damage in nonpregnant, pregnant, and newly born rabbits. *Cell Biochem. Biophys.* **56**, 39-47.
- Tukey, J.W. (1977). Easy summaries – numerical and graphical. In *Exploratory Data Analysis*, pp. 43-44. Addison-Wesley, Reading, MA.
- United States Code (USC) **42** §4321 *et seq.*
- Utteridge, T.D., Gebski, V., Finnie, J.W., Vernon-Roberts, B., and Kuchel, T.R. (2002). Long-term exposure of $\text{E}\mu\text{-Pim}1$ transgenic mice to 898.4 MHz microwaves does not increase lymphoma incidence. *Radiat. Res.* **158**, 357-364.
- Verschaeve, L., Juutilainen, J., Lagroye, I., Miyakoshi, J., Saunders, R., de Seze, R., Tenforde, T., van Rongen, E., Veyret, B., and Xu, Z. (2010). *In vitro* and *in vivo* genotoxicity of radiofrequency fields. *Mutat. Res.* **705**, 252-268.
- Veyret, B., Bouthet, C., Deschaux, P., de Seze, R., Geffard, M., Joussot-Dubien, J., le Diraison, M., Moreau, J.M., and Caristan, A. (1991). Antibody responses of mice exposed to low-power microwaves under combined, pulse-and-amplitude modulation. *Bioelectromagnetics* **12**, 47-56.
- Vijayalaxmi, and Prihoda, T.J. (2012). Genetic damage in human cells exposed to non-ionizing radiofrequency fields: A meta-analysis of the data from 88 publications (1990-2011). *Mutat. Res.* **749**, 1-16.
- Volkow, N.D., Tomasi, D., Wang, G.J., Vaska, P., Fowler, J.S., Telang, F., Alexoff, D., Logan, J., and Wong, C. (2011). Effects of cell phone radiofrequency signal exposure on brain glucose metabolism. *JAMA* **305**, 808-813.
- Wagner, P., Röschke, J., Mann, K., Hiller, W., and Frank, C. (1998). Human sleep under the influence of pulsed radiofrequency electromagnetic fields: A polysomnographic study using standardized conditions. *Bioelectromagnetics* **19**, 199-202.
- Wagner, P., Röschke, J., Mann, K., Fell, J., Hiller, W., Frank, C., and Grözinger, M. (2000). Human sleep EEG under the influence of pulsed radio frequency electromagnetic fields. Results from polysomnographies using submaximal high power flux densities. *Neuropsychobiology* **42**, 207-212.

Williams, D.A. (1971). A test for differences between treatment means when several dose levels are compared with a zero dose control. *Biometrics* **27**, 103-117.

Williams, D.A. (1972). The comparison of several dose levels with a zero dose control. *Biometrics* **28**, 519-531.

Williams, D.A. (1986). A note on Shirley's nonparametric test for comparing several dose levels with a zero-dose control. *Biometrics* **42**, 183-186.

Witt, K.L., Knapton, A., Wehr, C.M., Hook, G.J., Mirsalis, J., Shelby, M.D., and MacGregor, J.T. (2000). Micronucleated erythrocyte frequency in peripheral blood of B6C3F1 mice from short-term, prechronic, and chronic studies of the NTP Carcinogenesis Bioassay Program. *Environ. Mol. Mutagen.* **36**, 163-194.

Witt, K.L., Livanos, E., Kissling, G.E., Torous, D.K., Caspary, W., Tice, R.R., and Recio, L. (2008). Comparison of flow cytometry- and microscopy-based methods for measuring micronucleated reticulocyte frequencies in rodents treated with nongenotoxic and genotoxic chemicals. *Mutat. Res.* **649**, 101-113.

Wyde, M., Cesta, M., Blystone, C., Elmore, S., Foster, P., Hooth, M., Kissling, G., Malarkey, D., Stills, R., Stout, M., Walker, N., Witt, K., Wolfe, M., and Bucher, J. (2016). Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Cell Phone Radiofrequency Radiation in Hsd: Sprague Dawley® SD Rats (Whole Body Exposures). <<http://dx.doi.org/10.1101/055699>>

Wyde, M.E., Horn, T.L., Capstick, M.H., Ladbury, J.M., Koepke, G., Wilson, P.F., Kissling, G.E., Stout, M.D., Kuster, N., Melnick, R.L., Gauger, J., Bucher, J.R., and McCormick, D.L. (2018). Pilot Studies of the National Toxicology Program's Cell Phone Radiofrequency Radiation Reverberation Chamber Exposure System (publication pending).

Yakymenko, I., Tsybulin, O., Sidorik, E., Henshel, D., Kyrylenko, O., and Kyrylenko, S. (2016). Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation. *Electromagn. Biol. Med.* **35**, 186-202.

APPENDIX A

SUMMARY OF LESIONS IN MALE RATS

EXPOSED TO GSM-MODULATED CELL PHONE RFR FOR 2 YEARS

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TABLE A1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
<i>14-Week interim evaluation</i>	15	15	15	15
Early deaths				
Accidental deaths	1			1
Moribund	44	24	19	13
Natural deaths	20	21	21	16
Survivors				
Terminal euthanasia	25	45	50	60
Animals examined microscopically	100	100	100	100

Systems Examined at 14 Weeks with No Neoplasms Observed

Alimentary System
Cardiovascular System
Endocrine System
General Body System
Genital System
Hematopoietic System
Integumentary System
Musculoskeletal System
Nervous System
Respiratory System
Special Senses System
Urinary System

2-Year Study

Alimentary System

Esophagus	(90)	(89)	(90)	(90)
Intestine large, cecum	(75)	(75)	(79)	(80)
Intestine large, colon	(81)	(83)	(81)	(82)
Serosa, pheochromocytoma malignant, metastatic, adrenal medulla			1 (1%)	
Intestine large, rectum	(83)	(81)	(85)	(87)
Intestine small, duodenum	(81)	(82)	(79)	(79)
Adenocarcinoma		1 (1%)		1 (1%)
Intestine small, ileum	(78)	(76)	(78)	(76)
Intestine small, jejunum	(73)	(76)	(70)	(76)
Adenocarcinoma	2 (3%)	1 (1%)	1 (1%)	1 (1%)
Liver	(90)	(90)	(90)	(90)
Cholangioma		1 (1%)		
Hepatocellular adenoma		1 (1%)		2 (2%)
Hepatocellular carcinoma			1 (1%)	
Pheochromocytoma malignant, metastatic, adrenal medulla			1 (1%)	
Serosa, carcinoma, metastatic, intestine small, jejunum				1 (1%)
Mesentery	(39)	(19)	(17)	(7)
Pheochromocytoma malignant, metastatic, adrenal medulla			1 (6%)	
Oral mucosa	(0)	(2)	(0)	(2)
Squamous cell carcinoma		1 (50%)		1 (50%)

TABLE A1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Pancreas	(90)	(89)	(88)	(86)
Adenoma	13 (14%)	17 (19%)	14 (16%)	12 (14%)
Adenoma, multiple	5 (6%)	4 (4%)	4 (5%)	4 (5%)
Carcinoma	1 (1%)	1 (1%)	2 (2%)	
Carcinoma, metastatic, intestine small, jejunum				1 (1%)
Pheochromocytoma malignant, metastatic, adrenal medulla			1 (1%)	
Salivary glands	(90)	(90)	(90)	(90)
Schwannoma malignant		1 (1%)		
Sublingual gland, schwannoma malignant, metastatic, uncertain primary site	1 (1%)			
Stomach, forestomach	(90)	(90)	(90)	(90)
Sarcoma				1 (1%)
Squamous cell carcinoma				1 (1%)
Squamous cell papilloma	1 (1%)			
Stomach, glandular	(86)	(88)	(87)	(86)
Tooth	(0)	(1)	(1)	(0)
Odontoma			1 (100%)	
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Chemodectoma malignant		1 (1%)		
Blood vessel	(1)	(2)	(1)	(0)
Heart	(90)	(90)	(90)	(90)
Carcinoma, metastatic, adrenal cortex				1 (1%)
Chemodectoma malignant		1 (1%)		
Endocardium, schwannoma malignant	1 (1%)		1 (1%)	2 (2%)
Myocardium, schwannoma malignant	1 (1%)			3 (3%)
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(88)
Adenoma	1 (1%)	4 (4%)	2 (2%)	1 (1%)
Carcinoma		1 (1%)		1 (1%)
Adrenal medulla	(88)	(90)	(89)	(87)
Pheochromocytoma benign	8 (9%)	21 (23%)	19 (21%)	14 (16%)
Pheochromocytoma benign, multiple	1 (1%)		2 (2%)	
Pheochromocytoma, complex	1 (1%)			
Pheochromocytoma, malignant	1 (1%)	1 (1%)	4 (4%)	
Bilateral, pheochromocytoma benign	1 (1%)	2 (2%)	4 (4%)	
Bilateral, pheochromocytoma malignant		1 (1%)		
Islets, pancreatic	(90)	(89)	(86)	(85)
Adenoma	5 (6%)	12 (13%)	9 (10%)	10 (12%)
Adenoma, multiple		2 (1%)	1 (1%)	1 (1%)
Carcinoma	8 (9%)	13 (15%)	10 (12%)	5 (6%)
Carcinoma, multiple		2 (2%)		
Parathyroid gland	(83)	(87)	(87)	(81)
Adenoma	1 (1%)	1 (1%)		1 (1%)

TABLE A1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Endocrine System (continued)				
Pituitary gland	(89)	(90)	(90) 1 (1%)	(90)
Sarcoma, metastatic, eye				
Schawannoma malignant, metastatic, Harderian gland				1 (1%)
Schawannoma malignant, metastatic, trigeminal ganglion			1 (1%)	
Pars distalis, adenoma	17 (19%)	28 (31%)	25 (28%)	26 (29%)
Pars distalis, adenoma, multiple			1 (1%)	
Thyroid gland	(89)	(89)	(89)	(87)
Chemodectoma malignant, metastatic, tissue NOS		1 (1%)		
Bilateral, C-cell, adenoma			1 (1%)	1 (1%)
C-cell, adenoma	8 (9%)	9 (10%)	7 (8%)	7 (8%)
C-cell, carcinoma	2 (2%)	2 (2%)	2 (2%)	3 (3%)
Follicular cell, adenoma		1 (1%)	1 (1%)	
Follicular cell, adenoma, multiple		1 (1%)		
Follicular cell, carcinoma				1 (1%)
General Body System				
Tissue NOS	(3)	(4) 1 (25%)	(4)	(5)
Chemodectoma malignant		1 (25%)		
Abdominal, fat, hemangiosarcoma		1 (25%)		
Abdominal, fat, pheochromocytoma malignant, metastatic, adrenal medulla			1 (25%)	
Mediastinum, chemodectoma benign				1 (20%)
Mediastinum, schwannoma malignant	1 (33%)			
Genital System				
Bulbourethral gland	(1)	(0)	(0)	(0)
Coagulating gland	(0)	(0)	(0)	(1)
Ductus deferens	(1)	(0)	(0)	(0)
Epididymis	(90)	(90)	(90)	(90)
Penis	(0)	(0)	(0)	(1)
Preputial gland	(88)	(90)	(90)	(90)
Squamous cell carcinoma				1 (1%)
Prostate	(90)	(90)	(90)	(90)
Adenoma	2 (2%)	2 (2%)	6 (7%)	3 (3%)
Carcinoma			1 (1%)	
Seminal vesicle	(90)	(89)	(89)	(90)
Testis	(90)	(90)	(90)	(90)
Interstitial cell, adenoma	2 (2%)	2 (2%)	3 (3%)	2 (2%)

TABLE A1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hemangioma				1 (1%)
Lymph node	(25)	(22)	(18)	(12)
Mediastinal, pheochromocytoma malignant, metastatic, adrenal medulla				1 (6%)
Pancreatic, pheochromocytoma malignant, metastatic, adrenal medulla				1 (6%)
Lymph node, mandibular	(89)	(90)	(89)	(90)
Schwannoma, malignant, metastatic, salivary glands			1 (1%)	
Lymph node, mesenteric	(90)	(89)	(86)	(89)
Carcinoma, metastatic, intestine small, jejunum				1 (1%)
Hemangiosarcoma				1 (1%)
Spleen	(90)	(90)	(89)	(90)
Hemangiosarcoma	3 (3%)			1 (1%)
Capsule, carcinoma, metastatic, intestine small, jejunum				1 (1%)
Thymus	(88)	(86)	(88)	(86)
Alveolar/bronchiolar carcinoma, metastatic, lung		1 (1%)		
Thymoma benign		2 (2%)	1 (1%)	
Thymoma malignant				1 (1%)
Integumentary System				
Mammary gland	(82)	(76)	(82)	(82)
Adenocarcinoma			1 (1%)	
Fibroadenoma	2 (2%)	3 (4%)	4 (5%)	2 (2%)
Skin	(90)	(90)	(90)	(90)
Basal cell adenoma	1 (1%)	1 (1%)		1 (1%)
Basal cell carcinoma			1 (1%)	
Keratoacanthoma	5 (6%)	3 (3%)	9 (10%)	2 (2%)
Neural crest tumor			1 (1%)	
Sarcoma			1 (1%)	
Squamous cell papilloma	2 (2%)			
Sebaceous gland, adenoma	1 (1%)			
Subcutaneous tissue, fibroma	2 (2%)	5 (6%)	7 (8%)	5 (6%)
Subcutaneous tissue, fibrosarcoma	1 (1%)	3 (3%)	1 (1%)	2 (2%)
Subcutaneous tissue, hemangopericytoma			1 (1%)	
Subcutaneous tissue, hibernoma				1 (1%)
Subcutaneous tissue, lipoma	2 (2%)	3 (3%)	1 (1%)	2 (2%)
Subcutaneous tissue, malignant fibrous histiocytoma			1 (1%)	
Subcutaneous tissue, myxosarcoma			1 (1%)	
Subcutaneous tissue, sarcoma	2 (2%)	1 (1%)		1 (1%)
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Skeletal muscle	(90)	(90)	(90)	(90)
Diaphragm, carcinoma, metastatic, intestine small, jejunum				1 (1%)

TABLE A1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Nervous System				
Brain	(90)	(90)	(90)	(90)
Glioma malignant		3 (3%)	3 (3%)	2 (2%)
Sarcoma, metastatic, eye			1 (1%)	
Schwannoma malignant, metastatic, trigeminal ganglion			1 (1%)	
Meninges, granular cell tumor benign	1 (1%)	3 (3%)	3 (3%)	3 (3%)
Meninges, granular cell tumor malignant			1 (1%)	
Nerve trigeminal	(84)	(88)	(87)	(88)
Schwannoma malignant, metastatic, trigeminal ganglion			1 (1%)	
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Peripheral nerve, tibial	(88)	(89)	(90)	(88)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(75)	(73)	(77)	(77)
Sarcoma, metastatic, eye			1 (1%)	
Schwannoma malignant			1 (1%)	
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Alveolar/bronchiolar adenoma	1 (1%)	1 (1%)		
Alveolar/bronchiolar carcinoma		1 (1%)		
Carcinoma, metastatic, uncertain primary site		1 (1%)		
Sarcoma, metastatic, kidney				1 (1%)
Schwannoma malignant, metastatic, salivary glands		1 (1%)		
Nose	(89)	(90)	(90)	(89)
Sarcoma, metastatic, eye			1 (1%)	
Schwannoma malignant, metastatic, trigeminal ganglion			1 (1%)	
Schwannoma malignant, metastatic, uncertain primary site	1 (1%)			
Trachea	(90)	(88)	(87)	(86)
Chemodectoma malignant, metastatic, tissue NOS		1 (1%)		
Special Senses System				
Eye	(85)	(86)	(87)	(83)
Choroid, schwannoma malignant			1 (1%)	
Retrobulbar, sarcoma			1 (1%)	
Retrobulbar, schwannoma malignant, metastatic, trigeminal ganglion			1 (1%)	
Retrobulbar, schwannoma malignant, metastatic, uncertain primary site	2 (2%)		1 (1%)	
Harderian gland	(90)	(90)	(90)	(90)
Schwannoma malignant			1 (1%)	2 (2%)
Schwannoma malignant, metastatic, trigeminal ganglion			1 (1%)	
Schwannoma malignant, metastatic, uncertain primary site	2 (2%)		1 (1%)	
Lacrimal gland	(2)	(1)	(2)	(2)

TABLE A1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Urinary System				
Kidney	(90)	(90)	(90)	(90)
Hemangioma		1 (1%)		
Lipoma	1 (1%)	1 (1%)		
Oncocytoma benign	1 (1%)		1 (1%)	
Sarcoma			1 (1%)	1 (1%)
Bilateral, renal tubule, adenoma				1 (1%)
Bilateral, renal tubule, adenoma, multiple	1 (1%)			
Bilateral, renal tubule, carcinoma	1 (1%)			1 (1%)
Renal tubule, adenoma	1 (1%)	1 (1%)	3 (3%)	1 (1%)
Renal tubule, adenoma, multiple	1 (1%)			
Ureter	0	1	0	0
Urethra	0	0	1	0
Transitional epithelium, papilloma			1 (100%)	
Urinary bladder	(89)	(89)	(86)	(85)
Carcinoma, metastatic, prostate			1 (1%)	
Systemic Lesions				
Multiple organs ^b	(90)	(90)	(90)	(90)
Leukemia mononuclear				1 (1%)
Lymphoma malignant	2 (2%)	4 (4%)	4 (4%)	2 (2%)
Mesothelioma malignant	2 (2%)	1 (1%)	6 (7%)	1 (1%)
Neoplasm Summary				
Total animals with primary neoplasms ^c				
2-Year study	56	73	78	71
Total primary neoplasms	114	176	179	141
Total animals with benign neoplasms				
2-Year study	49	68	71	57
Total benign neoplasms	87	132	131	104
Total animals with malignant neoplasms	23	36	38	34
2-Year study	27	44	47	37
Total malignant neoplasms	2	4	5	3
2-Year study	6	6	20	8
Total animals with malignant neoplasms- uncertain primary site				
2-Year study	2	1	1	
Total animals with uncertain neoplasms- benign or malignant			1	
2-Year study				1
Total uncertain neoplasms				
2-Year study				1

^a Number of animals examined microscopically at the site and the number of animals with neoplasm

^b Number of animals with any tissue examined microscopically

^c Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE A2
Statistical Analysis of Primary Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Medulla: Benign Pheochromocytoma				
Overall rate ^a	10/88 (11%)	23/90 (26%)	25/89 (28%)	14/87 (16%)
Rate per litters ^b	8/35 (23%)	19/35 (54%)	21/35 (60%)	12/35 (34%)
Adjusted rate ^c	15.2%	29.9%	31.7%	18.3%
Terminal rate ^d	3/23 (13%)	13/45 (29%)	14/49 (29%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted poly-3 test ^e	P=0.472N	P=0.030	P=0.017	P=0.384
Litter C-A/Fisher's test ^f	P=0.365	P=0.007	P=0.002	P=0.214
Adrenal Medulla: Benign, Complex, or Malignant Pheochromocytoma				
Overall rate	11/88 (13%)	24/90 (27%)	28/89 (31%)	14/87 (16%)
Rate per litters	9/35 (26%)	19/35 (54%)	23/35 (66%)	12/35 (34%)
Adjusted rate	16.7%	31.1%	35.3%	18.3%
Terminal rate	3/23 (13%)	13/45 (29%)	15/49 (31%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted poly-3 test	P=0.409N	P=0.035	P=0.010	P=0.472
Litter C-A/Fisher's test	P=0.420	P=0.014	P<0.001	P=0.301
Heart: Malignant Schwannoma				
Overall rate	0/90 (0%)	2/90 (2%)	1/90 (1%)	5/90 (6%)
Rate per litters	0/35 (0%)	2/35 (6%)	1/35 (3%)	5/35 (14%)
Adjusted rate	0%	2.7%	1.3%	6.4%
Terminal rate	0/25 (0%)	2/45 (4%)	1/50 (2%)	3/60 (5%)
First incidence (days)	— ^g	730 (T)	730 (T)	582
Rao-Scott adjusted poly-3 test	P=0.041	P=0.297	P=0.540	P=0.080
Litter C-A/Fisher's test	P=0.013	P=0.246	P=0.500	P=0.027
Mammary Gland: Fibroadenoma or Adenocarcinoma				
Overall rate	2/90 (2%)	3/90 (3%)	5/90 (6%)	2/90 (2%)
Rate per litters	2/35 (6%)	3/35 (9%)	5/35 (14%)	2/35 (6%)
Adjusted rate	3%	4%	6.3%	2.6%
Terminal rate	1/25 (4%)	0/45 (0%)	3/50 (6%)	1/60 (2%)
First incidence (days)	440	320	454	544
Rao-Scott adjusted poly-3 test	P=0.504N	P=0.536	P=0.289	P=0.604N
Litter C-A/Fisher's test	P=0.581	P=0.500	P=0.214	P=0.693
Pancreas: Adenoma				
Overall rate	18/90 (20%)	21/89 (24%)	18/88 (20%)	16/86 (19%)
Rate per litters	16/35 (46%)	17/35 (49%)	13/34 (38%)	13/35 (37%)
Adjusted rate	26.8%	27.6%	23.5%	21.3%
Terminal rate	9/25 (36%)	13/45 (29%)	15/50 (30%)	14/60 (23%)
First incidence (days)	580	614	716	674
Rao-Scott adjusted poly-3 test	P=0.216N	P=0.523	P=0.396N	P=0.292N
Litter C-A/Fisher's test	P=0.204N	P=0.500	P=0.350N	P=0.314N
Pancreas: Adenoma or Carcinoma				
Overall rate	18/90 (20%)	22/89 (25%)	19/88 (22%)	16/86 (19%)
Rate per litters	16/35 (46%)	18/35 (51%)	14/34 (41%)	13/35 (37%)
Adjusted rate	26.8%	28.9%	24.7%	21.3%
Terminal rate	9/25 (36%)	13/45 (29%)	15/50 (30%)	14/60 (23%)
First incidence (days)	580	614	677	674
Rao-Scott adjusted poly-3 test	P=0.201N	P=0.459	P=0.457N	P=0.288N
Litter C-A/Fisher's test	P=0.189N	P=0.406	P=0.446N	P=0.314N

TABLE A2
**Statistical Analysis of Primary Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	14/89 (16%)	10/86 (12%)	11/85 (13%)
Rate per litters	5/35 (14%)	12/35 (34%)	9/34 (26%)	11/35 (31%)
Adjusted rate	7.6%	18.5%	13.2%	14.8%
Terminal rate	2/25 (8%)	10/45 (22%)	9/50 (18%)	11/60 (18%)
First incidence (days)	624	531	677	730 (T)
Rao-Scott adjusted poly-3 test	P=0.282	P=0.051	P=0.204	P=0.140
Litter C-A/Fisher's test	P=0.141	P=0.046	P=0.169	P=0.077
Pancreatic Islets: Carcinoma				
Overall rate	8/90 (9%)	15/89 (17%)	10/86 (12%)	5/85 (6%)
Rate per litters	8/35 (23%)	12/35 (34%)	10/34 (29%)	4/35 (11%)
Adjusted rate	12%	19.7%	13.1%	6.7%
Terminal rate	3/25 (12%)	7/45 (16%)	8/50 (16%)	4/60 (7%)
First incidence (days)	663	531	537	544
Rao-Scott adjusted poly-3 test	P=0.088N	P=0.173	P=0.517	P=0.220N
Litter C-A/Fisher's test	P=0.083N	P=0.214	P=0.365	P=0.171N
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	13/90 (14%)	27/89 (30%)	19/86 (22%)	16/85 (19%)
Rate per litters	12/35 (34%)	19/35 (54%)	17/34 (50%)	14/35 (40%)
Adjusted rate	19.4%	35.2%	24.8%	21.3%
Terminal rate	5/25 (20%)	16/45 (36%)	16/50 (32%)	15/60 (25%)
First incidence (days)	624	531	537	544
Rao-Scott adjusted poly-3 test	P=0.344N	P=0.032	P=0.282	P=0.462
Litter C-A/Fisher's test	P=0.518	P=0.074	P=0.140	P=0.402
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	17/89 (19%)	28/90 (31%)	26/90 (29%)	26/90 (29%)
Rate per litters	13/35 (37%)	23/35 (66%)	19/35 (54%)	22/35 (63%)
Adjusted rate	24.9%	35.2%	32.2%	32.4%
Terminal rate	5/25 (20%)	15/45 (33%)	17/50 (34%)	19/60 (32%)
First incidence (days)	527	309	537	384
Rao-Scott adjusted poly-3 test	P=0.301	P=0.126	P=0.216	P=0.210
Litter C-A/Fisher's test	P=0.064	P=0.015	P=0.115	P=0.028
Prostate Gland: Adenoma				
Overall rate	2/90 (2%)	2/90 (2%)	6/90 (7%)	3/90 (3%)
Rate per litters	2/35 (6%)	2/35 (6%)	5/35 (14%)	3/35 (9%)
Adjusted rate	3%	2.7%	7.7%	3.9%
Terminal rate	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	730 (T)	730 (T)
Rao-Scott adjusted poly-3 test	P=0.419	P=0.625N	P=0.224	P=0.566
Litter C-A/Fisher's test	P=0.342	P=0.693	P=0.214	P=0.500
Prostate Gland: Adenoma or Carcinoma				
Overall rate	2/90 (2%)	2/90 (2%)	7/90 (8%)	3/90 (3%)
Rate per litters	2/35 (6%)	2/35 (6%)	6/35 (17%)	3/35 (9%)
Adjusted rate	3%	2.7%	9%	3.9%
Terminal rate	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	717	730 (T)
Rao-Scott adjusted poly-3 test	P=0.412	P=0.626N	P=0.161	P=0.566
Litter C-A/Fisher's test	P=0.329	P=0.693	P=0.130	P=0.500

TABLE A2
Statistical Analysis of Primary Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Skin: Keratoacanthoma				
Overall rate	5/90 (6%)	3/90 (3%)	9/90 (10%)	2/90 (2%)
Rate per litters	5/35 (14%)	3/35 (9%)	7/35 (20%)	2/35 (6%)
Adjusted rate	7.4%	4%	11.6%	2.6%
Terminal rate	0/25 (0%)	2/45 (4%)	9/50 (18%)	2/60 (3%)
First incidence (days)	552	694	730 (T)	730 (T)
Rao-Scott adjusted poly-3 test	P=0.271N	P=0.332N	P=0.322	P=0.208N
Litter C-A/Fisher's test	P=0.256N	P=0.355N	P=0.376	P=0.214N
Skin: Squamous Cell Papilloma or Keratoacanthoma				
Overall rate	7/90 (8%)	3/90 (3%)	9/90 (10%)	2/90 (2%)
Rate per litters	7/35 (20%)	3/35 (9%)	7/35 (20%)	2/35 (6%)
Adjusted rate	10.4%	4%	11.6%	2.6%
Terminal rate	1/25 (4%)	2/45 (4%)	9/50 (18%)	2/60 (3%)
First incidence (days)	552	694	730 (T)	730 (T)
Rao-Scott adjusted poly-3 test	P=0.145N	P=0.164N	P=0.520	P=0.088N
Litter C-A/Fisher's test	P=0.113N	P=0.153N	P=0.617	P=0.075N
Skin: Squamous Cell Papilloma, Keratoacanthoma, Basal Cell Adenoma, or Basal Cell Carcinoma				
Overall rate	8/90 (9%)	4/90 (4%)	10/90 (11%)	3/90 (3%)
Rate per litters	8/35 (23%)	4/35 (11%)	8/35 (23%)	3/35 (9%)
Adjusted rate	11.9%	5.3%	12.8%	3.9%
Terminal rate	2/25 (8%)	3/45 (7%)	10/50 (20%)	2/60 (3%)
First incidence (days)	552	694	730 (T)	694
Rao-Scott test	P=0.145N	P=0.163N	P=0.526	P=0.090N
Litter C-A/Fisher's test	P=0.132N	P=0.171N	P=0.612	P=0.094N
Skin (Subcutaneous Tissue): Fibroma				
Overall rate	2/90 (2%)	5/90 (6%)	7/90 (8%)	5/90 (6%)
Rate per litters	2/35 (6%)	5/35 (14%)	7/35 (20%)	5/35 (14%)
Adjusted rate	3.1%	6.7%	8.9%	6.4%
Terminal rate	2/25 (8%)	3/45 (7%)	4/50 (8%)	2/60 (3%)
First incidence (days)	730 (T)	673	501	443
Rao-Scott test	P=0.306	P=0.256	P=0.129	P=0.277
Litter C-A/Fisher's test	P=0.214	P=0.214	P=0.075	P=0.214
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, Myxosarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	9/90 (10%)	10/90 (11%)	8/90 (9%)
Rate per litters	5/35 (14%)	9/35 (26%)	10/35 (29%)	8/35 (23%)
Adjusted rate	7.5%	11.7%	12.6%	10%
Terminal rate	2/25 (8%)	4/45 (9%)	5/50 (10%)	2/60 (3%)
First incidence (days)	567	291	501	443
Rao-Scott adjusted poly-3 test	P=0.428	P=0.268	P=0.218	P=0.383
Litter C-A/Fisher's test	P=0.293	P=0.185	P=0.122	P=0.270
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, Myxosarcoma, Malignant Fibrous Histiocytoma, or Hemangiopericytoma				
Overall rate	5/90 (6%)	9/90 (10%)	11/90 (12%)	8/90 (9%)
Rate per litters	5/35 (14%)	9/35 (26%)	11/35 (31%)	8/35 (23%)
Adjusted rate	7.5%	11.7%	13.8%	10%
Terminal rate	2/25 (8%)	4/45 (9%)	6/50 (12%)	2/60 (3%)
First incidence (days)	567	291	501	443
Rao-Scott adjusted poly-3 test	P=0.421	P=0.268	P=0.159	P=0.383
Litter C-A/Fisher's test	P=0.284	P=0.185	P=0.077	P=0.270

TABLE A2
**Statistical Analysis of Primary Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Thyroid Gland (C-cell): Adenoma				
Overall rate	8/89 (9%)	9/89 (10%)	8/89 (9%)	8/87 (9%)
Rate per litters	7/35 (20%)	8/35 (23%)	8/34 (24%)	8/35 (23%)
Adjusted rate	12.1%	12%	10.3%	10.6%
Terminal rate	6/25 (24%)	8/45 (18%)	6/50 (12%)	8/60 (13%)
First incidence (days)	498	576	636	730 (T)
Rao-Scott adjusted poly-3 test	P=0.416N	P=0.581N	P=0.460N	P=0.481N
Litter C-A/Fisher's test	P=0.456	P=0.500	P=0.474	P=0.500
Thyroid Gland (C-cell): Adenoma or Carcinoma				
Overall rate	10/89 (11%)	11/89 (12%)	10/89 (11%)	11/87 (13%)
Rate per litters	8/35 (23%)	8/35 (23%)	9/34 (26%)	11/35 (31%)
Adjusted rate	14.9%	14.6%	12.9%	14.5%
Terminal rate	6/25 (24%)	8/45 (18%)	8/50 (16%)	11/60 (18%)
First incidence (days)	498	576	636	730 (T)
Rao-Scott adjusted poly-3 test	P=0.512N	P=0.566N	P=0.457N	P=0.561N
Litter C-A/Fisher's test	P=0.214	P=0.612	P=0.472	P=0.296
All Organs: Malignant Schwannoma				
Overall rate	3/90 (3%)	3/90 (3%)	5/90 (6%)	7/90 (8%)
Rate per litters	3/35 (9%)	3/35 (9%)	5/35 (14%)	7/35 (20%)
Adjusted rate	4.5%	4%	6.4%	8.9%
Terminal rate	1/25 (4%)	2/45 (4%)	3/50 (6%)	4/60 (7%)
First incidence (days)	555	720	661	582
Rao-Scott adjusted poly-3 test	P=0.133	P=0.577N	P=0.435	P=0.238
Litter C-A/Fisher's test	P=0.073	P=0.663	P=0.355	P=0.153
All Organs: Malignant Mesothelioma				
Overall rate	2/90 (2%)	1/90 (1%)	6/90 (7%)	1/90 (1%)
Rate per litters	2/35 (6%)	1/35 (3%)	6/35 (17%)	1/35 (3%)
Adjusted rate	3%	1.3%	7.6%	1.3%
Terminal rate	1/25 (4%)	0/45 (0%)	1/50 (2%)	1/60 (2%)
First incidence (days)	645	705	550	730 (T)
Rao-Scott adjusted poly-3 test	P=0.485N	P=0.454N	P=0.228	P=0.446N
Litter C-A/Fisher's test	P=0.544N	P=0.500N	P=0.130	P=0.500N

TABLE A2
**Statistical Analysis of Primary Neoplasms in Male Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

(T) Terminal euthanasia

- a Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, heart, pancreas, pancreatic islets, pituitary gland, prostate gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
- b Number of litters with tumor-bearing animals/number of litters examined at site
- c Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
- d Observed incidence at terminal euthanasia
- e Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott adjusted poly-3 test is a modification of the Poly-3 test that also incorporates an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
- f The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
- g Not applicable; no neoplasms in animal group

TABLE A3a
Historical Incidence of Malignant Schwannoma of the Heart in Control Male Hsd:Sprague Dawley SD Rats^a

		Incidence in Controls
Overall Historical Incidence: All Routes		
Total (%)		2/240 (0.8%)
Mean ± standard deviation		1.0% ± 1.2%
Range		0%-2%

^a Data as of November 2017

TABLE A3b
Historical Incidence of Prostate Gland Neoplasms in Control Male Hsd:Sprague Dawley SD Rats^a

	Adenoma	Carcinoma	Adenoma or Carcinoma
Overall Historical Incidence: All Routes			
Total (%)	2/240 (0.8%)	0/240	2/240 (0.8%)
Mean ± standard deviation	0.6% ± 1.1%		0.6% ± 1.1%
Range	0%-2%		0%-2%

^a Data as of November 2017

TABLE A3c
Historical Incidence of Brain Neoplasms in Control Male Hsd:Sprague Dawley SD Rats^a

	Malignant Glioma	Benign Granular Cell Tumor	Malignant Granular Cell Tumor	Benign or Malignant Granular Cell Tumor
Overall Historical Incidence: All Routes				
Total (%)	2/190 (1.3%)	3/190 (1.7%)	0/240	3/190 (1.7%)
Mean ± standard deviation	1.3% ± 2.3%	1.7% ± 2.1%		1.7% ± 2.1%
Range	0%-4%	0%-4%		0%-4%

^a Data as of November 2017

TABLE A3d
Historical Incidence of Pituitary Gland (Pars Distalis) Adenoma
in Control Male Hsd:Sprague Dawley SD Rats^a

		Incidence in Controls
Overall Historical Incidence: All Routes		
Total (%)		47/239 (19.7%)
Mean ± standard deviation		19.8% ± 7.5%
Range		10%-28%

^a Data as of November 2017

TABLE A3e
Historical Incidence of Adrenal Medulla Neoplasms in Control Male Hsd:Sprague Dawley SD Rats^a

Benign Pheochromocytoma	Malignant Pheochromocytoma	Complex Pheochromocytoma	Benign, Malignant, or Complex Pheochromocytoma
Overall Historical Incidence: All Routes			
Total (%)	36/238 (15.1%)	8/238 (3.4%)	2/238 (0.8%)
Mean ± standard deviation	15.8% ± 6.5%	3.8% ± 2.0%	0.8% ± 1.0%
Range	10%-24%	1%-6%	0%-2%
			45/238 (18.9%)
			20.1% ± 7.1%
			13%-28%

^a Data as of November 2017

TABLE A3f
Historical Incidence of Pancreatic Islet Neoplasms in Control Male Hsd:Sprague Dawley SD Rats^a

Adenoma	Carcinoma	Adenoma or Carcinoma
Overall Historical Incidence: All Routes		
Total (%)	18/240 (7.5%)	8/240 (3.3%)
Mean ± standard deviation	7.9% ± 5.5%	2.2% ± 4.4%
Range	4%-16%	0%-9%
		26/240 (10.8%)
		10.1% ± 6.0%
		4%-16%

^a Data as of November 2017

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental deaths	1			1
Moribund	44	24	19	13
Natural deaths	20	21	21	16
Survivors				
Terminal euthanasia	25	45	50	60
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(10)	(10)
Intestine large, cecum	(10)	(10)	(10)	(10)
Intestine large, colon	(10)	(10)	(10)	(10)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)	1 (10%)		
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(10)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell	1 (10%)		3 (30%)	3 (30%)
Pancreas	(10)	(10)	(10)	(10)
Salivary glands	(10)	(10)	(10)	(10)
Stomach, forestomach	(10)	(10)	(10)	(10)
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy	2 (20%)	4 (40%)	3 (30%)	4 (40%)
Ventricle right, cardiomyopathy	1 (10%)	1 (10%)	5 (50%)	5 (50%)
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(9)	(10)	(10)	(8)
Pituitary gland	(10)	(10)	(10)	(10)
Pars distalis, cyst		1 (10%)	2 (20%)	2 (20%)
Rathke's cleft, cyst		1 (10%)	3 (30%)	3 (30%)
Thyroid gland	(10)	(10)	(10)	(10)
Ectopic thymus		1 (10%)	1 (10%)	2 (20%)
Genital System				
Epididymis	(10)	(10)	(10)	(10)
Preputial gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	7 (70%)	3 (30%)	1 (10%)	4 (40%)

^a Number of animals examined microscopically at the site and the number of animals with lesion

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
14-Week Interim Evaluation (continued)				
Genital System (continued)				
Prostate	(10)	(10)	(10)	(10)
Inflammation, chronic active		1 (10%)	1 (10%)	
Seminal vesicle	(10)	(10)	(10)	(10)
Testis	(10)	(10)	(10)	(10)
Germ cell, degeneration				1 (10%)
Hematopoietic System				
Bone marrow	(10)	(10)	(10)	(10)
Lymph node, mandibular	(10)	(10)	(10)	(10)
Hemorrhage	2 (20%)	2 (20%)	2 (20%)	1 (10%)
Hyperplasia, lymphocyte				4 (40%)
Proliferation, plasma cell				2 (20%)
Lymph node, mesenteric	(10)	(10)	(10)	(10)
Hemorrhage		2 (20%)		
Spleen	(10)	(10)	(10)	(10)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	5 (50%)	1 (10%)	2 (20%)	
Musculoskeletal System				
Bone	(10)	(10)	(10)	(10)
Tibia, fracture, chronic				1 (10%)
Skeletal muscle	(10)	(10)	(10)	(10)
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion			1 (10%)	
Inflammation, chronic active	1 (10%)	1 (10%)		
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(10)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Retina, developmental malformation				1 (10%)
Harderian gland	(10)	(10)	(10)	(10)
Inflammation, chronic active		3 (30%)	2 (20%)	2 (20%)
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Congestion			1 (10%)	
Nephropathy, chronic progressive	9 (90%)	8 (80%)	8 (80%)	7 (70%)
Pelvis, dilation			1 (10%)	
Renal tubule, dilation		1 (10%)		
Urinary bladder	(10)	(10)	(10)	(10)
Systems Examined with No Lesions Observed				
General Body System				
Integumentary System				
Nervous System				

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study				
Alimentary System				
Esophagus	(90)	(89)	(90)	(90)
Dilation	2 (2%)			
Hyperplasia	1 (1%)			
Arteriole, inflammation, chronic active		1 (1%)		
Artery, inflammation, chronic active			1 (1%)	
Intestine large, cecum	(75)	(75)	(79)	(80)
Edema	11 (15%)	1 (1%)		4 (5%)
Erosion	10 (13%)			3 (4%)
Inflammation, acute	10 (13%)	1 (1%)		2 (3%)
Inflammation, chronic active	1 (1%)			
Ulcer	6 (8%)			
Artery, inflammation, chronic active	20 (27%)	9 (12%)	5 (6%)	6 (8%)
Artery, mineral	1 (1%)			
Epithelium, erosion				1 (1%)
Epithelium, regeneration	14 (19%)			2 (3%)
Intestine large, colon	(81)	(83)	(81)	(82)
Edema		1 (1%)		1 (1%)
Erosion	1 (1%)			1 (1%)
Inflammation, acute	1 (1%)			
Ulcer	1 (1%)			
Artery, inflammation, chronic active	12 (15%)	5 (6%)	5 (6%)	5 (6%)
Artery, mineral	2 (2%)			
Epithelium, regeneration	5 (6%)			2 (2%)
Intestine large, rectum	(83)	(81)	(85)	(87)
Cyst			1 (1%)	
Edema	1 (1%)			1 (1%)
Erosion	1 (1%)			
Hyperplasia, lymphocyte	1 (1%)	1 (1%)		
Inflammation, acute	2 (2%)			
Artery, inflammation, chronic active	4 (5%)	7 (9%)	4 (5%)	2 (2%)
Epithelium, regeneration	3 (4%)			
Intestine small, duodenum	(81)	(82)	(79)	(79)
Dilation			1 (1%)	
Erosion	1 (1%)			
Ulcer	1 (1%)			
Intestine small, ileum	(78)	(76)	(78)	(76)
Artery, inflammation, chronic active	2 (3%)	1 (1%)		
Epithelium, regeneration	1 (1%)			
Intestine small, jejunum	(73)	(76)	(70)	(76)
Dilation			1 (1%)	
Liver	(90)	(90)	(90)	(90)
Angiectasis	1 (1%)		1 (1%)	
Basophilic focus	1 (1%)	1 (1%)		
Clear cell focus	8 (9%)	7 (8%)	22 (24%)	16 (18%)
Eosinophilic focus	12 (13%)	5 (6%)	2 (2%)	8 (9%)
Extramedullary hematopoiesis	5 (6%)	4 (4%)	1 (1%)	4 (4%)
Hepatodiaphragmatic nodule	1 (1%)		2 (2%)	2 (2%)
Infiltration cellular, mixed cell	3 (3%)	2 (2%)		5 (6%)
Mixed cell focus	32 (36%)	45 (50%)	50 (56%)	58 (64%)
Artery, inflammation, chronic active	2 (2%)	5 (6%)	1 (1%)	
Artery, mineral	1 (1%)			2 (2%)
Artery, thrombus		1 (1%)		
Bile duct, cyst	3 (3%)	3 (3%)	2 (2%)	
Bile duct, fibrosis				1 (1%)
Bile duct, hyperplasia	41 (46%)	35 (39%)	37 (41%)	33 (37%)
Centrilobular, hepatocyte, hypertrophy		1 (1%)		
Hepatocyte, degeneration	1 (1%)			

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Liver (continued)	(90)	(90)	(90)	(90)
Hepatocyte, degeneration, cystic			1 (1%)	
Hepatocyte, necrosis	5 (6%)	6 (7%)	8 (9%)	1 (1%)
Hepatocyte, vacuolization, cytoplasmic	6 (7%)	4 (4%)	9 (10%)	3 (3%)
Kupffer cell, pigment	1 (1%)	1 (1%)		1 (1%)
Periductal, cholangiofibrosis	2 (2%)	1 (1%)	1 (1%)	1 (1%)
Mesentery	(39)	(19)	(17)	(7)
Hemorrhage	1 (3%)			
Inflammation, chronic	2 (5%)			
Inflammation, chronic active		1 (5%)		
Necrosis	2 (5%)	3 (16%)	1 (6%)	
Neovascularization	1 (3%)	1 (5%)		
Arteriole, inflammation, chronic active		1 (5%)		
Artery, inflammation, chronic active	32 (82%)	12 (63%)	14 (82%)	5 (71%)
Artery, mineral	21 (54%)	4 (21%)	5 (29%)	2 (29%)
Vein, degeneration	1 (3%)			
Vein, inflammation, chronic active	1 (3%)	1 (5%)		1 (14%)
Oral mucosa	(0)	(2)	(0)	(2)
Hyperplasia				1 (50%)
Ulcer		1 (50%)		
Pancreas	(90)	(89)	(88)	(86)
Cyst	1 (1%)			
Inflammation, chronic active				1 (1%)
Thrombus	1 (1%)			
Acinus, atrophy	13 (14%)	16 (18%)	10 (11%)	11 (13%)
Acinus, hyperplasia	63 (70%)	58 (65%)	44 (50%)	32 (37%)
Artery, inflammation, chronic active	48 (53%)	28 (31%)	26 (30%)	14 (16%)
Artery, mineral	11 (12%)	3 (3%)	3 (3%)	1 (1%)
Salivary glands	(90)	(90)	(90)	(90)
Inflammation, chronic active		1 (1%)		
Artery, inflammation, chronic active	11 (12%)	7 (8%)	3 (3%)	1 (1%)
Artery, mineral	2 (2%)			
Duct, parotid gland, dilation	5 (6%)	3 (3%)	1 (1%)	4 (4%)
Duct, parotid gland, inflammation, acute	1 (1%)			
Parotid gland, atrophy	18 (20%)	16 (18%)	14 (16%)	14 (16%)
Parotid gland, inflammation, acute	2 (2%)	7 (8%)	3 (3%)	1 (1%)
Parotid gland, vacuolation, cytoplasmic	1 (1%)			
Sublingual gland, inflammation, acute			1 (1%)	
Submandibular gland, atrophy		1 (1%)	1 (1%)	
Stomach, forestomach	(90)	(90)	(90)	(90)
Cyst		1 (1%)		
Edema	5 (6%)	11 (12%)	3 (3%)	2 (2%)
Erosion		1 (1%)	1 (1%)	
Fibrosis				1 (1%)
Inflammation, acute	1 (1%)	1 (1%)		
Inflammation, chronic active	7 (8%)	14 (16%)	5 (6%)	6 (7%)
Mineral	1 (1%)		1 (1%)	
Necrosis				1 (1%)
Ulcer	6 (7%)	8 (9%)	3 (3%)	2 (2%)
Artery, inflammation, chronic active		4 (4%)	3 (3%)	
Epithelium, degeneration		1 (1%)		
Epithelium, hyperplasia	11 (12%)	21 (23%)	12 (13%)	11 (12%)
Epithelium, hyperplasia, atypical	1 (1%)			
Epithelium, hyperplasia, basal cell			1 (1%)	

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Stomach, glandular	(86)	(88)	(87)	(86)
Erosion	3 (3%)		2 (2%)	1 (1%)
Hemorrhage			1 (1%)	
Inflammation, granulomatous		1 (1%)		1 (1%)
Inflammation, acute	1 (1%)			
Inflammation, chronic active	1 (1%)	3 (3%)		
Mineral	31 (36%)	7 (8%)	8 (9%)	4 (5%)
Ulcer			1 (1%)	
Artery, inflammation, chronic active	3 (3%)	2 (2%)	2 (2%)	1 (1%)
Tooth	(0)	(1)	(1)	(0)
Dysplasia		1 (100%)		
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Aneurysm		1 (1%)		
Dilation		1 (1%)	3 (3%)	
Mineral	30 (33%)	7 (8%)	12 (13%)	6 (7%)
Blood vessel	(1)	(2)	(1)	(0)
Mineral	1 (100%)			
Pulmonary artery, inflammation, chronic active		1 (50%)	1 (100%)	
Pulmonary artery, necrosis		1 (50%)	1 (100%)	
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	79 (88%)	82 (91%)	78 (87%)	79 (88%)
Congestion	1 (1%)			
Hemorrhage				1 (1%)
Thrombus	1 (1%)			
Artery, infiltration cellular, histiocyte			1 (1%)	
Artery, inflammation, chronic active		5 (6%)	4 (4%)	2 (2%)
Artery, mineral	20 (22%)	7 (8%)	3 (3%)	2 (2%)
Artery, necrosis		1 (1%)		
Atrium, dilation	3 (3%)		1 (1%)	
Atrium, thrombus	1 (1%)	1 (1%)		
Atrium, myocardium, hypertrophy	1 (1%)			
Endocardium, hyperplasia, Schwann cell		1 (1%)		2 (2%)
Myocardium, mineral	9 (10%)	2 (2%)	4 (4%)	1 (1%)
Myocardium, necrosis	1 (1%)			
Valve, inflammation, chronic active	1 (1%)			
Ventricle right, cardiomyopathy	54 (60%)	62 (69%)	72 (80%)	74 (82%)
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(88)
Accessory adrenal cortical nodule	6 (7%)	7 (8%)	6 (7%)	4 (5%)
Angiectasis			1 (1%)	2 (2%)
Atrophy			1 (1%)	1 (1%)
Congestion			1 (1%)	
Degeneration	3 (3%)			
Degeneration, cystic			2 (2%)	2 (2%)
Hemorrhage			1 (1%)	
Hyperplasia	47 (52%)	46 (51%)	46 (51%)	45 (51%)
Hypertrophy	35 (39%)	43 (48%)	50 (56%)	54 (61%)
Necrosis	5 (6%)	3 (3%)	4 (4%)	
Thrombus	2 (2%)	1 (1%)		
Vacuolation, cytoplasmic	20 (22%)	32 (36%)	25 (28%)	22 (25%)

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Endocrine System (continued)				
Adrenal medulla	(88)	(90)	(89)	(87)
Degeneration, cystic		1 (1%)		
Hyperplasia	42 (48%)	24 (27%)	26 (29%)	35 (40%)
Thrombus	1 (1%)			
Islets, pancreatic	(90)	(89)	(86)	(85)
Hyperplasia	12 (13%)	5 (6%)	5 (6%)	7 (8%)
Parathyroid gland	(83)	(87)	(87)	(81)
Cyst				1 (1%)
Hyperplasia	51 (61%)	35 (40%)	46 (53%)	28 (35%)
Hyperplasia, focal			2 (2%)	1 (1%)
Pituitary gland	(89)	(90)	(90)	(90)
Necrosis			1 (1%)	
Craniopharyngeal duct, cyst	1 (1%)			
Pars distalis, cyst	5 (6%)	9 (10%)	15 (17%)	16 (18%)
Pars distalis, hyperplasia	32 (36%)	34 (38%)	35 (39%)	32 (36%)
Pars distalis, necrosis				1 (1%)
Pars intermedia, angiectasis	1 (1%)			
Pars intermedia, cyst	6 (7%)	5 (6%)	9 (10%)	6 (7%)
Pars intermedia, hyperplasia	1 (1%)	1 (1%)	2 (2%)	2 (2%)
Pars nervosa, cyst		1 (1%)		
Pars nervosa, developmental malformation			1 (1%)	1 (1%)
Pars nervosa, infiltration cellular, mixed cell				1 (1%)
Thyroid gland	(89)	(89)	(89)	(87)
Congestion				1 (1%)
Ectopic thymus			1 (1%)	
C-cell, hyperplasia	16 (18%)	24 (27%)	18 (20%)	14 (16%)
Follicle, cyst		1 (1%)	1 (1%)	1 (1%)
Follicle, hyperplasia, cystic	1 (1%)			
Follicular cell, hyperplasia		1 (1%)		
Follicular cell, hypertrophy		1 (1%)		1 (1%)
General Body System				
Tissues NOS	(3)	(4)	(4)	(5)
Inflammation, chronic active		1 (25%)		
Abdominal, fat, hemorrhage	1 (33%)			
Abdominal, fat, inflammation, chronic active		1 (25%)		
Fat, necrosis	2 (67%)		2 (50%)	3 (60%)
Mediastinum, inflammation, chronic active			1 (25%)	1 (20%)
Genital System				
Bulbourethral gland	(1)	(0)	(0)	(0)
Coagulating gland	(0)	(0)	(0)	(1)
Inflammation, chronic active				1 (100%)
Ductus deferens	(1)	(0)	(0)	(0)
Granuloma	1 (100%)			
Epididymis	(90)	(90)	(90)	(90)
Exfoliated germ cell	51 (57%)	26 (29%)	29 (32%)	15 (17%)
Granuloma sperm	1 (1%)			
Hypospermia	28 (31%)	20 (22%)	23 (26%)	8 (9%)
Inflammation, acute		1 (1%)		
Inflammation, chronic active		1 (1%)		
Artery, inflammation, chronic active	2 (2%)	2 (2%)	1 (1%)	2 (2%)

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Genital System (continued)				
Penis	(0)	(0)	(0)	(1)
Preputial gland	(88)	(90)	(90)	(90)
Atrophy	1 (1%)			
Hyperplasia	1 (1%)			
Inflammation, suppurative		1 (1%)	3 (3%)	
Inflammation, granulomatous	1 (1%)			
Inflammation, acute	1 (1%)			
Inflammation, chronic active	46 (52%)	48 (53%)	54 (60%)	52 (58%)
Artery, inflammation, chronic active	1 (1%)			1 (1%)
Duct, dilation	51 (58%)	53 (59%)	49 (54%)	51 (57%)
Duct, hyperplasia		1 (1%)		1 (1%)
Duct, mineral				1 (1%)
Prostate	(90)	(90)	(90)	(90)
Decreased secretory fluid	4 (4%)	6 (7%)	6 (7%)	2 (2%)
Hemorrhage	1 (1%)			
Inflammation cellular, mononuclear cell	1 (1%)			1 (1%)
Inflammation, suppurative		1 (1%)	1 (1%)	
Inflammation, acute	7 (8%)	3 (3%)	6 (7%)	4 (4%)
Inflammation, chronic active	6 (7%)	15 (17%)	9 (10%)	13 (14%)
Artery, inflammation, chronic active	1 (1%)	1 (1%)	1 (1%)	
Epithelium, hyperplasia	5 (6%)	13 (14%)	11 (12%)	11 (12%)
Seminal vesicle	(90)	(89)	(89)	(90)
Decreased secretory fluid	35 (39%)	18 (20%)	22 (25%)	11 (12%)
Degeneration				1 (1%)
Hemorrhage	1 (1%)			
Inflammation, acute	4 (4%)		3 (3%)	1 (1%)
Inflammation, chronic			1 (1%)	
Inflammation, chronic active	1 (1%)	4 (4%)	1 (1%)	1 (1%)
Artery, inflammation, chronic active	1 (1%)	1 (1%)		
Epithelium, hyperplasia	1 (1%)			
Testis	(90)	(90)	(90)	(90)
Cyst	1 (1%)			
Edema		2 (2%)	3 (3%)	2 (2%)
Inflammation, chronic active	2 (2%)			
Pigment	1 (1%)			
Artery, inflammation, chronic active	52 (58%)	40 (44%)	37 (41%)	20 (22%)
Germ cell, degeneration	51 (57%)	35 (39%)	42 (47%)	20 (22%)
Germinal epithelium, mineral		1 (1%)		
Interstitial cell, hyperplasia	1 (1%)	2 (2%)		4 (4%)
Rete testis, dilation	1 (1%)			
Seminiferous tubule, dilation	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Tunic, hemorrhage		1 (1%)		
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Fibrosis				1 (1%)
Hemorrhage		1 (1%)	3 (3%)	
Hypercellularity	15 (17%)	42 (47%)	32 (36%)	23 (26%)

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Lymph node	(25)	(22)	(18)	(12)
Artery, mediastinal, inflammation, chronic active		1 (5%)		1 (8%)
Artery, mediastinal, mineral		1 (5%)		
Bronchial, erythrophagocytosis			3 (17%)	
Iliac, erythrophagocytosis	2 (8%)	2 (9%)	1 (6%)	
Iliac, hyperplasia, lymphocyte	2 (8%)		1 (6%)	1 (8%)
Iliac, infiltration cellular, histiocyte	2 (8%)		1 (6%)	
Iliac, pigment			1 (6%)	
Iliac, proliferation, plasma cell	3 (12%)	1 (5%)	1 (6%)	2 (17%)
Iliac, lymphatic sinus, ectasia	5 (20%)	2 (9%)		1 (8%)
Lumbar, erythrophagocytosis	2 (8%)	1 (5%)	1 (6%)	1 (8%)
Lumbar, hemorrhage		1 (5%)		
Lumbar, hyperplasia, lymphocyte		1 (5%)		1 (8%)
Lumbar, proliferation, plasma cell		1 (5%)		1 (8%)
Lumbar, lymphatic sinus, ectasia		2 (9%)	1 (6%)	
Lymphatic sinus, mediastinal, ectasia	1 (4%)	1 (5%)		1 (8%)
Lymphatic sinus, renal, ectasia		3 (14%)	1 (6%)	1 (8%)
Mediastinal, congestion			2 (11%)	
Mediastinal, erythrophagocytosis	6 (24%)	5 (23%)	5 (28%)	6 (50%)
Mediastinal, hemorrhage	1 (4%)	1 (5%)		
Mediastinal, infiltration cellular, histiocyte		1 (5%)		
Pancreatic, erythrophagocytosis	3 (12%)	1 (5%)		2 (17%)
Pancreatic, hemorrhage	1 (4%)			
Pancreatic, hyperplasia, lymphocyte	1 (4%)			
Pancreatic, proliferation, plasma cell			1 (6%)	
Renal, erythrophagocytosis	8 (32%)	4 (18%)	3 (17%)	1 (8%)
Renal, hemorrhage		1 (5%)		
Renal, hyperplasia, lymphocyte		1 (5%)		1 (8%)
Renal, proliferation, plasma cell	2 (8%)			1 (8%)
Lymph node, mandibular	(89)	(90)	(89)	(90)
Atrophy, lymphoid		1 (1%)		
Congestion				3 (3%)
Erythrophagocytosis		2 (2%)	4 (4%)	3 (3%)
Hemorrhage		1 (1%)		
Hyperplasia, lymphocyte	41 (46%)	50 (56%)	54 (61%)	57 (63%)
Infiltration cellular, histiocyte				1 (1%)
Infiltration cellular, polymorphonuclear	2 (2%)			
Inflammation, suppurative				1 (1%)
Inflammation, chronic active				1 (1%)
Pigment			1 (1%)	1 (1%)
Proliferation, plasma cell	49 (55%)	67 (74%)	69 (78%)	68 (76%)
Lymphatic sinus, ectasia	16 (18%)	12 (13%)	20 (22%)	16 (18%)
Lymph node, mesenteric	(90)	(89)	(86)	(89)
Atrophy		1 (1%)		
Depletion cellular			1 (1%)	
Erythrophagocytosis	17 (19%)	7 (8%)	7 (8%)	8 (9%)
Hyperplasia, lymphocyte	2 (2%)		1 (1%)	4 (4%)
Infiltration cellular, histiocyte	1 (1%)		1 (1%)	
Infiltration cellular, polymorphonuclear	2 (2%)			
Proliferation, plasma cell				1 (1%)
Artery, inflammation, chronic active			1 (1%)	
Lymphatic sinus, ectasia		3 (3%)	2 (2%)	1 (1%)
Lymphocyte, depletion	2 (2%)			

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Spleen	(90)	(90)	(89)	(90)
Congestion				1 (1%)
Developmental malformation	1 (1%)			
Erythropagocytosis				1 (1%)
Extramedullary hematopoiesis	45 (50%)	58 (64%)	56 (63%)	64 (71%)
Hemorrhage			2 (2%)	
Hyperplasia, lymphocyte	5 (6%)	2 (2%)	2 (2%)	
Hyperplasia, plasma cell			1 (1%)	2 (2%)
Pigment	57 (63%)	62 (69%)	74 (83%)	74 (82%)
Arteriole, mineral	1 (1%)			
Artery, inflammation, chronic active		1 (1%)	4 (4%)	1 (1%)
Artery, mineral			1 (1%)	
Capsule, fibrosis		1 (1%)		
Red pulp, atrophy	26 (29%)	10 (11%)	10 (11%)	3 (3%)
White pulp, atrophy	30 (33%)	16 (18%)	13 (15%)	11 (12%)
Thymus	(88)	(86)	(88)	(86)
Atrophy	79 (90%)	71 (83%)	75 (85%)	78 (91%)
Congestion				1 (1%)
Cyst	10 (11%)	10 (12%)	9 (10%)	10 (12%)
Ectopic parathyroid gland	6 (7%)	1 (1%)	2 (2%)	3 (3%)
Ectopic thyroid	1 (1%)	4 (5%)		2 (2%)
Hemorrhage	2 (2%)	2 (2%)	2 (2%)	2 (2%)
Hyperplasia, epithelial	2 (2%)	4 (5%)	2 (2%)	2 (2%)
Thrombus			2 (2%)	
Artery, inflammation, chronic active	6 (7%)	3 (3%)	2 (2%)	1 (1%)
Integumentary System				
Mammary gland	(82)	(76)	(82)	(82)
Atrophy	1 (1%)		2 (2%)	
Galactocele	1 (1%)			
Hyperplasia		2 (3%)	5 (6%)	2 (2%)
Inflammation, granulomatous		1 (1%)		
Artery, inflammation, chronic active		1 (1%)		
Duct, dilation	3 (4%)	13 (17%)	3 (4%)	13 (16%)
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	3 (3%)	6 (7%)	8 (9%)	10 (11%)
Cyst epithelial inclusion, multifocal				1 (1%)
Hyperkeratosis		1 (1%)	2 (2%)	
Inflammation, chronic			1 (1%)	
Inflammation, chronic active	1 (1%)	1 (1%)	2 (2%)	1 (1%)
Ulcer	2 (2%)	3 (3%)	2 (2%)	
Artery, subcutaneous tissue, inflammation, chronic active	1 (1%)			
Epidermis, hyperplasia	1 (1%)	2 (2%)	2 (2%)	1 (1%)
Hair follicle, atrophy		1 (1%)	1 (1%)	
Hair follicle, dilation				1 (1%)
Lip, subcutaneous tissue, foreign body				1 (1%)
Lip, subcutaneous tissue, inflammation, chronic active				1 (1%)
Prepuce, cyst epithelial inclusion		1 (1%)		
Subcutaneous tissue, degeneration		1 (1%)		
Subcutaneous tissue, fibrosis				1 (1%)
Subcutaneous tissue, inflammation, suppurative				1 (1%)
	1 (1%)			

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Integumentary System (continued)				
Skin (continued)	(90)	(90)	(90)	(90)
Subcutaneous tissue, inflammation, acute		1 (1%)		
Subcutaneous tissue, inflammation, chronic	1 (1%)			
Subcutaneous tissue, inflammation, chronic active			2 (2%)	1 (1%)
				2 (2%)
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Fibrous osteodystrophy	46 (51%)	18 (20%)	14 (16%)	6 (7%)
Increased bone				1 (1%)
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	34 (38%)	49 (54%)	43 (48%)	37 (41%)
Mineral	2 (2%)			
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	7 (8%)	9 (10%)	4 (4%)	10 (11%)
Cyst		1 (1%)	1 (1%)	1 (1%)
Edema		2 (2%)	1 (1%)	
Hemorrhage	2 (2%)	1 (1%)	2 (2%)	
Infiltration cellular, mononuclear cell	1 (1%)			
Mineral	5 (6%)	4 (4%)	6 (7%)	2 (2%)
Necrosis	7 (8%)	3 (3%)	4 (4%)	3 (3%)
Vacuolation, cytoplasmic				
Brain stem, hemorrhage		1 (1%)		1 (1%)
Cerebellum, atrophy				2 (2%)
Choroid plexus, degeneration	1 (1%)			
Choroid plexus, mineral	3 (3%)	1 (1%)		
Glial cell, hyperplasia		2 (2%)	3 (3%)	1 (1%)
Meninges, hyperplasia	1 (1%)			
Meninges, hyperplasia, granular cell	1 (1%)		1 (1%)	
Meninges, metaplasia, osseous			1 (1%)	
Meninges, mineral		1 (1%)		
Perivascular, infiltration cellular, mononuclear cell		1 (1%)		
Pineal gland, infiltration cellular, mononuclear cell			1 (1%)	1 (1%)
Pineal gland, mineral	3 (3%)	10 (11%)	8 (9%)	3 (3%)
Pineal gland, vacuolation, cytoplasmic	12 (13%)	19 (21%)	20 (22%)	13 (14%)
Nerve trigeminal	(84)	(88)	(87)	(88)
Degeneration	63 (75%)	69 (78%)	65 (75%)	63 (72%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	86 (96%)	88 (98%)	90 (100%)	87 (97%)
Infiltration cellular, mononuclear cell	1 (1%)			
Peripheral nerve, tibial	(88)	(89)	(90)	(88)
Degeneration	84 (95%)	84 (94%)	90 (100%)	85 (97%)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Degeneration	30 (33%)	38 (42%)	41 (46%)	32 (36%)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Degeneration	21 (23%)	10 (11%)	17 (19%)	12 (13%)
Nerve, degeneration	79 (88%)	82 (91%)	87 (97%)	81 (90%)

