

Appendix B – Literature Review on the Health Impacts of the Hours of Service Rule Changes



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Appendix B – Literature Review on the Health Impacts of the Hours of Service Rule Changes

Introduction

PURPOSE OF THE REVIEW

As many as 4.5 million Americans work as commercial motor vehicle (CMV) drivers. While there is no such thing as a “typical” driver, drivers can be considered to belong to categories based on the type of vehicle driven, the schedule on which they drive, and the type of load they typically carry. Drivers differ in whether they work under a union arrangement, or as independent contractors or employees of non-unionized companies. The majority of drivers are male; 2009 Bureau of Labor Statistics (BLS) data record that in the category of “Driver/sales workers and truck drivers” 5.2 percent of workers are female, and women make up 5.9 percent of “Motor vehicle operators, other” (BLS 2009). We have a general sense of the age distribution of drivers (see Exhibit B-1) from a database of 64,000 drivers compiled by RoadReady:

Exhibit B-1. Age Distribution of Drivers

Age Category	# in Category	% in Category
20-29	9242	14%
30-39	18986	29%
40-49	20525	32%
50-59	12310	19%
60-69	3252	5%
70-79	280	0%
80-89	14	0%

This type of work is characterized by long hours, both per day and per week. It is sedentary and can involve sitting for 8 to 14 hours per day. Drivers often experience short sleep or intermittent sleep schedules. These factors lead to a concern over such health issues as obesity, obstructive sleep apnea, and chronic fatigue.

The Centers for Disease Control and Prevention (CDC) MMWR Morbidity and Mortality Weekly Report for February 2008 (CDC 2008) lists the following as being associated with insufficient rest: mental distress; depression; anxiety; **obesity**; hypertension; diabetes; high cholesterol; cigarette smoking; physical inactivity; heavy drinking; and cardiovascular disease. The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK 2010) links obesity/overweight to: diabetes; coronary heart disease; high blood cholesterol; stroke; hypertension; gallbladder disease; osteoarthritis; sleep apnea and other breathing problems; and some forms of cancer.

There is evidence to support the perception that drivers are more likely to be overweight or obese than are members of the general population (Exhibit B-2). Both of the driver populations listed in the exhibit are LTL drivers and, therefore, unlikely to work extreme hours although likely to drive at night.

The purpose of this review is to provide information to support proposed revisions of hours of work regulations, with the goal of increasing both driver safety and the safety of the public, as well as to maximize the health and well-being of CMV drivers. To conduct this review we reviewed the literature on health impacts and conditions among all persons; we did not restrict the reviewed literature to that directly concerned with CMV drivers because drivers are no less likely than other people to be subject to health impacts caused by lack of sleep and sedentary lifestyle.

Exhibit B-2. Obesity Prevalence Among Three Populations

FMCSA Fatigue Management Survey – 2,128 drivers (Dinges et al. 2005)		Impact on medical costs of truck drivers – 2,950 drivers (Martin et al. 2009)		National all adult males (Flegal et al. 2010)	
<i>BMI</i>	<i>% drivers</i>	<i>BMI</i>	<i>% drivers</i>	<i>BMI</i>	<i>% adult males</i>
<25	10.3	18.5-24.9	13	<25	27.7
25-29.9	39.9	25-29.9	30	25-29.9	40.1
30-34.9	26.3	≥30	55	30-34.9	17.3
35-39.9	11.3			35-39.9	10.7
40+	12.3			40+	4.2

STRUCTURE OF THE CHAPTER

This chapter is structured in two major sections. In the first section we present data from three studies of sleep and mortality from which we were able to extract data to produce a curve demonstrating the expected lost years of life based on typical hours of sleep. We discuss the implications of this model for Hours of Service regulations.

In the second section we examine research that suggests a chain of relationships between the “driver lifestyle” of long hours, protracted sitting, and moderate-to-severe sleep deprivation; obesity as a potential outcome of this lifestyle; and health problems and costs frequently linked to obesity. We present conclusions from this set of studies and we outline the need for additional evidence in this area. The studies referenced in this appendix are available in docket FMCSA-2004-19608.

METHODOLOGY

Section 1

For the analysis of sleep and mortality we performed a National Library of Medicine PubMed search using the following terms: sleep; rest; nap; circadian rhythm; parasomnia; insomnia; dyssomnia; hypersomnia; mortality; death; lifespan; years of life; and lifeyears. Search limits set were: search on title/abstract, publication date in past 10 years, human (non-animal) studies, English language. We also searched Google using the same set of keywords. We identified a number of studies of sleep duration and mortality. We selected only three for the final analysis because the three studies were the only ones that included information on the size and demographic makeup of the sample, the crude mortality rate (in person-years), and the confidence interval for risk of increased mortality in males and females.

For the statistical analyses of the Phase 1 sleep-hours data in the Ferrie study, we assumed that a response of “6” means 5.5 to 6.5 hours, etc. On that basis, we fitted a normal distribution to the Phase 1 “hours of sleep” frequency distribution and obtained a mean of 6.787 hours and a standard deviation of 0.768 hours.

To regress the mortality hazard ratios we calculated ‘exph’ and ‘exphh,’ the expected number of hours of sleep and the expected number of hours squared for each interval. Thus if the hours value is exactly N, then $\text{exph} = N$ and $\text{exphh} = N^2$. We then regressed the published estimated mortality ratio versus exph and exphh (and an intercept). This gives predicted values for the mortality ratio if the hours of sleep value is exactly N (an interval from N to N) or if the hours of sleep is reported as N, but is assumed to lie inside the interval from $N-0.5$ to $N+0.5$ and comes from the fitted normal distribution. The model is shown below. The two approaches give very similar predictions.

Although the fitted normal distribution to the hours of sleep is standard statistical modeling (assuming we are correct to treat a response of 6 as meaning from 5.5 to 6.5, etc.), the quadratic regression analysis is highly approximate because it does not take into account how the covariates affect the estimated mortality ratios. However, it should be a good approximation.

The following model was estimated for the distribution of hours of sleep, assuming “6” means 5.5-6.5 hours, and so forth. This model uses Phase 1 frequency distribution and best-fitting normal distribution.

Normally distributed:

Mean 6.787198

Standard Deviation 0.76828

Regression model for mortality hazard ratio assuming:

Hazard ratio = $a + b \cdot \text{exph} + c \cdot \text{exphh} + \text{error}$

Exph = expected value of hours of sleep if between from and to

Exphh = expected value of hours of sleep squared if between from and to

Error is normally distributed with mean zero

Parameter	Value	Standard Error	P-value
a	11.76028	1.0430	0.0078
b	-3.13766	0.3067	0.0094
c	0.227359	0.0219	0.0092

For example, if the hours of sleep is exactly 7, then $\text{exph} = 7$ and $\text{exphh} = 49$ and so the predicted hazard ratio = 0.937228

If the hours of sleep is the interval from 6.5 to 7.5, then:

$\text{Exph} = 6.971673$

$\text{Exphh} = 48.68249$

Predicted hazard ratio = 0.95392 (for the full set of predicted ratios, see Section 1 below)

Section 2

For the second section of this chapter we again searched PubMed with the following limits: publication date in past 10 years, English language, human (non-animal) studies, with the following keywords or phrases: sleep; health; “long hours;” “shift work;” obesity; fatigue; “sleep deprivation;” “sedentary work;” “sedentary lifestyle;” “truck drivers;” “short sleep duration;” “increased mortality;” and “health effects.” We also searched Ovid, Scopus, and Google Scholar using the phrases “short sleep duration;” “increased mortality;” and “health effects.” We reviewed studies and data from FMCSA that relate to fatigue or truck driver health to identify any statistics on obesity, high blood pressure (HBP), cardiovascular disease (CVD), obstructive sleep apnea (OSA), or related topics. We reviewed reference lists in the identified studies to determine whether additional titles would be useful.

