DOCUMENTS IN THIS PACKET INCLUDE:

LETTERS FROM CITIZENS TO THE
MAYOR OR CITY COUNCIL

RESPONSES FROM STAFF TO LETTERS FROM CITIZENS

ITEMS FROM MAYOR AND COUNCIL MEMBERS

ITEMS FROM OTHER COMMITTEES AND AGENCIES

ITEMS FROM CITY, COUNTY, STATE, AND REGIONAL AGENCIES

Prepared for: 3/26/2018

Document dates: 3/7/2018 – 3/14/2018

Set 1 of 2

Note: Documents for every category may not have been received for packet reproduction in a given week.

Carnahan, David

From: Jeanne Fleming <jfleming@metricus.net>
Sent: Monday, March 12, 2018 12:58 PM
To: Kniss, Liz (internal)
Cc: Council, City; Architectural Review Board; Clerk, City
Subject: Don't change the existing Wireless ordinance

Dear Mayor Kniss,

Thank you for ensuring that United Neighbors was notified promptly that City Council will next be considering the proposed changes to the Wireless ordinance on Monday, March 19th. We look forward to seeing you and your colleagues at that meeting.

When you meet, I urge you, on behalf of United Neighbors, to vote against changing the Wireless ordinance. As the City Attorney wrote to City Council in her 4/29/15 report regarding what is now the existing ordinance, this ordinance requires—and "require" is her word—that no new or collocated cell towers may be installed in Palo Alto without both an architectural review and conditional use permit findings. Yet now Senior Staff are recommending you allow the Director of Planning, if she so chooses, to bypass ARB and PTC review of these cell towers.

The fact is, now more than ever, both an architectural review and conditional use permit findings are necessary to protect residents from the juggernaut that is the telecom industry as it seeks to install commercial facilities next to our homes. Now is not the time--when applications for new cell towers here are at 150 and counting--to make it easier for Verizon, AT&T et al. to come into Palo Alto's residential neighborhoods and do as they please. In fact, most other California cities are currently in the process of making their Wireless ordinances *tougher*, not weakening them.

Please consider, what sense does it make to dispense with the experience, professionalism and depth of knowledge of the Architectural Review Board when dealing with this critical issue? And what possible virtue can there be in changing the ordinance so that, at the Director's discretion, the *only way* residents would be able make their views heard in a public forum—or to go face-to-face with the telecom companies—would be to pay to appeal proposed cell towers?

Why, in short, should the power to review cell tower applications be taken out of the hands of experts, removed from public forums, and turned over to Senior City Staff? Why should these decisions be made behind closed doors? And where's the transparency Palo Alto residents are entitled to expect from the people running the City?

The fact that the proposed changes to the code do not dictate that the Director bypass the ARB and the PTC cannot disguise the fact that these changes would permit the Director to do so—with no explanation to the public, to City Council or to anyone else..

The changes Staff want you to make to the existing code may be good for them by, for example, allowing them to avoid time-consuming preparations for ARB and PTC hearings. But these changes are bad for residents. And they fly in the face of good governance.

City of Palo Alto | City Clerk's Office | 3/12/2018 4:10 PM

I am aware, of course, that you are generally sympathetic to the interests of developers in Palo Alto. And I am aware, as well, that developers always want more infrastructure, and cell towers are infrastructure. But I'm counting on you to also care about the residents of Palo Alto and to protect them against the invasion of unnecessarily ugly, noisy, bulky and hazardous cell towers that Verizon and others are currently proposing.

Thank you for your attention. Please let me know if you have any questions.

Sincerely,

Jeanne Fleming

Jeanne Fleming, PhD
JFleming@Metricus.net
650-325-5151

Carnahan, David

From: Miriam Sedman <msedman@pacbell.net>
Sent: Monday, March 12, 2018 10:45 PM
To: Council, City
Cc: Clerk, City; Architectural Review Board
Subject: Don't change the existing Wireless ordinance

Dear City Council Members,

I urge you, on behalf of Palo Alto residents, to vote against changing the Wireless ordinance. As the City Attorney wrote to City Council in her 4/29/15 report regarding what is now the existing ordinance, this ordinance requires—and "require" is her word—that no new or collocated cell towers may be installed in Palo Alto without both an architectural review and conditional use permit findings. Yet now Senior Staff are recommending you allow the Director of Planning, if she so chooses, to bypass ARB and PTC review of these cell towers.

The fact is, now more than ever, both an architectural review and conditional use permit findings are necessary to protect residents. On a case-by-case basis it is absolutely necessary for the city to review requests to place cell phone towers in residential neighborhoods.

For example, a sign proposing a Verizon cell phone tower has appeared in the Triple El neighborhood and it makes no sense to install a cell phone tower in this neighborhood for the following reasons.

· The proposed location is central to homes where children, elderly and retirees are at home 24 hours a day and will be exposed to high levels of radiation (lots of data on this!)

· Verizon is optimizing their plan for ease of access and NOT to minimize exposure of radiation to citizens (This is just wrong).

· Our neighborhood has good Verizon coverage right now. Even the Verizon maps show that we are in good shape.

· The houses in the triple El neighborhood that have poles and wires already bear the burden of additional radiation and maintenance for the neighborhood; let alone that the poles and wires are unsightly. Why penalize the same homeowners for phones?

· There are just a whole lot better places to put these poles e.g. NOT in the middle of densely populated neighborhoods.

It is essential that you continue to protect the safety of Palo Alto residents and push back on utility companies who's primary concern is profit and who are not concerned with public safety or neighborhood well being.

Regards,
Miriam Sedman
915 Elsinore Dr.
Palo Alto, CA 94303

Carnahan, David

From: Janet Gu <janetlipingding1120@gmail.com>
Sent: Tuesday, March 13, 2018 12:08 AM
To: Council, City
Cc: Architectural Review Board
Subject: Vote against changing the ordinance please

Dear Mayor Kniss,

Thank you for ensuring that United Neighbors was notified promptly that City Council will next be considering the proposed changes to the Wireless ordinance on Monday, March 19th. We look forward to seeing you and your colleagues at that meeting.

When you meet, I urge you, on behalf of United Neighbors, to vote against changing the Wireless ordinance. As the City Attorney wrote to City Council in her 4/29/15 report regarding what is now the existing ordinance, this ordinance requires—and "require" is her word—that no new or collocated cell towers may be installed in Palo Alto without both an architectural review and conditional use permit findings. Yet now Senior Staff are recommending you allow the Director of Planning, if she so chooses, to bypass ARB and PTC review of these cell towers.

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Please consider, what sense does it make to dispense with the experience, professionalism and depth of knowledge of the Architectural Review Board when dealing with this critical issue? And what possible virtue can there be in changing the ordinance so that, at the Director's discretion, the *only way* residents would be able make their views heard in a public forum—or to go face-to-face with the telecom companies—would be to pay to appeal proposed cell towers?

Why, in short, should the power to review cell tower applications be taken out of the hands of experts, removed from public forums, and turned over to Senior City Staff? Why should these decisions be made behind closed doors? And where's the transparency Palo Alto residents are entitled to expect from the people running the City?

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Thank you for your attention. Please let me know if you have any questions.

Sincerely,

Janet Ding

City of Palo Alto | City Clerk's Office | 3/14/2018 7:52 AM

Carnahan, David

From: Ding Janet <janetding318@gmail.com>
Sent: Tuesday, March 13, 2018 12:10 AM
To: Council, City
Cc: Architectural Review Board
Subject: vote against changing the ordinance!

Dear Mayor Kniss,

Thank you for ensuring that United Neighbors was notified promptly that City Council will next be considering the proposed changes to the Wireless ordinance on Monday, March 19th. We look forward to seeing you and your colleagues at that meeting.

When you meet, I urge you, on behalf of United Neighbors, to vote against changing the Wireless ordinance. As the City Attorney wrote to City Council in her 4/29/15 report regarding what is now the existing ordinance, this ordinance requires—and "require" is her word—that no new or collocated cell towers may be installed in Palo Alto without both an architectural review and conditional use permit findings. Yet now Senior Staff are recommending you allow the Director of Planning, if she so chooses, to bypass ARB and PTC review of these cell towers.

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Thank you for your attention. Please let me know if you have any questions.

Sincerely,

Liping Ding

Carnahan, David

From: Liping Ding <ding.li@husky.neu.edu>
Sent: Tuesday, March 13, 2018 12:12 AM
To: Council, City
Cc: Architectural Review Board
Subject: vote against changing the ordinance!

Dear Mayor Kniss,

Thank you for ensuring that United Neighbors was notified promptly that City Council will next be considering the proposed changes to the Wireless ordinance on Monday, March 19th. We look forward to seeing you and your colleagues at that meeting.

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Thank you for your attention. Please let me know if you have any questions.

Sincerely,

Li Ding

Carnahan, David

From: Alan Cooper <akcooper@pacbell.net>
Sent: Tuesday, March 13, 2018 10:27 AM
To: Council, City; Architectural Review Board; Clerk, City
Subject: Do not change the existing Wireless ordinance

Dear Council Members,

I am a 50 year resident of Palo Alto, and am unhappy about the proliferation of cell towers in our residential neighborhoods. The towers are ugly, noisy and they emit radiation potentially affecting our health. I have a tower within a half block of my house and experience it every day.

I understand you will vote soon to make the process easier for companies to install these devices by allowing the Director of Planning to bypass ARB and PTC review of these cell towers.

I ask that you DO NOT approve the request by City Staff to take the ARB and PTC out of the approval process.

The ARB and PTC are part of the public approval process to assure transparency and allow maximum expert and public input on controversial issues, such as "implanting" cell towers into our neighborhoods.

Thank you,

Alan Cooper
270 Kellogg Ave
Palo Alto

Carnahan, David

From: Claudia <claudiaegriffin@gmail.com>
Sent: Wednesday, March 14, 2018 8:22 AM
To: Council, City
Subject: Wireless ordinance

Hello,
I am writing to you to urge you to vote against the change in the wireless ordinance.

Best regards,
Claudia

City of Palo Alto | City Clerk's Office | 3/14/2018 1:33 PM

Carnahan, David

From: Amrutha Kattamuri <vkattamuri@yahoo.com>
Sent: Wednesday, March 14, 2018 10:19 AM
To: Council, City; Clerk, City; Architectural Review Board; Scharff, Gregory (internal); Atkinson, Rebecca; French, Amy
Subject: A few more resources - Informative links on cell towers and more
Attachments: NTP Partial Finding Report May 2016.pdf

Dear All,

I would like to share a few more resources on this topic.

1. Here is a link to the repository of studies about impacts on health due to (chronic) exposure to radiation from wireless technologies (cell towers, WiFi Cell phones, smart meters, etc)

http://www.bioinitiative.org/

2. In May 2016, the United States National Toxicology Program, part of the U.S. Dept of Health and Human Services, released the results of an animal study on cell phone radiation.
Please see the attachment of the NTP study below.

Thanks,
Amrutha

About the National Toxicology Program (NTP): NTP is a federal, interagency program headquartered at NIEHS, whose goal is to safeguard the public by identifying substances in the environment that may affect human health. For more information about NTP and its programs, visit ntp.niehs.nih.gov.

About the National Institute of Environmental Health Sciences (NIEHS): NIEHS supports research to understand the effects of the environment on human health and is part of NIH. For more information on environmental health topics, visit www.niehs.nih.gov. Subscribe to one or more of the NIEHS news lists (www.niehs.nih.gov/news/newsroom/newslist/index.cfm) to stay current on NIEHS news, press releases, grant opportunities, training, events, and publications.

On Tuesday, March 13, 2018, 9:37:20 AM PDT, Amrutha Kattamuri <vkattamuri@yahoo.com> wrote:

Dear All,

I am sending the following links to articles/information/videos on cell towers for you to go through.

1. Senator Blumenthal, Representative Eshoo Urge FCC to Enforce Exposure Limits for Those Who Work Near Wireless Towers

Senator Blumenthal, Representative Eshoo Urge FCC to Enforce Exposure Limits for Those Who Work Near Wireless Towers

Senator Blumenthal, Representative Eshoo Urge FCC to Enforce Exposure Li...

2. This is the link to the new and latest study on cell towers (**Los Angeles was the study site in the United States)**

Cell Phone Towers are Largest Contributor to Environmental Radiofrequency Radiation

Cell Phone Towers are Largest Contributor to Environmental Radiofrequenc...

Study finds cell towers are largest contributor to environmental radiofrequency radiation exposure.

3. CA Dept of Health issues warnings on Cell Phone usage

California health officials release guidelines on cellphone radiation

California health officials release guidelines on cellphone radiation

State health officials aren't saying that cellphones pose health risks, but "the science is evolving"

4. Julie Watts report on cell towers

ConsumerWatch: 5G Cellphone Towers Signal Renewed Concerns Over Impacts on Health

City of Palo Alto | City Clerk's Office | 3/14/2018 1:33 PM

ConsumerWatch: 5G Cellphone Towers Signal Renewed Concerns Over Impacts ...

Wireless carriers are installing millions of towers across the country to enable the new, faster 5G cellphone te...

5. View four speakers from **1:46:38 through 2:01:15** using this slide presentation.View from **2:01:15 through 2:18:00** for Santa Rosa City Council members' comments.

City of Santa Rosa Council Meeting March 6, 2018

City of Santa Rosa Council Meeting March 6, 2018

City meeting agendas, packets, archives, and live stream are always available at https://santa-rosa.legistar.com

Thanks,
Amrutha

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)

Draft 5-19-2016

Table of Contents

Abstract

The US National Toxicology Program (NTP) has carried out extensive rodent toxicology and carcinogenesis studies of radiofrequency radiation (RFR) at frequencies and modulations used in the US telecommunications industry. This report presents partial findings from these studies. The occurrences of two tumor types in male Harlan Sprague Dawley rats exposed to RFR, malignant gliomas in the brain and schwannomas of the heart, were considered of particular interest, and are the subject of this report. The findings in this report were reviewed by expert peer reviewers selected by the NTP and National Institutes of Health (NIH). These reviews and responses to comments are included as appendices to this report, and revisions to the current document have incorporated and addressed these comments. Supplemental information in the form of 4 additional manuscripts has or will soon be submitted for publication. These manuscripts describe in detail the designs and performance of the RFR exposure system, the dosimetry of RFR exposures in rats and mice, the results to a series of pilot studies establishing the ability of the animals to thermoregulate during RFR exposures, and studies of DNA damage.

Capstick M, Kuster N, Kühn S, Berdinas-Torres V, Wilson P, Ladbury J, Koepke G, McCormick D, Gauger J, Melnick R. A radio frequency radiation reverberation chamber exposure system for rodents

Yijian G, Capstick M, McCormick D, Gauger J, Horn T, Wilson P, Melnick RL and Kuster N. Life time dosimetric assessment for mice and rats exposed to cell phone radiation

Wyde ME, Horn TL, Capstick M, Ladbury J, Koepke G, Wilson P, Stout MD, Kuster N, Melnick R, Bucher JR, and McCormick D. Pilot studies of the National Toxicology Program's cell phone radiofrequency radiation reverberation chamber exposure system

Smith-Roe SL, Wyde ME, Stout MD, Winters J, Hobbs CA, Shepard KG, Green A, Kissling GE, Tice RR, Bucher JR, Witt KL. Evaluation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure

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)

Draft 5-19-2016

SUMMARY

The purpose of this communication is to report partial findings from a series of radiofrequency radiation (RFR) cancer studies in rats performed under the auspices of the U.S. National Toxicology Program (NTP).[1] This report contains peer-reviewed, neoplastic and hyperplastic findings only in the brain and heart of Hsd:Sprague Dawley® SD® (HSD) rats exposed to RFR starting *in utero* and continuing throughout their lifetimes. These studies found low incidences of malignant gliomas in the brain and schwannomas in the heart of male rats exposed to RFR of the two types [Code Division Multiple Access (CDMA) and Global System for Mobile Communications (GSM)] currently used in U.S. wireless networks. Potentially preneoplastic lesions were also observed in the brain and heart of male rats exposed to RFR.

The review of partial study data in this report has been prompted by several factors. Given the widespread global usage of mobile communications among users of all ages, even a very small increase in the incidence of disease resulting from exposure to RFR could have broad implications for public health. There is a high level of public and media interest regarding the safety of cell phone RFR and the specific results of these NTP studies.

[1] NTP is a federal, interagency program, headquartered at the National Institute of Environmental Health Sciences, part of the National Institutes of Health, whose goal is to safeguard the public by identifying substances in the environment that may affect human health. For more information about NTP and its programs, visit http://ntp.niehs.nih.gov

Lastly, the tumors in the brain and heart observed at low incidence in male rats exposed to GSM- and CDMA-modulated cell phone RFR in this study are of a type similar to tumors observed in some epidemiology studies of cell phone use. These findings appear to support the International Agency for Research on Cancer (IARC) conclusions regarding the possible carcinogenic potential of RFR.[2]

It is important to note that this document reviews only the findings from the brain and heart and is not a complete report of all findings from the NTP's studies. Additional data from these studies in Hsd:Sprague Dawley® SD® (Harlan) rats and similar studies conducted in B6C3F$_1$/N mice are currently under evaluation and will be reported together with the current findings in two forthcoming NTP Technical Reports.

STUDY RATIONALE

Cell phones and other commonly used wireless communication devices transmit information via non-ionizing radiofrequency radiation (RFR). In 2013, IARC classified RFR as a *possible human carcinogen* based on "limited evidence" of an association between exposure to RFR from heavy wireless phone use and glioma and acoustic neuroma (vestibular schwannoma) in human epidemiology studies, and "limited evidence" for the carcinogenicity of RFR in experimental animals. While ionizing radiation is a well-accepted human carcinogen, theoretical arguments have been raised against the possibility that non-ionizing radiation could induce tumors (discussed in IARC, 2013). Given the extremely large number of people who use wireless

[2] IARC (International Agency for Research on Cancer). 2013. Non-Ionizing Radiation, Part 2: Radiofrequency Electromagnetic Fields. IARC Monogr Eval Carcinog Risk Hum 102. Available: http://monographs.iarc.fr/ENG/Monographs/vol102/mono102.pdf [accessed 26 May 2016].

communication devices, even a very small increase in the incidence of disease resulting from exposure to the RFR generated by those devices could have broad implications for public health.

DESCRIPTION OF THE NTP CELL PHONE RFR PROGRAM

RFR emitted by wireless communication devices, especially cell phones, was nominated to the NTP for toxicology and carcinogenicity testing by the U.S. Food and Drug Administration (FDA). After careful and extensive evaluation of the published literature and experimental efforts already underway at that time, the NTP concluded that additional studies were warranted to more clearly define any potential health hazard to the U.S. population. Due to the technical complexity of such studies, NTP staff worked closely with RFR experts from the National Institute of Standards and Technology (NIST). With support from NTP, engineers at NIST evaluated various types of RFR exposure systems and demonstrated the feasibility of using a specially designed exposure system (reverberation chambers), which resolved the inherent limitations identified in existing systems.

In general, NTP chronic toxicity/carcinogenicity studies expose laboratory rodents to a test article for up to 2 years and are designed to determine the potential for the agent tested to be hazardous and/or carcinogenic to humans.[3] For cell phone RFR, a program of study was designed to evaluate potential, long-term health effects of whole-body exposures. These studies were conducted in three phases: (1) a series of pilot studies to establish field strengths that do not raise body temperature, (2) 28-day toxicology studies in rodents exposed to various low-level field strengths, and (3) chronic toxicology and carcinogenicity studies. The studies were carried out under contract at IIT Research Institute (IITRI) in Chicago, IL following Good Laboratory

[3] Specifications for the Conduct of NTP Studies, http://ntp.niehs.nih.gov/ntp/test_info/finalntp_toxcarspecsjan2011.pdf

Practices (GLP). These studies were conducted in rats and mice using a reverberation chamber exposure system with two signal modulations [Code Division Multiple Access (CDMA) and Global System for Mobile Communications (GSM)] at two frequencies (900 MHz for rats and 1900 MHz for mice), the modulations and frequency bands that are primarily used in the United States.

STUDY DESIGN

Hsd:Sprague Dawley® SD® (Harlan) rats were housed in custom-designed reverberation chambers and exposed to cell phone RFR. Experimentally generated 900 MHz RF fields with either GSM or CDMA modulation were continuously monitored in real-time during all exposure periods via RF sensors located in each exposure chamber that recorded RF field strength (V/m). Animal exposure levels are reported as whole-body specific absorption rate (SAR), a biological measure of exposure based on the deposition of RF energy into an absorbing organism or tissue. SAR is defined as the energy (watts) absorbed per mass of tissue (kilograms). Rats were exposed to GSM- or CDMA-modulated RFR at 900 MHz with whole-body SAR exposures of 0, 1.5, 3, or 6 W/kg. RFR field strengths were frequently adjusted based on changes in body weight to maintain desired SAR levels.

Exposures to RFR were initiated *in utero* beginning with the exposure of pregnant dams (approximately 11-14 weeks of age) on Gestation Day (GD) 5 and continuing throughout gestation. After birth, dams and pups were exposed in the same cage through weaning on postnatal day (PND) 21, at which point the dams were removed and exposure of 90 pups per sex per group was continued for up to 106 weeks. Pups remained group-housed from PND 21 until they were individually housed on PND 35. Control and treatment groups were populated with no

more than 3 pups per sex per litter. All RF exposures were conducted over a period of approximately 18 hours using a continuous cycle of 10 minutes on (exposed) and 10 minutes off (not exposed), for a total daily exposure time of approximately 9 hours a day, 7 days/week. A single, common group of unexposed animals of each sex served as controls for both RFR modulations. These control rats were housed in identical reverberation chambers with no RF signal generation. Each chamber was maintained on a 12-hour light/dark cycle, within a temperature range of 72 ± 3°F, a humidity range of 50 ± 15%, and with at least 10 air changes per hour. Throughout the studies, all animals were provided *ad libitum* access to feed and water.

RESULTS

In pregnant rats exposed to 900 MHz GSM- or CDMA-modulated RFR, no exposure-related effects were observed on the percent of dams littering, litter size, or sex distribution of pups. Small, exposure-level-dependent reductions (up to 7%) in body weights compared to controls were observed throughout gestation and lactation in dams exposed to GSM- or CDMA-modulated RFR. In the offspring, litter weights tended to be lower (up to 9%) in GSM and CDMA RFR-exposed groups compared to controls. Early in the lactation phase, body weights of male and female pups were lower in the GSM-modulated (8%) and CDMA-modulated (15%) RFR groups at 6 W/kg compared to controls. These weight differences in the offspring for both GSM and CDMA exposures tended to lessen (6% and 10%, respectively) as lactation progressed. Throughout the remainder of the chronic study, no RFR exposure-related effects on body weights were observed in male and female rats exposed to RFR, regardless of modulation.