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Nervous System (continued)				
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	58 (64%)	68 (76%)	72 (80%)	69 (77%)
Hemorrhage, focal	1 (1%)			
Trigeminal ganglion	(75)	(73)	(77)	(77)
Degeneration	23 (31%)	25 (34%)	22 (29%)	15 (19%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Congestion	13 (14%)	15 (17%)	11 (12%)	10 (11%)
Cyst		1 (1%)		
Fibrosis		1 (1%)		
Foreign body	4 (4%)		2 (2%)	1 (1%)
Hemorrhage	3 (3%)	4 (4%)	3 (3%)	
Inflammation, suppurative	3 (3%)	1 (1%)	2 (2%)	1 (1%)
Inflammation, granulomatous			3 (3%)	
Inflammation, chronic active	2 (2%)	8 (9%)	5 (6%)	3 (3%)
Inflammation, subacute	2 (2%)			
Mineral			1 (1%)	
Alveolar epithelium, hyperplasia			1 (1%)	
Alveolus, infiltration cellular, histiocyte	37 (41%)	40 (44%)	43 (48%)	48 (53%)
Artery, inflammation, chronic active	3 (3%)	5 (6%)	6 (7%)	3 (3%)
Artery, mineral	1 (1%)			
Artery, mediastinum, inflammation, chronic active		2 (2%)		
Bronchiole, hyperplasia, epithelial			1 (1%)	
Epithelium, alveolus, hyperplasia	3 (3%)	3 (3%)	3 (3%)	1 (1%)
Interstitial, fibrosis			1 (1%)	
Interstitial, mineral	1 (1%)		2 (2%)	
Perivascular, inflammation, chronic active	1 (1%)			
Nose	(89)	(90)	(90)	(89)
Foreign body	5 (6%)	3 (3%)	2 (2%)	4 (4%)
Fungus		1 (1%)		
Hyperplasia, lymphocyte			2 (2%)	
Inflammation, suppurative	10 (11%)	12 (13%)	13 (14%)	10 (11%)
Inflammation, chronic active		1 (1%)	2 (2%)	
Mineral			1 (1%)	
Nasopharyngeal duct, respiratory epithelium, hyperplasia	1 (1%)			
Olfactory epithelium, accumulation, hyaline droplet	79 (89%)	87 (97%)	82 (91%)	81 (91%)
Olfactory epithelium, atrophy			1 (1%)	
Olfactory epithelium, hyperplasia			2 (2%)	
Olfactory epithelium, metaplasia, respiratory	3 (3%)			2 (2%)
Respiratory epithelium, accumulation, hyaline droplet	3 (3%)	6 (7%)	7 (8%)	2 (2%)
Respiratory epithelium, atrophy		2 (2%)	1 (1%)	1 (1%)
Respiratory epithelium, hyperplasia	3 (3%)	11 (12%)	14 (16%)	11 (12%)
Respiratory epithelium, hyperplasia, goblet cell	1 (1%)			
Respiratory epithelium, metaplasia, squamous			1 (1%)	
Respiratory epithelium, mineral	1 (1%)			1 (1%)
Septum, developmental malformation				

TABLE A4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Respiratory System (continued)				
Trachea	(90)	(88)	(87)	(86)
Artery, inflammation, chronic active		1 (1%)		1 (1%)
Artery, mineral	1 (1%)			
Epithelium, hyperplasia	1 (1%)			
Epithelium, metaplasia, squamous	1 (1%)			
Glands, inflammation, acute				1 (1%)
Special Senses System				
Eye	(85)	(86)	(87)	(83)
Retinal detachment	1 (1%)			
Anterior chamber, inflammation, acute	4 (5%)	5 (6%)	3 (3%)	2 (2%)
Cornea, degeneration		1 (1%)		
Cornea, fibrosis	1 (1%)	3 (3%)	4 (5%)	6 (7%)
Cornea, inflammation, acute	28 (33%)	33 (38%)	25 (29%)	25 (30%)
Cornea, neovascularization	10 (12%)	19 (22%)	20 (23%)	19 (23%)
Cornea, ulcer	6 (7%)	2 (2%)	1 (1%)	2 (2%)
Cornea, epithelium, degeneration		2 (2%)	2 (2%)	2 (2%)
Cornea, epithelium, hyperplasia	13 (15%)	17 (20%)	15 (17%)	20 (24%)
Cornea, epithelium, regeneration		2 (2%)	2 (2%)	
Lens, cataract		2 (2%)		2 (2%)
Retina, atrophy	6 (7%)	10 (12%)	12 (14%)	14 (17%)
Retina, degeneration	1 (1%)			
Retina, dysplasia		1 (1%)		
Retina, gliosis		1 (1%)		
Harderian gland	(90)	(90)	(90)	(90)
Atrophy	1 (1%)	1 (1%)	1 (1%)	
Cyst			1 (1%)	
Degeneration, cystic	2 (2%)			3 (3%)
Hyperplasia				3 (3%)
Hypertrophy		2 (2%)		1 (1%)
Inflammation, granulomatous			2 (2%)	
Inflammation, acute	2 (2%)			
Inflammation, chronic			2 (2%)	
Inflammation, chronic active	2 (2%)		1 (1%)	1 (1%)
Lacrimal gland	(2)	(1)	(2)	(2)
Inflammation, granulomatous			1 (50%)	
Metaplasia, Harderian gland	2 (100%)	1 (100%)	2 (100%)	2 (100%)
Urinary System				
Kidney	(90)	(90)	(90)	(90)
Infarct			1 (1%)	
Inflammation, suppurative			1 (1%)	1 (1%)
Mineral	1 (1%)		1 (1%)	1 (1%)
Nephropathy, chronic progressive	88 (98%)	89 (99%)	90 (100%)	89 (99%)
Thrombus	1 (1%)			
Artery, inflammation, chronic active				1 (1%)
Artery, mineral	2 (2%)	1 (1%)		
Pelvis, dilation	1 (1%)	2 (2%)	1 (1%)	
Pelvis, inflammation, suppurative		1 (1%)	1 (1%)	1 (1%)
Pelvis, inflammation, chronic active				1 (1%)

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Rats
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Urinary System (continued)				
Kidney (continued)	(90)	(90)	(90)	(90)
Renal tubule, accumulation, hyaline droplet			1 (1%)	1 (1%)
Renal tubule, cyst	18 (20%)	17 (19%)	14 (16%)	6 (7%)
Renal tubule, hyperplasia		2 (2%)	1 (1%)	2 (2%)
Renal tubule, hyperplasia, atypical	2 (2%)			
Renal tubule, hyperplasia, oncocytic	2 (2%)			
Urothelium, hyperplasia	1 (1%)	2 (2%)	1 (1%)	2 (2%)
Ureter	(0)	(1)	(0)	(0)
Dilation		1 (100%)		
Urethra	(0)	(0)	(1)	(0)
Urinary bladder	(89)	(89)	(86)	(85)
Dilation			1 (1%)	
Hemorrhage	2 (2%)			1 (1%)
Inflammation, suppurative				
Inflammation, acute	2 (2%)			
Inflammation, chronic active			1 (1%)	
Necrosis	1 (1%)		1 (1%)	
Artery, inflammation, chronic active			1 (1%)	
Muscularis, degeneration	1 (1%)			
Serosa, inflammation, chronic active			1 (1%)	
Urothelium, hyperplasia	1 (1%)	1 (1%)	2 (2%)	1 (1%)

APPENDIX B

SUMMARY OF LESIONS IN FEMALE RATS EXPOSED TO GSM-MODULATED CELL PHONE RFR FOR 2 YEARS

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TABLE B1
Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
<i>14-Week interim evaluation</i>	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	30	25	31	22
Natural deaths	11	10	11	11
Survivors				
Died last week of study	1	3	1	
Terminal euthanasia	47	52	47	57
Animals examined microscopically	100	100	100	100

Systems Examined at 14 Weeks with No Neoplasms Observed

Alimentary System

Cardiovascular System

Endocrine System

General Body System

Genital System

Hematopoietic System

Integumentary System

Musculoskeletal System

Nervous System

Respiratory System

Special Senses System

Urinary System

2-Year Study

Alimentary System

Esophagus	(90)	(90)	(90)	(90)
Intestine large, cecum	(84)	(83)	(83)	(84)
Serosa, adenocarcinoma, metastatic, pancreas			1 (1%)	
Intestine large, colon	(89)	(88)	(89)	(89)
Intestine large, rectum	(90)	(89)	(89)	(89)
Granular cell tumor benign	1 (1%)			
Serosa, schwannoma malignant		1 (1%)		
Intestine small, duodenum	(88)	(85)	(83)	(85)
Adenocarcinoma		1 (1%)		
Intestine small, ileum	(86)	(82)	(81)	(83)
Intestine small, jejunum	(83)	(82)	(81)	(84)
Leiomyosarcoma	1 (1%)	1 (1%)		
Serosa, sarcoma stromal, metastatic, uterus		1 (1%)		
Liver	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum		1 (1%)		
Adenocarcinoma, metastatic, pancreas			1 (1%)	
Carcinoma, metastatic, kidney		1 (1%)		
Carcinoma, metastatic, uncertain primary site		1 (1%)		
Hepatocellular adenoma	7 (8%)	1 (1%)	1 (1%)	3 (3%)

TABLE B1
Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Liver (continued)	(90)	(90)	(90)	(90)
Hepatocellular adenoma, multiple		1 (1%)		
Hepatocellular carcinoma		1 (1%)		
Sarcoma stromal, metastatic, uterus		1 (1%)		
Serosa, adenocarcinoma, metastatic, uterus		1 (1%)		
Mesentery	(4)	(5)	(5)	(5)
Adenocarcinoma, metastatic, intestine small, duodenum		1 (20%)		
Adenocarcinoma, metastatic, pancreas	1 (25%)		1 (20%)	
Adenocarcinoma, metastatic, uterus		1 (20%)		
Sarcoma stromal, metastatic, uterus			1 (20%)	
Schwannoma malignant, metastatic, ovary				1 (0%)
Oral mucosa	(1)	(0)	(0)	(0)
Squamous cell carcinoma	1 (100%)			
Pancreas	(90)	(90)	(90)	(87)
Adenocarcinoma, metastatic, intestine small, duodenum		1 (1%)		
Adenocarcinoma, metastatic, uterus		1 (1%)		
Carcinoma, metastatic, uncertain primary site		1 (1%)		
Sarcoma stromal metastatic, uterus		1 (1%)		
Schwannoma malignant, metastatic, ovary			1 (1%)	2 (2%)
Acinus, adenocarcinoma				1 (1%)
Salivary glands	(90)	(89)	(90)	(90)
Myoepithelioma			1 (1%)	
Schwannoma malignant				1 (1%)
Parotid gland, adenoma			1 (1%)	
Stomach, forestomach	(90)	(90)	(90)	(90)
Sarcoma	1 (1%)			
Squamous cell papilloma			1 (1%)	
Stomach, glandular	(90)	(89)	(90)	(89)
Sarcoma, metastatic, stomach, forestomach		1 (1%)		
Serosa, adenocarcinoma, metastatic, pancreas			1 (1%)	
Tongue	(1)	(0)	(0)	(0)
Squamous cell carcinoma	1 (100%)			
Tooth	(0)	(0)	(1)	(0)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Heart	(90)	(90)	(90)	(90)
Endocardium, schwannoma malignant			1 (1%)	
Epicardium, paraganglioma			1 (1%)	
Myocardium, schwannoma malignant			1 (1%)	

TABLE B1
Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(90)	(89)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum		1 (1%)		
Adenoma, metastatic, pancreas			1 (1%)	
Adenoma	1 (1%)	5 (6%)	1 (1%)	5 (6%)
Carcinoma	1 (1%)			
Sarcoma stromal, metastatic, uterus		1 (1%)		
Adrenal medulla	(86)	(90)	(90)	(86)
Adenocarcinoma, metastatic, pancreas			1 (1%)	
Pheochromocytoma benign	1 (1%)	3 (3%)	3 (3%)	1 (1%)
Pheochromocytoma complex		1 (1%)	1 (1%)	
Bilateral, pheochromocytoma benign				1 (1%)
Islets, pancreatic	(90)	(89)	(90)	(87)
Adenoma	5 (6%)	4 (4%)	3 (3%)	4 (5%)
Carcinoma	2 (2%)	1 (1%)	4 (4%)	4 (5%)
Parathyroid gland	(87)	(79)	(82)	(79)
Pituitary gland	(90)	(90)	(90)	(90)
Schwannoma malignant, metastatic, trigeminal ganglion		1 (1%)		
Pars distalis, adenoma	42 (47%)	30 (33%)	35 (39%)	32 (36%)
Pars distalis, adenoma, multiple	1 (1%)	3 (3%)	3 (3%)	
Pars distalis, carcinoma	1 (1%)	1 (1%)		1 (1%)
Thyroid gland	(90)	(88)	(90)	(88)
Carcinoma, metastatic, kidney		2 (2%)		
Bilateral, C-cell, adenoma		1 (1%)		1 (1%)
Bilateral C-cell, adenoma, multiple		1 (1%)		
C-cell, adenoma	6 (7%)	9 (10%)	8 (9%)	12 (14%)
C-cell, carcinoma			1 (1%)	1 (1%)
Follicular cell, carcinoma	1 (1%)			
General Body System				
Tissue NOS	(8)	(10)	(8)	(10)
Chemodectoma benign			1 (13%)	
Abdominal, schwannoma malignant	1 (13%)			
Fat, adenocarcinoma, metastatic, uterus			1 (13%)	
Mediastinum, paraganglioma		1 (10%)		
Genital System				
Clitoral gland	(87)	(85)	(86)	(87)
Squamous cell carcinoma			1 (1%)	
Ovary	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum		1 (1%)		
Adenocarcinoma, metastatic, pancreas			1 (1%)	
Cystadenoma	1 (1%)		1 (1%)	
Granulosa cell tumor benign	1 (1%)			
Granulosa cell tumor malignant	2 (2%)		1 (1%)	
Lymphangiosarcoma		1 (1%)		
Schwannoma malignant			1 (1%)	
Sertoli cell tumor benign	1 (1%)			
Sex cord stromal tumor, benign		1 (1%)	1 (1%)	
Tubulostromal carcinoma				1 (1%)

TABLE B1
Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Genital System (continued)				
Ovary (continued)	(90)	(90)	(90)	(90)
Bilateral, rete ovarii, adenoma		1 (1%)		1 (1%)
Periovarian tissue, sarcoma stromal, metastatic, uterus				
Oviduct	(1)	(0)	(0)	(0)
Uterus	(90)	(89)	(90)	(90)
Adenocarcinoma	3 (3%)	1 (1%)	2 (2%)	5 (6%)
Adenocarcinoma, metastatic, pancreas			1 (1%)	
Adenoma				1 (1%)
Hemangioma		1 (1%)		
Hemangiosarcoma	2 (2%)			
Leiomyosarcoma			1 (1%)	
Polyp stromal	15 (17%)	11 (12%)	10 (11%)	16 (18%)
Polyp stromal, multiple	1 (1%)	6 (7%)	1 (1%)	1 (1%)
Sarcoma stromal		1 (1%)	2 (2%)	
Schwannoma malignant	1 (1%)		1 (1%)	
Squamous cell carcinoma			2 (2%)	
Cervix, leiomyosarcoma	1 (1%)			
Cervix, malignant mixed mullerian tumor				1 (1%)
Cervix, polyp stromal		1 (1%)		
Cervix, schwannoma malignant	1 (1%)	1 (1%)		1 (1%)
Vagina	(2)	(3)	(1)	(1)
Granular cell tumor benign		1 (33%)		
Sarcoma, metastatic, uterus		1 (33%)		
Schwannoma malignant	1 (50%)			
Schwannoma malignant, metastatic, uterus	1 (50%)			
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Lymph node	(13)	(14)	(21)	(14)
Bronchial, adenocarcinoma, metastatic, intestine small, duodenum		1 (7%)		
Lumbar, basal cell carcinoma, metastatic, skin		1 (7%)		
Mediastinal, adenocarcinoma, metastatic, intestine small, duodenum		1 (7%)		
Mediastinal, adenocarcinoma, metastatic, pancreas			1 (5%)	
Lymph node, mandibular	(90)	(89)	(89)	(90)
Lymph node, mesenteric	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum		1 (1%)		
Hemangiosarcoma			1 (1%)	
Spleen	(90)	(90)	(90)	(90)
Hemangiosarcoma	1 (1%)			1 (1%)
Sarcoma stromal, metastatic, uterus		1 (1%)		
Capsule, adenocarcinoma, metastatic, intestine small, duodenum		1 (1%)		
Thymus	(87)	(86)	(88)	(86)
Schwannoma malignant			1 (1%)	
Thymoma benign	1 (1%)	1 (1%)		
Thymoma malignant	1 (1%)	1 (1%)		

TABLE B1
Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Integumentary System				
Mammary gland	(90)	(89)	(89)	(90)
Adenocarcinoma	9 (10%)	5 (6%)	8 (9%)	6 (7%)
Adenocarcinoma, multiple	1 (1%)	1 (1%)		
Adenoma	4 (4%)	5 (6%)	2 (2%)	2 (2%)
Adenoma, multiple	4 (4%)			
Fibroadenoma	34 (38%)	43 (48%)	38 (43%)	32 (36%)
Fibroadenoma, multiple	29 (32%)	25 (28%)	22 (25%)	30 (33%)
Myoepithelioma				1 (1%)
Skin	(90)	(90)	(90)	(90)
Basal cell carcinoma		1 (1%)		
Keratoacanthoma				1 (1%)
Pilomatrixoma			1 (1%)	
Squamous cell carcinoma			1 (1%)	1 (1%)
Squamous cell papilloma			1 (1%)	
Subcutaneous tissue, fibroma	2 (2%)	3 (3%)	2 (2%)	4 (4%)
Subcutaneous tissue, fibrosarcoma		1 (1%)		1 (1%)
Subcutaneous tissue, lipoma				1 (1%)
Subcutaneous tissue,				
malignant fibrous histiocytoma	1 (1%)			
Subcutaneous tissue, sarcoma	2 (2%)			
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Skeletal muscle	(90)	(90)	(90)	(90)
Sarcoma stromal, metastatic, uterus		1 (1%)		
Diaphragm, adenocarcinoma, metastatic, intestine small, duodenum			1 (1%)	
Diaphragm, adenocarcinoma, metastatic, pancreas				1 (1%)
Diaphragm, carcinoma, metastatic, uncertain primary site		1 (1%)		
Nervous System				
Brain	(90)	(90)	(90)	(90)
Carcinoma, metastatic, pituitary gland	1 (1%)	1 (1%)		
Glioma malignant				1 (1%)
Schwannoma malignant, metastatic, trigeminal ganglion		1 (1%)		
Meninges, carcinoma, metastatic, kidney		2 (2%)		
Meninges, granular cell tumor benign	1 (1%)	1 (1%)	2 (2%)	
Meninges, granular cell tumor malignant				1 (1%)
Nerve trigeminal	(84)	(88)	(89)	(90)
Schwannoma malignant, metastatic, trigeminal ganglion		1 (1%)		
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Peripheral nerve, tibial	(90)	(90)	(90)	(89)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Meninges, carcinoma, metastatic, kidney		1 (1%)		
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(81)	(79)	(80)	(79)
Schwannoma malignant		1 (1%)		

TABLE B1
Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, uterus			1 (1%)	
Alveolar/bronchiolar carcinoma		1 (1%)		
Basal cell carcinoma, metastatic, skin		1 (1%)		
Carcinoma, metastatic, thyroid gland			1 (1%)	
Carcinoma, metastatic,				1 (1%)
uncertain primary site		2 (2%)		
Sarcoma stromal, metastatic, uterus		1 (1%)		
Squamous cell carcinoma, metastatic, skin			1 (1%)	
Nose	(90)	(90)	(90)	(90)
Trachea	(89)	(90)	(89)	(87)
Special Senses System				
Eye	(88)	(85)	(87)	(87)
Retrobulbar, schwannoma malignant, metastatic, trigeminal ganglion		1 (1%)		
Harderian gland	(90)	(90)	(90)	(90)
Zymbal's gland	(0)	(0)	(0)	(1)
Squamous cell papilloma				1 (100%)
Urinary System				
Kidney	(90)	(90)	(90)	(89)
Adenocarcinoma, metastatic, intestine small, duodenum		1 (1%)		
Carcinoma, metastatic, uncertain primary site		1 (1%)		
Sarcoma stromal, metastatic, uterus		1 (1%)		
Bilateral, renal tubule, carcinoma	1 (1%)	1 (1%)		
Bilateral, renal tubule, carcinoma, multiple			1 (1%)	
Renal tubule, adenoma	1 (1%)	1 (1%)		
Renal tubule, adenoma, multiple				1 (1%)
Renal tubule, carcinoma, multiple				1 (1%)
Urinary bladder	(88)	(88)	(90)	(87)
Leiomyosarcoma	1 (1%)			
Schwannoma malignant, metastatic, ovary			1 (1%)	
Schwannoma malignant, metastatic, tissue NOS	1 (1%)			
Serosa, adenocarcinoma, metastatic, pancreas			1 (1%)	
Urothelium, carcinoma		1 (1%)		
Systemic Lesions				
Multiple organs ^b	(90)	(90)	(90)	(90)
Histiocytic sarcoma		1 (1%)		1 (1%)
Leukemia mononuclear		1 (1%)	1 (1%)	
Lymphoma malignant	5 (6%)	4 (4%)	2 (2%)	5 (6%)

TABLE B1
Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Neoplasm Summary				
Total animals with primary neoplasms ^c				
2-Year study	89	87	82	86
Total primary neoplasms				
2-Year study	202	189	175	184
Total animals with benign neoplasms				
2-Year study	82	83	75	80
Total benign neoplasms				
2-Year study	159	159	140	150
Total animals with malignant neoplasms				
2-Year study	37	28	30	28
Total malignant neoplasms				
2-Year study	43	33	34	34
Total animals with metastatic neoplasms				
2-Year study	5	10	5	2
Total metastatic neoplasms				
2-Year study	5	44	18	2
Total animals with malignant neoplasms- uncertain primary site				
2-Year study		2		
Total animals with uncertain neoplasms- benign or malignant				
2-Year study			1	
Total uncertain neoplasms				
2-Year study			1	

^a Number of animals examined microscopically at the site and the number of animals with neoplasm

^b Number of animals with any tissue examined microscopically

^c Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE B2
**Statistical Analysis of Primary Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Cortex: Adenoma				
Overall rate ^a	1/90 (1%)	5/90 (6%)	1/89 (1%)	5/90 (6%)
Rate per litters ^b	1/35 (3%)	5/35 (14%)	1/35 (3%)	4/35 (11%)
Adjusted rate ^c	1.4%	6.5%	1.4%	6.7%
Terminal rate ^d	1/48 (2%)	2/53 (4%)	1/48 (2%)	4/57 (7%)
First incidence (days)	737 (T)	464	737 (T)	651
Rao-Scott adjusted poly-3 test ^e	P=0.222	P=0.182	P=0.742N	P=0.174
Litter C-A/Fisher's test ^f	P=0.280	P=0.099	P=0.754	P=0.178
Liver: Hepatocellular Adenoma				
Overall rate	7/90 (8%)	2/90 (2%)	1/90 (1%)	3/90 (3%)
Rate per litters	6/35 (17%)	2/35 (6%)	1/35 (3%)	3/35 (9%)
Adjusted rate	10.1%	2.6%	1.4%	4%
Terminal rate	6/48 (13%)	2/53 (4%)	1/48 (2%)	3/57 (5%)
First incidence (days)	707	737 (T)	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.168N	P=0.106N	P=0.061N	P=0.183N
Litter C-A/Fisher's test	P=0.207N	P=0.130N	P=0.053N	P=0.239N
Liver: Hepatocellular Adenoma or Hepatocellular Carcinoma				
Overall rate	7/90 (8%)	3/90 (3%)	1/90 (1%)	3/90 (3%)
Rate per litters	6/35 (17%)	3/35 (9%)	1/35 (3%)	3/35 (9%)
Adjusted rate	10.1%	4%	1.4%	4%
Terminal rate	6/48 (13%)	2/53 (4%)	1/48 (2%)	3/57 (5%)
First incidence (days)	707	696	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.141N	P=0.171N	P=0.057N	P=0.176N
Litter C-A/Fisher's test	P=0.175N	P=0.239N	P=0.053N	P=0.239N
Mammary Gland: Fibroadenoma				
Overall rate	63/90 (70%)	68/90 (76%)	60/90 (67%)	62/90 (69%)
Rate per litters	31/35 (89%)	33/35 (94%)	33/35 (94%)	34/35 (97%)
Adjusted rate	77%	79.2%	71.7%	72.7%
Terminal rate	36/48 (75%)	39/53 (74%)	32/48 (67%)	38/57 (67%)
First incidence (days)	464	464	383	283
Rao-Scott adjusted poly-3 test	P=0.219N	P=0.445	P=0.287N	P=0.334N
Litter C-A/Fisher's test	P=0.134	P=0.337	P=0.337	P=0.178
Mammary Gland: Adenoma				
Overall rate	8/90 (9%)	5/90 (6%)	2/90 (2%)	2/90 (2%)
Rate per litters	7/35 (20%)	5/35 (14%)	2/35 (6%)	2/35 (6%)
Adjusted rate	11.3%	6.6%	2.8%	2.7%
Terminal rate	5/48 (10%)	4/53 (8%)	2/48 (4%)	1/57 (2%)
First incidence (days)	524	718	737 (T)	669
Rao-Scott adjusted poly-3 test	P=0.038N	P=0.271N	P=0.075N	P=0.065N
Litter C-A/Fisher's test	P=0.036N	P=0.376N	P=0.075N	P=0.075N
Mammary Gland: Fibroadenoma or Adenoma				
Overall rate	64/90 (71%)	69/90 (77%)	60/90 (67%)	62/90 (69%)
Rate per litters	31/35 (89%)	33/35 (94%)	33/35 (94%)	34/35 (97%)
Adjusted rate	77.7%	80.4%	71.7%	72.7%
Terminal rate	36/48 (75%)	40/53 (76%)	32/48 (67%)	38/57 (67%)
First incidence (days)	464	464	383	283
Rao-Scott adjusted poly-3 test	P=0.182N	P=0.413	P=0.260N	P=0.304N
Litter C-A/Fisher's test	P=0.134	P=0.337	P=0.337	P=0.178

TABLE B2
Statistical Analysis of Primary Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Mammary Gland: Adenocarcinoma				
Overall rate	10/90 (11%)	6/90 (7%)	8/90 (9%)	6/90 (7%)
Rate per litters	9/35 (26%)	6/35 (17%)	7/35 (20%)	6/35 (17%)
Adjusted rate	14.2%	7.8%	11.1%	7.9%
Terminal rate	6/48 (13%)	4/53 (8%)	5/48 (10%)	3/57 (5%)
First incidence (days)	622	332	489	489
Rao-Scott adjusted poly-3 test	P=0.218N	P=0.171N	P=0.379N	P=0.177N
Litter C-A/Fisher's test	P=0.284N	P=0.281N	P=0.388N	P=0.281N
Mammary Gland: Adenoma or Adenocarcinoma				
Overall rate	16/90 (18%)	11/90 (12%)	10/90 (11%)	8/90 (9%)
Rate per litters	13/35 (37%)	11/35 (31%)	9/35 (26%)	7/35 (20%)
Adjusted rate	22.2%	14.2%	13.9%	10.5%
Terminal rate	9/48 (19%)	8/53 (15%)	7/48 (15%)	4/57 (7%)
First incidence (days)	524	332	489	489
Rao-Scott adjusted poly-3 test	P=0.052N	P=0.153N	P=0.146N	P=0.049N
Litter C-A/Fisher's test	P=0.065N	P=0.401N	P=0.220N	P=0.093N
Mammary Gland: Fibroadenoma, Adenoma, or Adenocarcinoma				
Overall rate	66/90 (73%)	72/90 (80%)	64/90 (71%)	64/90 (71%)
Rate per litters	31/35 (89%)	34/35 (97%)	35/35 (100%)	34/35 (97%)
Adjusted rate	79.6%	82.3%	75.3%	74.4%
Terminal rate	36/48 (75%)	41/53 (77%)	33/48 (69%)	39/57 (68%)
First incidence (days)	464	332	383	283
Rao-Scott adjusted poly-3 test	P=0.166N	P=0.410	P=0.326N	P=0.282N
Litter C-A/Fisher's test	P=0.102	P=0.178	P=0.057	P=0.178
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	4/89 (4%)	3/90 (3%)	4/87 (5%)
Rate per litters	4/35 (11%)	4/35 (11%)	3/35 (9%)	4/35 (11%)
Adjusted rate	7.2%	5.3%	4.2%	5.5%
Terminal rate	5/48 (10%)	2/53 (4%)	2/48 (4%)	3/57 (5%)
First incidence (days)	737 (T)	699	621	669
Rao-Scott adjusted poly-3 test	P=0.422N	P=0.451N	P=0.346N	P=0.466N
Litter C-A/Fisher's test	P=0.555N	P=0.645	P=0.500N	P=0.645
Pancreatic Islets: Carcinoma				
Overall rate	2/90 (2%)	1/89 (1%)	4/90 (4%)	4/87 (5%)
Rate per litters	2/35 (6%)	1/35 (3%)	4/35 (11%)	4/35 (11%)
Adjusted rate	2.9%	1.3%	5.6%	5.5%
Terminal rate	1/48 (2%)	1/53 (2%)	2/48 (4%)	4/57 (7%)
First incidence (days)	711	737 (T)	699	737 (T)
Rao-Scott adjusted poly-3 test	P=0.188	P=0.467N	P=0.349	P=0.362
Litter C-A/Fisher's test	P=0.157	P=0.500N	P=0.337	P=0.337
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	7/90 (8%)	5/89 (6%)	7/90 (8%)	7/87 (8%)
Rate per litters	6/35 (17%)	5/35 (14%)	7/35 (20%)	7/35 (20%)
Adjusted rate	10.1%	6.7%	9.8%	9.6%
Terminal rate	6/48 (13%)	3/53 (6%)	4/48 (8%)	6/57 (11%)
First incidence (days)	711	699	621	669
Rao-Scott adjusted poly-3 test	P=0.487	P=0.323N	P=0.571N	P=0.558N
Litter C-A/Fisher's test	P=0.369	P=0.500N	P=0.500	P=0.500

TABLE B2
**Statistical Analysis of Primary Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	43/90 (48%)	33/90 (37%)	38/90 (42%)	32/90 (36%)
Rate per litters	28/35 (80%)	24/35 (69%)	26/35 (74%)	23/35 (66%)
Adjusted rate	57.1%	42.5%	51.2%	41.6%
Terminal rate	28/48 (58%)	23/53 (43%)	24/48 (50%)	24/57 (42%)
First incidence (days)	464	578	545	565
Rao-Scott adjusted poly-3 test	P=0.077N	P=0.049N	P=0.283N	P=0.038N
Litter C-A/Fisher's test	P=0.162N	P=0.206N	P=0.388N	P=0.141N
Pituitary Gland (Pars Distalis): Adenoma or Carcinoma				
Overall rate	44/90 (49%)	34/90 (38%)	38/90 (42%)	33/90 (37%)
Rate per litters	29/35 (83%)	24/35 (69%)	26/35 (74%)	23/35 (66%)
Adjusted rate	57.9%	43.8%	51.2%	42.8%
Terminal rate	28/48 (58%)	24/53 (45%)	24/48 (50%)	25/57 (44%)
First incidence (days)	464	578	545	565
Rao-Scott adjusted poly-3 test	P=0.082N	P=0.056N	P=0.250N	P=0.044N
Litter C-A/Fisher's test	P=0.111N	P=0.132N	P=0.281N	P=0.085N
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	4/90 (4%)	2/90 (2%)	5/90 (6%)
Rate per litters	5/35 (14%)	4/35 (11%)	2/35 (6%)	5/35 (14%)
Adjusted rate	7%	5.2%	2.8%	6.7%
Terminal rate	1/48 (2%)	3/53 (6%)	2/48 (4%)	4/57 (7%)
First incidence (days)	268	123	737 (T)	669
Rao-Scott adjusted poly-3 test	P=0.551N	P=0.449N	P=0.220N	P=0.586N
Litter C-A/Fisher's test	P=0.571	P=0.500N	P=0.214N	P=0.633
Thyroid Gland (C-cell): Adenoma				
Overall rate	6/90 (7%)	11/88 (13%)	8/90 (9%)	13/88 (15%)
Rate per litters	6/35 (17%)	10/35 (29%)	8/35 (23%)	12/35 (34%)
Adjusted rate	8.5%	14.7%	11.3%	17.6%
Terminal rate	3/48 (6%)	8/53 (15%)	6/48 (13%)	12/57 (21%)
First incidence (days)	608	699	695	656
Rao-Scott adjusted poly-3 test	P=0.108	P=0.188	P=0.383	P=0.091
Litter C-A/Fisher's test	P=0.096	P=0.197	P=0.383	P=0.085
Thyroid Gland (C-cell): Adenoma or Carcinoma				
Overall rate	6/90 (7%)	11/88 (13%)	9/90 (10%)	14/88 (16%)
Rate per litters	6/35 (17%)	10/35 (29%)	9/35 (26%)	13/35 (37%)
Adjusted rate	8.5%	14.7%	12.7%	18.9%
Terminal rate	3/48 (6%)	8/53 (15%)	7/48 (15%)	13/57 (23%)
First incidence (days)	608	699	695	656
Rao-Scott adjusted poly-3 test	P=0.068	P=0.186	P=0.290	P=0.061
Litter C-A/Fisher's test	P=0.054	P=0.197	P=0.281	P=0.053
Uterus: Stromal Polyp				
Overall rate	16/90 (18%)	18/90 (20%)	11/90 (12%)	17/90 (19%)
Rate per litters	11/35 (31%)	14/35 (40%)	10/35 (29%)	13/35 (37%)
Adjusted rate	22.7%	23.5%	15.4%	22.3%
Terminal rate	14/48 (29%)	14/53 (26%)	6/48 (13%)	11/57 (19%)
First incidence (days)	531	602	656	594
Rao-Scott adjusted poly-3 test	P=0.461N	P=0.530	P=0.216N	P=0.554N
Litter C-A/Fisher's test	P=0.452	P=0.309	P=0.500N	P=0.401

TABLE B2
Statistical Analysis of Primary Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Uterus: Stromal Polyp or Stromal Sarcoma				
Overall rate	16/90 (18%)	19/90 (21%)	13/90 (14%)	17/90 (19%)
Rate per litters	11/35 (31%)	15/35 (43%)	11/35 (31%)	13/35 (37%)
Adjusted rate	22.7%	24.7%	18.2%	22.3%
Terminal rate	14/48 (29%)	14/53 (26%)	8/48 (17%)	11/57 (19%)
First incidence (days)	531	602	656	594
Rao-Scott adjusted poly-3 test	P=0.451N	P=0.465	P=0.341N	P=0.553N
Litter C-A/Fisher's test	P=0.476	P=0.229	P=0.601	P=0.401
Uterus: Adenocarcinoma				
Overall rate	3/90 (3%)	1/90 (1%)	2/90 (2%)	5/90 (6%)
Rate per litters	2/35 (6%)	1/35 (3%)	2/35 (6%)	5/35 (14%)
Adjusted rate	4.3%	1.3%	2.8%	6.7%
Terminal rate	3/48 (6%)	1/53 (2%)	2/48 (4%)	5/57 (9%)
First incidence (days)	737 (T)	737 (T)	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.217	P=0.316N	P=0.500N	P=0.420
Litter C-A/Fisher's test	P=0.075	P=0.500N	P=0.693	P=0.214
All Organs: Malignant Schwannoma				
Overall rate	4/90 (4%)	1/90 (1%)	5/90 (6%)	2/90 (2%)
Rate per litters	3/35 (9%)	1/35 (3%)	5/35 (14%)	2/35 (6%)
Adjusted rate	5.7%	1.3%	7%	2.7%
Terminal rate	2/48 (4%)	0/53 (0%)	2/48 (4%)	1/57 (2%)
First incidence (days)	489	480	578	622
Rao-Scott adjusted poly-3 test	P=0.428N	P=0.212N	P=0.519	P=0.354N
Litter C-A/Fisher's test	P=0.563N	P=0.307N	P=0.355	P=0.500N
All Organs: Malignant Lymphoma				
Overall rate	5/90 (6%)	4/90 (4%)	2/90 (2%)	5/90 (6%)
Rate per litters	5/35 (14%)	3/35 (9%)	2/35 (6%)	5/35 (14%)
Adjusted rate	7%	5.2%	2.8%	6.5%
Terminal rate	0/48 (0%)	0/53 (0%)	0/48 (0%)	1/57 (2%)
First incidence (days)	268	545	450	299
Rao-Scott adjusted poly-3 test	P=0.537N	P=0.454N	P=0.229N	P=0.572N
Litter C-A/Fisher's test	P=0.518	P=0.355N	P=0.214N	P=0.633

TABLE B2
**Statistical Analysis of Primary Neoplasms in Female Rats Exposed to GSM-Modulated Cell Phone RFR
for 2 Years**

(T) Terminal euthanasia

- a Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, liver, pancreatic islets, pituitary gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
- b Number of litters with tumor-bearing animals/number of litters examined at site
- c Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
- d Observed incidence at terminal euthanasia
- e Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott adjusted poly-3 test is a modification of the Poly-3 test that also incorporates an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
- f The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
- g Value of statistic cannot be computed.

TABLE B3a
Historical Incidence of Malignant Schwannoma of the Heart
in Control Female Hsd:Sprague Dawley SD Rats^a

Incidence in Controls	
Overall Historical Incidence: All Routes	
Total	0/239

^a Data as of November 2017

TABLE B3b
Historical Incidence of Brain Neoplasms in Control Female Hsd:Sprague Dawley SD Rats^a

	Malignant Glioma	Benign Granular Cell Tumor	Malignant Granular Cell Tumor	Benign or Malignant Granular Cell Tumor
Overall Historical Incidence: All Routes				
Total (%)	1/190 (0.7%)	2/190 (1.0%)	0/240	2/190 (1.0%)
Mean ± standard deviation	0.7% ± 1.2%	1.0% ± 1.0%		1.0% ± 1.0%
Range	0%-2%	0%-2%		0%-2%

^a Data as of November 2017

TABLE B3c
Historical Incidence of Pituitary Gland (Pars Distalis) Adenoma
in Control Female Hsd:Sprague Dawley SD Rats^a

Incidence in Controls	
Overall Historical Incidence: All Routes	
Total (%)	98/240 (40.8%)
Mean ± standard deviation	39.4% ± 5.6%
Range	36%-48%

^a Data as of November 2017

TABLE B3d
Historical Incidence of Adrenal Medulla Neoplasms in Control Female Hsd:Sprague Dawley SD Rats^a

	Benign Pheochromocytoma	Malignant Pheochromocytoma	Complex Pheochromocytoma	Benign, Malignant, or Complex Pheochromocytoma
Overall Historical Incidence: All Routes				
Total (%)	4/235 (1.7%)	2/235 (0.9%)	0/235	6/235 (2.6%)
Mean ± standard deviation	1.8% ± 2.9%	1.0% ± 2.0%		2.8% ± 4.8%
Range	0%-6%	0%-4%		0%-10%

^a Data as of November 2017

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	30	25	31	22
Natural deaths	11	10	11	11
Survivors				
Died last week of study	1	3	1	
Terminal euthanasia	47	52	47	57
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(10)	(10)
Intestine large, cecum	(10)	(10)	(10)	(10)
Intestine large colon	(10)	(10)	(10)	(10)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)	1 (10%)		
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(10)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell	1 (10%)	1 (10%)	2 (20%)	1 (10%)
Pancreas	(10)	(10)	(10)	(10)
Salivary glands	(10)	(10)	(10)	(10)
Degeneration, cystic				1 (10%)
Stomach, forestomach	(10)	(10)	(10)	(10)
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy		2 (20%)	1 (10%)	2 (20%)
Ventricle right, cardiomyopathy			1 (10%)	
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(10)	(8)	(9)	(9)
Pituitary gland	(10)	(10)	(10)	(10)
Pars distalis, cyst	2 (20%)			
Pars intermedia, cyst	1 (10%)	1 (10%)		
Thyroid gland	(10)	(10)	(10)	(10)

^a Number of animals examined microscopically at the site and the number of animals with lesion

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
14-Week Interim Evaluation (continued)				
Genital System				
Clitoral gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	4 (40%)	5 (50%)	6 (60%)	4 (40%)
Ovary	(10)	(10)	(10)	(10)
Cyst				1 (10%)
Follicle, cyst	1 (10%)			
Uterus	(10)	(10)	(10)	(10)
Hematopoietic System				
Bone marrow	(10)	(10)	(10)	(10)
Lymph node, mandibular	(10)	(10)	(10)	(10)
Hemorrhage		2 (20%)		
Hyperplasia, lymphocyte				2 (20%)
Proliferation, plasma cell				3 (30%)
Lymph node, mesenteric	(10)	(10)	(10)	(10)
Spleen	(10)	(10)	(10)	(10)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	1 (10%)		1 (10%)	
Musculoskeletal System				
Bone	(10)	(10)	(10)	(10)
Skeletal muscle	(10)	(10)	(10)	(10)
Infiltration cellular, histiocyte			1 (10%)	
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion			1 (10%)	
Inflammation, chronic active			1 (10%)	
Alveolus, infiltration cellular, histiocyte	2 (20%)			
Epithelium alveolus, hyperplasia			1 (10%)	
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(10)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Harderian gland	(10)	(10)	(10)	(10)
Inflammation cellular, lymphocyte	1 (10%)			
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Nephropathy, chronic progressive	3 (30%)	4 (40%)	3 (30%)	2 (20%)
Urinary bladder	(10)	(10)	(10)	(10)
Systems Examined with No Lesions Observed				
General Body System				
Integumentary System				
Nervous System				

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Dilation			1 (1%)	
Intestine large, cecum	(84)	(83)	(83)	(84)
Serosa, inflammation, acute		1 (1%)		
Intestine large, colon	(89)	(88)	(89)	(89)
Intestine large, rectum	(90)	(89)	(89)	(89)
Hyperplasia, lymphocyte		1 (1%)		1 (1%)
Inflammation, acute				1 (1%)
Necrosis				1 (1%)
Epithelium, hyperplasia				1 (1%)
Epithelium, metaplasia, squamous			1 (1%)	1 (1%)
Intestine small, duodenum	(88)	(85)	(83)	(85)
Ectopic tissue				1 (1%)
Ulcer		1 (1%)		
Intestine small, ileum	(86)	(82)	(81)	(83)
Hyperplasia, lymphocyte	1 (1%)			1 (1%)
Necrosis, lymphoid				
Serosa, inflammation, acute		1 (1%)		
Intestine small, jejunum	(83)	(82)	(81)	(84)
Liver	(90)	(90)	(90)	(90)
Angiectasis	6 (7%)	4 (4%)	6 (7%)	6 (7%)
Basophilic focus	11 (12%)	17 (19%)	11 (12%)	8 (9%)
Clear cell focus	2 (2%)	3 (3%)	6 (7%)	6 (7%)
Congestion		1 (1%)	2 (2%)	
Eosinophilic focus	9 (10%)	26 (29%)	23 (26%)	23 (26%)
Extramedullary hematopoiesis	15 (17%)	19 (21%)	17 (19%)	12 (13%)
Fibrosis		1 (1%)		
Hepatodiaphragmatic nodule	1 (1%)		1 (1%)	
Infiltration cellular, histiocyte			2 (2%)	
Infiltration cellular, mixed cell	1 (1%)	4 (4%)	1 (1%)	2 (2%)
Infiltration cellular, mononuclear cell		1 (1%)		
Inflammation, acute		1 (1%)		1 (1%)
Inflammation, chronic		1 (1%)		
Inflammation, chronic active			2 (2%)	
Mixed cell focus	29 (32%)	23 (26%)	33 (37%)	28 (31%)
Pigment		1 (1%)		
Bile duct, cyst	11 (12%)	6 (7%)	5 (6%)	8 (9%)
Bile duct, fibrosis	1 (1%)			
Bile duct, hyperplasia	9 (10%)	5 (6%)	10 (11%)	9 (10%)
Bile duct, inflammation, chronic active			1 (1%)	
Centrilobular, hepatocyte, necrosis		3 (3%)		2 (2%)
Centrilobular, hepatocyte, vacuolation, cytoplasmic		1 (1%)		
Hepatocyte, degeneration			1 (1%)	
Hepatocyte, hypertrophy	2 (2%)	5 (6%)	2 (2%)	6 (7%)
Hepatocyte, increased mitoses	2 (2%)			
Hepatocyte, necrosis	4 (4%)	2 (2%)	5 (6%)	8 (9%)
Hepatocyte, vacuolation, cytoplasmic	1 (1%)	1 (1%)	1 (1%)	3 (3%)
Kupffer cell, hyperplasia	3 (3%)			
Kupffer cell, hypertrophy	2 (2%)			
Periductal, cholangiofibrosis	1 (1%)			1 (1%)
Serosa, inflammation, suppurative		1 (1%)		
Serosa, inflammation, chronic active	1 (1%)		1 (1%)	
Sinusoid, dilation			1 (1%)	

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Mesentery	(4)	(5)	(5)	(5)
Inflammation, chronic active	1 (25%)		1 (20%)	
Necrosis	1 (25%)	3 (60%)	2 (40%)	3 (60%)
Artery, inflammation, chronic active				1 (20%)
Oral mucosa	(1)	(0)	(0)	(0)
Pancreas	(90)	(90)	(90)	(87)
Ectopic liver	1 (1%)			
Inflammation, acute		1 (1%)		
Inflammation, chronic active	1 (1%)		2 (2%)	
Acinus, atrophy	5 (6%)	4 (4%)	4 (4%)	5 (6%)
Acinus, hyperplasia	1 (1%)	2 (2%)	5 (6%)	2 (2%)
Artery, inflammation, chronic active		2 (2%)	1 (1%)	1 (1%)
Periductal, cholangiofibrosis			7 (8%)	4 (5%)
Salivary glands	(90)	(89)	(90)	(90)
Duct, parotid gland, dilation	1 (1%)			1 (1%)
Parotid gland, atrophy	4 (4%)	11 (12%)	8 (9%)	4 (4%)
Parotid gland, inflammation, acute				1 (1%)
Parotid gland, vacuolation, cytoplasmic				1 (1%)
Sublingual gland, atrophy				2 (2%)
Sublingual gland, metaplasia			1 (1%)	
Submandibular gland, atrophy		1 (1%)	1 (1%)	
Stomach, forestomach	(90)	(90)	(90)	(90)
Edema	2 (2%)	2 (2%)	1 (1%)	
Erosion	2 (2%)			
Fibrosis	1 (1%)			
Inflammation, acute		1 (1%)		
Inflammation, chronic active	4 (4%)	5 (6%)		
Ulcer	1 (1%)	7 (8%)	2 (2%)	2 (2%)
Epithelium, hyperplasia	10 (11%)	14 (16%)	8 (9%)	8 (9%)
Epithelium, hyperplasia, basal cell	1 (1%)			1 (1%)
Stomach, glandular	(90)	(89)	(90)	(89)
Erosion	1 (1%)	1 (1%)		
Tongue	(1)	(0)	(0)	(0)
Tooth	(0)	(0)	(1)	(0)
Dysplasia			1 (100%)	
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Dilation			1 (1%)	
Mineral		2 (2%)	1 (1%)	
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	40 (44%)	30 (33%)	39 (43%)	27 (30%)
Atrium, myocardium, hypertrophy		1 (1%)		
Myocardium, hypertrophy		1 (1%)		
Myocardium, mineral		1 (1%)		
Myocardium, necrosis				1 (1%)
Myocardium, Schwann cell, hyperplasia				1 (1%)
Myocardium, ventricle right, degeneration			1 (1%)	
Vein, mineral		1 (1%)		
Ventricle right, cardiomyopathy	4 (4%)	9 (10%)	14 (16%)	15 (17%)

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(90)	(89)	(90)
Accessory adrenal cortical nodule	5 (6%)	7 (8%)	6 (7%)	6 (7%)
Angiectasis		1 (1%)		
Atrophy	1 (1%)	1 (1%)		
Cyst			1 (1%)	
Degeneration, cystic	22 (24%)	26 (29%)	36 (40%)	29 (32%)
Extramedullary hematopoiesis		1 (1%)	1 (1%)	
Hemorrhage		1 (1%)	1 (1%)	
Hyperplasia	14 (16%)	26 (29%)	40 (45%)	26 (29%)
Hypertrophy	52 (58%)	54 (60%)	51 (57%)	56 (62%)
Mineral			1 (1%)	
Necrosis	2 (2%)	4 (4%)	1 (1%)	2 (2%)
Pigment	1 (1%)			
Thrombus		3 (3%)	1 (1%)	
Vacuolation, cytoplasmic	18 (20%)	21 (23%)	11 (12%)	8 (9%)
Adrenal medulla	(86)	(90)	(90)	(86)
Hyperplasia	13 (15%)	19 (21%)	14 (16%)	25 (29%)
Necrosis	1 (1%)			
Islets, pancreatic	(90)	(89)	(90)	(87)
Ectopic tissue			1 (1%)	
Hyperplasia	15 (17%)	6 (7%)	11 (12%)	12 (14%)
Parathyroid gland	(87)	(79)	(82)	(79)
Cyst		1 (1%)		
Fibrosis	13 (15%)	4 (5%)	9 (11%)	6 (8%)
Hyperplasia		1 (1%)	2 (2%)	1 (1%)
Hyperplasia, focal	3 (3%)		2 (2%)	
Hypertrophy		1 (1%)		
Pituitary gland	(90)	(90)	(90)	(90)
Cyst	1 (1%)			
Pars distalis, angiectasis	2 (2%)			
Pars distalis, atrophy		1 (1%)		
Pars distalis, cyst	7 (8%)	3 (3%)	4 (4%)	4 (4%)
Pars distalis, hyperplasia	20 (22%)	26 (29%)	22 (24%)	22 (24%)
Pars distalis, vacuolation, cytoplasmic			1 (1%)	
Pars intermedia, cyst	3 (3%)	3 (3%)	2 (2%)	2 (2%)
Pars intermedia, hyperplasia	1 (1%)		1 (1%)	
Pars intermedia, vacuolation, cytoplasmic			1 (1%)	
Pars nervosa, cyst		1 (1%)		
Thyroid gland	(90)	(88)	(90)	(88)
C-cell, hyperplasia	28 (31%)	49 (56%)	45 (50%)	43 (49%)
Follicle, cyst	1 (1%)	2 (2%)		1 (1%)
General Body System				
Tissue NOS	(8)	(10)	(8)	(10)
Inflammation, chronic active	1 (13%)			
Abdominal, necrosis		1 (10%)		
Fat, necrosis	6 (75%)	8 (80%)	7 (88%)	9 (90%)

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Rats
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Genital System				
Clitoral gland	(87)	(85)	(86)	(87)
Hyperplasia, focal		3 (4%)		
Inflammation, suppurative	1 (1%)	1 (1%)		
Inflammation, granulomatous		1 (1%)		1 (1%)
Inflammation, acute			1 (1%)	1 (1%)
Inflammation, chronic active	28 (32%)	24 (28%)	32 (37%)	40 (46%)
Metaplasia, squamous		1 (1%)		
Duct, dilation	47 (54%)	47 (55%)	44 (51%)	40 (48%)
Ovary	(90)	(90)	(90)	(90)
Atrophy	72 (80%)	63 (70%)	66 (73%)	71 (79%)
Congestion	1 (1%)			
Cyst	22 (24%)	24 (27%)	23 (26%)	27 (30%)
Fibrosis		1 (1%)		
Inflammation, suppurative		1 (1%)		1 (1%)
Inflammation, chronic				2 (2%)
Inflammation, chronic active				1 (1%)
Necrosis				
Bursa, dilation	4 (4%)	5 (6%)	6 (7%)	6 (7%)
Interstitial cell, hyperplasia		2 (2%)		
Periovarian tissue, cyst			1 (1%)	
Periovarian tissue, hemorrhage			1 (1%)	
Periovarian tissue, inflammation,				
chronic active				1 (1%)
Rete ovarii, hyperplasia	15 (17%)	25 (28%)	13 (14%)	12 (13%)
Oviduct	(1)	(0)	(0)	(0)
Cyst	1 (100%)			
Uterus	(90)	(89)	(90)	(90)
Adenomyosis				1 (1%)
Angiectasis	1 (1%)	1 (1%)		
Cyst	5 (6%)	3 (3%)	11 (12%)	7 (8%)
Dilation	8 (9%)	7 (8%)	12 (13%)	4 (4%)
Fibrosis	1 (1%)	1 (1%)	1 (1%)	
Hemorrhage		3 (3%)	4 (4%)	1 (1%)
Hyperplasia, stromal		3 (3%)	1 (1%)	4 (4%)
Inflammation cellular, mononuclear cell			1 (1%)	
Inflammation, suppurative	4 (4%)	11 (12%)	6 (7%)	10 (11%)
Inflammation, acute	1 (1%)			2 (2%)
Inflammation, chronic active		2 (2%)	6 (7%)	1 (1%)
Pigment		1 (1%)		1 (1%)
Thrombus	1 (1%)	2 (2%)		1 (1%)
Artery, inflammation, chronic active				1 (1%)
Cervix, cyst			1 (1%)	1 (1%)
Cervix, hyperplasia, stromal	2 (2%)			
Cervix, serosa, fibrosis	1 (1%)			
Endometrium, hyperplasia, cystic	37 (41%)	33 (37%)	28 (31%)	39 (43%)
Epithelium, metaplasia, squamous	48 (53%)	38 (43%)	39 (43%)	45 (50%)
Serosa, fibrosis			1 (1%)	
Serosa, inflammation, suppurative		1 (1%)		
Vein, thrombus		1 (1%)		
Vagina	(2)	(3)	(1)	(1)
Exudate		1 (33%)		
Inflammation, chronic active				1 (100%)

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hypercellularity	56 (62%)	57 (63%)	55 (61%)	56 (62%)
Myelofibrosis				1 (1%)
Lymph node	(13)	(14)	(21)	(14)
Axillary, hyperplasia, lymphocyte				1 (7%)
Axillary, proliferation, plasma cell	1 (8%)			1 (7%)
Deep cervical, fibrosis				1 (7%)
Deep cervical, inflammation, chronic active				1 (7%)
Iliac, congestion				1 (7%)
Iliac, erythrophagocytosis	3 (23%)	1 (7%)	3 (14%)	2 (14%)
Iliac, hyperplasia, lymphocyte	1 (8%)		2 (10%)	2 (14%)
Iliac, infiltration cellular, histiocyte			1 (5%)	
Iliac, inflammation, acute	1 (8%)			
Iliac, pigment	1 (8%)		1 (5%)	1 (7%)
Iliac, proliferation, plasma cell	6 (46%)	1 (7%)	2 (10%)	
Iliac, lymphatic sinus, ectasia		1 (7%)	3 (14%)	
Inguinal, erythrophagocytosis	1 (8%)			
Inguinal, hyperplasia, lymphocyte		1 (7%)		
Inguinal, infiltration cellular, plasma cell		1 (7%)		
Inguinal, pigment			1 (5%)	
Inguinal, proliferation, plasma cell	1 (8%)			
Inguinal, lymphatic sinus, ectasia	1 (8%)			
Lumbar, erythrophagocytosis	1 (8%)	1 (7%)	1 (5%)	1 (7%)
Lumbar, hyperplasia, lymphocyte		2 (14%)		
Lumbar, infiltration cellular, histiocyte			1 (5%)	
Lumbar, inflammation, chronic active		1 (7%)		
Lumbar, proliferation, plasma cell		2 (14%)	1 (5%)	
Lumbar, lymphatic sinus, ectasia		1 (7%)		
Lymphatic sinus, mediastinal, ectasia			1 (5%)	
Lymphatic sinus, renal, ectasia		1 (7%)		
Mediastinal, congestion	1 (8%)		1 (5%)	
Mediastinal, erythrophagocytosis		3 (21%)	5 (24%)	3 (21%)
Mediastinal, hyperplasia, lymphocyte			2 (10%)	1 (7%)
Mediastinal, proliferation, plasma cell	1 (8%)		3 (14%)	
Pancreatic, erythrophagocytosis	1 (8%)	1 (7%)	1 (5%)	
Renal, erythrophagocytosis		2 (14%)	1 (5%)	2 (14%)
Renal, inflammation, chronic active		1 (7%)		
Lymph node, mandibular	(90)	(89)	(89)	(90)
Congestion				1 (1%)
Erythrophagocytosis		2 (2%)	4 (4%)	3 (3%)
Hemorrhage	1 (1%)			
Hyperplasia, lymphocyte	46 (51%)	40 (45%)	44 (49%)	51 (57%)
Hyperplasia, reticulum cell				1 (1%)
Infiltration cellular, histiocyte		1 (1%)		
Inflammation, chronic active			1 (1%)	
Proliferation, plasma cell	68 (76%)	57 (64%)	65 (73%)	56 (62%)
Lymphatic sinus, ectasia	1 (1%)	3 (3%)	7 (8%)	1 (1%)

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Lymph node, mesenteric	(90)	(90)	(90)	(90)
Atrophy	1 (1%)	1 (1%)		
Erythropagocytosis	1 (1%)	3 (3%)	3 (3%)	5 (6%)
Hemorrhage		1 (1%)		
Hyperplasia, lymphocyte		1 (1%)		1 (1%)
Infiltration cellular, histiocyte	2 (2%)	3 (3%)	2 (2%)	1 (1%)
Necrosis, lymphocyte				1 (1%)
Pigment				1 (1%)
Proliferation, plasma cell		1 (1%)	1 (1%)	
Lymphatic sinus, ectasia		1 (1%)		
Spleen	(90)	(90)	(90)	(90)
Accessory spleen			1 (1%)	
Congestion		2 (2%)	1 (1%)	
Developmental malformation			1 (1%)	
Extramedullary hematopoiesis	80 (89%)	77 (86%)	78 (87%)	78 (87%)
Hemorrhage			1 (1%)	
Hyperplasia, stromal	1 (1%)			
Pigment	74 (82%)	40 (44%)	47 (52%)	46 (51%)
Red pulp, atrophy	7 (8%)	5 (6%)	2 (2%)	1 (1%)
Red pulp, hyperplasia		2 (2%)		
White pulp, atrophy	3 (3%)	6 (7%)	6 (7%)	2 (2%)
Thymus	(87)	(86)	(88)	(86)
Atrophy	75 (86%)	70 (81%)	62 (70%)	61 (71%)
Cyst	39 (45%)	30 (35%)	33 (38%)	28 (33%)
Ectopic parathyroid gland	1 (1%)	1 (1%)	2 (2%)	
Ectopic thyroid		1 (1%)		1 (1%)
Hemorrhage	2 (2%)	2 (2%)	2 (2%)	3 (3%)
Hyperplasia, epithelial	55 (63%)	19 (22%)	19 (22%)	20 (23%)
Necrosis, lymphocyte				1 (1%)
Integumentary System				
Mammary gland	(90)	(89)	(89)	(90)
Galactocele	24 (27%)	18 (20%)	14 (16%)	10 (11%)
Hyperplasia	49 (54%)	41 (46%)	51 (57%)	28 (31%)
Hyperplasia, atypical				3 (3%)
Inflammation, granulomatous			1 (1%)	
Duct, dilation	56 (62%)	52 (58%)	55 (62%)	58 (64%)
Lymphatic, dilation			1 (1%)	
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	1 (1%)	2 (2%)		
Inflammation, acute		1 (1%)		
Inflammation, chronic active	1 (1%)			
Ulcer			1 (1%)	
Dermis, fibrosis		1 (1%)		
Epidermis, hyperplasia	2 (2%)			
Subcutaneous tissue, edema			1 (1%)	1 (1%)
Subcutaneous tissue, inflammation, chronic active			1 (1%)	1 (1%)

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Fibrosis		1 (1%)	1 (1%)	
Fibrous osteodystrophy		1 (1%)	2 (2%)	
Increased bone		1 (1%)	1 (1%)	
Cranium, fracture	1 (1%)			
Mandible, fracture	1 (1%)			
Maxilla, fracture	1 (1%)			
Vertebra, increased bone		1 (1%)		
Vertebra, inflammation, chronic active		1 (1%)		
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	3 (3%)	7 (8%)	4 (4%)	3 (3%)
Mineral				1 (1%)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	26 (29%)	16 (18%)	18 (20%)	11 (12%)
Congestion	1 (1%)	1 (1%)		
Cyst				1 (1%)
Edema	2 (2%)			2 (2%)
Hemorrhage		1 (1%)		1 (1%)
Mineral			1 (1%)	
Necrosis				1 (1%)
Pigment		1 (1%)		
Cerebellum, hemorrhage		1 (1%)		
Glial cell, hyperplasia	1 (1%)			
Hypothalamus, cyst		1 (1%)		
Meninges, hyperplasia	1 (1%)			
Meninges, hyperplasia, granular cell	1 (1%)		1 (1%)	
Pineal gland, mineral	1 (1%)	1 (1%)	3 (3%)	1 (1%)
Pineal gland, vacuolation, cytoplasmic	1 (1%)	3 (3%)	2 (2%)	1 (1%)
Nerve trigeminal	(84)	(88)	(89)	(90)
Degeneration	64 (76%)	71 (81%)	65 (73%)	74 (82%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	80 (89%)	84 (93%)	81 (90%)	84 (93%)
Infiltration cellular, mixed cell	1 (1%)			
Peripheral nerve, tibial	(90)	(90)	(90)	(89)
Degeneration	77 (86%)	83 (92%)	80 (89%)	80 (90%)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Degeneration	24 (27%)	29 (32%)	43 (48%)	23 (26%)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Cyst		1 (1%)		1 (1%)
Degeneration	10 (11%)	7 (8%)	13 (14%)	12 (13%)
Nerve, degeneration	74 (82%)	81 (90%)	70 (78%)	78 (87%)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	59 (66%)	64 (71%)	61 (68%)	65 (72%)
Trigeminal ganglion	(81)	(79)	(80)	(79)
Degeneration	33 (41%)	31 (39%)	28 (35%)	17 (22%)

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Congestion	3 (3%)	5 (6%)	5 (6%)	3 (3%)
Foreign body		2 (2%)		
Hemorrhage	1 (1%)	1 (1%)	4 (4%)	2 (2%)
Inflammation, suppurative	2 (2%)	1 (1%)		
Inflammation, granulomatous	1 (1%)	2 (2%)	3 (3%)	
Inflammation, chronic active	6 (7%)	8 (9%)	9 (10%)	8 (9%)
Pigment				1 (1%)
Alveolar epithelium, metaplasia, squamous			1 (1%)	1 (1%)
Alveolus, infiltration cellular, histiocyte	71 (79%)	75 (83%)	83 (92%)	82 (91%)
Alveolus, pigment			2 (2%)	1 (1%)
Artery, inflammation, chronic active	1 (1%)			
Bronchiole, hyperplasia			1 (1%)	
Epithelium alveolus, hyperplasia	2 (2%)	2 (2%)	6 (7%)	2 (2%)
Interstitium, fibrosis		1 (1%)		
Nose	(90)	(90)	(90)	(90)
Foreign body			1 (1%)	
Inflammation, suppurative	1 (1%)	2 (2%)	3 (3%)	1 (1%)
Inflammation, acute			1 (1%)	1 (1%)
Inflammation, chronic active			1 (1%)	
Nasopharyngeal duct, inflammation, chronic active			1 (1%)	
Nerve, olfactory epithelium, degeneration			1 (1%)	
Olfactory epithelium, accumulation, hyaline droplet	89 (99%)	86 (96%)	88 (98%)	87 (97%)
Olfactory epithelium, atrophy		1 (1%)	1 (1%)	
Olfactory epithelium, degeneration		1 (1%)		
Olfactory epithelium, metaplasia, respiratory	1 (1%)		1 (1%)	
Respiratory epithelium, accumulation, hyaline droplet	12 (13%)	8 (9%)	10 (11%)	10 (11%)
Respiratory epithelium, hyperplasia			1 (1%)	
Respiratory epithelium, metaplasia, squamous			2 (2%)	
Trachea	(89)	(90)	(89)	(87)
Inflammation, chronic active	1 (1%)			1 (1%)
Artery, inflammation, chronic active				
Epithelium, hyperplasia		1 (1%)		
Glands, cyst	1 (1%)			
Special Senses System				
Eye	(88)	(85)	(87)	(87)
Cornea, inflammation, acute	1 (1%)			
Cornea, epithelium, hyperplasia	1 (1%)			
Lens, cataract	1 (1%)			
Retina, atrophy	18 (20%)	15 (18%)	16 (18%)	13 (15%)
Retina, dysplasia	1 (1%)			
Sclera, inflammation, acute			1 (1%)	

TABLE B4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to GSM-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Special Senses System (continued)				
Harderian gland	(90)	(90)	(90)	(90)
Atrophy	13 (14%)	12 (13%)	15 (17%)	24 (27%)
Hyperplasia			1 (1%)	
Infiltration cellular, lymphocyte	2 (2%)			
Inflammation, granulomatous	7 (8%)	9 (10%)	9 (10%)	10 (11%)
Inflammation, acute		1 (1%)		
Inflammation, chronic	7 (8%)	4 (4%)	1 (1%)	2 (2%)
Inflammation, chronic active	1 (1%)	1 (1%)	2 (2%)	2 (2%)
Zymbal's gland	(0)	(0)	(0)	(1)
Urinary System				
Kidney	(90)	(90)	(90)	(89)
Ectopic tissue		1 (1%)		
Infarct		1 (1%)		
Inflammation, granulomatous				1 (1%)
Inflammation, acute	1 (1%)			
Inflammation, chronic active		1 (1%)	1 (1%)	
Mineral				1 (1%)
Necrosis		1 (1%)		
Nephropathy, chronic progressive	74 (82%)	61 (88%)	68 (76%)	59 (66%)
Artery, inflammation, chronic active	1 (1%)			1 (1%)
Pelvis, dilation	3 (3%)	2 (2%)	1 (1%)	
Renal tubule, accumulation, hyaline droplet		2 (2%)	1 (1%)	
Renal tubule, cyst	3 (3%)	2 (2%)	1 (1%)	1 (1%)
Renal tubule, hyperplasia, atypical		1 (1%)		1 (1%)
Renal tubule, hypertrophy		1 (1%)		
Renal tubule, necrosis		2 (2%)		
Renal tubule, pigment		1 (1%)		
Urothelium, hyperplasia				1 (1%)
Urinary bladder	(88)	(88)	(90)	(87)
Dilation	1 (1%)			
Edema		1 (1%)		
Hemorrhage		1 (1%)	1 (1%)	
Infiltration cellular, histiocyte				1 (1%)
Infiltration cellular, mononuclear cell				1 (1%)
Inflammation, acute	3 (3%)		1 (1%)	
Necrosis	1 (1%)			
Urothelium, hyperplasia	1 (1%)	1 (1%)		