Section 1. Sleep and Mortality

The data presented in this section are taken from three large-scale, long-term studies [Amagai et al. 2004; Ferrie et al. 2007; Tamakoshi et al. 2004]. Amagai et al. followed 11,325 participants over several years in a “population-based prospective study investigating risk factors for cardiovascular diseases, started in 1992. The authors report “A total of 495 deaths ... were observed during the average of 8.2-year follow-up period. After adjusting for age, systolic blood pressure, serum total cholesterol, body mass index, smoking habits, alcohol drinking habits, education, and marital status, the hazard ratios (95% confidence intervals) of all-cause mortality for individuals sleeping shorter than 6 hours and 9 hours or longer were 2.4 (1.3-4.2) and 1.1 (0.8-1.6) in males, and 0.7 (0.2-2.3) and 1.5 (1.0-2.4) in females, respectively, relative to those with 7-7.9 hours sleep” [Amagai et al. 2004, p.124].¹

Ferrie et al. (2007) followed 10,308 white-collar British civil servants in a prospective cohort study, with follow-up at 12 and 17 years. The authors report finding “U shaped associations ... between sleep (≤ 5 , 6, 7, 8, ≥ 9 hours) at Phase 1 and Phase 3 and subsequent all-cause, cardiovascular, and non-cardiovascular mortality” [Ferrie et al. 2007, p.1659]. The “U-shaped curve” represents the frequent finding that deviations toward less sleep or more sleep than 7-8 hours increases an individual’s risk of early mortality. Tamakoshi et al. (2004) enrolled 104,010 individuals in a study of cancer risk in rural Japanese residents, followed them for approximately 10 years, and found that for this sample, “Sleep duration at night of 7 hours was found to show the lowest mortality risk” [Tamakoshi et al. 2004, p.51]. Exhibit B-3 presents the results of the quantitative analysis of the Ferrie et al. 2007 data:

Exhibit B-3. Sleep – Mortality Risk Ratios (Ferrie *et al.* 2007)

Sleep Hours: From	Sleep Hours: To	Frequency	Observed Mortality Ratio	Sleep Hours: Midvalue	Expected Hours: exph	Expected Hours Squared: exphh	Predicted Mortality Ratio	Standard Error
Data points from Ferrie <i>et al.</i> 2007:								
0	5.5	587	1.61	2.75	5.18	26.94	1.62	0.06
5.5	6.5	2642	1.11	6	6.10	37.31	1.10	0.04
6.5	7.5	4884	1	7	6.97	48.68	0.95	0.05
7.5	8.5	1579	1.08	8	7.85	61.65	1.15	0.04
8.5	12	89	1.77	10.25	8.77	76.93	1.74	0.06
Fitted points assuming sleep is normally distributed:								
0.5	1.5			1	1.39	1.95	7.83	0.66
1.5	2.5			2	2.37	5.63	5.60	0.44
2.5	3.5			3	3.34	11.16	3.83	0.27
3.5	4.5			4	4.29	18.42	2.50	0.14
4.5	5.5			5	5.21	27.21	1.60	0.06
5.5	6.5			6	6.10	37.31	1.10	0.04
6.5	7.5			7	6.97	48.68	0.95	0.05
7.5	8.5			8	7.85	61.65	1.15	0.04

¹ For hazard ratios and odds ratios, if a confidence interval does not include 1, the result is statistically significant. For example, an odds ratio of 2 with CI of .8 – 3 is not statistically significant; and OR of 1.2, with a CI of 1.1-1.5 is significant.

Exhibit B-3. Sleep – Mortality Risk Ratios (Ferrie *et al.* 2007)

Sleep Hours: From	Sleep Hours: To	Frequency	Observed Mortality Ratio	Sleep Hours: Midvalue	Expected Hours: exph	Expected Hours Squared: exphh	Predicted Mortality Ratio	Standard Error
8.5	9.5			9	8.75	76.66	1.73	0.06
9.5	10.5			10	9.69	93.90	2.71	0.14
10.5	11.5			11	10.65	113.38	4.13	0.28
11.5	12.5			12	11.62	135.02	6.00	0.45
Fitted points assuming subjects sleep discrete numbers of hours:								
1	1			1	1.00	1.00	8.85	0.76
2	2			2	2.00	4.00	6.39	0.52
3	3			3	3.00	9.00	4.39	0.32
4	4			4	4.00	16.00	2.85	0.17
5	5			5	5.00	25.00	1.76	0.07
6	6			6	6.00	36.00	1.12	0.04
7	7			7	7.00	49.00	0.94	0.05
8	8			8	8.00	64.00	1.21	0.04
9	9			9	9.00	81.00	1.94	0.08
10	10			10	10.00	100.00	3.12	0.18
11	11			11	11.00	121.00	4.76	0.33
12	12			12	12.00	144.00	6.85	0.53

Mapping these values on a graph results in a U-shaped curve in which seven hours of sleep carries the lowest hazard ratio, and sleep periods of less than seven and more than seven hours show a progressively larger mortality hazard ratio.

Section 2: Driver Health Outcomes

For the population at large, researchers have spent much time and effort to understand the relationships between individual activities and habits and their possible eventual health outcomes. For example, a simple linear example of this kind is the causal relationship we now understand to exist between cigarette smoking and risk of lung cancer. We can expect these same relationships to hold true for commercial vehicle drivers, as drivers are a segment of the U.S. population and are subject to the same behavioral and genetic forces that act on non-drivers.

In reviewing possible outcomes of the “driver lifestyle” of long hours, protracted sitting, and moderate-to-severe sleep deprivation; we cannot posit a simple linear relationship between “lifestyle” and one or more health outcomes. Rather we need to view this relationship as a network of mutually-reinforcing effects that will result in varying levels of risk in terms of particular outcomes such as cardiovascular disease. Exhibit B-4 reflects current thinking on how this network of relationships acts on human health:

Exhibit B-4. Health habit and risk relationships

Long hours	→	Insufficient sleep	Insufficient sleep	→	Obesity
	→	Obesity		→	High blood pressure
	→	Cardiovascular disease		→	Diabetes
Sedentary pattern	→	Obesity	Obesity	→	Obstructive sleep apnea
	→	Metabolism		→	High blood pressure
	→	Increased risk of mortality		→	Cardiovascular disease
Obstructive sleep apnea	→	High blood pressure		→	Stroke
	→	Cardiovascular disease		→	Diabetes
	→	Diabetes		→	Arthritis
	→	Increased risk of mortality		→	Other disease

LONG HOURS AND INSUFFICIENT SLEEP

Artazcoz et al. (2009, p.521) looked at 7,103 salaried workers aged 16–64 in Spain to compare work hours with health-related behaviors. They categorized work hours as “less than 30 h (part-time), 30–40 (reference category), 41–50 and 51–60 h.” For men, longer work hours were associated with “shortage of sleep (aOR 1.42, 95% CI 1.09 to 1.85) and no leisure-time physical activity (aOR 2.43, 95% CI 1.64 to 3.60). Moreover, a gradient from standard working hours to 51–60 h a week was found for these six outcomes. Among women long working hours were only related to smoking and to shortage of sleep.”