At the end of the 2-year study, survival was lower in the control group of males than in all groups of male rats exposed to GSM-modulated RFR. Survival was also slightly lower in control females than in females exposed to 1.5 or 6 W/kg GSM-modulated RFR. In rats exposed to CDMA-modulated RFR, survival was higher in all groups of exposed males and in the 6 W/kg females compared to controls.

Brain

A low incidence of malignant gliomas and glial cell hyperplasia was observed in all groups of male rats exposed to GSM-modulated RFR (Table 1). In males exposed to CDMA-modulated RFR, a low incidence of malignant gliomas occurred in rats exposed to 6 W/kg (Table 1). Glial cell hyperplasia was also observed in the 1.5 W/kg and 6 W/kg CDMA-modulated exposure groups. No malignant gliomas or glial cell hyperplasias were observed in controls. There was not a statistically significant difference between the incidences of lesions in exposed male rats compared to control males for any of the GSM- or CDMA-modulated RFR groups. However, there was a statistically significant positive trend in the incidence of malignant glioma ($p < 0.05$) for CDMA-modulated RFR exposures.

Table 1. Incidence of brain lesions in male Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Malignant glioma [† ‡]	0[*]	3 (3.3%)	3 (3.3%)	2 (2.2%)	0	0	3 (3.3%)
Glial cell hyperplasia	0	2 (2.2%)	3 (3.3%)	1 (1.1%)	2 (2.2%)	0	2 (2.2%)

[§] Data presented as number of animals per group with lesions (percentage of animals per group with lesions).
[*] Significant SAR-dependent trend for CDMA exposures by poly-6 ($p < 0.05$). See appendix B
[†] Poly-6 survival adjusted rates for malignant gliomas were 0/53.48 in controls; GSM: 3/67.96 (4.4%), 3/72.10 (4.2%), and 2/72.65 (2.8%) in the 1.5, 3, and 6 W/kg groups, respectively; CDMA: 0/65.94, 0/73.08, and 3/57.49 (5.2%) for the 1.5, 3, and 6 W/kg groups, respectively.
[‡] Historical control incidence in NTP studies: 11/550 (2.0%), range 0-8%

In females exposed to GSM-modulated RFR, a malignant glioma was observed in a single rat exposed to 6 W/kg, and glial cell hyperplasia was observed in a single rat exposed to 3 W/kg (Table 2). In females exposed to CDMA-modulated RFR, malignant gliomas were observed in two rats exposed to 1.5 W/kg. Glial cell hyperplasia was observed in one female in each of the CDMA-modulation exposure groups (1.5, 3, and 6 W/kg). There was no glial cell hyperplasia or malignant glioma observed in any of the control females. Detailed descriptions of the malignant gliomas and glial cell hyperplasias are presented in Appendix C.

Table 2. Incidence of brain lesions in female Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Malignant glioma [‡]	0	0	0	1 (1.1%)	2 (2.2%)	0	0
Glial cell hyperplasia	0	0	1 (1.1%)	0	1 (1.1%)	1 (1.1%)	1 (1.1%)

[§] Data presented as number of animals per group with lesions (percentage of animals per group with lesions).
[‡] Historical control incidence in NTP studies: 1/540 (0.18%), range 0-2%

Heart

Cardiac schwannomas were observed in male rats in all exposed groups of both GSM- and CDMA-modulated RFR, while none were observed in controls (Table 3). For both modulations (GSM and CDMA), there was a significant positive trend in the incidence of schwannomas of the heart with respect to exposure SAR. Additionally, the incidence of schwannomas in the 6 W/kg males was significantly higher in CDMA-modulated RFR-exposed males compared to controls. The incidence of schwannomas in the 6 W/kg GSM-modulated RFR-exposed males was higher, but not statistically significant ($p = 0.052$) compared to controls. Schwann cell

hyperplasia of the heart was also observed in three males exposed to 6 W/kg CDMA-modulated RFR. In the GSM-modulation exposure groups, a single incidence of Schwann cell hyperplasia was observed in a 1.5 W/kg male.

Table 3. Incidence of heart lesions in male Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated cell phone RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Schwannoma [† ‡]	0[*]	2 (2.2%)	1 (1.1%)	5 (5.5%)	2 (2.2%)	3 (3.3%)	6 (6.6%)[**]
Schwann cell hyperplasia	0	1 (1.1%)	0	2 (2.2%)	0	0	3 (3.3%)

[§] Data presented as number of animals per group with lesions (percentage of animals per group with lesions).
[*] Significant SAR level-dependent trend for GSM and CDMA by poly-3 ($p < 0.05$). See appendix B
[**] Significantly higher than controls by poly-3 ($p < 0.05$)
[†] Poly-3 survival adjusted rates for schwannomas were 0/65.47 in controls; GSM: 2/74.87 (2.7%), 1/77.89 (1.3%), and 5/78.48 (6.4%) in the 1.5, 3, and 6 W/kg groups, respectively; CDMA: 2/74.05 (2.7%), 3/78.67 (3.8%), and 6/67.94 (8.8%) for the 1.5, 3, and 6 W/kg groups, respectively.
[‡] Historical control incidence in NTP studies: 9/699 (1.3%) range 0-6%

In females, schwannomas of the heart were also observed at 3 W/kg GSM-modulated RFR and 1.5 and 6 W/kg CDMA-modulated RFR. Schwann cell hyperplasia was observed in one female in each of the CDMA-modulation exposure groups (1.5, 3, and 6 W/kg).

Table 4. Incidence of heart lesions in female Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated cell phone RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Schwannoma[‡]	0	0	2 (2.2%)	0	2 (2.2%)	0	2 (2.2%)
Schwann cell hyperplasia	0	0	0	0	1 (1.1%)	1 (1.1%)	1 (1.1%)

[§] Data presented as number of animals per group with tumors (percentage of animals per group with tumors).
[‡] Historical control incidence in NTP studies: 4/699 (0.6 %), range 0-4%

Schwann cells are present in the peripheral nervous system and are distributed throughout the whole body, not just in the heart. Therefore, organs other than the heart were examined for schwannomas and Schwann cell hyperplasia. Several occurrences of schwannomas were observed in the head, neck, and other sites throughout the body of control and GSM and CDMA RFR-exposed male rats. In contrast to the significant increase in the incidence of schwannomas in the heart of exposed males, the incidence of schwannomas observed in other tissue sites of exposed males (GSM and CDMA modulations) was not significantly different than in controls (Table 5). Additionally, Schwann cell hyperplasia was not observed in any tissues other than the heart. The combined incidence of schwannomas from all sites was generally higher in GSM- and CDMA-modulated RFR exposed males, but not significantly different than in controls. The Schwann cell response to RFR appears to be specific to the heart of male rats.

Table 5. Incidence of schwannomas in male Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Heart[‡]	0[*]	2 (2.2%)	1 (1.1%)	5 (5.5%)	2 (2.2%)	3 (3.3%)	6 (6.6%)[**]
Other sites[†]	3 (3.3%)	1 (1.1%)	4 (4.4%)	2 (2.2%)	2 (2.2%)	1 (1.1%)	1 (1.1%)
All sites (total)	3 (3.3%)	3 (3.3%)	5 (5.5%)	7 (7.7%)	4 (4.4%)	4 (4.4%)	7 (7.7%)

[§] Data presented as number of animals per group with tumors (percentage of animals per group with tumors).
[*] Significant SAR level-dependent trend for GSM and CDMA, poly 3 test ($p < 0.05$)
[**] Significantly higher than controls, poly-3 test ($p < 0.05$)
[‡] Historical control incidence in NTP studies: 9/699 (1.3%), range 0-6%
[†] Mediastinum, thymus, and fat

In female rats, there was no statistically significant or apparent exposure-related effect on the incidence of schwannomas in the heart or the combined incidence in the heart or other sites (Table 6).

Table 6. Incidence of schwannomas in female Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

	Control	GSM			CDMA		
Schwannoma site	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Heart[‡]	0	0	2 (2.2%)	0	2 (2.2%)	0	2 (2.2%)
Other sites[†]	4 (4.4%)	1 (1.1%)	3 (3.3%)	1 (1.1%)	0	2 (2.2%)	2 (2.2%)
All sites (total)	4 (4.4%)	1 (1.1%)	5 (5.5%)	2 (2.2%)	2 (2.2%)	2 (2.2%)	4 (4.4%)

[§] Data presented as number of animals per group with tumors (percentage of animals per group with tumors).
[‡] Historical control incidence in NTP studies: 4/699 (0.6%), range 0-4%
[†] Ovary, uterus, vagina, thymus, abdomen, and clitoral gland

DISCUSSION

The two tumor types, which are the focus of this report, are malignant gliomas of the brain and schwannomas of the heart. Glial cells are a collection of specialized, non-neuronal, support cells whose functions include maintenance of homeostasis, formation of myelin, and providing support and protection for neurons of the peripheral nervous system (PNS) and the central nervous system (CNS). In the CNS, glial cells include astrocytes, oligodendrogliocytes, microglial cells, and ependymal cells. Schwann cells are classified as glial cells of the PNS. In the PNS, Schwann cells produce myelin and are analogous to oligodendrocytes of the CNS. Generally, glial neoplasms in the rat are aggressive, poorly differentiated, and usually classified as malignant.

In the heart, exposure to GSM or CDMA modulations of RFR in male rats resulted in a statistically significant, positive trend in the incidence of schwannomas. There was also a statistically significant, pairwise increase at the highest CDMA exposure level tested compared to controls. Schwann cell hyperplasias also occurred at the highest exposure level of CDMA-

modulated RFR. Schwann cell hyperplasia in the heart may progress to cardiac schwannomas. No Schwann cell hyperplasias or schwannomas of the heart were observed in the single, common control group of male rats. The historical control rate of schwannomas of the heart in male Harlan Sprague Dawley rats is 1.30% (7/539) and ranges from 0-6% for individual NTP studies (Table D2, Appendix D). The 5.5-6.6% observed in the 6 W/kg GSM- and CDMA-modulated RFR groups exceeds the historical incidence, and approaches or exceeds the highest rate observed in a single study (6%). The increase in the incidence of schwannomas in the heart of male rats in this study is likely the result of whole-body exposures to GSM- or CDMA-modulated RFR.

In the brain, there was a significant, positive trend in the incidences of malignant gliomas in males exposed to CDMA-modulated RFR, and a low incidence was observed in males at all exposure levels of GSM-modulated RFR that was not statistically different than in control males. Glial cell hyperplasia, a preneoplastic lesion distinctly different from gliosis, was also observed at low incidences in rats exposed to either GSM or CDMA modulation. Glial cell hyperplasia may progress to malignant glioma. Neither of these lesions was observed in the control group of male rats. Although not observed in the current control group, malignant gliomas have been observed in control male Harlan Sprague Dawley rats from other completed NTP studies. Currently in males, the historical control rate of malignant glioma for those studies is 2.0% (11/550) and ranges from 0-8% for individual studies (Table D1, Appendix D). The 2.2-3.3% observed in all of the GSM-modulation groups and in the 6 W/kg CDMA-modulated group only slightly exceeds the mean historical control rate and falls within the observed range.

The survival of the control group of male rats in the current study (28%) was relatively low compared to other recent NTP studies in Hsd:Sprague Dawley® SD® (Harlan) rats (average 47%, range 24-72%). If malignant gliomas or schwannomas are late-developing tumors, the absence of these lesions in control males in the current study could conceivably be related to the shorter longevity of control rats in this study. Appendix E lists the time on study for each animal with a malignant glioma or heart schwannoma. Most of the gliomas were observed in animals that died late in the study, or at the terminal sacrifice. However, a relatively high number of the heart schwannomas in exposed groups were observed by 90 weeks into the study, a time when approximately 60 of the 90 control male rats remained alive and at risk for developing a tumor.

CONCLUSIONS

Under the conditions of these 2-year studies, the hyperplastic lesions and glial cell neoplasms of the heart and brain observed in male rats are considered likely the result of whole-body exposures to GSM- or CDMA-modulated RFR. There is higher confidence in the association between RFR exposure and the neoplastic lesions in the heart than in the brain. No biologically significant effects were observed in the brain or heart of female rats regardless of modulation.

NEXT STEPS

The results reported here are limited to select findings of concern in the brain and heart and do not represent a complete reporting of all findings from these studies of cell phone RFR. The complete results for all NTP studies on the toxicity and carcinogenicity of GSM and CDMA-modulated RFR are currently being reviewed and evaluated according to the established NTP process and will be reported together with the current findings in two forthcoming NTP

Technical Reports. Given the large scale and scope of these studies, completion of this process is anticipated by fall 2017, and the draft NTP Technical Reports are expected to be available for peer review and public comment by the end of 2017. We anticipate that the results from a series of initial studies investigating the tolerance to various power levels of RFR, including measurements of body temperatures in both sexes of young and old rats and mice and in pregnant female rats, will be published in the peer-reviewed literature later in 2016.

APPENDIX A – CONTRIBUTORS

NTP CONTRIBUTORS

Participated in the evaluation and interpretation of results and the reporting of findings.

M.E. Wyde, Ph.D. (NTP study scientist)
M.F. Cesta, D.V.M., Ph.D. (NTP pathologist)
C.R. Blystone, Ph.D.
J.R. Bucher, Ph.D.
S.A. Elmore, D.V.M., M.S.
P.M. Foster, Ph.D.
M.J. Hooth, Ph.D.
G.E. Kissling, Ph.D.
D.E. Malarkey, D.V.M., Ph.D.
R.C. Sills, D.V.M., Ph.D.
M.D. Stout, Ph.D.
N.J. Walker, Ph.D.
K.L. Witt, M.S.
M.S. Wolfe, Ph.D.

APPENDIX B – STATISTICAL ANALYSIS

The Poly-k test (Bailer and Portier, 1988; Portier and Bailer, 1989; Piegorsch and Bailer, 1997) was used to assess neoplasm 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 lesion incidence at 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 sacrifice; if the animal died prior to terminal sacrifice 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, k, for a Weibull hazard function describing cumulative lesion incidence over time (Bailer and Portier, 1988). A further advantage of the Poly-k method is that it does not require lesion lethality assumptions.

Unless otherwise specified, the NTP uses a value of k=3 in the analysis of site-specific lesions (Portier et al., 1986). Bailer and Portier (1988) showed that the Poly-3 test gives valid results if the true value of k is anywhere in the range from 1 to 5. In addition, Portier et al. (1986) modeled a collection of relatively common tumors observed in control animals from two-year NTP rodent carcinogenicity studies, showing that the Weibull distribution with values of k ranging between 1 and 5 was a reasonable fit to tumor incidence in most cases. In cases of early tumor onset or late tumor onset, however, k=3 may not be the optimal choice. Tumors with early onset would require a value of k much less than 3, while tumors with late onset would require a value of k much greater than 3. In the current studies, malignant brain gliomas occurred only in animals surviving more than 88% of the length of the study. For these brain tumors, a Weibull distribution with k=6 is a better fit to survival time than with k=3 (Portier, 1986). Malignant schwannomas of the heart occurred in animals surviving at least 65% of the length of the study; a Weibull distribution with k=3 adequately fits these heart tumor incidences. Therefore, poly-6 tests were used for analyses of brain tumors and poly-3 tests were used for schwannomas.

Variation introduced by the use of risk weights, which reflect differential mortality, was accommodated by adjusting the variance of the Poly-k statistic as recommended by Bieler and Williams (1993) and a continuity correction modified from Thomas et al. (1977) was applied.

Tests of significance for tumors and nonneoplastic lesions included pairwise comparisons of each dosed group with controls and a test for an overall dose-related trend. Continuity-corrected Poly-k tests were used in the analysis of lesion incidence, and reported P values are one sided.

Body weights and litter weights were compared to the control group using analysis of variance and Dunnett's test (1955). The probability of survival was estimated by the product-limit procedure of Kaplan and Meier (1958). Statistical analyses for possible exposure-related effects on survival used Cox's (1972) method for testing two groups for equality and Tarone's (1975) life table test to identify exposure-related trends. Survival analysis p-values are two-sided.

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APPENDIX C – PATHOLOGY

Pathology data presented in this report on cell phone RFR were subjected to a rigorous peer review process. The primary goal of the NTP peer-review process is to reach consensus agreement on treatment-related findings, confirm the diagnosis of all neoplasms, and confirm any unusual lesions. At study termination, a complete necropsy and histopathology evaluation was conducted on every animal. The initial pathology examination was performed by a veterinary pathologist, who recorded all neoplastic and nonneoplastic lesions. This examination identified several potential treatment-related lesions in target organs of concern (brain and heart), which were chosen for immediate review.[1] The initial findings of glial cell tumors and hyperplasias in the brain and schwannomas, Schwann call hyperplasia, and schwannomas from all sites were subjected to an expedited, multilevel NTP pathology peer-review process. The data were locked[2] prior to receipt of the finalized, study-laboratory reports to ensure that the raw data did not change during the review.

The pathology peer review consisted of a quality assessment (QA) review of all slides with tissues from the central nervous system (7 sections of brain and 3 sections of spinal cord), trigeminal nerve and ganglion, and heart. Additionally, the schwannomas of the head and neck region were reviewed. The QA review of the central nervous system and head and neck schwannomas was performed by Dr. Margarita Gruebbel of Experimental Pathology Laboratories, Inc. (EPL), and the QA review of the hearts and trigeminal nerves and ganglia was performed by Dr. Cynthia Shackelford, EPL.

The QA review pathologists then met with Dr. Mark Cesta, NTP pathologist for these studies, and Dr. David Malarkey, head of the NTP Pathology Group, to review lesions and select slides for the Pathology Working Group (PWG) reviews. All PWG reviews were conducted blinded with respect to treatment group and only identified the test articles as “test agent A” or “test

[1] Pathology peer review of remaining lesions from the cell phone RFR studies continues and is not addressed in this report.

[2] Locking data refers to restricting access to the computer database so the data for a particular study cannot be changed.

agent B". Due to the large number of slides for review, the PWG was held in three separate sessions:

- January 29, 2016, for review of glial lesions in the brain and Schwann cell lesions in the heart
- February 11, 2016, for review of schwannomas of the head and neck
- February 12, 2016, for review of granular cell lesions of the brain

The reviewing PWG pathologists largely agreed on the diagnostic criteria for the lesions and on the diagnoses of schwannomas in the head and neck, and granular cell lesions in the brain. However, there was much discussion on the criteria for differentiating glial cell hyperplasia from malignant glioma and Schwann cell hyperplasia from schwannoma. The lack of PWG agreement on definitive criteria for the glial cell and Schwann cell lesions, and the requirement for a high level of confidence in the diagnoses prompted NTP to convene two additional PWGs (organized and conducted by the NTP pathologist, Dr. Mark Cesta) with selected experts in the organ under review. These second level PWG reviews were also conducted as noted above and held in two separate sessions:

- February 25, 2016, for review of glial lesions in the brain
- March 3, 2016, for review of cardiac schwannomas, schwannomas in other organs (except the head and neck), and right ventricular degeneration

In both PWGs, the participants came to consensus on the diagnoses of the lesions and the criteria used for those diagnoses. Participants of the individual PWGs are listed below.

Table C-1. NTP Pathology Working Group (PWG) Attendees

PWG member	Affiliation
January 29, 2016 - Evaluated glial lesions in the brain and Schwann cell lesions in the heart	
A.E. Brix, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences
G.P. Flake, M.D.	National Institute of Environmental Health Sciences
R.H. Garman, D.V.M.	Consultants in Veterinary Pathology, Inc. Monroeville, PA
M.M. Gruebbel, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC (observer)
R.A. Herbert, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
J.S. Hoane, D.V.M.	Charles River Laboratories, Inc. Durham, NC (contract study pathologist)
K.S. Janardhan, BVSc, MVSc, Ph.D.	Integrated Laboratory System
R. Kovi, BVSc, MVSc, Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC (observer)
D.E. Malarkey, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
R.A. Miller, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
J.P. Morrison, D.V.M.	Charles River Laboratories, Inc. Durham, NC

PWG member	Affiliation
A.R. Pandiri, BVSc & AH, Ph.D.	National Institute of Environmental Health Sciences
C.C. Shackelford, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC (observer)
J.A. Swenberg, D.V.M., Ph.D.	University of North Carolina – Chapel Hill, NC
G. Willson, BVMS, Dip RC Path, FRC Path, MRCVS	Experimental Pathology Laboratories, Inc. RTP, NC (PWG coordinator)
February 11, 2016 - Evaluated schwannomas of the head and neck	
A.E. Brix, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences
G.P. Flake, M.D.	National Institute of Environmental Health Sciences
M.M. Gruebbel, D.V.M., Ph.D.,	Experimental Pathology Laboratories, Inc. RTP, NC (PWG coordinator)
K.S. Janardhan, BVSc, MVSc, Ph.D.	Integrated Laboratory System RTP, NC
D.E. Malarkey, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
A.R. Pandiri, BVSc & AH, Ph.D.	National Institute of Environmental Health Sciences
R.R. Maronpot, D.V.M.	Experimental Pathology Laboratories, Inc. RTP, NC
February 12, 2016 - Evaluated granular cell lesions of the brain	
A.E. Brix, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences
M.M. Gruebbel, D.V.M., Ph.D.,	Experimental Pathology Laboratories, Inc. RTP, NC (PWG coordinator)
J.S. Hoane, D.V.M.	Charles River Laboratories, Inc. Durham, NC (contract study pathologist)
K.S. Janardhan, BVSc, MVSc, Ph.D.	Integrated Laboratory System RTP, NC
A.R. Pandiri, BVSc. & AH, Ph.D.	National Institute of Environmental Health Sciences
R.R. Moore, D.V.M.	Integrated Laboratory System RTP, NC
February 25, 2016 - Evaluated glial lesions in the brain	
D. Bigner, M.D., Ph.D.	Duke University Durham, NC
B. Bolon, D.V.M., MS, Ph.D.	GEMpath, Inc. Longmont, CO
V. Chen, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (observer)
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (PWG coordinator, NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences (observer)
G.P. Flake, M.D.	National Institute of Environmental Health Sciences (observer)
J.S. Hardisty, D.V.M.	Experimental Pathology Laboratories, Inc. RTP, NC
R.A. Herbert, D.V.M., Ph.D.,	National Institute of Environmental Health Sciences (observer)
R. Kovi, BVSc, MVSc, Ph.D.	Experimental Pathology Laboratories, Inc. (observer)
P.B. Little, D.V.M.	Experimental Pathology Laboratories, Inc.
D.E. Malarkey, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
J.P. Morrison, D.V.M., Ph.D.	Charles River Laboratories, Inc.
A. Sharma, BVSc, MVSc, MS, Ph.D.	Covance
March 3, 2016 - Evaluated heart lesions, and schwannomas in other organs (except head and neck)	
B. Berridge, D.V.M., Ph.D.	GlaxoSmithKline RTP, NC
M.C. Boyle, D.V.M., Ph.D.	Amgen Thousand Oaks, CA
V. Chen, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (observer)
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (PWG coordinator, NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences (observer)
M. Elwell, D.V.M., Ph.D.	Covance Chantilly, VA

PWG member	Affiliation
J.R. Hailey, D.V.M.	Covance Chantilly, VA
M. Novilla, D.V.M., MS, Ph.D.	SNBL Everett, WA

LESION DESCRIPTIONS

Brain

Malignant gliomas were infiltrative lesions, usually of modest size, with indistinct tumor margins. The neoplastic cells were typically very densely packed with more cells than neuropil. The cells were typically small and had round to oval, hyperchromatic nuclei. Mitoses were infrequent. In some of the neoplasms, invasion of the meninges, areas of necrosis surrounded by palisading neoplastic cells, cuffing of blood vessels, and neuronal satellitosis were observed. The malignant gliomas did not appear to arise from any specific anatomic subsite of the brain.

