



Partnership for Air Transportation  
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# **A Review of the Literature Related to Potential Health Effects of Aircraft Noise**

PARTNER Project 19 Final Report

prepared by  
Hales Swift

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## **EXECUTIVE SUMMARY**

Previous reports have dealt with the “health effects of noise” in some capacity. Many of these have considered various quality of life factors as the primary health effects. This is in line with the World Health Organization (WHO, 1946) definition of health, which reads, “Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.” Noise was seen as detracting primarily from mental well being (annoyance for instance) or social well being (speech interference). However, recent studies have demonstrated a possible relationship between noise exposure, such as that caused by aircraft, and the more physiological side of the WHO definition: disease or infirmity. Led in part by industrial and laboratory studies showing acute effects such as transient blood pressure increases, a number of other recent studies have been conducted showing a mixture of possible short and long-term cardiovascular effects of noise. While not all studies have resulted in significant findings, a pattern of increased incidence of cardiovascular effects, hypertension and ischemic heart disease in particular, seems to have emerged. These purported effects are only recently documented and thus models accounting for their hypothesized societal costs are still in early stages.

A feature of this literature survey is its emphasis on cardiovascular outcomes and an evaluation of the potential pathways from aircraft noise to health outcomes for possible cardiovascular endpoints. This is in contrast to most previous reports on this subject, which were not focused as much on potential mechanisms for the proposed or observed effects. Two potential pathways are discussed: sleep disruption and noise-induced stress; because both have been related to possible cardiovascular outcomes. The relationship between aircraft noise and annoyance, disturbance of communication, and disruption of learning leading to delays in reading proficiency or difficulties in remembering material, have been reviewed elsewhere and readers are referred to that literature. However, a brief review of these topics is given in this report. The focus on the two pathways primarily covered in this report is because of the results of recent studies in Europe focused on the health impacts of transportation noise. Through these studies researchers have shown nighttime aircraft noise to be more highly correlated to health impacts than twenty-four hour or day-time noise, and have found that observed effects in road noise studies have become stronger when house orientation and window opening habits at night have been taken into account. Research into health effects of industrial noise exposure as well as health effects associated with annoyance from community noise suggests that stress reactions, such as arousal of the cardiovascular system in response to a loud noise, may lead to negative cardiovascular outcomes as well. It has been proposed that repeated short-term increases of blood pressure and heart rate associated with these reactions may lead to changes in the functioning of the cardiovascular system and eventual hypertension. Thus, both sleep and stress, because they have been proposed as pathways leading from noise exposure to eventual cardiovascular outcomes, are of interest and have been focused on in this report.

There are several potential problems that arise in health studies, e.g., unaccounted for confounding factors; removal of the impacts of certain factors which are known to be risk factors for cardiovascular disease but might also be outcomes of the noise exposure; inaccurate prediction of exposure to noise sources of interest; difficulties disambiguating impacts of total noise exposure versus exposure to a particular noise source of interest. In addition, adequate control of other factors like air quality, which may also be influenced by noise producing infrastructure, may pose challenges and increase the diversity of expertise needed for an effective study. As an example of the potential difficulties of confounding, shortening, fragmenting, or changing the stage structure of sleep may lead to increases in the prevalence of obesity, diabetes, hypertension and heart disease — noted components of the metabolic syndrome. However, obesity and diabetes (also potentially outcomes of sleep problems) seem to have been examined primarily as confounders in these noise and health studies. In assessing relevant exposures, it is rare that an observed effect is clearly and solely attributable to the noise source of interest, as total noise exposure (in work, while commuting, at home or elsewhere in the community) is rarely assessed. Further, the actual exposure inside the home may deviate considerably from the outdoor level and predictions even of outdoor exposure may not be accurate, although the accuracy is steadily improving with increased computational capability. In

future studies, it will be important to be able to address all of these issues so that a more accurate assessment of aircraft noise impacts can be made.

Potential cardiovascular health effects of exposure to aircraft noise need to be quantified so that the economic and ease-of-travel benefits of airports can be weighed against potential health effects of noise and other pollutants. Currently, hedonic measures are used to assess the negative impacts of noise around airports, yet it does not seem likely that they would reflect the impacts of cardiovascular effects of noise, which are largely unknown in the general population. Some would argue that hedonic measures do not even reflect non health-related quality of life effects, because people need to experience noise exposure before they truly understand its impacts, and the ability to move is limited once a house has been purchased. Others make strong arguments in favor of hedonic measures. With regards to cardiovascular health effects, it may be possible to extend the usefulness of hedonic measures through education of the public concerning potential risks, enabling them to make more informed choices when purchasing their homes. Other possibilities include the use of the system of Disability Adjusted Life Years (DALY's), which has been recommended as an effective decision making tool where both quality of life and disease issues were being considered in balance alongside potential economic benefits. This system is also used for other disease-related and quality-of-life outcomes resulting from air quality and climate change. Thus, using such a system it might be possible to better understand the relative contributions of each of these three undesirable outcomes of aircraft operations.

The last two chapters of this report include a discussion of unanswered questions and ideas and recommendations for future research that would: (1) help further elucidate the relationship between aircraft noise and health; and (2) provide decision makers with the tools that they need to optimize policy with regards to noise producing infrastructure in order to minimize negative health outcomes. Additionally, ways to conduct studies that might help address these unanswered questions and the feasibility of those approaches is also discussed.