APPENDIX C
SUMMARY OF LESIONS IN MALE RATS
EXPOSED TO CDMA-MODULATED
CELL PHONE RFR FOR 2 YEARS

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TABLE C1
Summary of the Incidence of Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
<i>14-Week interim evaluation</i>	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	44	24	13	6
Natural deaths	20	23	21	41
Survivors				
Died last week of study			1	
Terminal euthanasia	25	43	55	43
Animals examined microscopically	100	100	100	100

Systems Examined at 14 Weeks with No Neoplasms Observed

Alimentary System

Cardiovascular System

Endocrine System

General Body System

Genital System

Hematopoietic System

Integumentary System

Musculoskeletal System

Nervous System

Respiratory System

Special Senses System

Urinary System

2-Year Study

Alimentary System

Esophagus	(90)	(90)	(90)	(90)
Intestine large, cecum	(75)	(76)	(74)	(68)
Intestine large, colon	(81)	(83)	(82)	(76)
Intestine large, rectum	(83)	(81)	(80)	(76)
Serosa, sarcoma, metastatic, skeletal muscle				1 (1%)
Intestine small, duodenum	(81)	(84)	(83)	(66)
Adenocarcinoma			1 (1%)	
Osteosarcoma			1 (1%)	
Serosa, sarcoma, metastatic, skeletal muscle				1 (2%)
Intestine small, ileum	(78)	(76)	(77)	(63)
Intestine small, jejunum	(73)	(73)	(75)	(62)
Adenocarcinoma	2 (3%)	1 (1%)		
Liver	(90)	(90)	(89)	(88)
Hepatocellular adenoma		2 (2%)	4 (4%)	
Hepatocellular carcinoma			1 (1%)	1 (1%)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Mesentery	(39)	(19)	(17)	(6)
Oral mucosa	(0)	(1)	(1)	(0)
Squamous cell carcinoma			1 (100%)	

TABLE C1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Pancreas	(90)	(88)	(87)	(78)
Adenoma	13 (14%)	16 (18%)	19 (22%)	5 (6%)
Adenoma, multiple	5 (6%)	6 (7%)	7 (8%)	2 (3%)
Carcinoma	1 (1%)	1 (1%)	2 (2%)	
Pheochromocytoma malignant, metastatic, adrenal medulla		1 (1%)		
Salivary glands	(90)	(90)	(90)	(86)
Parotid gland, adenoma			1 (1%)	
Sublingual gland, schwannoma malignant, metastatic, uncertain primary site	1 (1%)			
Stomach, forestomach	(90)	(90)	(89)	(90)
Squamous cell carcinoma			1 (1%)	
Squamous cell papilloma	1 (1%)			
Stomach, glandular	(86)	(86)	(85)	(78)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Blood vessel	(1)	(2)	(1)	(0)
Heart	(90)	(90)	(90)	(90)
Atrium, schwannoma malignant				1 (1%)
Endocardium, schwannoma malignant		1 (1%)		4 (4%)
Myocardium, schwannoma malignant		1 (1%)	3 (3%)	1 (1%)
Pericardium, schwannoma malignant, metastatic, thymus			1 (1%)	
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(89)
Adenoma	1 (1%)	3 (3%)		2 (2%)
Carcinoma		3 (3%)		
Adrenal medulla	(88)	(90)	(90)	(90)
Pheochromocytoma benign	8 (9%)	17 (19%)	19 (21%)	12 (13%)
Pheochromocytoma benign, multiple	1 (1%)	1 (1%)	2 (2%)	
Pheochromocytoma complex	1 (1%)			
Pheochromocytoma malignant	1 (1%)	3 (3%)	3 (3%)	1 (1%)
Bilateral, pheochromocytoma benign	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Islets, pancreatic	(90)	(88)	(87)	(79)
Adenoma	5 (6%)	11 (13%)	7 (8%)	7 (9%)
Adenoma, multiple			1 (1%)	
Carcinoma	8 (9%)	6 (7%)	13 (15%)	5 (6%)
Carcinoma, multiple				1 (1%)
Parathyroid gland	(83)	(83)	(83)	(82)
Adenoma	1 (1%)	1 (1%)	2 (2%)	1 (1%)
Pituitary gland	(89)	(90)	(90)	(90)
Schwannoma malignant, metastatic, tissue NOS		1 (1%)		1 (1%)
Schwannoma malignant, metastatic, uncertain primary site		1 (1%)		
Pars distalis, adenoma	17 (19%)	25 (28%)	33 (37%)	11 (12%)
Pars distalis, adenoma, multiple			1 (1%)	2 (2%)

TABLE C1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Endocrine System (continued)				
Thyroid gland	(89)	(87) 2 (2%)	(86)	(85)
Bilateral, C-cell, adenoma			1 (1%)	
Bilateral, C-cell, carcinoma			14 (16%)	11 (13%)
C-cell, adenoma	8 (9%)	12 (14%)	1 (1%)	4 (5%)
C-cell, carcinoma	2 (2%)			1 (1%)
Follicular cell, adenoma				
General Body System				
Tissue NOS	(3)	(1) 1 (100%)	(3)	(3) 1 (33%)
Schwannoma malignant				1 (33%)
Fat, schwannoma malignant				
Mediastinum, chemodectoma benign			1 (33%)	
Mediastinum, schwannoma malignant	1 (33%)			
Genital System				
Bulbourethral gland	(1)	(1)	(0)	(0)
Coagulating gland	(0)	(2)	(3)	(0)
Ductus deferens	(1)	(0)	(1)	(0)
Leiomyoma			1 (100%)	
Epididymis	(90)	(90)	(90)	(90)
Penis	(0)	(4)	(2)	(1)
Preputial gland	(88)	(88)	(89)	(89)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Prostate	(90)	(90)	(90)	(85)
Adenoma	2 (2%)		2 (2%)	1 (1%)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Seminal vesicle	(90)	(90)	(90)	(90)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Testis	(90)	(89)	(90)	(90)
Hemangioma			1 (1%)	
Interstitial cell, adenoma	2 (2%)	2 (2%)	1 (1%)	1 (1%)
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Pheochromocytoma malignant, metastatic, adrenal medulla			1 (1%)	
Sarcoma, metastatic, skeletal muscle				1 (1%)
Lymph node	(25)	(23)	(24)	(16)
Iliac, sarcoma, metastatic, skeletal muscle				1 (6%)
Lumbar, sarcoma, metastatic, skeletal muscle				1 (6%)
Lymph node, mandibular	(89)	(90)	(90)	(88)
Lymph node, mesenteric	(90)	(89)	(88)	(88)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Spleen	(90)	(90)	(90)	(85)
Hemangiosarcoma	3 (3%)			
Thymus	(88)	(85)	(87)	(82)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Schwannoma malignant				

TABLE C1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Integumentary System				
Mammary gland	(82)	(77)	(80)	(80)
Fibroadenoma	2 (2%)	5 (6%)	2 (3%)	1 (1%)
Skin	(90)	(90)	(90)	(90)
Basal cell adenoma	1 (1%)	1 (1%)		
Keratoacanthoma	5 (6%)	4 (4%)	5 (6%)	4 (4%)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Squamous cell carcinoma			3 (3%)	
Squamous cell papilloma	2 (2%)	1 (1%)	3 (3%)	1 (1%)
Trichoepithelioma				1 (1%)
Conjunctiva, sarcoma			1 (1%)	
Sebaceous gland, adenoma	1 (1%)			1 (1%)
Subcutaneous tissue, fibroma	2 (2%)	11 (12%)	6 (7%)	4 (4%)
Subcutaneous tissue, fibroma, multiple			1 (1%)	
Subcutaneous tissue, fibrosarcoma	1 (1%)		1 (1%)	
Subcutaneous tissue, hemangiosarcoma		1 (1%)		
Subcutaneous tissue, hibernoma			1 (1%)	
Subcutaneous tissue, lipoma	2 (2%)	5 (6%)	4 (4%)	1 (1%)
Subcutaneous tissue, malignant fibrous histiocytoma				1 (1%)
Subcutaneous tissue, myxosarcoma		2 (2%)		
Subcutaneous tissue, myxosarcoma, multiple			1 (1%)	
Subcutaneous tissue, neural crest tumor			1 (1%)	
Subcutaneous tissue, sarcoma	2 (2%)			
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Bone, vertebra	(0)	(0)	(1)	(0)
Skeletal muscle	(90)	(90)	(90)	(90)
Hemangioma		1 (1%)		
Hemangiosarcoma			1 (1%)	
Sarcoma				1 (1%)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Glioma malignant				3 (3%)
Meningioma benign			1 (1%)	
Schwannoma malignant, metastatic, tissue NOS		1 (1%)		1 (1%)
Schwannoma malignant, metastatic, uncertain primary site		1 (1%)		
Choroid plexus, granular cell tumor benign				1 (1%)
Meninges, granular cell tumor benign	1 (1%)		1 (1%)	1 (1%)
Meninges, granular cell tumor malignant		1 (1%)	1 (1%)	
Nerve trigeminal	(84)	(90)	(88)	(90)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Peripheral nerve, tibial	(88)	(90)	(90)	(89)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(75)	(77)	(79)	(83)
Schwannoma malignant, metastatic, tissue NOS		1 (1%)		

TABLE C1
Summary of the Incidence of Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR or 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Alveolar/bronchiolar adenoma	1 (1%)	2 (2%)	1 (1%)	
Alveolar/bronchiolar carcinoma			1 (1%)	
Carcinoma, metastatic, kidney				1 (1%)
Carcinoma, metastatic, thyroid gland			1 (1%)	
Hepatocellular carcinoma, metastatic, liver				1 (1%)
Pheochromocytoma malignant, metastatic, adrenal medulla			2 (2%)	
Sarcoma, metastatic, skeletal muscle				1 (1%)
Schwannoma malignant, metastatic, thymus			1 (1%)	
Nose	(89)	(90)	(90)	(87)
Schwannoma malignant, metastatic, tissue NOS				1 (1%)
Schwannoma malignant, metastatic, uncertain primary site	1 (1%)	1 (1%)	(88)	(72)
Trachea	(90)	(88)	(88)	
Special Senses System				
Eye	(85)	(83)	(81)	(72)
Schwannoma malignant, metastatic, uncertain primary site		1 (1%)		
Retrobulbar, schwannoma malignant, metastatic, uncertain primary site	2 (2%)			
Harderian gland	(90)	(90)	(90)	(89)
Schwannoma malignant, metastatic, tissue NOS		1 (1%)		1 (1%)
Schwannoma malignant, metastatic, uncertain primary site	2 (2%)	1 (1%)		
Lacrimal gland	(2)	(1)	(1)	(1)
Zymbal's gland	(0)	(0)	(1)	(1)
Adenoma			1 (100%)	1 (100%)
Urinary System				
Kidney	(90)	(90)	(90)	(87)
Lipoma	1 (1%)	2 (2%)	1 (1%)	
Liposarcoma			1 (1%)	
Nephroblastoma				1 (1%)
Oncocytoma benign	1 (1%)			
Bilateral, renal tubule, adenoma			1 (1%)	
Bilateral, renal tubule, adenoma, multiple	1 (1%)			
Bilateral, renal tubule, carcinoma	1 (1%)		1 (1%)	
Pelvis, urothelium, carcinoma				1 (1%)
Perirenal tissue, sarcoma, metastatic, skeletal muscle				1 (1%)
Renal tubule, adenoma	1 (1%)	1 (1%)		
Renal tubule, adenoma, multiple	1 (1%)			
Renal tubule, carcinoma				1 (1%)
Urinary bladder	(89)	(83)	(83)	(78)
Serosa, sarcoma, metastatic, skeletal muscle				1 (1%)

TABLE C1
**Summary of the Incidence of Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Systemic Lesions				
Multiple organs ^b	(90)	(90)	(90)	(90)
Histiocytic sarcoma				1 (1%)
Leukemia mononuclear		2 (2%)	4 (4%)	1 (1%)
Lymphoma malignant	2 (2%)	2 (2%)	1 (1%)	4 (4%)
Mesothelioma malignant	2 (2%)	2 (2%)	3 (3%)	1 (1%)
Neoplasm Summary				
Total animals with primary neoplasms ^c				
2-Year study	56	74	76	63
Total primary neoplasms				
2-Year study	114	160	193	108
Total animals with benign neoplasms				
2-Year study	49	66	69	49
Total benign neoplasms				
2-Year study	87	132	145	71
Total animals with malignant neoplasms				
2-Year study	23	25	41	34
Total malignant neoplasms				
2-Year study	27	27	48	37
Total animals with metastatic neoplasms				
2-Year study	2	3	5	4
Total metastatic neoplasms				
2-Year study	6	10	6	22
Total animals with malignant neoplasms- uncertain primary site				
2-Year study	2	1		
Total animals with uncertain neoplasms- benign or malignant			1	
2-Year study			1	
Total uncertain neoplasms				
2-Year study			1	

^a Number of animals examined microscopically at the site and the number of animals with neoplasm

^b Number of animals with any tissue examined microscopically

^c Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE C2
**Statistical Analysis of Primary Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Medulla: Benign Pheochromocytoma				
Overall rate ^a	10/88 (11%)	19/90 (21%)	22/90 (24%)	13/90 (14%)
Rate per litters ^b	8/35 (23%)	13/35 (37%)	17/35 (49%)	12/35 (34%)
Adjusted rate ^c	15.2%	25.4%	27.8%	19.1%
Terminal rate ^d	3/23 (13%)	14/43 (33%)	16/56 (29%)	9/43 (21%)
First incidence (days)	510	647	642	497
Rao-Scott adjusted poly-3 test ^e	P=0.440	P=0.121	P=0.070	P=0.363
Litter C-A/Fisher's test ^f	P=0.219	P=0.148	P=0.022	P=0.214
Adrenal Medulla: Benign, Complex, or Malignant Pheochromocytoma				
Overall rate	11/88 (13%)	21/90 (23%)	23/90 (26%)	14/90 (16%)
Rate per litters	9/35 (26%)	15/35 (43%)	18/35 (51%)	13/35 (37%)
Adjusted rate	16.7%	28.1%	29.1%	20.4%
Terminal rate	3/23 (13%)	16/43 (37%)	17/56 (30%)	9/43 (21%)
First incidence (days)	510	647	642	497
Rao-Scott adjusted poly-3 test	P=0.474	P=0.091	P=0.070	P=0.368
Litter C-A/Fisher's test	P=0.251	P=0.104	P=0.024	P=0.220
Heart: Schwannoma Malignant				
Overall rate	0/90 (0%)	2/90 (2%)	3/90 (3%)	6/90 (7%)
Rate per litters	0/35 (0%)	2/35 (6%)	3/35 (9%)	6/35 (17%)
Adjusted rate	0%	2.7%	3.8%	8.8%
Terminal rate	0/25 (0%)	2/43 (5%)	2/56 (4%)	3/43 (7%)
First incidence (days)	— ^g	730 (T)	642	488
Rao-Scott adjusted poly-3 test	P=0.011	P=0.273	P=0.175	P=0.030
Litter C-A/Fisher's test	P=0.006	P=0.246	P=0.120	P=0.012
Mammary Gland: Fibroadenoma				
Overall rate	2/90 (2%)	5/90 (6%)	2/90 (2%)	1/90 (1%)
Rate per litters	2/35 (6%)	5/35 (14%)	2/35 (6%)	1/35 (3%)
Adjusted rate	3%	6.7%	2.6%	1.5%
Terminal rate	1/25 (4%)	4/43 (9%)	2/56 (4%)	1/43 (2%)
First incidence (days)	440	550	730 (T)	730 (T)
Rao-Scott adjusted poly-3 test	P=0.229N	P=0.271	P=0.608N	P=0.468N
Litter C-A/Fisher's test	P=0.219N	P=0.214	P=0.693	P=0.500N
Pancreas: Adenoma				
Overall rate	18/90 (20%)	22/88 (25%)	26/87 (30%)	7/78 (9%)
Rate per litters	16/35 (46%)	16/35 (46%)	18/35 (51%)	7/35 (20%)
Adjusted rate	26.8%	29.6%	33.5%	11.2%
Terminal rate	9/25 (36%)	13/43 (30%)	20/56 (36%)	5/43 (12%)
First incidence (days)	580	568	577	621
Rao-Scott adjusted poly-3 test	P=0.034N	P=0.436	P=0.268	P=0.035N
Litter C-A/Fisher's test	P=0.015N	P=0.595	P=0.406	P=0.020N
Pancreas: Adenoma or Carcinoma				
Overall rate	18/90 (20%)	22/88 (25%)	27/87 (31%)	7/78 (9%)
Rate per litters	16/35 (46%)	16/35 (46%)	18/35 (51%)	7/35 (20%)
Adjusted rate	26.8%	29.6%	34.8%	11.2%
Terminal rate	9/25 (36%)	13/43 (30%)	21/56 (38%)	5/43 (12%)
First incidence (days)	580	568	577	621
Rao-Scott adjusted poly-3 test	P=0.037N	P=0.436	P=0.223	P=0.035N
Litter C-A/Fisher's test	P=0.015N	P=0.595	P=0.406	P=0.020N

TABLE C2
**Statistical Analysis of Primary Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	11/88 (13%)	8/87 (9%)	7/79 (9%)
Rate per litters	5/35 (14%)	8/35 (23%)	6/35 (17%)	7/35 (20%)
Adjusted rate	7.6%	15.1%	10.5%	11.2%
Terminal rate	2/25 (8%)	8/43 (19%)	7/56 (13%)	6/43 (14%)
First incidence (days)	624	605	726	665
Rao-Scott adjusted poly-3 test	P=0.458	P=0.167	P=0.396	P=0.364
Litter C-A/Fisher's test	P=0.413	P=0.270	P=0.500	P=0.376
Pancreatic Islets: Carcinoma				
Overall rate	8/90 (9%)	6/88 (7%)	13/87 (15%)	6/79 (8%)
Rate per litters	8/35 (23%)	6/35 (17%)	12/35 (34%)	6/35 (17%)
Adjusted rate	12%	8.3%	17%	9.5%
Terminal rate	3/25 (12%)	6/43 (14%)	10/56 (18%)	3/43 (7%)
First incidence (days)	663	730 (T)	642	582
Rao-Scott adjusted poly-3 test	P=0.512N	P=0.313N	P=0.268	P=0.411N
Litter C-A/Fisher's test	P=0.446N	P=0.383N	P=0.214	P=0.383N
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	13/90 (14%)	17/88 (19%)	18/87 (21%)	13/79 (16%)
Rate per litters	12/35 (34%)	14/35 (40%)	14/35 (40%)	11/35 (31%)
Adjusted rate	19.4%	23.3%	23.5%	20.6%
Terminal rate	5/25 (20%)	14/43 (33%)	14/56 (25%)	9/43 (21%)
First incidence (days)	624	605	642	582
Rao-Scott adjusted poly-3 test	P=0.510	P=0.357	P=0.343	P=0.506
Litter C-A/Fisher's test	P=0.395N	P=0.402	P=0.402	P=0.500N
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	17/89 (19%)	25/90 (28%)	34/90 (38%)	13/90 (14%)
Rate per litters	13/35 (37%)	18/35 (51%)	24/35 (69%)	12/35 (34%)
Adjusted rate	24.9%	32.7%	41.8%	19%
Terminal rate	5/25 (20%)	16/43 (37%)	22/56 (39%)	6/43 (14%)
First incidence (days)	527	605	471	567
Rao-Scott adjusted poly-3 test	P=0.226N	P=0.208	P=0.030	P=0.273N
Litter C-A/Fisher's test	P=0.398N	P=0.168	P=0.008	P=0.500N
Skin: Keratoacanthoma				
Overall rate	5/90 (6%)	4/90 (4%)	5/90 (6%)	4/90 (4%)
Rate per litters	5/35 (14%)	3/35 (9%)	5/35 (14%)	3/35 (9%)
Adjusted rate	7.4%	5.4%	6.4%	6%
Terminal rate	0/25 (0%)	2/43 (5%)	4/56 (7%)	3/43 (7%)
First incidence (days)	552	610	726	665
Rao-Scott adjusted poly-3 test	P=0.477N	P=0.430N	P=0.521N	P=0.490N
Litter C-A/Fisher's test	P=0.360N	P=0.355N	P=0.633	P=0.355N
Skin: Squamous Cell Papilloma or Keratoacanthoma				
Overall rate	7/90 (8%)	5/90 (6%)	8/90 (9%)	5/90 (6%)
Rate per litters	7/35 (20%)	4/35 (11%)	8/35 (23%)	4/35 (11%)
Adjusted rate	10.4%	6.7%	10.2%	7.5%
Terminal rate	1/25 (4%)	3/43 (7%)	6/56 (11%)	4/43 (9%)
First incidence (days)	552	610	717	665
Rao-Scott adjusted poly-3 test	P=0.413N	P=0.302N	P=0.577N	P=0.372N
Litter C-A/Fisher's test	P=0.308N	P=0.256N	P=0.500	P=0.256N

TABLE C2
Statistical Analysis of Primary Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Skin: Squamous Cell Papilloma, Keratoacanthoma, or Squamous Cell Carcinoma				
Overall rate	7/90 (8%)	5/90 (6%)	10/90 (11%)	5/90 (6%)
Rate per litters	7/35 (20%)	4/35 (11%)	10/35 (29%)	4/35 (11%)
Adjusted rate	10.4%	6.7%	12.8%	7.5%
Terminal rate	1/25 (4%)	3/43 (7%)	7/56 (13%)	4/43 (9%)
First incidence (days)	552	610	717	665
Rao-Scott adjusted poly-3 test	P=0.443N	P=0.297N	P=0.410	P=0.368N
Litter C-A/Fisher's test	P=0.341N	P=0.256N	P=0.289	P=0.256N
Skin: Squamous Cell Papilloma, Keratoacanthoma, Trichoepithelioma, Basal Cell Adenoma or Squamous Cell Carcinoma				
Overall rate	8/90 (9%)	6/90 (7%)	10/90 (11%)	6/90 (7%)
Rate per litters	8/35 (23%)	5/35 (14%)	10/35 (29%)	5/35 (14%)
Adjusted rate	11.9%	8%	12.8%	9%
Terminal rate	2/25 (8%)	4/43 (9%)	7/56 (13%)	4/43 (9%)
First incidence (days)	552	610	717	630
Rao-Scott adjusted poly-3 test	P=0.430N	P=0.296N	P=0.516	P=0.371N
Litter C-A/Fisher's test	P=0.334N	P=0.270N	P=0.393	P=0.270N
Skin (Subcutaneous Tissue): Lipoma				
Overall rate	2/90 (2%)	5/90 (6%)	4/90 (4%)	1/90 (1%)
Rate per litters	2/35 (6%)	3/35 (9%)	4/35 (11%)	1/35 (3%)
Adjusted rate	3.1%	6.7%	5.1%	1.5%
Terminal rate	2/25 (8%)	3/43 (7%)	3/56 (5%)	0/43 (0%)
First incidence (days)	730 (T)	693	726	713
Rao-Scott adjusted poly-3 test	P=0.331N	P=0.350	P=0.480	P=0.536N
Litter C-A/Fisher's test	P=0.370N	P=0.500	P=0.337	P=0.500N
Skin (Subcutaneous Tissue): Fibroma				
Overall rate	2/90 (2%)	11/90 (12%)	7/90 (8%)	4/90 (4%)
Rate per litters	2/35 (6%)	11/35 (31%)	7/35 (20%)	4/35 (11%)
Adjusted rate	3.1%	14.4%	8.8%	6%
Terminal rate	2/25 (8%)	5/43 (12%)	6/56 (11%)	2/43 (5%)
First incidence (days)	730 (T)	447	383	665
Rao-Scott adjusted poly-3 test	P=0.500N	P=0.025	P=0.143	P=0.328
Litter C-A/Fisher's test	P=0.500N	P=0.006	P=0.075	P=0.337
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, Myxosarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	13/90 (14%)	10/90 (11%)	5/90 (6%)
Rate per litters	5/35 (14%)	12/35 (34%)	10/35 (29%)	5/35 (14%)
Adjusted rate	7.5%	16.9%	12.6%	7.5%
Terminal rate	2/25 (8%)	6/43 (14%)	8/56 (14%)	3/43 (7%)
First incidence (days)	567	447	383	665
Rao-Scott adjusted poly-3 test	P=0.335N	P=0.081	P=0.236	P=0.600N
Litter C-A/Fisher's test	P=0.342N	P=0.046	P=0.122	P=0.633
Thyroid Gland (C-cell): Adenoma				
Overall rate	8/89 (9%)	14/87 (16%)	14/86 (16%)	11/85 (13%)
Rate per litters	7/35 (20%)	13/35 (37%)	13/34 (38%)	10/35 (29%)
Adjusted rate	12.1%	18.7%	18.5%	16.8%
Terminal rate	6/25 (24%)	5/43 (12%)	13/56 (23%)	8/43 (19%)
First incidence (days)	498	517	677	582
Rao-Scott adjusted poly-3 test	P=0.327	P=0.186	P=0.194	P=0.278
Litter C-A/Fisher's test	P=0.363	P=0.093	P=0.080	P=0.289

TABLE C2
**Statistical Analysis of Primary Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Thyroid Gland (C-cell): Carcinoma				
Overall rate	2/89 (2%)	0/87 (0%)	2/86 (2%)	4/85 (5%)
Rate per litters	1/35 (3%)	0/35 (0%)	1/34 (3%)	4/35 (11%)
Adjusted rate	3%	0%	2.6%	6.2%
Terminal rate	0/25 (0%)	0/43 (0%)	2/56 (4%)	3/43 (7%)
First incidence (days)	541	— ^g	730 (T)	717
Rao-Scott adjusted poly-3 test	P=0.173	P=0.339N	P=0.666N	P=0.398
Litter C-A/Fisher's test	P=0.034	P=0.500N	P=0.746	P=0.178
Thyroid Gland (C-cell): Adenoma or Carcinoma				
Overall rate	10/89 (11%)	14/87 (16%)	16/86 (19%)	15/85 (18%)
Rate per litters	8/35 (23%)	13/35 (37%)	13/34 (38%)	12/35 (34%)
Adjusted rate	14.9%	18.7%	21.1%	22.9%
Terminal rate	6/25 (24%)	5/43 (12%)	15/56 (27%)	11/43 (26%)
First incidence (days)	498	517	677	582
Rao-Scott adjusted poly-3 test	P=0.151	P=0.351	P=0.233	P=0.172
Litter C-A/Fisher's test	P=0.249	P=0.148	P=0.130	P=0.214
All Organs: Malignant Schwannoma				
Overall rate	3/90 (3%)	4/90 (4%)	4/90 (4%)	8/90 (9%)
Rate per litters	3/35 (9%)	4/35 (11%)	4/35 (11%)	7/35 (20%)
Adjusted rate	4.5%	5.4%	5.1%	11.6%
Terminal rate	1/25 (4%)	2/43 (5%)	2/56 (4%)	4/43 (9%)
First incidence (days)	555	573	619	153
Rao-Scott adjusted poly-3 test	P=0.075	P=0.551	P=0.582	P=0.136
Litter C-A/Fisher's test	P=0.100	P=0.500	P=0.500	P=0.153

TABLE C2
**Statistical Analysis of Primary Neoplasms in Male Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

(T) Terminal euthanasia

- a Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, heart, pancreas, pancreatic islets, pituitary gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
- b Number of litters with tumor-bearing animals/number of litters examined at site
- c Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
- d Observed incidence at terminal euthanasia
- e Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott adjusted poly-3 test is a modification of the Poly-3 test that also incorporates an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
- f The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
- g Not applicable; no neoplasms in animal group

TABLE C3a
Historical Incidence of Malignant Schwannoma of the Heart in Control Male Hsd:Sprague Dawley SD Rats^a

		Incidence in Controls
Overall Historical Incidence: All Routes		
Total (%)		2/240 (0.8%)
Mean ± standard deviation		1.0% ± 1.2%
Range		0%-2%

^a Data as of November 2017

TABLE C3b
Historical Incidence of Brain Neoplasms in Control Male Hsd:Sprague Dawley SD Rats^a

	Malignant Glioma	Benign Granular Cell Tumor	Malignant Granular Cell Tumor	Benign or Malignant Granular Cell Tumor
Overall Historical Incidence: All Routes				
Total (%)	2/190 (1.3%)	3/190 (1.7%)	0/240	3/190 (1.7%)
Mean ± standard deviation	1.3% ± 2.3%	1.7% ± 2.1%		1.7% ± 2.1%
Range	0%-4%	0%-4%		0%-4%

^a Data as of November 2017

TABLE C3c
**Historical Incidence of Pituitary Gland (Pars Distalis) Adenoma
in Control Male Hsd:Sprague Dawley SD Rats^a**

		Incidence in Controls
Overall Historical Incidence: All Routes		
Total (%)		47/239 (19.7%)
Mean ± standard deviation		19.8% ± 7.5%
Range		10%-28%

^a Data as of November 2017

TABLE C3d**Historical Incidence of Hepatocellular Neoplasms in Control Male Hsd:Sprague Dawley SD Rats^a**

	Adenoma	Carcinoma	Adenoma or Carcinoma
Overall Historical Incidence: All Routes			
Total (%)	1/240 (0.4%)	0/240	1/240 (0.4%)
Mean ± standard deviation	0.5% ± 1.0%		0.5% ± 1.0%
Range	0%-2%		0%-2%

^a Data as of November 2017

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	44	24	13	6
Natural deaths	20	23	21	41
Survivors				
Died last week of study			1	
Terminal euthanasia	25	43	55	43
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(9)	(10)
Intestine large, cecum	(10)	(10)	(9)	(10)
Intestine large, colon	(10)	(10)	(9)	(10)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)	1 (10%)		1 (10%)
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(9)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)
Hepatodiaphragmatic nodule			1 (10%)	
Infiltration cellular, mixed cell	1 (10%)	1 (10%)	1 (10%)	3 (30%)
Hepatocyte, necrosis			1 (10%)	
Pancreas	(10)	(10)	(10)	(10)
Salivary glands	(10)	(10)	(9)	(10)
Stomach, forestomach	(10)	(10)	(10)	(10)
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy	2 (20%)		3 (30%)	6 (60%)
Artery, inflammation, chronic active		1 (10%)	1 (10%)	
Ventricle right, cardiomyopathy	1 (10%)	5 (50%)	4 (40%)	4 (40%)
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(9)	(9)	(9)	(10)
Pituitary gland	(10)	(10)	(10)	(10)
Pars intermedia, cyst		1 (10%)		2 (20%)
Rathke's cleft, cyst		1 (10%)	1 (10%)	
Thyroid gland	(10)	(10)	(9)	(10)

^a Number of animals examined microscopically at the site and the number of animals with lesion

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
14-Week Interim Evaluation (continued)				
Genital System				
Epididymis	(10)	(10)	(10) 1 (10%)	(10)
Granuloma sperm				1 (10%)
Hypospermia				
Preputial gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	7 (70%)	3 (30%)	6 (60%)	3 (30%)
Prostate	(10)	(10)	(10)	(10)
Inflammation, chronic active				1 (10%)
Seminal vesicle	(10)	(10)	(10)	(10)
Testis	(10)	(10)	(10)	(10)
Germ cell, degeneration				1 (10%)
Hematopoietic System				
Bone marrow	(10)	(10)	(9)	(10)
Lymph node	(0)	(0)	(1)	(0)
Inguinal, pigment			1 (100%)	
Lymph node, mandibular	(10)	(10)	(9)	(10)
Hemorrhage	2 (20%)	1 (10%)		1 (10%)
Lymph node, mesenteric	(10)	(10)	(9)	(10)
Hemorrhage				1 (10%)
Spleen	(10)	(10)	(9)	(10)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	5 (50%)	3 (30%)	4 (40%)	2 (20%)
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion		1 (10%)		1 (10%)
Inflammation, chronic active	1 (10%)		1 (10%)	1 (10%)
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(9)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Retina, developmental malformation		1 (10%)		
Harderian gland	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte		1 (10%)		
Inflammation, chronic active		1 (10%)	3 (30%)	1 (10%)
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Congestion			2 (20%)	
Nephropathy, chronic progressive	9 (90%)	7 (70%)	8 (80%)	9 (90%)
Pelvis, dilation				1 (10%)
Urinary bladder	(10)	(10)	(10)	(10)

Systems Examined with No Lesions Observed

General Body System

Integumentary System

Musculoskeletal System

Nervous System

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Dilation	2 (2%)			
Hyperplasia	1 (1%)			
Intestine large, cecum	(75)	(76)	(74)	(68)
Edema	11 (15%)			
Erosion	10 (13%)	1 (1%)	1 (1%)	1 (1%)
Hemorrhage		1 (1%)		
Inflammation, acute	10 (13%)	1 (1%)		1 (1%)
Inflammation, chronic active	1 (1%)	1 (1%)		
Necrosis			1 (1%)	
Ulcer	6 (8%)			
Artery, inflammation, chronic active	20 (27%)	8 (11%)	7 (9%)	2 (3%)
Artery, mineral	1 (1%)			
Artery, thrombus			1 (1%)	
Epithelium, regeneration	14 (19%)	1 (1%)		1 (1%)
Intestine large, colon	(81)	(83)	(82)	(76)
Cyst		1 (1%)		
Erosion	1 (1%)	1 (1%)		
Inflammation, acute	1 (1%)			
Ulcer	1 (1%)			
Artery, inflammation, chronic active	12 (15%)	4 (5%)	5 (6%)	1 (1%)
Artery, mineral	2 (2%)			
Epithelium, regeneration	5 (6%)			
Intestine large, rectum	(83)	(81)	(80)	(76)
Edema	1 (1%)			
Erosion	1 (1%)			
Hyperplasia, lymphocyte	1 (1%)			
Inflammation, acute	2 (2%)			
Inflammation, chronic active		1 (1%)		
Artery, inflammation, chronic active	4 (5%)	1 (1%)	1 (1%)	1 (1%)
Epithelium, regeneration	3 (4%)			
Intestine small, duodenum	(81)	(84)	(83)	(66)
Dilation		1 (1%)		
Ectopic tissue		1 (1%)		
Erosion	1 (1%)			
Ulcer	1 (1%)	1 (1%)		
Artery, inflammation, chronic active			3 (4%)	
Intestine small, ileum	(78)	(76)	(77)	(63)
Congestion		1 (1%)		
Hemorrhage			1 (1%)	
Inflammation, acute		1 (1%)		
Artery, inflammation, chronic active	2 (3%)		1 (1%)	
Epithelium, regeneration	1 (1%)			
Intestine small, jejunum	(73)	(73)	(75)	(62)
Artery, inflammation, chronic active			1 (1%)	
Liver	(90)	(90)	(89)	(88)
Angiectasis	1 (1%)	1 (1%)		1 (1%)
Basophilic focus	1 (1%)		2 (2%)	
Clear cell focus	8 (9%)	4 (4%)	5 (6%)	5 (6%)
Eosinophilic focus	12 (13%)	5 (6%)	11 (12%)	4 (5%)
Extramedullary hematopoiesis	5 (6%)	4 (4%)	3 (3%)	1 (1%)
Hepatodiaphragmatic nodule	1 (1%)	1 (1%)		1 (1%)
Infiltration cellular, mixed cell	3 (3%)	1 (1%)	3 (3%)	2 (2%)
Mixed cell focus	32 (36%)	51 (57%)	47 (53%)	37 (42%)
Artery, inflammation, chronic active	2 (2%)	1 (1%)		
Artery, mineral	1 (1%)	1 (1%)		

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Liver (continued)	(90)	(90)	(89)	(88)
Bile duct, cyst	3 (3%)	5 (6%)	2 (2%)	1 (1%)
Bile duct, fibrosis				1 (1%)
Bile duct, hyperplasia	41 (46%)	33 (37%)	26 (29%)	14 (16%)
Hepatocyte, degeneration	1 (1%)		1 (1%)	1 (1%)
Hepatocyte, necrosis	5 (6%)	6 (7%)	6 (7%)	6 (7%)
Hepatocyte, vacuolation, cytoplasmic	6 (7%)	6 (7%)	7 (8%)	7 (8%)
Kupffer cell, pigment	1 (1%)			
Periductal, cholangiofibrosis	2 (2%)	2 (2%)	2 (2%)	
Mesentery	(39)	(19)	(17)	(6)
Fibrosis		1 (5%)		
Hemorrhage	1 (3%)			1 (17%)
Inflammation, chronic	2 (5%)			
Necrosis	2 (5%)	1 (5%)	1 (6%)	1 (17%)
Neovascularization	1 (3%)	2 (11%)	3 (18%)	
Artery, inflammation, chronic active	32 (82%)	16 (84%)	13 (76%)	3 (50%)
Artery, mineral	21 (54%)	5 (26%)	2 (12%)	
Vein, degeneration	1 (3%)			
Vein, inflammation, chronic active	1 (3%)	2 (11%)	1 (6%)	
Oral mucosa	(0)	(1)	(1)	(0)
Ulcer		1 (100%)		
Pancreas	(90)	(88)	(87)	(78)
Cyst	1 (1%)			1 (1%)
Inflammation, chronic active		1 (1%)		
Thrombus	1 (1%)	1 (1%)		
Acinus, atrophy	13 (14%)	9 (10%)	10 (11%)	8 (10%)
Acinus, hyperplasia	63 (70%)	55 (63%)	49 (56%)	28 (36%)
Artery, inflammation, chronic active	48 (53%)	28 (32%)	23 (26%)	5 (6%)
Artery, mineral	11 (12%)	2 (2%)		
Duct, crystals			1 (1%)	
Duct, inflammation, acute			1 (1%)	
Salivary glands	(90)	(90)	(90)	(86)
Artery, inflammation, chronic active	11 (12%)	6 (7%)	2 (2%)	1 (1%)
Artery, mineral	2 (2%)	1 (1%)	1 (1%)	
Duct, parotid gland, dilation	5 (6%)	1 (1%)	1 (1%)	
Duct, parotid gland, inflammation, acute	1 (1%)	1 (1%)		
Parotid gland, atrophy	18 (20%)	15 (17%)	8 (9%)	3 (3%)
Parotid gland, inflammation, acute	2 (2%)	4 (4%)	2 (2%)	
Parotid gland, vacuolation, cytoplasmic	1 (1%)	2 (2%)		
Sublingual gland, atrophy			1 (1%)	1 (1%)
Sublingual gland, mineral				1 (1%)
Submandibular gland, atrophy		2 (2%)		
Stomach, forestomach	(90)	(90)	(89)	(90)
Cyst		1 (1%)		
Edema	5 (6%)	5 (6%)	1 (1%)	1 (1%)
Erosion		1 (1%)		
Inflammation, acute	1 (1%)	1 (1%)	1 (1%)	
Inflammation, chronic				1 (1%)
Inflammation, chronic active	7 (8%)	4 (4%)	10 (11%)	1 (1%)
Mineral	1 (1%)	1 (1%)		
Ulcer	6 (7%)	8 (9%)	4 (4%)	1 (1%)
Artery, inflammation, chronic active		1 (1%)		
Epithelium, hyperplasia	11 (12%)	17 (19%)	11 (12%)	6 (7%)
Epithelium, hyperplasia, atypical	1 (1%)			
Epithelium, hyperplasia, basal cell			1 (1%)	1 (1%)

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Stomach, glandular	(86)	(86)	(85)	(78)
Erosion	3 (3%)	2 (2%)	3 (4%)	
Inflammation, acute	1 (1%)			
Inflammation, chronic active	1 (1%)			
Mineral	31 (36%)	9 (10%)	6 (7%)	1 (1%)
Necrosis			3 (4%)	
Artery, inflammation, chronic active	3 (3%)			
Artery, mineral			1 (1%)	
Epithelium, hyperplasia, focal				1 (1%)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Dilation		5 (6%)	1 (1%)	
Mineral	30 (33%)	8 (9%)	6 (7%)	2 (2%)
Blood vessel	(1)	(2)	(1)	(0)
Inflammation, chronic active			1 (100%)	
Mineral	1 (100%)			
Pulmonary artery, mineral		1 (50%)		
Pulmonary artery, necrosis		1 (50%)		
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	79 (88%)	84 (93%)	83 (92%)	85 (94%)
Congestion	1 (1%)			
Hemorrhage				1 (1%)
Inflammation, suppurative			1 (1%)	
Thrombus	1 (1%)		3 (3%)	
Artery, degeneration		1 (1%)		
Artery, inflammation, chronic active			2 (2%)	
Artery, mineral	20 (22%)	7 (8%)	2 (2%)	1 (1%)
Artery, pericardium, inflammation, chronic active				1 (1%)
Artery, pericardium, pigment		1 (1%)		
Atrium, dilation	3 (3%)	1 (1%)		4 (4%)
Atrium, thrombus	1 (1%)	5 (6%)		1 (1%)
Atrium, myocardium, hypertrophy	1 (1%)	1 (1%)		1 (1%)
Atrium, myocardium, necrosis		1 (1%)		
Atrium left, mineral			1 (1%)	
Endocardium, hyperplasia, Schwann cell				3 (3%)
Myocardium, mineral	9 (10%)	2 (2%)	1 (1%)	
Myocardium, necrosis	1 (1%)	1 (1%)		1 (1%)
Pericardium, hemorrhage			1 (1%)	
Valve, inflammation, chronic active	1 (1%)			
Ventricle right, cardiomyopathy	54 (60%)	45 (50%)	62 (69%)	74 (82%)
Ventricle right, dilation			1 (1%)	

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(89)
Accessory adrenal cortical nodule	6 (7%)	4 (4%)	7 (8%)	7 (8%)
Angiectasis		1 (1%)		
Atrophy		1 (1%)		1 (1%)
Degeneration	3 (3%)	1 (1%)	1 (1%)	2 (2%)
Degeneration, cystic		3 (3%)		1 (1%)
Extramedullary hematopoiesis			1 (1%)	
Hyperplasia	47 (52%)	42 (47%)	45 (50%)	44 (49%)
Hypertrophy	35 (39%)	42 (47%)	55 (61%)	44 (49%)
Necrosis	5 (6%)	5 (6%)	1 (1%)	1 (1%)
Pigment				1 (1%)
Thrombus	2 (2%)	2 (2%)	1 (1%)	
Vacuolation, cytoplasmic	20 (22%)	18 (20%)	21 (23%)	12 (13%)
Adrenal medulla	(88)	(90)	(90)	(90)
Hyperplasia	42 (48%)	34 (38%)	32 (36%)	21 (23%)
Thrombus	1 (1%)			
Islets, pancreatic	(90)	(88)	(87)	(79)
Hyperplasia	12 (13%)	15 (17%)	13 (15%)	12 (15%)
Parathyroid gland	(83)	(83)	(83)	(82)
Fibrosis			3 (4%)	
Hyperplasia	51 (61%)	35 (42%)	32 (39%)	17 (21%)
Hyperplasia, focal		1 (1%)		
Pituitary gland	(89)	(90)	(90)	(90)
Craniopharyngeal duct, cyst	1 (1%)			1 (1%)
Pars distalis, angiectasis				1 (1%)
Pars distalis, atrophy				1 (1%)
Pars distalis, cyst	5 (6%)	15 (17%)	7 (8%)	6 (7%)
Pars distalis, hyperplasia	32 (36%)	32 (36%)	34 (38%)	27 (30%)
Pars distalis, necrosis		1 (1%)		
Pars intermedia, angiectasis	1 (1%)	1 (1%)		
Pars intermedia, cyst	6 (7%)	1 (1%)	5 (6%)	7 (8%)
Pars intermedia, hyperplasia	1 (1%)	3 (3%)		2 (2%)
Pars nervosa, cyst		1 (1%)		
Thyroid gland	(89)	(87)	(86)	(85)
C-cell, hyperplasia	16 (18%)	17 (20%)	17 (20%)	22 (26%)
Follicle, cyst		2 (2%)		1 (1%)
Follicle, hyperplasia, cystic	1 (1%)			
General Body System				
Tissue NOS	(3)	(1)	(3)	(3)
Abdominal, fat, hemorrhage	1 (33%)			
Fat, hemorrhage			1 (33%)	
Fat, necrosis	2 (67%)		1 (33%)	1 (33%)
Genital System				
Bulbourethral gland	(1)	(1)	(0)	(0)
Coagulating gland	(0)	(2)	(3)	(0)
Inflammation, suppurative			1 (33%)	
Inflammation, chronic active		2 (100%)	2 (67%)	
Ductus deferens	(1)	(0)	(1)	(0)
Granuloma	1 (100%)			

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Genital System (continued)				
Epididymis	(90)	(90)	(90)	(90)
Exfoliated germ cell	51 (57%)	33 (37%)	33 (37%)	17 (19%)
Granuloma sperm	1 (1%)	1 (1%)		
Hypospermia	28 (31%)	24 (27%)	13 (14%)	13 (14%)
Inflammation, chronic				1 (1%)
Inflammation, chronic active			1 (1%)	
Artery, inflammation, chronic active	2 (2%)	3 (3%)	3 (3%)	3 (3%)
Artery, thrombus				1 (1%)
Tail, developmental malformation		1 (1%)		
Penis	(0)	(4)	(2)	(1)
Concretion		3 (75%)	2 (100%)	1 (100%)
Prolapse		1 (25%)		
Preputial gland	(88)	(88)	(89)	(89)
Atrophy	1 (1%)	1 (1%)		
Fibrosis			2 (2%)	
Hyperplasia	1 (1%)			
Inflammation, suppurative		1 (1%)		
Inflammation, granulomatous	1 (1%)			
Inflammation, acute	1 (1%)			
Inflammation, chronic active	46 (52%)	53 (60%)	46 (52%)	49 (55%)
Metaplasia, squamous			1 (1%)	
Artery, inflammation, chronic active	1 (1%)			
Duct, dilation	51 (58%)	54 (61%)	50 (56%)	48 (54%)
Duct, hyperplasia		1 (1%)		1 (1%)
Prostate	(90)	(90)	(90)	(85)
Decreased secretory fluid	4 (4%)	5 (6%)	7 (8%)	3 (4%)
Hemorrhage	1 (1%)		1 (1%)	
Infiltration cellular, mononuclear cell	1 (1%)			1 (1%)
Inflammation, acute	7 (8%)	9 (10%)	4 (4%)	2 (2%)
Inflammation, chronic active	6 (7%)	10 (11%)	10 (11%)	5 (6%)
Artery, inflammation, chronic active	1 (1%)		3 (3%)	
Artery, thrombus		1 (1%)		
Epithelium, hyperplasia	5 (6%)	11 (12%)	9 (10%)	15 (18%)
Seminal vesicle	(90)	(90)	(90)	(90)
Decreased secretory fluid	35 (39%)	34 (38%)	18 (20%)	7 (8%)
Developmental malformation			1 (1%)	
Dilation			1 (1%)	
Hemorrhage	1 (1%)		1 (1%)	
Hyperplasia, atypical				1 (1%)
Inflammation, acute	4 (4%)	1 (1%)	3 (3%)	1 (1%)
Inflammation, chronic active	1 (1%)	4 (4%)		
Artery, inflammation, chronic active	1 (1%)			
Epithelium, hyperplasia	1 (1%)			
Lumen, hemorrhage			1 (1%)	
Testis	(90)	(89)	(90)	(90)
Cyst	1 (1%)			
Edema		2 (2%)		
Inflammation, chronic active	2 (2%)			
Pigment	1 (1%)			
Artery, inflammation, chronic active	52 (58%)	37 (42%)	30 (33%)	12 (13%)
Germ cell, degeneration	51 (57%)	37 (42%)	31 (34%)	24 (27%)
Germinal epithelium, mineral		1 (1%)		
Interstitial cell, hyperplasia	1 (1%)	2 (2%)		1 (1%)
Rete testis, dilation	1 (1%)			
Seminiferous tubule, dilation	1 (1%)	1 (1%)	1 (1%)	

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hemorrhage		5 (6%)	3 (3%)	
Hypercellularity	15 (17%)	25 (28%)	18 (20%)	13 (14%)
Hypocellularity			1 (1%)	1 (1%)
Lymph node	(25)	(23)	(24)	(16)
Bronchial, erythrophagocytosis		2 (9%)		
Bronchial, hyperplasia, lymphocyte		1 (4%)		
Iliac, erythrophagocytosis	2 (8%)	2 (9%)	1 (4%)	
Iliac, hyperplasia, lymphocyte	2 (8%)		2 (8%)	
Iliac, infiltration cellular, histiocyte	2 (8%)	1 (4%)		
Iliac, pigment			1 (4%)	
Iliac, proliferation, plasma cell	3 (12%)		1 (4%)	
Iliac, lymphatic sinus, ectasia	5 (20%)	3 (13%)	1 (4%)	
Inguinal, hyperplasia, lymphocyte			1 (4%)	
Inguinal, lymphatic sinus, ectasia			1 (4%)	
Lumbar, erythrophagocytosis	2 (8%)	2 (9%)	1 (4%)	1 (6%)
Lumbar, proliferation, plasma cell		1 (4%)		
Lumbar, lymphatic sinus, ectasia		2 (9%)	1 (4%)	2 (13%)
Lymphatic sinus, mediastinal, ectasia	1 (4%)	1 (4%)	1 (4%)	1 (6%)
Lymphatic sinus, popliteal, ectasia		1 (4%)		
Lymphatic sinus, renal, ectasia		4 (17%)	3 (13%)	
Mediastinal, erythrophagocytosis	6 (24%)	7 (30%)	7 (29%)	3 (19%)
Mediastinal, extramedullary hematopoiesis			1 (4%)	
Mediastinal, hemorrhage	1 (4%)	1 (4%)	1 (4%)	1 (6%)
Mediastinal, hyperplasia, lymphocyte			1 (4%)	
Mediastinal, infiltration cellular, histiocyte		1 (4%)	1 (4%)	
Mediastinal, inflammation, acute		1 (4%)		
Mediastinal, pigment		1 (4%)		
Mediastinal, proliferation, plasma cell			1 (4%)	
Pancreatic, erythrophagocytosis	3 (12%)	1 (4%)	4 (17%)	3 (19%)
Pancreatic, hemorrhage	1 (4%)			
Pancreatic, hyperplasia, lymphocyte	1 (4%)			
Pancreatic, infiltration cellular, mixed cell				1 (6%)
Renal, erythrophagocytosis	8 (32%)	6 (26%)	4 (17%)	
Renal, hyperplasia, lymphocyte		1 (4%)		
Renal, infiltration cellular, mixed cell				1 (6%)
Renal, proliferation, plasma cell	2 (8%)			
Lymph node, mandibular	(89)	(90)	(90)	(88)
Congestion		1 (1%)	2 (2%)	
Erythrophagocytosis		3 (3%)	2 (2%)	1 (1%)
Hemorrhage			1 (1%)	
Hyperplasia, lymphocyte	41 (46%)	50 (56%)	52 (58%)	40 (45%)
Infiltration cellular, histiocyte		2 (2%)		1 (1%)
Infiltration cellular, polymorphonuclear	2 (2%)			
Necrosis, lymphocyte		1 (1%)		
Proliferation, plasma cell	49 (55%)	61 (68%)	62 (69%)	57 (65%)
Lymphatic sinus, ectasia	16 (18%)	24 (27%)	29 (32%)	14 (16%)
Lymph node, mesenteric	(90)	(89)	(88)	(88)
Erythrophagocytosis	17 (19%)	5 (6%)	5 (6%)	9 (10%)
Hyperplasia, lymphocyte	2 (2%)	3 (3%)	3 (3%)	3 (3%)
Infiltration cellular, histiocyte	1 (1%)			
Infiltration cellular, polymorphonuclear	2 (2%)			1 (1%)
Proliferation, plasma cell		1 (1%)		
Lymphatic sinus, ectasia		2 (2%)	3 (3%)	1 (1%)
Lymphocyte, depletion	2 (2%)			

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Spleen	(90)	(90)	(90)	(85)
Congestion			1 (1%)	
Developmental malformation	1 (1%)			
Extramedullary hematopoiesis	45 (50%)	60 (67%)	56 (62%)	48 (56%)
Hemorrhage		1 (1%)	1 (1%)	
Hyperplasia, lymphocyte	5 (6%)			
Necrosis			2 (2%)	
Pigment	57 (63%)	54 (60%)	64 (71%)	63 (74%)
Thrombus		1 (1%)		
Arteriole, mineral	1 (1%)			
Red pulp, atrophy	26 (29%)	14 (16%)	12 (13%)	13 (15%)
White pulp, atrophy	30 (33%)	11 (12%)	10 (11%)	24 (28%)
Thymus	(88)	(85)	(87)	(82)
Atrophy	79 (90%)	76 (89%)	80 (92%)	65 (79%)
Cyst	10 (11%)	10 (12%)	10 (11%)	17 (21%)
Ectopic parathyroid gland	6 (7%)	6 (7%)	7 (8%)	5 (6%)
Ectopic thyroid	1 (1%)			
Hemorrhage	2 (2%)	2 (2%)	2 (2%)	20 (24%)
Hyperplasia, epithelial	2 (2%)	2 (2%)	4 (5%)	4 (5%)
Artery, inflammation, chronic active	6 (7%)	3 (4%)	2 (2%)	1 (1%)
Integumentary System				
Mammary gland	(82)	(77)	(80)	(80)
Atrophy	1 (1%)	2 (3%)	3 (4%)	
Galactocele	1 (1%)	1 (1%)	2 (3%)	
Duct, dilation	3 (4%)	8 (10%)	9 (11%)	3 (4%)
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	3 (3%)	12 (13%)	3 (3%)	2 (2%)
Inflammation, suppurative		2 (2%)		
Inflammation, chronic active	1 (1%)	2 (2%)	2 (2%)	1 (1%)
Ulcer	2 (2%)	2 (2%)	4 (4%)	
Adnexa, atrophy				1 (1%)
Artery, subcutaneous tissue, inflammation, chronic active	1 (1%)			
Dermis, fibrosis				1 (1%)
Epidermis, hyperplasia	1 (1%)	1 (1%)		1 (1%)
Hair follicle, congestion				1 (1%)
Hair follicle, degeneration			1 (1%)	
Prepuce, hyperplasia		2 (2%)		1 (1%)
Prepuce, inflammation, acute				1 (1%)
Prepuce, inflammation, chronic active		1 (1%)		
Prepuce, ulcer		2 (2%)		1 (1%)
Subcutaneous tissue, hemorrhage		1 (1%)		
Subcutaneous tissue, inflammation, suppurative	1 (1%)		1 (1%)	2 (2%)
Subcutaneous tissue, inflammation, chronic	1 (1%)		1 (1%)	
Subcutaneous tissue, inflammation, chronic active			2 (2%)	
Subcutaneous tissue, necrosis			1 (1%)	

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Fibrous osteodystrophy	46 (51%)	20 (22%)	15 (17%)	5 (6%)
Cranium, inflammation, chronic active			1 (1%)	
Bone, vertebra	(0)	(0)	(1)	(0)
Developmental malformation			1 (100%)	
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	34 (38%)	35 (39%)	30 (33%)	26 (29%)
Inflammation, chronic active			1 (1%)	
Mineral	2 (2%)		1 (1%)	
Diaphragm, hernia		1 (1%)		
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	7 (8%)	12 (13%)	6 (7%)	3 (3%)
Edema		1 (1%)		
Hemorrhage	2 (2%)	3 (3%)		
Infiltration cellular, mononuclear cell	1 (1%)			
Inflammation, suppurative			1 (1%)	
Mineral	5 (6%)	3 (3%)	4 (4%)	4 (4%)
Necrosis	7 (8%)	7 (8%)	3 (3%)	
Choroid plexus, degeneration	1 (1%)			
Choroid plexus, mineral	3 (3%)	1 (1%)		
Glial cell, hyperplasia		2 (2%)		2 (2%)
Hypothalamus, cyst		3 (3%)		
Meninges, fibrosis		1 (1%)		
Meninges, hyperplasia	1 (1%)		1 (1%)	
Meninges, hyperplasia, granular cell	1 (1%)	1 (1%)		
Meninges, mineral		1 (1%)		
Pineal gland, mineral	3 (3%)	3 (3%)	2 (2%)	
Pineal gland, vacuolation, cytoplasmic	12 (13%)	6 (7%)	9 (10%)	4 (4%)
Nerve trigeminal	(84)	(90)	(88)	(90)
Degeneration	63 (75%)	66 (73%)	67 (76%)	49 (54%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	86 (96%)	90 (100%)	88 (98%)	84 (93%)
Infiltration cellular, histiocyte			1 (1%)	
Infiltration cellular, mononuclear cell	1 (1%)			
Peripheral nerve, tibial	(88)	(90)	(90)	(89)
Degeneration	84 (95%)	90 (100%)	89 (99%)	81 (91%)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Degeneration	30 (33%)	36 (40%)	42 (47%)	35 (39%)
Meninges, inflammation, suppurative			1 (1%)	
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Degeneration	21 (23%)	15 (17%)	21 (23%)	24 (27%)
Nerve, degeneration	79 (88%)	85 (94%)	83 (92%)	76 (84%)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	58 (64%)	69 (77%)	74 (82%)	62 (69%)
Hemorrhage, focal	1 (1%)			
Meninges, inflammation, suppurative			1 (1%)	
Trigeminal ganglion	(75)	(77)	(79)	(83)
Degeneration	23 (31%)	22 (29%)	21 (27%)	16 (19%)

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Congestion	13 (14%)	13 (14%)	11 (12%)	33 (37%)
Foreign body	4 (4%)	2 (2%)	1 (1%)	1 (1%)
Hemorrhage	3 (3%)	5 (6%)	2 (2%)	4 (4%)
Inflammation, suppurative	3 (3%)		1 (1%)	2 (2%)
Inflammation, granulomatous		6 (7%)	1 (1%)	
Inflammation, chronic		1 (1%)	1 (1%)	
Inflammation, chronic active	2 (2%)	1 (1%)	1 (1%)	
Inflammation, subacute	2 (2%)			
Metaplasia, osseous			1 (1%)	
Alveolus, infiltration cellular, histiocyte	37 (41%)	38 (42%)	42 (47%)	47 (52%)
Artery, inflammation, chronic active	3 (3%)	3 (3%)	1 (1%)	
Artery, mineral	1 (1%)			
Artery, mediastinum, inflammation, chronic active	2 (2%)			
Epithelium alveolus, hyperplasia	3 (3%)	2 (2%)	1 (1%)	1 (1%)
Interstitium, inflammation, chronic		5 (6%)		
Interstitium, inflammation, chronic active		1 (1%)		
Interstitium, mineral	1 (1%)	1 (1%)	1 (1%)	
Mediastinum, inflammation, suppurative			1 (1%)	
Perivascular, infiltration cellular, lymphocyte			1 (1%)	
Perivascular, inflammation, chronic active	1 (1%)			
Nose	(89)	(90)	(90)	(87)
Foreign body	5 (6%)	2 (2%)	3 (3%)	8 (9%)
Hyperplasia, lymphocyte		1 (1%)		
Inflammation, suppurative	10 (11%)	6 (7%)	10 (11%)	17 (20%)
Inflammation, chronic active				2 (2%)
Mineral				1 (1%)
Nasopharyngeal duct, respiratory epithelium, hyperplasia	1 (1%)			
Olfactory epithelium, accumulation, hyaline droplet	79 (89%)	88 (98%)	90 (100%)	76 (87%)
Olfactory epithelium, hyperplasia		1 (1%)		
Olfactory epithelium, metaplasia, respiratory	3 (3%)	2 (2%)	1 (1%)	4 (5%)
Respiratory epithelium, accumulation, hyaline droplet	3 (3%)	1 (1%)	2 (2%)	3 (3%)
Respiratory epithelium, hyperplasia	3 (3%)	4 (4%)	8 (9%)	7 (8%)
Respiratory epithelium, hyperplasia, goblet cell	1 (1%)			
Respiratory epithelium, mineral	1 (1%)			
Trachea	(90)	(88)	(88)	(72)
Artery, inflammation, chronic active		1 (1%)		
Artery, mineral	1 (1%)			
Epithelium, hyperplasia	1 (1%)			
Epithelium, metaplasia, squamous	1 (1%)			

TABLE C4
**Summary of the Incidence of Nonneoplastic Lesions in Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Special Senses System				
Eye	(85)	(83)	(81)	(72)
Phthisis bulbi			1 (1%)	
Retinal detachment	1 (1%)		1 (1%)	
Anterior chamber, inflammation, acute	4 (5%)	8 (10%)	5 (6%)	1 (1%)
Cornea, fibrosis	1 (1%)	2 (2%)	2 (2%)	4 (6%)
Cornea, inflammation, acute	28 (33%)	18 (22%)	19 (23%)	17 (24%)
Cornea, neovascularization	10 (12%)	14 (17%)	14 (17%)	21 (29%)
Cornea, ulcer	6 (7%)	1 (1%)		
Cornea, epithelium, degeneration			1 (1%)	2 (3%)
Cornea, epithelium, hyperplasia	13 (15%)	15 (18%)	17 (21%)	20 (28%)
Lens, cataract		1 (1%)		
Retina, atrophy	6 (7%)	17 (20%)	17 (21%)	8 (11%)
Retina, degeneration	1 (1%)			
Retina, dysplasia				1 (1%)
Harderian gland	(90)	(90)	(90)	(89)
Atrophy	1 (1%)	4 (4%)	2 (2%)	3 (3%)
Degeneration			1 (1%)	1 (1%)
Degeneration, cystic	2 (2%)	4 (4%)	1 (1%)	
Hyperplasia				2 (2%)
Infiltration cellular, lymphocyte		3 (3%)		3 (3%)
Inflammation, suppurative		1 (1%)		
Inflammation, granulomatous		5 (6%)	2 (2%)	
Inflammation, acute	2 (2%)	1 (1%)		
Inflammation, chronic		1 (1%)		1 (1%)
Inflammation, chronic active	2 (2%)	2 (2%)	1 (1%)	1 (1%)
Lacrimal gland	(2)	(1)	(1)	(1)
Metaplasia, harderian gland	2 (100%)	1 (100%)	1 (100%)	1 (100%)
Zymbal's gland	(0)	(0)	(1)	(1)
Urinary System				
Kidney	(90)	(90)	(90)	(87)
Mineral	1 (1%)		2 (2%)	
Necrosis			1 (1%)	
Nephropathy, chronic progressive	88 (98%)	90 (100%)	90 (100%)	86 (99%)
Thrombus	1 (1%)	1 (1%)	1 (1%)	
Artery, inflammation, chronic active			1 (1%)	
Artery, mineral	2 (2%)			
Pelvis, dilation	1 (1%)		1 (1%)	1 (1%)
Pelvis, inflammation, suppurative		1 (1%)	1 (1%)	
Pelvis, urothelium, hyperplasia		3 (3%)	1 (1%)	
Perirenal tissue, hemorrhage				1 (1%)
Perirenal tissue, thrombus			1 (1%)	
Renal tubule, accumulation, hyaline droplet				1 (1%)
Renal tubule, cyst	18 (20%)	17 (19%)	9 (10%)	6 (7%)
Renal tubule, hyperplasia, atypical	2 (2%)	1 (1%)	3 (3%)	
Renal tubule, hyperplasia, oncocytic	2 (2%)			
Renal tubule, inflammation, suppurative			1 (1%)	
Renal tubule, necrosis				1 (1%)
Urothelium, hyperplasia	1 (1%)		1 (1%)	

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Urinary System (continued)				
Urinary bladder	(89)	(83)	(83)	(78)
Dilation		1 (1%)		
Hemorrhage	2 (2%)	1 (1%)	2 (2%)	1 (1%)
Inflammation, acute	2 (2%)	1 (1%)	1 (1%)	
Inflammation, chronic active		2 (2%)		
Necrosis	1 (1%)		1 (1%)	
Artery, inflammation, chronic active		1 (1%)		1 (1%)
Muscularis, degeneration	1 (1%)			
Serosa, inflammation, chronic active			1 (1%)	
Urothelium, hyperplasia	1 (1%)	4 (5%)	2 (2%)	1 (1%)