Knauth (2007, p.127) conducted a literature review of “105 studies on the effects of extended daily working hours.” He produced a table of “Effects of extended shifts on duration or quality of sleep.” 13 studies cited “worse” sleep in shifts longer than 8 hours; 6 studies found no difference; eight studies found “better” sleep. He acknowledges that some of the studies had methodological problems, making a firm conclusion difficult.

LONG HOURS AND OBESITY

Di Milia and Mummery (2009, p.364) administered a survey to “804 Australian participants employed in the coal industry and 275 participants from a regional university.” “Participants were allocated into ... three groups based on the mean work duration per shift; ‘short’ (M=8.72 h±0.56), ‘medium’ (M=10.95 h±0.56) and ‘long’ (M=12.60 h±0.41).” Mean Body Mass Index (BMI) was significantly higher in shift workers than in day workers ($p<.001$). Mean BMI (12.60 h±0.41) was also significantly higher ($p<.001$) higher in the group working long daily hours followed by medium working hours (10.95 h±0.56) and short working hours (8.72 h±0.56).” The authors report “the most significant predictor of obesity was long working hours (OR=2.82, CI:1.10-7.19).”

Violanti et al. (2009, p.194) looked at “atypical work hours,” including midnight shifts, among 98 police officers and a possible relationship to metabolic syndrome (a group of metabolic risk factors for coronary heart disease and type 2 diabetes; it includes abdominal obesity). They report, “Stratification on sleep duration and overtime revealed significant associations between midnight shifts and the mean number of metabolic syndrome components among officers with less sleep ($p = .013$) and more overtime ($p = .007$). Results suggest shorter sleep duration and more overtime combined with midnight shift work may be important contributors to the metabolic syndrome.”

LONG HOURS AND CARDIOVASCULAR DISEASE

Chen et al. (2005, p.890) report on results from the Taxi Drivers’ Health Study from Taiwan. The authors used questionnaires to assess “driving time profiles” for 1,157 drivers; long driving time was defined as “self-reported monthly driving time” divided into quartiles (≤ 208 hours; 210-260 hours; 261-312 hours; and 318-450 hours). They measured whole blood cell (WBC) count as “a haematological marker for increased CVD risk” as it is a sign of “systemic inflammation and haemostatic alteration.” They report “After adjusting for conventional CVD risk factors” and a series of demographic factors such as alcohol drinking, “long driving time was still associated with significant increases in WBC and platelets, whereas the effect on haematocrit was diminished and became statistically non-significant.”

INSUFFICIENT SLEEP AND OBESITY

Banks and Dinges (2007, p.519) report that “laboratory studies of healthy adults subjected to sleep restriction have found adverse effects on endocrine functions, metabolic and inflammatory responses, suggesting that sleep restriction produces physiological consequences that may be unhealthy.”

Schoenborn and Adams (2008, p.1) reported on “the association between sleep and selected health risk behaviors using data from the 2004-2006 [National Health Interview Survey] NHIS.” They state, “Direction of causality cannot be determined with cross-sectional survey data. However, identifying health risk behaviors among adults with varying sleep durations can provide useful information on possible clustering of behaviors that are known to be associated

with unfavorable health outcomes.” Regarding sleep and obesity, “Overall, about one in four adults were obese (25%), based on self-reported height and weight. Adults who slept less than 6 hours had the highest rate of obesity (33%) and adults who slept 7 to 8 hours had the lowest (22%) ... This pattern was found for both men and women and across all age groups and most race/ethnicity groups studied. The association between sleep and obesity was less striking among adults aged 65 years and over than among younger adults” (p.3).

Van Cauter and Knutson (2008, p. S59) reviewed laboratory studies “indicating that sleep curtailment in young adults results in a constellation of metabolic and endocrine alterations, including decreased glucose tolerance, decreased insulin sensitivity, elevated sympathovagal balance, increased evening concentrations of cortisol, increased levels of ghrelin, decreased levels of leptin, and increased hunger and appetite.” They also reviewed cross-sectional and prospective epidemiological studies showing an increased risk of weight gain in short sleepers. They conclude, “Findings from laboratory studies in young adults and epidemiological studies in both children and adults converge to suggest that partial chronic sleep restriction, an increasingly prevalent behavior in modern society, may increase the risk of weight gain and play a role in the current epidemic of obesity” (p.S64).

Patel and Hu (2008, p.643) conducted a meta-analysis based on a literature search for “all articles published between 1966 and January 2007 using the search “sleep” AND (“duration” OR “hour” OR “hours”) AND (“obesity” OR “weight”) in the MEDLINE database.” “Thirty-six publications (31 cross-sectional, 5 prospective, and 0 experimental) were identified. Findings in both cross-sectional and cohort studies of children suggested short sleep duration is strongly and consistently associated with concurrent and future obesity. Results from adult cross-sectional analyses were more mixed with 17 of 23 studies supporting an independent association between short sleep duration and increased weight. In contrast, all three longitudinal studies in adults found a positive association between short sleep duration and future weight.”

Cappuccio et al. (2008, p.1) also performed a meta-analysis, using resources in addition to MEDLINE (EMBASE, AMED, CINAHL, PsychINFO, and “manual searches without language restrictions” from 1982). “Criteria for inclusion were: report of duration of sleep as exposure, BMI as continuous outcome and prevalence of obesity as categorical outcome, number of participants, age, and gender.” 36 population samples were included in the analysis, for 634,511 participants. They report “In children the pooled OR for short duration of sleep and obesity was 1.89 (1.46 to 2.43; $P < 0.0001$). In adults the pooled OR was 1.55 (1.43 to 1.68; $P < 0.0001$). There was no evidence of publication bias. In adults, the pooled β for short sleep duration was -0.35 (-0.57 to -0.12) unit change in BMI per hour of sleep change.” They state “Cross-sectional studies from around the world show a consistent increased risk of obesity amongst short sleepers in children and adults.”

INSUFFICIENT SLEEP AND HIGH BLOOD PRESSURE

Gangwisch et al. (2006, p.833) looked at the possibility of increased risk of hypertension in individuals with short sleep (but without sleep disorders). They “assessed whether short sleep duration would increase the risk for hypertension incidence by conducting longitudinal analyses of the first National Health and Nutrition Examination Survey ($n=4810$) using Cox proportional hazards models and controlling for covariates.” They found, “Sleep durations of ≤ 5 hours per night were associated with a significantly increased risk of hypertension (hazard ratio, 2.10; 95% CI, 1.58 to 2.79) in subjects between the ages of 32 and 59 years, and controlling for the

potential confounding variables only partially attenuated this relationship. The increased risk continued to be significant after controlling for obesity and diabetes.”