Glial cell hyperplasia consisted of small, proliferative, and poorly demarcated foci of poorly differentiated glial cells that accumulated and invaded into the surrounding parenchyma. In some cases, there was a small amount of perivascular cuffing. The hyperplastic cells appeared morphologically identical to those in the gliomas but were typically less dense with more neuropil than glial cells. There were no necrotic or degenerative elements present, so there was no evidence that the increased number of glial cells was a reaction to brain injury.

Heart

The intracardiac schwannomas were either endocardial or myocardial (intramural). The endocardial schwannomas lined the ventricles and atria and invaded into the myocardium. Two morphologic cell types were observed, but indistinct cell margins and eosinophilic cytoplasm were common to both types. Groups of cells with widely spaced small, round nuclei and moderate amounts of cytoplasm were interspersed among bands or sheets of parallel, elongated cells with thin, spindle-shaped, hyperchromatic nuclei. The myocardial schwannomas were typically less densely cellular and infiltrated amid, sometimes replacing, the cardiomyocytes. The cell types described for the endocardial neoplasms were both present, but in fewer numbers. In both subtypes of schwannomas, there was a minimal amount of cellular pleomorphism. In some larger neoplasms, Antoni type A and B patterns were present.

The Schwann cell hyperplasias were similar in appearance to the schwannomas, but were smaller and had less pleomorphism of the cells. In the case of the endocardial Schwann cell hyperplasia, there was no invasion of the myocardium.

APPENDIX D – HISTORICAL CONTROLS

Table D1. Incidence of astrocytoma, glioma, and/or oligodendroglioma in brains of male Harlan Sprague Dawley rats in NTP studies

Chemical	First dose	N	Control incidence
Dibutylphthalate	8/30/2010	49	4%
2-Hydroxy-4-methoxybenzophenone	11/8/2010	50	0%
p-Chloro-a,a,a-trifluorotoluene	1/17/2011	50	4%
Di-(2-ethylhexyl)phthalate	2/17/2011	50	8%
Di-(2-ethylhexyl)phthalate (perinatal)	6/27/2011	50	0%
Tris (chloroisopropyl) phosphate	12/12/2011	50	0%
Sodium tungstate	12/23/2011	50	4%
Resveratrol	5/7/2012	50	0%
Black cohosh	7/2/2012	50	2%
Radiofrequency radiation (GSM/CDMA)	9/16/2012	90	0%

Historical control rate: 11/550 (2.0%)

Table D2. Incidence of schwannoma in the heart of male Harlan Sprague Dawley rats in NTP studies

Chemical	First dose	N	Control incidence
Indole-3-carbinol	3/14/2007	50	2%
Perfluorooctanoic acid	6/19/2009	50	0%
Dietary zinc	9/3/2009	50	0%
Dibutylphthalate	8/30/2010	49	4%
2-Hydroxy-4-methoxybenzophenone	11/8/2010	50	2%
p-Chloro-a,a,a-trifluorotoluene	1/17/2011	50	0%
Di-(2-ethylhexyl)phthalate	2/17/2011	50	6%
Di-(2-ethylhexyl)phthalate (perinatal)	6/27/2011	50	4%
Tris (chloroisopropyl) phosphate	12/12/2011	50	0%
Sodium tungstate	12/23/2011	50	0%
Resveratrol	5/7/2012	50	0%
Black Cohosh	7/2/2012	50	0%
Radiofrequency radiation (GSM/CDMA)	9/16/2012	90	0%

Historical control rate: 9/699 (1.30%)

APPENDIX E – TIME ON STUDY TO APPEARANCE OF TUMORS

Malignant Glioma

SAR (W/kg)	Animal ID number	Time on study (weeks)
GSM-modulated exposed males		
1.5	717	105
	735	102
	786	104
3.0	924	101
	943	105
	1014	93
6.0	1135	104
	1137	102
CDMA-modulated exposed males		
6.0	1795	105
	1799	104
	1852	105
GSM-modulated exposed females		
6.0	1246	96
CDMA-modulated exposed females		
1.5	1463	105
	1474	105

Time to Malignant Schwannoma in Heart

SAR (W/kg)	Animal ID number	Length of survival (weeks)
GSM-modulated exposed males		
1.5	758	104
	801	105
3.0	931	105
6.0	1149	83
	1155	105
	1187	104
	1206	104
	1230	91
CDMA-modulated exposed males		
1.5	1364	105
	1352	105
3.0	1559	92
	1617	105
	1622	104
6.0	1801	76
	1821	70
	1829	104
	1833	89
	1849	104
	1860	105
GSM-modulated exposed females		
3.0	1037	105
	1077	83
CDMA-modulated exposed females		
1.5	1461	106
	1480	93
6.0	1888	105
	1965	106

APPENDIX F – REVIEWER'S COMMENTS

National Toxicology Program

Peer Review Charge and Summary Comments

Purpose: To provide independent peer review of an initial draft of this partial report. The peer reviewers were blind to the test agents under study. Introductory materials on RFR and details of the methods dealing with the field generation and animal housing were redacted from the version sent to the reviewers. The reviewers were provided a study data package, also blinded to test agents, containing basic in life study information such as body weight and survival curves and information concerning the generation of pups from the *in utero* exposures.

Report Title: Draft Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Test Articles A and B (and associated Study Data Package)

Reviewers' Names:

David Dorman, D.V.M., Ph.D., North Carolina State University
Russell Cattley, D.V.M., Ph.D., Auburn University
Michael Pino, D.V.M., Ph.D., Pathology consultant

Charge: To peer review the draft report and comment on whether the scientific evidence supports NTP's conclusion(s) for the study findings.

1. Scientific criticisms:
   a. Please comment on whether the information presented in the draft report, including presentation of data in any tables, is clearly and objectively presented. Please suggest any improvements.

   All three reviewers found the results to be clearly and objectively presented, although there were suggestions to provide historical control information for brain and heart lesions for female Harlan Sprague Dawley rats, clarify statements about the specific statistical tests used and the presence or lack of statistical significance of the brain

gliomas in the Results, and expand the conclusions statements to clarify the basis for the conclusions.

b. Please comment on whether NTP's scientific interpretations of the data are objective and reasonable. Please explain why or why not.

The reviewers stated that the NTP had performed an adequate and objective peer review of the pathology data, and the statistical approaches used were consistent with other NTP studies. The methods were described as objective and reasonable. The interpretations of the data, including the limitations, were also reasonable and objective. One reviewer found the data on schwannomas of the heart to be more compelling with respect to an association with treatment than the brain gliomas. This reviewer summarized the findings as:

> "In the heart the evidence for a carcinogenic effect can be based on 1) the presence of the tumors in all six of the test article groups versus none in the controls 2) the statistically significant trend for schwannomas with both compounds and the statistically significant increase in incidence in the 4X (top) dose for test article B; 3) the fact that the incidence of the tumors in both 4X dose groups approaches or exceeds the high end of the historical control range; and 4) the tumors in the 4X group of test article B are accompanied by a higher incidence of Schwann cell hyperplasia. Using the NTP's guide for levels of evidence for carcinogenic activity, I would consider the heart schwannomas as 'Some Evidence' of carcinogenic activity.
>
> The proliferative lesions in the brain are more difficult to interpret because 1) their low incidence that was well within the historical control range, 2) lack of clear dose response; and 3) lack of statistical significance (except for the significant exposure-dependent trend for test article B. . . . However, the presence of malignant gliomas and/or foci of glial cell hyperplasia in 5 of 6 test article groups for both sexes vs none in controls of either sex is suggestive of a test

article effect. . . .I would consider the malignant gliomas as 'Equivocal Evidence' of carcinogenic activity."

2. Please identify any Information that should be added or deleted:

One reviewer suggested that more information be given on the time when tumors were observed (e.g., at terminal necropsy, or early in the study) to help assess the possible impact of the decreased survival times in the control animals on tumor incidence. This reviewer also suggested a discussion of how the survival of control male rats in this study compared to the historical control data. There was also concern that the diagnostic criteria developed by the PWG and used in the current study would impact the historical control incidence rates reported in Table D.

3. The scientific evidence supports NTP's conclusion(s) for the study findings:

The NTP's overall draft conclusion was as follows: "Under the conditions of these studies, the observed hyperplastic lesions and neoplasms outlined in this partial report are considered likely the result of exposures to test article A and test article B. The findings in the heart were statistically stronger than the findings in the brain."

The reviewers had the option of agreeing, agreeing in principle, or disagreeing with the draft conclusions. All three reviewers agreed in principle, reiterating issues discussed above.

APPENDIX G – NIH REVIEWER'S COMMENTS

National Institutes of Health

Peer Review Charge and Reviewer's Comments

Purpose: To provide independent peer review of the pathology diagnoses and statistical evaluation of the partial findings from NTP's studies. Background materials included the draft NTP report, introductory materials on RFR, and details on the methods dealing with the field generation and statistical analyses references and guidance. The reviewers were provided a study data package, containing basic in life study information such as body weight and survival curves, information concerning the generation of pups from the *in utero* exposures, and raw pathology data.

Report Title: Draft Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Test Articles A and B (and associated Study Data Package)

Reviewers' Names:

Diana C. Haines, D.V.M., Frederick National Laboratory
Michael S. Lauer, M.D., Office of Extramural Research, NIH
Maxwell P. Lee, Ph.D., Laboratory of Cancer Biology and Genetics, NCI,
Aleksandra M. Michalowski, M.Sc., Ph.D., Laboratory of Cancer Biology and Genetics, NCI
R. Mark Simpson, D.V.M., Ph.D., Laboratory of Cancer Biology and Genetics, NCI
[Sixth reviewer's name and comments are withheld.]

Charge: To peer review the draft report, statistical analyses, and pathology data and comment on whether the scientific evidence supports NTP's conclusion(s) for the study findings.

Reviewer's comments and NTP responses to the comments are provided.

- Appendix G1: Reviewer's comments
- Appendix G2: NTP's responses to NIH reviewer's comments

Appendix G1: Reviewer's comments

Reviewer: Diana C. Haines, D.V.M., Frederick National Laboratory

April 5, 2016

Dr. Tabak,

I've always relied on experts, not myself, for statistical analysis, and so do not feel qualified to address the statistical methods used. My training and experience has been in veterinary pathology, including QA review of NTP studies, and serving on PWGs, so will give my opinion on the pathology interpretation (biological significance rather than statistical significance).

Having perused the 3 RFR Draft Report and the raw data, all appears to be in order, including QA of the histopathology (technique) as well as PWG review (diagnosis). Looking at the data, I agree with the report's conclusion: *Under the conditions of these studies, the hyperplastic lesions and neoplasms observed in male rats are considered likely the result of exposures to GSM- an CDMA-modulated RFR. The findings in the heart were statistically stronger than the findings in the brain.* But note, it is "considered likely" not "definitely is".

There may be also several caveats relating to "under the conditions of these studies", including how well the conditions recapitulate actual human exposure: whole body exposure from in utero to old age; 18.5 hours/day (10 min on/10 min off, for total of 9hr actual exposure); and dose[A]. I'm not physicist, so have to presume experts analyzed and accepted concept of the reverberation chamber, including "doses"[A] as being relevant to human exposure.

[A] Dosimetric Assessment paper: "As could be expected in a study following NTP protocols, the exposure levels for the rodents in this project exceed the limits for the wbSAR and psSAR defined in the IEEE Std C95.1-2005 safety standard for human exposure to mobile phone radiation. In the low dose exposure group the exposure level in the organs exceeds or is close to the localized SAR limit for the general public, except for a few low-water content tissues. More specifically, the psSAR over 1 g in the human head, is limited by the safety standards to <2W/kg, whereas, in the low dose rodents the SAR averaged over the whole brain is >2.4 W/kg for mice, and >1.3 W/kg for rats, hence similar to the limit. Furthermore, the psSAR and oSAR have larger uncertainty compared to the wbSAR. Deviations of the exposure level from the target dose, especially during the early exposure period, should be carefully evaluated in the interpretation of the final biological studies.

Results from the companion mouse study will hopefully add some insight.

Diana Copeland Haines, DVM
Diplomate, American College of Veterinary Pathologists
Senior Staff Pathologist, Pathology Section
Pathology/Histotechnology Laboratory
Leidos Biomedical Research, Inc.
Frederick National Laboratory for Cancer Research
P.O. Box B, Frederick, MD 21702
Phone: 301-846-5921 Fax: 301-846-1953
Diana.Haines@fnlcr.nih.gov
http://ncifrederick.cancer.gov/rtp/lasp/phl/

Appendix G1: Reviewer's comments

Reviewer: Michael S. Lauer, M.D., Office of Extramural Research, NIH

Michael S Lauer, MD (OER)

Review of NTP paper: "Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Cell Phone Radiofrequency Radiation (Whole Body Exposures)"
March 20, 2016

Summary of findings:

This is a partial report, a report which is presumably part of a larger set of studies involving 2 species (mice and rats), 2 sexes (male, female), and multiple tissue types, all based on 90-week studies of two different types (GSM and CDMA) of cell phone radiofrequency radiation (RFR). In this partial report, we are given findings regarding brain gliomas and heart schwannomas in male and female Harlan Sprague Dawley rats which were exposed exposed to control or 3 different levels (1.5, 3.0, 6.0) of two types (GSM and CDMA) of RFR. There were 90 rats in each group. Using the poly-3 test with the Bieler-Williams variance adjustment, the authors found a statistically significant increase in the rate of brain gliomas in males exposed to CDMA RFR. Using the poly-6 test, the authors found a statistically significant increase in the rates of heart schwannomas in males exposed to GSM and CDMA. There were no statistically significant differences in rates of gliomas or schwannomas in females; also there was no statistically significant increase in rates of gliomas in males exposed to GSM RFR.

Comments:

1) Why aren't we being told, at least at a high level, of the results of other experiments (i.e., male and female mice, tissues other than heart and brain, tumors other than glioma and schwannoma)? Given the multiple comparisons inherent in this kind of work (see pages 27-30 and Table 13 of the FDA guidance document), there is a high risk of false positive discoveries. In the absence of knowing other findings, we must worry about selective reporting bias.
2) I was able to reproduce the authors' positive P-value findings (see Appendix 1, R code) using the MCPAN R package. However, I'm getting slightly different values for adjusted denominators (also in Appendix 1).
3) I was able to reproduce the authors' findings of longer survival with RFR (see Appendix 1, R code).
4) I have a number of questions about the study design:
    a. Were control rats selected in utero like the exposed rats were?
    b. Were pregnant dams assigned to different groups by formal randomization? If not, why not?
    c. Why were pups in the same litter included? Did the authors take any steps in their analyses to account for the resulting absence of i.i.d?
    d. The authors state that at most 3 pups were chosen per litter. How were the 3 pups chosen (and the others presumably not used for this experiment)? Were the 3 pups that were chosen selected by formal randomization? If not, why not?

e. Were all analyses based on the intent-to-treat principle? Were there any crossovers? Were all rats accounted for by the end of the experiment and were all rats who started in the experiment included in the final analyses?
f. Blinding: The authors state that "All PWG reviewer were conducted blinded with respect to treatment group," but in the very next phrase write "only identifying the test articles as 'test agent A' or 'test agent B.'" Why was this information (test agent A or B) given? The blinding was not complete.

5) Sample size:
   a. Did the authors perform a prospective (that is before initiation of the work) sample size calculation? If so, what were the prior assumptions? In other words, why did the authors choose to study 90 rats in each group and why did they set the maximum duration to 90 weeks (instead of 104 weeks)?
   b. I used a publicly available simulation package[1] to calculate the study power for male rats based on the following (see Appendix 2, power calculation simulation studies):
      i. Control tumor rate of ~1.5%.
      ii. Risk ratio 2.5 in the group receiving the highest dose
      iii. 2-sided Alpha = 0.005 (based on Table 13 of the FDA guidance document). Note this low alpha of 0.005 for poly-k trend tests is recommended to minimize the risk of false positive discoveries.
      iv. Sample size of 90 for each group with one planned sacrifice.
      v. Low lethality with lethality parameters set according to study duration and Weibull shape parameter (see Table 3 of Moon et al[1]). When I re-ran the simulations using intermediate lethality, results were not materially changed.
      vi. Study duration 90 weeks
      vii. 5000 simulations
      viii. Note – I used dose levels of 0,1,2, and 4 because I was unable to adjust these on the web site (despite trying 3 different browsers).
   c. Based on these inputs, the recommendations in Table 13 of the FDA guidance document, and a sample size of 90 rats in each group, I find very low power (<5%, see Appendix 2). Even allowing for a risk ratio of 5.0 (a level that is clinically unlikely), the power for 2-sided alpha=0.005, k=3 and low lethality is only ~14% (see Appendix 2).
   d. The low power implies that there is a high risk of false positive findings[2], especially since the epidemiological literature questions the purported association between cell phone exposure and cancer.[3]

6) Summary: I am unable to accept the authors' conclusions:
   a. We need to know all other findings of these experiments (mice, other tumor types) given the risk of false positive findings and reporting bias. It would be helpful to have a copy of the authors' statistical code.
   b. We need to know whether randomization was employed to assign dams to specific groups (control and intervention).

c. We need to know whether randomization was employed to determine which pups from each litter were chosen for continued participation in the experiment.
d. We need to know whether there was a formal power/sample size calculation performed prior to initiation of the experiment. If not, why not? If yes, we need to see the details. In particular, we need to know whether the authors followed the recommendations of the FDA guidance document (in particular Table 13).
e. I suspect that this experiment is substantially underpowered and that the few positive results found reflect false positive findings.[2] The higher survival with RFR, along with the prior epidemiological literature, leaves me even more skeptical of the authors' claims.

References:

1. Moon H, Lee JJ, Ahn H, Nikolova RG. A Web-based Simulator for Sample Size and Power Estimation in Animal Carcinogenicity Studies. *J Stat Software; Vol 1, Issue 13* . 2002. doi:10.18637/jss.v007.i13.
2. Ioannidis JPA. Why most published research findings are false. Jantsch W, Schaffler F, eds. *PLoS Med*. 2005;2(8):e124. doi:10.1371/journal.pmed.0020124.
3. Frei P, Poulsen AH, Johansen C, Olsen JH, Steding-Jessen M, Schüz J. Use of mobile phones and risk of brain tumours: update of Danish cohort study. *BMJ*. 2011;343.