APPENDIX D

SUMMARY OF LESIONS IN FEMALE RATS

EXPOSED TO CDMA-MODULATED

CELL PHONE RFR FOR 2 YEARS

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TABLE D1
Summary of the Incidence of Neoplasms in Female Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	30	29	28	16
Natural deaths	11	15	12	13
Survivors				
Died last week of study	1	2		
Terminal euthanasia	47	44	50	61
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Integumentary System				
Mammary gland	(10)	(10)	(10)	(10)
Adenocarcinoma				1 (10%)
Skin	(10)	(10)	(10)	(10)
Systems Examined with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
2-Year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Intestine large, cecum	(84)	(82)	(86)	(80)
Intestine large, colon	(89)	(89)	(88)	(88)
Intestine large, rectum	(90)	(88)	(87)	(88)
Granular cell tumor benign	1 (1%)			
Intestine small, duodenum	(88)	(86)	(87)	(85)
Intestine small, ileum	(86)	(83)	(84)	(83)
Intestine small, jejunum	(83)	(81)	(84)	(79)
Leiomyosarcoma	1 (1%)			
Liver	(90)	(90)	(90)	(90)
Carcinoma, metastatic, adrenal cortex			1 (1%)	
Hepatocellular adenoma	7 (8%)	2 (2%)	2 (2%)	1 (1%)
Hepatocellular carcinoma				1 (1%)
Mesentery	(4)	(3)	(11)	(4)
Adenocarcinoma, metastatic, uterus	1 (25%)			
Oral mucosa	(1)	(1)	(0)	(0)
Squamous cell carcinoma	1 (100%)			
Pancreas	(90)	(90)	(90)	(89)
Salivary glands	(90)	(90)	(90)	(90)
Parotid gland, squamous cell carcinoma				1 (1%)

TABLE D1
Summary of the Incidence of Neoplasms in Female Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Stomach, forestomach	(90)	(90)	(90)	(90)
Sarcoma	1 (1%)			1 (1%)
Squamous cell papilloma				1 (1%)
Stomach, glandular	(90)	(90)	(89)	(88)
Sarcoma, metastatic, stomach, forestomach	1 (1%)			
Tongue	(1)	(0)	(0)	(0)
Squamous cell carcinoma	1 (100%)			
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Blood vessel	(0)	(0)	(0)	(1)
Heart	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, mammary gland			1 (1%)	
Endocardium, schwannoma malignant		1 (1%)		2 (2%)
Myocardium, schwannoma malignant		1 (1%)		
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(90)
Adenoma	1 (1%)	2 (2%)	2 (2%)	1 (1%)
Carcinoma	1 (1%)		1 (1%)	
Adrenal medulla	(86)	(89)	(87)	(88)
Pheochromocytoma benign	1 (1%)	7 (8%)	3 (3%)	4 (5%)
Pheochromocytoma complex			1 (1%)	
Pheochromocytoma malignant		2 (2%)	1 (1%)	
Islets, pancreatic	(90)	(89)	(90)	(88)
Adenoma	5 (6%)	4 (4%)	5 (6%)	4 (5%)
Carcinoma	2 (2%)	2 (2%)		2 (2%)
Parathyroid gland	(87)	(80)	(85)	(85)
Pituitary gland	(90)	(89)	(89)	(90)
Pars distalis, adenoma	42 (47%)	40 (45%)	30 (34%)	40 (44%)
Pars distalis, adenoma, multiple	1 (1%)	1 (1%)		
Pars distalis, carcinoma	1 (1%)	1 (1%)	1 (1%)	
Thyroid gland	(90)	(90)	(90)	(89)
Bilateral, C-cell, adenoma				1 (1%)
Bilateral, C-cell, carcinoma			1 (1%)	
C-cell, adenoma	6 (7%)	9 (10%)	3 (3%)	7 (8%)
C-cell, carcinoma		3 (3%)	2 (2%)	2 (2%)
Follicular cell, adenoma			1 (1%)	
Follicular cell, carcinoma	1 (1%)	1 (1%)		
General Body System				
Tissue NOS	(8)	(11)	(8)	(6)
Abdominal, schwannoma malignant	1 (13%)			
Abdominal, fat, lipoma			1 (13%)	

TABLE D1
**Summary of the Incidence of Neoplasms in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Genital System				
Clitoral gland	(87)	(88)	(89)	(86)
Carcinoma			1 (1%)	
Schwannoma malignant				1 (1%)
Ovary	(90)	(90)	(89)	(90)
Cystadenocarcinoma		1 (1%)		
Cystadenoma	1 (1%)			
Granulosa cell tumor benign	1 (1%)			
Granulosa cell tumor malignant	2 (2%)			
Sertoli cell tumor benign	1 (1%)			
Periovarian tissue, schwannoma malignant				1 (1%)
Rete ovarii, adenoma			1 (1%)	1 (1%)
Oviduct	(1)	(0)	(0)	(0)
Uterus	(90)	(90)	(90)	(90)
Adenocarcinoma	3 (3%)			3 (3%)
Adenoma			1 (1%)	1 (1%)
Carcinoma		1 (1%)		
Hemangiosarcoma	2 (2%)			
Leiomyosarcoma			2 (2%)	
Polyp, glandular		2 (2%)	2 (2%)	
Polyp stromal	15 (17%)	11 (12%)	9 (10%)	12 (13%)
Polyp stromal, multiple	1 (1%)	4 (4%)	4 (4%)	5 (6%)
Schwannoma malignant	1 (1%)			
Squamous cell carcinoma			1 (1%)	
Cervix, leiomyosarcoma	1 (1%)			
Cervix, polyp stromal			1 (1%)	
Cervix, schwannoma malignant	1 (1%)		2 (2%)	
Vagina	(2)	(1)	(0)	(1)
Polyp, stromal		1 (100%)		
Schwannoma malignant	1 (50%)			
Schwannoma malignant, metastatic, uterus	1 (50%)			
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Lymph node	(13)	(8)	(11)	(20)
Iliac, adenocarcinoma, metastatic, mammary gland				1 (5%)
Lymph node, mandibular	(90)	(90)	(89)	(90)
Lymph node, mesenteric	(90)	(90)	(90)	(89)
Spleen	(90)	(90)	(90)	(90)
Hemangiosarcoma	1 (1%)			
Thymus	(87)	(83)	(87)	(87)
Thymoma benign	1 (1%)		1 (1%)	
Thymoma malignant	1 (1%)			
Integumentary System				
Mammary gland	(90)	(90)	(90)	(90)
Adenocarcinoma	9 (10%)	7 (8%)	6 (7%)	3 (3%)
Adenocarcinoma, multiple	1 (1%)	1 (1%)		1 (1%)
Adenoma	4 (4%)	4 (4%)	1 (1%)	2 (2%)
Adenoma, multiple	4 (4%)			
Fibroadenoma	34 (38%)	40 (44%)	34 (38%)	32 (36%)
Fibroadenoma, multiple	29 (32%)	21 (23%)	29 (32%)	30 (33%)

TABLE D1
Summary of the Incidence of Neoplasms in Female Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Integumentary System (continued)				
Skin	(90)	(90)	(90)	(90)
Keratoacanthoma		1 (1%)	2 (2%)	1 (1%)
Squamous cell carcinoma, metastatic, salivary glands				1 (1%)
Subcutaneous tissue, fibroma	2 (2%)		1 (1%)	3 (3%)
Subcutaneous tissue, lipoma			1 (1%)	2 (2%)
Subcutaneous tissue, malignant fibrous histiocytoma	1 (1%)			
Subcutaneous tissue, sarcoma	2 (2%)			
Subcutaneous tissue, squamous cell carcinoma		2 (2%)		
Vulva, squamous cell carcinoma				1 (1%)
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Vertebra, chondroma			1 (1%)	
Skeletal muscle	(90)	(90)	(90)	(90)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Carcinoma, metastatic, pituitary gland	1 (1%)			
Glioma malignant		3 (3%)		
Meningioma malignant				1 (1%)
Neuroblastoma		1 (1%)		
Sarcoma		1 (1%)		
Meninges, granular cell tumor benign	1 (1%)	1 (1%)		2 (2%)
Pineal gland, pinealoma		1 (1%)		
Nerve trigeminal	(84)	(84)	(85)	(84)
Squamous cell carcinoma, metastatic, salivary glands				1 (1%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Peripheral nerve, tibial	(90)	(90)	(89)	(89)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(89)	(90)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(81)	(77)	(81)	(75)
Carcinoma, metastatic, pituitary gland			1 (1%)	
Squamous cell carcinoma, metastatic, salivary glands				1 (1%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, mammary gland		2 (2%)	1 (1%)	1 (1%)
Carcinoma, metastatic, adrenal cortex			1 (1%)	
Carcinoma, metastatic, thyroid gland		1 (1%)	1 (1%)	
Carcinoma, metastatic, uncertain primary site		1 (1%)		
Nose	(90)	(89)	(90)	(89)
Trachea	(89)	(88)	(89)	(89)
Carcinoma, metastatic, thyroid gland			1 (1%)	

TABLE D1
Summary of the Incidence of Neoplasms in Female Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Special Senses System				
Ear	(0)	(0)	(1) 1 (100%)	(1) 1 (100%)
Neural crest tumor				
Eye	(88)	(86)	(88)	(86)
Sarcoma		1 (1%)		
Harderian gland	(90)	(90)	(90)	(90)
Urinary System				
Kidney	(90)	(90)	(90)	(89)
Bilateral, renal tubule, carcinoma	1 (1%)		2 (2%)	
Renal tubule, adenoma	1 (1%)		1 (1%)	
Urinary bladder	(88)	(88)	(90)	(90)
Leiomyosarcoma	1 (1%)			
Schwannoma malignant, metastatic, tissue NOS	1 (1%)			
Systemic Lesions				
Multiple organs ^b	(90)	(90)	(90)	(90)
Histiocytic sarcoma			2 (2%)	
Leukemia mononuclear		3 (3%)		
Lymphoma malignant	5 (6%)	2 (2%)	4 (4%)	3 (3%)

TABLE D1
Summary of the Incidence of Neoplasms in Female Rats
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Neoplasm Summary				
Total animals with primary neoplasms ^c				
14-Week interim evaluation				1
2-Year study	89	88	82	85
Total primary neoplasms				
14-Week interim evaluation				1
2-Year study	202	185	164	174
Total animals with benign neoplasms				
2-Year study	82	84	78	79
Total benign neoplasms				
2-Year study	159	151	136	150
Total animals with malignant neoplasms				
14-Week interim evaluation				1
2-Year study	37	27	24	22
Total malignant neoplasms				
14-Week interim evaluation				1
2-Year study	43	34	27	23
Total animals with metastatic neoplasms				
2-Year study	5	4	6	2
Total metastatic neoplasms				
2-Year study	5	4	11	5
Total animals with malignant neoplasms- uncertain primary site				
2-Year study		1		
Total animals with uncertain neoplasms- benign or malignant				
2-Year study			1	1
Total uncertain neoplasms benign or malignant				
2-Year study			1	1

^a Number of animals examined microscopically at the site and the number of animals with neoplasm

^b Number of animals with any tissue examined microscopically

^c Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE D2
Statistical Analysis of Primary Neoplasms in Female Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Medulla: Benign Pheochromocytoma				
Overall rate ^a	1/86 (1%)	7/89 (8%)	3/87 (3%)	4/88 (5%)
Rate per litters ^b	1/35 (3%)	7/34 (21%)	3/35 (9%)	4/35 (11%)
Adjusted rate ^c	1.5%	9.6%	4.4%	5.2%
Terminal rate ^d	1/45 (2%)	5/44 (11%)	3/48 (6%)	4/60 (7%)
First incidence (days)	737 (T)	464	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test ^e	P=0.466	P=0.059	P=0.322	P=0.248
Litter C-A/Fisher's test ^f	P=0.379	P=0.025	P=0.307	P=0.178
Adrenal Medulla: Benign, Complex, or Malignant Pheochromocytoma				
Overall rate	1/86 (1%)	9/89 (10%)	5/87 (6%)	4/88 (5%)
Rate per litters	1/35 (3%)	9/34 (26%)	5/35 (14%)	4/35 (11%)
Adjusted rate	1.5%	12.3%	7.2%	5.2%
Terminal rate	1/45 (2%)	7/44 (16%)	4/48 (8%)	4/60 (7%)
First incidence (days)	737 (T)	464	652	737 (T)
Rao-Scott adjusted poly-3 test	P=0.546	P=0.022	P=0.126	P=0.242
Litter C-A/Fisher's test	P=0.457	P=0.006	P=0.099	P=0.178
Liver: Hepatocellular Adenoma				
Overall rate	7/90 (8%)	2/90 (2%)	2/90 (2%)	1/90 (1%)
Rate per litters	6/35 (17%)	2/34 (6%)	2/35 (6%)	1/35 (3%)
Adjusted rate	10.1%	2.7%	2.8%	1.3%
Terminal rate	6/48 (13%)	1/45 (2%)	2/50 (4%)	1/61 (2%)
First incidence (days)	707	493	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.042N	P=0.118N	P=0.125N	P=0.052N
Litter C-A/Fisher's test	P=0.039N	P=0.139N	P=0.130N	P=0.053N
Liver: Hepatocellular Adenoma or Hepatocellular Carcinoma				
Overall rate	7/90 (8%)	2/90 (2%)	2/90 (2%)	2/90 (2%)
Rate per litters	6/35 (17%)	2/34 (6%)	2/35 (6%)	2/35 (6%)
Adjusted rate	10.1%	2.7%	2.8%	2.5%
Terminal rate	6/48 (13%)	1/45 (2%)	2/50 (4%)	2/61 (3%)
First incidence (days)	707	493	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.089N	P=0.112N	P=0.118N	P=0.096N
Litter C-A/Fisher's test	P=0.108N	P=0.139N	P=0.130N	P=0.130N
Mammary Gland: Fibroadenoma				
Overall rate	63/90 (70%)	61/90 (68%)	63/90 (70%)	62/90 (69%)
Rate per litters	31/35 (89%)	32/34 (94%)	33/35 (94%)	31/35 (89%)
Adjusted rate	77%	73.3%	75.2%	73.1%
Terminal rate	36/48 (75%)	33/45 (73%)	36/50 (72%)	45/61 (74%)
First incidence (days)	464	300	268	492
Rao-Scott adjusted poly-3 test	P=0.356N	P=0.358N	P=0.462N	P=0.344N
Litter C-A/Fisher's test	P=0.505N	P=0.351	P=0.337	P=0.645
Mammary Gland: Adenoma				
Overall rate	8/90 (9%)	4/90 (4%)	1/90 (1%)	2/90 (2%)
Rate per litters	7/35 (20%)	4/34 (12%)	1/35 (3%)	2/35 (6%)
Adjusted rate	11.3%	5.4%	1.4%	2.5%
Terminal rate	5/48 (10%)	1/45 (2%)	1/50 (2%)	2/61 (3%)
First incidence (days)	524	647	737 (T)	737 (T)
Rao-Scott adjusted poly-3 test	P=0.035N	P=0.207N	P=0.039N	P=0.063N
Litter C-A/Fisher's test	P=0.034N	P=0.274N	P=0.027N	P=0.075N

TABLE D2
**Statistical Analysis of Primary Neoplasms in Female Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Mammary Gland: Fibroadenoma or Adenoma				
Overall rate	64/90 (71%)	62/90 (69%)	63/90 (70%)	62/90 (69%)
Rate per litters	31/35 (89%)	32/34 (94%)	33/35 (94%)	31/35 (89%)
Adjusted rate	77.7%	74.5%	75.2%	73.1%
Terminal rate	36/48 (75%)	33/45 (73%)	36/50 (72%)	45/61 (74%)
First incidence (days)	464	300	268	492
Rao-Scott adjusted poly-3 test	P=0.307N	P=0.386N	P=0.427N	P=0.312N
Litter C-A/Fisher's test	P=0.505N	P=0.351	P=0.337	P=0.645
Mammary Gland: Adenocarcinoma				
Overall rate	10/90 (11%)	8/90 (9%)	6/90 (7%)	4/90 (4%)
Rate per litters	9/35 (26%)	8/34 (24%)	5/35 (14%)	4/35 (11%)
Adjusted rate	14.2%	10.4%	8.2%	4.9%
Terminal rate	6/48 (13%)	1/45 (2%)	3/50 (6%)	2/61 (3%)
First incidence (days)	622	300	493	305
Rao-Scott adjusted poly-3 test	P=0.042N	P=0.330N	P=0.196N	P=0.055N
Litter C-A/Fisher's test	P=0.059N	P=0.528N	P=0.185N	P=0.109N
Mammary Gland: Adenoma or Adenocarcinoma				
Overall rate	16/90 (18%)	12/90 (13%)	7/90 (8%)	6/90 (7%)
Rate per litters	13/35 (37%)	12/34 (35%)	5/35 (14%)	6/35 (17%)
Adjusted rate	22.2%	15.5%	9.5%	7.4%
Terminal rate	9/48 (19%)	2/45 (4%)	4/50 (8%)	4/61 (7%)
First incidence (days)	524	300	493	305
Rao-Scott adjusted poly-3 test	P=0.009N	P=0.214N	P=0.041N	P=0.014N
Litter C-A/Fisher's test	P=0.017N	P=0.536N	P=0.027N	P=0.053N
Mammary Gland: Fibroadenoma, Adenoma, or Adenocarcinoma				
Overall rate	66/90 (73%)	68/90 (76%)	66/90 (73%)	66/90 (73%)
Rate per litters	31/35 (89%)	33/34 (97%)	34/35 (97%)	32/35 (91%)
Adjusted rate	79.6%	78.7%	78.1%	76.6%
Terminal rate	36/48 (75%)	33/45 (73%)	37/50 (74%)	47/61 (77%)
First incidence (days)	464	300	268	305
Rao-Scott adjusted poly-3 test	P=0.352N	P=0.518N	P=0.478N	P=0.393N
Litter C-A/Fisher's test	P=0.519	P=0.187	P=0.178	P=0.500
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	4/89 (4%)	5/90 (6%)	4/88 (5%)
Rate per litters	4/35 (11%)	4/34 (12%)	5/35 (14%)	4/35 (11%)
Adjusted rate	7.2%	5.5%	6.9%	5.1%
Terminal rate	5/48 (10%)	2/45 (4%)	3/50 (6%)	4/61 (7%)
First incidence (days)	737 (T)	647	627	737 (T)
Rao-Scott adjusted poly-3 test	P=0.405N	P=0.460N	P=0.585N	P=0.424N
Litter C-A/Fisher's test	P=0.558	P=0.629	P=0.500	P=0.645
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	7/90 (8%)	6/89 (7%)	5/90 (6%)	6/88 (7%)
Rate per litters	6/35 (17%)	6/34 (18%)	5/35 (14%)	6/35 (17%)
Adjusted rate	10.1%	8.2%	6.9%	7.6%
Terminal rate	6/48 (13%)	3/45 (7%)	3/50 (6%)	5/61 (8%)
First incidence (days)	711	647	627	702
Rao-Scott adjusted poly-3 test	P=0.367N	P=0.451N	P=0.347N	P=0.404N
Litter C-A/Fisher's test	P=0.538N	P=0.603	P=0.500N	P=0.624

TABLE D2
Statistical Analysis of Primary Neoplasms in Female Rats Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	43/90 (48%)	41/89 (46%)	30/89 (34%)	40/90 (44%)
Rate per litters	28/35 (80%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.1%	52.9%	39.5%	49%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)
First incidence (days)	464	578	493	626
Rao-Scott adjusted poly-3 test	P=0.156N	P=0.360N	P=0.026N	P=0.204N
Litter C-A/Fisher's test	P=0.203N	P=0.474N	P=0.058N	P=0.289N
Pituitary Gland (Pars Distalis): Adenoma or Carcinoma				
Overall rate	44/90 (49%)	42/89 (47%)	31/89 (35%)	40/90 (44%)
Rate per litters	29/35 (83%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.9%	54.1%	40.7%	49%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)
First incidence (days)	464	578	493	626
Rao-Scott adjusted poly-3 test	P=0.131N	P=0.381N	P=0.030N	P=0.180N
Litter C-A/Fisher's test	P=0.144N	P=0.360N	P=0.031N	P=0.197N
Skin (Subcutaneous Tissue): Fibroma, Sarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	0/90 (0%)	1/90 (1%)	3/90 (3%)
Rate per litters	5/35 (14%)	0/34 (0%)	1/35 (3%)	3/35 (9%)
Adjusted rate	7%	0%	1.4%	3.7%
Terminal rate	1/48 (2%)	0/45 (0%)	1/50 (2%)	1/61 (2%)
First incidence (days)	268	— ^g	737 (T)	550
Rao-Scott adjusted poly-3 test	P=0.413N	P=0.057N	P=0.142N	P=0.335N
Litter C-A/Fisher's test	P=0.426N	P=0.029N	P=0.099N	P=0.355N
Thyroid Gland (C-cell): Adenoma				
Overall rate	6/90 (7%)	9/90 (10%)	3/90 (3%)	8/89 (9%)
Rate per litters	6/35 (17%)	9/34 (26%)	3/35 (9%)	7/35 (20%)
Adjusted rate	8.5%	12.1%	4.2%	10.1%
Terminal rate	3/48 (6%)	6/45 (13%)	2/50 (4%)	6/61 (10%)
First incidence (days)	608	578	674	669
Rao-Scott adjusted poly-3 test	P=0.548N	P=0.326	P=0.232N	P=0.473
Litter C-A/Fisher's test	P=0.507N	P=0.259	P=0.239N	P=0.500
Thyroid Gland: (C-cell): Adenoma or Carcinoma				
Overall rate	6/90 (7%)	11/90 (12%)	6/90 (7%)	10/89 (11%)
Rate per litters	6/35 (17%)	11/34 (32%)	6/35 (17%)	9/35 (26%)
Adjusted rate	8.5%	14.8%	8.3%	12.6%
Terminal rate	3/48 (6%)	8/45 (18%)	4/50 (8%)	8/61 (13%)
First incidence (days)	608	578	643	669
Rao-Scott adjusted poly-3 test	P=0.390	P=0.175	P=0.579N	P=0.288
Litter C-A/Fisher's test	P=0.402	P=0.118	P=0.624	P=0.281
Uterus: Polyp Stromal				
Overall rate	16/90 (18%)	17/90 (19%)	16/90 (18%)	17/90 (19%)
Rate per litters	11/35 (31%)	13/34 (38%)	15/35 (43%)	13/35 (37%)
Adjusted rate	22.7%	23%	22%	21.2%
Terminal rate	14/48 (29%)	13/45 (29%)	12/50 (24%)	14/61 (23%)
First incidence (days)	531	605	631	587
Rao-Scott adjusted poly-3 test	P=0.437N	P=0.552	P=0.532N	P=0.490N
Litter C-A/Fisher's test	P=0.374	P=0.367	P=0.229	P=0.401

TABLE D2
**Statistical Analysis of Primary Neoplasms in Female Rats Exposed to CDMA-Modulated Cell Phone RFR
for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
All Organs: Malignant Lymphoma				
Overall rate	5/90 (6%)	2/90 (2%)	4/90 (4%)	3/90 (3%)
Rate per litters	5/35 (14%)	2/34 (6%)	4/35 (11%)	3/35 (9%)
Adjusted rate	7%	2.7%	5.5%	3.7%
Terminal rate	0/48 (0%)	1/45 (2%)	1/50 (2%)	0/61 (0%)
First incidence (days)	268	706	483	587
Rao-Scott adjusted poly-3 test	P=0.339N	P=0.209N	P=0.472N	P=0.297N
Litter C-A/Fisher's test	P=0.382N	P=0.226N	P=0.500N	P=0.355N

(T) Terminal euthanasia

- a Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, liver, pancreatic islets, pituitary gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
- b Number of litters with tumor-bearing animals/number of litters examined at site
- c Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
- d Observed incidence at terminal euthanasia
- e Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott adjusted poly-3 test is a modification of the Poly-3 test that also incorporates an adjustment for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
- f The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
- g Not applicable; no neoplasms in animal group.
- h Value of statistic cannot be computed.

TABLE D3a
Historical Incidence of Malignant Schwannoma of the Heart
in Control Female Hsd:Sprague Dawley SD Rats^a

Incidence in Controls	
Overall Historical Incidence: All Routes	
Total	0/239

^a Data as of November 2017

TABLE D3b
Historical Incidence of Malignant Glioma of the Brain in Control Female Hsd:Sprague Dawley SD Rats^a

Incidence in Controls	
Overall Historical Incidence: All Routes	
Total (%)	1/190 (0.7%)
Mean ± standard deviation	0.7% ± 1.2%
Range	0%-2%

^a Data as of November 2017

TABLE D3c
Historical Incidence of Pituitary Gland (Pars Distalis) Neoplasms
in Control Female Hsd:Sprague Dawley SD Rats^a

	Adenoma	Carcinoma	Adenoma or Carcinoma
Overall Historical Incidence: All Routes			
Total (%)	98/240 (40.8%)	1/240 (0.4%)	99/240 (41.3%)
Mean ± standard deviation	39.4% ± 5.6%	0.3% ± 0.6%	39.7% ± 6.2%
Range	36%-48%	0%-1%	36%-49%

^a Data as of November 2017

TABLE D3d**Historical Incidence of Adrenal Medulla Neoplasms in Control Female Hsd:Sprague Dawley SD Rats^a**

	Benign Pheochromocytoma	Malignant Pheochromocytoma	Complex Pheochromocytoma	Benign, Malignant, or Complex Pheochromocytoma
Overall Historical Incidence: All Routes				
Total (%)	4/235 (1.7%)	2/235 (0.9%)	0/235	6/235 (2.6%)
Mean ± standard deviation	1.8% ± 2.9%	1.0% ± 2.0%		2.8% ± 4.8%
Range	0%-6%	0%-4%		0%-10%

^a Data as of November 2017

TABLE D3e**Historical Incidence of Hepatocellular Neoplasms in Control Female Hsd:Sprague Dawley SD Rats^a**

	Adenoma	Carcinoma	Adenoma or Carcinoma
Overall Historical Incidence: All Routes			
Total (%)	11/240 (4.6%)	0/240	11/240 (4.6%)
Mean ± standard deviation	3.9% ± 3.2%		3.9% ± 3.2%
Range	0%-8%		0%-8%

^a Data as of November 2017

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	30	29	28	16
Natural deaths	11	15	12	13
Survivors				
Died last week of study	1	2		
Terminal euthanasia	47	44	50	61
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(10)	(10)
Intestine large, cecum	(10)	(10)	(10)	(10)
Intestine large, colon	(10)	(10)	(10)	(10)
Inflammation, chronic active				1 (10%)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)		2 (20%)	1 (10%)
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(10)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell	1 (10%)	1 (10%)		2 (20%)
Inflammation, chronic active				1 (10%)
Pancreas	(10)	(10)	(10)	(10)
Inflammation, chronic active				1 (10%)
Salivary glands	(10)	(10)	(10)	(10)
Inflammation, chronic active				1 (10%)
Stomach, forestomach	(10)	(10)	(10)	(10)
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Inflammation, chronic active				1 (10%)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy			2 (20%)	2 (20%)
Endocardium, inflammation, chronic active				1 (10%)
Ventricle right, cardiomyopathy			1 (10%)	1 (10%)
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(10)	(10)	(9)	(9)
Pituitary gland	(10)	(10)	(10)	(10)
Pars distalis, cyst	2 (20%)			
Pars intermedia, cyst	1 (10%)		2 (20%)	
Rathke's cleft, cyst		1 (10%)		
Thyroid gland	(10)	(10)	(10)	(10)
Ectopic thymus			1 (10%)	

^a Number of animals examined microscopically at the site and the number of animals with lesion

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
14-Week Interim Evaluation (continued)				
Genital System				
Clitoral gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	4 (40%)	4 (40%)	4 (40%)	2 (20%)
Ovary	(10)	(10)	(10)	(10)
Follicle, cyst	1 (10%)		1 (10%)	
Uterus	(10)	(10)	(10)	(10)
Hematopoietic System				
Bone marrow	(10)	(10)	(10)	(10)
Lymph node	(0)	(0)	(1)	(0)
Pigment			1 (100%)	
Lymph node, mandibular	(10)	(10)	(10)	(9)
Lymph node, mesenteric	(10)	(10)	(10)	(10)
Spleen	(10)	(10)	(10)	(10)
Extramedullary hematopoiesis				1 (10%)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	1 (10%)	2 (20%)	1 (10%)	
Hyperplasia, epithelial				1 (10%)
Integumentary System				
Mammary gland	(10)	(10)	(10)	(10)
Skin	(10)	(10)	(10)	(10)
Inflammation, chronic active				1 (10%)
Musculoskeletal System				
Bone	(10)	(10)	(10)	(10)
Skeletal muscle	(10)	(10)	(10)	(10)
Inflammation, chronic active				1 (10%)
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion				1 (10%)
Alveolus, infiltration cellular, histiocyte	2 (20%)			
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(10)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Conjunctiva, inflammation, chronic active				1 (10%)
Harderian gland	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte	1 (10%)			
Inflammation, chronic				1 (10%)
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell				1 (10%)
Inflammation, chronic active				1 (10%)
Nephropathy, chronic progressive	3 (30%)	3 (30%)	4 (40%)	4 (40%)
Urinary bladder	(10)	(10)	(10)	(10)

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
14-Week Interim Evaluation (continued)				
Systems Examined with No Lesions Observed				
General Body System				
Nervous System				
2-Year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Dilation		2 (2%)		
Inflammation, acute				1 (1%)
Muscularis, degeneration		1 (1%)		
Intestine large, cecum	(84)	(82)	(86)	(80)
Ulcer			1 (1%)	
Artery, inflammation, chronic active		2 (2%)		
Intestine large, colon	(89)	(89)	(88)	(88)
Diverticulum			1 (1%)	
Artery, inflammation, chronic active		1 (1%)		
Intestine large, rectum	(90)	(88)	(87)	(88)
Hyperplasia, lymphocyte			3 (3%)	
Artery, inflammation, chronic active		1 (1%)		
Intestine small, duodenum	(88)	(86)	(87)	(85)
Intestine small, ileum	(86)	(83)	(84)	(83)
Hyperplasia, lymphocyte	1 (1%)			
Intestine small, jejunum	(83)	(81)	(84)	(79)
Liver	(90)	(90)	(90)	(90)
Angiectasis	6 (7%)	3 (3%)	9 (10%)	3 (3%)
Basophilic focus	11 (12%)	11 (12%)	7 (8%)	15 (17%)
Clear cell Focus	2 (2%)	4 (4%)	7 (8%)	3 (3%)
Congestion				1 (1%)
Eosinophilic focus	9 (10%)	17 (19%)	10 (11%)	9 (10%)
Extramedullary hematopoiesis	15 (17%)	11 (12%)	13 (14%)	13 (14%)
Hepatodiaphragmatic nodule	1 (1%)			3 (3%)
Infiltration cellular, histiocyte		1 (1%)		
Infiltration cellular, mixed cell	1 (1%)	2 (2%)	4 (4%)	2 (2%)
Inflammation, granulomatous		1 (1%)		
Mitotic alteration				1 (1%)
Mixed cell focus	29 (32%)	17 (19%)	29 (32%)	35 (39%)
Pigment		1 (1%)		
Artery, inflammation, chronic active		1 (1%)		
Bile duct, cyst	11 (12%)	14 (16%)	6 (7%)	9 (10%)
Bile duct, fibrosis	1 (1%)	1 (1%)	4 (4%)	
Bile duct, hyperplasia	9 (10%)	10 (11%)	12 (13%)	7 (8%)
Hepatocyte, hypertrophy	2 (2%)	2 (2%)	1 (1%)	1 (1%)
Hepatocyte, increased mitoses	2 (2%)			
Hepatocyte, necrosis	4 (4%)	9 (10%)	7 (8%)	4 (4%)
Hepatocyte, vacuolation, cytoplasmic	1 (1%)	5 (6%)	5 (6%)	9 (10%)
Kupffer cell, hyperplasia	3 (3%)			1 (1%)
Kupffer cell, hypertrophy	2 (2%)			
Kupffer cell, pigment			1 (1%)	1 (1%)
Periductal, cholangiofibrosis	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Serosa, inflammation, chronic active	1 (1%)			
Mesentery	(4)	(3)	(11)	(4)
Hemorrhage			1 (9%)	
Inflammation, chronic active	1 (25%)			1 (25%)
Necrosis	1 (25%)	1 (33%)	5 (45%)	2 (50%)
Artery, inflammation, chronic active		2 (67%)	2 (18%)	
Vein, degeneration			1 (9%)	
Vein, inflammation, chronic active			1 (9%)	1 (25%)

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Oral mucosa	(1)	(1) 1 (100%)	(0)	(0)
Inflammation, chronic active				
Pancreas	(90)	(90)	(90)	(89)
Ectopic liver	1 (1%)		1 (1%)	
Inflammation, chronic active	1 (1%)			3 (3%) 1 (1%)
Necrosis				
Acinus, atrophy	5 (6%)	2 (2%)	6 (7%)	2 (2%)
Acinus, hyperplasia	1 (1%)	4 (4%)	2 (2%)	2 (2%)
Artery, inflammation, chronic active		5 (6%)		1 (1%)
Periductal, cholangiofibrosis		3 (3%)	2 (2%)	1 (1%)
Salivary glands	(90)	(90)	(90)	(90)
Degeneration		1 (1%)		
Artery, inflammation, chronic active		3 (3%)		
Duct, parotid gland, dilation	1 (1%)	1 (1%)	2 (2%)	
Duct, parotid gland, fibrosis			1 (1%)	
Parotid gland, atrophy	4 (4%)	7 (8%)	9 (10%)	1 (1%)
Parotid gland, fibrosis			2 (2%)	3 (3%)
Parotid gland, inflammation, suppurative		1 (1%)		
Parotid gland, inflammation, acute		1 (1%)	1 (1%)	
Parotid gland, mineral				1 (1%)
Parotid gland, vacuolation, cytoplasmic			1 (1%)	
Sublingual gland, atrophy		2 (2%)	3 (3%)	
Sublingual gland, fibrosis			1 (1%)	
Sublingual gland, metaplasia		1 (1%)		1 (1%)
Submandibular gland, atrophy			1 (1%)	1 (1%)
Stomach, forestomach	(90)	(90)	(90)	(90)
Cyst, squamous				1 (1%)
Edema	2 (2%)	2 (2%)	2 (2%)	3 (3%)
Erosion	2 (2%)	1 (1%)		
Fibrosis	1 (1%)	1 (1%)	1 (1%)	
Inflammation, acute				1 (1%)
Inflammation, chronic active	4 (4%)	5 (6%)	2 (2%)	1 (1%)
Ulcer	1 (1%)	3 (3%)	3 (3%)	3 (3%)
Epithelium, hyperplasia	10 (11%)	11 (12%)	8 (9%)	8 (9%)
Epithelium, hyperplasia, basal cell	1 (1%)	1 (1%)	2 (2%)	
Stomach, glandular	(90)	(90)	(89)	(88)
Cyst		1 (1%)		
Erosion	1 (1%)		1 (1%)	1 (1%)
Artery, inflammation, chronic active		1 (1%)		
Tongue	(1)	(0)	(0)	(0)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Blood vessel	(0)	(0)	(0)	(1)
Pulmonary artery, degeneration				1 (100%)
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	40 (44%)	43 (48%)	33 (37%)	45 (50%)
Artery, inflammation, chronic		1 (1%)		
Artery, mineral		1 (1%)		
Artery, necrosis		1 (1%)		
Atrium, endocardium, hyperplasia, Schwann cell			1 (1%)	
Endocardium, hyperplasia, Schwann cell		1 (1%)		1 (1%)
Epicardium, inflammation, acute			1 (1%)	
Ventricle right, cardiomyopathy	4 (4%)	7 (8%)	9 (10%)	9 (10%)

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(90)
Accessory adrenal cortical nodule	5 (6%)	7 (8%)	6 (7%)	12 (13%)
Atrophy	1 (1%)		2 (2%)	
Degeneration, cystic	22 (24%)	19 (21%)	18 (20%)	19 (21%)
Extramedullary hematopoiesis			1 (1%)	
Hyperplasia	14 (16%)	31 (34%)	26 (29%)	19 (21%)
Hypertrophy	52 (58%)	55 (61%)	56 (62%)	50 (56%)
Necrosis	2 (2%)	2 (2%)	2 (2%)	4 (4%)
Pigment	1 (1%)		1 (1%)	
Vacuolation, cytoplasmic	18 (20%)	17 (19%)	11 (12%)	14 (16%)
Adrenal medulla	(86)	(89)	(87)	(88)
Hyperplasia	13 (15%)	20 (22%)	20 (23%)	18 (20%)
Hypertrophy				1 (1%)
Necrosis	1 (1%)			1 (1%)
Islets, pancreatic	(90)	(89)	(90)	(88)
Hyperplasia	15 (17%)	12 (13%)	14 (16%)	13 (15%)
Parathyroid gland	(87)	(80)	(85)	(85)
Cyst			2 (2%)	
Fibrosis	13 (15%)	11 (14%)	6 (7%)	10 (12%)
Hyperplasia		2 (3%)		3 (4%)
Hyperplasia, focal	3 (3%)		2 (2%)	
Hypertrophy, focal				1 (1%)
Pituitary gland	(90)	(89)	(89)	(90)
Angiectasis		1 (1%)		
Atrophy			1 (1%)	
Cyst	1 (1%)		1 (1%)	
Fibrosis			1 (1%)	
Pigment			2 (2%)	
Pars distalis, angiectasis	2 (2%)			
Pars distalis, cyst	7 (8%)	5 (6%)	3 (3%)	1 (1%)
Pars distalis, hyperplasia	20 (22%)	22 (25%)	26 (29%)	22 (24%)
Pars distalis, vacuolation, cytoplasmic			1 (1%)	
Pars intermedia, cyst	3 (3%)	3 (3%)	1 (1%)	3 (3%)
Pars intermedia, hyperplasia	1 (1%)		1 (1%)	
Pars nervosa, developmental malformation				1 (1%)
Thyroid gland	(90)	(90)	(90)	(89)
C-cell, hyperplasia	28 (31%)	30 (33%)	34 (38%)	38 (43%)
C-cell, hypoplasia				1 (1%)
Follicle, cyst	1 (1%)		1 (1%)	
Follicular cell, hyperplasia		1 (1%)		
General Body System				
Tissue NOS	(8)	(11)	(8)	(6)
Cyst		1 (9%)		
Inflammation, chronic active	1 (13%)	1 (9%)		
Abdominal, fat, necrosis		5 (45%)	3 (38%)	2 (33%)
Fat, necrosis	6 (75%)	4 (36%)	4 (50%)	3 (50%)
Mediastinum, cyst				1 (17%)
Mediastinum, hemorrhage		1 (9%)		
Mediastinum, inflammation, chronic		1 (9%)		

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Genital System				
Clitoral gland	(87)	(88)	(89)	(86)
Hyperplasia		1 (1%)		
Inflammation, suppurative	1 (1%)	1 (1%)		
Inflammation, granulomatous				1 (1%)
Inflammation, acute		1 (1%)		
Inflammation, chronic			1 (1%)	1 (1%)
Inflammation, chronic active	28 (32%)	43 (49%)	35 (39%)	42 (49%)
Duct, dilation	47 (54%)	64 (73%)	65 (73%)	60 (70%)
Duct, hyperplasia		3 (3%)	4 (4%)	1 (1%)
Ovary	(90)	(90)	(89)	(90)
Atrophy	72 (80%)	69 (77%)	56 (63%)	77 (86%)
Congestion	1 (1%)			
Cyst	22 (24%)	27 (30%)	23 (26%)	34 (38%)
Fibrosis		1 (1%)	1 (1%)	1 (1%)
Hemorrhage				1 (1%)
Inflammation, chronic		1 (1%)	1 (1%)	
Inflammation, chronic active			1 (1%)	
Pigment				1 (1%)
Bursa, dilation	4 (4%)	4 (4%)	2 (2%)	1 (1%)
Follicle, cyst				1 (1%)
Periovarian tissue, cyst			1 (1%)	
Rete ovarii, cyst			1 (1%)	
Rete ovarii, hyperplasia	15 (17%)	17 (19%)	14 (16%)	11 (12%)
Oviduct	(1)	(0)	(0)	(0)
Cyst	1 (100%)			
Uterus	(90)	(90)	(90)	(90)
Adenomyosis		2 (2%)	2 (2%)	
Angiectasis	1 (1%)			
Cyst	5 (6%)	6 (7%)	7 (8%)	11 (12%)
Dilation	8 (9%)	10 (11%)	11 (12%)	8 (9%)
Fibrosis	1 (1%)		1 (1%)	
Hemorrhage			1 (1%)	4 (4%)
Infiltration cellular, plasma cell		1 (1%)		
Inflammation, suppurative	4 (4%)	11 (12%)	8 (9%)	12 (13%)
Inflammation, acute	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Inflammation, chronic active			4 (4%)	1 (1%)
Thrombus	1 (1%)	1 (1%)		
Cervix, hyperplasia, stromal	2 (2%)	1 (1%)	1 (1%)	1 (1%)
Cervix, thrombus		1 (1%)		
Cervix, epithelium, hyperplasia		1 (1%)		
Cervix, serosa, fibrosis	1 (1%)			
Endometrium, hyperplasia, cystic	37 (41%)	43 (48%)	35 (39%)	46 (51%)
Epithelium, metaplasia, squamous	48 (53%)	39 (43%)	28 (31%)	46 (51%)
Glands, dilation		1 (1%)		
Vagina	(2)	(1)	(0)	(1)
Cyst				1 (100%)
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Fibrosis		2 (2%)		
Hypercellularity	56 (62%)	52 (58%)	43 (48%)	43 (48%)

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Lymph node	(13)	(8) 1 (13%)	(11)	(20)
Erythrophagocytosis				1 (5%)
Axillary, erythrophagocytosis				
Axillary, proliferation, plasma cell	1 (8%)		1 (9%)	
Bronchial, erythrophagocytosis			1 (9%)	
Bronchial, proliferation, plasma cell			1 (9%)	
Deep cervical, erythrophagocytosis				1 (5%)
Iliac, erythrophagocytosis	3 (23%)	3 (38%)	1 (9%)	3 (15%)
Iliac, hyperplasia, lymphocyte	1 (8%)		1 (9%)	6 (30%)
Iliac, infiltration cellular, histiocyte				1 (5%)
Iliac, inflammation, acute	1 (8%)			
Iliac, pigment	1 (8%)			3 (15%)
Iliac, proliferation, plasma cell	6 (46%)	1 (13%)	2 (18%)	5 (25%)
Iliac, lymphatic sinus, ectasia		1 (13%)	1 (9%)	5 (25%)
Inguinal, erythrophagocytosis	1 (8%)			
Inguinal, hyperplasia, lymphocyte			1 (9%)	
Inguinal, pigment			1 (9%)	
Inguinal, proliferation, plasma cell	1 (8%)			
Inguinal, lymphatic sinus, ectasia	1 (8%)	1 (13%)	1 (9%)	
Lumbar, erythrophagocytosis	1 (8%)	2 (25%)		
Lumbar, hyperplasia, lymphocyte				1 (5%)
Lumbar, lymphatic sinus, ectasia				1 (5%)
Lymphatic sinus, renal, ectasia		1 (13%)		
Mediastinal, congestion	1 (8%)			
Mediastinal, erythrophagocytosis		2 (25%)	4 (36%)	4 (20%)
Mediastinal, proliferation, plasma cell	1 (8%)			
Pancreatic, erythrophagocytosis	1 (8%)		1 (9%)	
Pancreatic, infiltration cellular, histiocyte				1 (5%)
Renal, erythrophagocytosis		2 (25%)		
Lymph node, mandibular	(90)	(90)	(89)	(90)
Congestion		2 (2%)		1 (1%)
Erythrophagocytosis		1 (1%)	2 (2%)	3 (3%)
Hemorrhage	1 (1%)			
Hyperplasia, lymphocyte	46 (51%)	49 (54%)	45 (51%)	43 (48%)
Infiltration cellular, histiocyte				1 (1%)
Pigment				1 (1%)
Proliferation, plasma cell	68 (76%)	68 (76%)	58 (65%)	56 (62%)
Lymphatic sinus, ectasia	1 (1%)	2 (2%)	2 (2%)	3 (3%)
Lymph node, mesenteric	(90)	(90)	(90)	(89)
Atrophy	1 (1%)			
Erythrophagocytosis	1 (1%)	3 (3%)	2 (2%)	
Hyperplasia, lymphocyte			1 (1%)	
Infiltration cellular, histiocyte	2 (2%)			1 (1%)
Lymphatic sinus, ectasia			1 (1%)	1 (1%)
Spleen	(90)	(90)	(90)	(90)
Accessory spleen		1 (1%)		
Extramedullary hematopoiesis	80 (89%)	74 (82%)	79 (88%)	82 (91%)
Fibrosis		1 (1%)		
Hemorrhage		1 (1%)		1 (1%)
Hyperplasia, lymphocyte		1 (1%)		
Hyperplasia, stromal	1 (1%)		1 (1%)	
Pigment	74 (82%)	79 (88%)	77 (86%)	79 (88%)
Red pulp, atrophy	7 (8%)	11 (12%)	13 (14%)	6 (7%)
White pulp, atrophy	3 (3%)	3 (3%)	4 (4%)	1 (1%)

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Thymus	(87)	(83)	(87)	(87)
Atrophy	75 (86%)	67 (81%)	74 (85%)	63 (72%)
Cyst	39 (45%)	34 (41%)	34 (39%)	45 (52%)
Ectopic parathyroid gland	1 (1%)	2 (2%)	2 (2%)	2 (2%)
Hemorrhage	2 (2%)	5 (6%)	5 (6%)	3 (3%)
Hyperplasia, epithelial	55 (63%)	59 (71%)	54 (62%)	38 (44%)
Hyperplasia, lymphocyte			1 (1%)	
Artery, inflammation, chronic active		2 (2%)	1 (1%)	
Integumentary System				
Mammary gland	(90)	(90)	(90)	(90)
Galactocele	24 (27%)	17 (19%)	17 (19%)	10 (11%)
Hyperplasia	49 (54%)	50 (56%)	46 (51%)	34 (38%)
Inflammation, granulomatous			2 (2%)	
Inflammation, acute				1 (1%)
Inflammation, chronic active			2 (2%)	1 (1%)
Duct, dilation	56 (62%)	61 (68%)	51 (57%)	70 (78%)
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	1 (1%)	1 (1%)	3 (3%)	1 (1%)
Hyperkeratosis		1 (1%)		
Inflammation, chronic active	1 (1%)			1 (1%)
Ulcer			1 (1%)	
Epidermis, hyperplasia	2 (2%)			
Lymphatic, subcutaneous tissue, angiectasis				1 (1%)
Subcutaneous tissue, inflammation, chronic active		1 (1%)		
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Fibrous osteodystrophy		1 (1%)		
Cranium, fracture	1 (1%)			
Mandible, fracture	1 (1%)			
Maxilla, fracture	1 (1%)			
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	3 (3%)	7 (8%)	10 (11%)	2 (2%)
Diaphragm, hernia				1 (1%)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	26 (29%)	31 (34%)	16 (18%)	20 (22%)
Congestion	1 (1%)			
Cyst		1 (1%)		
Edema	2 (2%)	1 (1%)		
Hemorrhage		1 (1%)		
Mineral		1 (1%)	1 (1%)	
Pigment			1 (1%)	
Cerebrum, degeneration			1 (1%)	
Choroid plexus, mineral		1 (1%)		
Glial cell, hyperplasia			1 (1%)	1 (1%)
Meninges, hyperplasia	1 (1%)		1 (1%)	
Meninges, hyperplasia, granular cell	1 (1%)			1 (1%)
Meninges, mineral			1 (1%)	

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Nervous System (continued)				
Brain (continued)	(90)	(90)	(90)	(90)
Neuron, necrosis		1 (1%)		
Pineal gland, infiltration cellular, mononuclear cell			1 (1%)	
Pineal gland, mineral	1 (1%)			
Pineal gland, vacuolation, cytoplasmic	1 (1%)	(84)	(85)	(84)
Nerve trigeminal	(84)			
Degeneration	64 (76%)	70 (83%)	64 (75%)	72 (86%)
Gliosis			1 (1%)	
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	80 (89%)	83 (92%)	83 (92%)	89 (99%)
Infiltration cellular, mixed cell	1 (1%)			
Peripheral nerve, tibial	(90)	(90)	(89)	(89)
Degeneration	77 (86%)	77 (86%)	83 (93%)	86 (97%)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Degeneration	24 (27%)	29 (32%)	22 (24%)	35 (39%)
Spinal cord, lumbar	(90)	(90)	(89)	(90)
Degeneration	10 (11%)	11 (12%)	15 (17%)	12 (13%)
Nerve, degeneration	74 (82%)	77 (86%)	77 (87%)	80 (89%)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	59 (66%)	64 (71%)	59 (66%)	70 (78%)
Trigeminal ganglion	(81)	(77)	(81)	(75)
Degeneration	33 (41%)	21 (27%)	22 (27%)	28 (37%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Congestion	3 (3%)	12 (13%)	9 (10%)	5 (6%)
Foreign body		1 (1%)		1 (1%)
Hemorrhage	1 (1%)	6 (7%)		1 (1%)
Inflammation, suppurative	2 (2%)			1 (1%)
Inflammation, granulomatous	1 (1%)	5 (6%)	1 (1%)	2 (2%)
Inflammation, chronic active	6 (7%)	6 (7%)	6 (7%)	11 (12%)
Alveolar epithelium, hyperplasia		1 (1%)		
Alveolar epithelium, metaplasia, squamous		1 (1%)		2 (2%)
Alveolus, infiltration cellular, histiocyte	71 (79%)	77 (86%)	84 (93%)	81 (90%)
Artery, inflammation, chronic active	1 (1%)			
Artery, muscularis, hyperplasia				1 (1%)
Bronchus, hyperplasia		1 (1%)		
Epithelium alveolus, hyperplasia	2 (2%)	2 (2%)	3 (3%)	1 (1%)
Pleura, inflammation, acute			1 (1%)	
Nose	(90)	(89)	(90)	(89)
Foreign body		1 (1%)	1 (1%)	2 (2%)
Inflammation, suppurative	1 (1%)	3 (3%)	1 (1%)	3 (3%)
Inflammation, acute		1 (1%)		
Inflammation, chronic active		1 (1%)		
Nerve, degeneration		1 (1%)		
Olfactory epithelium, accumulation, hyaline droplet	89 (99%)	89 (100%)	86 (96%)	86 (97%)
Olfactory epithelium, hyperplasia			1 (1%)	
Olfactory epithelium, metaplasia, respiratory	1 (1%)			2 (2%)
Olfactory epithelium, metaplasia, squamous		1 (1%)		

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Respiratory System (continued)				
Nose (continued)	(90)	(89)	(90)	(89)
Respiratory epithelium, accumulation, hyaline droplet	12 (13%)	19 (21%)	22 (24%)	11 (12%)
Respiratory epithelium, hyperplasia		1 (1%)		3 (3%)
Respiratory epithelium, metaplasia, squamous				1 (1%)
Trachea	(89)	(88)	(89)	(89)
Inflammation, chronic active	1 (1%)	1 (1%)		
Epithelium, hyperplasia		1 (1%)		
Epithelium, metaplasia, squamous		1 (1%)		
Glands, cyst	1 (1%)	1 (1%)		2 (2%)
Special Senses System				
Ear	(0)	(0)	(1)	(1)
Eye	(88)	(86)	(88)	(86)
Anterior chamber, exudate		1 (1%)		
Anterior chamber, inflammation, acute			1 (1%)	2 (2%)
Anterior chamber, iris, synechia		1 (1%)		
Choroid, inflammation, chronic active			1 (1%)	
Cornea, fibrosis		1 (1%)		
Cornea, inflammation, acute	1 (1%)	2 (2%)	1 (1%)	2 (2%)
Cornea, inflammation, chronic active		1 (1%)		
Cornea, neovascularization		1 (1%)		
Cornea, ulcer				1 (1%)
Cornea, epithelium, hyperplasia	1 (1%)	2 (2%)		1 (1%)
Lens, cataract	1 (1%)	3 (3%)		
Retina, atrophy	18 (20%)	17 (20%)	18 (20%)	18 (21%)
Retina, dysplasia	1 (1%)	1 (1%)	1 (1%)	3 (3%)
Harderian gland	(90)	(90)	(90)	(90)
Atrophy	13 (14%)	15 (17%)	16 (18%)	17 (19%)
Cyst				1 (1%)
Hyperplasia				1 (1%)
Hypertrophy				1 (1%)
Infiltration cellular, lymphocyte	2 (2%)			1 (1%)
Inflammation, granulomatous	7 (8%)	6 (7%)	4 (4%)	9 (10%)
Inflammation, chronic	7 (8%)	1 (1%)	1 (1%)	2 (2%)
Inflammation, chronic active	1 (1%)	4 (4%)	2 (2%)	
Urinary System				
Kidney	(90)	(90)	(90)	(89)
Inflammation, acute	1 (1%)			
Nephropathy, chronic progressive	74 (82%)	76 (84%)	76 (84%)	65 (73%)
Artery, inflammation, chronic active	1 (1%)			
Pelvis, dilation	3 (3%)		2 (2%)	
Pelvis, inflammation, suppurative		2 (2%)		
Pelvis, mineral				1 (1%)
Pelvis, urothelium, hyperplasia		1 (1%)		
Renal tubule, cyst	3 (3%)	2 (2%)		
Renal tubule, hyperplasia			1 (1%)	
Renal tubule, necrosis		1 (1%)		

TABLE D4
**Summary of the Incidence of Nonneoplastic Lesions in Female Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 2 Years**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
2-Year Study (continued)				
Urinary System (continued)				
Urinary bladder	(88)	(88)	(90)	(90)
Dilation	1 (1%)			
Edema		3 (3%)		
Fibrosis			1 (1%)	
Hemorrhage		1 (1%)	1 (1%)	
Infiltration cellular, histiocyte		1 (1%)	1 (1%)	
Inflammation, acute	3 (3%)	2 (2%)		
Inflammation, chronic active		1 (1%)		
Necrosis	1 (1%)			
Artery, inflammation, chronic active		1 (1%)		
Urothelium, hyperplasia	1 (1%)			

APPENDIX E

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GENETIC TOXICOLOGY

COLLECTION OF TISSUE SAMPLES FOR GENOTOXICITY TESTING

Exposures ceased at 7 a.m. on the day of necropsy at 14 weeks after weaning. Thirty-five male rats (five sham controls, 15 that were exposed to CDMA, and 15 that were exposed to GSM) were necropsied approximately 2 to 4 hours after cessation of exposure and 35 female rats (five sham controls, 15 that were exposed to CDMA, and 15 that were exposed to GSM) were necropsied approximately 5 to 7 hours after cessation of exposure. Animals were necropsied in the following order: one animal from each exposure group starting with the sham control group, moving through each of the exposed groups for each of the radiofrequency modulations in turn, then rotating back to the sham control group; animals were necropsied in numerical order within each exposure group. Five different tissues (cerebrum, frontal cortex, hippocampus, liver, and blood leukocytes) were collected from each animal for the comet assay. Because blood was examined in both the micronucleus and the comet assays, a single tube of blood was collected per animal by retroorbital bleeding, and the sample was divided into two aliquots, one that was processed for the comet assay and the other for the micronucleus assay.

COMET ASSAY

For preparation of samples for the comet assay, a 50 µL sample of blood was transferred to a tube containing 1 mL of freshly prepared cold mincing buffer [Mg^{+2} , Ca^{+2} , and phenol free Hank's Balanced Salt Solution (Life Technologies, Carlsbad, CA) with 20 mM ethylenediaminetetraacetic acid (EDTA) pH 7.3 to 7.5 and 10% v/v fresh dimethyl sulfoxide (DMSO)]. The liver and the hippocampus, cerebellum, and frontal cortex sections of the brain were rinsed with cold mincing buffer to remove residual blood and held on ice briefly (≤ 5 minutes) until processed. Small portions (3 to 4 mm) of the left lobe of the liver and each brain section were placed in tubes containing cold mincing solution and rapidly minced until finely dispersed. All samples prepared for the comet assay were immediately flash frozen in liquid nitrogen (Recio *et al.*, 2010) and subsequently transferred to a -80° C freezer for storage until shipment by overnight courier on dry ice to the analytical laboratory. Upon receipt, all samples were immediately placed in a -80° C freezer for storage until further processing.

Blood and tissue samples were thawed on ice and maintained on ice during slide preparation. Just prior to use, each cell suspension was shaken gently to mix the cells and placed back on ice for 15 to 30 seconds to allow clumps to settle. A portion of the supernatant was empirically diluted with 0.5% low melting point agarose (Lonza, Walkersville, MD) dissolved in Dulbecco's phosphate buffer (Ca^{+2} , Mg^{+2} , and phenol free) at 37° C and layered onto each well of a 2-well CometSlide™ (Trevigen, Gaithersburg, MD). Slides were immersed in cold lysing solution [2.5 M NaCl, 100 mM Na₂EDTA, 10 mM tris(hydroxymethyl)aminomethane (Tris), pH 10, containing freshly added 10% DMSO (Fisher Scientific, Pittsburgh, PA) and 1% Triton X-100] overnight in a refrigerator, protected from light. The following day, the slides were rinsed in 0.4 M Trizma base (pH 7.5), randomly placed onto the platform of a horizontal electrophoresis unit and treated with cold alkali solution (300 mM NaOH, 1 mM Na₂EDTA, pH>13) for 20 minutes to allow DNA unwinding, then electrophoresed at 4° to 9° C for 20 minutes at 25 V (0.7 V/cm), with a current of approximately 300 mA. Following electrophoresis, slides were neutralized with 0.4 M Trizma base (pH 7.5) for 5 minutes and then dehydrated by immersion in absolute ethanol (Pharmco-AAPER, Shelbyville, KY) for at least 5 minutes and allowed to air dry. Slides were prepared in a laboratory with a relative humidity of no more than 60% and stored at room temperature in a desiccator with a relative humidity of no more than 60% until stained and scored; stained slides were stored in a desiccator. NaCl, Na₂EDTA, Triton X-100, and Trizma base were purchased from Sigma-Aldrich (St. Louis, MO); NaOH was purchased from Fisher Scientific (Pittsburgh, PA).

After staining with SYBR® Gold (Molecular Probes, Life Technologies, Grand Island, NY), slides, independently coded to mask treatment, were scored using Comet Assay IV Imaging Software, Version 4.3.1 (Perceptive Instruments, Ltd., Suffolk, UK) validated for GLP Part 11 compliance. In the alkaline (pH>13) comet assay, when damaged nuclear DNA fragments, it undergoes unidirectional migration through the agarose gel within an electrical field, forming an image that resembles a comet, and the greater the amount of fragmentation, the greater the amount of DNA migration that will occur. The image analysis software partitions the intensity of the fluorescent signal of the DNA in the entire comet image into the percent that is attributable to the comet head and the percent attributable

to the tail. Manual adjustment of the automated detection of head and tail features is sometimes required. To evaluate DNA damage levels, the extent of DNA migration was characterized for 100 scorable comet figures per animal/tissue as percent tail DNA (intensity of all tail pixels divided by the total intensity of all pixels in the comet, expressed as a percentage).

Comet figures are classified during the scoring process as scorable (evaluated for percent tail DNA), non-scoreable (due to inability to evaluate percent tail DNA, e.g. if comets overlapped), and “hedgehog.” Hedgehogs either have no defined head, i.e., all DNA appears to be in the tail, or the head and tail appear to be separated. Hedgehogs may represent cells that have sustained high levels of DNA damage and are apoptotic, although certain data suggest they may represent cells with high levels of repairable DNA damage (Rundell *et al.*, 2003; Lorenzo *et al.*, 2013). The frequency of hedgehogs (%HH) was determined by tabulating the number observed in a separate group of 100 cells per animal/tissue.

When rat samples were scored, a marked interanimal variation in percent tail DNA and high %HH values were observed in some tissues, yet the range of percent tail DNA values appeared to be truncated at approximately 65%. To better understand these observations, rat slides were reanalyzed by scoring 150 cells/tissue per animal, as recommended by the OECD guideline (OECD, 2014). In this rescore of the rat samples, all scorable cells were included in the sample of 150 analyzed cells, regardless of the apparent level of DNA damage estimated by the scorer prior to software analysis of the images; highly damaged cells that were unscorable using the software (true HH) were not included. For the 150-cell scoring method, the %HH was not independently determined due to limitations at the time in the comet assay software arising from the added number of cells scored. Therefore, %HH was estimated by dividing the number of comets having more than 90% tail DNA by 150.

Although there was no concurrent positive control group in these cell phone RFR studies, slides were made with human TK6 cells treated with ethyl methanesulfonate (standard positive control compound for the comet assay) and were included in each electrophoresis run with each slide set as an internal technical positive control.

MICRONUCLEUS ASSAY

For the micronucleus assay, sampling schedules were as described for the comet assay. At 14 weeks after weaning, blood samples (approximately 200 µL) obtained by retroorbital bleeding (one sample per rat) were placed into EDTA tubes and immediately refrigerated. The samples were sent on the day of collection to the analytical laboratory well insulated on cold packs via overnight delivery. Upon arrival, blood samples were diluted in anticoagulant (heparin) and fixed in ice cold methanol (Sigma-Aldrich, St. Louis, MO) according to instructions provided with the MicroFlow^{PLUS} Kit (Litron Laboratories, Rochester, NY). Fixed blood samples were stored in a -80° C freezer for at least 3 days prior to analysis by flow cytometry.

Flow cytometric analysis of red blood cell samples was performed using MicroFlow^{PLUS} Kit reagents and a FACSCalibur™ dual-laser bench top system (Becton Dickinson Biosciences, San Jose, CA) as described by MacGregor *et al.* (2006) and Witt *et al.* (2008). Both mature [normochromic erythrocytes (NCEs)] and immature [reticulocytes; polychromatic erythrocytes (PCEs)] erythrocytes were analyzed for the presence of micronuclei. Immature erythrocytes are distinguished by the presence of an active transferrin receptor (CD-71) on the cell surface. For each sample, 20,000 (\pm 2,000) immature CD71-positive erythrocytes were analyzed by flow cytometry to determine the frequency of micronucleated reticulocytes. Aggregates were excluded on the basis of forward and side scatter, platelets were excluded based on staining with an anti-CD61 antibody, and nucleated leukocytes were excluded on the basis of intense propidium iodide staining. Typically, more than one million NCEs (CD-71 negative) were enumerated concurrently during PCE analysis, allowing for calculation of the percentage of PCEs among total erythrocytes as a measure of bone marrow toxicity.

DATA ANALYSIS FOR THE COMET AND MICRONUCLEUS ASSAYS

Data from both the comet and the micronucleus assays were analyzed using the same statistical methods (Kissling *et al.*, 2007). Mean percent tail DNA was calculated for each cell type for each animal; likewise, mean micronucleated PCEs/1,000 PCEs and micronucleated NCEs/1,000 NCEs, as well as % PCEs, were calculated for each animal. These data are summarized in the tables as mean \pm standard error of the mean. Levene's test was used

to determine if variances among exposed groups were equal at $P=0.05$. When variances were equal, linear regression analysis was used to test for linear trend and Williams' test was used to evaluate pairwise differences of each exposed group with the sham control group. When variances were unequal, nonparametric methods were used to analyze the data; Jonckheere's test was used to evaluate linear trend and Dunn's test was used to assess the significance of pairwise differences of each exposed group with the sham control group. To maintain the overall significance level at 0.05, the trend as well as the pairwise differences from the sham control group were declared statistically significant if $P<0.025$. A result was considered positive if the trend test was significant and if at least one exposed group was significantly elevated over the sham control group, or if two or more exposed groups were significantly increased over the corresponding sham control group. A response was considered equivocal if only the trend test was significant or if only a single exposed group was significantly increased over the sham control.

RESULTS

Twenty tissue samples obtained from animals in the 14-week interim evaluation study were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, five tissues). Results are reported based on the standard 100-cell scoring approach in use at the time these data were collected; data obtained using a 150-cell scoring approach, recommended in a recently adopted international guideline for the *in vivo* comet assay, are noted for comparison. The only clear positive result was observed in hippocampus cells of male rats exposed to the CDMA modulation (Tables E1 and E2). Data obtained using the 150-cell scoring approach did not meet the statistical criteria for a positive result, although the mean percent tail DNA values were elevated over the sham controls in all exposure groups, and the values increased with increasing dose level (Table E3). An exposure-related increase in DNA damage was seen in the cells of the frontal cortex of male rats exposed to the CDMA modulation (Table E1). However, although the trend test was significant ($P=0.004$), no individual exposure groups were significantly elevated over the sham control group and the result was therefore judged to be equivocal. Data obtained using the 15-cell scoring approach showed a similar pattern of response in the male frontal cortex (CDMA) and were also considered to be equivocal based on a significant trend test ($P=0.005$) (Table E3). For male rat blood leukocytes exposed to either the CDMA or GSM modulation (Tables E1 and E2), results from scoring 100 cells were negative; however, these leukocyte samples showed equivocal responses with the 150-cell method due to a significant trend test ($P=0.012$) or pairwise test ($P=0.021$) for CDMA- and GSM-exposed rats, respectively (Tables E3 and E4). No statistically significant increases in the percent tail DNA were observed in any of the female rat samples scored with the 100-cell approach (Tables E5 and E6). The 150-cell scoring approach yielded a significant trend test ($P=0.013$) in peripheral blood leukocytes of female rats exposed to the CDMA modulation, but these results were driven by data from a single animal (Table E7).