INSUFFICIENT SLEEP AND DIABETES

Hayashino et al. (2007, p.1) looked at the relationship between sleep quality and quantity and the risk of developing diabetes among “healthy workers” in Japan. “Of the 6509 participants included in the current analysis, the average age (range) and body-mass index at baseline were 38.2 (19-69) years and 22.6 kg/m², suggesting that the study population consisted of relatively young and lean workers” (p.3). Although they found no connection between length of sleep and diabetes, “For participants who often experienced difficulty in initiating sleep, the multivariate-adjusted hazard ratios for diabetes were 1.42 (95% CI, 1.05-1.91) in participants with a medium frequency of difficulty initiating sleep, and 1.61 (95% CI, 1.00-2.58) for those with a high frequency, with a statistically significant linear trend” (p.1).

Gottlieb et al. (2005, p.863) “assessed the cross-sectional relation of usual sleep time to diabetes mellitus (DM) and [impaired glucose tolerance (IGT)] among participants in the Sleep Heart Health Study.” They report that “Compared with those sleeping 7 to 8 hours per night, subjects sleeping 5 hours or less and 6 hours per night had adjusted odds ratios for DM of 2.51 (95% confidence interval, 1.57-4.02) and 1.66 (95% confidence interval, 1.15-2.39), respectively. Adjusted odds ratios for IGT were 1.33 (95% confidence interval, 0.83-2.15) and 1.58 (95% confidence interval, 1.15-2.18), respectively. Subjects sleeping 9 hours or more per night also had increased odds ratios for DM and IGT.”

SEDENTARY PATTERN AND OBESITY

Caban et al. (2005, p.1) produced a report on obesity rates across professional categories in the United States. Their report is based on self-reported weight and height collected annually on US workers age 18 or over, from the 1986 to 1995 and the 1997 to 2002 National Health Interview Surveys. The authors used survey responses to calculate annual occupation-specific prevalence rates for obesity. They report “pooled obesity prevalence rates were highest in motor vehicle operators (31.7% in men; 31.0% in women).” “During the period from 1986 to 1995, the highest pooled obesity rates were observed for male workers employed as motor vehicle operators (19.8%) ...for female workers, the highest pooled obesity rates were among motor vehicle operators (22.6%)” (p.5). “In the period from 1997 to 2002, the highest pooled obesity rates were observed for male workers employed as motor vehicle operators (31.7%) ... for female workers, those employed as motor vehicle operators (31.0%).”

Mummery et al. (2005, p.91) looked at “occupational sitting time” and BMI among 1,579 full-time Australian workers. Within the sample, mean sitting time for men was 209 minutes. The authors report “Univariate analyses showed significant associations between occupational sitting time and BMI of > or = 25 in men but not in women.” “The odds ratio for BMI > or = 25 was 1.92 (CI 1.17-3.17) in men who reported sitting for >6 hours/day compared with those who sat for <45 minutes/day.”

Dahl et al. (2009, p.345) report that a 10-year follow-up study beginning in 1994 was done to “examine standardized hospital treatment ratios (SHR) of lifestyle related diseases in a cohort of long haul truck drivers in Denmark compared with SHRs among other truck drivers and the working population at large.” They found that “Compared to the working population at large both long haul and other drivers had a statistical significant elevated risk for being hospitalized for obesity (SHR:254, 95% CI: 127-454) and diabetes mellitus (SHR:140, 95% CI: 104-185).”

“Personal lifestyle and working conditions are supposed to be tightly interwoven in long haul truck driving, but when compared to other truck drivers this does not reflect major differences in lifestyle related diseases, with the exception of a significantly lower risk for alcohol-related diseases and a possibly higher risk for lung cancer. All truck drivers had an increased risk of hospital treatment for diseases related to excess caloric intake and lack of exercise.”

Healy et al. (2008, p.661) looked at the flip side of sedentary behavior/obesity. They followed 168 participants in the Australian Diabetes, Obesity and Lifestyle study to see whether those who had more frequent breaks in their sedentary time as measured over seven consecutive days (although experiencing the same overall amount of sedentary time) would show better scores in terms of several health measures including BMI and resting blood pressure. They report, “Independent of total sedentary time and moderate-to-vigorous intensity activity time, increased breaks in sedentary time were beneficially associated with waist circumference (standardized β = -0.16, 95% CI -0.31 to -0.02, P = 0.026), BMI (β = -0.19, -0.35 to -0.02, P = 0.026), triglycerides (β = -0.18, -0.34 to -0.02, P = 0.029), and 2-h plasma glucose (β = -0.18, -0.34 to -0.02, P = 0.025).”

SEDENTARY PATTERN AND METABOLISM

Hamilton et al. (2007, p.2655) looked at sedentary time and its relationship to mortality, CV disease, Type 2 diabetes, metabolic syndrome and obesity. The authors go beyond the usual examination of levels of exercise and look at the cellular processes involved in extended sitting (as opposed to “the normally high volume of intermittent nonexercise physical activity in everyday life”). They experimented in the laboratory with “reducing normal spontaneous standing and ambulatory time” to see the effect on a protein “important for controlling plasma triglyceride catabolism, HDL cholesterol, and other metabolic risk factors.” They found, “Experimentally reducing normal spontaneous standing and ambulatory time had a much greater effect on LPL regulation than adding vigorous exercise training on top of the normal level of nonexercise activity.” They conclude “the average nonexercising person may become even more metabolically unfit in the coming years if they sit too much.”

SEDENTARY PATTERN AND INCREASED RISK OF MORTALITY

Katzmarzyk et al. (2009, p.998) “prospectively examined sitting time and mortality in a representative sample of 17,013 Canadians 18-90 [years] of age.” Subjects were followed for an average of 12 years; 1,832 deaths occurred during the period. Sitting time was characterized as “almost none of the time,” “one fourth of the time,” “half of the time,” three fourths of the time,” and “almost all of the time.” The authors report, “After adjustment for potential confounders, there was a progressively higher risk of mortality across higher levels of sitting time from all causes (hazard ratios (HR): 1.00, 1.00, 1.11, 1.36, 1.54; P for trend <0.0001) and CVD (HR: 1.00, 1.01, 1.22, 1.47, 1.54; P for trend <0.0001) but not cancer.” This held true independent of leisure-time activity.

OBESITY AND HEALTH OUTCOMES

Mokdad et al. (2008, p.76) reviewed data from the 2001 Behavioral Risk Factor Surveillance System (BRFSS) to look for associations between obesity and health risk factors. They defined overweight and obesity as follows: overweight – BMI 25 through 29.9; obesity – BMI 30 – 39.9; BMI 40 or higher. They report, “Overweight and obesity were significantly associated with diabetes, high blood pressure, high cholesterol, asthma, arthritis, and poor health status. Compared with adults with normal weight, adults with a BMI of 40 or higher had an odds ratio

(OR) of 7.37 (95% confidence interval [CI], 6.39-8.50) for diagnosed diabetes, 6.38 (95% CI, 5.67-7.17) for high blood pressure, 1.88 (95% CI, 1.67-2.13) for high cholesterol levels, 2.72 (95% CI, 2.38-3.12) for asthma, 4.41 (95% CI, 3.91-4.97) for arthritis, and 4.19 (95% CI, 3.68-4.76) for fair or poor health.”