# 1. INTRODUCTION

For most people, sound is an important and meaningful contributor to the experience of their environment and their daily activities. However, unwanted sound may interrupt activity where quiet is desirable, distract concentration, reduce the quality of communication, and contribute to the stress of individuals (Berglund and Lindvall, 1995; WHO, 2000). Much research has focused on addressing the fairly clear quality of life effects associated with annoyance by intrusive noises. A second prominent field of noise study has been sleep disturbance (see, for example, Passchier-Vermeer and Passchier, 2000; Finegold, Harris, von Gierke, 1994; Finegold and Elias, 2002). In part, this has been of interest because noise during sleep disrupts sleep (Basner, 2008) and may produce increased annoyance through remembered awakenings (Basner, Samel, Isermann, 2006). The difficulties, stresses and annoyance potentially arising as a result of unwanted noise exposure during waking or sleep have sometimes been termed “health effects” (see, for example, (WHO,2005); also for illustration, see Figure 1). This is consistent with the World Health Organization (WHO) definition of health which includes “complete physical, mental, and social well-being and not merely the absence of disease or infirmity” (WHO, 1946). However, at the time of many of these studies, sleep, while seen as psychologically beneficial, was not universally seen as a physiological necessity (Spiegel, Leproult, Van Cauter, 1999).

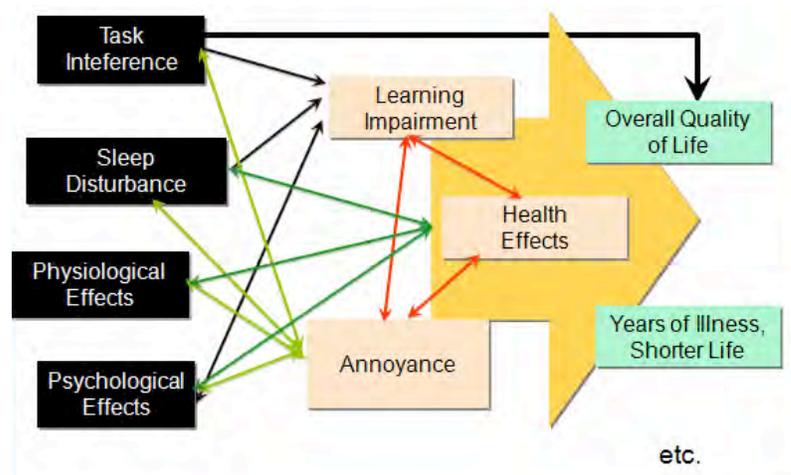


Figure 1: Broader Health Effects of Noise Context

Recently, observed changes in blood pressure in response to noise in the industrial setting have led to environmental studies focused on possible cardiovascular health effects of exposure to noise (see Babisch’s 2006 meta-analysis for a list of such studies). While not all studies have led to significant findings, a pattern of cardiovascular health effects of noise seems to have emerged. In recent studies increased prevalence of hypertension (see, for example, the Hypertension and Exposure to Noise near Airports (HYENA) study) and heart disease has been found in people exposed to higher levels of road and aircraft noise (Jarup, Babisch, Houthuijs, et al, 2008; Passchier-Vermeer and Passchier, 2000; van Kempen, Kruijze, Boshuizen, Ameling, Staatsen, de Hollander, 2002). A possible effect of noise on the development of ischemic heart disease was a main focus of a recent meta-analysis by Babisch (2006). Although ultimately not achieving statistical significance in the dose-response relationship (derived from a number of studies with similar designs), results of Babisch’s meta-analysis support the hypothesis that increases in environmental noise exposure may lead to increased prevalence of ischemic heart disease. Babisch has used his dose-response

relationship to evaluate the cost of possible health effects (WHO, 2005). Additional support for the hypothetical influence of noise on the prevalence of ischemic heart disease comes from industrial studies (Davies, Teschke, Kennedy, Hodgson, Hertzman, Demers, 2005; Ising, Babisch, Kruppa, Lindthammer, Wiens, 1997). These observed cardiovascular health effects are not as well-known or widely studied as the more direct and immediate quality of life effects (e.g. annoyance) and are fairly recently reported, whereas other consequences of noise (again, annoyance) may be both more intuitive and longer studied, see, for example, (Mestre, 2008). Thus, in examining these lesser known hypothesized effects, this report will primarily focus on health as the absence of infirmity and disease. As a result, while they will be mentioned here, many of the quality of life issues traditionally studied and reported in the literature dealing with health effects of noise will not be dealt with at length here in favor of the potentially more serious, but perhaps less well-known, consequences.

Another difference between this report and many of the previous reports is an emphasis on potential avenues for the development of the proposed illnesses. The works mentioned earlier, such as the HYENA study and Babisch's meta-analysis, were focused primarily on outcomes, although the researchers engaged in a brief discussion of the potential mechanisms at play. An interesting feature of both of these studies is their apparent support of a potential role of sleep disturbance in the genesis of cardiovascular health effects. An important part of Babisch's case for the existence of an effect of noise on heart disease is centered on the important role of modifiers of exposure and their catalyzing impact on the observed health effects. Stronger relationships occurred when modifiers such as window opening increased noise exposure during the night (similar effects of window opening were not observed during the day) or increased noise in the bedroom rather than the living room (Babisch, 2006). In the HYENA study, researchers found significant relationships between road-noise averaged over twenty-four hours and hypertension incidence, and nighttime aircraft noise averaged over nighttime hours and hypertension incidence. Thus, two of the seminal studies in the field seem to support a possible role of sleep disruption in mediating possible noise-induced health effects.

These results may, however, seem counterintuitive because the exposure measure was an energy averaged metric. Sleep is primarily disturbed by individual events rather than ambient levels and so the averaging of exposure energy throughout the 8-hr nighttime interval might be expected to produce a large decrease in the sensitivity of the measure to factors that influence sleep disturbance. Though the results seem to have achieved statistical significance, it may be desirable to use more event-sensitive measures in future studies as time averaged energy metrics may not be optimally sensitive to events that might lead to potential awakening (Basner, Samel, Isermann, 2006). {this is challenging but necessary for progress, need to look at cumulative effects of multiple events} A stronger result might thus have been seen with more event oriented metrics. Ideally, such a metric could also take into account the timing of the events and the history of any preceding sleep disturbance.