In contrast to what was seen in the mice, a high degree of interanimal variability was observed in the percent tail DNA values in rats within a treatment group, and this level of variability reduced the statistical power to detect increases in DNA migration, although the magnitudes of the increases observed in some rats suggested these were treatment-related effects. To rule out any influence from technical artifacts or protocol features, percent tail DNA values and percent hedgehogs were correlated to the position of slides in the electrophoresis chambers, the interval from exposure cessation to tissue collection, and the date of slide preparation; no patterns emerged for any of these variables and the level of DNA damage observed.

Similar to what was seen in the mice, no significant increases in micronucleated red blood cells or in the percentage of reticulocytes were observed in rats of either sex exposed to either modulation of cell phone RFR (Tables E9).

TABLE E1
DNA Damage in Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 19 Weeks (100-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^b
Frontal Cortex				
Sham Control ^d	0	6.18 ± 0.72		2.00 ± 0.71
CDMA	1.5	6.00 ± 0.48	1.000	1.00 ± 0.77
	3	9.51 ± 1.17	0.081	10.60 ± 3.89
	6	12.78 ± 3.96	0.049	12.20 ± 6.84
		P=0.004 ^e		
Hippocampus				
Sham Control	0	5.88 ± 0.39		3.40 ± 1.21
CDMA	1.5	8.06 ± 1.20	0.135	3.80 ± 2.33
	3	8.16 ± 0.98	0.151	6.20 ± 2.56
	6	10.42 ± 2.18	0.019	4.40 ± 2.98
		P=0.014		
Cerebellum				
Sham Control	0	5.57 ± 0.92		0.40 ± 0.24
CDMA	1.5	5.60 ± 0.71	1.000	1.80 ± 0.80
	3	10.70 ± 3.66	0.504	9.40 ± 6.81
	6	10.58 ± 3.52	0.731	8.00 ± 3.91
		P=0.156		
Liver				
Sham Control	0	13.81 ± 2.88		33.60 ± 17.89
CDMA	1.5	22.99 ± 2.77	0.081	68.60 ± 15.70
	3	16.04 ± 2.14	0.098	7.80 ± 0.86
	6	20.79 ± 3.10	0.057	41.10 ± 14.80
		P=0.154		
Peripheral Blood				
Sham Control	0	1.48 ± 0.29		0.20 ± 0.20
CDMA	1.5	1.22 ± 0.45	0.596	0.80 ± 0.80
	3	2.13 ± 0.34	0.156	0.40 ± 0.40
	6	2.08 ± 0.43	0.166	1.40 ± 1.17
		P=0.071		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' or Dunn's test.

^d No CDMA radiofrequency radiation exposure

^e Dose-related trend; significant at P≤0.025 by linear regression or Jonckheere's test.

TABLE E2
DNA Damage in Male Rats Exposed to GSM-Modulated Cell Phone RFR for 19 Weeks (100-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^b
Frontal Cortex				
Sham Control ^d	0	6.18 ± 0.72		2.00 ± 0.71
GSM	1.5	6.98 ± 0.42	0.465	1.40 ± 0.51
	3	8.66 ± 1.96	0.247	8.20 ± 2.69
	6	6.30 ± 0.32	1.000	3.00 ± 1.55
		P=0.343 ^e		
Hippocampus				
Sham Control	0	5.88 ± 0.39		3.40 ± 1.21
GSM	1.5	11.82 ± 2.68	0.092	4.80 ± 2.84
	3	9.64 ± 1.27	0.111	4.80 ± 1.53
	6	11.69 ± 3.92	0.072	10.20 ± 7.98
		P=0.103		
Cerebellum				
Sham Control	0	5.57 ± 0.92		0.40 ± 0.24
GSM	1.5	7.36 ± 2.48	0.295	2.40 ± 1.91
	3	6.37 ± 0.77	0.354	3.40 ± 1.17
	6	8.48 ± 1.85	0.149	5.00 ± 2.86
		P=0.132		
Liver				
Sham Control	0	13.81 ± 2.88		33.60 ± 17.89
GSM	1.5	13.26 ± 2.38	0.547	21.00 ± 12.30
	3	13.09 ± 2.32	0.634	28.40 ± 15.07
	6	14.49 ± 2.71	0.536	24.80 ± 16.13
		P=0.404		
Peripheral Blood				
Sham Control	0	1.48 ± 0.29		0.20 ± 0.20
GSM	1.5	1.83 ± 0.63	0.352	3.20 ± 2.71
	3	1.78 ± 0.33	0.419	1.20 ± 0.49
	6	1.50 ± 0.27	0.446	0.40 ± 0.24
		P=0.550		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' or Dunn's test.

^d No GSM radiofrequency radiation exposure

^e Dose-related trend; significant at P≤0.025 by linear regression or Jonckheere's test.

TABLE E3
DNA Damage in Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 19 Weeks (150-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^{b,d}
Frontal Cortex				
Sham Control ^e	0	9.73 ± 0.81		0.27 ± 0.27
CDMA	1.5	8.24 ± 0.39	1.000	0.13 ± 0.13
	3	18.77 ± 3.27	0.043	2.53 ± 1.29
	6	23.62 ± 8.66	0.092	3.20 ± 1.72
		P=0.005 ^f		
Hippocampus				
Sham Control	0	8.99 ± 1.55		1.07 ± 0.45
CDMA	1.5	12.27 ± 2.21	0.244	0.40 ± 0.27
	3	15.46 ± 2.25	0.107	2.53 ± 0.90
	6	16.77 ± 5.44	0.069	2.40 ± 1.44
		P=0.043		
Cerebellum				
Sham Control	0	4.90 ± 0.82		0.00 ± 0.00
CDMA	1.5	6.33 ± 1.00	0.681	0.27 ± 0.16
	3	13.75 ± 6.01	0.504	2.93 ± 2.20
	6	15.86 ± 5.91	0.163	2.40 ± 1.07
		P=0.061		
Liver				
Sham Control	0	25.71 ± 8.71		1.73 ± 1.73
CDMA	1.5	55.41 ± 7.91	0.136	14.67 ± 5.57
	3	19.11 ± 2.28	0.164	0.80 ± 0.49
	6	40.01 ± 7.90	0.114	9.07 ± 7.10
		P=0.385		
Peripheral Blood				
Sham Control	0	0.69 ± 0.20		0.00 ± 0.00
CDMA	1.5	1.16 ± 0.47	0.295	0.00 ± 0.00
	3	1.83 ± 0.74	0.121	0.13 ± 0.13
	6	2.57 ± 0.80	0.026	0.00 ± 0.00
		P=0.012		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010) and OECD (2014). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6. Statistical analysis runs Levene's test to determine if variances of dosed groups are equal ($P = 0.05$). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at $P \leq 0.025$ by Williams' or Dunn's test.

^d Percent hedgehogs = estimated as the number of comets with >90% tail DNA/150

^e No CDMA radiofrequency radiation exposure

^f Dose-related trend; significant at $P \leq 0.025$ by linear regression or Jonckheere's test.

TABLE E4
DNA Damage in Male Rats Exposed to GSM-Modulated Cell Phone RFR for 19 Weeks (150-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^{b,d}
Frontal Cortex				
Sham Control ^e	0	9.73 ± 0.81		0.27 ± 0.27
GSM	1.5	11.96 ± 1.65	0.634	0.40 ± 0.27
	3	17.98 ± 5.12	0.545	1.20 ± 0.57
	6	9.57 ± 1.57	1.000	0.13 ± 0.13
		P=0.500 ^f		
Hippocampus				
Sham Control	0	8.99 ± 1.55		1.07 ± 0.45
GSM	1.5	17.24 ± 4.09	0.186	0.27 ± 0.16
	3	14.77 ± 2.54	0.227	1.47 ± 0.57
	6	21.32 ± 9.55	0.080	3.60 ± 2.03
		P=0.076		
Cerebellum				
Sham Control	0	4.90 ± 0.82		0.00 ± 0.00
GSM	1.5	9.43 ± 4.69	0.190	1.33 ± 1.17
	3	8.66 ± 2.17	0.232	1.47 ± 0.68
	6	12.11 ± 3.89	0.088	1.07 ± 1.07
		P=0.076		
Liver				
Sham Control	0	25.71 ± 8.71		1.73 ± 1.73
GSM	1.5	23.27 ± 9.43	0.539	4.13 ± 3.64
	3	25.15 ± 8.43	0.604	0.40 ± 0.40
	6	28.25 ± 10.55	0.534	4.93 ± 3.94
		P=0.390		
Peripheral Blood				
Sham Control	0	0.69 ± 0.20		0.00 ± 0.00
GSM	1.5	3.97 ± 2.75	0.146	0.27 ± 0.27
	3	1.97 ± 0.35	0.021	0.00 ± 0.00
	6	1.28 ± 0.23	0.272	0.00 ± 0.00
		P=0.089		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010) and OECD (2014). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6. Statistical analysis runs Levene's test to determine if variances of dosed groups are equal ($P = 0.05$). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at $P \leq 0.025$ by Williams' or Dunn's test.

^d Percent hedgehogs = estimated as the number of comets with >90% tail DNA/150

^e No GSM radiofrequency radiation exposure

^f Dose-related trend; significant at $P \leq 0.025$ by linear regression or Jonckheere's test.

TABLE E5
DNA Damage in Female Rats Exposed to CDMA-Modulated Cell Phone RFR for 19 Weeks
(100-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^b
Frontal Cortex				
Sham Control ^d	0	7.03 ± 1.21		3.80 ± 1.46
CDMA	1.5	12.70 ± 5.15	0.205	19.00 ± 15.04
	3	9.50 ± 2.27	0.249	9.80 ± 5.12
	6	13.00 ± 3.63	0.150	25.40 ± 11.44
		P=0.166 ^e		
Hippocampus				
Sham Control	0	13.14 ± 1.20		9.00 ± 15.73
CDMA	1.5	14.943 ± 0.704	0.346	8.40 ± 1.96
	3	15.237 ± 1.967	0.379	9.40 ± 2.89
	6	19.107 ± 5.269	0.126	21.20 ± 11.12
		P=0.080		
Cerebellum				
Sham Control	0	5.94 ± 0.98		3.80 ± 1.07
CDMA	1.5	4.91 ± 0.58	0.671	2.00 ± 1.05
	3	5.46 ± 0.83	0.747	2.00 ± 0.63
	6	5.86 ± 0.84	0.650	1.20 ± 0.37
		P=0.421		
Liver				
Sham Control	0	10.09 ± 0.87		7.00 ± 1.87
CDMA	1.5	15.26 ± 3.35	0.634	33.40 ± 15.11
	3	11.49 ± 2.05	1.000	12.40 ± 3.59
	6	18.35 ± 3.44	0.163	31.40 ± 12.33
		P=0.113		
Peripheral Blood				
Sham Control	0	3.15 ± 0.40		0.20 ± 0.20
CDMA	1.5	3.77 ± 1.19	0.371	1.20 ± 0.80
	3	4.13 ± 0.54	0.361	0.40 ± 0.40
	6	6.06 ± 2.18	0.082	9.80 ± 8.81
		P=0.048		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6. Statistical analysis runs Levene's test to determine if variances of dosed groups are equal ($P = 0.05$). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at $P \leq 0.025$ by Williams' or Dunn's test.

^d No CDMA radiofrequency radiation exposure

^e Dose-related trend; significant at $P \leq 0.025$ by linear regression or Jonckheere's test.

TABLE E6
DNA Damage in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 19 Weeks (100-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^b
Frontal Cortex				
Sham Control ^d	0	7.03 ± 1.21		3.80 ± 1.46
GSM	1.5	4.87 ± 0.47	0.820	2.20 ± 0.73
	3	6.18 ± 0.67	0.843	5.60 ± 2.36
	6	6.74 ± 0.74	0.723	6.40 ± 2.73
		P=0.386 ^e		
Hippocampus				
Sham Control	0	13.14 ± 1.20		9.00 ± 2.58
GSM	1.5	13.22 ± 1.56	0.936	7.25 ± 3.20
	3	17.67 ± 3.64	0.351	19.50 ± 7.89
	6	13.21 ± 1.03	1.000	10.00 ± 3.81
		P=0.334		
Cerebellum				
Sham Control	0	5.94 ± 0.98		3.80 ± 1.07
GSM	1.5	5.69 ± 0.75	0.662	2.00 ± 0.71
	3	4.62 ± 0.85	0.749	0.60 ± 0.24
	6	6.62 ± 0.96	0.381	2.40 ± 1.03
		P=0.302		
Liver				
Sham Control	0	10.09 ± 0.87		7.00 ± 1.87
GSM	1.5	9.91 ± 2.60	1.000	13.20 ± 11.23
	3	9.46 ± 2.07	1.000	17.00 ± 14.76
	6	18.99 ± 6.20	1.000	35.20 ± 19.42
		P=0.394		
Peripheral Blood				
Sham Control	0	3.15 ± 0.40		0.20 ± 0.20
GSM	1.5	2.80 ± 0.33	0.593	0.80 ± 0.49
	3	3.39 ± 0.68	0.447	0.60 ± 0.24
	6	3.93 ± 0.63	0.203	1.00 ± 0.32
		P=0.093		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6. Statistical analysis runs Levene's test to determine if variances of dosed groups are equal ($P = 0.05$). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at $P \leq 0.025$ by Williams' or Dunn's test.

^d No GSM radiofrequency radiation exposure

^e Dose-related trend; significant at $P \leq 0.025$ by linear regression or Jonckheere's test.

TABLE E7
DNA Damage in Female Rats Exposed to CDMA-Modulated Cell Phone RFR for 19 Weeks
(150-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^{b,d}
Frontal Cortex				
Sham Control ^e	0	12.23 ± 2.18		0.40 ± 0.16
CDMA	1.5	25.37 ± 12.96	0.782	8.67 ± 7.67
	3	18.70 ± 5.28	0.634	1.87 ± 0.88
	6	33.49 ± 11.14	0.092	7.20 ± 5.62
		P=0.035 ^f		
Hippocampus				
Sham Control	0	18.08 ± 1.30		0.83 ± 0.32
CDMA	1.5	20.58 ± 2.056	0.531	1.07 ± 0.34
	3	20.63 ± 1.920	0.382	1.33 ± 0.21
	6	29.55 ± 9.439	0.218	6.53 ± 5.23
		P=0.068		
Cerebellum				
Sham Control	0	4.93 ± 1.09		0.00 ± 0.00
CDMA	1.5	4.61 ± 1.61	0.621	0.53 ± 0.53
	3	3.89 ± 0.43	0.709	0.13 ± 0.13
	6	5.88 ± 0.63	0.342	0.27 ± 0.16
		P=0.249		
Liver				
Sham Control	0	12.41 ± 1.64		0.13 ± 0.13
CDMA	1.5	26.15 ± 8.57	0.145	4.00 ± 3.67
	3	16.17 ± 2.17	0.176	0.67 ± 0.42
	6	26.65 ± 6.91	0.059	2.00 ± 1.17
		P=0.102		
Peripheral Blood				
Sham Control	0	3.32 ± 0.09		0.13 ± 0.13
CDMA	1.5	4.45 ± 1.53	1.000	0.40 ± 0.27
	3	3.94 ± 0.40	0.465	0.13 ± 0.13
	6	12.76 ± 7.59	0.028	2.93 ± 2.77
		P=0.013		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010) and OECD (2014). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6. Statistical analysis runs Levene's test to determine if variances of dosed groups are equal ($P = 0.05$). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at $P \leq 0.025$ by Williams' or Dunn's test.

^d Percent hedgehogs = estimated as the number of comets with >90% tail DNA/150

^e No CDMA radiofrequency radiation exposure

^f Dose-related trend; significant at $P \leq 0.025$ by linear regression or Jonckheere's test.

TABLE E8
DNA Damage in Female Rats Exposed to GSM-Modulated Cell Phone RFR for 19 Weeks (150-Cell Method)^a

	Dose (W/kg)	Percent Tail DNA ^b	P Value ^c	Percent Hedgehogs ^{b,d}
Frontal Cortex				
Sham Control ^e	0	12.23 ± 2.18		0.40 ± 0.16
GSM	1.5	6.28 ± 1.00	0.856	0.00 ± 0.00
	3	9.83 ± 1.11	0.877	0.67 ± 0.21
	6	13.74 ± 2.79	0.376	0.13 ± 0.13
		P=0.137 ^f		
Hippocampus				
Sham Control	0	18.08 ± 1.296		0.83 ± 0.32
GSM	1.5	17.54 ± 3.59	1.000	1.50 ± 1.29
	3	28.08 ± 7.00	0.662	3.66 ± 1.40
	6	18.19 ± 3.35	1.000	2.93 ± 1.53
		P=0.534		
Cerebellum				
Sham Control	0	4.93 ± 1.09		0.00 ± 0.00
GSM	1.5	5.11 ± 0.63	0.731	0.00 ± 0.00
	3	3.51 ± 0.74	1.000	0.00 ± 0.00
	6	6.54 ± 2.33	1.000	0.27 ± 0.16
		P=0.705		
Liver				
Sham Control	0	12.41 ± 1.64		0.13 ± 0.13
GSM	1.5	17.05 ± 7.24	1.000	0.93 ± 0.62
	3	14.06 ± 5.68	1.000	0.27 ± 0.16
	6	26.03 ± 10.69	1.000	4.00 ± 3.23
		P=0.580		
Peripheral Blood				
Sham Control	0	3.32 ± 0.09		0.13 ± 0.13
GSM	1.5	3.07 ± 0.43	1.000	0.27 ± 0.16
	3	2.82 ± 0.52	1.000	0.13 ± 0.13
	6	3.86 ± 0.76	1.000	0.40 ± 0.16
		P=0.580		

^a Study was performed at ILS, Inc. The detailed protocol is presented by Recio *et al.* (2010) and OECD (2014). Groups of five rats per exposure group; exposure began *in utero* on gestation day 6. Statistical analysis runs Levene's test to determine if variances of dosed groups are equal ($P = 0.05$). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at $P \leq 0.025$ by Williams' or Dunn's test.

^d Percent hedgehogs = estimated as the number of comets with >90% tail DNA/150

^e No GSM radiofrequency radiation exposure

^f Dose-related trend; significant at $P \leq 0.025$ by linear regression or Jonckheere's test.

TABLE E9
Frequency of Micronuclei in Peripheral Blood Erythrocytes of Rats Following Exposure to CDMA- or GSM-Modulated Cell Phone RFR for 19 Weeks^a

Dose (W/kg)	Number of Rats with Erythrocytes Scored	Micronucleated		Micronucleated		PCEs ^b (%)	P Value ^c
		PCEs/ 1,000 PCEs ^b	P Value ^c	NCEs/ 1,000 NCEs ^b	P Value ^c		
Male							
Sham Control ^d	0	5	0.84 ± 0.10	0.33 ± 0.11		0.96 ± 0.05	
CDMA	1.5	5	0.56 ± 0.02	0.989	0.12 ± 0.02	1.000	0.84 ± 0.07 0.588
	3	5	0.55 ± 0.06	0.997	0.13 ± 0.02	1.000	0.89 ± 0.05 0.700
	6	5	0.43 ± 0.07	0.998	0.13 ± 0.05	1.000	1.00 ± 0.07 0.741
			P=0.999 ^e		P=0.970		P=0.389
GSM	1.5	5	0.61 ± 0.11	0.920	0.14 ± 0.04	1.000	1.04 ± 0.03 0.352
	3	5	0.60 ± 0.11	0.961	0.08 ± 0.02	1.000	1.01 ± 0.06 0.425
	6	5	0.49 ± 0.08	0.972	0.13 ± 0.02	1.000	1.09 ± 0.06 0.114
			P=0.985		P=0.911		P=0.123
Female							
Sham Control	0	5	0.62 ± 0.07	0.13 ± 0.04		0.66 ± 0.08	
CDMA	1.5	5	0.54 ± 0.08	1.000	0.18 ± 0.03	0.263	0.93 ± 0.17 0.337
	3	5	0.72 ± 0.12	0.778	0.16 ± 0.02	0.316	0.74 ± 0.12 0.406
	6	5	0.51 ± 0.04	1.000	0.19 ± 0.06	0.219	0.83 ± 0.05 0.297
			P=0.541		P=0.212		P=0.430
GSM	1.5	5	0.61 ± 0.10	0.519	0.20 ± 0.04	0.377	0.77 ± 0.07 0.376
	3	5	0.70 ± 0.08	0.495	0.11 ± 0.02	0.447	0.74 ± 0.09 0.455
	6	5	0.59 ± 0.07	0.525	0.13 ± 0.03	0.476	1.00 ± 0.03 0.010
			P=0.566		P=0.737		P=0.008

^a Study was performed at ILS, Inc. The detailed protocol is presented by Witt *et al.* (2008). Exposure began *in utero* on gestation day 6; NCE=normochromic erythrocyte; PCE=polychromatic erythrocyte. Statistical analysis runs Levene's test to determine if variances of dosed groups are equal ($P = 0.05$). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons. Note that the test for %PCE is a two-sided test, while the other two tests are one-sided.

^b Mean ± standard error

^c Pairwise comparison with the sham control group; exposed group values are significant at $P \leq 0.025$ by Williams' or Dunn's test.

^d No CDMA or GSM radiofrequency radiation exposure

^e Dose-related trend significant at $P \leq 0.025$ by linear regression or Jonckheere's test

APPENDIX F

CLINICAL PATHOLOGY RESULTS

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TABLE F1
Clinical Pathology Data for Rats at the 14-Week Interim Evaluation
in the 2-Year GSM-Modulated Cell Phone RFR Study^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Male				
Hematology				
Hematocrit (%)	52.0±0.3	51.7±0.2	51.8±0.6	52.4±0.4
Manual hematocrit (%)	50±0	50±0	50±1	51±1
Hemoglobin (g/dL)	16.5±0.1	16.5±0.1	16.6±0.2	16.7±0.2
Erythrocytes (10 ⁶ /µL)	8.88±0.07	8.92±0.05	9.15±0.10	8.94±0.08
Reticulocytes (10 ⁶ /µL)	270.4±3.9	259.4±7.3	258.2±7.0	265.6±9.5
Nucleated erythrocytes/100 leukocytes	0.00±0.00	0.00±0.00	0.00±0.00	0.10±0.10
Mean cell volume (fL)	58.5±0.4	58.0±0.5	56.7±0.5	58.6±0.5
Mean cell hemoglobin (pg)	18.6±0.1	18.5±0.1	18.2±0.2	18.8±0.2
Mean cell hemoglobin concentration (g/dL)	31.7±0.1	31.9±0.1	32.0±0.2	32.0±0.2
Platelets (10 ³ /µL)	894±41	896±34	954±57	896±24
Leukocytes (10 ³ /µL)	9.34±0.50	9.23±0.45	9.38±0.52	9.63±0.65
Segmented neutrophils (10 ³ /µL)	1.19±0.09	1.02±0.06	1.29±0.11	1.33±0.11
Lymphocytes (10 ³ /µL)	7.66±0.48	7.78±0.43	7.68±0.53	7.87±0.59
Monocytes (10 ³ /µL)	0.26±0.03	0.22±0.02	0.20±0.02	0.22±0.02
Basophils (10 ³ /µL)	0.05±0.01	0.05±0.00	0.05±0.01	0.05±0.01
Eosinophils (10 ³ /µL)	0.10±0.01	0.08±0.01	0.09±0.01	0.09±0.01
Large unstained cells (10 ³ /µL)	0.09±0.01	0.09±0.01	0.07±0.01	0.08±0.01
Clinical Chemistry				
Urea nitrogen (mg/dL)	18.1±0.6	17.6±0.4	17.7±0.4	18.1±0.5
Creatinine (mg/dL)	0.50±0.01**	0.54±0.02	0.54±0.01*	0.56±0.01**
Glucose (mg/dL)	135±6	134±4	127±2	128±3
Total protein (g/dL)	6.1±0.1	6.2±0.1	6.1±0.1	6.3±0.1
Albumin (g/dL)	3.6±0.0	3.7±0.1	3.7±0.0	3.7±0.0
Cholesterol (mg/dL)	92±2	86±3	87±3	88±4
Triglycerides (mg/dL)	88±6	88±10	98±6	96±6
Alanine aminotransferase (IU/L)	55±2	49±1	57±4	51±2
Alkaline phosphatase (IU/L)	284±12	316±19	315±19	318±16
Creatine kinase (IU/L)	277±83	467±161	284±68	664±266
Sorbitol dehydrogenase (IU/L)	33±1	33±1	34±1	33±1
Bile salt/acids (µmol/L)	27.3±3.8	20.5±3.1	33.0±4.8	29.6±4.8

TABLE F1
**Clinical Pathology Data for Rats at the 14-Week Interim Evaluation
in the 2-Year GSM-Modulated Cell Phone RFR Study**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Female				
Hematology				
Hematocrit (%)	48.1±0.5	49.1±0.5	48.6±0.4	48.7±0.6
Manual hematocrit (%)	47±0	49±0*	49±0**	49±0*
Hemoglobin (g/dL)	15.6±0.2	15.9±0.1	15.8±0.1	15.8±0.1
Erythrocytes (10 ⁶ /µL)	8.15±0.07	8.33±0.12	8.29±0.06	8.18±0.11
Reticulocytes (10 ⁶ /µL)	245.7±14.9	232.8±6.3	241.9±13.8	273.1±17.6
Nucleated erythrocytes/100 leukocytes	0.00±0.00	0.00±0.00	0.00±0.00	0.00±0.00
Mean cell volume (fL)	58.9±0.3	59.0±0.6	58.7±0.6	59.5±0.5
Mean cell hemoglobin (pg)	19.1±0.1	19.2±0.2	19.0±0.2	19.3±0.2
Mean cell hemoglobin concentration (g/dL)	32.4±0.1	32.5±0.2	32.5±0.2	32.3±0.2
Platelets (10 ³ /µL)	963±40	933±33	1,022±38	954±55
Leukocytes (10 ³ /µL)	8.80±0.50	7.90±0.67	6.86±0.51*	7.34±0.41
Segmented neutrophils (10 ³ /µL)	1.15±0.12	0.88±0.09	0.93±0.13	1.02±0.10
Lymphocytes (10 ³ /µL)	7.22±0.40	6.66±0.61	5.60±0.40**	6.00±0.37
Monocytes (10 ³ /µL)	0.19±0.02	0.15±0.01	0.16±0.02	0.14±0.01
Basophils (10 ³ /µL)	0.05±0.01	0.04±0.01	0.03±0.00*	0.03±0.00*
Eosinophils (10 ³ /µL)	0.11±0.02	0.10±0.01	0.08±0.01	0.09±0.01
Large unstained cells (10 ³ /µL)	0.09±0.01	0.07±0.01	0.06±0.01**	0.06±0.01**
Clinical Chemistry				
Urea nitrogen (mg/dL)	17.5±0.9	18.1±0.8	16.9±0.5	15.9±0.9
Creatinine (mg/dL)	0.57±0.03	0.56±0.03	0.56±0.02	0.63±0.02
Glucose (mg/dL)	151±9	146±9	141±5	146±6
Total protein (g/dL)	6.2±0.1	6.2±0.1	6.3±0.1	6.3±0.1
Albumin (g/dL)	3.8±0.0	3.8±0.0	3.9±0.0	3.9±0.1
Cholesterol (mg/dL)	78±4	72±3	69±3	64±3**
Triglycerides (mg/dL)	70±9	51±4	61±8	44±5*
Alanine aminotransferase (IU/L)	48±2	41±2	43±2	41±2
Alkaline phosphatase (IU/L)	277±24	273±16	265±13	226±12
Creatine kinase (IU/L)	209±35	295±54	218±27	275±68
Sorbitol dehydrogenase (IU/L)	31±4	28±5	28±4	32±4
Bile salt/acids (µmol/L)	33.7±6.1	22.8±3.7	28.0±4.5	31.5±6.3

* Significantly different ($P \leq 0.05$) from the sham control group by Dunn's or Shirley's test

** $P \leq 0.01$

^a Data are presented as mean ± standard error. Statistical analysis performed by Jonckheere (trend) and Shirley's or Dunn's (pairwise) tests.

TABLE F2
Clinical Pathology Data for Rats at the 14-Week Interim Evaluation
in the 2-Year CDMA-Modulated Cell Phone RFR Study^a

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Male				
Hematology				
Hematocrit (%)	52.0±0.3	51.9±0.4	51.4±0.5	51.9±0.8
Manual hematocrit (%)	50±0	50±1	50±1	50±1 ^b
Hemoglobin (g/dL)	16.5±0.1	16.6±0.1	16.6±0.1	16.7±0.3
Erythrocytes (10 ⁶ /µL)	8.88±0.07	8.92±0.07	8.91±0.10	8.82±0.13
Reticulocytes (10 ⁶ /µL)	270.4±3.9	266.8±5.7	257.4±5.6	274.8±15.1
Nucleated erythrocytes/100 leukocytes	0.00±0.00	0.00±0.00	0.00±0.00	0.00±0.00
Mean cell volume (fL)	58.5±0.4	58.2±0.4	57.7±0.5	58.9±0.5
Mean cell hemoglobin (pg)	18.6±0.1	18.7±0.1	18.6±0.1	18.9±0.1
Mean cell hemoglobin concentration (g/dL)	31.7±0.1	32.0±0.2	32.3±0.2	32.1±0.2
Platelets (10 ³ /µL)	894±41	839±26	911±22	926±35
Leukocytes (10 ³ /µL)	9.34±0.50	10.46±0.64	10.08±0.55	9.52±0.88
Segmented neutrophils (10 ³ /µL)	1.19±0.09	1.06±0.14	1.11±0.06	1.03±0.10
Lymphocytes (10 ³ /µL)	7.66±0.48	8.94±0.64	8.49±0.52	8.09±0.82
Monocytes (10 ³ /µL)	0.26±0.03	0.22±0.03	0.21±0.03	0.20±0.03
Basophils (10 ³ /µL)	0.05±0.01	0.06±0.01	0.05±0.00	0.05±0.01
Eosinophils (10 ³ /µL)	0.10±0.01	0.10±0.01	0.13±0.04	0.07±0.01
Large unstained cells (10 ³ /µL)	0.09±0.01	0.08±0.01	0.09±0.01	0.07±0.01
Clinical Chemistry				
Urea nitrogen (mg/dL)	18.1±0.6	17.2±0.4	17.5±0.4	18.5±0.6
Creatinine (mg/dL)	0.50±0.01**	0.50±0.01	0.54±0.02	0.59±0.02**
Glucose (mg/dL)	135±6	133±5	149±8	154±11
Total protein (g/dL)	6.1±0.1	6.1±0.0	6.3±0.1	6.2±0.1
Albumin (g/dL)	3.6±0.0*	3.6±0.0	3.6±0.0	3.7±0.0
Cholesterol (mg/dL)	92±2	89±2	92±3	88±3
Triglycerides (mg/dL)	88±6	84±7	87±5	72±5
Alanine aminotransferase (IU/L)	55±2	48±2	52±3	50±2
Alkaline phosphatase (IU/L)	284±12	287±16	300±15	309±26
Creatine kinase (IU/L)	277±83	265±31	387±109	309±64
Sorbitol dehydrogenase (IU/L)	33±1	31±1	33±2	35±1
Bile salt/acids (µmol/L)	27.3±3.8	36.3±4.5	33.7±5.0	30.1±5.4

TABLE F2
**Clinical Pathology Data for Rats at the 14-Week Interim Evaluation
in the 2-Year CDMA-Modulated Cell Phone RFR Study**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Female				
Hematology				
Hematocrit (%)	48.1±0.5	48.9±0.3	48.7±0.4	48.4±0.4
Manual hematocrit (%)	47±0	48±0	48±0*	48±0
Hemoglobin (g/dL)	15.6±0.2	15.8±0.1	15.8±0.1	15.7±0.1
Erythrocytes (10 ⁶ /µL)	8.15±0.07	8.13±0.08	8.23±0.03	8.17±0.08
Reticulocytes (10 ⁶ /µL)	245.7±14.9	282.5±10.3	268.0±19.8	267.9±14.4
Nucleated erythrocytes/100 leukocytes	0.00±0.00	0.00±0.00	0.00±0.00	0.00±0.00
Mean cell volume (fL)	58.9±0.3	60.2±0.4	59.2±0.5	59.3±0.7
Mean cell hemoglobin (pg)	19.1±0.1	19.5±0.2	19.2±0.1	19.3±0.2
Mean cell hemoglobin concentration (g/dL)	32.4±0.1	32.4±0.1	32.4±0.2	32.6±0.2
Platelets (10 ³ /µL)	963±40	978±36	989±38	983±33
Leukocytes (10 ³ /µL)	8.80±0.50	8.12±0.83	7.82±0.56	8.83±0.97
Segmented neutrophils (10 ³ /µL)	1.15±0.12	0.96±0.15	0.98±0.11	1.28±0.28
Lymphocytes (10 ³ /µL)	7.22±0.40	6.75±0.67	6.47±0.48	7.12±0.68
Monocytes (10 ³ /µL)	0.19±0.02	0.20±0.03	0.18±0.03	0.19±0.03
Basophils (10 ³ /µL)	0.05±0.01	0.04±0.01	0.03±0.00	0.04±0.01
Eosinophils (10 ³ /µL)	0.11±0.02	0.09±0.01	0.09±0.01	0.12±0.03
Large unstained cells (10 ³ /µL)	0.09±0.01	0.07±0.01	0.06±0.01*	0.08±0.01
Clinical Chemistry				
Urea nitrogen (mg/dL)	17.5±0.9	17.9±0.5	17.0±0.6	16.7±0.5
Creatinine (mg/dL)	0.57±0.03	0.58±0.03	0.56±0.01	0.59±0.02
Glucose (mg/dL)	151±9	145±9	140±5	141±7
Total protein (g/dL)	6.2±0.1	6.2±0.1	6.3±0.1	6.2±0.1
Albumin (g/dL)	3.8±0.0	3.8±0.0	3.9±0.0	3.8±0.0
Cholesterol (mg/dL)	78±4	71±2	73±3	71±2
Triglycerides (mg/dL)	70±9	61±5	54±3	70±8
Alanine aminotransferase (IU/L)	48±2	43±2	44±2	45±1
Alkaline phosphatase (IU/L)	277±24	248±16	257±18	252±17
Creatine kinase (IU/L)	209±35	212±36	217±41	418±97
Sorbitol dehydrogenase (IU/L)	31±4	31±4	28±4	34±5
Bile salt/acids (µmol/L)	33.7±6.1	36.8±5.1	32.7±5.8	29.9±5.6

* Significantly different ($P \leq 0.05$)

** $P \leq 0.01$

a Data are presented as mean ± standard error. Statistical analysis performed by Jonckheere (trend) and Shirley's or Dunn's (pairwise) tests.

b n=9

APPENDIX G

MEAN BODY TEMPERATURES, ORGAN WEIGHTS, AND ORGAN-WEIGHT-TO-BODY-WEIGHT RATIOS

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TABLE G1
Mean Body Temperatures for Rats by Litter in the 28-Day GSM-Modulated Cell Phone RFR Study^a

Day ^b	Sham Control		3 W/kg		6 W/kg		9 W/kg	
	Temperature (° C)	No. Litters Measured						
Male								
16	37.4 ± 0.2	4	37.0 ± 0.1	4	37.2 ± 0.2	4	37.2 ± 0.1	4
20	37.6 ± 0.1	4	37.0 ± 0.1**	4	37.2 ± 0.2	4	37.4 ± 0.1	4
27	37.3 ± 0.2	4	37.0 ± 0.1	4	37.2 ± 0.0	3	37.4 ± 0.1	4
16-27 ^c	37.5 ± 0.1	4	37.0 ± 0.0*	4	37.2 ± 0.1	4	37.4 ± 0.1	4
Female								
16	38.0 ± 0.3	4	37.0 ± 0.2**	4	37.0 ± 0.1*	4	37.4 ± 0.1	4
20	38.1 ± 0.2	4	37.6 ± 0.1	4	37.0 ± 0.1**	4	37.6 ± 0.1	4
27	37.9 ± 0.2	4	37.8 ± 0.3	4	37.3 ± 0.3	4	37.6 ± 0.0	4
16-27 ^c	38.0 ± 0.2*	4	37.4 ± 0.1	4	37.1 ± 0.1**	4	37.5 ± 0.0	4

* Significantly different ($P \leq 0.05$) from the sham control group by mixed effects models

** ($P \leq 0.01$)

a Temperatures are given as mean ± standard error. Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

b Postnatal day

c Average was calculated as the mean of the litter means of the individual animal averages over the time range.

TABLE G2
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats by Litter
in the 28-Day GSM-Modulated Cell Phone RFR Study^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	4	4	4	4
Males				
Necropsy body wt.	251 ± 6**	230 ± 2*	227 ± 2*	202 ± 7**
R. Adrenal gland				
Absolute	0.0253 ± 0.0012*	0.0236 ± 0.0009	0.0240 ± 0.0006	0.0198 ± 0.0021
Relative	0.10 ± 0.00	0.10 ± 0.00	0.11 ± 0.00	0.10 ± 0.01
Brain				
Absolute	1.76 ± 0.02	1.78 ± 0.02	1.75 ± 0.03	1.66 ± 0.05
Relative	7.02 ± 0.08**	7.73 ± 0.12*	7.73 ± 0.11*	8.25 ± 0.21**
Heart				
Absolute	1.02 ± 0.03**	0.94 ± 0.02	0.88 ± 0.03*	0.80 ± 0.04**
Relative	4.06 ± 0.08	4.07 ± 0.11	3.89 ± 0.11	3.97 ± 0.09
R. Kidney				
Absolute	1.10 ± 0.05**	1.04 ± 0.01	0.98 ± 0.03	0.84 ± 0.03**
Relative	4.36 ± 0.12	4.52 ± 0.02	4.30 ± 0.12	4.16 ± 0.06
Liver				
Absolute	12.05 ± 0.35**	11.04 ± 0.36	10.77 ± 0.16	9.75 ± 0.40**
Relative	47.93 ± 0.66	47.99 ± 1.17	47.47 ± 0.69	48.30 ± 1.18
Lung				
Absolute	2.23 ± 0.13*	1.93 ± 0.07	1.77 ± 0.09	1.81 ± 0.17
Relative	8.93 ± 0.69	8.40 ± 0.36	7.82 ± 0.43	9.00 ± 0.96
R. Testis				
Absolute	1.473 ± 0.056*	1.504 ± 0.038	1.455 ± 0.047	1.324 ± 0.028
Relative	5.86 ± 0.14*	6.54 ± 0.13*	6.42 ± 0.23*	6.56 ± 0.10*
Thymus				
Absolute	0.742 ± 0.060	0.520 ± 0.027*	0.672 ± 0.068	0.584 ± 0.017
Relative	2.95 ± 0.23	2.26 ± 0.10	2.96 ± 0.31	2.91 ± 0.14

TABLE G2
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats by Litter
in the 28-Day GSM-Modulated Cell Phone RFR Study

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	4	4	4	4
Females				
Necropsy body wt.	168 ± 3	161 ± 5	167 ± 5	155 ± 4
R. Adrenal gland				
Absolute	0.0299 ± 0.0027	0.0263 ± 0.0019	0.0280 ± 0.0004	0.0253 ± 0.0017
Relative	0.18 ± 0.01	0.16 ± 0.01	0.17 ± 0.01	0.16 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.64 ± 0.02	1.68 ± 0.03	1.61 ± 0.02
Relative	9.77 ± 0.22	10.27 ± 0.30	10.05 ± 0.22	10.36 ± 0.16
Heart				
Absolute	0.73 ± 0.02	0.69 ± 0.02	0.71 ± 0.02	0.68 ± 0.02
Relative	4.35 ± 0.09	4.31 ± 0.07	4.27 ± 0.04	4.37 ± 0.06
R. Kidney				
Absolute	0.76 ± 0.02	0.73 ± 0.02	0.75 ± 0.02	0.68 ± 0.01
Relative	4.49 ± 0.10	4.53 ± 0.10	4.47 ± 0.07	4.38 ± 0.04
Liver				
Absolute	7.65 ± 0.20	7.29 ± 0.28	7.74 ± 0.21	7.10 ± 0.21
Relative	45.53 ± 0.65	45.36 ± 0.78	46.28 ± 0.22	45.71 ± 0.88
Lung				
Absolute	1.52 ± 0.08	1.52 ± 0.03	1.50 ± 0.10	1.38 ± 0.04
Relative	9.03 ± 0.51	9.49 ± 0.51	8.93 ± 0.56	8.92 ± 0.43
Thymus				
Absolute	0.453 ± 0.059	0.344 ± 0.015	0.444 ± 0.032	0.405 ± 0.035
Relative	2.71 ± 0.38	2.13 ± 0.05	2.64 ± 0.15	2.61 ± 0.23

* Significantly different ($P \leq 0.05$) from the sham control group by mixed models

** $P \leq 0.01$

a Organ weights (absolute weights) and body weights are given in grams; means are calculated as means of litter means; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

TABLE G3
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats
in the 28-Day GSM-Modulated Cell Phone RFR Study^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	249 ± 3**	231 ± 2*	227 ± 2*	205 ± 6**
R. Adrenal gland				
Absolute	0.0247 ± 0.0011*	0.0237 ± 0.0012	0.0238 ± 0.0009	0.0206 ± 0.0013
Relative	0.10 ± 0.00	0.10 ± 0.00	0.11 ± 0.00	0.10 ± 0.01
Brain				
Absolute	1.75 ± 0.01	1.77 ± 0.02	1.75 ± 0.02	1.69 ± 0.03
Relative	7.05 ± 0.07**	7.68 ± 0.12*	7.73 ± 0.08*	8.29 ± 0.17**
Heart				
Absolute	1.02 ± 0.02**	0.94 ± 0.02	0.90 ± 0.02*	0.82 ± 0.03**
Relative	4.09 ± 0.06	4.06 ± 0.08	3.95 ± 0.06	4.02 ± 0.08
R. Kidney				
Absolute	1.08 ± 0.03**	1.04 ± 0.01	0.98 ± 0.02	0.85 ± 0.03**
Relative	4.32 ± 0.09	4.51 ± 0.04	4.33 ± 0.08	4.17 ± 0.05
Liver				
Absolute	11.85 ± 0.22**	11.25 ± 0.22	10.86 ± 0.18	9.81 ± 0.30**
Relative	47.66 ± 0.63	48.61 ± 0.66	47.84 ± 0.56	47.98 ± 0.77
Lung				
Absolute	2.24 ± 0.12*	1.90 ± 0.09	1.81 ± 0.08	1.87 ± 0.12
Relative	9.05 ± 0.56	8.00 ± 0.37	8.00 ± 0.37	9.25 ± 0.70
R. Testis				
Absolute	1.464 ± 0.036*	1.516 ± 0.039	1.482 ± 0.029	1.330 ± 0.041
Relative	5.88 ± 0.11*	6.55 ± 0.14*	6.54 ± 0.14*	6.50 ± 0.09*
Thymus				
Absolute	0.757 ± 0.041	0.536 ± 0.019*	0.637 ± 0.036	0.576 ± 0.013
Relative	3.04 ± 0.15	2.31 ± 0.07	2.81 ± 0.16	2.83 ± 0.10

TABLE G3
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats
in the 28-Day GSM-Modulated Cell Phone RFR Study

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	10	10	10	10
Females				
Necropsy body wt.	167 ± 2	163 ± 4	168 ± 3	156 ± 3
R. Adrenal gland				
Absolute	0.0285 ± 0.0019	0.0266 ± 0.0016	0.0280 ± 0.0012	0.0259 ± 0.0017
Relative	0.17 ± 0.01	0.16 ± 0.01	0.17 ± 0.01	0.17 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.64 ± 0.02	1.67 ± 0.02	1.60 ± 0.02
Relative	9.86 ± 0.15	10.13 ± 0.23	9.96 ± 0.14	10.31 ± 0.14
Heart				
Absolute	0.72 ± 0.01	0.70 ± 0.02	0.72 ± 0.02	0.68 ± 0.01
Relative	4.34 ± 0.07	4.26 ± 0.05	4.25 ± 0.05	4.34 ± 0.05
R. Kidney				
Absolute	0.74 ± 0.02	0.73 ± 0.02	0.74 ± 0.02	0.68 ± 0.01
Relative	4.46 ± 0.06	4.48 ± 0.07	4.43 ± 0.05	4.38 ± 0.07
Liver				
Absolute	7.55 ± 0.14	7.44 ± 0.24	7.80 ± 0.19	7.04 ± 0.14
Relative	45.35 ± 0.44	45.56 ± 0.64	46.38 ± 0.40	45.18 ± 0.42
Lung				
Absolute	1.52 ± 0.07	1.50 ± 0.05	1.54 ± 0.09	1.36 ± 0.04
Relative	9.11 ± 0.40	9.22 ± 0.37	9.16 ± 0.44	8.78 ± 0.38
Thymus				
Absolute	0.474 ± 0.036	0.349 ± 0.017	0.444 ± 0.025	0.386 ± 0.021
Relative	2.86 ± 0.23	2.13 ± 0.07	2.63 ± 0.12	2.48 ± 0.13

* Significantly different ($P \leq 0.05$) from the sham control group by mixed models

** $P \leq 0.01$

a Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

TABLE G4
**Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats at the 14-Week Interim Evaluation
in the 2-Year GSM-Modulated Cell Phone RFR Study^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	444 ± 9*	426 ± 10	430 ± 7	410 ± 7*
Brain				
Absolute	1.92 ± 0.02	1.94 ± 0.02	1.94 ± 0.02	1.88 ± 0.02
Relative	4.34 ± 0.08	4.58 ± 0.11	4.53 ± 0.06	4.59 ± 0.06
R. Epididymis				
Absolute	0.6423 ± 0.0278	0.6370 ± 0.0191	0.6245 ± 0.0173	0.6332 ± 0.0312
Relative	1.45 ± 0.06	1.50 ± 0.05	1.46 ± 0.06	1.54 ± 0.06
L. Epididymis				
Absolute	0.6404 ± 0.0208	0.6430 ± 0.0178	0.6219 ± 0.0168	0.6435 ± 0.0290
Relative	1.44 ± 0.05	1.51 ± 0.04	1.45 ± 0.05	1.56 ± 0.05
Heart				
Absolute	1.52 ± 0.03	1.46 ± 0.03	1.42 ± 0.04	1.44 ± 0.03
Relative	3.42 ± 0.06	3.42 ± 0.04	3.30 ± 0.05	3.51 ± 0.04
R. Kidney				
Absolute	1.59 ± 0.04*	1.43 ± 0.06*	1.50 ± 0.02	1.41 ± 0.04*
Relative	3.58 ± 0.09	3.37 ± 0.11	3.50 ± 0.06	3.44 ± 0.07
L. Kidney				
Absolute	1.58 ± 0.04**	1.43 ± 0.05*	1.47 ± 0.02*	1.35 ± 0.05**
Relative	3.57 ± 0.09*	3.34 ± 0.09	3.42 ± 0.05	3.28 ± 0.09*
Liver				
Absolute	16.49 ± 0.31	14.97 ± 0.46	15.23 ± 0.35	14.85 ± 0.59*
Relative	37.17 ± 0.61	35.12 ± 0.63	35.47 ± 0.73	36.12 ± 0.97
Lung				
Absolute	2.20 ± 0.06	2.08 ± 0.06	2.03 ± 0.06	2.00 ± 0.08
Relative	4.96 ± 0.15	4.89 ± 0.13	4.73 ± 0.16	4.88 ± 0.17
R. Testis				
Absolute	2.071 ± 0.051	1.982 ± 0.049	2.055 ± 0.041	1.930 ± 0.056
Relative	4.68 ± 0.16	4.66 ± 0.11	4.80 ± 0.15	4.70 ± 0.10
L. Testis				
Absolute	2.083 ± 0.059	2.028 ± 0.051	2.049 ± 0.039	1.954 ± 0.055
Relative	4.71 ± 0.17	4.77 ± 0.11	4.79 ± 0.15	4.76 ± 0.08
Thymus				
Absolute	0.349 ± 0.023	0.361 ± 0.026	0.372 ± 0.024	0.360 ± 0.017
Relative	0.79 ± 0.05	0.84 ± 0.04	0.86 ± 0.05	0.87 ± 0.03

TABLE G4
**Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats at the 14-Week Interim Evaluation
in the 2-Year GSM-Modulated Cell Phone RFR Study**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Females				
Necropsy body wt.	274 ± 8*	249 ± 6*	265 ± 8	244 ± 5**
Brain				
Absolute	1.79 ± 0.02	1.80 ± 0.03	1.81 ± 0.02	1.78 ± 0.02
Relative	6.58 ± 0.18*	7.28 ± 0.14**	6.90 ± 0.18	7.33 ± 0.11**
Heart				
Absolute	1.02 ± 0.03	0.95 ± 0.02	0.98 ± 0.03	0.94 ± 0.03
Relative	3.74 ± 0.09	3.84 ± 0.08	3.70 ± 0.06	3.87 ± 0.04
R. Kidney				
Absolute	0.97 ± 0.03**	0.91 ± 0.02	0.89 ± 0.02*	0.85 ± 0.02**
Relative	3.54 ± 0.08	3.65 ± 0.05	3.37 ± 0.10	3.50 ± 0.07
L. Kidney				
Absolute	0.97 ± 0.04**	0.87 ± 0.02*	0.88 ± 0.02*	0.83 ± 0.02**
Relative	3.53 ± 0.11	3.51 ± 0.04	3.33 ± 0.08	3.41 ± 0.08
Liver				
Absolute	10.07 ± 0.56*	8.41 ± 0.25*	9.36 ± 0.40	8.03 ± 0.22**
Relative	36.55 ± 1.16**	33.85 ± 0.61	35.27 ± 0.63	32.94 ± 0.46**
Lung				
Absolute	1.85 ± 0.05*	1.67 ± 0.05	1.90 ± 0.07	1.56 ± 0.03**
Relative	6.79 ± 0.24	6.74 ± 0.18	7.22 ± 0.29	6.42 ± 0.11
R. Ovary				
Absolute	0.0598 ± 0.0042	0.0581 ± 0.0047	0.0569 ± 0.0038	0.0565 ± 0.0025
Relative	0.22 ± 0.02	0.23 ± 0.02	0.21 ± 0.01	0.23 ± 0.01
L. Ovary				
Absolute	0.0511 ± 0.0031	0.0496 ± 0.0040	0.0532 ± 0.0045	0.0521 ± 0.0035
Relative	0.19 ± 0.01	0.20 ± 0.01	0.20 ± 0.01	0.21 ± 0.01
Thymus				
Absolute	0.299 ± 0.016*	0.244 ± 0.013*	0.274 ± 0.020	0.234 ± 0.013*
Relative	1.10 ± 0.06	0.98 ± 0.05	1.03 ± 0.07	0.96 ± 0.04

* Significantly different ($P \leq 0.05$)

** $P \leq 0.01$

a Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

TABLE G5
Mean Body Temperatures for Rats by Litter in the 28-Day CDMA-Modulated Cell Phone RFR Study^a

Day ^b	Sham Control		3 W/kg		6 W/kg		9 W/kg	
	Temperature (° C)	No. Litters Measured						
Male								
16	37.4 ± 0.2	4	37.0 ± 0.0	4	37.1 ± 0.0	4	37.2 ± 0.1	4
20	37.6 ± 0.1	4	37.2 ± 0.1	4	37.1 ± 0.1	4	37.5 ± 0.2	4
27	37.3 ± 0.2	4	36.8 ± 0.1	4 ^d	37.0 ± 0.1	4	37.4 ± 0.1	4
16-27 ^c	37.5 ± 0.1	4	37.0 ± 0.0	4	37.1 ± 0.0	4	37.4 ± 0.1	4
Female								
16	38.0 ± 0.3	4	37.2 ± 0.2*	3	37.4 ± 0.1	4	37.5 ± 0.1	4
20	38.1 ± 0.2*	4	38.1 ± 0.2	4	37.5 ± 0.2	4	37.7 ± 0.2	4
27	37.9 ± 0.2	4	37.1 ± 0.1	4	37.9 ± 0.3	4	37.9 ± 0.2	4
16-27 ^c	38.0 ± 0.2	4	37.5 ± 0.1	4	37.6 ± 0.2	4	37.7 ± 0.1	4

* Significantly different ($P \leq 0.05$) from the sham control group by mixed effects models with the Dam ID as the random effect

^a Temperatures are given as mean ± standard error. Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

^b Postnatal day

^c Average was calculated as the mean of the litter means of the individual animal averages over the time range.

^d One animal's value was excluded from the litter means because it was an outlier.

TABLE G6
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats by Litter
in the 28-Day CDMA-Modulated Cell Phone RFR Study^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	4	4	4	4
Males				
Necropsy body wt.	251 ± 6**	232 ± 4	234 ± 7	216 ± 3**
R. Adrenal gland				
Absolute	0.0253 ± 0.0012	0.0242 ± 0.0021	0.0249 ± 0.0019	0.0228 ± 0.0008
Relative	0.10 ± 0.00	0.10 ± 0.01	0.11 ± 0.00	0.11 ± 0.00
Brain				
Absolute	1.76 ± 0.02	1.75 ± 0.02	1.74 ± 0.02	1.72 ± 0.02
Relative	7.02 ± 0.08*	7.53 ± 0.13*	7.47 ± 0.19	7.98 ± 0.06**
Heart				
Absolute	1.02 ± 0.03*	0.92 ± 0.01	0.95 ± 0.05	0.89 ± 0.04
Relative	4.06 ± 0.08	3.95 ± 0.06	4.05 ± 0.12	4.12 ± 0.13
R. Kidney				
Absolute	1.10 ± 0.05**	1.04 ± 0.02	1.00 ± 0.03	0.91 ± 0.04*
Relative	4.36 ± 0.12	4.46 ± 0.04	4.27 ± 0.09	4.20 ± 0.13
Liver				
Absolute	12.05 ± 0.35**	10.97 ± 0.22	11.25 ± 0.38	10.23 ± 0.17**
Relative	47.93 ± 0.66	47.20 ± 0.99	48.11 ± 0.68	47.49 ± 0.74
Lung				
Absolute	2.23 ± 0.13	1.98 ± 0.16	1.94 ± 0.09	1.96 ± 0.04
Relative	8.93 ± 0.69	8.53 ± 0.76	8.30 ± 0.49	9.13 ± 0.26
R. Testis				
Absolute	1.473 ± 0.056	1.444 ± 0.024	1.456 ± 0.050	1.410 ± 0.038
Relative	5.86 ± 0.14**	6.22 ± 0.08	6.23 ± 0.04	6.54 ± 0.08**
Thymus				
Absolute	0.742 ± 0.060**	0.639 ± 0.011	0.646 ± 0.023	0.553 ± 0.025**
Relative	2.95 ± 0.23	2.75 ± 0.04	2.77 ± 0.11	2.57 ± 0.12

TABLE G6
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats by Litter
in the 28-Day CDMA-Modulated Cell Phone RFR Study

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	4	4	4	4
Females				
Necropsy body wt.	168 ± 3	165 ± 5	169 ± 8	161 ± 4
R. Adrenal gland				
Absolute	0.0299 ± 0.0027*	0.0250 ± 0.0003	0.0268 ± 0.0009	0.0242 ± 0.0016
Relative	0.18 ± 0.01	0.15 ± 0.00	0.16 ± 0.01	0.15 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.68 ± 0.02	1.63 ± 0.01	1.63 ± 0.03
Relative	9.77 ± 0.22	10.23 ± 0.21	9.76 ± 0.42	10.14 ± 0.14
Heart				
Absolute	0.73 ± 0.02	0.71 ± 0.02	0.72 ± 0.03	0.70 ± 0.01
Relative	4.35 ± 0.09	4.34 ± 0.07	4.29 ± 0.10	4.33 ± 0.08
R. Kidney				
Absolute	0.76 ± 0.02*	0.75 ± 0.02	0.73 ± 0.03	0.69 ± 0.02*
Relative	4.49 ± 0.10*	4.53 ± 0.03	4.32 ± 0.07	4.26 ± 0.08
Liver				
Absolute	7.65 ± 0.20	7.50 ± 0.16	7.87 ± 0.42	7.25 ± 0.07
Relative	45.53 ± 0.65	45.64 ± 1.19	46.54 ± 0.53	45.11 ± 0.70
Lung				
Absolute	1.52 ± 0.08	1.48 ± 0.06	1.46 ± 0.06	1.41 ± 0.04
Relative	9.03 ± 0.51	8.98 ± 0.45	8.78 ± 0.73	8.76 ± 0.34
Thymus				
Absolute	0.453 ± 0.059*	0.444 ± 0.030	0.388 ± 0.024	0.380 ± 0.024
Relative	2.71 ± 0.38	2.69 ± 0.10	2.31 ± 0.17	2.36 ± 0.09

* Significantly different ($P \leq 0.05$) from the sham control group

** $P \leq 0.01$

a Organ weights (absolute weights) and body weights are given in grams as means of litter means; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

TABLE G7
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats
in the 28-Day CDMA-Modulated Cell Phone RFR Study^a

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	249 ± 3**	231 ± 3	236 ± 6	215 ± 3**
R. Adrenal gland				
Absolute	0.0247 ± 0.0011	0.0254 ± 0.0016	0.0253 ± 0.0013	0.0223 ± 0.0007
Relative	0.10 ± 0.00	0.11 ± 0.01	0.11 ± 0.00	0.10 ± 0.00
Brain				
Absolute	1.75 ± 0.01	1.75 ± 0.01	1.75 ± 0.01	1.72 ± 0.02
Relative	7.05 ± 0.07**	7.59 ± 0.11*	7.43 ± 0.14	8.01 ± 0.07**
Heart				
Absolute	1.02 ± 0.02*	0.91 ± 0.01	0.97 ± 0.04	0.90 ± 0.02
Relative	4.09 ± 0.06	3.95 ± 0.05	4.08 ± 0.09	4.16 ± 0.09
R. Kidney				
Absolute	1.08 ± 0.03**	1.03 ± 0.02	1.00 ± 0.03	0.91 ± 0.03*
Relative	4.32 ± 0.09	4.46 ± 0.05	4.22 ± 0.07	4.24 ± 0.08
Liver				
Absolute	11.85 ± 0.22**	11.05 ± 0.21	11.38 ± 0.33	10.29 ± 0.17**
Relative	47.66 ± 0.63	47.80 ± 0.58	48.13 ± 0.59	47.88 ± 0.47
Lung				
Absolute	2.24 ± 0.12	1.92 ± 0.10	1.91 ± 0.09	1.95 ± 0.08
Relative	9.05 ± 0.56	8.34 ± 0.49	8.07 ± 0.35	9.10 ± 0.42
R. Testis				
Absolute	1.464 ± 0.036	1.438 ± 0.023	1.473 ± 0.036	1.407 ± 0.034
Relative	5.88 ± 0.11**	6.22 ± 0.08	6.23 ± 0.06	6.54 ± 0.12**
Thymus				
Absolute	0.757 ± 0.041**	0.639 ± 0.017	0.658 ± 0.026	0.568 ± 0.022**
Relative	3.04 ± 0.15	2.77 ± 0.06	2.79 ± 0.10	2.64 ± 0.11

TABLE G7
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats
in the 28-Day CDMA-Modulated Cell Phone RFR Study

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	10	10	10	10
Females				
Necropsy body wt.	167 ± 2	163 ± 4	172 ± 6	159 ± 3
R. Adrenal gland				
Absolute	0.0285 ± 0.0019*	0.0250 ± 0.0007	0.0265 ± 0.0012	0.0236 ± 0.0011
Relative	0.17 ± 0.01	0.15 ± 0.00	0.16 ± 0.01	0.15 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.67 ± 0.01	1.64 ± 0.01	1.61 ± 0.01
Relative	9.86 ± 0.15	10.33 ± 0.19	9.62 ± 0.33	10.15 ± 0.16
Heart				
Absolute	0.72 ± 0.01	0.71 ± 0.02	0.74 ± 0.02	0.69 ± 0.01
Relative	4.34 ± 0.07	4.34 ± 0.06	4.29 ± 0.08	4.33 ± 0.06
R. Kidney				
Absolute	0.74 ± 0.02*	0.73 ± 0.02	0.74 ± 0.02	0.67 ± 0.01*
Relative	4.46 ± 0.06*	4.51 ± 0.03	4.32 ± 0.06	4.23 ± 0.06
Liver				
Absolute	7.55 ± 0.14	7.52 ± 0.16	8.04 ± 0.32	7.23 ± 0.17
Relative	45.35 ± 0.44	46.25 ± 0.64	46.77 ± 0.55	45.45 ± 0.74
Lung				
Absolute	1.52 ± 0.07	1.50 ± 0.07	1.44 ± 0.04	1.41 ± 0.06
Relative	9.11 ± 0.40	9.23 ± 0.37	8.45 ± 0.41	8.85 ± 0.37
Thymus				
Absolute	0.474 ± 0.036*	0.432 ± 0.021	0.400 ± 0.025	0.369 ± 0.017
Relative	2.86 ± 0.23	2.65 ± 0.08	2.35 ± 0.16	2.32 ± 0.10

* Significantly different ($P \leq 0.05$)

** $P \leq 0.01$

a Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

TABLE G8
**Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats at the 14-Week Interim Evaluation
in the 2-Year CDMA-Modulated Cell Phone RFR Study^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	444 ± 9**	440 ± 8	435 ± 4	411 ± 8**
Brain				
Absolute	1.92 ± 0.02	1.96 ± 0.02	1.97 ± 0.04	1.92 ± 0.02
Relative	4.34 ± 0.08**	4.47 ± 0.08	4.53 ± 0.10	4.67 ± 0.08*
R. Epididymis				
Absolute	0.6423 ± 0.0278	0.6298 ± 0.0256	0.6370 ± 0.0162 ^b	0.5817 ± 0.0353
Relative	1.45 ± 0.06	1.44 ± 0.07	1.46 ± 0.04 ^b	1.42 ± 0.09
L. Epididymis				
Absolute	0.6404 ± 0.0208	0.6673 ± 0.0137	0.6491 ± 0.0155	0.5866 ± 0.0428
Relative	1.44 ± 0.05	1.52 ± 0.05	1.49 ± 0.03	1.43 ± 0.11
Heart				
Absolute	1.52 ± 0.03	1.52 ± 0.04	1.51 ± 0.03	1.43 ± 0.04
Relative	3.42 ± 0.06	3.46 ± 0.06	3.47 ± 0.06	3.49 ± 0.07
R. Kidney				
Absolute	1.59 ± 0.04**	1.51 ± 0.04	1.51 ± 0.05	1.38 ± 0.05**
Relative	3.58 ± 0.09	3.45 ± 0.09	3.47 ± 0.11	3.36 ± 0.13
L. Kidney				
Absolute	1.58 ± 0.04**	1.53 ± 0.04	1.49 ± 0.04	1.35 ± 0.04**
Relative	3.57 ± 0.09	3.49 ± 0.06	3.43 ± 0.09	3.29 ± 0.12
Liver				
Absolute	16.49 ± 0.31**	15.66 ± 0.39	15.57 ± 0.37	14.13 ± 0.36**
Relative	37.17 ± 0.61**	35.64 ± 0.57	35.79 ± 0.64	34.35 ± 0.45**
Lung				
Absolute	2.20 ± 0.06	2.06 ± 0.07	2.29 ± 0.12	2.09 ± 0.09
Relative	4.96 ± 0.15	4.70 ± 0.18	5.26 ± 0.25	5.12 ± 0.30
R. Testis				
Absolute	2.071 ± 0.051	2.104 ± 0.045	2.087 ± 0.044	1.784 ± 0.152
Relative	4.68 ± 0.16	4.80 ± 0.14	4.81 ± 0.12	4.37 ± 0.38
L. Testis				
Absolute	2.083 ± 0.059	2.116 ± 0.055	2.102 ± 0.036	1.836 ± 0.160
Relative	4.71 ± 0.17	4.83 ± 0.14	4.84 ± 0.10	4.50 ± 0.40
Thymus				
Absolute	0.349 ± 0.023	0.368 ± 0.018	0.387 ± 0.026	0.364 ± 0.031
Relative	0.79 ± 0.05	0.84 ± 0.04	0.89 ± 0.06	0.88 ± 0.07

TABLE G8
**Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats at the 14-Week Interim Evaluation
in the 2-Year CDMA-Modulated Cell Phone RFR Study**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Females				
Necropsy body wt.	274 ± 8	264 ± 5	264 ± 6	252 ± 4*
Brain				
Absolute	1.79 ± 0.02	1.79 ± 0.02	1.85 ± 0.02	1.77 ± 0.02
Relative	6.58 ± 0.18	6.81 ± 0.13	7.02 ± 0.15	7.05 ± 0.09
Heart				
Absolute	1.02 ± 0.03	0.97 ± 0.01	0.98 ± 0.02	0.94 ± 0.02*
Relative	3.74 ± 0.09	3.67 ± 0.05	3.73 ± 0.07	3.73 ± 0.08
R. Kidney				
Absolute	0.97 ± 0.03	0.91 ± 0.02	0.91 ± 0.02	0.84 ± 0.02**
Relative	3.54 ± 0.08	3.45 ± 0.07	3.47 ± 0.06	3.35 ± 0.08
L. Kidney				
Absolute	0.97 ± 0.04	0.87 ± 0.02*	0.89 ± 0.02*	0.82 ± 0.02**
Relative	3.53 ± 0.11	3.28 ± 0.05	3.38 ± 0.06	3.26 ± 0.09
Liver				
Absolute	10.07 ± 0.56	8.92 ± 0.28	9.19 ± 0.28	8.76 ± 0.20*
Relative	36.55 ± 1.16	33.72 ± 0.59	34.80 ± 0.64	34.82 ± 0.76
Lung				
Absolute	1.85 ± 0.05	1.80 ± 0.07	1.75 ± 0.08	1.72 ± 0.06
Relative	6.79 ± 0.24	6.83 ± 0.26	6.68 ± 0.35	6.84 ± 0.25
R. Ovary				
Absolute	0.0598 ± 0.0042	0.0578 ± 0.0039	0.0587 ± 0.0029	0.0566 ± 0.0053
Relative	0.22 ± 0.02	0.22 ± 0.01	0.22 ± 0.01	0.22 ± 0.02
L. Ovary				
Absolute	0.0511 ± 0.0031	0.0521 ± 0.0030	0.0547 ± 0.0035	0.0495 ± 0.0055
Relative	0.19 ± 0.01	0.20 ± 0.01	0.21 ± 0.01	0.20 ± 0.02
Thymus				
Absolute	0.299 ± 0.016	0.282 ± 0.008	0.274 ± 0.015	0.278 ± 0.015
Relative	1.10 ± 0.06	1.07 ± 0.03	1.04 ± 0.05	1.11 ± 0.06

* Significantly different ($P \leq 0.05$)

** $P \leq 0.01$

a Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (Relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. (statistical significance in the Sham Control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant trend test).

b n=9

APPENDIX H

REPRODUCTIVE TISSUE EVALUATIONS

TABLE H1	Summary of Reproductive Tissue Evaluations for Male Rats Exposed to GSM-Modulated Cell Phone RFR for 14 Weeks.....	H-2
TABLE H2	Summary of Reproductive Tissue Evaluations for Male Rats Exposed to CDMA-Modulated Cell Phone RFR for 14 Weeks	H-2

TABLE H1
**Summary of Reproductive Tissue Evaluations for Male Rats
 Exposed to GSM-Modulated Cell Phone RFR for 14 Weeks^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Weights (g)				
Necropsy body wt	444 ± 9▲	426 ± 10	430 ± 7	410 ± 7*
L. Cauda epididymis	0.266 ± 0.011	0.271 ± 0.008	0.257 ± 0.009	0.269 ± 0.011
L. Epididymis	0.640 ± 0.021	0.643 ± 0.018	0.622 ± 0.017	0.643 ± 0.029
L. Testis	2.083 ± 0.059	2.028 ± 0.051	2.049 ± 0.039	1.954 ± 0.055
Spermatid measurements				
Spermatid heads (10 ⁶ /testis)	305.1 ± 11.2	280.3 ± 8.9	263.4 ± 12.3	297.4 ± 12.5
Spermatid heads (10 ⁶ /g testis)	147.3 ± 6.0	138.6 ± 4.2	128.5 ± 5.2	152.7 ± 6.8
Epididymal spermatozoal measurements				
Sperm motility (%)	91.5 ± 1.4	74.0 ± 8.0	91.5 ± 1.4	89.4 ± 2.2
Sperm (10 ⁶ /cauda epididymis)	247.7 ± 68.9	285.5 ± 71.3	283.7 ± 57.5	220.3 ± 34.9
Sperm (10 ⁶ /g cauda epididymis)	909.3 ± 243.5	1,081.1 ± 282.6	1,085.0 ± 210.7	834.8 ± 139.5

▲ Significant trend (P≤0.05) by Jonckheere's test

* Significantly different (P≤0.05) from the sham control group by Williams' or Dunnett's test

^a Data are presented as mean ± standard error. Pairwise differences from the sham control group are tested for significance by Williams' or Dunnett's test (tissue weights) or by Shirley's or Dunn's test (spermatid and epididymal spermatozoal measurements).