Lenz et al. (2009, p.641) reviewed 27 meta-analyses (international) and 15 cohort studies (German) to determine whether overweight and obesity elevate morbidity and mortality. They did not find an elevated mortality rate, but in both overweight and obese individuals the risk for certain disease-specific morbidity was elevated: “The overall mortality of overweight persons (body mass index [BMI] 25-29.9 kg/m²) is no higher than that of persons of normal weight (BMI 18.5-24.9 kg/m²), but their mortality from individual diseases is elevated, diminished or unchanged, depending on the particular disease.” Disease-specific risk areas include cardiovascular risk, Type 2 diabetes, orthopedic complications, neoplastic diseases, asthma, renal diseases, and gastroesophageal reflux disease. The studies reviewed by Lenz et al. indicate that, “Morbidity and mortality are markedly influenced by” demographic characteristics such as age, sex, ethnic origin, and social status.

Finkelstein (2010, p. 336) presented data from the National Health Interview Survey Linked Mortality Files to estimate life expectancies by levels of weight, age, race, gender, and smoking status. Obesity levels II (BMI 35 <40) were significantly associated with the loss of 4 to 5 years of life for whites. Obesity levels III (BMI 40+) were significantly associated with the loss of 5 to 10 years across both races. Smoking status made little difference.

Grotle et al. (2008, n.p.) explored the possible relationship between obesity and osteoarthritis in the knee, hip, and hand among 1,854 Norwegians aged 24-76 years. The authors followed participants for 10 years and included 1,675 persons in the analysis. The authors defined obesity as BMI of 30 and above; osteoarthritis was self-reported. “At 10-years follow-up the incidence rates were 5.8 percent (CI 4.3-7.3) for hip OA, 7.3 percent (CI 5.7-9.0) for knee OA, and 5.6 percent (CI 4.2-7.1) for hand OA. When adjusting for age, gender, work status and leisure time activities, a high BMI (>30) was significantly associated with knee OA (OR 2.81; 95% CI 1.32-5.96), and a dose-response relationship was found for this association. Obesity was also significantly associated with hand OA (OR 2.59; 1.08-6.19), but not with hip OA (OR 1.11; 0.41-2.97). There was no statistically significant interaction effect between BMI and gender, age or any of the other confounding variables.”

OBSTRUCTIVE SLEEP APNEA AND HIGH BLOOD PRESSURE

Okada et al. (2006, p.891) studied 207 men (age 30 to 76) who had undergone health screenings. Based on polysomnography, 29 percent were considered to have sleep-disordered breathing with hypopnea. “The frequency of obesity (BMI≥25), hypertension, hypercholesterolemia, fasting blood glucose level, and HbA1c were significantly higher in patients with SDB than in normal individuals (AHI<5 times/h).” “The results ... suggest that as SDB becomes severe, it becomes more closely linked to the onset of lifestyle-related illnesses, such as hypertension, hypercholesterolemia, and abnormal glucose metabolism.”

OBSTRUCTIVE SLEEP APNEA AND CARDIOVASCULAR DISEASE

Chami et al. (2008, n.p.) “assessed the relation of SDB to LV morphology and systolic function in a community based sample of middle-aged and older adults.” They report “A polysomnographically derived apnea-hypopnea index (AHI) and hypoxemia index (percent of sleep time with oxyhemoglobin saturation <90%) were used to quantify SDB severity. LV mass

index was significantly associated with both AHI and hypoxemia index after adjustment for age, sex, ethnicity, study site, body mass index, current and prior smoking ... etc.” They conclude “In a community-based cohort, SDB is associated with echocardiographic evidence of increased LV mass and reduced LV systolic function.”

Mehra et al. (2006, p.910) report that for 6,441 members of the Sleep Heart Health Study, “individuals with severe sleep-disordered breathing have two-to fourfold higher odds of complex arrhythmias than those without sleep-disordered breathing even after adjustment for potential confounders.”

OBSTRUCTIVE SLEEP APNEA AND DIABETES

Seicean et al. (2008, p.1001) looked for a possible association between “sleep-disordered breathing [SDB],” diabetes precursors (impaired fasting glucose – IFG and impaired glucose tolerance – IGT), and “occult diabetes” among 2,588 study participants aged 52 to 96 years. “SDB was observed in 209 non overweight and 1,036 overweight/obese participants. SDB groups had significantly higher adjusted prevalence and adjusted odds of IFG, IFG plus IGT, and occult diabetes. The adjusted odds ratio for all subjects was 1.3 (95% CI 1.1-1.6) for IFG, 1.2 (1.0-1.4) for IGT, 1.4 (1.1-2.7) for IFG plus IGT, and 1.7 (1.1-2.7) for occult diabetes.” Associations held even after adjusting for age, sex, race, BMI, waist circumference. The authors conclude “The significant association ... suggests the importance of SDB as a risk factor for clinically important levels of metabolic dysfunction.”

Marshall et al. (2009, p.15) examined sleep apnea as an independent risk factor for diabetes. Among 295 study participants, “at baseline moderate severe OSA [obstructive sleep apnea] was associated with a univariate, but not multivariate, increased risk of diabetes (odds ratio = 4.37, 95% CL = 1.12, 17.12). Longitudinally, moderate-severe OSA was a significant univariate and independent risk factor for incident diabetes (fully adjusted OR = 13.45, 95% CL = 1.59, 114.11).”

OBSTRUCTIVE SLEEP APNEA AND INCREASED RISK OF MORTALITY

Marshall et al. (2008, p.1079) examined whether OSA “is an independent risk factor for all-cause mortality in a community-based sample free from clinical referral bias.” “Among the 380 participants ... moderate-to-severe OSA was independently associated with greater risk of all-cause mortality (fully adjusted hazard ratio [HR] = 6.24, 95% CL 2.01, 19.39) than non-OSA ($n = 285$, 22 deaths). Mild OSA (RDI 5 to <15/hr) was not an independent risk factor for higher mortality (HR = 0.47, 95% CL 0.17, 1.29).” The authors conclude, “Moderate-to-severe sleep apnea is independently associated with a large increased risk of all-cause mortality in this community-based sample.”

Punjabi et al. (2009, p.1) reported on the relationship between sleep-disordered breathing and mortality among 6,441 men and women participating in the Sleep Heart Health Study and concluded, “Sleep-disordered breathing is associated with all-cause mortality and specifically that due to coronary artery disease, particularly in men aged 40-70 years with severe sleep-disordered breathing.”

COSTS OF NEGATIVE HEALTH OUTCOMES

The potential costs of negative health outcomes can be measured in two ways: the actual dollar costs of medical care and associated costs for particular health problems; or increased

mortality. Below we present a brief overview of selected studies which give some idea of the range of costs that may be experienced by overweight or obese persons.