Appendix 1: Attempted replication of positive findings

```
# Review of NTP paper on cell phone RFR and certain cancers
# Attempt to reproduce the positive findings
# Data from Larry Tabak
# Code by Mike Lauer

setwd("~/Desktop/Files to save")

library(MCPAN)
library(rms)
library(Hmisc)

# Read in CDMA NTP data

CDMA <- read.csv("~/Desktop/Files to save/NTP CDMA Raw Tumor Data.csv")

# Survival and treatment group, adjusting for sex, by Cox proportional hazards

CDMA$status<-1
CDMA$S<-Surv(CDMA$Removal.Day, CDMA$status)
f<-cph(S~Treatment+Sex, data=CDMA)
f

# Survival greater (better) for 3.0W, P=0.0157, for 6.0W, P=0.0260

# Table 1 -- Poly-3 test for malignant glioma in males CDMA

males_CDMA<-subset(CDMA, Sex=='M')

poly3test(time=males_CDMA$Removal.Day, status=males_CDMA$Brain.Glioma.Malignant,
      f=males_CDMA$Dose, k=3, type='Williams', method='BW', alternative='greater')

# P=0.039

poly3ci(time=males_CDMA$Removal.Day, status=males_CDMA$Brain.Glioma.Malignant,
      f=males_CDMA$Dose, k=3, type='Williams', method='BW', alternative='greater')
```

Call result:

```
Sample estimates, using poly- 3 -adjustment
                    0         1.5       3       6
x                   0.0000  0.0000  0.0000  3.0000
n                  90.0000 90.0000 90.0000 90.0000
adjusted n         63.8258 72.3688 76.6821 64.8154
adjusted estimate  0.0000  0.0000  0.0000  0.0463

# Table 3 -- Poly-6 test for malignant Schwannoma in males CDMA

poly3test(time=males_CDMA$Removal.Day,
        status=males_CDMA$Heart.Schwannoma.Malignant, f=males_CDMA$Dose, k=6,
        type='Williams', method='BW', alternative='greater')

# P=0.0005

poly3ci(time=males_CDMA$Removal.Day,
        status=males_CDMA$Heart.Schwannoma.Malignant,f=males_CDMA$Dose,
        k=3,type='Williams', method='BW')

Call result:

Sample estimates, using poly- 3 -adjustment
                    0     1.5       3       6
x                 0.0000  2.0000  3.0000  6.0000
n                90.0000 90.0000 90.0000 90.0000
adjusted n         63.8258 72.3971 77.0575 66.5582
adjusted estimate  0.0000  0.0276  0.0389  0.0901

# Read in GSM NTP data

GSM <- read.csv("~/Desktop/Files to save/NTP GSM Raw Tumor data.csv")

# Survival and treatment group, adjusting for sex, by Cox proportional hazards

GSM$status<-1
GSM$S<-Surv(GSM$Removal.Day, GSM$status)
f<-cph(S~Treatment+Sex, data=GSM)
f

# Survival greater (better) for 6.0W, P=0.0048

males_GSM<-subset(GSM, Sex=='M')

# Table 3 -- Poly-6 test for malignant Schwannomas in males GSM
```

```
poly3test(time=males_GSM$Removal.Day, status=males_GSM$Heart.Schwannoma.Malignant,
        f=males_CDMA$Dose, k=6, type='Williams', method='BW', alternative='greater')

# P=0.004

poly3ci(time=males_GSM$Removal.Day, status=males_GSM$Heart.Schwannoma.Malignant,
        f=males_CDMA$Dose, k=3, type='Williams', method='BW', alternative='greater')
```

Call result:

Sample estimates, using poly- 3 -adjustment

	0	1.5	3	6
x	0.0000	2.0000	1.0000	5.0000
n	90.0000	90.0000	90.0000	90.0000
adjusted n	63.8258	73.1547	76.1127	77.0723
adjusted estimate	0.0000	0.0273	0.0131	0.0649

Appendix 2: Simulations for power calculations

Power Simulations for NTP Cell Phone RFR paper (from
https://biostatistics.mdanderson.org/acss/Login.aspx and
https://www.jstatsoft.org/article/view/v007i13)[1]
Michael Lauer, MD (OER)
March 19, 2016

1) For malignant gliomas (Table 1), P = 0.005, HR = 2.5, k=3

The University of Texas M. D. Anderson Cancer Center
Sample Size and Power Estimation for Animal Carcinogenicity Studies

Reference: "A Web-based Simulator for Sample Size and Power
Estimation in Animal Carcinogenicity Studies."
Hojin Moon, J. Jack Lee, Hongshik Ahn and Rumiana G. Nikolova,
Journal of Statistical Software. (2002)[1]

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 3.00
Hazard ratio(s) of dose vs. control group
1.50 2.00 2.50
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 23.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990
average lethality = 0.0816

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0060	0.0000	0.0000
67	0.0002	0.0002	0.0334	0.0000	0.0000
78	0.0003	0.0005	0.0729	0.0000	0.0000
90	0.0005	0.0023	0.1855	0.0094	0.6887

dose group 1:
average tumor rate = 0.0225
average competing risks survival rate = 0.7000
average lethality = 0.0784

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0003	0.0002	0.0325	0.0000	0.0000
78	0.0004	0.0008	0.0720	0.0000	0.0000
90	0.0007	0.0034	0.1851	0.0145	0.6842

dose group 2:
average tumor rate = 0.0297
average competing risks survival rate = 0.6997
average lethality = 0.0772

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0004	0.0003	0.0331	0.0000	0.0000
78	0.0005	0.0012	0.0721	0.0000	0.0000
90	0.0010	0.0045	0.1829	0.0191	0.6790

dose group 3:
average tumor rate = 0.0366
average competing risks survival rate = 0.7007
average lethality = 0.0772

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0005	0.0003	0.0330	0.0000	0.0000

78	0.0006	0.0013	0.0716	0.0000	0.0000
90	0.0012	0.0054	0.1812	0.0238	0.6749

Positive Trend (Power): 0.0238

2) For malignant Schwannomas (Table 3), P = 0.005, HR = 2.5, k=6

The University of Texas M. D. Anderson Cancer Center
Sample Size and Power Estimation for Animal Carcinogenicity Studies

Reference: "A Web-based Simulator for Sample Size and Power
Estimation in Animal Carcinogenicity Studies."
Hojin Moon, J. Jack Lee, Hongshik Ahn and Rumiana G. Nikolova,
Journal of Statistical Software. (2002)[1]

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 6.00
Hazard ratio(s) of dose vs. control group
1.50 2.00 2.50
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 45.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990
average lethality = 0.0631

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0060	0.0000	0.0000
67	0.0001	0.0001	0.0335	0.0000	0.0000
78	0.0002	0.0003	0.0732	0.0000	0.0000
90	0.0005	0.0019	0.1859	0.0096	0.6887

dose group 1:
average tumor rate = 0.0225
average competing risks survival rate = 0.7000
average lethality = 0.0602

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0059	0.0000	0.0000
67	0.0001	0.0001	0.0326	0.0000	0.0000
78	0.0003	0.0005	0.0723	0.0000	0.0000
90	0.0006	0.0029	0.1856	0.0148	0.6842

dose group 2:
average tumor rate = 0.0297
average competing risks survival rate = 0.6997
average lethality = 0.0582

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0059	0.0000	0.0000
67	0.0002	0.0001	0.0333	0.0000	0.0000
78	0.0004	0.0007	0.0726	0.0000	0.0000
90	0.0009	0.0038	0.1837	0.0195	0.6790

dose group 3:
average tumor rate = 0.0366
average competing risks survival rate = 0.7007
average lethality = 0.0588

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0059	0.0000	0.0000
67	0.0003	0.0001	0.0332	0.0000	0.0000
78	0.0005	0.0007	0.0722	0.0000	0.0000
90	0.0011	0.0046	0.1821	0.0243	0.6749

Positive Trend (Power): 0.0230

3) For further consideration, P = 0.005, HR = 5, k=3

The University of Texas M. D. Anderson Cancer Center
Sample Size and Power Estimation for Animal Carcinogenicity Studies

Reference: "A Web-based Simulator for Sample Size and Power
Estimation in Animal Carcinogenicity Studies."
Hojin Moon, J. Jack Lee, Hongshik Ahn and Rumiana G. Nikolova,
Journal of Statistical Software. (2002) In Press.

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 3.00
Hazard ratio(s) of dose vs. control group
2.00 3.50 5.00
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 23.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990

average lethality = 0.0816

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0060	0.0000	0.0000
67	0.0002	0.0002	0.0334	0.0000	0.0000
78	0.0003	0.0005	0.0729	0.0000	0.0000
90	0.0005	0.0023	0.1855	0.0094	0.6887

dose group 1:
average tumor rate = 0.0301
average competing risks survival rate = 0.7000
average lethality = 0.0743

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0004	0.0003	0.0324	0.0000	0.0000
78	0.0005	0.0011	0.0717	0.0000	0.0000
90	0.0009	0.0045	0.1839	0.0194	0.6789

dose group 2:
average tumor rate = 0.0515
average competing risks survival rate = 0.6997
average lethality = 0.0774

sacrifice time	d	a1	b1	a2	b2
45	0.0002	0.0000	0.0058	0.0000	0.0000
67	0.0007	0.0006	0.0328	0.0000	0.0000
78	0.0009	0.0020	0.0713	0.0000	0.0000
90	0.0017	0.0076	0.1795	0.0331	0.6638

dose group 3:
average tumor rate = 0.0727
average competing risks survival rate = 0.7007
average lethality = 0.0804

sacrifice time	d	a1	b1	a2	b2
45	0.0003	0.0000	0.0059	0.0000	0.0000
67	0.0010	0.0006	0.0327	0.0000	0.0000
78	0.0013	0.0028	0.0701	0.0000	0.0000
90	0.0025	0.0107	0.1755	0.0470	0.6496

Positive Trend (Power): 0.1420

4) For further consideration, same as in baseline (1) but with intermediate lethality

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 3.00
Hazard ratio(s) of dose vs. control group
1.50 2.00 2.50
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 225.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990
average lethality = 0.3936

sacrifice time	d	a1	b1	a2	b2
45	0.0004	0.0000	0.0060	0.0000	0.0000
67	0.0014	0.0001	0.0334	0.0000	0.0000
78	0.0014	0.0004	0.0729	0.0000	0.0000
90	0.0019	0.0015	0.1855	0.0063	0.6887

dose group 1:
average tumor rate = 0.0225
average competing risks survival rate = 0.7000
average lethality = 0.3852

sacrifice time	d	a1	b1	a2	b2
45	0.0006	0.0000	0.0059	0.0000	0.0000
67	0.0022	0.0001	0.0325	0.0000	0.0000
78	0.0020	0.0006	0.0720	0.0000	0.0000
90	0.0029	0.0023	0.1851	0.0097	0.6842

dose group 2:
average tumor rate = 0.0297
average competing risks survival rate = 0.6997
average lethality = 0.3839

sacrifice time	d	a1	b1	a2	b2
45	0.0008	0.0000	0.0059	0.0000	0.0000
67	0.0029	0.0003	0.0331	0.0000	0.0000
78	0.0027	0.0008	0.0721	0.0000	0.0000
90	0.0039	0.0031	0.1829	0.0127	0.6790

dose group 3:
average tumor rate = 0.0366
average competing risks survival rate = 0.7007
average lethality = 0.3897

sacrifice time	d	a1	b1	a2	b2
45	0.0009	0.0000	0.0059	0.0000	0.0000
67	0.0037	0.0003	0.0330	0.0000	0.0000
78	0.0033	0.0009	0.0716	0.0000	0.0000
90	0.0048	0.0037	0.1812	0.0157	0.6749

Positive Trend (Power): 0.0219

References:

1. Moon H, Lee JJ, Ahn H, Nikolova RG. A Web-based Simulator for Sample Size and Power Estimation in Animal Carcinogenicity Studies. *J Stat Software; Vol 1, Issue 13* . 2002. doi:10.18637/jss.v007.i13.
2. Ioannidis JPA. Why most published research findings are false. Jantsch W, Schaffler F, eds. *PLoS Med*. 2005;2(8):e124. doi:10.1371/journal.pmed.0020124.
3. Frei P, Poulsen AH, Johansen C, Olsen JH, Steding-Jessen M, Schüz J. Use of mobile phones and risk of brain tumours: update of Danish cohort study. *BMJ*. 2011;343.

Appendix G1: Reviewer's comments

Reviewer: Maxwell P. Lee, Ph.D., Laboratory of Cancer Biology and Genetics, NCI

I think the study was well designed and the analyses and results were clearly presented.

My main concern is the control data. Since the main finding was the increased incidence rates of heart schwannomas and brain gliomas in male Harlan Spragu Dawley rats exposed to GSM- or CDMA-modulated cell phone RFR, my analyses and evaluation below were focused on the male rats.

My concern regarding the control data came from the following two considerations. First, we need to consider sample variation. The incidence rates of the current controls for brain gliomas and heart schwannomas were 0. However, the historical controls were 1.67% for gliomas (range 0-8%) and 1.30% for schwannomas (0-6%). Given that there were substantial variations among the historical controls and the concurrent control is at the lowest end of the range, it is important to evaluate how different estimates of control incidence rates may impact the results of analyses. Supplementary Table S1 shows that for gliomas with 1.7% incidence rate we have 40%, 37%, 17%, and 6% of chance to observe 0 tumor, 1 tumor, 2 tumors, and greater than 2 tumors, respectively; heart schwannomas has similar distribution. Given the low incidence rate and moderate sample size of the control, even after observing 0 tumor in the current study, the 'true' incidence rate may be higher than 0. If we were repeating the experiment, we may see some control studies have 1 or more tumors. Second, it is puzzling why the control had short survival rate. Given that most of the gliomas and heart schwannomas are late-developing tumors, it is possible that if the controls were living longer some tumors might develop. Although the use of poly-3 (or poly-6) test intended to adjust the number of rats used in the study, it is still important to re-evaluate the analysis by considering the incidence rate in controls not being 0.

Therefore I have performed the analyses using the original data as well as the data modified by adding 1 tumor to the control. I implemented the poly-3 (or poly-6) trend test in R using the formula described in the file, Poly3 correction factor[1].docx.

The results are summarized in Table 1 for brain gliomas

Table 1. Incidence of brain gliomas in male rats exposed to GSM- or CDMA-modulated RFR, comparing control data with 0 vs. 1 tumor.

RFR	W/kg				pvalue
	0	1.5	3	6	
GSM	0	3	3	2	0.9771
GSM	1	3	3	2	0.8668
CDMA	0	0	0	3	0.0233
CDMA	1	0	0	3	0.1077

Poly-6 adjusted rates were used in the chi-square trend test. The 1st and 3rd rows correspond to the original data with 0 tumor observed in the control group (Th numbers in Table 1 here are identical to those in Table 1 in the original report). The test is significant for CDMA exposures (pvalue = 0.0233). However, it is not significant after adding 1 tumor to the control group (pvalue = 0.1077, the 4th row).

Similar analysis was performed for heart schwannomas. The results are summarized in Table 2.

Table 2. Incidence of heart schwannomas in male rats exposed to GSM- or CDMA-modulated RFR, comparing control data with 0 vs. 1 tumor.

RFR	W/kg				pvalue
	0	1.5	3	6	
GSM	0	2	1	5	0.0431
GSM	1	2	1	5	0.1079
CDMA	0	2	3	6	0.0144
CDMA	1	2	3	6	0.0365

Poly-3 adjusted rates were used in the chi-square trend test. The 1st and 3rd rows correspond to the original data with 0 tumor observed in the control group (Th numbers in Table 2 here are identical to those in Table 3 in the original report). The tests are significant for both GSM (pvalue = 0.0431) and CDMA (pvalue = 0.0144) exposures. However, only CDMA exposure remains significant after adding 1 tumor to the control group (pvalue = 0.0365, the 4th row).

Since the incidence of heart schwannomas in the 6 W/kg males was significantly higher in CDMA exposed males than the control group in the original report, I also analyzed the impact of adding 1 tumor to the control group

Table 3. Incidence of heart schwannomas in male rats exposed to 6 W/kg CDMA-modulated RFR, comparing control data with 0 vs. 1 tumor.

RFR	W/kg		pvalue
	0	6	
CDMA	0	6	0.0381
CDMA	1	6	0.0986

Poly-3 adjusted rates were used in the chi-square trend test. The 1st row corresponds to the original data with 0 tumor observed in the control group. The test was significant for CDMA exposures (pvalue = 0.0381). However, it was not significant after adding 1 tumor to the control group (pvalue = 0.0986, the 2nd row).

Conclusions

Increased incidence of heart schwannomas in male rats exposed to GSM- or CDMA-modulated RFR is statistically significant by the chi-square trend test. The evidence is better for CDMA exposure than GSM exposure. I think additional experiments are needed to assess if the incidence of brain gliomas in male rats exposed to GSM- or CDMA-modulated RFR is significantly higher than the control group or not.

My additional comments are summarized below.

1. I compared poly-3 adjusted number from Table 3 in the original repor versus the poly-3 adjusted number that I calculated using the raw data from the excel files. Supplementary Figure S1 shows that these two sets of numbers agre with each other in general. This is in contrast to the comparison for poly-6 adjusted number from Table 1 in the original report versus the poly-6 adjusted number that I calculated using the raw data from the excel files (Supplementary Figure S2). In fact, the adjusted rat numbers from Table 1 and Table 3 of the original report look quite similar (Supplementary Figure S3). This suggests that the poly-3 adjusted number was used in the footnotes in both Table 1 and Table 3 in the original report.

2. I noted that in Table S2 the adjusted numbers in from.original.report and poly3 are identical at Dose 0 and 1.5 for both CDMA and GSM as well as at Dose 3 for GSM but differ slightly in the other treatment doses for heart schwannomas. One possible cause of the difference is that the version of the raw data in the excel files differs from that used to generate the original report. The second possibility is typ in the footnote in Table 3. I also generated Table S3 that has the poly-6 adjusted numbers for brain gliomas. The two sets of the poly-6 adjusted numbers are ver different.

3. There are a couple of errors in the footnote of Table in the original report. 2/74.05 (5%) should be 2/74.05 (2.7%). 3/78.67 (4%) should be 3/78.67 (3.8%).

Supplementary Information

Table S1. Expected percentage of observing different numbers of tumors in the controls based on binomial distribution.

	0 tumor	1 tumor	2 tumors	>2 tumors
control for glioma	40%	37%	17%	6%
control for heart schwannoma	43%	37%	15%	5%

The percentage was calculated with 1.7% historical control rate for male rats (gliomas) and with poly-6 adjusted animal number, 53. Similarly, the percentage was calculated with 1.3% historical control rate for male (heart schwannoma) and with poly-3 adjusted animal number, 65.

Table S2. The poly-3 adjusted rat numbers in Table in the original report and those calculated from the raw data.

RFR	Dose	from.original.report	poly3
CDMA	0	65.47	65.47
CDMA	1.5	74.05	74.05
CDMA	3	78.67	78.35
CDMA	6	67.94	66.24
GSM	0	65.47	65.47
GSM	1.5	74.87	74.87
GSM	3	77.89	77.89
GSM	6	78.48	77.66

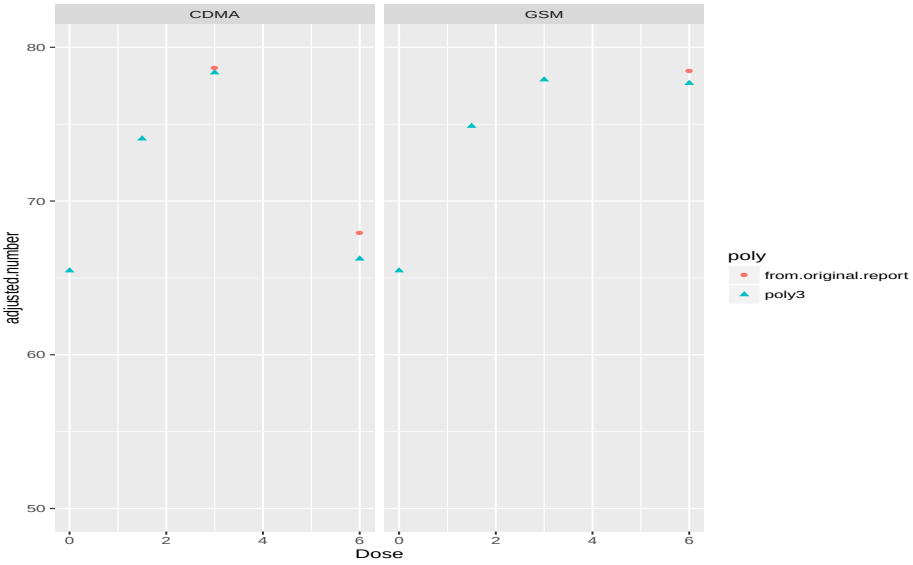
The numbers in from.original.report refers to the poly-3 adjusted rat number from Table 3 in the original report. The numbers in poly3 refers to the poly-3 adjusted rat numbers that I calculated from the raw data for heart schwannoma.

Table S3. The poly-6 adjusted rat numbers in Table in the original report and those calculated from the raw data.

RFR	Dose	from.original.report	poly6
CDMA	0	65.47	53.48
CDMA	1.5	74.05	65.94
CDMA	3	78.35	73.08
CDMA	6	66.24	57.5
GSM	0	65.47	53.48
GSM	1.5	74.93	67.84
GSM	3	78.27	71.43
GSM	6	77.1	72.55

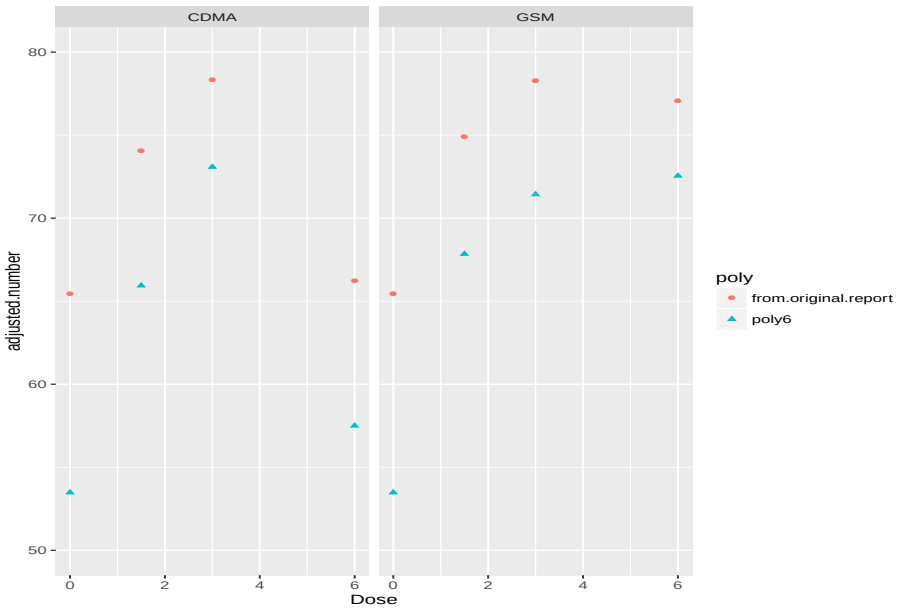
The numbers in from.original.report refers to the poly-6 adjusted rat number from Table 1 in the original report. The numbers in poly6 refers to the poly-6 adjusted rat numbers that I calculated from the raw data for brain gliomas.

Figure S1. Comparison of poly-3 adjusted rat numbers between those from the original report versus those calculated from the raw data.



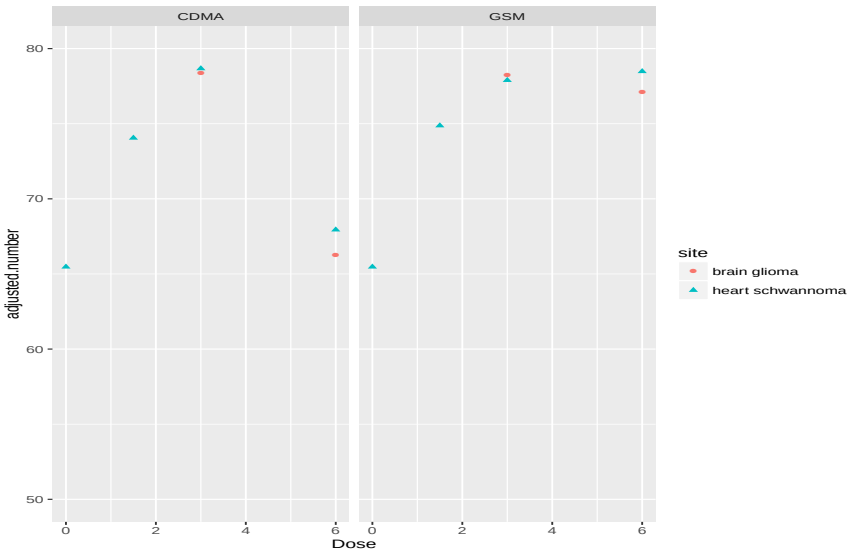
The poly-3 adjusted rat number from Table 3 of the original report is compare with the poly-3 adjusted rat number that I calculated from the raw data for heart schwannomas experiment

Figure S2. Comparison of poly-6 adjusted rat numbers between those from the original report versus those calculated from the raw data.