It is important to note that the use of event-centered metrics poses significant difficulties from a policy standpoint. Although an event-related metric might predict sleep disturbance more successfully, the exact relationships between inputs and outputs is not always as easily interpreted as an energy-averaged metric. Thus, further research is necessary both in the development of event-based sleep disturbance prediction techniques and in the development of guidance on how such a tool might be efficiently applied. An attempt to address awakenings resulting from acoustics events (measured in ASEL) and percentages awakened is the basis for the methodology described in the ANSI sleep disturbance standard, which is based on the work of Anderson and Miller. It also takes into account changes in the probability of awakening with time of night of the noise event (ANSI, 2008).

In other studies, focused on the direct effects of disrupted or shortened sleep (not necessarily caused by noise), researchers have found increased prevalence of heart disease and hypertension—the two outcomes examined in Babisch's meta-analysis and the HYENA study, respectively. Thus, reduced quantity and quality of sleep may lead to the results observed. In addition, reduced quantity and quality of sleep may also lead to increases in weight gain, prevalence of obesity, and increased occurrence of diabetes, which do not seem to have been studied with regard to noise—they were considered as confounders or exclusionary criteria in some studies (Babisch, 2006), but do not seem to have been considered as potential outcomes.

Accordingly, in this report, multiple types of sleep disruption caused by aircraft noise are discussed, as well as some of the potential consequences of these types of disruption for health outcomes. Several of the potential pathways from exposure to noise to health effects discussed in this report are mediated in some way by changes in sleep. Sleep disruption and deprivation arouses the sympathetic nervous system. It may also lead to changes in the hunger regulating hormones leptin and ghrelin and resultant increases in appetite and weight gain. Adiposity is a well-known risk factor for cardiovascular and metabolic outcomes. Disruption of nighttime sleep may serve to impair the nocturnal reduction in blood pressure seen in most healthy individuals as well as disrupt normal glucose management. Impairment of sleep has also been linked to increased incidence of hypertension, heart disease and diabetes. An illustration of the pathways between noise, sleep disturbance and health effects is shown in Figure 2.

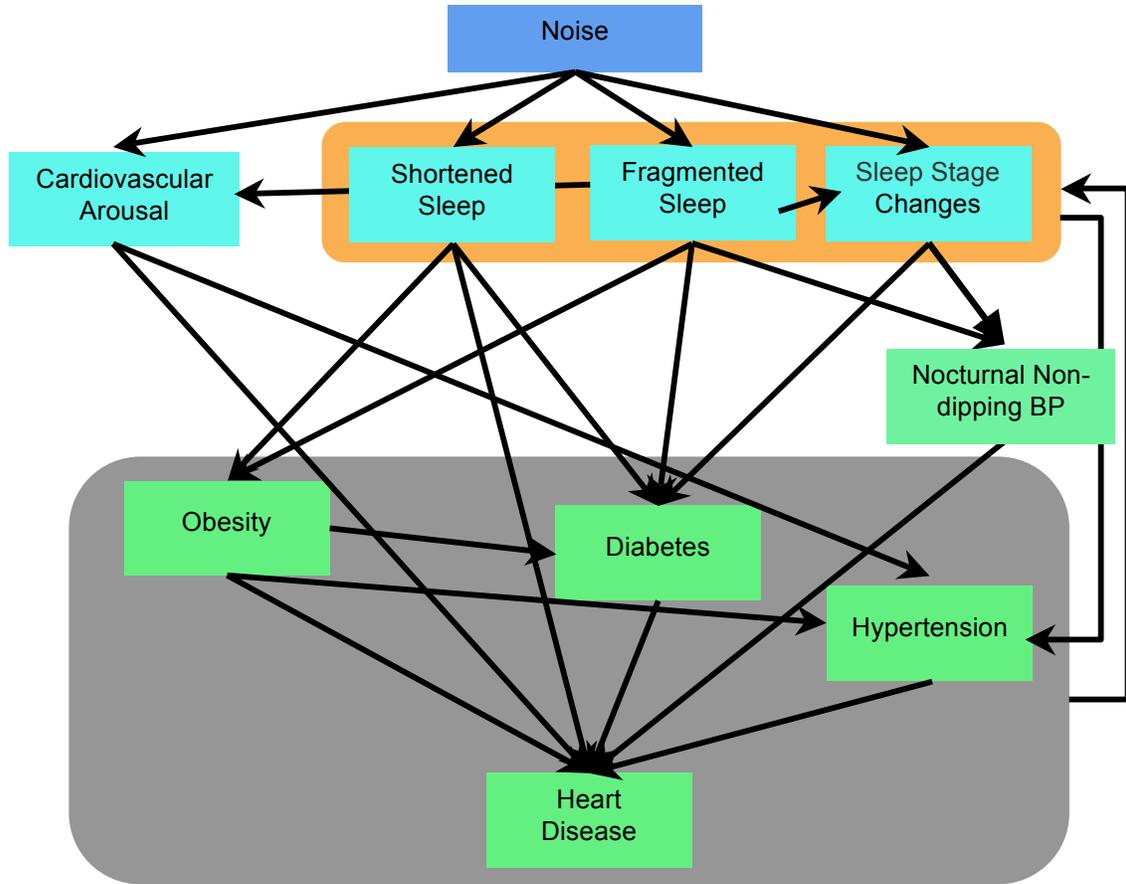


Figure 2: Proposed potential pathways for the health effects of noise through sleep disturbance.

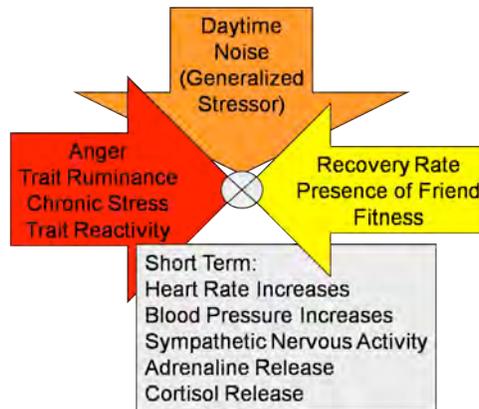


Figure 3: Schematic of potential responses and modifiers to stress reactions induced by noise.