Cost of overweight or obesity

Martin et al. (2009, p. 180) conducted a study among drivers for a large national transportation logistics company; the study was a “retrospective cross sectional study design in which BMI was measured at baseline and costs were ascertained in the 1 year follow-up period. Costs and disease prevalences were compared across normal weight, overweight, and obese subjects.” The study *n* was 2,950. The authors report, “Unadjusted trimmed total cost for overweight subjects (\$1613) and obese subjects (\$1792) were significantly higher than for normal weight subjects (\$1012; $P < 0.05$). After multivariate adjustment, obese and overweight subjects had on average, \$591 ($P=0.031$) and \$383 ($P=0.188$) higher total trimmed health care cost than normal weight subjects.” “Both overweight and obese individuals had higher health care costs and higher prevalence of hyperlipidemia, diabetes, and hypertension than their normal weight counterparts.”

Banno et al. (2008, p.247) discuss additional expenses incurred by obese women with and without sleep apnea, compared against normal weight controls. “Obese women are heavier users of health services than normal weight controls. Obese women with [obstructive sleep apnea syndrome] OSAS use significantly more health services than obese controls.” (p.247). “Physician fees, in Canadian dollars, one year before diagnosis in the OSAS cases were higher than in obese controls: $\$547.49 \pm 34.79$ vs $\$246.85 \pm 20.88$ ($P < 0.0001$).” “Physician visits one year before diagnosis in the OSAS cases were more frequent than in the obese controls: 13.2 ± 0.73 visits vs 7.26 ± 0.49 visits ($P < 0.0001$).”

Schulte et al. (2008, p.560) present an overview of the interaction between occupational hazards and obesity. In terms of cost, they cite studies that have measured “the annual direct medical and absenteeism costs in the US attributable to excess weight” as being between \$175 to \$2,027 for men and \$588 to \$2,164 for persons with BMI from 25 to over 40.

Rosekind et al. (2010, p.91) conducted a web-based anonymous survey of employees at “four US-based companies.” They used the survey responses to classify employees into sleep-disturbed groups based on criteria for insomnia and insufficient sleep syndrome. They used responses from the Work Limitations Questionnaire as a basis for assessing productivity losses and costs among respondents. The authors conclude, “Fatigue-related productivity losses were estimated to cost \$1967/employee annually.”

Hauner (2009, p. 639) cites a report of a study on BMI and cause-specific mortality in 900,000 adults published in 2009 which “showed an average loss of 2 to 4 years of life with a BMI between 30 and 34.9 kg/m², and a BMI between 40 and 45 kg/m² shortened life by an average of 8 to 10 years.”

DISCUSSION

The research cited here, along with other studies that have reached similar conclusions, supports the view that the effects of a sedentary lifestyle and insufficient sleep put individuals at risk for overweight or obesity. Overweight and obesity in turn contribute to a range of negative health effects that may be damaging by themselves, or may lead to other health problems. The policy implications of this view suggest that employment rules favoring a more active lifestyle and more adequate sleep could lead to overall health benefits.

471 A number of researchers have noted the need for further work to refine our understanding of the
472 role of sleep in maintaining health. Grandner and Patel (2009, p. 146) point out that “research
473 needs to address the role of individual differences regarding sleep duration preferences. We
474 need to differentiate between natural (possibly healthy) short/long sleep and
475 insufficient/overextended sleep.” Similarly, “We need to conduct community-based intervention
476 studies to assess the effect of modifying sleep times on health outcomes and mortality.”

477 Czeisler (2009, p.249-275), in his review of current knowledge on medical and genetic
478 differences in the effect of sleep loss on individual performance, notes these effects may be
479 related to age, to the effects of food, drugs or pharmacological agents, work schedules, sleep
480 disorders, family responsibilities, psychiatric disorders, or other factors. In writing about work
481 schedules for physicians, Czeisler emphasizes the need to better understand the medical and
482 genetic basis of individual differences, and calls for the integration of this understanding into
483 policy-setting for work schedules and hours. Van Dongen and Belenky (2009, p.518) note “trait
484 individual variability in vulnerability to performance impairment due to sleep loss” and they state:
485 “Judiciously selecting or monitoring individuals in specific tasks or occupations, within legally
486 and ethically acceptable boundaries, has the potential to improve operational performance and
487 productivity, reduce errors and accidents, and save lives.”

488 The Mollicone et al. (2008, p.833) study is one example of another direction for continued
489 research – sleep scheduling to maximize sleep benefits while supporting work schedules. The
490 authors studied 90 individuals assigned to “a range of sleep/wake scenarios with chronically
491 reduced nocturnal sleep, augmented with a diurnal nap.” They conclude “The results suggest
492 that reductions in total daily sleep result in a near-linear accumulation of impairment regardless
493 of whether sleep is scheduled as a consolidated nocturnal sleep period or split into a nocturnal
494 anchor sleep period and a diurnal nap” making split sleep schedules feasible for work requiring
495 restricted night-time sleep.

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References

- Amagai, Y., Ishikawa, S., Gotoh, T., Doi, Y., Kayaba, K., Nakamura, Y., and Kajii, E., "Sleep Duration and Mortality in Japan: the Jichi Medical School Cohort Study," *Journal of Epidemiology*, Vol. 14, No. 4, July 2004, pp.124-128. Available in the docket: FMCSA-2004-19608-3955.
- Artazcoz, L., Cortés, I., Escribà-Agüir, V., Cascant, L., and Villegas, R., "Understanding the Relationship of Long Working Hours with Health Status and Health-Related Behaviours," *Journal of Epidemiology and Community Health*, Vol. 63, No. 7, July 2009, pp. 521-527. Available in the docket: FMCSA-2004-19608-3998.
- Banks, S. and Dinges, D.F., "Behavioral and Physiological Consequences of Sleep Restriction," *Journal of Clinical Sleep Medicine*, Vol. 3, No. 5, August 15, 2007, pp. 519-528. Available in the docket: FMCSA-2004-19608-3957.
- Banno, K., Ramsey, C., Walld, R., and Kryger, M., "Expenditure on Health Care in Obese Women With and Without Sleep Apnea," *Sleep*, Vol. 32, No. 2, May 2009, pp. 247-252. Available in the docket: FMCSA-2004-19608-3958.
- Bureau of Labor Statistics, "Labor Force Statistics from the Current Population Survey (2009)." Retrieved August 18, 2010, from: <http://www.bls.gov/cps/tables.htm>. Available in the docket: FMCSA-2004-19608-4023.1.
- Caban, A., Lee, D., Fleming, L., Gómez-Marín, O., LeBlanc, W., and Pitman, T., "Obesity in US Workers: the National Health Interview Survey, 1986 to 2002," *American Journal of Public Health*, Vol. 95, No. 9, September 2005, pp. 1614-1622. Available in the docket: FMCSA-2004-19608-3961.
- Cappuccio, F., Taggart, F., Ngianga-Bakwin, K., Currie, A., Peile, E., Stranges, S., and Miller, M., "Meta-Analysis of Short Sleep Duration and Obesity in Children and Adults," *Sleep*, Vol.31, No. 5, May 2008, pp. 619-626. Available in the docket: FMCSA-2004-19608-3962.
- Centers for Disease Control and Prevention (CDC), "Perceived Insufficient Rest or Sleep – Four States, 2006," *Morbidity and Mortality Weekly Report*, Vol. 29, No. 57(08), February 2008, pp. 200-203. Available in the docket: FMCSA-2004-19608-3964.
- Chami, H., Devereux, R., Gottdeiner, J., Mahra, R., Roman, M., Benjamin, E., and Gottleib, D., "Left Ventricular Morphology and Systolic Function in Sleep-Disordered Breathing," *Circulation*, Vol. 117, No. 20, May 2008, p. 2599. Available in the docket: FMCSA-2004-19608-3963.
- Chen, J.C., Chen, Y.J., Chang, W.P., and Christiani, -D.C., "Long Driving Time Is Associated with Haematological Markers of Increased Cardiovascular Risk in Taxi Drivers," *Occupational and Environmental Medicine*, Vol. 62, No. 12, December 2005, pp. 890–894. Available in the docket: FMCSA-2004-19608-3965.