The poly-6 adjusted rat number from Table 1 of the original report is compare with the poly-6 adjusted rat number that I calculated from the raw data for brain gliomas experiment

Figure S3. Comparison of poly-6 adjusted rat numbers between those from the original report versus those calculated from the raw data.



The adjusted rat numbers from Table 1 and Table 3 of the original report are compared with each other.

Appendix G1: Reviewer's comments

Reviewer: Aleksandra M. Michalowski, M.Sc., Ph.D., Laboratory of Cancer Biology and Genetics, NCI

REVIEWER COMMENTS

Reviewer's Name:
Aleksandra M. Michalowski, Ph.D., M.Sc., National Cancer Institute/LCBG

Report Title:
Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Cell Phone Radiofrequency Radiation (Whole Body Exposures); Draft 3-16-2016

Charge: To peer review the draft report and comment on whether the scientific evidence supports NTP's conclusion(s) for the study findings.

1. Scientific criticisms:

   a. *Please comment on whether the information presented in the draft report, including presentation of data in any tables, is clearly and objectively presented. Please suggest any improvements.*

Overall, the information included in the report is presented in a comprehensive and accurate manner. Specifically, the experimental design and conditions are sufficiently documented and the choice of statistical approaches is explained; the results are well organized and necessary details are provided.

Nevertheless, a few additions could be suggested:

(1) Appendix tables for all poly-k tests performed could be added. I believe this would enhance the presentation of the adjusted rates and the strength of the statistical evidence. As a possible example I prepared the below table using the R package *MCPAN* and its *poly3test()* function.

poly-3	Heart Schwannoma Malignant, Male				Heart Schwannoma Malignant, Female			
CDMA exposure	0	1.5	3	6	0	1.5	3	6
X	0	2	3	6	0	2	0	2
N	90	90	90	90	90	90	90	90
adjusted n	63.8	72.4	77.1	66.6	67.9	71.8	70.3	78.0
Dunnett contrast	–	1.5 - 0	3 - 0	6 - 0	–	1.5 - 0	3 - 0	6 - 0
Estimate	0	0.03	0.04	0.09	0	0.03	0	0.03
Statistic	–	1.24	1.58	2.45	–	1.26	0	1.24
p-value	–	0.2704	0.1542	**0.0209**	–	0.2466	0.7992	0.2562
Williams contrast	–	(6,3,1.5) - 0	(6,3) - 0	6 - 0	–	(6,3,1.5) - 0	(6,3) - 0	6 - 0
Estimate	0	0.05	0.06	0.09	0	0.02	0.01	0.03
Statistic	–	2.78	2.75	2.45	–	1.27	0.88	1.24
p-value	–	**0.0056**	**0.0060**	**0.0138**	–	0.1661	0.2871	0.1744

(2) In the portion of the text describing poly-k test results, p-values are given for significant pairwise comparisons; I would also give the p-values estimated for the significant trends (maximum test).

(3) Information could be included regarding the software or programming environment used for the computations.

(4) In the portion of the text describing differences in survival at the end of the study between control and RFR-exposed animals (page 5§2) the compared characteristic is not named (median survival, TSAC?) and also no numerical values of the estimates or the range of differences are given. I would add numbers in the text or an Appendix table showing the group survival estimates described in this paragraph.

Median survival

CDMA	Female	Male	GSM	Female	Male
0	737	662.5	0	737	662.5
1.5	734	719	1.50	738	729
3	737	731	3	737	730
6	738.5	717	6	738	731

TSAC percentage

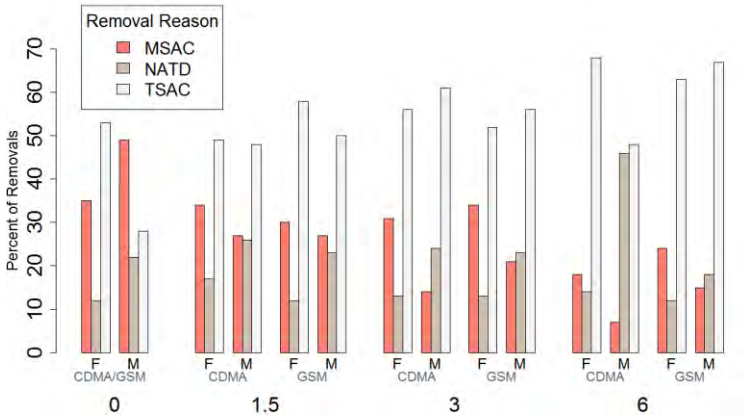
CDMA	Female	Male	GSM	Female	Male
0	53	28	0	53	28
1.5	49	48	1.5	58	50
3	56	61	3	52	56
6	68	48	6	63	67

b. *Please comment on whether NTP's scientific interpretations of the data are objective and reasonable. Please explain why or why not.*

Appropriate statistical design and methods were applied in accord with the FDA/NTP guidelines for conducting long-term rodent carcinogenicity studies and analyses. The results and limiting issues were objectively discussed. The critical issue of shorter survival in the male control group was addressed with regard to the percentage of animals surviving to terminal sacrifice in historical control data (avg. 47%, range 24% to 72%) and the possible impact of the observed age of tumor occurrence on the statistical inference.

I believe detailed information about animal selection and randomization procedures should be given so that the potential for allocation bias could be judged. As shown in the figure below, the lower survival rate to terminal sacrifice (28%) in the male control is accompanied by the higher rate of moribund sacrifice (49%); in the male group exposed to CDMA with 6 W/kg, a higher rate of natural death was observed (46%).

It has been reported that insufficient randomization can lead to differences in survival rates. As an example, in a carcinogenicity study on aspartame it was suggested that lack of randomization to different rooms may have possibly been the cause of low survival rates (27%) in the control female group due to a high background infection rate (EFSA, 2006; Magnuson, B., Williams, G.M., 2008).



2. Please identify any information that should be added or deleted:

A statement of the required statistical significance level should be added. FDA guidance suggests the use of significance levels of 0.025 and 0.005 for tests for positive trends in incidence rates of rare tumors and common tumors, respectively; for testing pairwise differences in tumor incidence the use of significance levels of 0.05 and 0.01 is recommended for rare and common tumors, respectively. If power calculations to determine the required sample size were performed, the results should also be included.

3. The scientific evidence supports NTP's conclusion(s) for the study findings:

The NTP's overall draft conclusion was as follows: "Under the conditions of these studies, the observed hyperplastic lesions and neoplasms outlined in this partial report are considered likely the result of exposures to test article A and test article B. The findings in the heart were statistically stronger than the findings in the brain."

In my view, the results support the conclusion of likely carcinogenic effect of the RFR-exposure on Schwannoma heart lesions in male Harlan Sprague Dawley rats.

Possible carcinogenic effects in the brain are marginal and are not sufficiently supported by statistical evidence in the male Harlan Sprague Dawley rats.

In the female Harlan Sprague Dawley rats very few lesions were observed in either site and statistical significance was not reached at all.

Appendix G1: Reviewer's comments

Reviewer: R. Mark Simpson, D.V.M., Ph.D., Laboratory of Cancer Biology and Genetics, NCI

Analysis of National Toxicology Program (NTP) study evaluating risk in rat lifetime exposure to GSM or CDMA RFR.

Notes:

The NTP study document acknowledges several study limitations [page 10, discussion section]. Potential limitations should prominently factor into considerations regarding the context of the findings, as well as their interpretation and application.

Working list of limitations potentially impacting NTP study interpretations

- Difficulty in achieving diagnostic consensus in lesions classifications of rare, unusual, and incompletely understood lesion association
- Document appears to indicate that the second Pathology Working Group (PWG) empaneled to review and obtain lesion classification consensus, following the inability of the initial PWG to do so, may have reviewed different lesions sets
- No record of clinical disease manifestations due to lesions involving heart and brain [note lesions in heart and brain are mutually exclusive; affected rats have either one or the other and do not appear to have the involvement of both organs together (appendix E)]
- Lesions, including malignancies, do not appear to materially shorten lifespan, except for a subgroup of rats (less than 1/3 of affected rats) with malignant Schwannomas in heart
- Lack of shortened lifespan as a consequence of malignancy for the majority of affected rats contrasts with shortened lifespan of male control rats for which there is absence of attributable cause of death. The survival of the control group of male rats in the current study (28%) was relatively low compared to other recent NTP studies (avg 47%, range 24 to 72%).
    - Creates greater reliance on statistical controlling for survival disparities and reliance on historical controls
- Reliance on historical controls made up of rats of different genetic strain background, held under different environmental conditions
- Absence of data on incidence of more frequently expected tumor occurrences in rats (background lesions)

Documenting the nature of the brain and cardiac lesions observed in RFR exposed rats and placing them into test article exposure-related context, in contrast to potential for their occurring spontaneously, are important and challenging goals. The NTP study limitations make the interpretation of reasonable risk more complicated. NTP acknowledgements of study limitations appear factored into one of NTP's reviewer's study conclusion, i.e., findings represent "some evidence" for a test article effect in statistically significant trend for Schwannomas; an opinion which is coupled with a conclusion for "equivocal evidence" of an effect in relation to malignant gliomas of the brain [NTP Appendix F, Reviewer Comments].

The summation from Appendix F reviewers regarding existence of test article effect is less than conclusive. The NTP study documents a series of cytoproliferative changes

in heart and brain. The nature of some of the changes is challenging diagnostically and appears to be incompletely understood. These findings are presented in the absence of complete analysis of the entire consequences of the study effects. For example, no potential significance for test article effect context is given to any of granular cell proliferative lesions of the brain, a finding mentioned only as a contrast to what was less well understood pathologically (NTP Appendix C, Pathology). It is noteworthy that the lesion types analyzed in the NTP RFR study under review are uncommon historically in rats, in the organs discussed. Furthermore, the malignancies of neuroglia appear to be paired with the occurrence of poorly understood changes involving neuroglial cell hyperplasias in the central and peripheral nervous systems. Little information can be gleaned from the literature about the nature and significance of these latter proliferative changes, interpreted by NTP as nonneoplastic and non-inflammation-reactive neuroglial cell in nature. Although unclear in the NTP study document, it is plausible that the particular lesion constellation, along with the relative novelty of some lesions, contributed to the lack of consensus regarding the nature of the lesions on the part of the initial PWG study pathologists. Concern raised by one of the reviewers (Appendix F, Reviewer Comments) regarding how this difficulty in ability to classify lesions might impact comparisons to historical control lesion incidence data (NTP Table D) is certainly principled.

The extraordinary PWG process, presumably posed by the difficult diagnostic interpretations, has the potential to influence the reliance on historical controls. In this regard, study limitations concerning determination of whether or not there is a test article effect include the substantially poor survival of male rats in the control group. The survival of the control group of male rats in the study under review (28%) was relatively low compared to other recent NTP studies (avg 47%, range 24 to 72%). This apparently led to greater statistical construction to account for the impact of study matched controls, and created increased reliance upon historical data of rare tumor incidences in control animals taken from other chronic carcinogenicity studies. NTP acknowledges a limitation in using the historical incident data and a small study match control group due to poor survivability. There are potential sources of variability when using historical controls of different rat strains and fluctuating study conditions (environment, vehicle, route of exposure, etc.), as is the case here. It seems less than clear what appropriate background lesion incidence is, as NTP indicates some data involve other strains of rats. The range of lesion incidence in historical controls could mean that the true incidence of some lesions varies considerably and might be considered rare or more common depending upon the incidence rate.

The guidance manual on Statistical Aspects of the Design, Analysis and Interpretation of Chronic Rodent Carcinogenicity Studies of Pharmaceuticals by the FDA provided for this review discusses applying comparisons using historical control lesion incidences at some length [beginning page 27, line 996]. Considering lesions as being rare or more common appears to influence selection of the level of statistical significance for comparisons. It appears that analysis for significant differences in tumor incidence between the control and the dose groups for these NTP studies has been established at the 0.05 level (NTP Tables 1,3,5). Interpretations of trend tests may be influenced by the choice of decision rule applied. Such choices can result in

about twice as large overall false positive error as that associated with control-high pairwise comparison tests [page 28, line 1012-1026]. The FDA guidance manual [page 31, line 1136] highlights concern regarding reliance upon historical control incidence data, stating that using historical control data in the interpretation of statistical test results is not very satisfactory because the range of historical control rates is usually too wide. This is especially true in situations in which the historical tumor rates of most studies used are clustered together, but a few other studies give rates far away from the cluster. When the range of historical control data is simply calculated as the difference between the maximum and the minimum of the historical control rates, the range does not consider the shape of the distribution of the rates. These circumstances may impose some limitations on optimal risk assessment designs.

Somewhat paradoxically then, NTP study limitations including that imposed due to reliance upon less than optimal historical control lesion incidence data for much of the comparisons between treated and untreated rats, is confronted by existence of a difficult to classify and incompletely understood lesion constellation interpreted to include neuroglial cell hyperplasia. Notwithstanding, this confounding proliferative lesion occurring in the context along with malignancies of apparently similar histogeneses, sustains a level of concern for a rare injury mechanism related to test article effect. Additional information about the study together with an assessment of the statistical analyses may enhance the value of this analysis.

R. Mark Simpson, D.V.M., Ph.D.

Appendix G2: NTP's Responses to NIH Reviewer's Comments

NTP Responses to Pathology Reviewer' Comments April 12, 2016

Reviewers: R. Mark Simpson, D.V.M., Ph.D. and Diana Copeland Haines, D.V.M.

Responses Relating to the Pathology Review Process

Drafts of the PWG reports are provided. As described in the PWG report, the specific task of the first PWG (January 29th 2016) was to: 1) confirm the presence of glial cell hyperplasia and malignant gliomas in the brain and Schwann cell hyperplasia and schwannomas in the heart; 2) develop specific diagnostic criteria in the brain for distinguishing glial cell hyperplasia from malignant glioma and gliosis, and in the heart for distinguishing between Schwann cell hyperplasia and schwannoma. The PWG participants confirmed the malignant gliomas and schwannomas, but the criteria for distinguishing between hyperplasia and neoplasia differed between the participants.

In order to clearly establish specific diagnostic criteria for the differentiation between hyperplastic and neoplastic lesions in the brain and heart, two additional PWGs were convened. The participants for the second (February 25, 2016) and third (March 3, 2016) PWGs were selected based on their distinguished expertise in the fields of neuropathology and cardiovascular pathology, respectively. Some of the participants were leaders in the International Harmonization of Nomenclature and Diagnostic Criteria initiative. The neuropathology experts of the second PWG confirmed the malignant gliomas in the brain, established diagnostic criteria for glial cell hyperplasia, and agreed that the hyperplastic lesions are within a continuum leading to malignant glioma. The cardiovascular pathology experts of the third PWG established specific diagnostic criteria for Schwann cell hyperplasia and schwannoma in the endocardium and myocardium, and reviewed and confirmed all cases of Schwann cell hyperplasia and schwannoma observed in these studies. The outcome of the PWG provided very high degree of confidence in the diagnoses.

The participants of the first PWG (January 29th 2016) only reviewed a subset of the glial lesions that were observed in the studies. The review for the second PWG (February 25, 2016) included all glial lesions in the studies including the subset that was reviewed in the first PWG.

Responses Relating to Considerations of Historical Control Data

For NTP toxicology and carcinogenicity studies, the concurrent controls are always the primary comparison group. However, historical control information is useful particularly in instances when there is differential survival between controls and exposed groups, as was observed in the RFR studies. Rates for glial cell neoplasms and heart schwannomas from control groups of male Harlan Sprague Dawley rats from other recently completed NTP studies are presented in Appendix D of the 3-16-2016 draft report. While Harlan Sprague Dawley rats are an outbred strain, they are considered single genetic strain in the same sense as other outbred strains, such as the Long-Evans or Wistar rat. Therefore, these historical control tumor rates are applicable to this study. However, it's important to note that the studies listed in Appendix D were carried out at laboratories other than the RFR studies, and under different housing and environmental conditions. At the time of the 3-16-201 draft report, not all of these studies had undergone a complete pathology peer review. In the past several weeks NTP pathologists have reviewed brain and heart slides from these male rat control groups, and have confirmed, with few exceptions, the low rates of hyperplastic and neoplastic lesions reported in Appendix D, applying the diagnostic criteria established during the PWGs outlined in Appendix C.

NTP Comments on Statistical Issues Raised by the Reviewers April 12, 2016

Given the multiple comparisons inherent in this kind of work, there is a high risk of false positive discoveries (Michael S. Lauer).

Although the NTP conducts statistical tests on multiple cancer endpoints in any given study, numerous authors have shown that the study-wide false positive rate does not greatly exceed 0.05 (Fears et al., 1977; Haseman, 1983; Office of Science and Technology Policy, 1985; Haseman, 1990; Haseman and Elwell, 1996; Lin and Rahman, 1998; Rahman and Lin, 2008; Kissling et al., 2014). One reason for this is that NTP's carcinogenicity decisions are not based solely on statistics and in many instances statistically significant findings are not concluded to be due to the test agent. Many factors go into this determination including whether there were pre-neoplastic lesions, whether there was a dose-response relationship, biological plausibility, background rates and variability of the tumor, etc. Additionally, with rare tumors especially, the actual false positive rate of each individual test is well below 0.05, due to the discrete nature of the data, so the cumulative false positive rate from many such tests is less than person would expect by multiplying 0.05 by the number of tests conducted (Fears et al., 1977; Haseman, 1983; Kissling et al., 2015).

I'm getting slightly different values for poly-k adjusted denominators (Michael S. Lauer).

I compared poly---3 adjusted number from Table 3 in the original report versus the poly---3 adjusted number that I calculated using the raw data from the excel files. Supplementary Figure S1 shows that these two sets of numbers agree with each other in general. This is in contrast to the comparison for poly---6 adjusted number from Table 1 in the original report versus the poly---6 adjusted number that I calculated using the raw data from the excel files (Supplementary Figure S2). In fact, the adjusted rat numbers from Table 1 and Table 3 of the original report look quite similar (Supplementary Figure S3). This suggests that the poly---3 adjusted number was used in the footnotes in both Table 1 and Table 3 in the original report. (Max Lee)

I noted that in Table S2 the adjusted numbers in from.original.report and poly3 are identical at Dose 0 and 1.5 for both CDMA and GSM as well as at Dose 3 for GSM but differ slightly in the other treatment doses for heart schwannomas. One possible cause of the difference is that the version of the raw data in the excel files differs from that used to generate the original report. The second possibility is typo in the footnote in Table 3. I also generated Table S3 that has the poly---6 adjusted numbers for brain gliomas. The two sets of the poly---6 adjusted numbers are very different. (Max Lee)

Information could be included regarding the software or programming environment used for the computations. (Aleksandra M. Michalowski)

The adjusted denominators in Table of the original report were labeled as poly-6 denominators, but were actually poly-3 denominators. This error was noted and brought to Dr Tabak's attention by Dr. Bucher in a March 22 email.

The p-values and adjusted denominators calculated by NTP are correct, except as noted for Table 1, and were calculated using validated poly-k software. This software is coded in Java and is embedded within NTP's TDMSE (Toxicology Data Management System Enterprise) system. Poly-k

calculations conducted by the reviewers in R may vary slightly from the NTP's calculation due to selection of study length and the NTP's use of the Bieler-Williams variance adjustment and a continuity correction. In his calculations, Dr. Lauer used 90 weeks as the study length, whereas the actual study length was 10 weeks. It is not apparent from the R documentation that the Bieler-Williams adjustment or the continuity correction is incorporated into the poly-3 calculations in R. In his calculations, Dr. Lee used two-sided p-values. In NTP statistical tests for carcinogenicity, the expectation is that if the test article is carcinogenic, tumor rates should increase with increasing exposure; thus, the NTP employs one-sided tests and p-values are one-sided. Using one-sided p-values in Dr. Lee's Table 1, the GSM trend if there were brain glioma in the control group remains nonsignificant, but the CDMA trend approaches 0.05 (p = 0.054) if there were brain glioma in the control group. In Dr. Lee's Table 2, the one-sided p-value for the GSM trend if there were 1 heart schwannoma in the control group approaches 0.05 (p = 0.054) and the one-sided p-value for the CDMA trend in heart schwannomas remains significant at = 0.018 if there were 1 heart schwannoma in the control group. In Dr. Lee's Table 3, the one-sided p-value for the CDMA pairwise comparison is significant at p = 0.049 if there were 1 heart schwannoma in the control group.

statement of the required statistical significance level should be added. FDA guidance suggests the use of significance levels of 0.025 an 0.005 for tests for positive trends in incidence rates of rare tumors and common tumors, respectively; for testing pairwise differences in tumor incidence the use of significance levels of 0.05 and 0.01 is recommended for rare and common tumors, respectively. (Aleksandra M. Michalowski)

Although the FDA guidance suggests lowering the significance level for most tests of trend and pairwise differences, this guidance is based o a misunderstanding of findings reported by Haseman (1983). In this paper, Haseman discusses several rules proposed by others for setting the significance level lower than 0.05. *If* these rules are rigidly followed, Haseman showed that study conclusions will be consistent with the NTP's more complex decision-making process, for which 0.05 is the nominal significance level and p-values are taken into consideration along with other factors (outlined above in response to comment 1) in determining whether the tumor increase is biologically significant. The NTP does not strictly adhere to specific statistical significance level in determining whether a carcinogenic effect is present.

Appendix tables for all poly-k tests performed could be added. (Aleksandra M. Michalowski)

Dr. Michalowski proposed a sample table. The rows corresponding to X, N, adjusted n are already included in the tables or appear the footnotes in the tables. The rows corresponding to "Dunnett contrast" and "Williams contrast" are not appropriate for dichotomous tumor data. Both Dunnett's test and Williams' test assume that the data are continuous and normally distributed.

In the portion of the text describing poly-k test results, p-values are given for significant pairwise comparisons; I would also give the p-values estimated for the significant trends. (Aleksandra M. Michalowski)

Indicators of significant trends are given in the tables in the form of asterisks next to control group tumor counts.