The above list of maladies that may potentially result from sleep disruption and also contribute to heart disease is far from all-inclusive. Apart from yet unknown potential mediators, several possible pathways mentioned in the literature have not been investigated in this report and may be valuable to include at a later date. These include the action of the hypothalamic-pituitary-adrenal (HPA) axis mentioned in several studies (for example, Gottlieb, Punjabi, Newman, Resnick, Redline, Baldwin, Nieto, 2005), a postulated role of cholesterol (Ekstedt, Akerstedt, Soderstrom, 2004), as well as the role of stress (considered as a potential outcome or analogue of noise annoyance). Possible immune system effects of sleep disruption, while treated briefly in this report, may justify further examination.

In addition to potential nighttime effects, noise exposure during the day is also seen as a potential pathway to the development of cardiovascular disease, and may lead to important vascular changes. Researchers studying effects of industrial noise exposure have found significant changes in ambulatory blood pressure and other cardiovascular parameters. Both transient and sustained effects seen by researchers were interpreted as possibly promoting development of hypertension (Chang, Su, Lin, Jain, Chan, 2007; Chang, Jain, Wang, Chan, 2003), a known risk factor for heart disease. Additionally, increased incidence of myocardial infarction has been seen in industrial workers exposed to higher noise levels (Davies, Teschke, Kennedy, Hodgson, Hertzman, Demers, 2005; Ising, Babisch, Kruppa, Lindthammer, Wiens, 1997). However, many of the exposure levels in the industrial studies (85+dBA in some cases) were of larger magnitude than is typical of aircraft noise exposure. Also, much of potential aircraft exposure comes in a different set of environments; work versus home, for instance. Thus, it is not entirely clear how aircraft noise exposure at home or during leisure might compare with industrial levels of exposure while working given the environmental, exposure level, and task differences.

Long and short term physiological changes in response to noise exposure while awake, (Chang, Su, Lin, Jain, Chan, 2007; Chang, Jain, Wang, Chan, 2003) and short term physiological changes due to noise exposure while sleeping (Griefahn, Brode, Marks, Basner, 2008; Haralabidis et al, 2008) have been observed. However, significant questions still need to be answered. These relate to: the intensity of the effects; the relative contributions of exposure in multiple settings (including work, transit, recreation, home and others) from multiple sources or from differing types of sources (aircraft vs. other environmental exposure); and, the relative importance of exposure while awake and exposure while asleep. These answers are especially valuable for policy makers as they can help inform decision making about when to schedule flights in order to minimize potential negative health impacts. Also, another important question remains concerning whether there are particularly susceptible or vulnerable groups of people that are affected more strongly than the general population.

Future research may address the remaining potential sleep health effects (obesity and diabetes) by estimating the likely effect sizes with sleep disruption, types and degrees consistent with aircraft and

determining whether they are large enough to require further study and consideration when evaluating the cost of noise. A next step in this direction, if warranted, might be the re-evaluation of data from previously conducted studies to determine the degree of presence or absence of the predicted, but previously unlooked-for, effects. Some of these effects have been considered as potential confounders. Thus, data on them might already exist. Babisch (2006), for instance, mentions studies that have adjusted for obesity. At the very least, care should be taken in adjusting models for variables that may be mediating a causal relationship between noise-induced sleep changes and heart disease or hypertension. Disambiguating the effects of noise exposure while waking and sleeping may be challenging and may require multiple studies, each addressing a part of the overall picture or a single one with 24-hr exposure monitoring and blood pressure monitoring. Possible approaches might include an ambulatory blood pressure and a noise exposure study over a prolonged period of time. A large population study might make use of some of the Health Effects and Risks of Transportation System (HEARTS) methodology in which predictions of exposure to noise, pollution, etc. while following a daily routine are calculated and summed. This could allow a higher level of prediction of individual exposure in future studies to limit exposure misestimation, misclassification and misattribution of causes. The HEARTS methodology also allows for the assessment of contributions from multiple possible environmental stressors (e. g., noise and air pollution). Selander, Nilsson, Bluhm, Rosenlund, Lindqvist, Nise and Pershagen (2009) also examined the combined effects of air quality and noise, a logical pairing because of their strong covariance. Studies by Hoffman, Moebus, Stang, Beck, Dragano, Mohlenkamp, Schmermund, Memmesheimer, Mann, Erbel and Jockel (2006), and Tonne, Melly, Mittleman, Coull, Goldberg, and Schwartz (2007) in which both variables were not simultaneously considered show associations between nearness to infrastructure, but ambiguities remain as to the relative contributions of air quality and noise exposure factors to any outcomes. Air pollution has been looked upon as often co-varying with noise (WHO, 2005) and future efforts could potentially benefit from means to systematically take both noise and air pollution variables into account. Similar measurements of exposure could be accomplished if people were willing to allow tracking through GPS, or long-term noise dosimetry, though some subjects or businesses might find this unacceptably intrusive.

In decision-making processes involving potential positive and negative consequences of noise producing infrastructure, the reactions and decisions of the people experiencing the exposure may provide a useful tool to quantify the cost of the exposure. The more apparent quality of life issues have, to some extent, already been taken into account in people's decision-making processes in choosing where to live and whether to remain there over a period of time. Factors such as excess noise or nearness to infrastructure that is useful for travel or business may influence consumer decisions to buy or leave, influencing housing prices through supply and demand. Through the system of hedonic indicators, measures which suggest how quality of life has been influenced by infrastructure, housing prices, for example, are used as a barometer to gauge cost and benefit. Predictions based on this system seek to optimize advantages for all parties. By quantifying how people are both helped and harmed by the presence of the noise-producing infrastructure, such as an airport, decision makers can predict how current operations and future changes might affect quality of life. However, use of hedonic methods to quantify health effects has the potential limitation that homeowners may only react to the forces that they know to be relevant and may not take into account other significant effects, which while less obvious, may be more serious. A related issue with the use of housing prices to quantify the cost of noise is that when people purchase their houses near an airport, they may understand neither how noise will affect their quality of life nor the severity of the exposure. After moving into a house, the annoyance impact will be more fully understood, but it is less likely that they will be able to move at that point. Other methods of costing such as administering surveys asking how much residents are willing to pay for a given reduction or increase in some factor, for instance a given reduction in noise exposure are also used. These methods are collectively referred to (not surprisingly) as the Willingness To Pay or "WTP" method.