- Czeisler, C., “Medical and Genetic Differences in the Adverse Impact of Sleep Loss on Performance: Ethical Considerations for the Medical Profession,” *Transactions of the American Clinical and Climatological Association*. Vol. 120, 2009, pp. 249-285. Available in the docket: FMCSA-2004-19608-3966.
- Dahl, S., Kaerlev, L., Jensen, A., Tüchsen, F., Hannerz, H., Nielsen, P.S., and Olsen, J., “Hospitalization for Lifestyle Related Diseases in Long Haul Drivers Compared with Other Truck Drivers and the Working Population at Large,” *Work*, Vol. 33, 2009, pp. 345-353. Available in the docket: FMCSA-2004-19608-4000.
- Di Milia, L. and Mummery, K., “The Association Between Job Related Factors, Short Sleep and Obesity,” *Industrial Health*, Vol. 47, 2009, pp. 363–368. Available in the docket: FMCSA-2004-19608-3967.
- Dinges, D., Maislin, G., Krueger, G., Brewster, R., and Carroll, R., “Pilot Test of Fatigue Management Technologies,” 2005. Available in the docket: FMCSA-2004-19608-4025.
- Ferrie, J., Shipley, M., Cappuccio, F., Brunner, E., Miller, M., Kumari, M., and Marmot, M., “A Prospective Study of Change in Sleep Duration: Associations with Mortality in the Whitehall II Cohort,” *Sleep*, Vol. 30, No. 12, 2007, pp. 1659-1666. Available in the docket: FMCSA-2004-19608-3969.
- Finkelstein, E.A., Brown, D.S., Wraga, L.A., Allaire, B. T., and Hoerger, T.J., “Individual and Aggregate Years-of-Life-Lost Associated with Overweight and Obesity,” *Obesity*, Vol. 18, No. 2, February 2010, pp. 333-339. Available in the docket: FMCSA-2004-19608-4006.
- Flegal, K.M., Carroll, M.D., Ogden, C.L. and Johnson, C.L., “Prevalence and Trends in Obesity Among U.S. Adults, 1999-2008,” *Journal of the American Medical Association*, Vol. 303, No. 3, 2010, pp. 235-241. Available in the docket: FMCSA-2004-19608-3970.
- Gangwisch, J., Heymsfield, S., Boden-Albala, B., Buijs, R., Kreier, F., Pickering, T., Rundle, A., Zammit, G., and Malaspina, D., “Short Sleep Duration as a Risk Factor for Hypertension: Analyses of the First National Health and Nutrition Examination Survey,” *Hypertension: Journal of the American Heart Association*, Vol. 47, April 2006, pp. 833-839. Available in the docket: FMCSA-2004-19608-3972.
- Gottlieb, D., Punjabi, N., Newman, A., Resnick, H., Redline, S., Baldwin, C., and Nieto, J., “Association of Sleep Time with Diabetes Mellitus and Impaired Glucose Tolerance,” *Archives of Internal Medicine*, Vol. 165, April 2005, pp. 863-868. Available in the docket: FMCSA-2004-19608-3973.
- Grandner, M. and Patel, N., “From Sleep Duration to Mortality: Implications of Meta-Analysis and Future Directions,” *Journal of Sleep Research*, Vol. 18, 2009, pp. 145-147. Available in the docket: FMCSA-2004-19608-3974.
- Grotle, M., Hagen, K., Natvig, B., Dahl, F., and Kvien, T., “Obesity and Osteoarthritis in Knee, Hip and/or Hand: An Epidemiological Study in the General Population with 10 years Follow-Up,” *Bio Med Central Musculoskeletal Disorders*, Vol. 9, No. 132, October 2008, n.p.. Available in the docket: FMCSA-2004-19608-3975.

- Hamilton, M. T., Hamilton, D. G., and Zderic, T. W., "Role of Low Energy Expenditure and Sitting in Obesity, Metabolic Syndrome, Type 2 Diabetes, and Cardiovascular Disease," *Diabetes*, Vol. 56, No. 11, November 1, 2007, pp. 2655 - 2667. Available in the docket: FMCSA-2004-19608-3976.
- Hauner, H., "Overweight – Not Such a Big Problem," *Deutsches Ärzteblatt International*, Vol. 106, No. 40, 2009, pp. 630-640. Available in the docket: FMCSA-2004-19608-3979.
- Hayashino, Y., Fukuhara, S., Suzukamo, Y., Okamura, T., Tanaka, T., and Ueshima, H., "Relation Between Sleep Quality and Quantity, Quality of Life, and Risk of Developing Diabetes in Healthy Workers in Japan: the High-Risk and Population Strategy for Occupational Health Promotion (HIPOP-OHP) Study," *Bio Med Central Public Health*, Vol. 7, No. 129, June 2007, n.p.. Available in the docket: FMCSA-2004-19608-3980.
- Healy, G., Dunstan, D., Salmon, J., Cerin, E., Shaw, J., Zimmet, P., and Owen, N., "Breaks in Sedentary Time: Beneficial Associations with Metabolic Risk," *Diabetes Care*, Vol. 31, No. 4, April 2008, pp. 661-666. Available in the docket: FMCSA-2004-19608-3981.
- Katzmarzyk, P.T., Church, T.S., Craig, C.L., and Bouchard, C., "Sitting Time and Mortality from All Causes, Cardiovascular Disease, and Cancer," *Medicine and Science in Sports and Exercise*, Vol. 41, No. 5, May 2009, pp. 998-1005. Available in the docket: FMCSA-2004-19608-4001.
- Knauth, P., "Extended Work Periods," *Industrial Health*, Vol. 45, 2007, pp. 126-136. Available in the docket: FMCSA-2004-19608-4009.
- Lenz, M., Richter, T., and Mühlhauser, I., "The Morbidity and Mortality Associated with Overweight and Obesity in Adulthood," *Deutsches Ärzteblatt International*, Vol. 106, No.40, 2009, pp. 641-648. Available in the docket: FMCSA-2004-19608-4012.
- Marshall, N., Wong, K., Liu, P., Cullen, S., Knuiman, M., and Grunstein, R., "Sleep Apnea as an Independent Risk Factor for All-Cause Mortality: the Busselton Health Study," *Sleep*, Vol. 31, No. 8, 2008, pp. 1079-1085. Available in the docket: FMCSA-2004-19608-4013.
- Marshall, N., Wong, K., Phillips, C., Liu, P., Knuiman, M., and Grunstein, R., "Is Sleep Apnea an Independent Risk Factor for Prevalent and Incident Diabetes in the Busselton Health Study?" *Journal of Clinical Sleep Medicine*, Vol. 5, No. 1, 2009, pp. 15-20. Available in the docket: FMCSA-2004-19608-4014.
- Martin, B.C., Church, T.S., Bonnell, R., Ben-Joseph, R., and Borgstadt, T., "The Impact of Overweight and Obesity on the Direct Medical Costs of Truck Drivers," *Journal of Occupational and Environmental Medicine*, Vol. 51, No. 2, February 2009, pp. 180 –184. Available in the docket: FMCSA-2004-19608-4004.
- Mehra, R., Benjamin, E., Shahar, E., Gottlieb, D., Nawabit, R., Kirchner, H. Sahadevan, J., and Redline, S., "Association of Nocturnal Arrhythmias with Sleep-Disordered Breathing: The Sleep Heart Health Study," *American Journal of Respiratory and Critical Care Medicine*, Vol. 173, 2006, pp. 910-916. Available in the docket: FMCSA-2004-19608-4015.
- Mokdad, A.H., Ford, E.S., Bowman, B.A., Dietz, W.H., Vinicor, F., Bales, V.S., and Marks, J.S., "Prevalence of Obesity, Diabetes, and Obesity-Related Health Risk Factors, 2001,"