TABLE H2
**Summary of Reproductive Tissue Evaluations for Male Rats
 Exposed to CDMA-Modulated Cell Phone RFR for 14 Weeks^a**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Weights (g)				
Necropsy body wt	444 ± 9▲▲	440 ± 8	435 ± 4	411 ± 8**
L. Cauda epididymis	0.266 ± 0.011	0.284 ± 0.008	0.274 ± 0.009	0.249 ± 0.017
L. Epididymis	0.640 ± 0.021	0.667 ± 0.014	0.649 ± 0.015	0.587 ± 0.043
L. Testis	2.083 ± 0.059	2.116 ± 0.055	2.102 ± 0.036	1.836 ± 0.160
Spermatid measurements				
Spermatid heads (10 ⁶ /testis)	305.1 ± 11.2	281.0 ± 12.5	280.7 ± 10.1	253.7 ± 30.1
Spermatid heads (10 ⁶ /g testis)	147.3 ± 6.0	133.9 ± 7.6	133.6 ± 4.5	129.5 ± 12.9
Epididymal spermatozoal measurements				
Sperm motility (%)	91.5 ± 1.4	90.9 ± 1.0	88.7 ± 4.0	81.9 ± 9.2
Sperm (10 ⁶ /cauda epididymis)	247.7 ± 68.9	206.0 ± 36.4	243.9 ± 36.4	201.8 ± 29.7
Sperm (10 ⁶ /g cauda epididymis)	909.3 ± 243.5	742.9 ± 140.7	906.2 ± 144.4	775.8 ± 106.6

▲▲ Significant trend (P≤0.01) by Jonckheere's test

** Significantly different (P≤0.01) from the sham control group by Williams' test

^a Data are presented as mean ± standard error. Pairwise differences from the sham control group are tested for significance by Williams' or Dunnett's test (tissue weights) or by Shirley's or Dunn's test (spermatid and epididymal spermatozoal measurements).

APPENDIX I

GSM- AND CDMA-MODULATED CELL PHONE RFR EXPOSURE DATA

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OVERVIEW

Exposure data include SAR (W/kg) (Tables I1 and I5), chamber field strength (V/m) (Tables I2 and I6), and E- and H-field measurements (V/m) (Tables I3, I4, I7, and I8). For the medium- and high-dose GSM chambers, where a second E-field probe was used, the H-field measurements were converted from E-field measurements (E-field divided by 377). Fields were measured continuously throughout the studies and measurements automatically recorded approximately every 20 seconds. For every 20 second interval, the SAR was calculated based on the average H- and/or E-field data. The data presented for each exposure parameter include the mean and standard deviation [expressed in decibels (dB), W/kg or V/m], the total number of measurements recorded during the identified period of exposure (>50,000 calculated SAR per month, and more than 1.4 million over the course of the 2-year studies); the lowest (minimum) and highest measurement (maximum) recorded during the given exposure period; the number of measurements that were within the acceptable range; and the ratio of all measurements within range. The data reported for SAR also include the range of animal body weights (g) over the indicated time period of exposure, and the selected target SAR (W/kg) for each group. The data reported for field strengths (chamber, E-field, and H-field) include the target range of the field required to maintain appropriate SAR exposures. The minimum and maximum exposure values reported represent a single recorded measurement over the 2-year exposure period. The SAR and chamber-field in the sham and exposure chambers were within the target ranges (defined as ± 2 dB) for >99.97% of recorded measurements over the course of the 2-year study; $\geq 99.25\%$ of E- and H-field exposures in the sham and exposure chambers were within the target ranges.

The dB is a mathematical transformation of a number or numerical ratio using base 10 logarithms. Multiplication of ratios is transformed into addition of dBs; raising a number to a power is transformed into multiplication of dBs.

In general, $\text{dB}(\text{power}) = 10 \times \log(R)$ and $\text{dB}(\text{field}) = 20 \times \log(R)$. The formulas differ by a factor of two because power or SAR varies as the square of the fields. For SAR (in W/kg), the decibel formula is calculated as:

$$\text{SAR(dB)} = 10 \times \log(\text{SAR}_M/\text{SAR}_T)$$

where SAR_M is the measured value and SAR_T is the target value, and

$$-2 \text{ dB} = 10 \times \log(\text{SAR}_L/\text{SAR}_T), \text{ where } \text{SAR}_L \text{ (low)} = \text{SAR}_T \times 10^{-0.2}$$

$$+2 \text{ dB} = 10 \times \log(\text{SAR}_H/\text{SAR}_T), \text{ where } \text{SAR}_H \text{ (high)} = \text{SAR}_T \times 10^{0.2}$$

On this basis, the ± 2 dB range specified by the NTP translates to the following ranges for each SAR used in the 2-year study:

Target SAR (W/kg)	Acceptable SAR Range (W/kg; ± 2 dB)
1.5	0.95 to 2.38
3	1.89 to 4.75
6	3.79 to 9.51

TABLE II
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – SAR^a

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 8 to 31, 2012								
<i>Male</i>								
Ch06 GSM High	238.7 to 355.6	6.00	5.96	0.30/0.07	2.553	11.257	42880/42893	1.000
Ch05 GSM Med	238.7 to 355.5	3.00	2.99	0.25/0.06	1.347	4.987	42890/42893	1.000
Ch09 GSM Low	239.9 to 358.9	1.50	1.49	0.23/0.05	0.654	2.544	42890/42893	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	42893/42893	1.000
September 1 to 30, 2012								
<i>Female</i>								
Ch17 GSM High	58.0 to 303.9	6.00	5.99	0.30/0.07	3.154	11.061	52929/52959	0.999
Ch16 GSM Med	58.7 to 303.9	3.00	3.00	0.27/0.06	1.634	5.394	52953/52959	1.000
Ch20 GSM Low	59.4 to 303.9	1.50	1.49	0.28/0.07	0.823	3.032	52938/52959	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52959/52959	1.000
<i>Male</i>								
Ch06 GSM High	60.9 to 290.7	6.00	5.99	0.33/0.08	1.483	25.815	52932/52959	0.999
Ch05 GSM Med	62.0 to 302.3	3.00	3.00	0.31/0.07	1.646	10.693	52926/52959	0.999
Ch09 GSM Low	63.9 to 309.8	1.50	1.50	0.30/0.07	0.737	5.732	52920/52959	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52959/52959	1.000
October 1 to 31, 2012								
<i>Female</i>								
Ch17 GSM High	117.8 to 202.2	6.00	5.97	0.27/0.07	4.018	9.604	55337/55339	1.000
Ch16 GSM Med	118.8 to 201.8	3.00	2.98	0.25/0.06	1.870	4.575	55338/55339	1.000
Ch20 GSM Low	120.1 to 205.4	1.50	1.49	0.25/0.06	0.947	2.556	55332/55335	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55339/55339	1.000
<i>Male</i>								
Ch06 GSM High	137.9 to 306.4	6.00	5.94	0.30/0.07	3.814	9.705	55349/55351	1.000
Ch05 GSM Med	139.5 to 313.7	3.00	2.98	0.26/0.06	2.110	4.395	55351/55351	1.000
Ch09 GSM Low	143.1 to 318.0	1.50	1.48	0.24/0.06	1.089	2.162	55339/55339	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55351/55351	1.000
November 1 to 30, 2012								
<i>Female</i>								
Ch17 GSM High	207.1 to 246.3	6.00	5.96	0.27/0.06	4.468	8.847	53853/53853	1.000
Ch16 GSM Med	208.5 to 246.7	3.00	2.98	0.23/0.05	2.407	4.141	53853/53853	1.000
Ch20 GSM Low	210.9 to 246.6	1.50	1.49	0.24/0.06	0.990	2.587	53842/53853	1.000
Ch15 Sham	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53853/53853	1.000
<i>Male</i>								
Ch06 GSM High	313.9 to 389.2	6.00	5.95	0.29/0.07	4.132	8.526	53853/53853	1.000
Ch05 GSM Med	324.3 to 400.0	3.00	2.99	0.27/0.06	2.271	3.920	53853/53853	1.000
Ch09 GSM Low	329.8 to 402.5	1.50	1.49	0.22/0.05	1.178	2.057	53853/53853	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53853/53853	1.000

^a Ch=chamber (e.g., Ch17=Chamber 17)

TABLE II
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
December 1 to 31, 2012								
<i>Female</i>								
Ch17 GSM High	246.3 to 259.5	6.00	5.93	0.28/0.07	3.770	8.873	55607/55608	1.000
Ch16 GSM Med	246.7 to 257.1	3.00	2.97	0.23/0.06	2.283	4.025	55608/55608	1.000
Ch20 GSM Low	246.6 to 261.6	1.50	1.49	0.20/0.05	1.164	2.069	55608/55608	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55608/55608	1.000
<i>Male</i>								
Ch06 GSM High	389.2 to 419.3	6.00	5.97	0.30/0.07	4.198	8.500	55608/55608	1.000
Ch05 GSM Med	400.0 to 429.8	3.00	2.99	0.28/0.07	2.109	3.863	55608/55608	1.000
Ch09 GSM Low	402.5 to 432.3	1.50	1.48	0.23/0.06	1.119	1.916	55608/55608	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55608/55608	1.000
January 1 to 31, 2013								
<i>Female</i>								
Ch17 GSM High	259.5 to 275.8	6.00	5.95	0.27/0.07	4.415	8.955	55618/55618	1.000
Ch16 GSM Med	257.1 to 273.1	3.00	2.98	0.23/0.06	2.271	3.975	55619/55619	1.000
Ch20 GSM Low	261.6 to 279.0	1.50	1.49	0.21/0.05	1.148	2.082	55617/55617	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55619/55619	1.000
<i>Male</i>								
Ch06 GSM High	419.3 to 458.6	6.00	5.97	0.32/0.08	4.278	8.641	55619/55619	1.000
Ch05 GSM Med	429.8 to 464.1	3.00	2.97	0.27/0.06	2.242	3.984	55619/55619	1.000
Ch09 GSM Low	432.3 to 469.8	1.50	1.48	0.25/0.06	1.152	2.394	55618/55619	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55619/55619	1.000
February 1 to 28, 2013								
<i>Female</i>								
Ch17 GSM High	275.8 to 286.0	6.00	5.96	0.26/0.06	4.471	8.310	50082/50082	1.000
Ch16 GSM Med	273.1 to 282.0	3.00	2.98	0.24/0.06	2.326	3.891	50082/50082	1.000
Ch20 GSM Low	279.0 to 287.8	1.50	1.49	0.20/0.05	1.221	1.992	50082/50082	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	50082/50082	1.000
<i>Male</i>								
Ch06 GSM High	458.6 to 482.5	6.00	5.98	0.33/0.08	4.282	8.327	50082/50082	1.000
Ch05 GSM Med	464.1 to 493.7	3.00	3.00	0.28/0.07	2.362	3.887	50082/50082	1.000
Ch09 GSM Low	469.8 to 496.2	1.50	1.49	0.24/0.06	1.137	1.931	50082/50082	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	50082/50082	1.000
March 1 to 31, 2013								
<i>Female</i>								
Ch17 GSM High	286.0 to 296.4	6.00	5.93	0.27/0.06	4.613	8.141	55704/55704	1.000
Ch16 GSM Med	282.0 to 292.9	3.00	2.97	0.25/0.06	2.166	3.911	55704/55704	1.000
Ch20 GSM Low	287.8 to 297.0	1.50	1.48	0.20/0.05	1.198	1.965	55704/55704	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000
<i>Male</i>								
Ch06 GSM High	482.5 to 503.9	6.00	5.93	0.34/0.08	4.278	8.763	55704/55704	1.000
Ch05 GSM Med	493.7 to 513.0	3.00	3.01	0.28/0.07	2.351	4.000	55704/55704	1.000
Ch09 GSM Low	496.2 to 517.8	1.50	1.49	0.24/0.06	1.150	2.068	55704/55704	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000

TABLE I1
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stddev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
April 1 to 30, 2013								
<i>Female</i>								
Ch17 GSM High	296.4 to 305.2	6.00	5.97	0.28/0.07	4.614	8.286	53719/53719	1.000
Ch16 GSM Med	292.9 to 300.8	3.00	2.99	0.25/0.06	2.254	3.933	53719/53719	1.000
Ch20 GSM Low	297.0 to 307.9	1.50	1.49	0.20/0.05	1.215	2.087	53719/53719	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53719/53719	1.000
<i>Male</i>								
Ch06 GSM High	503.9 to 523.3	6.00	5.91	0.33/0.08	4.395	8.282	53721/53721	1.000
Ch05 GSM Med	513.0 to 531.8	3.00	3.00	0.29/0.07	2.310	4.013	53721/53721	1.000
Ch09 GSM Low	517.8 to 538.7	1.50	1.49	0.25/0.06	1.207	1.911	53719/53719	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53722/53722	1.000
May 1 to 31, 2013								
<i>Female</i>								
Ch17 GSM High	305.2 to 314.8	6.00	5.93	0.29/0.07	3.468	8.502	52758/52762	1.000
Ch16 GSM Med	300.8 to 309.8	3.00	2.98	0.26/0.06	1.676	3.984	52760/52762	1.000
Ch20 GSM Low	307.9 to 316.0	1.50	1.48	0.22/0.05	0.817	1.957	52759/52762	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52762/52762	1.000
<i>Male</i>								
Ch06 GSM High	523.3 to 539.7	6.00	5.96	0.35/0.08	3.356	8.858	52788/52789	1.000
Ch05 GSM Med	531.8 to 550.9	3.00	3.00	0.29/0.07	1.145	3.948	52788/52790	1.000
Ch09 GSM Low	538.7 to 557.8	1.50	1.48	0.30/0.07	0.849	2.306	52778/52788	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52790/52790	1.000
June 1 to 30, 2013								
<i>Female</i>								
Ch17 GSM High	314.8 to 322.9	6.00	5.98	0.30/0.07	3.712	8.162	53536/53537	1.000
Ch16 GSM Med	309.8 to 317.7	3.00	2.97	0.26/0.06	2.049	3.927	53537/53537	1.000
Ch20 GSM Low	316.0 to 324.2	1.50	1.50	0.22/0.05	1.068	1.913	53537/53537	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53537/53537	1.000
<i>Male</i>								
Ch06 GSM High	539.7 to 556.0	6.00	5.93	0.35/0.08	3.758	8.744	53543/53544	1.000
Ch05 GSM Med	550.9 to 566.9	3.00	2.97	0.29/0.07	2.164	4.004	53544/53544	1.000
Ch09 GSM Low	557.8 to 574.0	1.50	1.49	0.25/0.06	1.099	1.953	53537/53537	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53544/53544	1.000
July 1 to 31, 2013								
<i>Female</i>								
Ch17 GSM High	322.9 to 338.2	6.00	5.99	0.29/0.07	4.603	8.053	55527/55527	1.000
Ch16 GSM Med	316.3 to 334.5	3.00	3.00	0.25/0.06	2.330	3.798	55527/55527	1.000
Ch20 GSM Low	324.2 to 341.9	1.50	1.49	0.21/0.05	1.186	1.863	55527/55527	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55527/55527	1.000
<i>Male</i>								
Ch06 GSM High	556.0 to 585.8	6.00	5.92	0.35/0.08	4.259	8.691	55531/55531	1.000
Ch05 GSM Med	566.9 to 596.0	3.00	2.96	0.29/0.07	2.249	4.043	55531/55531	1.000
Ch09 GSM Low	574.0 to 607.9	1.50	1.48	0.27/0.06	1.119	2.044	55527/55527	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55531/55531	1.000

TABLE II
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 1 to 31, 2013								
<i>Female</i>								
Ch17 GSM High	338.2 to 344.5	6.00	5.98	0.29/0.07	4.360	7.931	55952/55952	1.000
Ch16 GSM Med	334.5 to 343.0	3.00	2.99	0.26/0.06	2.294	4.001	55952/55952	1.000
Ch20 GSM Low	341.9 to 348.4	1.50	1.49	0.20/0.05	1.195	1.858	55952/55952	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55952/55952	1.000
<i>Male</i>								
Ch06 GSM High	585.8 to 597.1	6.00	5.89	0.34/0.08	3.819	8.404	55955/55955	1.000
Ch05 GSM Med	596.0 to 606.8	3.00	2.98	0.28/0.07	2.303	3.856	55955/55955	1.000
Ch09 GSM Low	607.9 to 619.4	1.50	1.48	0.25/0.06	1.167	1.959	55952/55952	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55955/55955	1.000
September 1 to 30, 2013								
<i>Female</i>								
Ch17 GSM High	344.5 to 349.8	6.00	5.95	0.28/0.07	3.800	8.065	53696/53696	1.000
Ch16 GSM Med	343.0 to 352.4	3.00	2.98	0.28/0.07	1.822	4.020	53694/53696	1.000
Ch20 GSM Low	348.4 to 355.8	1.50	1.49	0.22/0.05	1.073	1.968	53696/53696	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53696/53696	1.000
<i>Male</i>								
Ch06 GSM High	597.1 to 607.0	6.00	5.94	0.34/0.08	4.168	8.242	53703/53703	1.000
Ch05 GSM Med	606.8 to 617.7	3.00	2.97	0.30/0.07	1.936	4.044	53703/53703	1.000
Ch09 GSM Low	619.4 to 633.9	1.50	1.49	0.25/0.06	1.029	1.959	53696/53696	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53703/53703	1.000
October 1 to 31, 2013								
<i>Female</i>								
Ch17 GSM High	349.8 to 360.2	6.00	5.96	0.36/0.09	3.397	9.676	56717/56748	0.999
Ch16 GSM Med	352.4 to 361.2	3.00	2.97	0.36/0.09	1.520	4.314	56693/56748	0.999
Ch20 GSM Low	355.8 to 366.5	1.50	1.48	0.26/0.06	0.926	1.910	56745/56747	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	56748/56748	1.000
<i>Male</i>								
Ch06 GSM High	607.0 to 615.0	6.00	5.93	0.38/0.09	3.232	8.512	56799/56807	1.000
Ch05 GSM Med	617.7 to 628.2	3.00	3.00	0.34/0.08	2.071	4.172	56807/56807	1.000
Ch09 GSM Low	633.9 to 642.8	1.50	1.49	0.27/0.06	1.027	2.186	56748/56748	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	56807/56807	1.000
November 1 to 30, 2013								
<i>Female</i>								
Ch17 GSM High	360.2 to 368.2	6.00	5.98	0.29/0.07	3.849	8.198	55323/55323	1.000
Ch16 GSM Med	361.2 to 371.8	3.00	3.00	0.28/0.07	1.907	3.925	55323/55323	1.000
Ch20 GSM Low	366.5 to 375.8	1.50	1.50	0.22/0.05	1.085	2.056	55323/55323	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55323/55323	1.000
<i>Male</i>								
Ch06 GSM High	615.0 to 625.1	6.00	5.94	0.33/0.08	4.105	8.973	55332/55332	1.000
Ch05 GSM Med	628.2 to 635.6	3.00	2.99	0.30/0.07	1.778	3.941	55331/55332	1.000
Ch09 GSM Low	642.8 to 648.2	1.50	1.48	0.24/0.06	1.105	2.003	55329/55329	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55332/55332	1.000

TABLE II
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight		Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio							
	Range [g]															
December 1 to 31, 2013																
<i>Female</i>																
Ch17 GSM High	368.2 to 376.2	6.00	6.00	0.30/0.07	4.541	8.484	53099/53099	1.000								
Ch16 GSM Med	371.8 to 380.7	3.00	2.99	0.27/0.06	2.251	3.819	53099/53099	1.000								
Ch20 GSM Low	375.8 to 383.8	1.50	1.49	0.21/0.05	1.251	1.897	53057/53057	1.000								
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53099/53099	1.000								
<i>Male</i>																
Ch06 GSM High	625.1 to 631.2	6.00	5.93	0.33/0.08	4.449	7.962	53111/53111	1.000								
Ch05 GSM Med	635.6 to 641.1	3.00	2.98	0.32/0.08	2.021	3.936	53111/53111	1.000								
Ch09 GSM Low	648.2 to 653.2	1.50	1.49	0.22/0.05	1.207	1.908	53099/53099	1.000								
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53111/53111	1.000								
January 1 to 31, 2014																
<i>Female</i>																
Ch17 GSM High	376.2 to 390.8	6.00	5.98	0.30/0.07	4.226	8.481	55626/55626	1.000								
Ch16 GSM Med	380.7 to 392.7	3.00	2.97	0.26/0.06	1.924	3.798	55626/55626	1.000								
Ch20 GSM Low	383.8 to 394.6	1.50	1.48	0.21/0.05	1.081	2.172	55621/55621	1.000								
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55626/55626	1.000								
<i>Male</i>																
Ch06 GSM High	631.2 to 641.5	6.00	5.90	0.33/0.08	4.292	8.752	55629/55629	1.000								
Ch05 GSM Med	641.1 to 651.7	3.00	2.99	0.33/0.08	2.235	4.217	55629/55629	1.000								
Ch09 GSM Low	653.2 to 662.0	1.50	1.49	0.23/0.05	1.181	1.899	55627/55627	1.000								
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55629/55629	1.000								
February 1 to 28, 2014																
<i>Female</i>																
Ch17 GSM High	390.8 to 401.7	6.00	5.96	0.30/0.07	4.639	8.584	51974/51974	1.000								
Ch16 GSM Med	392.7 to 399.4	3.00	2.98	0.26/0.06	2.235	3.989	51974/51974	1.000								
Ch20 GSM Low	394.6 to 410.5	1.50	1.50	0.20/0.05	1.226	1.894	51974/51974	1.000								
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	51974/51974	1.000								
<i>Male</i>																
Ch06 GSM High	641.5 to 650.2	6.00	5.92	0.33/0.08	4.376	8.314	51980/51980	1.000								
Ch05 GSM Med	651.7 to 668.4	3.00	2.97	0.31/0.07	2.183	4.076	51980/51980	1.000								
Ch09 GSM Low	662.0 to 676.6	1.50	1.49	0.23/0.05	1.192	1.888	51974/51974	1.000								
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	51980/51980	1.000								
March 1 to 31, 2014																
<i>Female</i>																
Ch17 GSM High	401.7 to 410.4	6.00	6.01	0.30/0.07	3.728	8.431	55703/55704	1.000								
Ch16 GSM Med	399.4 to 408.6	3.00	2.97	0.28/0.07	1.933	3.849	55704/55704	1.000								
Ch20 GSM Low	410.5 to 420.7	1.50	1.49	0.21/0.05	0.992	2.005	55704/55704	1.000								
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000								
<i>Male</i>																
Ch06 GSM High	650.2 to 658.2	6.00	5.92	0.34/0.08	4.216	9.271	55704/55704	1.000								
Ch05 GSM Med	668.4 to 679.5	3.00	2.95	0.33/0.08	2.187	4.010	55704/55704	1.000								
Ch09 GSM Low	676.6 to 688.8	1.50	1.50	0.23/0.05	1.190	1.982	55704/55704	1.000								
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000								

TABLE II
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
April 1 to 30, 2014								
<i>Female</i>								
Ch17 GSM High	410.4 to 422.9	6.00	5.98	0.34/0.08	3.545	8.198	53640/53644	1.000
Ch16 GSM Med	408.6 to 418.1	3.00	2.99	0.30/0.07	1.875	3.870	53643/53644	1.000
Ch20 GSM Low	420.7 to 431.1	1.50	1.48	0.21/0.05	1.096	1.840	53643/53643	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53644/53644	1.000
<i>Male</i>								
Ch06 GSM High	658.2 to 662.2	6.00	5.83	0.34/0.08	0.734	8.341	53646/53649	1.000
Ch05 GSM Med	676.3 to 679.5	3.00	2.97	0.32/0.08	2.265	4.191	53649/53649	1.000
Ch09 GSM Low	687.5 to 688.8	1.50	1.51	0.24/0.06	1.181	1.930	53645/53645	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53649/53649	1.000
May 1 to 31, 2014								
<i>Female</i>								
Ch17 GSM High	422.9 to 435.5	6.00	5.96	0.32/0.08	3.891	8.517	55604/55604	1.000
Ch16 GSM Med	418.1 to 429.3	3.00	2.99	0.27/0.06	2.189	4.205	55604/55604	1.000
Ch20 GSM Low	431.1 to 437.8	1.50	1.48	0.21/0.05	1.086	1.993	55602/55602	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55604/55604	1.000
<i>Male</i>								
Ch06 GSM High	662.2 to 667.4	6.00	5.88	0.32/0.08	4.364	8.818	55604/55604	1.000
Ch05 GSM Med	676.3 to 679.2	3.00	2.83	0.32/0.08	2.015	4.391	55604/55604	1.000
Ch09 GSM Low	687.5 to 691.2	1.50	1.50	0.25/0.06	1.195	1.976	55604/55604	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55604/55604	1.000
June 1 to 30, 2014								
<i>Female</i>								
Ch17 GSM High	435.5 to 453.7	6.00	5.98	0.32/0.08	4.053	8.457	53771/53771	1.000
Ch16 GSM Med	429.3 to 440.7	3.00	2.98	0.28/0.07	1.449	3.889	53767/53771	1.000
Ch20 GSM Low	437.8 to 452.4	1.50	1.49	0.22/0.05	0.702	2.034	53763/53767	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53771/53771	1.000
<i>Male</i>								
Ch06 GSM High	667.3 to 670.7	6.00	5.91	0.33/0.08	3.215	9.767	53769/53773	1.000
Ch05 GSM Med	670.5 to 679.2	3.00	2.89	0.35/0.08	0.391	4.215	53750/53774	1.000
Ch09 GSM Low	672.8 to 691.2	1.50	1.51	0.25/0.06	0.292	1.988	53767/53773	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53774/53774	1.000
July 1 to 31, 2014								
<i>Female</i>								
Ch17 GSM High	458.1 to 465.3	6.00	5.96	0.32/0.08	3.511	8.111	55599/55601	1.000
Ch16 GSM Med	443.4 to 447.0	3.00	2.98	0.28/0.07	1.763	4.162	55600/55601	1.000
Ch20 GSM Low	451.7 to 467.3	1.50	1.49	0.21/0.05	0.990	1.973	55601/55601	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55601/55601	1.000
<i>Male</i>								
Ch06 GSM High	667.3 to 670.3	6.00	5.87	0.32/0.08	3.459	8.639	55602/55603	1.000
Ch05 GSM Med	659.4 to 670.5	3.00	2.96	0.32/0.08	1.787	4.205	55602/55603	1.000
Ch09 GSM Low	659.5 to 672.8	1.50	1.50	0.24/0.06	0.990	1.960	55601/55601	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55603/55603	1.000

TABLE II
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 1 to 31, 2014								
<i>Female</i>								
Ch17 GSM High	464.7 to 473.8	6.00	5.92	0.30/0.07	3.387	8.139	54345/54354	1.000
Ch16 GSM Med	447.0 to 453.0	3.00	2.98	0.27/0.07	1.868	4.001	54353/54354	1.000
Ch20 GSM Low	461.2 to 475.4	1.50	1.49	0.21/0.05	1.112	1.875	54354/54354	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	54354/54354	1.000
<i>Male</i>								
Ch06 GSM High	655.0 to 670.3	6.00	5.90	0.32/0.08	4.391	8.661	54358/54358	1.000
Ch05 GSM Med	638.8 to 659.4	3.00	2.98	0.30/0.07	2.247	4.095	54358/54358	1.000
Ch09 GSM Low	653.6 to 660.1	1.50	1.49	0.22/0.05	1.171	1.819	54354/54354	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	54358/54358	1.000
September 1 to 30, 2014								
<i>Female</i>								
Ch17 GSM High	471.2 to 475.1	6.00	5.94	0.30/0.07	2.537	11.960	52084/52088	1.000
Ch16 GSM Med	453.0 to 471.6	3.00	2.98	0.26/0.06	1.802	5.349	52085/52088	1.000
Ch20 GSM Low	461.2 to 476.1	1.50	1.48	0.20/0.05	1.070	2.533	52086/52088	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52088/52088	1.000
<i>Male</i>								
Ch06 GSM High	650.2 to 655.0	6.00	5.95	0.30/0.07	2.313	11.724	52074/52088	1.000
Ch05 GSM Med	636.4 to 638.8	3.00	2.98	0.25/0.06	2.239	4.851	52087/52088	1.000
Ch09 GSM Low	644.0 to 653.6	1.50	1.48	0.22/0.05	0.916	2.468	52085/52088	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52088/52088	1.000
August 8, 2012 to September 30, 2014								
<i>Female</i>								
Ch17 GSM High	58.0 to 475.1	6.00	5.97	0.30/0.07	2.537	11.960	1400145/1400254	1.000
Ch16 GSM Med	58.7 to 471.6	3.00	2.98	0.27/0.06	1.449	5.394	1400178/1400255	1.000
Ch20 GSM Low	59.4 to 476.1	1.50	1.49	0.22/0.05	0.702	3.032	1400140/1400194	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	1400255/1400255	1.000
<i>Male</i>								
Ch06 GSM High	60.9 to 670.7	6.00	5.93	0.33/0.08	0.734	25.815	1400345/1400419	1.000
Ch05 GSM Med	62.0 to 679.5	3.00	2.97	0.30/0.07	0.391	10.693	1400356/1400421	1.000
Ch09 GSM Low	63.9 to 691.2	1.50	1.49	0.25/0.06	0.292	5.732	1400229/1400291	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	1400422/1400422	1.000

TABLE I2**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field^a**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
August 8 to 31, 2012							
<i>Male</i>							
Ch06 GSM High	246.10 to 283.80	261.74	0.30/9.31	163.93	357.66	85760/85786	1.000
Ch05 GSM Med	174.00 to 200.70	185.30	0.25/5.39	119.06	238.06	85780/85786	1.000
Ch09 GSM Low	123.10 to 141.90	131.09	0.23/3.52	82.96	172.00	85780/85786	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	85786/85786	1.000
September 1 -30, 2012							
<i>Female</i>							
Ch17 GSM High	192.60 to 269.00	237.79	0.30/8.24	153.47	362.88	105858/105918	0.999
Ch16 GSM Med	136.20 to 190.20	167.23	0.26/5.13	105.81	241.99	105906/105918	1.000
Ch20 GSM Low	096.10 to 134.50	118.05	0.27/3.74	75.08	175.15	105876/105918	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
<i>Male</i>							
Ch06 GSM High	192.60 to 266.50	232.35	0.32/8.68	106.40	443.91	105868/105918	1.000
Ch05 GSM Med	136.20 to 190.20	164.81	0.30/5.71	103.62	285.70	105858/105918	0.999
Ch09 GSM Low	096.30 to 134.50	116.84	0.29/3.90	75.03	209.17	105848/105918	0.999
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
<i>Female</i>							
Ch17 GSM High	193.80 to 232.80	214.26	0.28/6.95	164.06	288.93	110674/110678	1.000
Ch16 GSM Med	137.00 to 164.60	151.43	0.25/4.49	112.41	203.02	110676/110678	1.000
Ch20 GSM Low	098.50 to 116.40	108.14	0.25/3.20	79.98	143.57	110662/110670	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110678/110678	1.000
<i>Male</i>							
Ch06 GSM High	201.30 to 269.00	238.81	0.30/8.48	160.53	326.54	110698/110702	1.000
Ch05 GSM Med	142.40 to 192.20	170.55	0.26/5.18	127.50	224.85	110702/110702	1.000
Ch09 GSM Low	103.10 to 135.90	121.57	0.24/3.47	89.23	153.28	110678/110678	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
<i>Female</i>							
Ch17 GSM High	232.80 to 250.70	242.22	0.27/7.66	200.64	298.50	107706/107706	1.000
Ch16 GSM Med	164.60 to 177.30	171.90	0.24/4.71	147.26	201.43	107706/107706	1.000
Ch20 GSM Low	118.50 to 125.40	122.10	0.24/3.47	99.98	161.64	107684/107706	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000
<i>Male</i>							
Ch06 GSM High	271.80 to 292.20	282.73	0.30/9.97	238.55	347.86	107706/107706	1.000
Ch05 GSM Med	194.20 to 208.20	201.89	0.27/6.40	176.39	237.97	107706/107706	1.000
Ch09 GSM Low	137.40 to 148.20	143.02	0.22/3.68	123.60	164.44	107706/107706	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000

^a Ch=chamber (e.g., Ch17=Chamber 17)

TABLE I2**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
December 1 to 31, 2012							
<i>Female</i>							
Ch17 GSM High	250.70 to 254.90	253.13	0.28/8.43	202.44	310.55	111214/111216	1.000
Ch16 GSM Med	177.30 to 180.20	179.20	0.24/4.96	155.02	209.16	111216/111216	1.000
Ch20 GSM Low	125.40 to 129.20	127.62	0.21/3.08	112.48	149.98	111216/111216	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
<i>Male</i>							
Ch06 GSM High	292.20 to 298.00	295.52	0.31/10.70	248.48	353.56	111216/111216	1.000
Ch05 GSM Med	208.20 to 211.70	210.50	0.28/6.92	177.40	240.12	111216/111216	1.000
Ch09 GSM Low	148.20 to 150.20	148.91	0.24/4.13	129.81	170.40	111216/111216	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	254.90 to 261.40	257.58	0.28/8.39	220.72	312.88	111236/111236	1.000
Ch16 GSM Med	180.20 to 184.80	182.39	0.24/5.03	157.70	211.28	111238/111238	1.000
Ch20 GSM Low	129.20 to 130.70	129.57	0.21/3.21	114.20	153.83	111234/111234	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
<i>Male</i>							
Ch06 GSM High	298.00 to 302.10	298.69	0.33/11.42	250.82	361.83	111238/111238	1.000
Ch05 GSM Med	211.70 to 214.00	212.38	0.28/6.89	182.92	247.58	111238/111238	1.000
Ch09 GSM Low	150.20 to 151.40	150.50	0.26/4.49	132.92	190.45	111236/111238	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
<i>Female</i>							
Ch17 GSM High	261.40 to 264.00	261.98	0.27/8.15	228.00	307.30	100164/100164	1.000
Ch16 GSM Med	184.80 to 186.70	185.40	0.25/5.38	164.18	212.70	100164/100164	1.000
Ch20 GSM Low	130.70 to 132.00	131.16	0.20/3.13	117.78	150.45	100164/100164	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
<i>Male</i>							
Ch06 GSM High	302.10 to 304.00	302.15	0.33/11.82	256.65	357.92	100164/100164	1.000
Ch05 GSM Med	214.00 to 215.50	214.86	0.28/7.04	190.61	244.53	100164/100164	1.000
Ch09 GSM Low	151.40 to 152.40	151.39	0.24/4.22	132.24	172.35	100164/100164	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	264.00 to 266.50	264.63	0.27/8.42	231.60	307.67	111408/111408	1.000
Ch16 GSM Med	186.70 to 188.40	187.36	0.25/5.58	160.59	215.77	111408/111408	1.000
Ch20 GSM Low	132.00 to 133.20	132.42	0.21/3.18	119.43	152.94	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch06 GSM High	304.00 to 305.60	303.27	0.35/12.40	258.25	370.03	111408/111408	1.000
Ch05 GSM Med	215.50 to 216.80	216.06	0.28/7.06	190.20	250.00	111408/111408	1.000
Ch09 GSM Low	152.40 to 153.30	152.35	0.24/4.31	133.03	178.38	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000

TABLE I2**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
April 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	266.50 to 269.00	267.57	0.28/8.86	235.78	314.88	107438/107438	1.000
Ch16 GSM Med	188.40 to 190.20	189.34	0.26/5.68	164.80	217.69	107438/107438	1.000
Ch20 GSM Low	133.20 to 134.50	133.66	0.21/3.22	120.28	157.64	107438/107438	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107438/107438	1.000
<i>Male</i>							
Ch06 GSM High	305.60 to 307.60	305.35	0.34/12.25	263.80	362.58	107442/107442	1.000
Ch05 GSM Med	216.80 to 218.30	217.81	0.29/7.44	191.48	252.37	107442/107442	1.000
Ch09 GSM Low	153.30 to 154.40	153.44	0.25/4.47	137.33	173.95	107438/107438	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	269.00 to 271.80	269.81	0.30/9.50	206.91	323.99	105516/105524	1.000
Ch16 GSM Med	190.20 to 190.20	189.49	0.27/5.91	142.11	219.08	105520/105524	1.000
Ch20 GSM Low	134.50 to 135.90	135.04	0.23/3.57	100.46	155.42	105518/105524	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105524/105524	1.000
<i>Male</i>							
Ch06 GSM High	307.60 to 308.80	307.28	0.36/12.95	230.79	374.97	105576/105578	1.000
Ch05 GSM Med	218.30 to 220.10	219.52	0.29/7.56	135.92	251.57	105576/105580	1.000
Ch09 GSM Low	154.40 to 155.60	154.43	0.31/5.53	117.02	191.30	105556/105576	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	271.80 to 274.70	273.16	0.31/9.87	215.41	319.42	107072/107074	1.000
Ch16 GSM Med	190.20 to 192.20	191.37	0.27/6.01	159.04	220.20	107074/107074	1.000
Ch20 GSM Low	135.90 to 137.40	136.69	0.22/3.56	115.55	154.63	107074/107074	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107074/107074	1.000
<i>Male</i>							
Ch06 GSM High	308.80 to 311.30	308.73	0.36/12.96	246.21	375.54	107086/107088	1.000
Ch05 GSM Med	220.10 to 221.00	220.30	0.29/7.53	188.37	256.20	107088/107088	1.000
Ch09 GSM Low	155.60 to 157.00	156.15	0.26/4.68	134.23	178.92	107074/107074	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107088/107088	1.000
July 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	274.70 to 277.70	273.91	0.29/9.29	239.88	317.27	111054/111054	1.000
Ch16 GSM Med	192.20 to 196.40	193.72	0.25/5.73	172.83	217.88	111054/111054	1.000
Ch20 GSM Low	137.40 to 140.40	138.40	0.21/3.44	123.31	156.56	111054/111054	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111054/111054	1.000
<i>Male</i>							
Ch06 GSM High	311.30 to 315.30	311.45	0.36/13.11	264.23	378.17	111062/111062	1.000
Ch05 GSM Med	221.00 to 223.90	222.10	0.30/7.70	193.61	259.58	111062/111062	1.000
Ch09 GSM Low	157.00 to 159.00	157.04	0.27/5.00	136.56	186.11	111054/111054	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111062/111062	1.000

TABLE I2**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
August 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	277.70 to 280.80	277.49	0.29/9.56	236.42	318.87	111904/111904	1.000
Ch16 GSM Med	196.40 to 198.50	196.28	0.26/5.94	171.49	226.49	111904/111904	1.000
Ch20 GSM Low	140.40 to 140.40	139.96	0.21/3.34	125.38	156.35	111904/111904	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111904/111904	1.000
<i>Male</i>							
Ch06 GSM High	315.30 to 316.70	312.96	0.35/12.91	252.28	374.25	111910/111910	1.000
Ch05 GSM Med	223.90 to 224.90	222.92	0.29/7.53	197.54	253.53	111910/111910	1.000
Ch09 GSM Low	159.00 to 159.70	158.28	0.25/4.71	140.65	182.22	111904/111904	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	280.80 to 280.80	279.69	0.29/9.35	223.60	325.76	107392/107392	1.000
Ch16 GSM Med	198.50 to 200.70	198.49	0.29/6.69	154.85	229.99	107388/107392	1.000
Ch20 GSM Low	140.40 to 141.90	140.30	0.22/3.60	118.81	160.92	107392/107392	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107392/107392	1.000
<i>Male</i>							
Ch06 GSM High	316.70 to 318.10	314.99	0.35/12.78	265.80	370.62	107406/107406	1.000
Ch05 GSM Med	224.90 to 225.90	224.37	0.30/8.01	181.13	261.80	107406/107406	1.000
Ch09 GSM Low	159.70 to 161.00	159.38	0.26/4.76	132.09	182.21	107392/107392	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107406/107406	1.000
October 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	280.80 to 286.80	281.83	0.37/12.27	215.71	356.81	113434/113496	0.999
Ch16 GSM Med	200.70 to 202.80	200.67	0.37/8.80	144.28	241.44	113388/113496	0.999
Ch20 GSM Low	141.90 to 143.40	141.96	0.27/4.41	112.11	161.75	113490/113494	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113496/113496	1.000
<i>Male</i>							
Ch06 GSM High	318.10 to 319.40	316.70	0.40/14.75	20.95	379.83	113600/113614	1.000
Ch05 GSM Med	225.90 to 226.80	226.15	0.34/9.08	188.65	268.19	113614/113614	1.000
Ch09 GSM Low	161.00 to 161.70	160.60	0.27/5.14	134.24	194.14	113496/113496	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	286.80 to 286.80	286.14	0.29/9.81	229.63	335.12	110646/110646	1.000
Ch16 GSM Med	202.80 to 204.80	203.21	0.28/6.65	161.63	233.48	110646/110646	1.000
Ch20 GSM Low	143.40 to 144.80	143.51	0.22/3.73	122.76	168.98	110646/110646	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110646/110646	1.000
<i>Male</i>							
Ch06 GSM High	319.40 to 320.80	318.24	0.33/12.45	263.77	389.98	110664/110664	1.000
Ch05 GSM Med	226.80 to 227.70	226.96	0.30/8.01	175.08	260.66	110662/110664	1.000
Ch09 GSM Low	161.70 to 161.70	161.02	0.25/4.62	139.23	187.46	110658/110658	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110664/110664	1.000

TABLE I2
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
December 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	286.80 to 289.60	287.63	0.30/10.26	251.13	340.91	106198/106198	1.000
Ch16 GSM Med	204.80 to 206.70	205.25	0.27/6.51	176.82	231.75	106198/106198	1.000
Ch20 GSM Low	144.80 to 146.10	144.94	0.21/3.57	132.18	162.32	106114/106114	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106198/106198	1.000
<i>Male</i>							
Ch06 GSM High	320.80 to 322.10	319.56	0.33/12.50	276.96	370.51	106222/106222	1.000
Ch05 GSM Med	227.70 to 228.70	227.45	0.32/8.64	186.67	262.62	106222/106222	1.000
Ch09 GSM Low	161.70 to 162.40	161.63	0.22/4.24	145.51	182.95	106198/106198	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	289.60 to 294.50	291.48	0.31/10.54	247.48	345.55	111252/111252	1.000
Ch16 GSM Med	206.70 to 208.20	206.91	0.27/6.43	166.99	234.62	111252/111252	1.000
Ch20 GSM Low	146.10 to 147.30	146.17	0.22/3.73	125.15	177.44	111242/111242	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111252/111252	1.000
<i>Male</i>							
Ch06 GSM High	322.10 to 323.40	320.41	0.34/12.88	272.03	391.85	111258/111258	1.000
Ch05 GSM Med	228.70 to 229.60	228.70	0.33/8.88	198.03	272.01	111258/111258	1.000
Ch09 GSM Low	162.40 to 163.00	162.49	0.23/4.34	143.93	184.14	111254/111254	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
<i>Female</i>							
Ch17 GSM High	294.50 to 296.40	295.29	0.30/10.52	260.64	355.30	103948/103948	1.000
Ch16 GSM Med	208.20 to 208.20	207.59	0.27/6.51	179.98	240.45	103948/103948	1.000
Ch20 GSM Low	147.30 to 149.00	148.04	0.20/3.53	133.39	166.89	103948/103948	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103948/103948	1.000
<i>Male</i>							
Ch06 GSM High	323.40 to 324.80	322.09	0.33/12.65	277.08	381.92	103960/103960	1.000
Ch05 GSM Med	229.60 to 230.60	229.42	0.32/8.48	197.45	269.79	103960/103960	1.000
Ch09 GSM Low	163.00 to 163.70	163.19	0.23/4.39	145.88	183.61	103948/103948	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103960/103960	1.000
March 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	296.40 to 298.00	297.00	0.31/10.72	234.16	352.11	111404/111408	1.000
Ch16 GSM Med	208.20 to 209.60	208.52	0.28/6.90	168.58	237.91	111408/111408	1.000
Ch20 GSM Low	149.00 to 149.70	148.83	0.21/3.69	121.71	172.98	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch06 GSM High	324.80 to 324.80	321.89	0.35/13.20	271.97	403.29	111408/111408	1.000
Ch05 GSM Med	230.60 to 231.50	229.35	0.33/8.95	197.64	267.59	111408/111408	1.000
Ch09 GSM Low	163.70 to 164.40	163.82	0.23/4.36	145.79	188.13	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000

TABLE I2**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	298.00 to 299.30	298.09	0.34/12.08	230.03	349.80	107280/107288	1.000
Ch16 GSM Med	209.60 to 210.70	209.72	0.31/7.56	166.05	238.56	107286/107288	1.000
Ch20 GSM Low	149.70 to 150.20	149.58	0.22/3.77	128.89	166.30	107286/107286	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107288/107288	1.000
<i>Male</i>							
Ch06 GSM High	324.80 to 326.10	321.85	0.35/13.36	114.48	385.94	107292/107298	1.000
Ch05 GSM Med	231.50 to 231.50	229.97	0.33/8.93	201.12	273.57	107298/107298	1.000
Ch09 GSM Low	164.40 to 164.40	164.03	0.24/4.54	145.21	185.62	107290/107290	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	299.30 to 300.40	300.03	0.33/11.60	242.81	359.23	111208/111208	1.000
Ch16 GSM Med	210.70 to 211.70	211.00	0.27/6.71	180.74	250.53	111208/111208	1.000
Ch20 GSM Low	150.20 to 150.20	149.77	0.22/3.81	128.27	173.78	111204/111204	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
<i>Male</i>							
Ch06 GSM High	326.10 to 326.10	323.75	0.32/12.29	279.16	396.81	111208/111208	1.000
Ch05 GSM Med	231.50 to 231.50	224.46	0.33/8.72	189.71	280.03	111208/111208	1.000
Ch09 GSM Low	164.40 to 165.10	164.89	0.25/4.91	147.41	189.52	111208/111208	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
June 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	300.40 to 302.10	300.88	0.33/11.52	247.80	357.96	107542/107542	1.000
Ch16 GSM Med	211.70 to 213.10	212.09	0.29/7.21	147.07	242.76	107534/107542	1.000
Ch20 GSM Low	150.20 to 151.00	150.43	0.22/3.91	103.10	175.53	107526/107534	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107542/107542	1.000
<i>Male</i>							
Ch06 GSM High	326.10 to 327.40	324.69	0.34/12.95	239.60	417.63	107538/107546	1.000
Ch05 GSM Med	231.50 to 231.50	226.88	0.37/9.99	83.52	274.36	107500/107548	1.000
Ch09 GSM Low	163.70 to 165.10	164.18	0.26/4.92	72.17	190.10	107534/107546	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	302.10 to 302.70	301.45	0.33/11.55	230.64	352.78	111198/111202	1.000
Ch16 GSM Med	213.10 to 213.10	212.21	0.28/7.05	163.42	251.13	111200/111202	1.000
Ch20 GSM Low	151.00 to 151.40	150.88	0.21/3.77	122.47	172.90	111202/111202	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111202/111202	1.000
<i>Male</i>							
Ch06 GSM High	326.10 to 327.40	323.48	0.33/12.46	248.53	392.76	111204/111206	1.000
Ch05 GSM Med	229.60 to 231.50	229.53	0.33/8.75	178.63	274.03	111204/111206	1.000
Ch09 GSM Low	162.40 to 163.70	163.30	0.24/4.55	132.94	187.07	111202/111202	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111206/111206	1.000

TABLE I2**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	302.70 to 303.40	301.67	0.31/10.80	228.28	353.86	108690/108708	1.000
Ch16 GSM Med	213.10 to 213.60	212.27	0.28/6.99	168.24	246.20	108706/108708	1.000
Ch20 GSM Low	151.40 to 151.70	151.14	0.21/3.78	130.78	169.86	108708/108708	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108708/108708	1.000
<i>Male</i>							
Ch06 GSM High	324.80 to 327.40	323.73	0.33/12.36	280.03	389.80	108716/108716	1.000
Ch05 GSM Med	227.70 to 229.60	228.01	0.31/8.28	198.54	268.03	108716/108716	1.000
Ch09 GSM Low	162.40 to 163.00	162.22	0.22/4.20	144.61	179.04	108708/108708	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	303.40 to 303.40	302.18	0.30/10.67	197.54	428.95	104168/104176	1.000
Ch16 GSM Med	213.60 to 214.50	212.76	0.26/6.52	166.50	286.87	104170/104176	1.000
Ch20 GSM Low	151.40 to 151.70	151.10	0.21/3.63	128.30	197.41	104172/104176	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
<i>Male</i>							
Ch06 GSM High	324.80 to 324.80	322.76	0.31/11.68	201.44	453.53	104148/104176	1.000
Ch05 GSM Med	227.70 to 227.70	226.73	0.25/6.74	196.47	289.19	104174/104176	1.000
Ch09 GSM Low	161.70 to 162.40	161.10	0.22/4.12	126.78	208.08	104170/104176	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
<i>Female</i>							
Ch17 GSM High	192.60 to 303.40	276.19	0.30/9.74	153.47	428.95	2800286/2800508	1.000
Ch16 GSM Med	136.20 to 214.50	195.10	0.27/6.17	105.81	286.87	2800358/2800510	1.000
Ch20 GSM Low	096.10 to 151.70	138.52	0.22/3.59	75.08	197.41	2800278/2800388	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800510/2800510	1.000
<i>Male</i>							
Ch06 GSM High	192.60 to 327.40	305.54	0.34/12.09	20.95	453.53	2800696/2800838	1.000
Ch05 GSM Med	136.20 to 231.50	217.06	0.31/7.84	83.52	289.19	2800718/2800842	1.000
Ch09 GSM Low	096.30 to 165.10	154.30	0.25/4.47	72.17	209.17	2800466/2800582	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800844/2800844	1.000

TABLE I3**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – E-Field^a**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 8 to 31, 2012							
<i>Male</i>							
Ch06 GSM High	246.1 to 283.8	246.42	0.40/11.69	154.4	330.2	85692/85786	0.999
Ch05 GSM Med	174.0 to 200.7	192.97	0.37/8.36	124.3	252.8	85768/85786	1.000
Ch09 GSM Low	123.1 to 141.9	119.12	0.32/4.49	74.9	162.8	85742/85786	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	85786/85786	1.000
September 1 to 30, 2012							
<i>Female</i>							
Ch17 GSM High	192.6 to 269.0	231.49	0.54/14.83	141.4	395.5	105788/105918	0.999
Ch16 GSM Med	136.2 to 190.2	161.13	0.37/7.09	101.6	244.1	105894/105918	1.000
Ch20 GSM Low	96.1 to 134.5	112.92	0.39/5.13	71.8	170.4	105878/105918	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
<i>Male</i>							
Ch06 GSM High	192.6 to 266.5	221.52	0.45/11.71	104.2	445.8	105824/105918	0.999
Ch05 GSM Med	136.2 to 190.2	174.00	0.45/9.23	110.2	323.0	105652/105918	0.997
Ch09 GSM Low	96.3 to 134.5	109.88	0.38/4.91	68.9	195.7	105852/105918	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
October 1 to 31, 2012							
<i>Female</i>							
Ch17 GSM High	193.8 to 232.8	193.51	0.44/10.00	137.3	285.2	109918/110678	0.993
Ch16 GSM Med	137.0 to 164.6	138.97	0.34/5.51	108.8	184.5	110640/110678	1.000
Ch20 GSM Low	98.5 to 116.4	106.63	0.36/4.47	75.3	141.4	110658/110670	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110678/110678	1.000
<i>Male</i>							
Ch06 GSM High	201.3 to 269.0	222.93	0.40/10.56	140.8	316.8	110628/110702	0.999
Ch05 GSM Med	142.4 to 192.2	173.85	0.38/7.74	128.3	226.9	110700/110702	1.000
Ch09 GSM Low	103.1 to 135.9	108.94	0.30/3.85	80.3	143.4	110474/110678	0.998
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110702/110702	1.000
November 1 to 30, 2012							
<i>Female</i>							
Ch17 GSM High	232.8 to 250.7	222.28	0.40/10.48	178.3	284.9	107584/107706	0.999
Ch16 GSM Med	164.6 to 177.3	156.73	0.35/6.43	129.5	196.0	107652/107706	0.999
Ch20 GSM Low	118.5 to 125.4	116.79	0.61/8.50	85.0	170.5	107364/107706	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000
<i>Male</i>							
Ch06 GSM High	271.8 to 292.2	263.30	0.42/13.17	212.1	339.0	107672/107706	1.000
Ch05 GSM Med	194.2 to 208.2	205.84	0.42/10.20	164.2	257.0	107706/107706	1.000
Ch09 GSM Low	137.4 to 148.2	127.09	0.31/4.62	105.4	150.3	107422/107706	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000

^a Ch=chamber (e.g., Ch17=Chamber 17)

TABLE I3**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2012							
<i>Female</i>							
Ch17 GSM High	250.7 to 254.9	232.81	0.41/11.12	177.9	303.9	110988/111216	0.998
Ch16 GSM Med	177.3 to 180.2	165.11	0.38/7.41	135.9	208.6	111180/111216	1.000
Ch20 GSM Low	125.4 to 129.2	112.68	0.32/4.18	94.9	136.5	110810/111216	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
<i>Male</i>							
Ch06 GSM High	292.2 to 298.0	275.64	0.45/14.54	220.5	356.3	111140/111216	0.999
Ch05 GSM Med	208.2 to 211.7	219.30	0.44/11.41	173.0	271.3	111208/111216	1.000
Ch09 GSM Low	148.2 to 150.2	132.15	0.32/4.89	113.4	153.0	110840/111216	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
January 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	254.9 to 261.4	237.27	0.41/11.43	193.2	293.0	111138/111238	0.999
Ch16 GSM Med	180.2 to 184.8	167.60	0.34/6.66	141.7	198.4	111220/111238	1.000
Ch20 GSM Low	129.2 to 130.7	115.91	0.32/4.36	99.7	140.3	111140/111234	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
<i>Male</i>							
Ch06 GSM High	298.0 to 302.1	279.82	0.47/15.40	220.4	362.2	111106/111238	0.999
Ch05 GSM Med	211.7 to 214.0	221.30	0.45/11.71	179.9	275.1	111236/111238	1.000
Ch09 GSM Low	150.2 to 151.4	135.72	0.33/5.19	114.5	174.3	111180/111238	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
February 1 to 28, 2013							
<i>Female</i>							
Ch17 GSM High	261.4 to 264.0	244.04	0.40/11.51	201.9	309.0	100140/100164	1.000
Ch16 GSM Med	184.8 to 186.7	174.32	0.38/7.89	145.5	216.4	100160/100164	1.000
Ch20 GSM Low	130.7 to 132.0	116.79	0.32/4.36	99.0	141.3	100012/100164	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
<i>Male</i>							
Ch06 GSM High	302.1 to 304.0	276.46	0.45/14.58	221.6	347.2	99924/100164	0.998
Ch05 GSM Med	214.0 to 215.5	227.95	0.47/12.61	186.4	279.7	100128/100164	1.000
Ch09 GSM Low	151.4 to 152.4	136.55	0.32/5.04	114.7	160.3	100106/100164	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
March 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	264.0 to 266.5	248.07	0.39/11.49	197.3	308.4	111392/111408	1.000
Ch16 GSM Med	186.7 to 188.4	178.55	0.38/7.88	147.1	212.8	111396/111408	1.000
Ch20 GSM Low	132.0 to 133.2	117.42	0.32/4.39	99.1	143.9	111142/111408	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
<i>Male</i>							
Ch06 GSM High	304.0 to 305.6	278.61	0.46/15.06	224.1	380.8	111090/111408	0.997
Ch05 GSM Med	215.5 to 216.8	229.50	0.45/12.26	186.4	283.8	111380/111408	1.000
Ch09 GSM Low	152.4 to 153.3	136.42	0.31/4.94	115.9	162.5	111286/111408	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000

TABLE I3
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	266.5 to 269.0	250.31	0.41/11.95	207.4	314.9	107404/107438	1.000
Ch16 GSM Med	188.4 to 190.2	178.92	0.40/8.46	147.1	219.1	107426/107438	1.000
Ch20 GSM Low	133.2 to 134.5	119.19	0.32/4.45	101.7	149.3	107286/107438	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107438/107438	1.000
<i>Male</i>							
Ch06 GSM High	305.6 to 307.6	280.59	0.47/15.43	229.2	372.4	107104/107442	0.997
Ch05 GSM Med	216.8 to 218.3	231.77	0.46/12.63	189.1	288.7	107402/107442	1.000
Ch09 GSM Low	153.3 to 154.4	137.80	0.32/5.12	111.5	163.3	107268/107438	0.998
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107444/107444	1.000
May 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	269.0 to 271.8	256.26	0.44/13.37	184.1	324.2	105446/105524	0.999
Ch16 GSM Med	190.2 to 190.2	179.56	0.38/8.02	138.8	219.5	105504/105524	1.000
Ch20 GSM Low	134.5 to 135.9	121.06	0.33/4.64	90.0	145.1	105262/105524	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105524/105524	1.000
<i>Male</i>							
Ch06 GSM High	307.6 to 308.8	278.54	0.46/15.09	206.9	359.6	104946/105578	0.994
Ch05 GSM Med	218.3 to 220.1	237.22	0.49/13.63	151.8	290.6	105468/105580	0.999
Ch09 GSM Low	154.4 to 155.6	137.77	0.43/7.00	88.7	209.9	104226/105576	0.987
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105580/105580	1.000
June 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	271.8 to 274.7	252.40	0.44/13.06	194.3	343.6	106884/107074	0.998
Ch16 GSM Med	190.2 to 192.2	180.43	0.40/8.49	138.4	219.4	106978/107074	0.999
Ch20 GSM Low	135.9 to 137.4	121.62	0.33/4.72	97.5	143.3	106782/107074	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107074/107074	1.000
<i>Male</i>							
Ch06 GSM High	308.8 to 311.3	275.67	0.44/14.27	212.0	349.6	105800/107088	0.988
Ch05 GSM Med	220.1 to 221.0	236.86	0.48/13.34	191.9	295.4	107012/107088	0.999
Ch09 GSM Low	155.6 to 157.0	140.60	0.33/5.40	119.5	168.0	106942/107074	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107088/107088	1.000
July 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	274.7 to 277.7	253.80	0.40/11.87	205.8	314.3	110994/111054	0.999
Ch16 GSM Med	192.2 to 196.4	182.06	0.39/8.41	151.1	219.7	111034/111054	1.000
Ch20 GSM Low	137.4 to 140.4	122.97	0.32/4.66	105.2	149.3	110832/111054	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111054/111054	1.000
<i>Male</i>							
Ch06 GSM High	311.3 to 315.3	278.80	0.44/14.64	223.1	369.0	109898/111062	0.990
Ch05 GSM Med	221.0 to 223.9	237.76	0.50/14.21	189.2	300.6	110936/111062	0.999
Ch09 GSM Low	157.0 to 159.0	140.17	0.33/5.51	115.0	170.4	110664/111054	0.996
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111062/111062	1.000

TABLE I3**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	277.7 to 280.8	257.67	0.43/12.95	208.3	319.3	111830/111904	0.999
Ch16 GSM Med	196.4 to 198.5	182.52	0.39/8.42	151.0	222.3	111850/111904	1.000
Ch20 GSM Low	140.4 to 140.4	124.67	0.33/4.84	104.3	150.3	111676/111904	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111904/111904	1.000
<i>Male</i>							
Ch06 GSM High	315.3 to 316.7	279.34	0.45/14.72	215.4	372.8	110478/111910	0.987
Ch05 GSM Med	223.9 to 224.9	237.25	0.49/13.81	192.1	293.2	111860/111910	1.000
Ch09 GSM Low	159.0 to 159.7	142.43	0.32/5.41	120.6	169.9	111800/111904	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111910/111910	1.000
September 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	280.8 to 280.8	262.26	0.42/13.14	204.1	327.2	107316/107392	0.999
Ch16 GSM Med	198.5 to 200.7	185.57	0.39/8.61	139.3	226.1	107296/107392	0.999
Ch20 GSM Low	140.4 to 141.9	125.73	0.33/4.86	106.5	148.1	107242/107392	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107392/107392	1.000
<i>Male</i>							
Ch06 GSM High	316.7 to 318.1	281.16	0.48/15.95	224.7	390.2	105756/107406	0.985
Ch05 GSM Med	224.9 to 225.9	238.83	0.50/14.05	187.3	293.1	107356/107406	1.000
Ch09 GSM Low	159.7 to 161.0	142.21	0.32/5.37	119.3	173.7	107192/107392	0.998
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107406/107406	1.000
October 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	280.8 to 286.8	266.84	0.48/15.15	202.3	349.4	113156/113496	0.997
Ch16 GSM Med	200.7 to 202.8	191.15	0.50/11.29	128.8	245.9	113068/113496	0.996
Ch20 GSM Low	141.9 to 143.4	126.25	0.35/5.17	96.2	153.1	112706/113494	0.993
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113496/113496	1.000
<i>Male</i>							
Ch06 GSM High	318.1 to 319.4	282.54	0.53/17.77	38.2	423.6	110868/113614	0.976
Ch05 GSM Med	225.9 to 226.8	241.27	0.54/15.50	186.8	302.8	113456/113614	0.999
Ch09 GSM Low	161.0 to 161.7	143.81	0.35/5.96	116.8	176.8	113012/113496	0.996
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113614/113614	1.000
November 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	286.8 to 286.8	273.00	0.45/14.54	204.0	343.4	110616/110646	1.000
Ch16 GSM Med	202.8 to 204.8	192.79	0.42/9.55	148.3	241.8	110586/110646	0.999
Ch20 GSM Low	143.4 to 144.8	127.11	0.34/5.01	108.4	153.5	110198/110646	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110646/110646	1.000
<i>Male</i>							
Ch06 GSM High	319.4 to 320.8	287.34	0.48/16.25	225.8	357.9	109554/110664	0.990
Ch05 GSM Med	226.8 to 227.7	241.65	0.50/14.42	165.6	302.3	110582/110664	0.999
Ch09 GSM Low	161.7 to 161.7	143.34	0.34/5.76	114.0	178.7	110244/110658	0.996
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110664/110664	1.000