The potential cardiovascular effects of noise mentioned above are largely unknown to the general public and so may be safely assumed not to play a role in individual or population decisions regarding home buying or quality of life self-assessment. Hence, hedonic indicators are unlikely to adequately quantify their impacts. Several options exist for taking into account the potential cardiovascular outcomes. Education of the public might allow individuals to take these possibilities into account, thus allowing the system of hedonic indicators

to have continued and increased usefulness, however, this might lead to a disproportionately negative view of infrastructure. The cost of potential health outcomes, as well as quality of life outcomes, could be evaluated by using the DALY (Disability Adjusted Life Years) system (WHO, 2005). Because this measure is used widely to predict and evaluate the cost of other health outcomes, it helps us when trying to make comparisons between the total impact on health of various exposure increases or interventions. For example, for communities near airports, the health effects cost of chemical and particulate exposure can be compared to the health effects cost of noise. Some controversy concerning this system exists; for example, it is seen as focusing disproportionate attention on measureable outcomes and posing difficulties for dealing with comorbidities (Lyttekens, 2003). Nevertheless, the system is seen as valuable in weighing potential choices where both positives and negatives. Members of the ICAO CAEP noise panel noted that DALYs were applicable to noise and were well-developed for air quality issues. However, others of the panel felt that DALYs were not yet widely agreed upon for use in noise (Maurice and Lee (eds) 2009) result.

In conclusion, the potential cardiovascular and metabolic health effects of noise-exposure appear to be deserving of future exploration. This is especially true because of the potentially serious nature of the health effects and the valuable protective planning ability that such research and the resulting predictive tools may produce. The lesser-explored areas mentioned above—cholesterol, immune function, and the HPA axis they relate to noise-affected sleep—may also be valuable areas of future research and provide information which may be used to enhance policy and support health. Methods to quantify the potential health effects of noise remain a topic of ongoing discussion.

Due to the interdisciplinary nature of the material presented in this report, and the potential audience for the report, it was deemed appropriate to include sections describing some of the more important and potentially unfamiliar methods and terminology used later in the report. Included in this section is information on different noise metrics, prediction methods and sources of error. Also included are explanations of ambulatory blood pressure and other physiological metrics and methods. If the reader is already familiar with the information in a particular subsection, these sections can be skipped. The next major section of the report deals with sleep loss. The structure of the middle part of the report (Sections 3 and 4) was designed to mimic the proposed order of the generated effects: noise to physiological changes in the body and/or sleep disturbance to health effects: increased incidence of heart disease and hypertension, along with obesity and diabetes. Results of environmental studies (such as HYENA) support a possible role of sleep disturbance in the observed outcomes of increased hypertension with increased nighttime aircraft noise exposure. The next chapter of the report is focused on some of the effects that noise has on overall quality of life. Following that is a section on some of the unanswered questions and future research that needs to be conducted in order to answer some of these questions. The report ends with a discussion on how potential cardiovascular health effects of noise may be taken into account in cost-benefit analyses.

## 2. METHODS OF STUDYING HEALTH EFFECTS

As many readers may be unfamiliar with some of the disciplines that are needed to study health effects of noise, some background information useful for understanding later portions of the text are included. The glossary given in Appendix B may also be a useful reference for those unfamiliar with terminology and measures used in this field of research.

### 2.1 Types of Health/Epidemiological Studies

A **Cross-Sectional Study** is often likened to a snap shot of the population, in that the presence of some particular outcome is measured along with known and suspected risk factors to see if the risk factors are found more or less often in the subjects with the outcome than in the general population. If a suspected risk factor is seen more often in the subjects exhibiting the outcome condition than in subjects without the outcome condition then the proposed risk factor and the outcome condition may be said to be “correlated”. Here correlation is used to indicate that some systematic relationship may exist, rather than the more limited mathematical definition of correlation that indicates the presence of a linear relationship between the two variables. Correlation tends to support the hypothesis of a relationship between a risk factor and a given outcome whereas the absence of a correlation would tend to refute the hypothesis. Although correlation may provide supporting evidence of causation it is not itself a sufficient proof of such. With cross-sectional studies we are not able to determine causality, but only state whether a condition and hypothetical cause are seen together. Cross-sectional studies have the advantage of usually being less costly because the population is only measured once and does not require follow-up, thus, there is a much smaller time investment and difficulties following subjects over time are avoided. Additionally, a larger study population is more manageable at less cost in this type of study design so there may be more opportunity to reach statistical significance for smaller effects.

A **Longitudinal Study**, also called a **Cohort Study**, involves repeated measurement of a variable in a chosen (often initially disease free) population in order to measure how many of the people in the population, get the disease with respect to some variable of interest. If the subjects are identified at the beginning of the experiment and followed forward, the study is called a **Prospective Study**, whereas if the study is constructed after the fact, perhaps by using medical records, then it is referred to as a **Retrospective**, or “backward-looking” study. At a generally higher cost and greater difficulty (due to the repeated measures, difficulties of maintaining contact with the test population and longer time commitment), in a longitudinal study it is possible to make a stronger case for causation because it is possible to show whether a proposed factor causes the development of the disease. Thus, longitudinal studies are an important method to identify risk factors in epidemiological studies and prospective epidemiologic studies are preferred for assessing causation..

A **Case-Control Study** involves matching people diagnosed with the disease with similar people from the same population (from which the diseased people were chosen) in order to examine what factors may have influenced genesis of the disease. Factors found to be more prevalent in the case sample would then be seen as potential causative or associated factors.