There are a couple of errors in the footnote of Table 3 in the original report. 2/74.05 (5%) should be 2/74.05 (2.7%). 3/78.67 (4%) should be 3/78.67 (3.8%). (Max Lee)

Thank you for pointing this out. The percentages will be corrected in our final report.

Were control rats selected in utero like the exposed rats were? Were pregnant dams assigned to different groups by formal randomization? How were the pups per litter chosen? (Michael S. Lauer).

believe detailed information about animal selection and randomization procedures should be given so that the potential for allocation bias could be judged. (Aleksandra M. Michalowski)

Pregnant dams were assigned to groups, including the control group, using formal randomization that sought to also equalize mean body weights across groups. The three pups per sex per litter were selected using formal randomization, as well. Tumors in the heart and brain were not observed in littermates, indicating that there was no litter-based bias in the results.

Were all analyses based on the intent-to-treat principle? Were there any crossovers? Were all rats accounted for by the end of the experiment an were all rats who started in the experiment included in the final analyses? (Michael S. Lauer)

The intent-to-treat principle is not relevant to this animal experiment, in which all animals that were assigned to treatment group received the full and equal treatment of that group. There were no crossovers. All animals that started the experiment were accounted for by the end of the experiment and included in the final analyses.

The PWG review blinding was not complete. (Michael S. Lauer)

PWG reviewers were blinded to the identity of the test article and the level of exposure but were not blinded to the fact that there were two different, yet related, test articles (modulations of cell phone RFR), to emphasize the fact that there was a common control group.

Did the authors perform a prospective sample size calculation? (Michael S. Lauer)

If power calculations to determine the required sample size were performed, the results should also be included. (Aleksandra M. Michalowski)

Sample size calculations were conducted for this study. However, for detecting carcinogenesis, sample size and power will depend on the baseline (control) tumor rate and the expected magnitude of the increase in tumors. For example, at 80% power, sample size requirements will be quite different for detecting a 2-fold increase in a rare tumor having a spontaneous occurrence of 0.5% compared to 2-fold increase in a more common tumor having a spontaneous occurrence of 10%. Because many different tumor types having wide range of spontaneous occurrence are involved in these studies, there is no “one-size-fits-all” sample size; rather, the sample size is a

compromise among several factors, including obtaining reasonable power to detect moderate to large increases for most tumor types, while staying within budgets of time, space, and funding. A sample of 90 animals per sex per group was selected as providing as much statistical power as possible across the spectrum of tumors, under the constraints imposed by the exposure system.

The NTP's carcinogenicity studies are similar in structure to the OECD's 45 Guideline for carcinogenicity studies and the FDA's guidance for rodent carcinogenicity studies of pharmaceuticals. These guidelines recommend at least 50 animals of each sex per group, but also mention that an increase in group size provides relatively little increase in statistical power. In the NTP's RFR studies, the group sizes were 90 animals of each sex per group, nearly twice as many as the minimum recommendation. Increasing the group sizes further provides diminishing returns, for which additional animals do not substantially increase power.

The low power implies that there is high risk of false positive findings (citing Ioannidis, 2005). … suspect that this experiment is substantially underpowered and that the few positive results found reflect false positive findings (citing Ioannidis, 2005). (Michael S. Lauer)

It is true that the power is low for detecting moderate increases above a low background tumor rate of approximately – %, as was seen in the brain and heart tumors. However, this low power does not correspond to high risk of false positive findings. The paper by Ioannidis that was cited correctly states that when studies are small or effect sizes are small (i.e., statistical power is low), "the less likely the research findings are to be true." Research findings can be "not true" if the result is a false positive or a false negative. With low statistical power, false negatives are much more likely than false positives. Therefore, the vast majority of false research findings in a low power situation will result from the failure to detect an effect when it exists. The false positive rate on any properly constructed statistical test will not exceed its significance level, alpha. By definition, the significance level of a statistical test is its false positive rate, and it is typically selected by the researcher, often at a low fixed value such as 0.05 or 5%.

If we were repeating the experiment, we may see some control studies have 1 or more tumors. (Max Lee) (Dr. Lee also presented analyses of the male rat data, inserting hypothetical data on one tumor-bearing animal in the control group.)

In light of the historical control data, Dr. Lee demonstrated that several associations became less or not significant with the insertion of a tumor data point in the control group. While we appreciate that some other studies had one or more tumors, the NTP considers the concurrent control group as the most important comparator to the treated groups. We took the historical control tumor rates into account in a more subjective manner in our interpretation of the findings. In 2010, we asked to adopt more formal method of incorporating historical control data in our statistical testing, but our Board of Scientific Counselors voted against adopting the method.

It is puzzling why the control had short survival rate. Given that most of the gliomas and heart schwannomas are late-developing tumors, it is possible that if the controls were living longer some tumors might develop. Although the use of poly-3 (or poly-6) test intended to adjust the number of rats

used in the study, it is still important to re-evaluate the analysis by considering the incidence rate in controls not being 0. (Max Lee)

We do not know why the male rat control group had a low survival rate. We generally d observe lower survival rates in studies such as the RFR studies in which animals are singly- rather than group housed. While some tumors might possibly have arisen in controls if they lived longer, it was notable that n glial cell or Schwann cell hyperplasias were found in these animals as well.

The poly-k (e.g., poly-3 or poly-6) test was developed to adjust for the fact that not all animals survive to the end of a two-year study, and survival rates may differ among groups. The test is essentially Cochran-Armitage trend test in which the denominator of the tumor rate in each group is adjusted downward to better reflect the number of animal-years at risk during the study. Each animal that develops the tumor or survives to the end of the study is counted as one animal. Each animal that does not develop the tumor and dies (or is moribund sacrificed) before the end of the study is counted as a fractional animal. The fraction is calculated as the proportion of the study that it survived, raised to the k-th power; = 3 or = 6 in this study. The survival-adjusted tumor rate in each group is then the number of animals having the tumor of interest divided by the total count of animals at risk of developing the tumor in the group. These survival-adjusted rates are used in the Cochran-Armitage formula to provide the poly-k test for dose-related trends and pairwise comparisons with the control group.

The poly-k test has been shown to yield valid inferences about tumor rates in NTP two-year rat and mouse carcinogenicity studies (Bailer and Portier, 1988; Portier and Bailer, 1989; Portier et al., 1986). Its theoretical basis is that tumor incidence, while not directly observed unless the tumor is immediately lethal, follows a Weibull distribution with a shape parameter, k. Verification using NTP studies has shown that if k is between 1 and 5, setting k = 3 yields a valid statistical test (Portier and Bailer, 1989; Portier et al, 1986). Thus, most of the time, the NTP uses the poly-3 test. If tumor type is late-occurring, as we observed with the brain gliomas, k = 6 is a better fit to the data and the poly-6 test has more validity.

In the portion of the text describing differences in survival at the end of the study between control and RFR-exposed animals the compared characteristic is not named an also n numerical values of the estimates or the range of differences are given. I would ad numbers in the text of a Appendix table showing the group survival estimates described in this paragraph. (Aleksandra M. Michalowski)

The Statistical Methods section describes the method for comparing survival distributions between the control and RFR-exposed groups, namely, Tarone's (1975) life table test to identify exposure-related trends in survival and Cox's (1972) method for testing two groups for equality of survival distributions.

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ADDITIONAL RESPONSE:

Dear All,

Thanks again for all your helpful comments on the NTP RFR studies. I did want to follow up on one remaining point of disagreement that Mike Lauer alluded to in his comments about low powered studies. Although we agree that our study design had low power to detect statistically significant neoplastic effects in the brain and heart, which occurred with both RFR modulations in male rats, we disagree over the assertion that low power in and of itself, creates false positive results. We cited a handful of publications outlining the statistical arguments against this with specific respect to the NTP rodent cancer study design in our response to comments document sent earlier. Although Mike referred to the example of positive findings in underpowered epidemiology studies that could not be replicated in larger follow up studies, there is a growing literature alluding to this problem with respect to experimental animal studies as well. An example is a relatively recent article by one of our collaborators in CAMARADES, Malcolm MacLeod.

http://www.nature.com/news/2011/110928/full/477511a.html

It's important to distinguish between low power to detect effects, and the constellation of other factors that often accompany low powered experimental animal studies in contributing to this problem. We've addressed this issue in a recent editorial, and these factors are captured in our published systematic review process for evaluating study quality in environmental health sciences (Rooney et al., 2014).

http://ehp.niehs.nih.gov/wp-content/uploads/122/7/ehp.1408671.pdf

http://ehp.niehs.nih.gov/wp-content/uploads/122/7/ehp.1307972.pdf

Table 1 in the Rooney et al. report outlines risk of bias considerations that commonly plague studies carried out by academic researchers that are accounted for in NTP studies.

I provide these examples to assure you that we are completely cognizant of these issues and take them very seriously. Again, we appreciate the help you've provided in assuring that we appropriately interpret and communicate our findings.

Best
John Bucher

Carnahan, David

From: Sea <paloaltolife@gmail.com>
Sent: Friday, March 09, 2018 8:08 AM
To: ctraboard@googlegroups.com; fbalin@gmail.com; schmitta@pacbell.net; madjensen@sbcglobal.net; Summa, Doria; rstolee@gmail.com; estolee@gmail.com; Keene, James; Council, City; mikez@siliconvalleybuilder.com
Subject: A great thriving market Foothill Produce in Los Altos potential for 2100 El Camino Real

Please pursue, invite, discuss, consider. Contact Saira 650-919-3459 daughter and father owners. she is a UCLA graduate. They own/operates 3 plus stores. Great produce from farm to town with great prices like milkPail de martini segona. Community will benefit from having fresh produce and lower prices to be affordable to renters students and stanford based foreign and visitors.

Again, Foothill Produce, Phillipe markets; Nearest is one on Foothill expressway south Los Altos near cross section of Homestead and Foothill expressway shopping center next to chevron gas trader Joe's.

Regards

SeReddy
Innovation•Integrity•Inclusion.
Walk the Talk

747 Stanford Avenue
Palo Alto Ca 94306
Paloaltolife@gmail.com
650-465-3535
949-857-2000

Sent from my iPhone


PRODUCE


TRADER JOE'S

Carnahan, David

From: Margaret Rosenbloom <margaret_rosenbloom@hotmail.com>
Sent: Tuesday, March 13, 2018 11:21 AM
To: Planning Commission
Cc: Council, City
Subject: Affordable housing for Palo Alto

I am writing to express my support for the Affordable Housing Overlay as written, as this will make it possible to develop the affordable housing our city so badly needs. The proposed amendments re distance from transit, amount of parking, height, and low income eligibility levels may meet the interests of some in the city but will cause delay and diminishment in what could be a major breakthrough in Palo Alto's housing impasse.

Palo Alto can not afford to remain stuck in a mindset of being a small town but must face the reality and demands of the current housing-jobs crunch. The Affordable Housing Overlay is a significant effort towards this goal and I urge the PTC to refer the ordnance as written to the City Council for their consideration and action under guidelines of the Comprehensive Plan.

Margaret J. Rosenbloom 650-328-1712

Carnahan, David

From: Aram James <abjpd1@gmail.com>
Sent: Sunday, March 11, 2018 1:06 PM
To: Council, City
Cc: Van Der Zwaag, Minka; council@redwoodcity.org; jseybert@redwoodcity.org; gkirby@redwoodcity.org; Perron, Zachary; dcbertini@menlopark.org; citycouncil@menlopark.org; Watson, Ron
Subject: Aram James (DJ-1-12-18).pdf
Attachments: Aram James (DJ-1-12-18).pdf

FYI:
>
> Hi Minka,
>
> I would very much like the Palo Alto Human Relationship Commission to draft a letter, to Santa Clara County Sheriff Laurie Smith, and to the Santa Clara County Board of Supervisors, opposing the purchase and use of Tasers in our jails. Thanks so much.
>
> Best regards,
>
> Aram B. James
> 415-370-5056
>
> P.S. Please see our letter below setting forth our reasons for opposing the use of Tasers in our jails. I provided a hard copy of our letter to the HRC at Thursday's meeting, and requested that the HRC consider writing a letter as suggested above. There was no response to my request.
>
>
>
> Sent from my iPhone

LOS ANGELES & SAN FRANCISCO
Daily Journal
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FRIDAY, JANUARY 12, 2018
PERSPECTIVE

There's no excuse for Taser use in our jails

By Aram James
and Richard Konda


New York Times News Service

Watchdogs across the country are organizing to oppose police practices that run contrary to community values and our constitutional rights. In Santa Clara County in the spring of 2017, Sheriff Laurie Smith, a longtime opponent of Tasers, in a surprising shift of policy, announced her plan to introduce Tasers into the Santa Clara County jails. Local civil rights organizations immediately began organizing to resist the sheriff's call for Tasers.

One of our first strategies was to ensure that members of the community were as fully informed as possible regarding the risks that Tasers pose to human life. We reviewed materials on Tasers and got ourselves current on the nuances of the issue. Next, we scheduled meetings over a seven-month period with key elected and non-elected officials who we felt could influence the sheriff's decision. This included members of the Santa Clara County Board of Supervisors who ultimately will vote to decide whether the sheriff will be allowed to purchase Tasers. We also met with the elected Santa Clara County district attorney, the Santa Clara County public defender and the county counsel. And most importantly, we met with the sheriff and her staff to open up a dialogue on this critical issue.

Here are some of the arguments and information we provided much of which came from a recent fivepart series by Reuters ("Shock Tactics: Inside the Taser, the Weapon that Transformed Policing") and the Bar Association of San Francisco.

Taser-Related Deaths and Litigation

Critically important to convincing our sheriff of the inappropriateness of bringing Tasers to the jails is Reuter's recent finding that the death toll associated with Tasers is substantially more than previously reported by mainstream civil rights organizations like Amnesty International. Using rigorous journalistic standards, Reuters documented 1,005 deaths related to Taser use by law enforcement.

In addition, Reuters completed a thorough examination of the monies paid out by cities across the country in Taser related litigation. Reuters identified and reviewed 442 wrongful death lawsuits in which Tasers were a factor that may have caused death. "In 120 of the 442 cases or 27%, the Taser was the only force alleged in the claim; in the remaining 322 cases, the stun guns were alleged to have been part of a broader array of police force. More than three-fifths of the 366 of the concluded lawsuits against governments, or 232, resulted in judgments or settlements for the plaintiffs: 220 settlements and 12 judgments. *Reuters was able to determine payouts in 193 cases, totaling $172 million paid by cities and their insurers*. That dollar figure does not include three dozen cases in which settlements remained confidential or were unavailable." (Emphasis added.)

These findings regarding the cost of litigation should trouble any law enforcement agency, city or county contemplating the purchase of Tasers.

Taser Warnings

Historically the manufacturer had very few warnings regarding the safety of its weapon. Increasingly and in order to shift liability to cities and police departments, Taser now has a 4,500-word, seven-page warning. The warning advises users not to deploy the Taser in the area of the face, eyes, neck, chest, heart and the genitals. And not to Taser a variety of populations including the frail, mentally ill, pregnant women and those with heart problems. *By warning police departments regarding the risk of death and serious injury when a Taser is improperly used, the manufacturer has effectively shifted liability from itself to police departments and municipalities.*

Reuters also explored in detail the progression of Taser warnings that includes a comprehensive interactive guide.

The progression of increasingly restrictive warnings issued by Taser has led some police agencies to either shelve Tasers all together or not to purchase them at all after having reviewed the extensive warnings. Ed Davis, former Boston police chief from 2006-2013, in ultimately declining to purchase Tasers for his department said the following: The warnings "made the weapon impractical to use, and it gave a lot of us the impression that we weren't getting the full story. I didn't want to take the risk. The potential litigation costs absolutely were a factor."

The tragic death of Everette Howard, a young African-American student, is a case examined in the Reuter's series. One Taser blast by University of Cincinnati police officer Richard Haas, a certified Taser instructor, resulted in Everette Howard's death. "Haas fired his stun gun. One electrified dart hit below Howard's lower left chest, the other near his waist. The 18 year-old collapsed, unconscious, and was pronounced dead at the hospital." Haas subsequently said, "I did not in my wildest dreams expect this kid to die."

As part of his role as a certified Taser trainer, Haas acknowledged that he had studied the Taser safety warnings over a 10-year period and noted that they had become more complex over the years. Ironically, the Taser blast that killed Everette Howard was the first time Haas had deployed a Taser in the field. He ultimately concluded, "it seemed like it was getting harder and harder to use the Taser." The University of Cincinnati ultimately settled the Howard's family wrongful death lawsuit for $2 million. Taser was not sued in the matter.

In another case explored by Reuters, Linwood Lambert was

tasered some 20 times by South Boston, Virginia, police officers. He died. There was substantial evidence that the three officers involved ignored the manufacturer's warning regarding the risk of repeatedly tasering victims. In addition, the officers ignored other warnings issued by the manufacturer. Under oath at a deposition, one of three officers involved, Corporal Tiffany Bratton, acknowledged that she was aware of the manufacturer's warnings. In a chilling statement, she said, "If I read and abided by every single warning ... I would not Taser anyone."

Catch-22

More and more attention is being paid by commentators to the fact that the use of Tasers is a Catch-22. Failure by police departments to follow closely the ever growing restrictions on the use of Tasers issued by the manufacturer has resulted in unnecessary deaths and a huge increase in the costs of litigation borne by municipalities. On the other hand, where police departments are closely complying with the manufacturer's complex warnings, they are finding it increasingly impractical to use Tasers. The Oakland Police Department has over 700 police officers on their force, all are armed with Tasers. The Bar Association of San Francisco Criminal Justice Task Force, Committee on Tasers contacted the Oakland Police Department to determine how frequently Tasers were deployed.

"To help answer some of the questions, the BASF also reached out to the Oakland Police Department (OPD) to determine how often Tasers are used, and how often they are effective. It is well known that LAPD reports 47% efficacy, but LAPD far exceeds the size of SFPD. The OPD which is closer in size to the SFPD, reported that in 2015 Tasers were deployed on just 37 occasions and 32 times in 2016. Oakland reported for each year, the efficacy was 50%." Other studies have confirmed that where warnings are complied with the use of Tasers drops dramatically. Similarly, numerous studies have confirmed that Tasers have an unacceptably high failure rate putting both the officers and intended victim at risk.

Moreover, Tasers are not effective. Michael Leonesio, a retired Oakland peace officer, provided answers to questions posed by the Bar Association of San Francisco. "Given the warnings issued by Taser International, does this diminish the weapon's efficacy and/or circumstances otherwise warranting Taser use[?] … Answer: The latest manufacturer warnings and trainings, as well as the Courts and current case law decisions, have absolutely limited the circumstances when a TASER, can and/or, should be used. Combine this with the fact that the new generation weapons are generating only half the electrical output of the previous generations, and I question the current weapons' ability for consistent, reliable, subject incapacitation."

Worth the Cost?

In June 2017, Taser expert Michael Leonesio, was called as an expert witness before the San Francisco Police Commission on the potential costs of outfitting all members of the SFPD with Tasers. "During his testimony, he estimated the first year in costs to San Francisco at $8,000 to $10,000 per officer which included the purchase price, maintenance, training and oversight. Assuming a department size of 2,200 officers, the cost is between $17.6 million and $22 million." Clearly, the sheriff and the Santa Clara County Board of Supervisors need to consider the cost factors raised above before expending millions of tax payer dollars on a weapon that is increasingly seen as impractical to use.

Final Argument

Tasers kill on the average of one person per week in the United States. According to the Reuters series, nine out of 10 who die are unarmed. Tasers are unsafe to use in jails because of the substantial risk of injury or death to both inmates and correction officers. The strongest single piece of evidence of this lack of safety is the 1,005 Taser related deaths reported in the Reuters fivepart series on Tasers. Equally powerful evidence of why Tasers should be banned is the ever growing list of restrictions/warnings issued by the manufacturer themselves regarding the serious risks of injury and death related to the use of Tasers.

The millions that would be spent in arming the correctional officers in the jails with Tasers would be better spent on hiring more and better trained correctional officers. Finally, given the recommendations of the Santa Clara County Blue Ribbon Commission on Improving Custody Operations, the purchase and use of Tasers in the jails runs counter to the community's loud and repeated calls for a more humane approach to incarceration.

Call to Action

When your community is faced with a questionable police practice be it the use of Tasers, inhumane jail conditions, unconstitutional surveillance tactics, racially discriminatory police enforcement; be confident that there is a way to organize your community to effectively challenge these issues. Meet early and often with the community and with your local elected officials. Provide them with the necessary information to fully educate them on the issues. Call on your local district attorney, who is the chief, law enforcement officer in every community, to support your efforts to challenge and end police practices that diminish public trust for local law enforcement. Remember police practices are not some obscure body of knowledge that we the community need sit back and passively accept. We can in fact make a difference.

Aram James *is a retired Santa Clara County deputy public defender, a member of CJA and a co-founder of the Albert Cobarrubias Justice Project (ACJP), a grassroots legal advocacy organization located in San Jose.*

Richard Konda *is an attorney and executive director of the Asian Law Alliance and the Chairperson of the Coalition for Justice and Accountability (CJA). Konda and James have challenged the use of Tasers by law enforcement for more than a decade.*

Carnahan, David

From: Debbie Nichols <debbiegailnichols@gmail.com>
Sent: Tuesday, March 13, 2018 4:14 PM
To: Kniss, Liz (external); Council, City; Lait, Jonathan; Gitelman, Hillary; Keene, James
Subject: Baptist Church CUP Request

Dear Mayor Kniss, Palo Alto City Council, Jonathan Lait, Hillary Gitelman, and City Manager James Keene

I am writing to voice my opposition to the Community Center designation for the Baptist Church, 305 N California Ave., Palo Alto.

The church is essentially operating as a "for profit" commercial building in Old Palo Alto. On Sundays, the church has less than 30 parishioners attending services.