TABLE I3
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	286.8 to 289.6	274.74	0.47/15.25	222.7	356.4	106174/106198	1.000
Ch16 GSM Med	204.8 to 206.7	194.33	0.40/9.11	158.6	234.0	106188/106198	1.000
Ch20 GSM Low	144.8 to 146.1	129.74	0.31/4.64	109.5	154.2	106008/106114	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106198/106198	1.000
<i>Male</i>							
Ch06 GSM High	320.8 to 322.1	286.88	0.47/15.93	231.5	370.7	104998/106222	0.988
Ch05 GSM Med	227.7 to 228.7	241.29	0.50/14.36	188.7	304.7	106184/106222	1.000
Ch09 GSM Low	161.7 to 162.4	140.55	0.32/5.22	119.5	166.0	105262/106198	0.991
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106222/106222	1.000
January 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	289.6 to 294.5	273.60	0.44/14.08	213.1	345.2	111208/111252	1.000
Ch16 GSM Med	206.7 to 208.2	197.35	0.40/9.31	156.6	238.4	111238/111252	1.000
Ch20 GSM Low	146.1 to 147.3	131.21	0.31/4.77	110.5	164.6	111166/111242	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111252/111252	1.000
<i>Male</i>							
Ch06 GSM High	322.1 to 323.4	286.59	0.45/15.24	232.0	364.4	109952/111258	0.988
Ch05 GSM Med	228.7 to 229.6	241.27	0.53/15.06	187.2	311.8	111150/111258	0.999
Ch09 GSM Low	162.4 to 163.0	141.77	0.32/5.40	114.6	169.4	110336/111254	0.992
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111258/111258	1.000
February 1 to 28, 2014							
<i>Female</i>							
Ch17 GSM High	294.5 to 296.4	278.51	0.43/14.19	224.9	353.9	103924/103948	1.000
Ch16 GSM Med	208.2 to 208.2	198.50	0.40/9.28	165.0	239.6	103946/103948	1.000
Ch20 GSM Low	147.3 to 149.0	132.92	0.31/4.85	112.3	157.0	103880/103948	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103948/103948	1.000
<i>Male</i>							
Ch06 GSM High	323.4 to 324.8	290.93	0.45/15.63	229.9	371.2	103072/103960	0.991
Ch05 GSM Med	229.6 to 230.6	241.40	0.50/14.31	197.7	300.3	103924/103960	1.000
Ch09 GSM Low	163.0 to 163.7	143.34	0.32/5.39	119.8	171.2	103444/103948	0.995
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103960/103960	1.000
March 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	296.4 to 298.0	281.01	0.42/13.93	212.2	352.2	111382/111408	1.000
Ch16 GSM Med	208.2 to 209.6	200.57	0.41/9.73	161.8	246.5	111400/111408	1.000
Ch20 GSM Low	149.0 to 149.7	133.58	0.32/4.93	106.6	154.5	111256/111408	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
<i>Male</i>							
Ch06 GSM High	324.8 to 324.8	290.82	0.48/16.36	223.2	381.3	110096/111408	0.988
Ch05 GSM Med	230.6 to 231.5	242.18	0.53/15.28	194.0	312.6	111336/111408	0.999
Ch09 GSM Low	163.7 to 164.4	144.23	0.32/5.33	122.6	175.8	111024/111408	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000

TABLE I3**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	298.0 to 299.3	279.24	0.44/14.60	206.0	347.1	107100/107288	0.998
Ch16 GSM Med	209.6 to 210.7	202.29	0.43/10.22	153.5	249.1	107228/107288	0.999
Ch20 GSM Low	149.7 to 150.2	134.98	0.32/5.09	111.6	156.3	107172/107286	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107288/107288	1.000
<i>Male</i>							
Ch06 GSM High	324.8 to 326.1	293.70	0.49/17.15	81.1	373.8	106068/107298	0.989
Ch05 GSM Med	231.5 to 231.5	242.46	0.53/15.16	196.6	306.2	107220/107298	0.999
Ch09 GSM Low	164.4 to 164.4	144.09	0.32/5.48	119.7	170.6	106748/107290	0.995
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107298/107298	1.000
May 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	299.3 to 300.4	285.77	0.45/15.19	226.9	363.3	111176/111208	1.000
Ch16 GSM Med	210.7 to 211.7	193.86	0.39/8.95	154.6	239.6	111128/111208	0.999
Ch20 GSM Low	150.2 to 150.2	134.45	0.34/5.30	108.5	158.8	111012/111204	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
<i>Male</i>							
Ch06 GSM High	326.1 to 326.1	296.46	0.46/16.14	237.1	375.6	110684/111208	0.995
Ch05 GSM Med	231.5 to 231.5	207.41	0.47/11.55	162.7	306.7	110450/111208	0.993
Ch09 GSM Low	164.4 to 165.1	153.28	0.43/7.71	123.4	195.4	111094/111208	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
June 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	300.4 to 302.1	288.24	0.47/16.20	235.2	380.3	107530/107542	1.000
Ch16 GSM Med	211.7 to 213.1	200.74	0.42/9.91	138.6	261.5	107508/107542	1.000
Ch20 GSM Low	150.2 to 151.0	134.74	0.33/5.24	95.9	177.0	107376/107534	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107542/107542	1.000
<i>Male</i>							
Ch06 GSM High	326.1 to 327.4	295.01	0.46/16.13	225.9	406.2	106940/107546	0.994
Ch05 GSM Med	231.5 to 231.5	207.94	0.45/11.15	77.9	266.6	106192/107548	0.987
Ch09 GSM Low	163.7 to 165.1	152.64	0.39/7.06	64.5	187.5	107418/107546	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107548/107548	1.000
July 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	302.1 to 302.7	289.35	0.45/15.51	220.3	362.7	111178/111202	1.000
Ch16 GSM Med	213.1 to 213.1	199.95	0.39/9.14	160.5	244.0	111182/111202	1.000
Ch20 GSM Low	151.0 to 151.4	135.12	0.31/4.98	99.5	156.3	111052/111202	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111202/111202	1.000
<i>Male</i>							
Ch06 GSM High	326.1 to 327.4	289.86	0.44/15.18	198.9	378.9	110010/111206	0.989
Ch05 GSM Med	229.6 to 231.5	215.61	0.41/10.50	174.4	265.3	111174/111206	1.000
Ch09 GSM Low	162.4 to 163.7	152.19	0.36/6.35	125.4	187.1	111176/111202	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111206/111206	1.000

TABLE I3
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	302.7 to 303.4	290.91	0.45/15.30	205.3	369.6	108668/108708	1.000
Ch16 GSM Med	213.1 to 213.6	200.65	0.39/9.18	153.8	243.3	108630/108708	0.999
Ch20 GSM Low	151.4 to 151.7	134.02	0.36/5.60	102.4	159.8	107888/108708	0.992
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108708/108708	1.000
<i>Male</i>							
Ch06 GSM High	324.8 to 327.4	290.86	0.45/15.32	231.0	374.5	107704/108716	0.991
Ch05 GSM Med	227.7 to 229.6	215.05	0.41/10.32	175.0	270.0	108704/108716	1.000
Ch09 GSM Low	162.4 to 163.0	153.30	0.37/6.76	128.0	181.8	108702/108708	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108716/108716	1.000
September 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	303.4 to 303.4	294.02	0.46/15.88	194.5	436.8	104160/104176	1.000
Ch16 GSM Med	213.6 to 214.5	204.29	0.41/9.82	167.9	279.2	104168/104176	1.000
Ch20 GSM Low	151.4 to 151.7	138.14	0.33/5.32	111.3	184.1	104052/104176	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
<i>Male</i>							
Ch06 GSM High	324.8 to 324.8	299.88	0.49/17.46	204.7	441.0	103900/104176	0.997
Ch05 GSM Med	227.7 to 227.7	224.63	0.61/16.43	174.7	308.7	104148/104176	1.000
Ch09 GSM Low	161.7 to 162.4	158.10	0.37/6.84	127.6	210.1	104170/104176	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
August 8, 2012 to September 30, 2014							
<i>Female</i>							
Ch17 GSM High	192.6 to 303.4	260.77	0.47/14.52	137.3	436.8	2797766/2800508	0.999
Ch16 GSM Med	136.2 to 214.5	184.16	0.41/8.99	101.6	279.2	2799216/2800510	1.000
Ch20 GSM Low	96.1 to 151.7	124.97	0.40/5.95	71.8	184.1	2794544/2800388	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800510/2800510	1.000
<i>Male</i>							
Ch06 GSM High	192.6 to 327.4	278.30	0.48/15.93	38.2	445.8	2779842/2800838	0.993
Ch05 GSM Med	136.2 to 231.5	224.32	0.65/17.31	77.9	323.0	2797270/2800842	0.999
Ch09 GSM Low	96.3 to 165.1	139.28	0.40/6.61	64.5	210.1	2792562/2800582	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800844/2800844	1.000

TABLE I4**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – H-Field^a**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 8 to 31, 2012							
<i>Female</i>							
Ch17 GSM High	0.65 to 0.75	0.687	0.36/0.029	0.50	0.97	85734/85786	0.999
Ch16 GSM Med	0.46 to 0.53	0.505	0.30/0.018	0.40	0.67	85774/85786	1.000
Ch20 GSM Low	0.33 to 0.38	0.370	0.30/0.013	0.29	0.50	85714/85786	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	85786/85786	1.000
September 1 to 30, 2012							
<i>Female</i>							
Ch17 GSM High	0.51 to 0.71	0.647	0.59/0.046	0.42	0.97	105690/105918	0.998
Ch16 GSM Med	0.36 to 0.51	0.460	0.41/0.022	0.29	0.68	105834/105918	0.999
Ch20 GSM Low	0.26 to 0.36	0.327	0.37/0.014	0.21	0.50	105824/105918	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105918/105918	1.000
<i>Male</i>							
Ch06 GSM High	0.51 to 0.71	0.645	0.41/0.031	0.29	1.17	105800/105918	0.999
Ch05 GSM Med	0.36 to 0.51	0.413	0.37/0.018	0.26	0.66	105892/105918	1.000
Ch09 GSM Low	0.26 to 0.36	0.328	0.38/0.015	0.21	0.59	105716/105918	0.998
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
<i>Female</i>							
Ch17 GSM High	0.51 to 0.62	0.623	0.46/0.034	0.46	0.85	110476/110678	0.998
Ch16 GSM Med	0.36 to 0.44	0.435	0.39/0.020	0.30	0.59	110654/110678	1.000
Ch20 GSM Low	0.26 to 0.31	0.291	0.36/0.012	0.22	0.40	110646/110670	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110678/110678	1.000
<i>Male</i>							
Ch06 GSM High	0.53 to 0.71	0.676	0.42/0.033	0.47	0.90	110666/110702	1.000
Ch05 GSM Med	0.38 to 0.51	0.444	0.35/0.018	0.32	0.61	110702/110702	1.000
Ch09 GSM Low	0.27 to 0.36	0.356	0.35/0.015	0.24	0.46	110620/110678	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
<i>Female</i>							
Ch17 GSM High	0.62 to 0.67	0.695	0.45/0.037	0.56	0.92	107658/107706	1.000
Ch16 GSM Med	0.44 to 0.47	0.496	0.39/0.022	0.42	0.60	107694/107706	1.000
Ch20 GSM Low	0.31 to 0.33	0.338	0.59/0.024	0.27	0.44	107688/107706	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107706/107706	1.000
<i>Male</i>							
Ch06 GSM High	0.72 to 0.78	0.801	0.43/0.041	0.63	1.04	107688/107706	1.000
Ch05 GSM Med	0.52 to 0.55	0.525	0.38/0.023	0.44	0.65	107706/107706	1.000
Ch09 GSM Low	0.36 to 0.39	0.422	0.35/0.017	0.35	0.51	107682/107706	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107706/107706	1.000

^a Ch=chamber (e.g., Ch17=Chamber 17)

TABLE I4
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2012							
<i>Female</i>							
Ch17 GSM High	0.67 to 0.68	0.725	0.44/0.037	0.58	0.90	111152/111216	0.999
Ch16 GSM Med	0.47 to 0.48	0.513	0.41/0.025	0.42	0.63	111202/111216	1.000
Ch20 GSM Low	0.33 to 0.34	0.378	0.34/0.015	0.33	0.46	111172/111216	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111216/111216	1.000
<i>Male</i>							
Ch06 GSM High	0.78 to 0.79	0.837	0.43/0.043	0.69	1.04	111200/111216	1.000
Ch05 GSM Med	0.55 to 0.56	0.535	0.37/0.023	0.45	0.64	111216/111216	1.000
Ch09 GSM Low	0.39 to 0.40	0.439	0.37/0.019	0.36	0.52	111182/111216	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	0.68 to 0.69	0.737	0.44/0.039	0.60	0.95	111146/111236	0.999
Ch16 GSM Med	0.48 to 0.49	0.523	0.38/0.023	0.44	0.64	111226/111238	1.000
Ch20 GSM Low	0.34 to 0.35	0.380	0.34/0.015	0.32	0.47	111214/111234	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111238/111238	1.000
<i>Male</i>							
Ch06 GSM High	0.79 to 0.80	0.842	0.44/0.043	0.70	1.05	111228/111238	1.000
Ch05 GSM Med	0.56 to 0.57	0.540	0.33/0.021	0.45	0.64	111238/111238	1.000
Ch09 GSM Low	0.40 to 0.40	0.438	0.36/0.019	0.37	0.58	111134/111238	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
<i>Female</i>							
Ch17 GSM High	0.69 to 0.70	0.742	0.43/0.037	0.61	0.91	100134/100164	1.000
Ch16 GSM Med	0.49 to 0.50	0.521	0.37/0.023	0.43	0.61	100164/100164	1.000
Ch20 GSM Low	0.35 to 0.35	0.386	0.35/0.016	0.33	0.48	100150/100164	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	100164/100164	1.000
<i>Male</i>							
Ch06 GSM High	0.80 to 0.81	0.870	0.47/0.049	0.72	1.08	100074/100164	0.999
Ch05 GSM Med	0.57 to 0.57	0.535	0.36/0.023	0.45	0.64	100160/100164	1.000
Ch09 GSM Low	0.40 to 0.40	0.441	0.37/0.019	0.37	0.53	100152/100164	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	0.70 to 0.71	0.746	0.41/0.036	0.63	0.93	111390/111408	1.000
Ch16 GSM Med	0.50 to 0.50	0.520	0.37/0.023	0.43	0.63	111408/111408	1.000
Ch20 GSM Low	0.35 to 0.35	0.391	0.34/0.016	0.33	0.46	111396/111408	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch06 GSM High	0.81 to 0.81	0.870	0.46/0.047	0.72	1.06	111368/111408	1.000
Ch05 GSM Med	0.57 to 0.58	0.537	0.36/0.022	0.45	0.64	111402/111408	1.000
Ch09 GSM Low	0.40 to 0.41	0.446	0.36/0.019	0.38	0.54	111396/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000

TABLE I4**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	0.71 to 0.71	0.756	0.43/0.038	0.62	0.91	107424/107438	1.000
Ch16 GSM Med	0.50 to 0.51	0.530	0.39/0.025	0.44	0.62	107438/107438	1.000
Ch20 GSM Low	0.35 to 0.36	0.393	0.34/0.016	0.34	0.46	107430/107438	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107438/107438	1.000
<i>Male</i>							
Ch06 GSM High	0.81 to 0.82	0.876	0.47/0.049	0.70	1.10	107396/107442	1.000
Ch05 GSM Med	0.58 to 0.58	0.541	0.35/0.022	0.46	0.64	107440/107442	1.000
Ch09 GSM Low	0.41 to 0.41	0.449	0.37/0.019	0.39	0.54	107406/107438	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	0.71 to 0.72	0.752	0.44/0.039	0.55	0.94	105516/105524	1.000
Ch16 GSM Med	0.51 to 0.51	0.529	0.40/0.025	0.39	0.62	105522/105524	1.000
Ch20 GSM Low	0.36 to 0.36	0.395	0.33/0.015	0.29	0.46	105514/105524	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105524/105524	1.000
<i>Male</i>							
Ch06 GSM High	0.82 to 0.82	0.891	0.49/0.052	0.68	1.17	105416/105578	0.998
Ch05 GSM Med	0.58 to 0.58	0.535	0.35/0.022	0.32	0.64	105564/105580	1.000
Ch09 GSM Low	0.41 to 0.41	0.454	0.41/0.022	0.36	0.60	105438/105576	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	0.72 to 0.73	0.780	0.46/0.043	0.60	0.95	107056/107074	1.000
Ch16 GSM Med	0.51 to 0.51	0.537	0.39/0.025	0.45	0.65	107070/107074	1.000
Ch20 GSM Low	0.36 to 0.36	0.403	0.35/0.016	0.34	0.47	107056/107074	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107074/107074	1.000
<i>Male</i>							
Ch06 GSM High	0.82 to 0.83	0.907	0.50/0.053	0.71	1.14	106866/107088	0.998
Ch05 GSM Med	0.58 to 0.59	0.540	0.34/0.021	0.46	0.64	107084/107088	1.000
Ch09 GSM Low	0.41 to 0.42	0.455	0.37/0.020	0.37	0.54	107054/107074	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107088/107088	1.000
July 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	0.73 to 0.74	0.780	0.45/0.041	0.65	0.95	111028/111054	1.000
Ch16 GSM Med	0.51 to 0.52	0.545	0.40/0.025	0.46	0.65	111054/111054	1.000
Ch20 GSM Low	0.36 to 0.37	0.408	0.34/0.016	0.34	0.48	111044/111054	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111054/111054	1.000
<i>Male</i>							
Ch06 GSM High	0.83 to 0.84	0.913	0.49/0.053	0.73	1.18	110836/111062	0.998
Ch05 GSM Med	0.59 to 0.59	0.548	0.35/0.023	0.45	0.66	111048/111062	1.000
Ch09 GSM Low	0.42 to 0.42	0.461	0.40/0.022	0.39	0.57	110988/111054	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111062/111062	1.000

TABLE I4
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	0.74 to 0.75	0.789	0.44/0.041	0.64	0.97	111878/111904	1.000
Ch16 GSM Med	0.52 to 0.53	0.557	0.40/0.026	0.46	0.66	111902/111904	1.000
Ch20 GSM Low	0.37 to 0.37	0.412	0.32/0.016	0.36	0.48	111892/111904	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111904/111904	1.000
<i>Male</i>							
Ch06 GSM High	0.84 to 0.84	0.919	0.49/0.053	0.74	1.15	111660/111910	0.998
Ch05 GSM Med	0.59 to 0.60	0.553	0.35/0.023	0.48	0.66	111910/111910	1.000
Ch09 GSM Low	0.42 to 0.42	0.462	0.38/0.021	0.38	0.55	111884/111904	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	0.75 to 0.75	0.788	0.44/0.041	0.63	0.98	107388/107392	1.000
Ch16 GSM Med	0.53 to 0.53	0.561	0.42/0.028	0.45	0.68	107386/107392	1.000
Ch20 GSM Low	0.37 to 0.38	0.411	0.34/0.016	0.35	0.49	107382/107392	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107392/107392	1.000
<i>Male</i>							
Ch06 GSM High	0.84 to 0.84	0.925	0.50/0.055	0.76	1.15	107170/107406	0.998
Ch05 GSM Med	0.60 to 0.60	0.557	0.35/0.023	0.46	0.66	107386/107406	1.000
Ch09 GSM Low	0.42 to 0.43	0.468	0.38/0.021	0.37	0.56	107326/107392	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107406/107406	1.000
October 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	0.75 to 0.76	0.787	0.50/0.047	0.60	1.00	113450/113496	1.000
Ch16 GSM Med	0.53 to 0.54	0.558	0.48/0.032	0.42	0.67	113476/113496	1.000
Ch20 GSM Low	0.38 to 0.38	0.418	0.39/0.019	0.33	0.49	113476/113494	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	113496/113496	1.000
<i>Male</i>							
Ch06 GSM High	0.84 to 0.85	0.931	0.56/0.062	0.60	1.34	113016/113614	0.995
Ch05 GSM Med	0.60 to 0.60	0.560	0.39/0.025	0.46	0.69	113554/113614	0.999
Ch09 GSM Low	0.43 to 0.43	0.471	0.38/0.021	0.39	0.59	113456/113496	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
<i>Female</i>							
Ch17 GSM High	0.76 to 0.76	0.794	0.43/0.041	0.62	0.99	110632/110646	1.000
Ch16 GSM Med	0.54 to 0.54	0.567	0.41/0.028	0.45	0.69	110644/110646	1.000
Ch20 GSM Low	0.38 to 0.38	0.424	0.34/0.017	0.35	0.50	110622/110646	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110646/110646	1.000
<i>Male</i>							
Ch06 GSM High	0.85 to 0.85	0.926	0.49/0.054	0.74	1.17	110468/110664	0.998
Ch05 GSM Med	0.60 to 0.60	0.563	0.35/0.023	0.48	0.68	110664/110664	1.000
Ch09 GSM Low	0.43 to 0.43	0.474	0.35/0.020	0.41	0.58	110642/110658	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110664/110664	1.000

TABLE I4**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2013							
<i>Female</i>							
Ch17 GSM High	0.76 to 0.77	0.797	0.48/0.046	0.63	0.99	106186/106198	1.000
Ch16 GSM Med	0.54 to 0.55	0.573	0.41/0.028	0.47	0.69	106196/106198	1.000
Ch20 GSM Low	0.38 to 0.39	0.425	0.32/0.016	0.38	0.49	106106/106114	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	106198/106198	1.000
<i>Male</i>							
Ch06 GSM High	0.85 to 0.85	0.934	0.48/0.053	0.78	1.19	106006/106222	0.998
Ch05 GSM Med	0.60 to 0.61	0.567	0.37/0.024	0.44	0.67	106114/106222	0.999
Ch09 GSM Low	0.43 to 0.43	0.485	0.34/0.019	0.42	0.57	106114/106198	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	0.77 to 0.78	0.821	0.46/0.045	0.68	1.04	111214/111252	1.000
Ch16 GSM Med	0.55 to 0.55	0.574	0.40/0.027	0.45	0.68	111252/111252	1.000
Ch20 GSM Low	0.39 to 0.39	0.427	0.34/0.017	0.35	0.51	111232/111242	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111252/111252	1.000
<i>Male</i>							
Ch06 GSM High	0.85 to 0.86	0.940	0.48/0.054	0.79	1.18	110934/111258	0.997
Ch05 GSM Med	0.61 to 0.61	0.573	0.38/0.026	0.48	0.68	111256/111258	1.000
Ch09 GSM Low	0.43 to 0.43	0.486	0.36/0.020	0.42	0.58	111152/111254	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
<i>Female</i>							
Ch17 GSM High	0.78 to 0.79	0.828	0.45/0.044	0.69	1.05	103936/103948	1.000
Ch16 GSM Med	0.55 to 0.55	0.575	0.37/0.025	0.49	0.67	103948/103948	1.000
Ch20 GSM Low	0.39 to 0.40	0.433	0.32/0.016	0.37	0.51	103938/103948	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	103948/103948	1.000
<i>Male</i>							
Ch06 GSM High	0.86 to 0.86	0.937	0.48/0.053	0.77	1.17	103800/103960	0.998
Ch05 GSM Med	0.61 to 0.61	0.577	0.38/0.025	0.48	0.67	103956/103960	1.000
Ch09 GSM Low	0.43 to 0.43	0.486	0.35/0.020	0.42	0.56	103898/103948	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	103960/103960	1.000
March 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	0.79 to 0.79	0.830	0.47/0.046	0.66	1.04	111398/111408	1.000
Ch16 GSM Med	0.55 to 0.56	0.574	0.38/0.026	0.45	0.66	111408/111408	1.000
Ch20 GSM Low	0.40 to 0.40	0.435	0.32/0.016	0.36	0.54	111402/111408	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch06 GSM High	0.86 to 0.86	0.936	0.48/0.053	0.78	1.18	111174/111408	0.998
Ch05 GSM Med	0.61 to 0.61	0.574	0.37/0.025	0.49	0.68	111408/111408	1.000
Ch09 GSM Low	0.43 to 0.44	0.486	0.34/0.020	0.41	0.58	111356/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000

TABLE I4
Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	0.79 to 0.79	0.841	0.49/0.049	0.63	1.04	107254/107288	1.000
Ch16 GSM Med	0.56 to 0.56	0.576	0.41/0.027	0.46	0.69	107288/107288	1.000
Ch20 GSM Low	0.40 to 0.40	0.436	0.33/0.017	0.38	0.50	107286/107286	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107288/107288	1.000
<i>Male</i>							
Ch06 GSM High	0.86 to 0.87	0.928	0.46/0.050	0.39	1.17	107206/107298	0.999
Ch05 GSM Med	0.61 to 0.61	0.577	0.37/0.025	0.49	0.68	107298/107298	1.000
Ch09 GSM Low	0.44 to 0.44	0.488	0.36/0.021	0.41	0.57	107212/107290	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	0.79 to 0.80	0.834	0.47/0.047	0.67	1.03	111186/111208	1.000
Ch16 GSM Med	0.56 to 0.56	0.605	0.40/0.029	0.49	0.74	111192/111208	1.000
Ch20 GSM Low	0.40 to 0.40	0.438	0.33/0.017	0.37	0.53	111196/111204	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111208/111208	1.000
<i>Male</i>							
Ch06 GSM High	0.87 to 0.87	0.931	0.47/0.051	0.78	1.23	111120/111208	0.999
Ch05 GSM Med	0.61 to 0.61	0.641	0.45/0.034	0.51	0.77	111208/111208	1.000
Ch09 GSM Low	0.44 to 0.44	0.468	0.37/0.021	0.40	0.56	111204/111208	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111208/111208	1.000
June 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	0.80 to 0.80	0.832	0.46/0.046	0.67	1.03	107526/107542	1.000
Ch16 GSM Med	0.56 to 0.57	0.593	0.40/0.028	0.41	0.69	107516/107542	1.000
Ch20 GSM Low	0.40 to 0.40	0.441	0.34/0.018	0.29	0.52	107508/107534	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107542/107542	1.000
<i>Male</i>							
Ch06 GSM High	0.87 to 0.87	0.940	0.47/0.053	0.67	1.16	107402/107546	0.999
Ch05 GSM Med	0.61 to 0.61	0.652	0.49/0.038	0.24	0.85	107538/107548	1.000
Ch09 GSM Low	0.43 to 0.44	0.466	0.35/0.019	0.21	0.55	107532/107546	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	0.80 to 0.80	0.832	0.48/0.048	0.64	1.05	111188/111202	1.000
Ch16 GSM Med	0.57 to 0.57	0.595	0.40/0.028	0.44	0.70	111200/111202	1.000
Ch20 GSM Low	0.40 to 0.40	0.442	0.33/0.017	0.37	0.52	111186/111202	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111202/111202	1.000
<i>Male</i>							
Ch06 GSM High	0.87 to 0.87	0.947	0.47/0.052	0.79	1.28	110960/111206	0.998
Ch05 GSM Med	0.61 to 0.61	0.646	0.45/0.034	0.46	0.80	111194/111206	1.000
Ch09 GSM Low	0.43 to 0.43	0.463	0.34/0.018	0.36	0.54	111202/111202	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111206/111206	1.000

TABLE I4**Summary of GSM-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
August 1 to 31, 2014							
<i>Female</i>							
Ch17 GSM High	0.80 to 0.81	0.829	0.43/0.042	0.62	1.05	108694/108708	1.000
Ch16 GSM Med	0.57 to 0.57	0.594	0.40/0.028	0.47	0.71	108708/108708	1.000
Ch20 GSM Low	0.40 to 0.40	0.446	0.39/0.020	0.36	0.53	108626/108708	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	108708/108708	1.000
<i>Male</i>							
Ch06 GSM High	0.86 to 0.87	0.946	0.46/0.051	0.78	1.17	108550/108716	0.998
Ch05 GSM Med	0.60 to 0.61	0.639	0.43/0.032	0.53	0.78	108704/108716	1.000
Ch09 GSM Low	0.43 to 0.43	0.454	0.34/0.018	0.39	0.54	108708/108708	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
<i>Female</i>							
Ch17 GSM High	0.81 to 0.81	0.823	0.45/0.043	0.53	1.12	104166/104176	1.000
Ch16 GSM Med	0.57 to 0.57	0.587	0.40/0.028	0.44	0.78	104168/104176	1.000
Ch20 GSM Low	0.40 to 0.40	0.435	0.33/0.017	0.37	0.56	104156/104176	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	104176/104176	1.000
<i>Male</i>							
Ch06 GSM High	0.86 to 0.86	0.917	0.52/0.056	0.53	1.24	104040/104176	0.999
Ch05 GSM Med	0.60 to 0.60	0.607	0.60/0.044	0.48	0.76	104174/104176	1.000
Ch09 GSM Low	0.43 to 0.43	0.435	0.34/0.018	0.33	0.56	104170/104176	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
<i>Female</i>							
Ch17 GSM High	0.51 to 0.81	0.774	0.49/0.045	0.42	1.12	2799438/2800508	1.000
Ch16 GSM Med	0.36 to 0.57	0.547	0.42/0.027	0.29	0.78	2800262/2800510	1.000
Ch20 GSM Low	0.26 to 0.40	0.403	0.41/0.020	0.21	0.56	2799794/2800388	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	2800510/2800510	1.000
<i>Male</i>							
Ch06 GSM High	0.51 to 0.87	0.883	0.49/0.051	0.29	1.34	2796716/2800838	0.999
Ch05 GSM Med	0.36 to 0.61	0.556	0.53/0.035	0.24	0.85	2800538/2800842	1.000
Ch09 GSM Low	0.26 to 0.44	0.449	0.42/0.022	0.21	0.60	2799272/2800582	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	2800844/2800844	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – SAR^a

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 8 to 31, 2012								
<i>Male</i>								
Ch07 IS95 High	237.9 to 358.4	6.00	5.97	0.17/0.04	2.109	8.613	42889/42893	1.000
Ch08 IS95 Med	238.8 to 354.1	3.00	2.99	0.17/0.04	1.077	4.524	42889/42893	1.000
Ch10 IS95 Low	239.3 to 361.1	1.50	1.50	0.17/0.04	0.593	2.223	42889/42893	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	42893/42893	1.000
September 1 to 30, 2012								
<i>Female</i>								
Ch19 IS95 High	55.9 to 303.9	6.00	5.99	0.19/0.04	4.373	9.406	52959/52959	1.000
Ch18 IS95 Med	59.6 to 303.9	3.00	2.99	0.19/0.05	2.018	4.860	52956/52959	1.000
Ch21 IS95 Low	59.3 to 303.9	1.50	1.50	0.20/0.05	1.103	2.524	52958/52959	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52959/52959	1.000
<i>Male</i>								
Ch07 IS95 High	59.2 to 294.9	6.00	5.99	0.21/0.05	4.555	15.811	52958/52959	1.000
Ch08 IS95 Med	63.0 to 306.6	3.00	3.00	0.25/0.06	2.116	10.629	52923/52959	0.999
Ch10 IS95 Low	62.1 to 309.8	1.50	1.50	0.25/0.06	0.882	5.060	52930/52959	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52959/52959	1.000
October 1 to 31, 2012								
<i>Female</i>								
Ch19 IS95 High	114.0 to 197.6	6.00	5.98	0.17/0.04	4.850	8.028	55335/55335	1.000
Ch18 IS95 Med	120.7 to 206.2	3.00	2.99	0.16/0.04	2.437	4.124	55335/55335	1.000
Ch21 IS95 Low	119.7 to 205.3	1.50	1.49	0.18/0.04	1.089	2.099	55335/55335	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55339/55339	1.000
<i>Male</i>								
Ch07 IS95 High	132.0 to 301.0	6.00	5.96	0.18/0.04	4.878	7.243	55351/55351	1.000
Ch08 IS95 Med	141.5 to 312.6	3.00	2.98	0.19/0.04	2.297	4.094	55351/55351	1.000
Ch10 IS95 Low	140.4 to 313.4	1.50	1.49	0.19/0.05	1.118	1.985	55339/55339	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55351/55351	1.000
November 1 to 30, 2012								
<i>Female</i>								
Ch19 IS95 High	204.3 to 239.5	6.00	5.99	0.18/0.04	4.441	9.544	53852/53853	1.000
Ch18 IS95 Med	211.8 to 248.7	3.00	2.98	0.15/0.04	2.556	3.879	53853/53853	1.000
Ch21 IS95 Low	210.9 to 249.2	1.50	1.49	0.16/0.04	1.205	2.100	53853/53853	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53853/53853	1.000
<i>Male</i>								
Ch07 IS95 High	310.9 to 383.3	6.00	5.99	0.17/0.04	5.166	7.287	53853/53853	1.000
Ch08 IS95 Med	323.1 to 396.1	3.00	2.99	0.17/0.04	2.432	4.100	53853/53853	1.000
Ch10 IS95 Low	324.6 to 399.7	1.50	1.49	0.17/0.04	1.251	1.926	53853/53853	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53853/53853	1.000

^a Ch=chamber (e.g., Ch19=Chamber 19)

TABLE I5**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – SAR**

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stddev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
December 1 to 31, 2012								
<i>Female</i>								
Ch19 IS95 High	239.5 to 251.0	6.00	5.96	0.16/0.04	4.865	7.908	55608/55608	1.000
Ch18 IS95 Med	248.7 to 262.3	3.00	2.98	0.15/0.04	2.463	3.959	55608/55608	1.000
Ch21 IS95 Low	249.2 to 261.3	1.50	1.49	0.17/0.04	1.129	2.178	55608/55608	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55608/55608	1.000
<i>Male</i>								
Ch07 IS95 High	383.3 to 412.5	6.00	5.99	0.18/0.04	5.000	7.717	55608/55608	1.000
Ch08 IS95 Med	396.1 to 426.2	3.00	2.99	0.16/0.04	2.506	3.711	55608/55608	1.000
Ch10 IS95 Low	399.7 to 428.6	1.50	1.50	0.17/0.04	1.228	1.934	55608/55608	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55608/55608	1.000
January 1 to 31, 2013								
<i>Female</i>								
Ch19 IS95 High	250.9 to 270.7	6.00	5.98	0.16/0.04	4.937	7.523	55617/55617	1.000
Ch18 IS95 Med	262.3 to 280.6	3.00	3.00	0.28/0.07	1.426	7.073	55545/55617	0.999
Ch21 IS95 Low	261.3 to 277.7	1.50	1.49	0.17/0.04	1.188	2.066	55617/55617	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55619/55619	1.000
<i>Male</i>								
Ch07 IS95 High	412.5 to 452.2	6.00	6.01	0.18/0.04	4.995	7.462	55619/55619	1.000
Ch08 IS95 Med	426.2 to 463.0	3.00	2.98	0.16/0.04	2.592	3.759	55619/55619	1.000
Ch10 IS95 Low	428.6 to 462.1	1.50	1.49	0.17/0.04	1.279	1.862	55619/55619	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55619/55619	1.000
February 1 to 28, 2013								
<i>Female</i>								
Ch19 IS95 High	270.7 to 279.9	6.00	6.00	0.16/0.04	5.200	7.619	50082/50082	1.000
Ch18 IS95 Med	280.6 to 290.1	3.00	2.98	0.14/0.03	2.609	3.700	50082/50082	1.000
Ch21 IS95 Low	277.7 to 287.3	1.50	1.49	0.17/0.04	1.151	2.119	50082/50082	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	50082/50082	1.000
<i>Male</i>								
Ch07 IS95 High	452.2 to 472.2	6.00	5.98	0.18/0.04	5.098	7.180	50082/50082	1.000
Ch08 IS95 Med	463.0 to 491.2	3.00	2.99	0.17/0.04	2.585	3.770	50082/50082	1.000
Ch10 IS95 Low	462.1 to 488.9	1.50	1.49	0.17/0.04	1.253	1.964	50082/50082	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	50082/50082	1.000
March 1 to 31, 2013								
<i>Female</i>								
Ch19 IS95 High	279.9 to 288.7	6.00	5.98	0.16/0.04	4.893	7.540	55704/55704	1.000
Ch18 IS95 Med	290.1 to 300.4	3.00	2.99	0.15/0.03	2.491	3.827	55704/55704	1.000
Ch21 IS95 Low	287.3 to 299.3	1.50	1.48	0.17/0.04	1.183	2.207	55704/55704	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000
<i>Male</i>								
Ch07 IS95 High	472.2 to 495.2	6.00	6.00	0.18/0.04	5.039	7.261	55704/55704	1.000
Ch08 IS95 Med	491.2 to 512.3	3.00	3.00	0.17/0.04	2.563	3.628	55704/55704	1.000
Ch10 IS95 Low	488.9 to 510.6	1.50	1.50	0.17/0.04	1.257	1.872	55704/55704	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
April 1 to 30, 2013								
<i>Female</i>								
Ch19 IS95 High	288.7 to 299.5	6.00	5.95	0.16/0.04	4.951	8.160	53719/53719	1.000
Ch18 IS95 Med	300.4 to 310.6	3.00	2.98	0.15/0.03	2.534	3.787	53719/53719	1.000
Ch21 IS95 Low	299.3 to 308.0	1.50	1.49	0.18/0.04	1.154	2.118	53719/53719	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53719/53719	1.000
<i>Male</i>								
Ch07 IS95 High	495.2 to 511.4	6.00	6.01	0.21/0.05	4.216	9.118	53721/53721	1.000
Ch08 IS95 Med	512.3 to 534.0	3.00	2.99	0.17/0.04	2.515	3.740	53721/53721	1.000
Ch10 IS95 Low	510.6 to 530.6	1.50	1.50	0.17/0.04	1.260	1.985	53719/53719	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53722/53722	1.000
May 1 to 31, 2013								
<i>Female</i>								
Ch19 IS95 High	299.5 to 308.8	6.00	5.98	0.16/0.04	4.581	7.657	52762/52762	1.000
Ch18 IS95 Med	310.6 to 320.6	3.00	3.00	0.15/0.03	2.318	3.891	52762/52762	1.000
Ch21 IS95 Low	308.0 to 315.8	1.50	1.48	0.18/0.04	1.117	2.169	52762/52762	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52762/52762	1.000
<i>Male</i>								
Ch07 IS95 High	511.4 to 530.9	6.00	5.99	0.19/0.04	1.318	7.206	52788/52789	1.000
Ch08 IS95 Med	534.0 to 552.1	3.00	2.99	0.17/0.04	2.358	3.621	52788/52788	1.000
Ch10 IS95 Low	530.6 to 550.3	1.50	1.49	0.18/0.04	1.073	1.934	52762/52762	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52790/52790	1.000
June 1 to 30, 2013								
<i>Female</i>								
Ch19 IS95 High	308.8 to 316.4	6.00	5.97	0.16/0.04	5.110	7.720	53537/53537	1.000
Ch18 IS95 Med	320.6 to 332.1	3.00	3.00	0.15/0.04	2.543	4.014	53537/53537	1.000
Ch21 IS95 Low	315.8 to 328.3	1.50	1.50	0.19/0.05	1.215	2.164	53537/53537	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53537/53537	1.000
<i>Male</i>								
Ch07 IS95 High	530.9 to 547.2	6.00	5.93	0.19/0.04	4.899	7.410	53542/53542	1.000
Ch08 IS95 Med	552.1 to 569.1	3.00	2.95	0.27/0.06	1.304	3.920	53286/53542	0.995
Ch10 IS95 Low	550.3 to 566.3	1.50	1.48	0.17/0.04	1.274	1.860	53537/53537	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53544/53544	1.000
July 1 to 31, 2013								
<i>Female</i>								
Ch19 IS95 High	316.4 to 332.3	6.00	6.02	0.16/0.04	5.171	8.001	55527/55527	1.000
Ch18 IS95 Med	332.1 to 351.4	3.00	2.98	0.14/0.03	2.558	3.934	55527/55527	1.000
Ch21 IS95 Low	328.3 to 346.1	1.50	1.49	0.18/0.04	1.202	2.127	55527/55527	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55527/55527	1.000
<i>Male</i>								
Ch07 IS95 High	547.2 to 577.0	6.00	5.93	0.18/0.04	4.797	7.368	55528/55528	1.000
Ch08 IS95 Med	569.1 to 599.6	3.00	2.97	0.17/0.04	2.531	3.655	55528/55528	1.000
Ch10 IS95 Low	566.3 to 597.9	1.50	1.49	0.18/0.04	1.246	1.938	55527/55527	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55531/55531	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 1 to 31, 2013								
<i>Female</i>								
Ch19 IS95 High	332.3 to 342.8	6.00	5.99	0.16/0.04	5.082	7.893	55952/55952	1.000
Ch18 IS95 Med	351.4 to 360.3	3.00	2.97	0.15/0.03	2.477	3.781	55952/55952	1.000
Ch21 IS95 Low	346.1 to 356.6	1.50	1.48	0.18/0.04	1.168	2.076	55952/55952	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55952/55952	1.000
<i>Male</i>								
Ch07 IS95 High	577.0 to 590.3	6.00	5.98	0.19/0.04	5.067	7.207	55952/55952	1.000
Ch08 IS95 Med	599.6 to 613.4	3.00	2.99	0.18/0.04	2.116	4.677	55952/55952	1.000
Ch10 IS95 Low	597.9 to 610.1	1.50	1.50	0.18/0.04	1.264	1.981	55952/55952	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55955/55955	1.000
September 1 to 30, 2013								
<i>Female</i>								
Ch19 IS95 High	342.8 to 349.6	6.00	5.97	0.17/0.04	5.099	7.749	53696/53696	1.000
Ch18 IS95 Med	360.3 to 366.3	3.00	2.99	0.15/0.03	2.569	3.855	53696/53696	1.000
Ch21 IS95 Low	356.6 to 363.7	1.50	1.48	0.18/0.04	1.149	2.224	53696/53696	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53696/53696	1.000
<i>Male</i>								
Ch07 IS95 High	590.3 to 594.1	6.00	5.98	0.20/0.05	3.989	7.867	53696/53696	1.000
Ch08 IS95 Med	613.4 to 623.8	3.00	2.99	0.16/0.04	2.588	3.626	53696/53696	1.000
Ch10 IS95 Low	610.1 to 620.5	1.50	1.50	0.18/0.04	1.237	1.814	53696/53696	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53703/53703	1.000
October 1 to 31, 2013								
<i>Female</i>								
Ch19 IS95 High	349.6 to 358.7	6.00	5.96	0.18/0.04	3.243	7.477	56746/56747	1.000
Ch18 IS95 Med	366.3 to 377.2	3.00	2.99	0.19/0.04	1.170	3.898	56706/56748	0.999
Ch21 IS95 Low	363.7 to 375.4	1.50	1.49	0.19/0.04	0.762	2.325	56746/56747	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	56748/56748	1.000
<i>Male</i>								
Ch07 IS95 High	594.1 to 603.9	6.00	5.95	0.19/0.05	3.609	8.358	56747/56748	1.000
Ch08 IS95 Med	623.8 to 632.5	3.00	2.97	0.18/0.04	1.622	3.648	56747/56748	1.000
Ch10 IS95 Low	620.5 to 631.5	1.50	1.49	0.19/0.04	0.769	1.850	56747/56748	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	56807/56807	1.000
November 1 to 30, 2013								
<i>Female</i>								
Ch19 IS95 High	358.7 to 366.5	6.00	5.96	0.21/0.05	3.622	7.586	55322/55323	1.000
Ch18 IS95 Med	377.2 to 385.8	3.00	3.00	0.19/0.05	0.910	7.500	55319/55323	1.000
Ch21 IS95 Low	375.4 to 384.9	1.50	1.49	0.23/0.05	0.811	2.262	55322/55323	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55323/55323	1.000
<i>Male</i>								
Ch07 IS95 High	603.9 to 611.0	6.00	5.93	0.21/0.05	2.254	7.558	55329/55330	1.000
Ch08 IS95 Med	632.5 to 640.5	3.00	2.99	0.23/0.05	2.092	4.959	55326/55329	1.000
Ch10 IS95 Low	631.5 to 641.9	1.50	1.50	0.22/0.05	1.059	2.127	55329/55329	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55332/55332	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stddev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
December 1 to 31, 2013								
<i>Female</i>								
Ch19 IS95 High	366.5 to 374.5	6.00	6.00	0.19/0.05	4.178	7.659	53057/53057	1.000
Ch18 IS95 Med	385.8 to 393.5	3.00	2.97	0.16/0.04	2.156	3.649	53099/53099	1.000
Ch21 IS95 Low	384.9 to 395.7	1.50	1.49	0.19/0.05	0.995	2.069	53057/53057	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53099/53099	1.000
<i>Male</i>								
Ch07 IS95 High	611.0 to 614.6	6.00	5.95	0.20/0.05	0.989	7.282	53106/53108	1.000
Ch08 IS95 Med	640.5 to 646.3	3.00	2.96	0.22/0.05	1.270	4.996	53020/53106	0.998
Ch10 IS95 Low	641.9 to 645.9	1.50	1.48	0.20/0.05	1.015	1.773	53099/53099	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53111/53111	1.000
January 1 to 31, 2014								
<i>Female</i>								
Ch19 IS95 High	374.5 to 386.3	6.00	5.98	0.17/0.04	4.565	8.091	55621/55621	1.000
Ch18 IS95 Med	393.5 to 405.6	3.00	2.98	0.14/0.03	2.430	3.687	55626/55626	1.000
Ch21 IS95 Low	395.7 to 408.4	1.50	1.48	0.16/0.04	1.107	2.088	55621/55621	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55626/55626	1.000
<i>Male</i>								
Ch07 IS95 High	614.6 to 625.5	6.00	5.91	0.20/0.05	4.782	7.279	55627/55627	1.000
Ch08 IS95 Med	646.3 to 661.2	3.00	2.97	0.17/0.04	2.235	3.510	55627/55627	1.000
Ch10 IS95 Low	645.9 to 658.9	1.50	1.49	0.18/0.04	1.169	1.845	55627/55627	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55629/55629	1.000
February 1 to 28, 2014								
<i>Female</i>								
Ch19 IS95 High	386.3 to 401.1	6.00	5.95	0.17/0.04	4.403	7.540	51974/51974	1.000
Ch18 IS95 Med	405.6 to 417.2	3.00	2.99	0.15/0.04	2.379	3.683	51974/51974	1.000
Ch21 IS95 Low	408.4 to 423.0	1.50	1.49	0.18/0.04	1.006	2.073	51974/51974	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	51974/51974	1.000
<i>Male</i>								
Ch07 IS95 High	625.5 to 637.7	6.00	5.92	0.19/0.04	4.726	7.209	51974/51974	1.000
Ch08 IS95 Med	661.2 to 671.2	3.00	2.98	0.16/0.04	2.288	3.559	51974/51974	1.000
Ch10 IS95 Low	658.9 to 672.1	1.50	1.50	0.18/0.04	1.137	1.770	51974/51974	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	51980/51980	1.000
March 1 to 31, 2014								
<i>Female</i>								
Ch19 IS95 High	401.1 to 415.9	6.00	5.99	0.16/0.04	5.177	8.398	55704/55704	1.000
Ch18 IS95 Med	417.2 to 426.8	3.00	2.99	0.13/0.03	2.635	3.825	55704/55704	1.000
Ch21 IS95 Low	423.0 to 433.7	1.50	1.48	0.16/0.04	1.217	2.018	55704/55704	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000
<i>Male</i>								
Ch07 IS95 High	637.7 to 640.9	6.00	5.90	0.19/0.05	4.851	7.304	55704/55704	1.000
Ch08 IS95 Med	671.2 to 679.3	3.00	2.99	0.16/0.04	2.519	3.608	55704/55704	1.000
Ch10 IS95 Low	672.1 to 678.5	1.50	1.50	0.17/0.04	1.262	1.804	55704/55704	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55704/55704	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
April 1 to 30, 2014								
<i>Female</i>								
Ch19 IS95 High	415.9 to 430.6	6.00	5.95	0.16/0.04	4.801	7.534	53643/53643	1.000
Ch18 IS95 Med	426.8 to 440.1	3.00	2.98	0.13/0.03	0.964	3.533	53643/53644	1.000
Ch21 IS95 Low	433.7 to 439.9	1.50	1.48	0.16/0.04	1.132	2.033	53643/53643	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53644/53644	1.000
<i>Male</i>								
Ch07 IS95 High	640.9 to 646.7	6.00	5.88	0.19/0.04	1.418	7.181	53646/53647	1.000
Ch08 IS95 Med	679.3 to 681.0	3.00	3.00	0.16/0.04	2.308	3.621	53646/53646	1.000
Ch10 IS95 Low	674.4 to 678.5	1.50	1.50	0.17/0.04	0.894	1.966	53643/53644	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53649/53649	1.000
May 1 to 31, 2014								
<i>Female</i>								
Ch19 IS95 High	430.6 to 435.9	6.00	5.92	0.15/0.04	4.846	7.433	55602/55602	1.000
Ch18 IS95 Med	440.1 to 447.7	3.00	2.98	0.13/0.03	2.611	3.979	55604/55604	1.000
Ch21 IS95 Low	439.9 to 448.6	1.50	1.49	0.17/0.04	1.185	2.158	55602/55602	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55604/55604	1.000
<i>Male</i>								
Ch07 IS95 High	640.5 to 644.4	6.00	5.91	0.17/0.04	4.656	7.371	55604/55604	1.000
Ch08 IS95 Med	681.0 to 682.7	3.00	3.01	0.16/0.04	2.370	3.513	55604/55604	1.000
Ch10 IS95 Low	674.4 to 676.1	1.50	1.49	0.17/0.04	1.210	1.822	55604/55604	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55604/55604	1.000
June 1 to 30, 2014								
<i>Female</i>								
Ch19 IS95 High	435.9 to 447.6	6.00	5.92	0.21/0.05	3.191	10.116	53730/53767	0.999
Ch18 IS95 Med	447.7 to 462.8	3.00	2.98	0.14/0.03	2.589	3.773	53770/53771	1.000
Ch21 IS95 Low	448.6 to 454.5	1.50	1.49	0.16/0.04	1.221	2.005	53767/53767	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53771/53771	1.000
<i>Male</i>								
Ch07 IS95 High	632.7 to 640.5	6.00	5.96	0.18/0.04	4.891	7.698	53773/53773	1.000
Ch08 IS95 Med	682.7 to 691.1	3.00	3.00	0.16/0.04	2.562	3.605	53773/53773	1.000
Ch10 IS95 Low	669.2 to 676.1	1.50	1.50	0.17/0.04	1.255	1.785	53771/53771	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	53774/53774	1.000
July 1 to 31, 2014								
<i>Female</i>								
Ch19 IS95 High	450.1 to 455.1	6.00	5.99	0.16/0.04	5.236	7.748	55601/55601	1.000
Ch18 IS95 Med	466.7 to 473.2	3.00	2.97	0.12/0.03	2.635	3.904	55601/55601	1.000
Ch21 IS95 Low	454.0 to 456.9	1.50	1.49	0.16/0.04	1.221	3.108	55598/55601	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55601/55601	1.000
<i>Male</i>								
Ch07 IS95 High	633.3 to 638.9	6.00	5.97	0.17/0.04	5.044	7.429	55601/55601	1.000
Ch08 IS95 Med	675.8 to 689.3	3.00	3.01	0.15/0.04	2.618	3.671	55601/55601	1.000
Ch10 IS95 Low	661.6 to 671.6	1.50	1.49	0.16/0.04	1.273	1.786	55601/55601	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	55603/55603	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight							
	Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 1 to 31, 2014								
<i>Female</i>								
Ch19 IS95 High	455.1 to 457.3	6.00	5.99	0.17/0.04	4.318	7.906	54354/54354	1.000
Ch18 IS95 Med	470.8 to 473.2	3.00	2.97	0.13/0.03	2.314	3.894	54354/54354	1.000
Ch21 IS95 Low	451.0 to 456.9	1.50	1.49	0.18/0.04	1.046	2.193	54354/54354	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	54354/54354	1.000
<i>Male</i>								
Ch07 IS95 High	622.8 to 633.3	6.00	5.94	0.18/0.04	4.629	7.561	54354/54354	1.000
Ch08 IS95 Med	659.4 to 675.8	3.00	2.98	0.17/0.04	2.209	3.803	54354/54354	1.000
Ch10 IS95 Low	649.7 to 661.6	1.50	1.49	0.16/0.04	1.090	1.855	54354/54354	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	54358/54358	1.000
September 1 to 30, 2014								
<i>Female</i>								
Ch19 IS95 High	454.2 to 455.7	6.00	5.99	0.18/0.04	4.062	10.317	52080/52088	1.000
Ch18 IS95 Med	467.7 to 471.7	3.00	2.97	0.14/0.03	2.195	4.559	52088/52088	1.000
Ch21 IS95 Low	445.6 to 453.5	1.50	1.49	0.16/0.04	0.880	2.711	52086/52088	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52088/52088	1.000
<i>Male</i>								
Ch07 IS95 High	620.1 to 622.8	6.00	5.93	0.16/0.04	4.185	9.677	52087/52088	1.000
Ch08 IS95 Med	648.3 to 659.4	3.00	2.97	0.16/0.04	2.066	4.741	52088/52088	1.000
Ch10 IS95 Low	661.1 to 665.3	1.50	1.48	0.15/0.03	1.038	2.242	52088/52088	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	52088/52088	1.000
August 8, 2012 to September 30, 2014								
<i>Female</i>								
Ch19 IS95 High	55.9 to 457.3	6.00	5.97	0.17/0.04	2.650	10.317	1400143/1400194	1.000
Ch18 IS95 Med	59.6 to 473.2	3.00	2.98	0.16/0.04	0.910	7.500	1400123/1400249	1.000
Ch21 IS95 Low	59.3 to 456.9	1.50	1.49	0.18/0.04	0.653	3.108	1400183/1400194	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	1400255/1400255	1.000
<i>Male</i>								
Ch07 IS95 High	59.2 to 646.7	6.00	5.96	0.19/0.04	0.989	15.811	1400312/1400324	1.000
Ch08 IS95 Med	63.0 to 691.1	3.00	2.98	0.18/0.04	1.077	10.629	1399933/1400319	1.000
Ch10 IS95 Low	62.1 to 678.5	1.50	1.49	0.18/0.04	0.593	5.060	1400227/1400262	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	-/0.00	0.000	0.000	1400422/1400422	1.000

TABLE I6**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field^a**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
August 8 to 31, 2012							
<i>Male</i>							
Ch07 IS95 High	246.10 to 283.80	262.13	0.17/5.16	149.00	313.07	85778/85786	1.000
Ch08 IS95 Med	174.00 to 200.70	185.32	0.17/3.59	106.47	226.74	85778/85786	1.000
Ch10 IS95 Low	123.10 to 143.40	131.39	0.17/2.54	78.99	159.09	85778/85786	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	85786/85786	1.000
September 1 -30, 2012							
<i>Female</i>							
Ch19 IS95 High	192.60 to 269.00	237.99	0.18/4.99	178.91	298.61	105918/105918	1.000
Ch18 IS95 Med	135.90 to 190.20	167.15	0.19/3.68	125.16	210.93	105912/105918	1.000
Ch21 IS95 Low	096.30 to 134.50	118.94	0.19/2.66	87.59	154.09	105916/105918	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
<i>Male</i>							
Ch07 IS95 High	192.20 to 266.50	235.04	0.21/5.62	177.28	375.72	105916/105918	1.000
Ch08 IS95 Med	136.20 to 190.20	165.57	0.23/4.50	127.08	284.85	105854/105918	0.999
Ch10 IS95 Low	096.30 to 134.50	116.96	0.23/3.16	82.06	196.54	105864/105918	0.999
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
<i>Female</i>							
Ch19 IS95 High	193.80 to 228.70	213.07	0.17/4.14	177.92	257.59	110670/110670	1.000
Ch18 IS95 Med	139.30 to 164.60	152.45	0.16/2.82	126.67	185.53	110670/110670	1.000
Ch21 IS95 Low	096.90 to 116.40	107.57	0.18/2.21	85.78	135.10	110670/110670	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110678/110678	1.000
<i>Male</i>							
Ch07 IS95 High	201.30 to 269.00	238.68	0.19/5.16	184.54	291.28	110702/110702	1.000
Ch08 IS95 Med	145.90 to 192.20	171.66	0.19/3.79	127.64	224.82	110702/110702	1.000
Ch10 IS95 Low	103.10 to 135.90	121.30	0.20/2.77	91.15	149.99	110678/110678	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
<i>Female</i>							
Ch19 IS95 High	232.80 to 246.10	240.74	0.18/4.97	207.64	304.40	107704/107706	1.000
Ch18 IS95 Med	167.60 to 177.30	172.96	0.15/3.08	157.53	202.06	107706/107706	1.000
Ch21 IS95 Low	118.50 to 125.40	121.96	0.16/2.33	106.52	148.67	107706/107706	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000
<i>Male</i>							
Ch07 IS95 High	271.80 to 292.20	282.34	0.17/5.61	252.88	319.04	107706/107706	1.000
Ch08 IS95 Med	194.20 to 208.20	201.56	0.17/4.01	176.59	233.27	107706/107706	1.000
Ch10 IS95 Low	137.40 to 147.30	142.68	0.17/2.76	129.54	163.57	107706/107706	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000

^a Ch=chamber (e.g., Ch19=Chamber 19)

TABLE I6**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2012							
<i>Female</i>							
Ch19 IS95 High	246.10 to 254.90	252.54	0.16/4.78	226.29	291.17	111216/111216	1.000
Ch18 IS95 Med	177.30 to 182.70	180.85	0.15/3.18	163.62	209.74	111216/111216	1.000
Ch21 IS95 Low	125.40 to 129.20	127.68	0.17/2.52	110.99	155.56	111216/111216	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
<i>Male</i>							
Ch07 IS95 High	292.20 to 298.00	296.07	0.18/6.03	268.82	336.88	111216/111216	1.000
Ch08 IS95 Med	208.20 to 211.70	210.45	0.16/3.97	191.98	235.34	111216/111216	1.000
Ch10 IS95 Low	147.30 to 149.70	148.95	0.17/2.90	134.40	168.66	111216/111216	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	254.90 to 261.40	258.29	0.16/4.89	236.85	292.38	111234/111234	1.000
Ch18 IS95 Med	182.70 to 186.70	184.88	0.25/5.50	125.89	280.35	111090/111234	0.999
Ch21 IS95 Low	129.20 to 130.70	129.67	0.17/2.50	115.01	151.52	111234/111234	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
<i>Male</i>							
Ch07 IS95 High	298.00 to 302.10	299.87	0.18/6.32	271.01	336.24	111238/111238	1.000
Ch08 IS95 Med	211.70 to 214.00	212.58	0.16/3.99	198.00	240.49	111238/111238	1.000
Ch10 IS95 Low	149.70 to 151.40	150.39	0.18/3.07	138.67	168.35	111238/111238	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
<i>Female</i>							
Ch19 IS95 High	261.40 to 261.40	261.07	0.16/4.80	243.09	294.24	100164/100164	1.000
Ch18 IS95 Med	186.70 to 188.40	187.39	0.14/3.14	174.17	209.88	100164/100164	1.000
Ch21 IS95 Low	130.70 to 132.00	131.14	0.17/2.53	115.67	155.65	100164/100164	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
<i>Male</i>							
Ch07 IS95 High	302.10 to 303.40	302.43	0.18/6.36	278.47	332.36	100164/100164	1.000
Ch08 IS95 Med	214.00 to 215.50	214.43	0.17/4.19	199.44	240.83	100164/100164	1.000
Ch10 IS95 Low	151.40 to 152.00	151.48	0.17/2.95	138.83	173.82	100164/100164	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	261.40 to 264.00	262.72	0.16/4.86	235.81	294.38	111408/111408	1.000
Ch18 IS95 Med	188.40 to 190.20	189.26	0.15/3.30	173.24	214.74	111408/111408	1.000
Ch21 IS95 Low	132.00 to 133.20	132.32	0.17/2.63	117.29	162.11	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch07 IS95 High	303.40 to 304.70	303.64	0.18/6.44	278.43	334.23	111408/111408	1.000
Ch08 IS95 Med	215.50 to 216.80	215.84	0.17/4.25	198.63	238.10	111408/111408	1.000
Ch10 IS95 Low	152.00 to 153.30	152.54	0.18/3.12	139.06	171.02	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000

TABLE I6**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	264.00 to 266.50	265.43	0.16/5.03	239.94	308.04	107438/107438	1.000
Ch18 IS95 Med	190.20 to 192.20	191.35	0.15/3.29	176.86	215.07	107438/107438	1.000
Ch21 IS95 Low	133.20 to 134.50	133.62	0.18/2.84	117.93	159.75	107438/107438	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107438/107438	1.000
<i>Male</i>							
Ch07 IS95 High	304.70 to 306.60	305.73	0.21/7.39	256.68	377.45	107442/107442	1.000
Ch08 IS95 Med	216.80 to 218.30	217.43	0.17/4.28	198.25	242.05	107442/107442	1.000
Ch10 IS95 Low	153.30 to 154.40	153.76	0.17/3.11	141.43	176.11	107438/107438	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	266.50 to 269.00	268.19	0.16/5.04	234.94	303.73	105524/105524	1.000
Ch18 IS95 Med	192.20 to 194.20	193.45	0.15/3.32	170.21	220.53	105524/105524	1.000
Ch21 IS95 Low	134.50 to 135.90	135.05	0.18/2.89	117.44	161.67	105524/105524	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105524/105524	1.000
<i>Male</i>							
Ch07 IS95 High	306.60 to 308.80	307.80	0.19/6.77	144.64	338.21	105576/105578	1.000
Ch08 IS95 Med	218.30 to 220.10	219.28	0.17/4.41	195.00	241.33	105576/105576	1.000
Ch10 IS95 Low	154.40 to 155.60	155.02	0.18/3.21	131.56	176.60	105524/105524	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	269.00 to 271.80	271.14	0.16/5.18	251.16	308.73	107074/107074	1.000
Ch18 IS95 Med	194.20 to 196.40	195.78	0.15/3.44	180.58	226.84	107074/107074	1.000
Ch21 IS95 Low	135.90 to 137.40	136.65	0.19/3.09	123.25	164.46	107074/107074	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107074/107074	1.000
<i>Male</i>							
Ch07 IS95 High	308.80 to 310.00	309.00	0.19/6.87	281.09	345.71	107084/107084	1.000
Ch08 IS95 Med	220.10 to 221.00	219.62	0.30/7.81	145.05	251.44	106572/107084	0.995
Ch10 IS95 Low	155.60 to 156.30	155.84	0.18/3.19	144.54	174.62	107074/107074	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107088/107088	1.000
July 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	271.80 to 277.70	274.50	0.16/5.16	254.24	316.25	111054/111054	1.000
Ch18 IS95 Med	196.40 to 200.70	198.18	0.15/3.36	184.01	227.52	111054/111054	1.000
Ch21 IS95 Low	137.40 to 140.40	138.38	0.18/2.96	124.13	165.13	111054/111054	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111054/111054	1.000
<i>Male</i>							
Ch07 IS95 High	310.00 to 314.00	311.52	0.19/6.78	280.42	347.54	111056/111056	1.000
Ch08 IS95 Med	221.00 to 223.90	222.27	0.17/4.39	205.37	246.82	111056/111056	1.000
Ch10 IS95 Low	156.30 to 158.40	157.32	0.18/3.25	144.08	179.70	111054/111054	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111062/111062	1.000