In a **Laboratory Study**, some variable or set of variables are strictly controlled for, and the responses of individuals, or groups of individuals, to some condition or stimuli are measured. Examples of this type of study, referred to in this report, are the experimental sleep disturbance studies in which some psychological or physiological variable is measured after sleep loss or fragmentation has been induced. These have the advantage of a high level of control, but the disadvantage of short time periods, high costs, and occasional difficulties differentiating between effects due to the experimental variable under investigation and the effects resulting purely from subjects’ reaction to the laboratory experience itself. Longer-term laboratory studies are difficult to conduct and raise concerns about the ethics of exposing subjects to noise that disturbs sleep over a long period of time, a circumstance potentially harmful to their health.\*

## 2.2 Noise Measurements

Basner (2008) found that integrated A-weighted energy-based metrics were not the most accurate predictors of sleepiness resulting from a night of aircraft noise. This result is not surprising, in that with these measurements many very quiet events unlikely to disturb sleep are evaluated as being equal to very few, very loud events where the likelihood of awakening is very high. For sleep disturbance, noise inside the bedroom is clearly important, so outdoor noise measures or predictions, even if single event-focused, may not be reflective of community sleep disturbance. Window opening, bedroom orientation to noise sources, wall and window construction will modify the characteristics of the sounds, so there is the potential of having a large variation in responses for the same outdoor noise measurement, even if (as we know is not the case) everyone responded in exactly the same way to the same sounds (Flindell, Bullmore, Robertson, Wright, Turner, Birch, Jiggins, Berry, Davidson, Dix, 2000). There is also evidence that people respond differently to noise events that rise to the maximum level more quickly than events that reach the maximum level more slowly. In extreme cases (e.g., sonic booms) the sounds will evoke a startle response (Thackray, 1972). Even at slower rates of increase, such as with aircraft landings and take-offs near airports, Brink, Wirth and Schierz (2006) found differences in people's responses even though the Lmax values were very similar. Sounds that fluctuate, e.g., alarms, tend to continually demand attention, and sounds that have meaning are both difficult to ignore, for example, speech is also difficult to tune out, not only because it fluctuates in level but also because it contains information. Similarly, people find it difficult to tune out the sound of a baby crying at night.

### 2.2.1 Types of Noise Metrics Used in Noise and Health Studies

Several classes of noise metrics exist for quantifying noise exposure. However, only a limited set of these are used in noise health studies and most are based on average energy of the sound that the person is being exposed to. In sleep studies, researchers also use Maximum A-weighted level or A-weighted Sound Exposure Level. In many studies, the metrics used are time-averaged frequency-weighted sound pressure levels where the averaging time may vary from fractions of seconds (as is the case when assessing maximum levels) to a year. The shorter averaging is used in metrics that quantify the impact of single events, where the energy in the time period immediately adjacent the peak level is thought to be important. The most popular weighting is A-weighting which is derived from an equal loudness contour (tones at different frequencies deemed equally loud to a 40 dB tone at 1000 Hz.). This weighting attenuates low frequencies and very high frequencies to better match the parameters of human hearing for low-level sounds (~40 phon). At higher noise levels, C-weighting should be used because this weighting has less attenuation at low frequencies similar to the behavior of the auditory system at higher sound levels (Beranek and Ver, 2005). It is also used frequently in some form for assessing blast noise, in which case it may be useful accounting for the contribution of rattling and shaking of the house (CHPPM, 2001). It should also be noted that these metrics could relate to indoor or outdoor measurements, though that is not always explicitly stated, leading to some ambiguity. Building facades tend to attenuate high frequency components of sounds more than low frequencies. Thus, outdoor and indoor levels may lead to differing noise exposures. Measurement of flyover noise is additionally complicated by the variety of levels, both high and low, which should necessitate the use of a weighting that varies dynamically with level as in the case of loudness metrics.

### 2.2.2 Types of Noise Metrics

The average (weighted) sound pressure level  $L_{eq}$  is given by:

$$L_{eq}(t_1, t_2) = 10 \cdot \log_{10} \left( \frac{1}{t_2 - t_1} \int_{t_1}^{t_2} p^2(t) / p_0^2 dt \right) \quad (1)$$

where  $p_0^2$  is the reference pressure and  $p$  is the (often) A-weighted (the weighting is applied using a filter) acoustic pressure and  $t_1$  and  $t_2$  are the beginning and ending measurement times. While the simplicity of the single number resulting from this is attractive, the averaging removes detailed temporal information so that significantly different exposure profiles may still give the same result. Sometimes the type of weighting is

explicitly contained in the variable, e.g., average A-weighted sound pressure level:  $L_{Aeq}$ ;  $L_{night}$ , sometimes more explicitly written as  $L_{22-7}$  which is always A-weighted; and  $L_{day}$ , sometimes written as  $L_{16hr}$  or more explicitly as  $L_{7-22}$ , which again is always A-weighted. The ANSI standard which explains proper use of acoustic terminology (ANSI, 1994) indicates that the correct symbol for A-weighted time-averaged equivalent continuous sound level is  $L_{Aeq}$  or  $L_{AeqT}$ , where T is the time interval measured. However, in some literature the A is assumed and suppressed and also the eq. For most community noise studies, A-weighting is assumed.

Day-night average sound level ( $L_{dn}$  or *DNL*) is, similar to  $L_{Aeq}$ , in that it is an average energy based metric. It differs from  $L_{Aeq}$  in the addition of a 10-dB penalty on sounds occurring during the nighttime. This is added to account for sensitivity to noise associated with these hours (for example, people may attempt to sleep and thus desire a quieter condition) and also because background levels at night are typically lower and thus aircraft may be more noticeable. The exact hours defined as nighttime vary somewhat between countries and may run between 2300 and 0700 or 2200 and 0600 (Jarup et al, 2007). In some countries a smaller penalty (e.g., 5dB) is also applied during the evening hours; these metrics are denoted by  $L_{den}$  though the timing of the evening period may vary from country to country.  $L_{max}$  (or  $L_{Amax}$ ) is the maximum (A-weighted) sound level experienced in a given period of time, measured on a fast setting on a sound level meter, and is often used in sleep disturbance studies to quantify the impact of a single event on sleep. Sound exposure level (SEL) is also often used to quantify single noise events. It is the total (weighted) energy of the noise within some time interval of the peak reading or over a defined event (ANSI, 1994). This event definition is sometimes given as all energy exposure while the waveform is within e.g. 10 dB of the peak level. Again, it is typically based on A-weighted sound pressure levels. A-weighted SEL and  $L_{Amax}$  are typically indoor levels in sleep disturbance studies.