Out of curiosity, I googled 305 N California Ave (the address of the church) and found 44 organizations and groups that are currently or have recently operated out of the church on a weekly or multi weekly basis. Below is a list of the groups. I believe this list is only the tip of the iceberg of the multitude of activities at the church.
>>>>>>>>>>>>
>>>>>>>>>>>> Tuesday Night Tango

Dances with Latin Flair
TH Tango
Klezmer Dance Party
Interlocked Square Dances
>>>>>>>>>>>> Justice in Palo Alto
Dance Maven
Joy Dance
Meatless Monday Dinners
>>>>>>>>>>>> Community Childcare
>>>>>>>>>>>> Synapse School
>>>>>>>>>>>> Palo Alto Mens Group
>>>>>>>>>>>> Peninsula Peace and Justice Center Peninsula English
>>>>>>>>>>>> Country Dance Do the Bay Foursquare Concerts Raggazzi Boys
>>>>>>>>>>>> Chorus Dr Joellen Werne LIVE Silicon Valley Arts and
>>>>>>>>>>>> Entertainment Palo Alto New Church Lots of concerts from
>>>>>>>>>>>> all sorts of groups, such as Tangos for Piano Gourmet
>>>>>>>>>>>> Vegetarian Dinners Nordic Footnotes Scandinavian Dance
>>>>>>>>>>>> Parties Stanford International Folk Dances Dr Jill Cooper,
>>>>>>>>>>>> MFT Peninsula Macrobiotic Community Pacific Association of
>>>>>>>>>>>> Challenge Enthusiasts Battfield Without Borders
Mozart Music School
>>>>>>>>>>>> Cities for CEDAW, the UN, and Women's Activism Globally
>>>>>>>>>>>> Multifaith Prayers for Peace Senior Book Club Benefit
>>>>>>>>>>>> Concert for Irans Quake Victims Dr John Smolowe Palo Alto
>>>>>>>>>>>> Philharmonic Fall Chamber Concert Baroque Music Concert
>>>>>>>>>>>> Peng Piano Academy Festival East European Folklife Center
>>>>>>>>>>>> Bay Choral Guild rehearsals Corey Head, Voice Teacher
>>>>>>>>>>>> rehearsal schedule Resounding Achord Productions The Happy

>>>>>>>>>>>> Body
ISing
>>>>>>>>>>>> Mosaic South Bay

The church has so many non-church related activities that it has removed the pews from the chapel to essentially make it a multipurpose room.
Also, no church staff member or private security person is at the church during evening events. The church is unattended.

The church parking lot only has 5 parking spaces. The PA Planning Dept told a neighbor that the church sold off its parking lot many years ago. As a result, the church inconveniences the neighbors and creates hazardous conditions with cars double parked in the Bryant Street Bike Boulevard lane, cars that are idling, and cars blocking homeowners driveways.

If the church became a community center, especially with functions until 11 pm on weekends, who would monitor the activities? Who could neighbors call for noise infractions late at night? No city staff employee is available after 5 pm.

I urge the City Council to deny the CUP request. Thank you.

Debbie Nichols
Resident, Old Palo Alto

>>>>>>>>>>>>
>>>>>>>>>

Carnahan, David

From: Eric Nordman <eric.nordman12@gmail.com>
Sent: Monday, March 12, 2018 3:44 PM
To: Council, City
Subject: Bike and Pedestrian Project Funding
Attachments: 20180312_Letter to City Council.pdf

City Council:
Attached is my letter in pdf form.
Sincerely,
Eric Nordman

Letter to City Council: Bicycle and Pedestrian Improvements.
March 12, 2018

Dear Council Members:

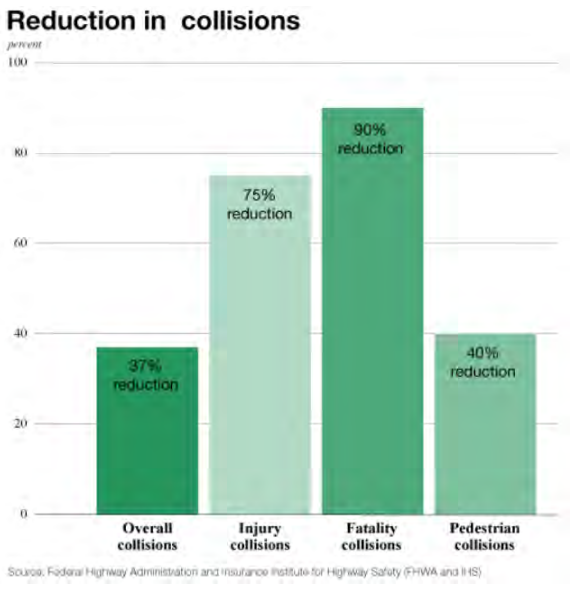
The comprehensive plan states: "The key to a sustainable transportation system lies in providing more options and more convenience so that people will more readily choose not to drive." Bicycle and pedestrian improvements are about as sustainable as one can get.

With rising construction costs and new unbudgeted projects there is financial pressure to cut or delay implementation of projects.

In the echo chamber of social media, the Ross Road Bicycle Boulevard changes are being heavily criticized before they are even completed.

The changes on Ross Road are more extensive than those on Bryant. Some of this is due to efforts to improve pedestrian safety. However, some is due to the direction by city council to not use diversions which were the key element on Bryant. Diversions are harder to implement in areas of the city that have few parallel streets and may not have been appropriate for Ross. However, to reduce costs for future Bike Boulevard projects, Council should lift this prohibition.

I understand there is now a petition to stop the roundabouts with the claim that they are unsafe. That is not the situation elsewhere. Seattle has installed over 1000 mini-roundabouts or traffic circles in the last 30 years and has seen a crash reduction of more than 70%. To the right is statistics from the FHWA. From Palo Alto's Comprehensive Plan: "Traffic circles have been shown to reduce collisions and are considered more bicycle-friendly than traditional two- or four-way stops controls."



Bryant was contentious when it was built, but has proven to be highly successful. On Bryant, bicyclists typically ride in a straight line to the left of parked cars. Cars, pass on the left as they would another car. It's a safe and

low stress situation for both bicyclists and drivers. While change is hard, I'm hopeful that in a year or so. the users of Ross Road will adopt similar habits.

Another critical bike and pedestrian project is the Charleston/Arastradero corridor which was passed unanimously by council. It also has driver safety implications, especially to the neighborhoods south of Charleston. This project has been in development for well over a decade and should be implemented.

Please continue implementation of the 2012 Bicycle and Pedestrian Transportation Plan.

Sincerely,

Eric Nordman

Carnahan, David

From: Arnout Boelens <a.m.p.boelens@gmail.com>
Sent: Tuesday, March 13, 2018 10:13 AM
To: Council, City
Cc: Ben@bikesiliconvalley.org
Subject: Bike Projects Funding

Dear city councel,

My name is Arnout Boelens and my wife and I have been living in Palo Alto for slightly over a year. I commute daily from Midtown to Stanford University by bike and my wife from Midtown to Downtown Palo Alto.

First of all I would like to thank you for your work to make Palo Alto a great biking city. My wife and I actually do not own a car and only use our bikes to get around. Whenever I do drive around in a car I realize that thanks to the bike boulevards biking is the quickest and least stressful way to get around town. Of course the weather helps too.

To get even more people on bikes and reduce traffic congestion I think it is essential that the Palo Alto moves forward with the bike projects on Amarillo Avenue, Bryant Street, East Meadow Drive, Montrose Avenue, Moreno Avenue, Louis Road, Palo Alto Avenue, and Ross Road. I feel especially a good connection to the new Highway 101 over crossing is important for people visiting the Baylands and people commuting to Google, Intuit, and many more companies by bike. If people do not feel safe during part of their commute because there are no traffic calming measures they might rather go by car, adding to to already bad rush hour traffic. Also the connection of the Bryant bike boulevard to Mountain View is currently a clear gap in the coverage of the cycling network and would help many commuters.

I hope that despite the gap in funding for infrastructure projects the city council will continue its commitment to keep Palo Alto a gold level Bicycle Friendly Community. Last but not least because investment in bike infrastructure is so much more cost efficient than spending on car infrastructure.

Kind regards,

Arnout Boelens

Carnahan, David

From: Joanna Teubert <jmsteubert@gmail.com>
Sent: Thursday, March 08, 2018 7:46 PM
To: Council, City
Cc: Ben@bikesiliconvalley.org
Subject: Biking in Palo Alto

To whom it may concern:

I would like to voice my support for continuous improvement to the bicycle infrastructure in Palo Alto. I use my bicycle to commute to work in Palo Alto and have been for three years. I appreciate being able to use bicycle friendly roads and reduce overall congestion. Please continue to keep bicycle transportation in mind on road projects. Thank you very much.

==========

Joanna Teubert
jmsteubert@gmail.com

Carnahan, David

From: Aram James <abjpd1@gmail.com>
Sent: Saturday, March 10, 2018 8:51 PM
To: chuckjagoda1@gmail.com; wilpf.peninsula.paloalto@gmail.com; stevendlee@alumni.duke.edu; HRC; Council, City; cindy.chavez@bos.sccgov.org; paloaltofreepress@gmail.com; council@redwoodcity.org; Perron, Zachary; Keene, James; griffinam@sbcglobal.net; molly.o'neal@pdo.sccgov.org; Stump, Molly; mdiaz@redwoodcity.org; gkirby@redwoodcity.org; cbolanos@co.sanmateo.ca.us; dave.cortese@bos.sccgov.org
Cc: rpichon@scscourt.org; roberta.ahlquist@sjsu.edu; mharris@scscourt.org; bwalsh@scscourt.org; supervisor.yeager@bos.sccgov.org; sscott@scscourt.org; aflint@scscourt.org; jrosen@da.sccgov.org; swagstaffe@smcgov.org; dcbertini@menlopark.org; mike.wasserman@bos.sccgov.org; joe.simitian@bos.sccgov.org; dryan@scscourt.org; jseybert@redwoodcity.org; jsylva@da.sccgov.org; Kilpatrick, Brad; myraw@smcba.org; 51swampdog@gmail.com; citycouncil@menlopark.org; bos@smcgov.org; price@padailypost.com; allison@padailypost.com; stephanie@dslextreme.com
Subject: Blacks leaving white evangelical churches —no one wants to seriously discuss racism - Trump their guy?

https://mobile.nytimes.com/2018/03/09/us/blacks-evangelical-churches.amp.html

Sent from my iPhone

Carnahan, David

From: Jyotsna Nimkar <jnimkar@gmail.com>
Sent: Saturday, March 10, 2018 7:51 PM
To: Council, City
Cc: Architectural Review Board; Clerk, City
Subject: Calling your attention to a conflict of interest

Dear Mayor Kniss, Vice-Mayor Filseth and Members of City Council:

I am writing to more formally bring to your attention something I believe you will be as troubled by as I am, and it is this: When a local property owner called the City because he was concerned about the level of radiation being emitted by a cell phone tower adjacent to his property, the City hired Verizon's engineering firm—the same firm Verizon has hired to persuade residents they have nothing to be concerned about when it comes to cell tower radiation—to conduct what was supposed to be an "independent" evaluation of whether the cell tower was in compliance with FCC guidelines.

Here's what occurred: In April of last year, the owner of the office building at 635 Bryant Street in Palo Alto contacted the City because he was concerned about the level of radiation being emitted by a nearby Verizon/Crown Castle cell tower. Specifically, he wrote to the Planning Department to request that the City obtain an "independent" (I am quoting from his email to the City) evaluation of radiation levels at the site. The Planning Department agreed to do so, and the work was carried out in May. But instead of getting an independent evaluation, the Planning Department hired Hammett & Edison—the same firm to whom Verizon is paying millions of dollars to support its applications to install over 100 new cell towers in Palo Alto. (By the way, Crown Castle is also a Hammett & Edison client.)

Of course I'm not qualified to evaluate Hammett & Edison's work. But what I do know is that Hammett & Edison had a massive conflict of interest in accepting this project, and one has to question the ethics of a company that would accept such an assignment. In addition, one also has to question the judgment of Hillary Gitelman and her staff, who commissioned Hammett & Edison to do the job.

I realize that Hammett & Edison has government clients and, in particular, it has, I believe, worked for Palo Alto in the past. But there is no gray area here: Hammett & Edison is being well paid by Verizon—as it was in May, 2017—to support Verizon's applications to install dozens and dozens of cell towers here. Moreover, its mission for Verizon includes assuaging any concerns Palo Altans may have that cell towers represent a health risk. *How could the City possibly justify hiring this firm to look into whether any cell tower is emitting more radiation than the FCC permits?*

I know you, as individuals, care a great deal about our city and its residents. I trust that you will be as appalled as I was to learn that a standard bearer for the telecom industry was hired to determine whether a Verizon cell tower is in compliance with FCC radiation standards.

Sincerely,

Jyotsna Nimkar

Jyotsna Nimkar

jnimkar@gmail.com

650-3419711

Carnahan, David

From: JIM POPPY <jamespoppy@comcast.net>
Sent: Tuesday, March 13, 2018 3:23 PM
To: Tanaka, Greg; Scharff, Gregory (internal); Kniss, Liz (internal); DuBois, Tom; Kou, Lydia; Wolbach, Cory; Filseth, Eric (Internal); Fine, Adrian; Holman, Karen; Keene, James; Council, City; French, Amy
Subject: Castilleja mailers contain false information.
Attachments: Castilleja-Myth-v-Fact.pdf

Hello Palo Alto Councilmembers, City Manager, and Planning Department,

Castilleja has mailed flyers to Palo Alto residents with false information.

Please read the attached so that you may better understand some of the facts and Castilleja's long history of avoiding the truth.

Regards,

Jim Poppy

135 Melville Ave

Setting the Record Straight: Myths vs Facts

Castilleja

Castilleja School has mailed flyers to Palo Alto residents filled with misrepresentations about their expansion plans.

Here are some facts which help illustrate Castilleja's long history of avoiding the truth.

MYTH

"After 100 years in its current location, these are not viable options, especially given today's real estate climate. Splitting the campus is the least environmentally-sustainable option..."

FACT

Castilleja has always been fixated on being located near Stanford. Castilleja stated in one of the first neighborhood meetings (September 18, 2014) that the board would not consider moving or splitting the campus, because of its proximity to Stanford. There was no mention of cost or environmental concerns. The topic was never allowed to be considered in neighborhood meetings.

"Reaffirmed, stay where we are, the value of the Stanford synergies, not move or split the school" *(page 24 from the public document below published on the Palo Alto government website)*

https://www.cityofpaloalto.org/civicax/filebank/documents/53964

Castilleja has wealthy donors who are standing by to donate funds for the garage construction. They could easily find a location in the Bay Area for the school. Many private schools have relocated in the recent past. Harker School has flourished with multiple campuses. If Castilleja truly wants to educate more women, they would find more space, instead of alienating the community for the sake of 115 more students. 75% of the student body does not live in Palo Alto.

MYTH

"Head of School Nanci Kauffman came forward to report that the School was over-enrolled. Castilleja paid a fine of $265,000 and began reductions to achieve a new City-authorized 438-student cap..."

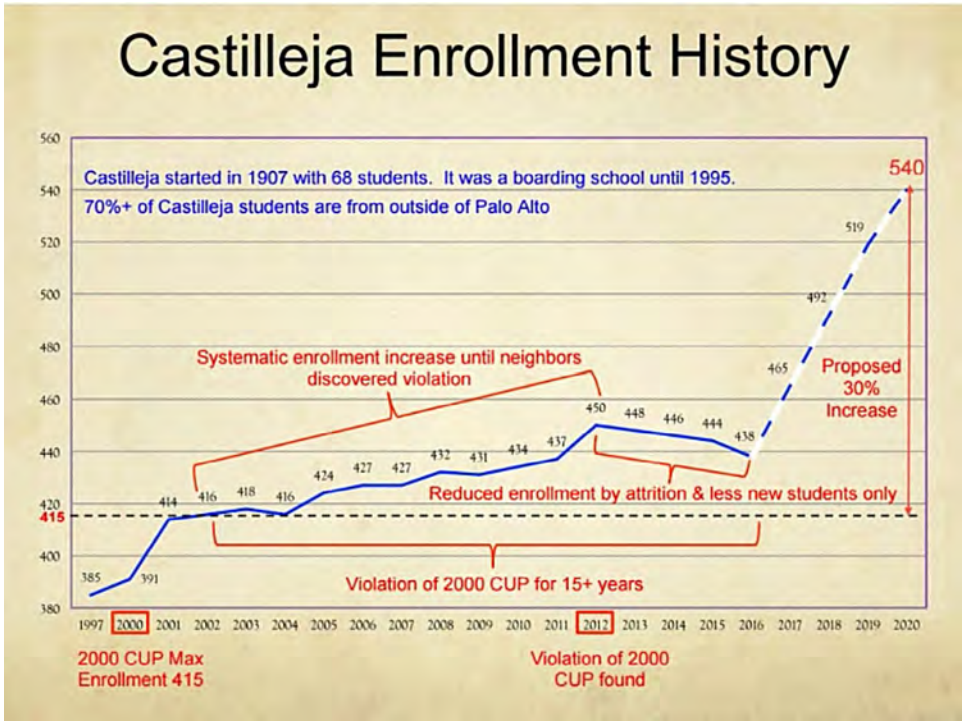
FACT

Castilleja began violating the terms of their permit in 2001 with systematic increases in enrollment each year. In 2013, this was inadvertently exposed at a public meeting at the school. It was reported in the PA Daily Post the next day.

The City directed the school to reduce enrollment to 415 but gave them a *1-year exemption* in 2015 to keep enrollment at 438 as they explored the possibility of a garage entrance and exit on Embarcadero Road. In 2016, when the City informed the school that the entrance/exit on Embarcadero was not feasible, they failed to re-enforce the 415 statute.

In 2017 City Manager Jim Keene instructed the school to continue the enrollment reduction to 415 starting in 2018-2019.

The one-time fine of $265,000 is a tiny fraction of the $1.2 million PER YEAR that they receive from their over-enrollment.



Palo Alto Weekly
www.PaloAltoOnline.com

PA Weekly Editorial, June 2017: "Castilleja's Unwise Stubbornness."

https://www.paloaltoonline.com/news/2017/06/16/editorial-castillejas-unwise-stubbornness

MYTH

"The number of car trips will stay the same due to the school's strict Traffic Demand Management (TDM) plan. This has reduced the number of car trips by nearly 20% since 2012. The underground garage features a driveway aligned with Melville Ave and is well-integrated with the surrounding landscape."

FACT

Castilleja's TDM program is self-administered and self-reported. Since it began, the only data provided has been data from the school. And it is only focused on "peak times" during the school day, conveniently omitting the 100+ events held by the school each year (more than allowed, another permit violation).

The plan for the underground garage is severely flawed. Cars would be forced to enter in one location on Bryant, which is a bike boulevard and already a dangerous intersection. Cars would then be forced back onto Embarcadero Road heading east when exiting. More likely, the garage would be avoided and students would be dropped off and picked up on surrounding streets.

The garage is Castilleja's Trojan Horse. Once they have more parking, they can have larger events and enrollment and greater impacts to the neighborhood and surrounding community.

Also, train electrification and the Stanford expansion are on the horizon, which will severely impact the area surrounding the school for years, possibly a decade or longer. The Castilleja project EIR is unlikely to incorporate these major impacts.

EVENTS PER SCHOOL YEAR 2017-18	
Castilleja	**119**
Pinewood	12
Stratford, Palo Alto	0
Stratford, Crestmoor	7
Hillbrook	11

MYTH

"Only 6 trees are being contemplated for removal. In fact, Castilleja's plan was designed specifically to minimize the impact on trees on its property, and includes the addition of at least 20 net new trees."

FACT

Castilleja's original plan from June 2016 indicated that 156 trees would be affected, and 58 removed, including many heritage oaks and redwoods. After public outcry, the plans were modified. The school now only admits to removing 6 trees but they plan to transplant 45 trees, many of which are stands of trees with intertwined roots. A former City arborist has stated that any transplanted trees are unlikely to thrive.

Residents would like to keep mature trees, and not have to live next to or in view of a large cement exit to the garage. Any new trees will take generations to replace those removed.

MYTH

"Since 2012, the school has held more than 30 community meetings. This has provided neighbors the opportunity to learn about the plan and share feedback that has been incorporated into the school's proposal."

FACT

Because of their many years of permit violations, Castilleja is required by The City to hold public meetings twice a year with neighbors. Several neighbors met as a "small working group" to provide specific input and recommendations, none of which were incorporated into the plans presented in June 2016.

In 2017 the school's PR firm told local media that "neighbors have always insisted on a garage" which prompted 47 households within a two block radius of the school to sign petitions against the proposed garage. The school has since dropped this falsehood from their PR efforts.

PNQLnow.org
Protect Neighborhood Quality of Life

PNQLnow.org is a grassroots organization of Palo Alto residents who oppose Castilleja's expansion plans to remove homes and trees, and to add a massive concrete underground garage that would funnel all cars into the Bryant Bike Boulevard and empty into the neighborhood. We would like the school to engage in open and honest dialog with neighbors.

The City has asked Castilleja and neighbors to enter into mediation. Here are our guiding principles as we enter into the mediation process:

GUIDING PRINCIPLES

1. Retain the quiet enjoyment, appearance and livability of our R-1 zoned residential neighborhood

2. Establish an open, honest, and trusting relationship between Castilleja and its neighbors

Please visit pnqlnow.org for more information. If you would like a yard sign or donate time/money, send us an email at info@pnqlnow.org.

Carnahan, David

From: Robert Moss <bmoss33@att.net>
Sent: Monday, March 12, 2018 3:25 PM
To: Architectural Review Board
Cc: Council, City; Planning Commission
Subject: Cell Towers in Residential Neighborhoods

Please reject the request by Verizon to allow cell towers in residential neighborhoods. People currently aren't complaining that they can't get adequate wireless phone service from their homes, and the intrusion of an industrial product (which is what the cell tower equipment is) in residential areas is unnecessary and intrusive. If there is any expansion of the cell system any and all electrical equipment should be buried underground, not sitting on sidewalks and curbs. Verizon has admitted hat the towers aren't needed to serve homes in the areas, but will be mainly for visitors who are using cell phones locally. That attitude puts transients over residents, and it is the city's responsibility to prioritize protecting residents, not commercial interests..

Yours very sincerely,

Bob Moss

Carnahan, David

From: Karin Thorne <karint32@gmail.com>
Sent: Monday, March 12, 2018 9:05 PM
To: Architectural Review Board; Council, City; Clerk, City
Subject: Cell Towers

March 12, 2018

TO: Architectural Review Board

I am sending this email out of my concern for Verizon's plans to locate cell towers in close proximity to our homes.

With the increased need of 5G communication for our ever growing cellular appetite , we cannot rush into quick solutions that are eyesores and emit sounds that are objectionable. According to a New York Times article by Allan Holmes on March 2, 2018, "residents across the country are just now beginning to understand the harms that hasty and insensitive small cell developments can inflict on their communities." To approve an antenna that emits noise and to construct a unit that is aesthetically objectionable would go against the sense of pride that neighbors share for our quiet tree lined streets.