- 647 *Journal of the American Medical Association*, Vol. 289, No. 1, January 2003, pp. 76-79.
648 Available in the docket: FMCSA-2004-19608-4016.
649
- 650 Mollicone, D. J., Van Dongen, H.P., Rogers, N.L., and Dinges, D.F., "Response Surface
651 Mapping of Neurobehavioral Performance: Testing the Feasibility of Split Sleep
652 Schedules for Space Operations," *Acta Astronaut*, Vol. 63, No. 7-10, 2008, pp. 833-840.
653 Available in the docket: FMCSA-2004-19608-4017.
654
- 655 Mummery, W., Schofield, G., Steele, R., Eakin, E., and Brown, W., "Occupational Sitting Time
656 and Overweight and Obesity in Australian Workers," *American Journal of Preventive
657 Medicine*, Vol. 29, No. 2, August 2005, pp. 91-97. Available in the docket: FMCSA-
658 2004-19608-4002.
659
- 660 National Institute of Diabetes and Digestive and Kidney Diseases, "Overweight and Obesity
661 Statistics," February 2010. Available in the docket: FMCSA-2004-19608-3982.
662
- 663 Okada, M., Takamizawa, A., Tsushima, K., Urushihata, K., Fujimoto, K., and Kubo, K.,
664 "Relationship Between Sleep-Disordered Breathing and Lifestyle-Related Illnesses in
665 Subjects Who Have Undergone Health-Screening," *Internal Medicine* (The Japanese
666 Society of Internal Medicine), Vol. 45, No. 15, 2006, pp. 891-896. Available in the
667 docket: FMCSA-2004-19608-3983.
668
- 669 Patel, S. and Hu, F., "Short Sleep Duration and Weight Gain: A Systematic Review," *Obesity*,
670 Vol.16, No. 3, March 2008, pp. 643-653. Available in the docket: FMCSA-2004-19608-
671 3984.
672
- 673 Punjabi, N., Caffo, B., Goodwin, J., Gottlieb, D., Newman, A., O'Connor, G., Rapoport, D.,
674 Redline, S., Resnick, H., Robbins, J., Shahar, E., Unruh, M., and Samet, J., "Sleep-
675 Disordered Breathing and Mortality: A Prospective Cohort Study," *PLoS Medicine*, Vol.
676 6, No. 8, August 2009, n.p.. Available in the docket: FMCSA-2004-19608-3997.
677
- 678 Rosekind, M., Gregory, K., Mallis, M., Brandt, S., Seal, B., and Lerner, D., "The Cost of Poor
679 Sleep: Workplace Productivity Loss and Associated Costs," *Journal of Occupational and
680 Environmental Medicine*, Vol. 51, No. 1, January 2010, pp. 91-98. Available in the
681 docket: FMCSA-2004-19608-4003.
682
- 683 Schoenborn, C. & Adams, P., "Sleep Duration as a Correlate of Smoking, Alcohol Use, Leisure-
684 Time Physical Inactivity, and Obesity Among Adults: United States, 2004-2006," National
685 Center for Health Statistics (CDC), NCHS Health E-Stats, May 2008. Available in the
686 docket: FMCSA-2004-19608-3985.
687
- 688 Schulte, P., Wagner, G., Downes, A., and Miller, D., "A Framework for the Concurrent
689 Consideration of Occupational Hazards and Obesity," *The Annals of Occupational
690 Hygiene*, Vol. 52, No. 7, September 2008, pp. 555-566. Available in the docket: FMCSA-
691 2004-19608-3988.
692
- 693 Seicean, S., Kirchner, H., Gottlieb, D., Punjabi, N., Resnick, H., Sanders, M., Budhiraja, R.,
694 Singer, M. and Redline, S., "Sleep-Disordered Breathing and Impaired Glucose
695 Metabolism in Normal-Weight and Overweight/Obese Individuals," *Diabetes Care*, Vol.
696 31, No. 5, May 2008, pp. 1001-1006. Available in the docket: FMCSA-2004-19608-3989.
697

- 698 Tamakoshi, A. and Ohno, Y., “Self-Reported Sleep Duration as a Predictor of All-Cause
699 Mortality: Results from the JACC Study, Japan,” *Sleep*, Vol. 27, No. 1, 2004, pp. 51-54.
700 Available in the docket: FMCSA-2004-19608-4018.
701
- 702 Van Cauter, E. and Knutson, K.L., “Sleep and the Epidemic of Obesity in Children and Adults,”
703 *European Journal of Endocrinology*, Vol. 159, 2008, pp. S59–S66. Available in the
704 docket: FMCSA-2004-19608-3991.
705
- 706 Van Dongen, H. and Belenky, G., “Individual Differences in Vulnerability to Sleep Loss in the
707 Work Environment,” *Industrial Health*, Vol. 47, 2009, pp. 518-526. Available in the
708 docket: FMCSA-2004-19608-3992.
709
- 710 Violanti, J., Burchfiel, C., Hartley, T., Mnatsakanova, A., Fekedulegn, D., Andrew, M., Charles,
711 L., and Vila, B., “Atypical Work Hours and Metabolic Syndrome Among Police Officers,”
712 *Archives of Environmental and Occupational Health*, Vol. 64, No. 3, Fall 2009, pp. 194-
713 201. Available in the docket: FMCSA-2004-19608-4005.
714