There are many other measures of noise. In recent years, models of loudness perception through time (Zwicker and Fastl, 1998 & Glasberg and Moore, 2002) have been used to quantify human response to sound, particularly in product design. These models of loudness capture more elements of human auditory processing than simple A- or C-weightings. However, their use in environmental noise, research, particularly in sleep disturbance and physiological health studies, has been very limited and therefore, they are not described here. This does not indicate that it is not important to include their use in future studies, as more accurate models may aid in our understanding of the relationships between noise, annoyance, sleep disturbance and health outcomes.

### **2.2.3 Method of Measurement and Prediction of Noise Exposure**

In studies of the impacts of noise around airports, the noise is usually quantified through predictive models such as the Integrated Noise Model (INM) and the Noise Model Simulation (NMSIM) program recently released by Wyle Laboratories. Occasionally, studies will include actual measurements at a property façade although extensive measurements at many locations may greatly increase the cost, difficulty and time required. For studies where nighttime sleep disturbance is of high interest the gold standard for noise measurement is in-bedroom noise measurement. This is rarely performed, both because of the cost and intrusiveness and also because indoor sources of noise cannot be easily excluded without recording and identifying the source of each noise disturbance; a tedious, but necessary, procedure. For a complete understanding of the effects of aircraft noise exposure, it is necessary to quantify exposure to these other noise sources, so that health outcomes can be correctly assigned to particular sources.

The Integrated Noise Model (INM) (FAA, 2008) predicts aircraft noise, as quantified by predominantly A-weighted sound pressure level based metrics in the vicinity of airports. It was developed based on the algorithm and framework from the SAE AIR 1845 Standard, which uses Noise-Power-Distance (NPD) data to estimate noise. It accounts for the specific operating mode, thrust setting, source-receiver geometry, acoustic directivity and other environmental factors. The Integrated Noise Model has been a major workhorse in the aircraft noise prediction field since before the advent of the PC and is the program most commonly used for environmental impact analysis around airports, especially in the U.S..

The most recent version of the Noise Modeling Simulator (NMSIM, 2009) from Wyle Laboratories is capable of modeling both air and ground noise sources including trains, airplanes and automobiles. It has advantages over other popular methods of simulating aircraft noise in its ability to account for terrain shielding effects and its computation of a complete spectral time history at any measurement point. It is able to assess the effects on an observer of multiple sources of exposure, account for various types of terrain and atmospheric patterns and create animations of noise levels resulting from aircraft flyovers.

Measurement at the residence initially seems fairly self-explanatory but entails some unexpected complications. Measurement has advantages over prediction in terms of precision of noise levels, accounting for any unexpected variables or unpredicted exposures such as contributions from neighbors. However, these advantages come with the disadvantage that while very specifically accurate, a noise measurement over a short period of time may not be representative of the exposure of a location over a longer period of time. Thus, a disconnect may occur between specific and characteristic accuracy. Also, it is difficult to isolate exposures of interest such as aircraft from other exposures such as neighbor noise, so in the same process in which measurement increases accuracy it may also limit specificity. The same may be said of indoor measurements where the noise recorded may be produced by the sleeper himself or by a partner as in loud snoring. These drawbacks may be mitigated in the case of studies of short-term effects by recording and identifying noise sources allowing both high accuracy and specificity but this practice is currently prohibitively expensive for use in longer-term studies as well as potentially invasive. Future developments in technology may eventually allow for cost-effective long term monitoring and evaluation of noise sources at or in a residence. Such a tool would allow an unprecedented level of exposure assessment and would significantly aid researchers in answering many of the lingering questions about health effects of noise.

#### **2.2.4 Sources of Error in Noise Studies**

Several possible sources of error must be accounted for in studies addressing potential health effects of noise—noise mis-measurement or mis-estimation, inadequate representation of multiple sources of individual exposure, differences in outdoor versus indoor exposure, coping methods that change noise exposure such as, e.g., closing windows, and duration of time in a residence. Determining an individual's relevant noise exposure is a task complicated by remaining ambiguities concerning the relative importance of waking vs. sleeping, and leisure vs. work vs. travel exposure. Also, in most instances detailed information about a subject's daily exposure is not available to researchers. Thus, many studies have been focused on either the working or the home environment, though exposure in other environments has occasionally been viewed as a confounder (Chang, Jain, Wang, Chan, 2003; Babisch, 2006). Levels in the sleeping chamber may be affected by how a house is oriented relative to noise sources such as roads. Also, individual habits concerning window opening may be an important modifier of exposure to noise as they may change the amount of sound transmitted from outside to inside. Housing construction techniques will also play a significant role in determining outdoor-indoor sound transmission. Indoor measurements of exposure are desirable where possible. However, simply measuring average or maximum indoor measurements may result in noise measurements that are not very closely related to the noise caused by the source of interest because other indoor noise sources may be present and dominating the measurements. So, both outdoor and indoor measures may be desirable in order to limit misattribution of noise exposure from indoor sources to outdoor ones. Most current theories dealing with health effects of noise assume that an induction effect is involved, wherein subjects must be exposed to noise over a prolonged period of time before noticeable health effects may begin to emerge. Thus, many noise studies have a requirement of a certain amount of time in residence in order to be considered for participation in the study (Babisch, 2006, Jarup et al, 2007). Accurate assessments of noise exposure over longer time periods is challenging unless details of airport operations, community developments, house and house use changes and other noise modifying or noise generating sources are available.

## 2.3 Other Measures of Interest

### 2.3.1 Measures of Sleep

Investigators have used many different methods of measuring sleep disturbance and sleep in general.