I urge the ARB to deny approval to the proposed cell towers unless Verizon's designs a) call for all the equipment except the antenna to be located completely underground and b) comply with Palo Alto's noise ordinances.

Thank you,

Karin Thorne
625 Lowell Ave.
Palo Alto, CA 94301

Carnahan, David

From: Pat Markevitch <pat@magic.com>
Sent: Monday, March 12, 2018 1:03 PM
To: Council, City
Subject: Charleston Arastradero Plan
Attachments: Terman Arastradero Plan Support Ltr 2015.pdf; Terman PTA letter Re Traffic Adaptive.pdf

March 7, 2018

Honorable Palo Alto City Council Members,

Over many years, the Terman Middle School PTA has supported the Charleston-Arastradero Plan. Please note the attached letter that the Terman PTA wrote in support when Council approved the Charleston-Arastradero project in 2015. In addition, we also have attached the letter we sent in 2017 to support the city's grant application for traffic adaptive signal timing equipment funding for the project. The Terman Middle School PTA has repeatedly supported the project through its various phases of development.

We are writing to affirm our strong support of the project as the city considers its infrastructure funding priorities. Please move the project forward expediently this Spring as planned. After more than a decade of working in partnership with the City of Palo Alto and PAUSD on this project, we look forward to construction of the long-awaited school commute safety hardscape improvements.

Thank you for your ongoing support of Safe Routes to School.

Sincerely,
Terman PTA Executive Board

TERMAN MIDDLE SCHOOL PTA

655 ARASTRADERO ROAD
PALO ALTO, CA 94306

Home of the Tigers

April 15, 2015

Dear City Council and Planning & Transportation Commissioners,

Terman Middle School directly abuts Charleston/Arastradero Roads. With very few exceptions, students who commute to this site must use this City of Palo Alto School Commute Corridor for some portion of their school commute. The Terman Middle School PTA actively encourages alternative commutes, so we consider the safety of this corridor to be a very high priority.

Terman Middle School PTA Traffic Safety Representatives and administrators have participated on the Charleston/Arastradero Stakeholders Group and the City School Traffic Safety Committee, providing comment and support for the Charleston/Arastradero Plan for more than a decade. Paint striping was adequate for a restriping trial of road operations and it provided some safety improvements. It is time to put the hardscape improvements in place that will deliver the lion's share of safety benefits to all users.

We ask you to **approve the Concept Plan Lines** which fine tune the striping plan that exists now on the corridor. These plans provide planted medians, intersection and signal improvements, bulb-outs, multi-use paths, buffered bicycle lanes, a new dedicated auto right turn lane into the Terman Middle School campus from east bound Arastradero, and other built enhancements. The project is a key component of the south Palo Alto bike boulevard network, safely connecting PAUSD corridor schools to community residences and after-school destinations.

We look forward to a safer street environment for all road users—people who drive, people who bike, and people who walk.

Thank you for City of Palo Alto's partnership in creating safer routes to school.

Sincerely,

Donna M. Pioppi, President
Terman Middle School PTA

Ms. Celeste Fiore

Valley Transportation Authority Programming & Grants

3331 North First Street

San Jose, CA 95134

February, 27, 2017

Dear Ms. Fiore,

We are writing to offer support for the City of Palo Alto grant application for Charleston/Arastradero Adaptive Traffic Signal Timing.

Terman Middle School directly abuts Charleston/Arastradero Road. With very few exceptions, students who commute to this site must use this City of Palo Alto School Commute Corridor for some portion of their school commute. One average, 279 students bicycle to Terman each day. Many also walk to school or ride a bus. Terman Middle School PTA actively encourages alternative commutes, so we consider the safety of this corridor for foot-powered student commuters to be a high priority. Adaptive Signal Timing is a key component of the City of Palo Alto's multi-modal improvement plan for this sensitive arterial.

Active traffic signal management supports safe and efficient roadways. The Adaptive Traffic Signal Timing on Charleston/Arastradero will improve traffic operations by improving travel time, reducing congestion, and reducing idling, especially during the morning peak hour when crosstown commuter traffic and school commute traffic converges, creating severe congestion. Traffic signal enhancements will minimize the number of drivers slowing down suddenly, which also causes pollution. In turn, air quality around our schools and in Palo Alto will be improved.

Adaptive traffic signal timing would enable completion of the transformation of this school commute corridor that carries nearly 20,000 car trips per day and many hundreds of students on foot and on bicycles to nearby schools.

Please approve the City of Palo Alto application for funding. We look forward to a safer street environment for all road users—people who drive, people who bike, and people who walk.

Thank you.

Sincerely,

Cheryl Phillips

2016-17 Executive Vice President (acting president),

Terman Middle School PTA

Carnahan, David

From: E Nigenda <enigenda1@gmail.com>
Sent: Monday, March 12, 2018 4:21 AM
To: Friend, Gil; Keene, James; Council, City
Subject: Cities Emit 60% More Carbon Than Thought

The carbon footprint of some of the world's biggest cities is 60 percent larger than previously estimated when all the products and services a city consumes are included, according to a new analysis.

https://news.nationalgeographic.com/2018/03/city-consumption-greenhouse-gases-carbon-c40-spd/

Carnahan, David

From: Randy Mont-Reynaud <rmontreynaud@gmail.com>
Sent: Wednesday, March 14, 2018 9:21 AM
To: Planning Commission; Council, City
Subject: Constructive Ideas for Affordable Housing
Attachments: AFFORDABLE HOUSING.pdf

Dear Planners and Council Members,

Affordable housing? Let's think about this. And think again. What sort of place is this, anyway? I've been living here for 38 years, raised 3 children, have been a student, a renter, a homeowner, then a renter again…Like many, I am now a working grandmother, and I'd surely hope there will be room for me to stay in our small city. Where do you stand?

In my many classrooms, my goal has always been to educate the heart. I'll speak to that now.

Our community, MOST of our community I should hope, is in favor of providing Affordable Housing - AND IN SHORT ORDER. Please don't disappoint? No one on the council, committee or street should not be on board with the proposed relaxed standards that will promote construction (can we say, immediate construction?) of Affordable Housing so seniors, families with disabled children, low income workers, veterans and disabled young adults can remain where they have grown up or grown old.

I hope not to be disappointed that our community, would want their legacy - or part of it - to be deployed to PAY IT FORWARD, and sustain us.

Among the proposals are the relaxed standards (Relax! Just Do It!) greater square footage, flexibility on retail requirements, parking standards…But, no need to build us a Taj Mahal folks, just four walls and a roof, some indoor plumbing would be great…

And, **here's a constructive idea** (pun intended) Why not consider taking over some existing apartment complexes (whose landlord's may be kvetching about their profit margins!) — Could the city consider BUYING up existing apartment complexes, and converting them into Affordable Housing?

Having lived here for 38 years, I've seen many changes but I hope hearts and minds are still in the spirit of the Civl Rights Movement.…Facilitate provision for our diverse population. Keep Palo Alto diverse, and not divided. Please Build back better, more and soon!

r

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With warmest regards,

Randy Mont-Reynaud, PhD

ISAIAH 58: "'Is not this the kind of fasting I have chosen: to loose the chains of injustice and untie the cords of the yoke, to set the oppressed free and break every yoke?"

650 858 1558 (cell)
Our 501 c-3 is "If Pigs Could Fly - Haiti" Visit us here:
www.ifpigscouldflyhaiti.org
And here is my blog: http://www.haitinextdoor.com/

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Carnahan, David

From: Susan Thomsen <susan@thomsenhome.com>
Sent: Monday, March 12, 2018 4:00 PM
To: Architectural Review Board; Council, City; Clerk, City
Subject: FW: [CPNA] Cell towers in residential neighborhoods

To whom it may concern:

I am writing to urge you not to approve the proposed cell towers in residential neighborhoods unless the mobile service providers comply with Palo Alto's noise ordinances, locate all equipment except the antennas completely underground, and locate the antennas away from single family homes.

Thank you for your consideration.

Sincerely,
Susan Thomsen
1701 Edgewood Drive
Palo Alto, CA 94303

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Carnahan, David

From: Lily Huang Liao <lilyhuangliao@gmail.com>
Sent: Monday, March 12, 2018 3:38 PM
To: Council, City; Clerk, City; Architectural Review Board
Subject: Re: [CPNA] Cell towers in residential neighborhoods

To whom it may concern.

I'm wring to express my strong objections to approval to the proposed cell towers in residential neighborhoods. Period.
Even if mobile service providers:

a) locate all the equipment, except the antennas completely underground and
b) comply with Palo Alto's noise ordinances.

The only exception I can support, is if

a) providers locate all the equipment except the antennas completely underground and
b) comply with Palo Alto's noise ordinances, AND
c) antennas are located in a considerable distances from residential buildings,
    a. example would be parks
    b. bridges over creeks, etc.

Sincerely,
Lily Huang
2330 Cowper Street,
Palo Alto, CA 94301

Carnahan, David

From: From: BBRetiredNow@aol.com <bbretirednow@aol.com>
Sent: Monday, March 12, 2018 3:31 PM
To: Architectural Review Board
Cc: Council, City
Subject: Fwd: [CPNA] Cell towers in residential neighborhoods

I am writing to plead with the board to deny Verizon's plans to install cell towers in Palo Alto's residential neighborhoods! My additional concern is that this is described as "its first wave" of towers, and I would like us to avoid the slippery slope.

If their aim is to erect the towers cheaply, the towers would be larger and require more noisy supportive equipment, none of which would bring anything good to our residential areas.

We are already dealing with the city council thinking of ways to bring more density and congestion to the area, and I hope we can stop the invasion of ugly towers and equipment threatening our neighborhoods.

Thanks,
Barbara Bogner

Carnahan, David

From: Roman Kagarlitsky <rak@renderx.com>
Sent: Monday, March 12, 2018 3:12 PM
To: 'ed ATT'
Cc: Architectural Review Board; Council, City; Clerk, City; 'crescent-park'
Subject: RE: [CPNA] Cell towers in residential neighborhoods

Noise and possible electric influence on various subjects and objects.
I'm sorry. I don't have time to get into lengthy discussion, but this is my position.
Obviously, Ed, of course, if you want to – you can support any equipment anywhere.
But I don't.

Roman.

From: crescent-park-pa@googlegroups.com [mailto:crescent-park-pa@googlegroups.com] **On Behalf Of** ed ATT
Sent: Monday, March 12, 2018 2:50 PM
To: Roman Kagarlitsky
Cc: arb@cityofpaloalto.org; city.council@cityofpaloalto.org; city.clerk@cityofpaloalto.org; crescent-park
Subject: Re: [CPNA] Cell towers in residential neighborhoods

Why?

Ed

> On Mar 12, 2018, at 2:26 PM, Roman Kagarlitsky <rak@renderx.com> wrote:
>
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> I'm wring to express my strong objections to approval to the proposed cell towers in residential neighborhoods.
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> c) antennas are located in a considerable distances from residential buildings,
> a. example would be parks
> b. bridges over creeks, etc.
>
> Roman Kagarlitsky
> 641 Fulton Street,
> Palo Alto, CA 94301

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You received this message because you are subscribed to the Google Groups "Crescent Park PA" group.
To unsubscribe from this group and stop receiving emails from it, send an email to crescent-park-pa+unsubscribe@googlegroups.com.
To post to this group, send email to crescent-park-pa@googlegroups.com.
Visit this group at https://groups.google.com/group/crescent-park-pa.
For more options, visit https://groups.google.com/d/optout.

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You received this message because you are subscribed to the Google Groups "Crescent Park PA" group.
To unsubscribe from this group and stop receiving emails from it, send an email to crescent-park-pa+unsubscribe@googlegroups.com.
To post to this group, send email to crescent-park-pa@googlegroups.com.
Visit this group at https://groups.google.com/group/crescent-park-pa.
For more options, visit https://groups.google.com/d/optout.

Carnahan, David

From: seawaif1@aol.com
Sent: Saturday, March 10, 2018 2:54 PM
To: CSD
Cc: Keene, James; Council, City
Subject: Dog Training Areas in Palo Alto

Re: Dog Training Areas

We are a group of six responsible local residents who have highly trained dogs (a golden retriever, an Australian shepherd, two Weimaraners, two collies, and two pit bull mixes) that compete in American Kennel Club obedience competitions. The exercises we practice involve jumps, scent work, retrieves, heel work, response to hand signals, recalls - skills that are accomplished with the dog working at a distance from where we stand and can only be done off leash.

One of our dogs is an extremely accomplished pit bull mix; this dog has won many ribbons and participated in the highly regarded AKC National Obedience Invitational Tournament, not once but twice. Another of our members has participated in this event with a previous dog. All of these dogs have been in intensive training for years.

We are asking for permission to use a small area at local parks for training practice once or twice per week, starting around 9:00 am, finishing around 11:00 am. Our dogs need to be comfortable working in many different locations, so we try to rotate around various areas to give them that experience. We define our working space of 40 x 50 feet with posts and/or gates; the only time the dogs need to be off leash is within the confines of that area. We set up away from the children's play areas. These dogs do not run out of the training area to visit passersby or other dogs, so they are not a bother to other park users. While waiting for their turn to practice, the dogs are confined in crates, or warming up on leash just outside the confines of the practice ring.

Currently, there are no public locations where we can do this without the potential for a visit from Animal Control, yet local communities allow AKC obedience & agility competitive matches and trials from time to time where the off leash exercises take place within ring setups like these (Cuesta Park; Greer Park; Mitchell Park; De Anza College). Most of us are members of Deep Peninsula Dog Training Club, Inc., that holds dog training classes Monday evenings at Rengstorff Park. Dogs do work off leash in some of these classes, many of which we all have attended; two of us have actually taught these classes.

It has become increasingly difficult to find areas in our local parks to train our dogs. Unfortunately, leash laws prohibit our mode of training even within the well-defined boundaries of our set-up. Dog parks are not suitable because there is no way we can prevent off-leash dogs from running into our training area, harassing our dogs, and at times, peeing on our equipment. We do need an area close to available parking because of the necessity of transporting ring gates and jumps. We are responsible dog owners and are extremely conscientious about "picking up after our dogs." We even pick up after other dogs that have left their calling cards in the area.

There are benefits to the community to allowing us to practice in public. People walking by are amazed by the abilities of our dogs. They often stop to ask about them and their training. A couple of us carry business cards for Deep Peninsula DTC because so many people want to know where they can find classes for their own dogs. The demonstration of well-trained dogs responding to the quiet directions of their owners promotes responsible ownership in others. Surely, the positive publicity we offer the public on training days and information about training classes in the area would offset any inconvenience we might create.

Mountain View used to have a permit system for just such training requirements. Can you help us?

Sincerely,

Anne Robinson (Menlo Park) seawaif1@aol.com

Judy Cummings (Los Altos Hills)

Linda Kirk (Los Altos Hills)

Nan Daley (Woodside)

Ferol Larsen (Palo Alto)

Mara Wildfeuer (Mountain View)

Carnahan, David

From: Matt | Willow Glen Electric <matt@willowglenelectric.com>
Sent: Monday, March 12, 2018 3:33 PM
To: Hoyt, George
Cc: Scharff, Gregory (internal); Council, City; Virginia Neff
Subject: Electrical Panel Upgrade permit price change

Hi George,

I'm inquiring about the recent change to Residential Electrical Panel Upgrade pricing in Palo Alto. It recently came to our attention that the City Council (CCed here w/ the Mayor) voted to approve the new fee schedule that came into effect on 2/1/18. Based on our conversation w/ the Building Department today, previous panel upgrades were priced according to amperage (0-200 Amps, 201-400 Amps, 401-800 Amps.) On 2/1/18 this fee structure changed to lump all upgrades from 0-800 Amps together. This has caused the cost of simple residential panel upgrade permit to skyrocket from $138 in January to $373, just for the panel upgrade line item alone. Our most recent permit in Palo Alto for a residential 200 Amp service upgrade w/ 50 Amp EV charger cost our company $622, which is the most we've ever paid in 22+ jurisdictions for this type of project. If we did this same project just a few miles south in Los Altos, we'd only pay $75 total for this permit.

Is there anything you can do to help fix this? We pass these permit costs on directly to our customers, but this revised permit cost seems a bit excessive.

Best regards,

Matt Londre
Sr. Project Manager
Willow Glen Electric, Inc.
(408) 705-3663 - cell
(408) 289-9725 ext. 1 - office

Carnahan, David

From: Joan Zwiep <joan@hosterfamily.com>
Sent: Monday, March 12, 2018 1:53 PM
To: Council, City
Subject: FBC Conditional Use Permit

Dear City Council Members,
This email is to let you know that I am opposed to allowing the First Baptist Church on California Avenue be turned into a Community Center. I hope that you deny their use of such a permit. Traffic and parking are the two top issues for Palo Alto citizens. Using a church as a full-time community center will only exacerbate the problems we have with cars and parking. There have already been two accidents due to the church. I live near California Avenue and I have noticed an increase traffic. We want our neighborhoods to be safe and to stay as neighborhoods.
Thank you.
Joan Zwiep
2345 Byron Street
Palo Alto, CA 94301
650-322-1246

Carnahan, David

From: Bonnie Flanagan <bonnie.m.flanagan@gmail.com>
Sent: Tuesday, March 13, 2018 11:17 AM
To: Council, City; Keene, James; Gitelman, Hillary; Lait, Jonathan; Owen, Graham
Subject: FBC CUP

Dear Mayor Kniss and City Council:

I was unable to make the meeting at Jordan Middle School on March 7th regarding First Baptist Church's request to become a Community Center, but I've been reading comments in both the Palo Alto Online & the PA Daily Post. Many are concerned with traffic & safety issues which was the focus of my February 1st letter to the Council.

My concerned for the safety of our children riding bikes in this area continues. The situation at Ross Road regarding safety for bikers/cars will be mirrored when the California Avenue/Bryant roundabout & Bryant Street bike path is completed.

Since my earlier letter there has been an accident on Bryant, a situation that sadly the neighborhood had expected to see happen, though hoped would not. One car had just picked up children from the daycare. The car had two small girls in the back seat where the impact occurred & airbags went off! The car that hit them zipped around CA Ave down Bryant. I was told, though I don't know for sure, that the car causing the accident was a parent picking up students from the church. To my knowledge nobody came over to help from the church & yet the back door closest to the accident was opened; students came out to look, but no adult. VERY scared little girls. I've included pictures below.

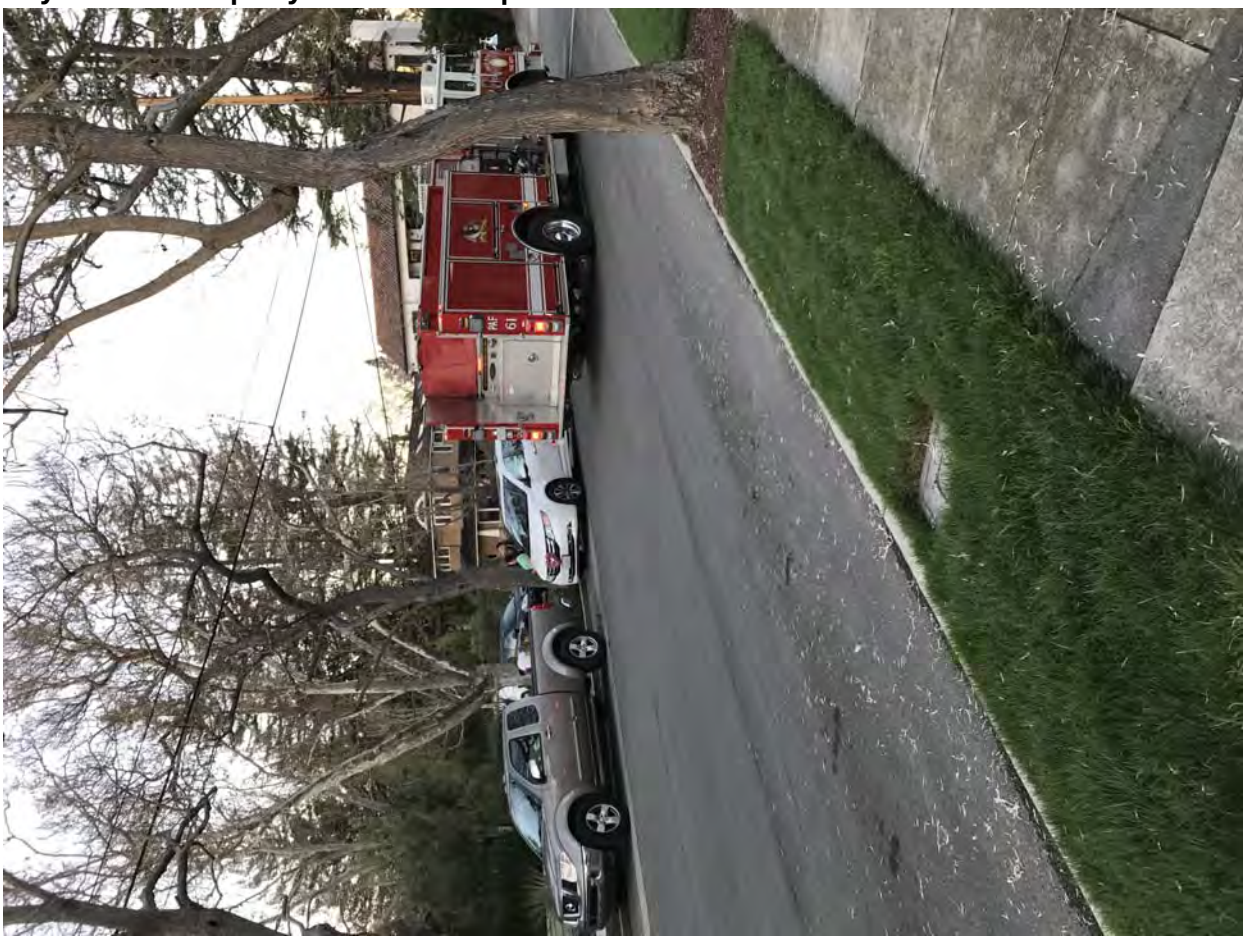
If a community center, roundabout & bike path converge in that area it's difficult to believe more accidents won't occur — as they say "it's an accident waiting to happen."

Please carefully consider what can be at FBC that does not impact child/bike safety & traffic.

Thank you,

Below are a few pictures:
1) accident on Bryant; car coming out of daycare hit by car coming down Bryant


STOP



2) 3 large buses returning iSing students to FBC; a Thursday when students are biking home


Royal
Coach Tours