TABLE I6**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	277.70 to 280.80	277.82	0.16/5.30	255.25	318.10	111904/111904	1.000
Ch18 IS95 Med	200.70 to 202.80	200.65	0.15/3.46	182.96	226.03	111904/111904	1.000
Ch21 IS95 Low	140.40 to 141.90	140.10	0.18/2.95	123.98	165.26	111904/111904	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111904/111904	1.000
<i>Male</i>							
Ch07 IS95 High	314.00 to 316.70	313.45	0.19/6.88	288.22	345.98	111904/111904	1.000
Ch08 IS95 Med	223.90 to 225.90	223.44	0.18/4.66	189.39	281.55	111904/111904	1.000
Ch10 IS95 Low	158.40 to 159.70	158.24	0.18/3.30	145.14	183.22	111904/111904	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	280.80 to 280.80	280.29	0.17/5.48	259.03	319.31	107392/107392	1.000
Ch18 IS95 Med	202.80 to 202.80	202.31	0.15/3.44	187.61	229.81	107392/107392	1.000
Ch21 IS95 Low	141.90 to 143.40	141.65	0.19/3.06	124.62	173.37	107392/107392	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107392/107392	1.000
<i>Male</i>							
Ch07 IS95 High	316.70 to 316.70	315.70	0.21/7.59	257.86	362.09	107392/107392	1.000
Ch08 IS95 Med	225.90 to 226.80	225.46	0.16/4.24	209.57	250.04	107392/107392	1.000
Ch10 IS95 Low	159.70 to 160.40	159.61	0.18/3.40	146.04	175.95	107392/107392	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107406/107406	1.000
October 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	280.80 to 283.80	281.41	0.18/5.86	206.58	313.65	113492/113494	1.000
Ch18 IS95 Med	202.80 to 204.80	203.01	0.20/4.82	126.61	231.08	113412/113496	0.999
Ch21 IS95 Low	143.40 to 144.80	143.35	0.19/3.19	102.19	178.48	113492/113494	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113496/113496	1.000
<i>Male</i>							
Ch07 IS95 High	316.70 to 318.10	315.94	0.20/7.20	245.25	373.22	113494/113496	1.000
Ch08 IS95 Med	226.80 to 227.70	226.35	0.18/4.72	167.22	250.80	113494/113496	1.000
Ch10 IS95 Low	160.40 to 161.00	160.43	0.19/3.55	115.17	178.59	113494/113496	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	283.80 to 286.80	284.53	0.22/7.15	222.74	322.36	110644/110646	1.000
Ch18 IS95 Med	204.80 to 206.70	205.34	0.19/4.62	114.00	327.33	110638/110646	1.000
Ch21 IS95 Low	144.80 to 146.10	144.71	0.23/3.95	107.65	179.75	110644/110646	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110646/110646	1.000
<i>Male</i>							
Ch07 IS95 High	318.10 to 319.40	316.80	0.21/7.75	195.44	357.91	110658/110660	1.000
Ch08 IS95 Med	227.70 to 228.70	227.67	0.23/6.06	191.58	294.96	110652/110658	1.000
Ch10 IS95 Low	161.00 to 161.70	161.12	0.22/4.20	136.29	193.18	110658/110658	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110664/110664	1.000

TABLE I6**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	286.80 to 289.60	287.74	0.20/6.62	239.22	323.90	106114/106114	1.000
Ch18 IS95 Med	206.70 to 208.20	206.92	0.16/3.94	175.52	229.96	106198/106198	1.000
Ch21 IS95 Low	146.10 to 147.30	146.20	0.20/3.39	119.22	173.18	106114/106114	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106198/106198	1.000
<i>Male</i>							
Ch07 IS95 High	319.40 to 319.40	317.36	0.21/7.68	129.45	351.31	106212/106216	1.000
Ch08 IS95 Med	228.70 to 228.70	227.86	0.24/6.43	149.29	296.06	106040/106212	0.998
Ch10 IS95 Low	161.70 to 161.70	161.31	0.20/3.83	133.47	176.37	106198/106198	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	289.60 to 292.20	290.54	0.17/5.74	255.37	335.23	111242/111242	1.000
Ch18 IS95 Med	208.20 to 209.60	208.51	0.14/3.48	188.48	232.86	111252/111252	1.000
Ch21 IS95 Low	147.30 to 148.20	147.24	0.17/2.87	126.66	175.22	111242/111242	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111252/111252	1.000
<i>Male</i>							
Ch07 IS95 High	319.40 to 320.80	318.05	0.20/7.53	284.68	354.25	111254/111254	1.000
Ch08 IS95 Med	228.70 to 230.60	229.35	0.17/4.50	198.04	248.14	111254/111254	1.000
Ch10 IS95 Low	161.70 to 162.40	161.88	0.18/3.41	143.19	179.92	111254/111254	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
<i>Female</i>							
Ch19 IS95 High	292.20 to 296.40	294.48	0.17/5.90	254.46	333.00	103948/103948	1.000
Ch18 IS95 Med	209.60 to 210.70	209.78	0.15/3.68	187.04	232.71	103948/103948	1.000
Ch21 IS95 Low	148.20 to 149.70	148.69	0.18/3.11	122.51	175.92	103948/103948	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103948/103948	1.000
<i>Male</i>							
Ch07 IS95 High	320.80 to 322.10	319.38	0.19/7.15	285.45	352.56	103948/103948	1.000
Ch08 IS95 Med	230.60 to 231.50	230.65	0.16/4.37	202.15	252.10	103948/103948	1.000
Ch10 IS95 Low	162.40 to 163.70	163.15	0.19/3.52	142.50	177.01	103948/103948	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103960/103960	1.000
March 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	296.40 to 298.00	296.72	0.16/5.55	275.93	351.43	111408/111408	1.000
Ch18 IS95 Med	210.70 to 211.70	210.79	0.13/3.27	197.92	238.93	111408/111408	1.000
Ch21 IS95 Low	149.70 to 150.20	149.49	0.16/2.76	134.93	173.56	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch07 IS95 High	322.10 to 323.40	320.78	0.19/7.24	291.74	354.86	111408/111408	1.000
Ch08 IS95 Med	231.50 to 231.50	231.09	0.16/4.30	212.10	253.84	111408/111408	1.000
Ch10 IS95 Low	163.70 to 163.70	163.52	0.17/3.29	150.14	179.49	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000

TABLE I6**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	298.00 to 300.40	299.05	0.16/5.69	269.69	337.86	107286/107286	1.000
Ch18 IS95 Med	211.70 to 213.10	212.13	0.14/3.33	120.89	231.36	107286/107288	1.000
Ch21 IS95 Low	150.20 to 150.20	149.60	0.16/2.80	130.96	175.51	107286/107286	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107288/107288	1.000
<i>Male</i>							
Ch07 IS95 High	323.40 to 323.40	321.14	0.19/7.22	157.72	354.94	107292/107294	1.000
Ch08 IS95 Med	231.50 to 232.50	231.60	0.16/4.24	203.00	254.30	107292/107292	1.000
Ch10 IS95 Low	163.70 to 163.70	163.39	0.17/3.27	126.33	187.37	107286/107288	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	300.40 to 300.40	299.44	0.16/5.43	270.97	335.58	111204/111204	1.000
Ch18 IS95 Med	213.10 to 213.10	212.29	0.13/3.28	198.92	245.53	111208/111208	1.000
Ch21 IS95 Low	150.20 to 150.70	150.10	0.17/2.90	134.01	180.82	111204/111204	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
<i>Male</i>							
Ch07 IS95 High	323.40 to 323.40	321.82	0.18/6.66	285.79	359.61	111208/111208	1.000
Ch08 IS95 Med	232.50 to 232.50	231.90	0.16/4.25	205.74	250.46	111208/111208	1.000
Ch10 IS95 Low	163.70 to 163.70	163.33	0.17/3.31	146.99	180.37	111208/111208	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
June 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	300.40 to 301.30	299.48	0.21/7.42	219.87	391.50	107460/107534	0.999
Ch18 IS95 Med	213.10 to 214.00	213.08	0.15/3.59	26.52	239.11	107540/107542	1.000
Ch21 IS95 Low	150.70 to 151.00	150.40	0.16/2.76	136.04	174.27	107534/107534	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107542/107542	1.000
<i>Male</i>							
Ch07 IS95 High	322.10 to 323.40	320.57	0.18/6.62	290.40	364.33	107546/107546	1.000
Ch08 IS95 Med	232.50 to 233.40	232.22	0.16/4.18	213.90	253.72	107546/107546	1.000
Ch10 IS95 Low	163.00 to 163.70	163.46	0.17/3.26	149.73	178.52	107542/107542	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	302.10 to 302.10	301.20	0.16/5.68	281.66	342.63	111202/111202	1.000
Ch18 IS95 Med	214.00 to 214.50	213.60	0.12/3.09	201.33	245.08	111202/111202	1.000
Ch21 IS95 Low	151.00 to 151.00	150.44	0.16/2.86	136.02	217.00	111196/111202	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111202/111202	1.000
<i>Male</i>							
Ch07 IS95 High	322.10 to 322.10	320.71	0.17/6.37	294.89	357.89	111202/111202	1.000
Ch08 IS95 Med	231.50 to 232.50	231.74	0.15/4.11	216.21	256.04	111202/111202	1.000
Ch10 IS95 Low	163.00 to 163.70	163.06	0.16/3.07	150.77	178.56	111202/111202	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111206/111206	1.000

TABLE I6
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	In Range/ Ratio
August 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	302.10 to 302.10	301.12	0.17/6.05	255.77	346.10	108708/108708	1.000
Ch18 IS95 Med	214.50 to 214.50	213.73	0.14/3.37	188.69	244.76	108708/108708	1.000
Ch21 IS95 Low	151.00 to 151.00	150.40	0.18/3.13	125.88	182.30	108708/108708	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108708/108708	1.000
<i>Male</i>							
Ch07 IS95 High	320.80 to 322.10	319.89	0.18/6.73	282.50	361.05	108708/108708	1.000
Ch08 IS95 Med	229.60 to 231.50	230.28	0.17/4.59	198.60	258.77	108708/108708	1.000
Ch10 IS95 Low	161.70 to 163.00	162.21	0.17/3.17	138.29	181.98	108708/108708	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	302.10 to 302.10	301.19	0.17/6.11	248.08	395.37	104162/104176	1.000
Ch18 IS95 Med	214.00 to 214.50	213.67	0.14/3.50	183.75	264.84	104176/104176	1.000
Ch21 IS95 Low	150.70 to 151.00	150.24	0.17/2.89	115.49	202.67	104172/104176	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
<i>Male</i>							
Ch07 IS95 High	320.80 to 320.80	319.75	0.16/6.09	268.63	408.47	104174/104176	1.000
Ch08 IS95 Med	228.70 to 229.60	228.28	0.16/4.23	190.37	288.41	104174/104176	1.000
Ch10 IS95 Low	163.00 to 163.00	162.73	0.15/2.84	136.12	200.08	104176/104176	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
<i>Female</i>							
Ch19 IS95 High	192.60 to 302.10	275.55	0.17/5.49	167.01	395.37	2800288/2800388	1.000
Ch18 IS95 Med	135.90 to 214.50	197.17	0.16/3.62	26.52	327.33	2800246/2800498	1.000
Ch21 IS95 Low	096.30 to 151.00	138.76	0.18/2.87	82.90	217.00	2800366/2800388	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800510/2800510	1.000
<i>Male</i>							
Ch07 IS95 High	192.20 to 323.40	304.76	0.19/6.72	129.45	408.47	2800624/2800648	1.000
Ch08 IS95 Med	136.20 to 233.40	218.09	0.18/4.65	106.47	296.06	2799872/2800638	1.000
Ch10 IS95 Low	096.30 to 163.70	154.21	0.18/3.24	78.99	200.08	2800458/2800524	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800844/2800844	1.000

TABLE I7**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – E-Field^a**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 8 to 31, 2012							
<i>Male</i>							
Ch07 IS95 High	246.1 to 283.8	249.97	0.24/6.98	136.7	310.0	85778/85786	1.000
Ch08 IS95 Med	174.0 to 200.7	177.30	0.22/4.61	102.5	212.3	85778/85786	1.000
Ch10 IS95 Low	123.1 to 143.4	124.27	0.21/3.05	77.3	151.3	85778/85786	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	85786/85786	1.000
September 1 to 30, 2012							
<i>Female</i>							
Ch19 IS95 High	192.6 to 269.0	239.48	0.24/6.85	181.6	297.4	105910/105918	1.000
Ch18 IS95 Med	135.9 to 190.2	157.66	0.26/4.84	114.7	202.6	105916/105918	1.000
Ch21 IS95 Low	96.3 to 134.5	115.70	0.45/6.14	80.0	157.2	105916/105918	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
<i>Male</i>							
Ch07 IS95 High	192.2 to 266.5	225.11	0.25/6.52	167.5	349.5	105916/105918	1.000
Ch08 IS95 Med	136.2 to 190.2	159.21	0.33/6.15	123.1	288.1	105874/105918	1.000
Ch10 IS95 Low	96.3 to 134.5	113.59	0.28/3.67	81.2	192.6	105910/105918	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
October 1 to 31, 2012							
<i>Female</i>							
Ch19 IS95 High	193.8 to 228.7	216.72	0.28/7.06	179.0	264.8	110670/110670	1.000
Ch18 IS95 Med	139.3 to 164.6	140.84	0.24/3.88	121.3	173.0	110670/110670	1.000
Ch21 IS95 Low	96.9 to 116.4	93.80	0.20/2.16	74.5	118.2	110610/110670	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110678/110678	1.000
<i>Male</i>							
Ch07 IS95 High	201.3 to 269.0	227.53	0.30/8.03	173.4	294.1	110702/110702	1.000
Ch08 IS95 Med	145.9 to 192.2	148.83	0.23/3.99	112.0	203.3	110456/110702	0.998
Ch10 IS95 Low	103.1 to 135.9	118.65	0.31/4.25	88.8	149.3	110678/110678	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110702/110702	1.000
November 1 to 30, 2012							
<i>Female</i>							
Ch19 IS95 High	232.8 to 246.1	245.80	0.29/8.41	206.0	317.3	107678/107706	1.000
Ch18 IS95 Med	167.6 to 177.3	158.36	0.23/4.24	139.2	187.4	107706/107706	1.000
Ch21 IS95 Low	118.5 to 125.4	104.95	0.20/2.39	92.6	131.4	107610/107706	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000
<i>Male</i>							
Ch07 IS95 High	271.8 to 292.2	273.49	0.30/9.67	234.5	337.5	107706/107706	1.000
Ch08 IS95 Med	194.2 to 208.2	174.47	0.22/4.47	152.3	208.3	107632/107706	0.999
Ch10 IS95 Low	137.4 to 147.3	136.25	0.27/4.29	117.0	159.9	107706/107706	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000

^a Ch=chamber (e.g., Ch19=Chamber 19)

TABLE I7**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2012							
<i>Female</i>							
Ch19 IS95 High	246.1 to 254.9	262.13	0.28/8.64	229.3	312.2	111216/111216	1.000
Ch18 IS95 Med	177.3 to 182.7	166.16	0.24/4.65	145.9	193.7	111216/111216	1.000
Ch21 IS95 Low	125.4 to 129.2	110.20	0.20/2.58	94.7	141.2	111082/111216	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
<i>Male</i>							
Ch07 IS95 High	292.2 to 298.0	284.03	0.30/9.99	246.4	335.2	111216/111216	1.000
Ch08 IS95 Med	208.2 to 211.7	183.33	0.25/5.27	162.5	217.1	111120/111216	0.999
Ch10 IS95 Low	147.3 to 149.7	141.52	0.29/4.73	122.4	163.2	111216/111216	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
January 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	254.9 to 261.4	265.88	0.28/8.61	235.6	306.1	111234/111234	1.000
Ch18 IS95 Med	182.7 to 186.7	167.70	0.28/5.41	108.4	259.5	111168/111234	0.999
Ch21 IS95 Low	129.2 to 130.7	111.49	0.20/2.54	96.5	132.2	111104/111234	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
<i>Male</i>							
Ch07 IS95 High	298.0 to 302.1	286.80	0.30/9.97	250.6	333.8	111238/111238	1.000
Ch08 IS95 Med	211.7 to 214.0	190.71	0.25/5.56	167.5	226.3	111236/111238	1.000
Ch10 IS95 Low	149.7 to 151.4	140.71	0.28/4.62	122.8	164.2	111238/111238	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
February 1 to 28, 2013							
<i>Female</i>							
Ch19 IS95 High	261.4 to 261.4	266.69	0.25/7.85	238.4	306.1	100164/100164	1.000
Ch18 IS95 Med	186.7 to 188.4	172.86	0.21/4.27	154.2	198.3	100164/100164	1.000
Ch21 IS95 Low	130.7 to 132.0	113.76	0.20/2.71	99.6	137.0	100046/100164	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
<i>Male</i>							
Ch07 IS95 High	302.1 to 303.4	290.09	0.31/10.66	253.4	342.9	100164/100164	1.000
Ch08 IS95 Med	214.0 to 215.5	198.37	0.27/6.30	173.0	229.6	100164/100164	1.000
Ch10 IS95 Low	151.4 to 152.0	139.08	0.26/4.29	124.1	165.2	100164/100164	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
March 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	261.4 to 264.0	268.11	0.26/8.10	228.7	311.7	111408/111408	1.000
Ch18 IS95 Med	188.4 to 190.2	173.32	0.22/4.37	154.8	200.4	111408/111408	1.000
Ch21 IS95 Low	132.0 to 133.2	114.08	0.20/2.65	100.5	139.9	111270/111408	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
<i>Male</i>							
Ch07 IS95 High	303.4 to 304.7	292.56	0.30/10.38	254.0	342.3	111408/111408	1.000
Ch08 IS95 Med	215.5 to 216.8	201.55	0.25/5.99	178.1	229.9	111408/111408	1.000
Ch10 IS95 Low	152.0 to 153.3	141.64	0.28/4.60	123.3	171.0	111408/111408	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000

TABLE I7**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	264.0 to 266.5	273.12	0.27/8.76	236.1	324.4	107438/107438	1.000
Ch18 IS95 Med	190.2 to 192.2	176.82	0.21/4.26	161.1	204.2	107438/107438	1.000
Ch21 IS95 Low	133.2 to 134.5	114.99	0.21/2.82	98.1	141.4	107112/107438	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107438/107438	1.000
<i>Male</i>							
Ch07 IS95 High	304.7 to 306.6	292.26	0.31/10.78	239.3	382.8	107440/107442	1.000
Ch08 IS95 Med	216.8 to 218.3	202.70	0.26/6.24	181.2	235.9	107442/107442	1.000
Ch10 IS95 Low	153.3 to 154.4	141.39	0.26/4.34	124.8	165.1	107438/107438	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107444/107444	1.000
May 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	266.5 to 269.0	277.77	0.28/9.13	233.4	325.0	105524/105524	1.000
Ch18 IS95 Med	192.2 to 194.2	176.25	0.21/4.38	152.7	205.0	105522/105524	1.000
Ch21 IS95 Low	134.5 to 135.9	116.63	0.23/3.07	98.1	140.2	105172/105524	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105524/105524	1.000
<i>Male</i>							
Ch07 IS95 High	306.6 to 308.8	294.02	0.31/10.66	136.5	344.2	105576/105578	1.000
Ch08 IS95 Med	218.3 to 220.1	205.31	0.28/6.81	180.4	240.7	105576/105576	1.000
Ch10 IS95 Low	154.4 to 155.6	144.69	0.30/5.15	120.2	171.5	105522/105524	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105580/105580	1.000
June 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	269.0 to 271.8	281.70	0.27/8.80	248.5	327.6	107074/107074	1.000
Ch18 IS95 Med	194.2 to 196.4	179.48	0.22/4.60	160.6	214.7	107074/107074	1.000
Ch21 IS95 Low	135.9 to 137.4	117.99	0.22/2.96	105.2	146.6	106772/107074	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107074/107074	1.000
<i>Male</i>							
Ch07 IS95 High	308.8 to 310.0	294.60	0.32/10.97	256.1	354.5	107084/107084	1.000
Ch08 IS95 Med	220.1 to 221.0	196.86	0.37/8.48	117.2	227.5	106566/107084	0.995
Ch10 IS95 Low	155.6 to 156.3	144.10	0.30/5.01	125.1	172.4	107074/107074	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107088/107088	1.000
July 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	271.8 to 277.7	287.00	0.27/8.92	256.8	342.3	111054/111054	1.000
Ch18 IS95 Med	196.4 to 200.7	180.29	0.21/4.33	163.3	209.1	111054/111054	1.000
Ch21 IS95 Low	137.4 to 140.4	120.20	0.20/2.84	106.0	145.9	110840/111054	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111054/111054	1.000
<i>Male</i>							
Ch07 IS95 High	310.0 to 314.0	298.96	0.32/11.20	255.1	350.2	111056/111056	1.000
Ch08 IS95 Med	221.0 to 223.9	200.01	0.25/5.80	176.8	237.8	111052/111056	1.000
Ch10 IS95 Low	156.3 to 158.4	146.34	0.28/4.77	129.0	173.4	111054/111054	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111062/111062	1.000

TABLE I7**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	277.7 to 280.8	285.33	0.28/9.36	249.9	335.8	111904/111904	1.000
Ch18 IS95 Med	200.7 to 202.8	184.94	0.21/4.62	166.9	215.2	111904/111904	1.000
Ch21 IS95 Low	140.4 to 141.9	122.00	0.21/2.95	106.6	148.1	111758/111904	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111904/111904	1.000
<i>Male</i>							
Ch07 IS95 High	314.0 to 316.7	298.56	0.31/10.91	257.5	354.7	111904/111904	1.000
Ch08 IS95 Med	223.9 to 225.9	202.11	0.25/5.95	177.1	254.5	111902/111904	1.000
Ch10 IS95 Low	158.4 to 159.7	145.77	0.30/5.20	125.4	175.7	111898/111904	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111910/111910	1.000
September 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	280.8 to 280.8	290.94	0.28/9.36	258.5	341.8	107392/107392	1.000
Ch18 IS95 Med	202.8 to 202.8	184.37	0.22/4.62	165.6	215.7	107392/107392	1.000
Ch21 IS95 Low	141.9 to 143.4	122.62	0.22/3.14	105.5	152.3	107002/107392	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107392/107392	1.000
<i>Male</i>							
Ch07 IS95 High	316.7 to 316.7	302.84	0.33/11.56	247.3	354.3	107378/107392	1.000
Ch08 IS95 Med	225.9 to 226.8	205.80	0.25/5.96	181.1	237.0	107392/107392	1.000
Ch10 IS95 Low	159.7 to 160.4	148.83	0.30/5.18	128.0	173.1	107392/107392	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107406/107406	1.000
October 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	280.8 to 283.8	291.26	0.28/9.50	218.6	333.1	113492/113494	1.000
Ch18 IS95 Med	202.8 to 204.8	178.28	0.33/6.85	102.4	215.2	113226/113496	0.998
Ch21 IS95 Low	143.4 to 144.8	124.81	0.23/3.29	89.9	151.7	113288/113494	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113496/113496	1.000
<i>Male</i>							
Ch07 IS95 High	316.7 to 318.1	300.02	0.30/10.59	228.1	355.7	113492/113496	1.000
Ch08 IS95 Med	226.8 to 227.7	207.25	0.27/6.42	155.1	243.1	113494/113496	1.000
Ch10 IS95 Low	160.4 to 161.0	148.27	0.29/5.10	100.6	175.3	113482/113496	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113614/113614	1.000
November 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	283.8 to 286.8	296.51	0.31/10.60	230.6	338.6	110646/110646	1.000
Ch18 IS95 Med	204.8 to 206.7	176.06	0.25/5.07	102.4	304.9	109814/110646	0.992
Ch21 IS95 Low	144.8 to 146.1	126.33	0.25/3.76	92.3	158.7	110042/110646	0.995
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110646/110646	1.000
<i>Male</i>							
Ch07 IS95 High	318.1 to 319.4	299.24	0.31/10.89	186.9	352.4	110646/110660	1.000
Ch08 IS95 Med	227.7 to 228.7	204.39	0.28/6.62	171.0	265.5	110464/110658	0.998
Ch10 IS95 Low	161.0 to 161.7	148.39	0.31/5.42	124.1	187.1	110620/110658	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110664/110664	1.000

TABLE I7**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	286.8 to 289.6	297.13	0.29/10.16	241.1	344.8	106114/106114	1.000
Ch18 IS95 Med	206.7 to 208.2	173.06	0.22/4.50	140.4	199.2	104002/106198	0.979
Ch21 IS95 Low	146.1 to 147.3	126.41	0.22/3.28	101.1	150.9	105674/106114	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106198/106198	1.000
<i>Male</i>							
Ch07 IS95 High	319.4 to 319.4	299.40	0.31/10.95	120.2	350.5	106202/106216	1.000
Ch08 IS95 Med	228.7 to 228.7	203.24	0.31/7.46	114.4	258.8	105908/106212	0.997
Ch10 IS95 Low	161.7 to 161.7	149.99	0.32/5.63	122.6	178.0	106162/106198	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106222/106222	1.000
January 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	289.6 to 292.2	299.08	0.27/9.44	261.9	355.6	111242/111242	1.000
Ch18 IS95 Med	208.2 to 209.6	175.32	0.20/4.07	155.1	198.6	110204/111252	0.991
Ch21 IS95 Low	147.3 to 148.2	127.63	0.19/2.87	108.0	153.6	111094/111242	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111252/111252	1.000
<i>Male</i>							
Ch07 IS95 High	319.4 to 320.8	300.75	0.32/11.25	259.2	355.8	111254/111254	1.000
Ch08 IS95 Med	228.7 to 230.6	205.87	0.26/6.19	173.4	239.9	111232/111254	1.000
Ch10 IS95 Low	161.7 to 162.4	149.74	0.30/5.23	128.5	178.9	111254/111254	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111258/111258	1.000
February 1 to 28, 2014							
<i>Female</i>							
Ch19 IS95 High	292.2 to 296.4	306.93	0.27/9.79	257.6	355.4	103948/103948	1.000
Ch18 IS95 Med	209.6 to 210.7	180.40	0.22/4.53	157.1	208.9	103744/103948	0.998
Ch21 IS95 Low	148.2 to 149.7	129.10	0.21/3.15	106.5	160.4	103764/103948	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103948/103948	1.000
<i>Male</i>							
Ch07 IS95 High	320.8 to 322.1	305.52	0.31/11.28	266.4	355.0	103948/103948	1.000
Ch08 IS95 Med	230.6 to 231.5	206.84	0.24/5.88	179.0	237.2	103940/103948	1.000
Ch10 IS95 Low	162.4 to 163.7	153.91	0.32/5.79	129.9	180.0	103946/103948	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103960/103960	1.000
March 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	296.4 to 298.0	306.62	0.26/9.46	271.2	388.9	111406/111408	1.000
Ch18 IS95 Med	210.7 to 211.7	179.74	0.21/4.34	161.9	205.3	111206/111408	0.998
Ch21 IS95 Low	149.7 to 150.2	130.12	0.20/2.98	116.8	156.9	111388/111408	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
<i>Male</i>							
Ch07 IS95 High	322.1 to 323.4	304.23	0.32/11.46	264.8	365.8	111408/111408	1.000
Ch08 IS95 Med	231.5 to 231.5	206.39	0.24/5.85	184.2	235.2	111408/111408	1.000
Ch10 IS95 Low	163.7 to 163.7	153.63	0.30/5.35	133.9	178.2	111408/111408	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000

TABLE I7**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	298.0 to 300.4	306.92	0.27/9.72	264.0	362.4	107286/107286	1.000
Ch18 IS95 Med	211.7 to 213.1	179.35	0.20/4.08	100.9	207.4	106834/107288	0.996
Ch21 IS95 Low	150.2 to 150.2	130.62	0.20/3.09	115.5	156.4	107260/107286	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107288/107288	1.000
<i>Male</i>							
Ch07 IS95 High	323.4 to 323.4	304.21	0.29/10.50	154.1	364.7	107292/107294	1.000
Ch08 IS95 Med	231.5 to 232.5	206.77	0.23/5.63	175.3	235.2	107290/107292	1.000
Ch10 IS95 Low	163.7 to 163.7	153.19	0.28/4.94	117.4	180.0	107284/107288	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107298/107298	1.000
May 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	300.4 to 300.4	310.23	0.26/9.46	278.7	361.9	111204/111204	1.000
Ch18 IS95 Med	213.1 to 213.1	181.02	0.20/4.21	160.2	213.0	110928/111208	0.997
Ch21 IS95 Low	150.2 to 150.7	130.02	0.20/3.01	116.2	160.0	111138/111204	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
<i>Male</i>							
Ch07 IS95 High	323.4 to 323.4	301.79	0.28/9.78	260.5	350.8	111208/111208	1.000
Ch08 IS95 Med	232.5 to 232.5	207.87	0.24/5.84	183.8	239.2	111204/111208	1.000
Ch10 IS95 Low	163.7 to 163.7	156.28	0.33/6.00	133.8	188.0	111208/111208	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
June 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	300.4 to 301.3	305.71	0.35/12.62	201.8	415.4	107452/107534	0.999
Ch18 IS95 Med	213.1 to 214.0	181.65	0.22/4.69	28.4	210.4	107186/107542	0.997
Ch21 IS95 Low	150.7 to 151.0	131.33	0.20/3.01	117.0	158.9	107524/107534	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107542/107542	1.000
<i>Male</i>							
Ch07 IS95 High	322.1 to 323.4	300.92	0.27/9.67	258.0	353.6	107546/107546	1.000
Ch08 IS95 Med	232.5 to 233.4	206.99	0.24/5.68	181.2	235.7	107534/107546	1.000
Ch10 IS95 Low	163.0 to 163.7	155.78	0.29/5.34	134.9	182.9	107542/107542	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107548/107548	1.000
July 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	302.1 to 302.1	309.91	0.27/9.74	274.2	367.1	111202/111202	1.000
Ch18 IS95 Med	214.0 to 214.5	180.86	0.18/3.69	163.2	213.4	110936/111202	0.998
Ch21 IS95 Low	151.0 to 151.0	133.65	0.20/3.14	118.5	198.6	111190/111202	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111202/111202	1.000
<i>Male</i>							
Ch07 IS95 High	322.1 to 322.1	298.86	0.28/9.65	257.8	360.2	111202/111202	1.000
Ch08 IS95 Med	231.5 to 232.5	206.55	0.22/5.37	183.6	236.4	111200/111202	1.000
Ch10 IS95 Low	163.0 to 163.7	153.76	0.28/5.01	134.7	175.0	111202/111202	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111206/111206	1.000

TABLE I7**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – E-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	302.1 to 302.1	301.60	0.26/9.21	252.8	352.3	108708/108708	1.000
Ch18 IS95 Med	214.5 to 214.5	183.36	0.21/4.42	158.5	222.5	108518/108708	0.998
Ch21 IS95 Low	151.0 to 151.0	131.88	0.24/3.69	107.2	164.6	108494/108708	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108708/108708	1.000
<i>Male</i>							
Ch07 IS95 High	320.8 to 322.1	297.12	0.28/9.67	245.4	349.2	108704/108708	1.000
Ch08 IS95 Med	229.6 to 231.5	207.72	0.26/6.27	171.0	246.6	108676/108708	1.000
Ch10 IS95 Low	161.7 to 163.0	153.23	0.27/4.91	127.2	175.6	108706/108708	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108716/108716	1.000
September 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	302.1 to 302.1	300.56	0.26/9.05	247.7	395.6	104162/104176	1.000
Ch18 IS95 Med	214.0 to 214.5	187.43	0.23/4.98	166.7	242.6	104170/104176	1.000
Ch21 IS95 Low	150.7 to 151.0	138.88	0.32/5.13	115.5	199.1	104172/104176	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
<i>Male</i>							
Ch07 IS95 High	320.8 to 320.8	299.37	0.22/7.51	250.7	395.7	104174/104176	1.000
Ch08 IS95 Med	228.7 to 229.6	212.35	0.31/7.69	182.0	276.7	104176/104176	1.000
Ch10 IS95 Low	163.0 to 163.0	155.34	0.25/4.55	129.6	193.5	104176/104176	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
August 8, 2012 to September 30, 2014							
<i>Female</i>							
Ch19 IS95 High	192.6 to 302.1	282.98	0.30/9.79	164.5	415.4	2800246/2800388	1.000
Ch18 IS95 Med	135.9 to 214.5	175.09	0.37/7.72	28.4	304.9	2794118/2800498	0.998
Ch21 IS95 Low	96.3 to 151.0	121.89	0.36/5.19	74.5	199.1	2796040/2800388	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800510/2800510	1.000
<i>Male</i>							
Ch07 IS95 High	192.2 to 323.4	289.61	0.31/10.58	120.2	395.7	2800580/2800648	1.000
Ch08 IS95 Med	136.2 to 233.4	197.37	0.33/7.73	102.5	288.1	2799062/2800638	0.999
Ch10 IS95 Low	96.3 to 163.7	144.71	0.32/5.37	77.3	193.5	2800404/2800524	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800844/2800844	1.000

TABLE I8**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – H-Field^a**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 8 to 31, 2012							
<i>Female</i>							
Ch19 IS95 High	0.65 to 0.75	0.699	0.15/0.012	0.45	0.83	85780/85786	1.000
Ch18 IS95 Med	0.46 to 0.53	0.510	0.17/0.010	0.35	0.62	85780/85786	1.000
Ch21 IS95 Low	0.33 to 0.38	0.342	0.16/0.006	0.22	0.42	85780/85786	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	85786/85786	1.000
September 1 to 30, 2012							
<i>Female</i>							
Ch19 IS95 High	0.51 to 0.71	0.627	0.21/0.016	0.46	0.81	105918/105918	1.000
Ch18 IS95 Med	0.36 to 0.51	0.469	0.31/0.017	0.34	0.58	105798/105918	0.999
Ch21 IS95 Low	0.26 to 0.36	0.324	0.49/0.019	0.24	0.43	105844/105918	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105918/105918	1.000
<i>Male</i>							
Ch07 IS95 High	0.51 to 0.71	0.650	0.25/0.019	0.49	1.07	105906/105918	1.000
Ch08 IS95 Med	0.36 to 0.51	0.456	0.31/0.016	0.34	0.75	105820/105918	0.999
Ch10 IS95 Low	0.26 to 0.36	0.319	0.27/0.010	0.22	0.53	105758/105918	0.998
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
<i>Female</i>							
Ch19 IS95 High	0.51 to 0.61	0.555	0.25/0.016	0.45	0.67	110670/110670	1.000
Ch18 IS95 Med	0.37 to 0.44	0.435	0.25/0.013	0.35	0.53	110668/110670	1.000
Ch21 IS95 Low	0.26 to 0.31	0.322	0.25/0.009	0.26	0.40	110606/110670	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110678/110678	1.000
<i>Male</i>							
Ch07 IS95 High	0.53 to 0.71	0.663	0.23/0.018	0.50	0.82	110702/110702	1.000
Ch08 IS95 Med	0.39 to 0.51	0.516	0.27/0.017	0.38	0.65	110676/110702	1.000
Ch10 IS95 Low	0.27 to 0.36	0.329	0.28/0.011	0.24	0.42	110678/110678	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
<i>Female</i>							
Ch19 IS95 High	0.62 to 0.65	0.625	0.26/0.019	0.54	0.78	107706/107706	1.000
Ch18 IS95 Med	0.45 to 0.47	0.498	0.23/0.013	0.44	0.58	107706/107706	1.000
Ch21 IS95 Low	0.31 to 0.33	0.369	0.24/0.011	0.32	0.45	107640/107706	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107706/107706	1.000
<i>Male</i>							
Ch07 IS95 High	0.72 to 0.78	0.772	0.20/0.018	0.69	0.87	107706/107706	1.000
Ch08 IS95 Med	0.52 to 0.55	0.607	0.27/0.019	0.51	0.71	107688/107706	1.000
Ch10 IS95 Low	0.36 to 0.39	0.396	0.25/0.012	0.35	0.46	107706/107706	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107706/107706	1.000

^a Ch=chamber (e.g., Ch19=Chamber 19)

TABLE I8**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2012							
<i>Female</i>							
Ch19 IS95 High	0.65 to 0.68	0.644	0.24/0.018	0.56	0.74	111216/111216	1.000
Ch18 IS95 Med	0.47 to 0.49	0.519	0.24/0.015	0.45	0.61	111214/111216	1.000
Ch21 IS95 Low	0.33 to 0.34	0.385	0.24/0.011	0.33	0.47	111128/111216	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111216/111216	1.000
<i>Male</i>							
Ch07 IS95 High	0.78 to 0.79	0.817	0.21/0.020	0.74	0.95	111216/111216	1.000
Ch08 IS95 Med	0.55 to 0.56	0.630	0.28/0.020	0.56	0.72	111210/111216	1.000
Ch10 IS95 Low	0.39 to 0.40	0.415	0.26/0.013	0.37	0.48	111216/111216	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	0.68 to 0.69	0.665	0.22/0.017	0.59	0.77	111234/111234	1.000
Ch18 IS95 Med	0.49 to 0.50	0.536	0.31/0.019	0.38	0.81	111018/111234	0.998
Ch21 IS95 Low	0.34 to 0.35	0.392	0.25/0.011	0.35	0.46	111170/111234	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111238/111238	1.000
<i>Male</i>							
Ch07 IS95 High	0.79 to 0.80	0.830	0.21/0.021	0.76	0.92	111238/111238	1.000
Ch08 IS95 Med	0.56 to 0.57	0.622	0.27/0.020	0.57	0.70	111238/111238	1.000
Ch10 IS95 Low	0.40 to 0.40	0.425	0.25/0.013	0.38	0.48	111238/111238	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
<i>Female</i>							
Ch19 IS95 High	0.69 to 0.69	0.678	0.21/0.017	0.60	0.77	100164/100164	1.000
Ch18 IS95 Med	0.50 to 0.50	0.536	0.20/0.012	0.48	0.60	100164/100164	1.000
Ch21 IS95 Low	0.35 to 0.35	0.394	0.24/0.011	0.34	0.47	100088/100164	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	100164/100164	1.000
<i>Male</i>							
Ch07 IS95 High	0.80 to 0.81	0.835	0.23/0.023	0.75	0.92	100164/100164	1.000
Ch08 IS95 Med	0.57 to 0.57	0.611	0.26/0.018	0.55	0.71	100164/100164	1.000
Ch10 IS95 Low	0.40 to 0.40	0.435	0.25/0.013	0.39	0.50	100164/100164	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	0.69 to 0.70	0.683	0.21/0.017	0.61	0.76	111408/111408	1.000
Ch18 IS95 Med	0.50 to 0.51	0.544	0.22/0.014	0.50	0.64	111406/111408	1.000
Ch21 IS95 Low	0.35 to 0.35	0.399	0.24/0.011	0.35	0.49	111336/111408	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch07 IS95 High	0.81 to 0.81	0.835	0.21/0.021	0.76	0.92	111408/111408	1.000
Ch08 IS95 Med	0.57 to 0.58	0.610	0.22/0.016	0.56	0.67	111408/111408	1.000
Ch10 IS95 Low	0.40 to 0.41	0.433	0.25/0.013	0.39	0.50	111408/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000

TABLE I8**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	0.70 to 0.71	0.684	0.24/0.019	0.61	0.78	107438/107438	1.000
Ch18 IS95 Med	0.51 to 0.51	0.546	0.20/0.013	0.49	0.61	107438/107438	1.000
Ch21 IS95 Low	0.35 to 0.36	0.404	0.25/0.012	0.36	0.49	107356/107438	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107438/107438	1.000
<i>Male</i>							
Ch07 IS95 High	0.81 to 0.81	0.847	0.25/0.024	0.73	1.04	107426/107442	1.000
Ch08 IS95 Med	0.58 to 0.58	0.616	0.23/0.016	0.56	0.70	107442/107442	1.000
Ch10 IS95 Low	0.41 to 0.41	0.441	0.25/0.013	0.39	0.51	107438/107438	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	0.71 to 0.71	0.686	0.23/0.019	0.62	0.79	105524/105524	1.000
Ch18 IS95 Med	0.51 to 0.52	0.559	0.22/0.014	0.50	0.65	105520/105524	1.000
Ch21 IS95 Low	0.36 to 0.36	0.407	0.26/0.013	0.35	0.49	105446/105524	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105524/105524	1.000
<i>Male</i>							
Ch07 IS95 High	0.81 to 0.82	0.853	0.23/0.022	0.41	0.95	105576/105578	1.000
Ch08 IS95 Med	0.58 to 0.58	0.619	0.24/0.018	0.55	0.69	105576/105576	1.000
Ch10 IS95 Low	0.41 to 0.41	0.439	0.27/0.014	0.38	0.51	105524/105524	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	0.71 to 0.72	0.691	0.22/0.018	0.63	0.80	107074/107074	1.000
Ch18 IS95 Med	0.52 to 0.52	0.563	0.23/0.015	0.50	0.64	107074/107074	1.000
Ch21 IS95 Low	0.36 to 0.36	0.412	0.27/0.013	0.36	0.50	106962/107074	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107074/107074	1.000
<i>Male</i>							
Ch07 IS95 High	0.82 to 0.82	0.858	0.22/0.022	0.78	0.95	107084/107084	1.000
Ch08 IS95 Med	0.58 to 0.59	0.643	0.34/0.025	0.44	0.74	107080/107084	1.000
Ch10 IS95 Low	0.41 to 0.42	0.445	0.29/0.015	0.40	0.51	107074/107074	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107088/107088	1.000
July 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	0.72 to 0.74	0.695	0.22/0.017	0.63	0.78	111054/111054	1.000
Ch18 IS95 Med	0.52 to 0.53	0.573	0.20/0.013	0.52	0.66	111054/111054	1.000
Ch21 IS95 Low	0.36 to 0.37	0.415	0.25/0.012	0.36	0.50	110982/111054	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111054/111054	1.000
<i>Male</i>							
Ch07 IS95 High	0.82 to 0.83	0.860	0.23/0.023	0.78	0.97	111056/111056	1.000
Ch08 IS95 Med	0.59 to 0.59	0.649	0.23/0.018	0.59	0.73	111056/111056	1.000
Ch10 IS95 Low	0.42 to 0.42	0.446	0.25/0.013	0.40	0.51	111054/111054	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111062/111062	1.000

TABLE I8**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	0.74 to 0.75	0.717	0.23/0.019	0.63	0.82	111904/111904	1.000
Ch18 IS95 Med	0.53 to 0.54	0.574	0.20/0.014	0.52	0.66	111904/111904	1.000
Ch21 IS95 Low	0.37 to 0.38	0.420	0.25/0.012	0.37	0.49	111844/111904	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111904/111904	1.000
<i>Male</i>							
Ch07 IS95 High	0.83 to 0.84	0.871	0.24/0.024	0.80	0.98	111904/111904	1.000
Ch08 IS95 Med	0.59 to 0.60	0.649	0.24/0.019	0.53	0.82	111894/111904	1.000
Ch10 IS95 Low	0.42 to 0.42	0.453	0.28/0.015	0.40	0.53	111904/111904	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	0.75 to 0.75	0.715	0.22/0.018	0.64	0.82	107392/107392	1.000
Ch18 IS95 Med	0.54 to 0.54	0.584	0.21/0.015	0.53	0.67	107392/107392	1.000
Ch21 IS95 Low	0.38 to 0.38	0.426	0.25/0.013	0.37	0.52	107314/107392	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107392/107392	1.000
<i>Male</i>							
Ch07 IS95 High	0.84 to 0.84	0.871	0.24/0.024	0.70	1.01	107392/107392	1.000
Ch08 IS95 Med	0.60 to 0.60	0.650	0.23/0.017	0.59	0.72	107392/107392	1.000
Ch10 IS95 Low	0.42 to 0.43	0.452	0.27/0.014	0.39	0.51	107392/107392	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107406/107406	1.000
October 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	0.75 to 0.75	0.720	0.22/0.019	0.52	0.82	113492/113494	1.000
Ch18 IS95 Med	0.54 to 0.54	0.604	0.33/0.024	0.40	0.69	113448/113496	1.000
Ch21 IS95 Low	0.38 to 0.38	0.429	0.28/0.014	0.30	0.54	113444/113494	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	113496/113496	1.000
<i>Male</i>							
Ch07 IS95 High	0.84 to 0.84	0.880	0.23/0.024	0.70	1.04	113496/113496	1.000
Ch08 IS95 Med	0.60 to 0.60	0.651	0.25/0.019	0.48	0.73	113494/113496	1.000
Ch10 IS95 Low	0.43 to 0.43	0.458	0.26/0.014	0.34	0.52	113496/113496	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
<i>Female</i>							
Ch19 IS95 High	0.75 to 0.76	0.723	0.25/0.021	0.57	0.84	110644/110646	1.000
Ch18 IS95 Med	0.54 to 0.55	0.622	0.25/0.018	0.33	0.93	110634/110646	1.000
Ch21 IS95 Low	0.38 to 0.39	0.433	0.31/0.016	0.33	0.53	110610/110646	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110646/110646	1.000
<i>Male</i>							
Ch07 IS95 High	0.84 to 0.85	0.887	0.25/0.026	0.54	0.99	110658/110660	1.000
Ch08 IS95 Med	0.60 to 0.61	0.666	0.30/0.024	0.55	0.87	110572/110658	0.999
Ch10 IS95 Low	0.43 to 0.43	0.461	0.29/0.016	0.39	0.53	110658/110658	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	110664/110664	1.000

TABLE I8**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
December 1 to 31, 2013							
<i>Female</i>							
Ch19 IS95 High	0.76 to 0.77	0.738	0.23/0.020	0.62	0.84	106114/106114	1.000
Ch18 IS95 Med	0.55 to 0.55	0.639	0.24/0.018	0.55	0.72	106170/106198	1.000
Ch21 IS95 Low	0.39 to 0.39	0.440	0.27/0.014	0.36	0.52	106088/106114	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	106198/106198	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.85	0.889	0.25/0.026	0.34	0.99	106212/106216	1.000
Ch08 IS95 Med	0.61 to 0.61	0.670	0.29/0.023	0.48	0.88	106204/106212	1.000
Ch10 IS95 Low	0.43 to 0.43	0.458	0.29/0.016	0.37	0.52	106198/106198	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	0.77 to 0.78	0.748	0.22/0.019	0.65	0.84	111242/111242	1.000
Ch18 IS95 Med	0.55 to 0.56	0.641	0.21/0.016	0.58	0.71	111246/111252	1.000
Ch21 IS95 Low	0.39 to 0.39	0.443	0.24/0.012	0.37	0.52	111212/111242	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111252/111252	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.85	0.890	0.25/0.026	0.77	1.00	111254/111254	1.000
Ch08 IS95 Med	0.61 to 0.61	0.671	0.24/0.019	0.59	0.74	111254/111254	1.000
Ch10 IS95 Low	0.43 to 0.43	0.462	0.26/0.014	0.40	0.54	111254/111254	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
<i>Female</i>							
Ch19 IS95 High	0.78 to 0.79	0.748	0.21/0.018	0.63	0.84	103948/103948	1.000
Ch18 IS95 Med	0.56 to 0.56	0.634	0.22/0.016	0.55	0.71	103946/103948	1.000
Ch21 IS95 Low	0.39 to 0.40	0.446	0.25/0.013	0.37	0.54	103908/103948	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	103948/103948	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.85	0.884	0.25/0.026	0.79	0.98	103948/103948	1.000
Ch08 IS95 Med	0.61 to 0.61	0.675	0.22/0.018	0.58	0.76	103948/103948	1.000
Ch10 IS95 Low	0.43 to 0.43	0.457	0.27/0.014	0.40	0.52	103948/103948	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	103960/103960	1.000
March 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	0.79 to 0.79	0.761	0.22/0.020	0.68	0.87	111408/111408	1.000
Ch18 IS95 Med	0.56 to 0.56	0.642	0.22/0.016	0.59	0.72	111404/111408	1.000
Ch21 IS95 Low	0.40 to 0.40	0.448	0.23/0.012	0.40	0.52	111388/111408	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.86	0.895	0.25/0.026	0.80	1.02	111408/111408	1.000
Ch08 IS95 Med	0.61 to 0.61	0.678	0.23/0.018	0.62	0.75	111408/111408	1.000
Ch10 IS95 Low	0.43 to 0.43	0.460	0.25/0.013	0.41	0.52	111408/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111408/111408	1.000

TABLE I8
Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
April 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	0.79 to 0.80	0.772	0.22/0.020	0.69	0.89	107286/107286	1.000
Ch18 IS95 Med	0.56 to 0.57	0.650	0.20/0.015	0.37	0.72	107284/107288	1.000
Ch21 IS95 Low	0.40 to 0.40	0.447	0.24/0.012	0.39	0.53	107262/107286	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107288/107288	1.000
<i>Male</i>							
Ch07 IS95 High	0.86 to 0.86	0.897	0.22/0.023	0.43	0.99	107292/107294	1.000
Ch08 IS95 Med	0.61 to 0.62	0.680	0.22/0.017	0.61	0.74	107292/107292	1.000
Ch10 IS95 Low	0.43 to 0.43	0.460	0.24/0.013	0.36	0.53	107288/107288	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	0.80 to 0.80	0.766	0.20/0.018	0.69	0.86	111204/111204	1.000
Ch18 IS95 Med	0.57 to 0.57	0.646	0.21/0.016	0.59	0.74	111204/111208	1.000
Ch21 IS95 Low	0.40 to 0.40	0.451	0.25/0.013	0.40	0.55	111114/111204	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111208/111208	1.000
<i>Male</i>							
Ch07 IS95 High	0.86 to 0.86	0.907	0.24/0.025	0.81	1.01	111208/111208	1.000
Ch08 IS95 Med	0.62 to 0.62	0.679	0.23/0.018	0.60	0.74	111208/111208	1.000
Ch10 IS95 Low	0.43 to 0.43	0.452	0.28/0.015	0.40	0.52	111208/111208	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111208/111208	1.000
June 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	0.80 to 0.80	0.778	0.24/0.022	0.57	1.00	107446/107534	0.999
Ch18 IS95 Med	0.57 to 0.57	0.649	0.21/0.016	0.60	0.72	107540/107542	1.000
Ch21 IS95 Low	0.40 to 0.40	0.450	0.23/0.012	0.40	0.52	107524/107534	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107542/107542	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.86	0.902	0.22/0.023	0.82	1.01	107546/107546	1.000
Ch08 IS95 Med	0.62 to 0.62	0.683	0.24/0.019	0.62	0.75	107546/107546	1.000
Ch10 IS95 Low	0.43 to 0.43	0.454	0.25/0.013	0.41	0.51	107542/107542	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	0.80 to 0.80	0.776	0.22/0.019	0.71	0.88	111202/111202	1.000
Ch18 IS95 Med	0.57 to 0.57	0.653	0.20/0.015	0.61	0.73	111188/111202	1.000
Ch21 IS95 Low	0.40 to 0.40	0.444	0.23/0.012	0.40	0.62	111186/111202	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111202/111202	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.85	0.909	0.23/0.024	0.84	1.03	111202/111202	1.000
Ch08 IS95 Med	0.61 to 0.62	0.681	0.22/0.017	0.61	0.75	111202/111202	1.000
Ch10 IS95 Low	0.43 to 0.43	0.457	0.24/0.013	0.42	0.51	111202/111202	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	111206/111206	1.000

TABLE I8**Summary of CDMA-Modulated Cell Phone RFR Exposure Data in Rats – H-Field**

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2014							
<i>Female</i>							
Ch19 IS95 High	0.80 to 0.80	0.797	0.22/0.020	0.67	0.91	108708/108708	1.000
Ch18 IS95 Med	0.57 to 0.57	0.647	0.22/0.016	0.58	0.73	108702/108708	1.000
Ch21 IS95 Low	0.40 to 0.40	0.448	0.27/0.014	0.38	0.54	108676/108708	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	108708/108708	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.85	0.909	0.24/0.025	0.80	1.01	108708/108708	1.000
Ch08 IS95 Med	0.61 to 0.61	0.671	0.26/0.020	0.57	0.77	108708/108708	1.000
Ch10 IS95 Low	0.43 to 0.43	0.454	0.25/0.013	0.39	0.53	108708/108708	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
<i>Female</i>							
Ch19 IS95 High	0.80 to 0.80	0.801	0.23/0.022	0.65	1.06	104152/104176	1.000
Ch18 IS95 Med	0.57 to 0.57	0.636	0.25/0.018	0.52	0.77	104166/104176	1.000
Ch21 IS95 Low	0.40 to 0.40	0.429	0.33/0.017	0.31	0.55	104170/104176	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	104176/104176	1.000
<i>Male</i>							
Ch07 IS95 High	0.85 to 0.85	0.902	0.20/0.021	0.76	1.12	104170/104176	1.000
Ch08 IS95 Med	0.61 to 0.61	0.648	0.32/0.024	0.53	0.80	104174/104176	1.000
Ch10 IS95 Low	0.43 to 0.43	0.451	0.24/0.013	0.38	0.55	104174/104176	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
<i>Female</i>							
Ch19 IS95 High	0.51 to 0.80	0.711	0.25/0.021	0.45	1.06	2800266/2800388	1.000
Ch18 IS95 Med	0.36 to 0.57	0.582	0.38/0.026	0.33	0.93	2800006/2800498	1.000
Ch21 IS95 Low	0.26 to 0.40	0.413	0.38/0.019	0.22	0.62	2799016/2800388	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	2800510/2800510	1.000
<i>Male</i>							
Ch07 IS95 High	0.51 to 0.86	0.849	0.24/0.024	0.34	1.12	2800596/2800648	1.000
Ch08 IS95 Med	0.36 to 0.62	0.633	0.32/0.024	0.29	0.88	2800368/2800638	1.000
Ch10 IS95 Low	0.26 to 0.43	0.434	0.29/0.015	0.21	0.55	2800350/2800524	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	-/0.000	0.00	0.00	2800844/2800844	1.000

APPENDIX J

INGREDIENTS, NUTRIENT COMPOSITION, AND CONTAMINANT LEVELS IN NTP-2000 RAT AND MOUSE RATION

TABLE J1	Ingredients of NTP-2000 Rat and Mouse Ration.....	J-2
TABLE J2	Vitamins and Minerals in NTP-2000 Rat and Mouse Ration.....	J-2
TABLE J3	Nutrient Composition of NTP-2000 Rat and Mouse Ration.....	J-3
TABLE J4	Contaminant Levels in NTP-2000 Rat and Mouse Ration.....	J-4

TABLE J1
Ingredients of NTP-2000 Rat and Mouse Ration

Ingredients	Percent by Weight
Ground hard winter wheat	22.26
Ground #2 yellow shelled corn	22.18
Wheat middlings	15.0
Oat hulls	8.5
Alfalfa meal (dehydrated, 17% protein)	7.5
Purified cellulose	5.5
Soybean meal (49% protein)	5.0
Fish meal (60% protein)	4.0
Corn oil (without preservatives)	3.0
Soy oil (without preservatives)	3.0
Dried brewer's yeast	1.0
Calcium carbonate (USP)	0.9
Vitamin premix ^a	0.5
Mineral premix ^b	0.5
Calcium phosphate, dibasic (USP)	0.4
Sodium chloride	0.3
Choline chloride (70% choline)	0.26
Methionine	0.2

^a Wheat middlings as carrier

^b Calcium carbonate as carrier

TABLE J2
Vitamins and Minerals in NTP-2000 Rat and Mouse Ration^a

	Amount	Source
Vitamins		
A	4,000 IU	Stabilized vitamin A palmitate or acetate
D	1,000 IU	D-activated animal sterol
K	1.0 mg	Menadione sodium bisulfite complex
α -Tocopheryl acetate	100 IU	
Niacin	23 mg	
Folic acid	1.1 mg	
<i>d</i> -Pantothenic acid	10 mg	<i>d</i> -Calcium pantothenate
Riboflavin	3.3 mg	
Thiamine	4 mg	Thiamine mononitrate
B ₁₂	52 μ g	
Pyridoxine	6.3 mg	Pyridoxine hydrochloride
Biotin	0.2 mg	<i>d</i> -Biotin
Minerals		
Magnesium	514 mg	Magnesium oxide
Iron	35 mg	Iron sulfate
Zinc	12 mg	Zinc oxide
Manganese	10 mg	Manganese oxide
Copper	2.0 mg	Copper sulfate
Iodine	0.2 mg	Calcium iodate
Chromium	0.2 mg	Chromium acetate

^a Per kg of finished product

TABLE J3
Nutrient Composition of NTP-2000 Rat and Mouse Ration

Nutrient	Mean ± Standard Deviation	Range	Number of Samples
Protein (% by weight)	14.4 ± 0.38	13.9 – 15.1	17
Crude fat (% by weight)	8.4 ± 0.37	7.7 – 9.2	17
Crude fiber (% by weight)	9.4 ± 0.41	8.6 – 9.9	17
Ash (% by weight)	4.9 ± 0.13	4.7 – 5.1	17
Amino Acids (% of total diet)			
Arginine	0.794 ± 0.070	0.67 – 0.97	26
Cystine	0.220 ± 0.022	0.15 – 0.25	26
Glycine	0.700 ± 0.038	0.62 – 0.80	26
Histidine	0.344 ± 0.074	0.27 – 0.68	26
Isoleucine	0.546 ± 0.041	0.43 – 0.66	26
Leucine	1.092 ± 0.063	0.96 – 1.24	26
Lysine	0.700 ± 0.110	0.31 – 0.86	26
Methionine	0.408 ± 0.043	0.26 – 0.49	26
Phenylalanine	0.621 ± 0.048	0.47 – 0.72	26
Threonine	0.508 ± 0.040	0.43 – 0.61	26
Tryptophan	0.153 ± 0.027	0.11 – 0.20	26
Tyrosine	0.413 ± 0.063	0.28 – 0.54	26
Valine	0.663 ± 0.040	0.55 – 0.73	26
Essential Fatty Acids (% of total diet)			
Linoleic	3.95 ± 0.242	3.49 – 4.55	26
Linolenic	0.31 ± 0.030	0.21 – 0.35	26
Vitamins			
Vitamin A (IU/kg)	3,899 ± 77	2,820 – 5,450	17
Vitamin D (IU/kg)	1,000 ^a		
α-Tocopherol (ppm)	79.7 ± 20.42	27.0 – 124.0	26
Thiamine (ppm) ^b	11.8 ± 17.85	6.6 – 81.0	17
Riboflavin (ppm)	8.1 ± 2.91	4.20 – 17.50	26
Niacin (ppm)	78.9 ± 8.52	66.4 – 98.2	26
Pantothenic acid (ppm)	26.7 ± 11.63	17.4 – 81.0	26
Pyridoxine (ppm) ^b	9.7 ± 2.09	6.44 – 14.3	26
Folic acid (ppm)	1.59 ± 0.45	1.15 – 3.27	26
Biotin (ppm)	0.32 ± 0.10	0.20 – 0.704	26
Vitamin B ₁₂ (ppb)	51.8 ± 36.6	18.3 – 174.0	26
Choline (ppm) ^b	2,665 ± 631	1,160 – 3,790	26
Minerals			
Calcium (%)	0.903 ± 0.070	0.697 – 1.01	17
Phosphorus (%)	0.553 ± 0.026	0.510 – 0.596	17
Potassium (%)	0.669 ± 0.030	0.626 – 0.733	26
Chloride (%)	0.386 ± 0.037	0.300 – 0.474	26
Sodium (%)	0.193 ± 0.024	0.160 – 0.283	26
Magnesium (%)	0.216 ± 0.057	0.185 – 0.490	26
Sulfur (%)	0.170 ± 0.029	0.116 – 0.209	14
Iron (ppm)	190.5 ± 38.0	135 – 311	26
Manganese (ppm)	50.7 ± 9.72	21.0 – 73.1	26
Zinc (ppm)	58.2 ± 26.89	43.3 – 184.0	26
Copper (ppm)	7.44 ± 2.60	3.21 – 16.3	26
Iodine (ppm)	0.514 ± 0.195	0.158 – 0.972	26
Chromium (ppm)	0.674 ± 0.265	0.330 – 1.380	25
Cobalt (ppm)	0.235 ± 0.157	0.094 – 0.864	24

^a From formulation

^b As hydrochloride (thiamine and pyridoxine) or chloride (choline)

TABLE J4
Contaminant Levels in NTP-2000 Rat and Mouse Ration^a

	Mean ± Standard Deviation ^b	Range	Number of Samples
Contaminants			
Arsenic (ppm)	0.20 ± 0.039	0.14 – 0.28	17
Cadmium (ppm)	0.05 ± 0.004	0.04 – 0.06	17
Lead (ppm)	0.21 ± 0.027	0.07 – 1.19	17
Mercury (ppm)	<0.02		17
Selenium (ppm)	0.17 ± 0.024	0.10 – 0.20	17
Aflatoxins (ppb)	<5.00		17
Nitrate nitrogen (ppm) ^c	18.76 ± 9.49	10.0 – 45.9	17
Nitrite nitrogen (ppm) ^c	0.61		17
BHA (ppm) ^d	<1.0		17
BHT (ppm) ^d	<1.0		17
Aerobic plate count (CFU/g)	<10.0		17
Coliform (MPN/g)	3.0		17
<i>Escherichia coli</i> (MPN/g)	<10		17
<i>Salmonella</i> (MPN/g)	Negative		17
Total nitrosoamines (ppb) ^e	9.2 ± 5.55	0.0 – 19.9	17
<i>N</i> -Nitrosodimethylamine (ppb) ^e	1.3 ± 1.04	0.0 – 3.0	17
<i>N</i> -Nitrosopyrrolidine (ppb) ^e	8.0 ± 5.02	0.0 – 18.6	17
Pesticides (ppm)			
α-BHC	<0.01		17
β-BHC	<0.02		17
γ-BHC	<0.01		17
δ-BHC	<0.01		17
Heptachlor	<0.01		17
Aldrin	<0.01		17
Heptachlor epoxide	<0.01		17
DDE	<0.01		17
DDD	<0.01		17
DDT	<0.01		17
HCB	<0.01		17
Mirex	<0.01		17
Methoxychlor	<0.05		17
Dieldrin	<0.01		17
Endrin	<0.01		17
Telodrin	<0.01		17
Chlordane	<0.05		17
Toxaphene	<0.10		17
Estimated PCBs	<0.20		17
Ronnel	<0.01		17
Ethion	<0.02		17
Trithion	<0.05		17
Diazinon	<0.10		17
Methyl chlorpyrifos	0.16 ± 0.179	0.02 – 0.686	17
Methyl parathion	<0.02		17
Ethyl parathion	<0.02		17
Malathion	0.117 ± 0.140	0.02 – 0.585	17
Endosulfan I	<0.01		17
Endosulfan II	<0.01		17
Endosulfan sulfate	<0.03		17

^a All samples were irradiated. CFU=colony-forming units; MPN=most probable number; BHC=hexachlorocyclohexane or benzene hexachloride

^b For values less than the limit of detection, the detection limit is given as the mean.

^c Sources of contamination: alfalfa, grains, and fish meal

^d Sources of contamination: soy oil and fish meal

^e All values were corrected for percent recovery.

APPENDIX K SENTINEL ANIMAL PROGRAM

METHODS.....	K-2
RESULTS	K-3

SENTINEL ANIMAL PROGRAM

METHODS

Rodents used in the National Toxicology Program are produced in optimally clean facilities to eliminate potential pathogens that may affect study results. The Sentinel Animal Program is part of the periodic monitoring of animal health that occurs during the evaluation of test agents. Under this program, the disease state of the rodents is monitored via sera or feces from extra (sentinel) or dosed animals in the study rooms. The sentinel animals and the study animals are subject to identical environmental conditions. Furthermore, the sentinel animals come from the same production source and weanling groups as the animals used for the studies of test agents.

Blood samples were collected and allowed to clot and the serum was separated. All samples were processed appropriately with serology testing performed by IDEXX BioResearch [formerly Research Animal Diagnostic Laboratory (RADIL), University of Missouri], Columbia, MO for determination of the presence of pathogens. The laboratory methods and agents for which testing was performed are tabulated below; the times at which samples were collected during the studies are also listed.

Blood was collected from five rats per sex per time point except for the following:

- 2-year study, Arrival collection: 10 females
- 2-year study, 4-week collection: 10 females
- 2-year study, 12-month collection: six females
- 2-year study, End of study collection: 10 males and 10 females

<u>Method and Test</u>	<u>Time of Collection</u>
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28-Day Study

Multiplex Fluorescent Immunoassay

H-1 (Toolan's H-1 virus)	Study termination
KRV (Kilham's rat virus)	Study termination
<i>Mycoplasma pulmonis</i>	Study termination
PVM (pneumonia virus of mice)	Study termination
RCV/SDA (rat coronavirus/sialodacryoadenitis virus)	Study termination
RMV (rat minute virus)	Study termination
RPV (rat parvovirus)	Study termination
RTV (rat theilovirus)	Study termination
Sendai	Study termination
TMEV (Theiler's murine encephalomyelitis virus)	Study termination

2-Year Study

Multiplex Fluorescent Immunoassay

H-1	Arrival ^a , 4 weeks ^b , 9 weeks ^c , 6, 12, and 18 months, study termination
KRV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
<i>M. pulmonis</i>	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
PVM	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
RCV/SDA	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
RMV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination

2-Year Study (continued)

Multiplex Fluorescent Immunoassay (continued)

RPV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
RTV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
Sendai	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
TMEV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination

^a Age-matched non-pregnant females^b Time-mated females that did not have a litter^c Offspring, 3 weeks post weaning**RESULTS**

All test results were negative.