On the most precise end, sleep scientists have used the combination of electroencephalogram (monitors brainwave patterns), electrocardiogram (monitors heart rate), electromyogram (monitors muscle activity), and electro-oculogram (measures eye movements) to determine the presence of clinically defined sleep, as well as the stage of sleep a person is experiencing; monitoring these variables simultaneously is known as **polysomnography**. Polysomnography lets researchers know how much time is spent in each sleep stage and awake as well as when important changes occur in sleep structure, such as the transition from one stage to another. While the detailed structural information about sleep of polysomnography has many advantages, it requires monitoring and scoring of sleep stages by technicians, may be costly to obtain and is not available in all studies for cost or other reasons. In the future, automated scoring may allow it to be more widely and economically used.

Other methods of measuring sleep may lack physiologically detailed information on sleep stage architecture, but still give an accurate idea of some types of arousal or awakening. These include **wrist actigraphy**, which involves the placing of a bracelet with accelerometers or similar sensors on the wrist of a person being monitored to determine their motility (whether they are active or dormant). Other methods have included the placing of **accelerometers** beneath the posts of the bed to monitor the movements of those sleeping. This method is able to detect heart rate and breathing via recoil effects as well as detect when the subject is moving (Brink, Muller, Schierz, 2006). In studies by Fidell (1995), "behavioral awakening" was measured in subjects via their **pushing a button** when they recognized that they had awakened.

Some studies such as that of Ohrstrom (1995) measure awakenings using a **survey the morning after** exposure to noise events. This method may be vulnerable to sleep state misperception (where a sleeping person thinks they are awake when they in fact are sleeping as clinically measured) leading to extra awakenings being reported or on the other hand unremembered awakenings being excluded.

**Cardiac arousals**, which may occur both with and without resulting changes in sleep stage or awakening in response to acoustic stimuli, have also been studied as a potential avenue for the development of road-noise induced cardiovascular disease (Griefahn, Brode, Marks, Basner, 2008). They can be observed using an ECG.

The data that is gained from polysomnography may be used to describe the average fragmentation of sleep over a night using variables such as the **sleep disturbance index (SDI)**. This is calculated by dividing the total number of arousals and awakenings in a night by the total length of sleep, thus it gives the average number of disturbances per hour. **Sleep fragmentation index (SFI)** is similarly defined as the number of awakenings and shifts to stage one sleep in a night (Morrell, Finn, Kim, Peppard, Badr, Young, 2000).

For studies involving sleep apnea a similar measure exist called the **apnea-hypopnea index (AHI)** which is the number of apneas (a respiratory event involving total stoppages of airflow) and hypopneas (a type of respiratory event involving large reduction in airflow volume) per hour, or similarly the **apnea index** may be calculated using just the number of apneas divided by the total length of sleep time. These measures are useful in giving a single-number description of the sleep or respiratory disturbance experienced by an individual.

Nocturnal decrease in blood pressure (a normal and healthy phenomenon) may be monitored via **ambulatory blood pressure monitoring (or ABPM)**. In this method, blood pressure is repeatedly sampled at a desired interval of time and the values recorded in order to determine an individual subject's 24-hr blood pressure profile and determine if they are experiencing the normal dip in blood pressure during sleep. It is also useful for noting short-term increases in blood pressure which may be associated with recent noise exposure. The measurement may be done via an automated inflatable cuff system, and allows recordings while sleeping or while performing daytime tasks, although some tasks such as heavy lifting may interfere

with measurements (Chang, Jain, Wang, Chan, 2003; Haralabidis, Dimakopoulou, Vigna-Taglianti, Giampaolo, Borgini, Dudley, Pershagen, Bluhm, Houthuijs, Babisch, Velonakis, Katsouyanni, and Jarup et al, 2008).

### **2.3.2 Tiredness, Sleepiness and Sleep Quality**

Another important set of measures are those used to determine **sleepiness, tiredness or sleep quality**.

Sleepiness has been determined subjectively through **surveys** including questionnaire items that addressed changes in mood and feelings of tiredness; one important example of this is the **Stanford Sleepiness Scale** in which a subject is asked to rate their subjective sleepiness on a scale ranging from “Feeling active, vital, alert, or wide awake” at the most alert, to “No longer fighting sleep, sleep onset soon; having dream-like thoughts” at the sleepest (Dement, 2009). Another similar measure is the **Epworth Sleepiness Scale (ESS)**. In this instrument the subject is asked to rate their likelihood of falling asleep in a number of contexts involving various degrees of mental stimulation. It has been found to be correlated with Multiple Sleep Latency Test scores (see below) (Johns, 1991). Another instrument is the Pittsburgh Sleep Quality Index (PSQI) developed by Buysse and his colleagues at the University of Pittsburgh. (Buysse et al., 2008). It uses a set of 19 self-rated questions and 5 questions rated by a bed fellow in order to measure sleep quality.

Performance measures examine how performance in some task may be diminished when sleep is shortened or otherwise disturbed. These may be useful in predicting the magnitude of any decrease in task performance at work or increase in accidents due to fatigue. Performance on various types of **psychomotor tasks**, e.g., a subject’s reaction time in performing a vigilance task, is typically assessed.

The “gold standard” for sleepiness as it is often called, is the **multiple sleep latency test**, or **MSLT**. In this test the time required for a subject to fall asleep is measured while they are monitored using polysomnography. This length of time to fall asleep is the measure and this tends to decrease as subject sleep debt increases. The test is repeated several times to account for differences in circadian alerting at different times of the day.

A second test of objective sleepiness, found to be highly correlated with the results of the multiple sleep latency test is the **pupillographic sleepiness test** or **PST** (Basner, 2008; Danker-Hopfe, Kraemer, Dorn, Schmidt, Ehlert, Herrmann, 2001). In this test the subject is asked to look at an light emitting diode (LED) while the motion of the subject’s pupil is measured using an infrared light in visual darkness. The amount of motility (motion) of the pupil in dilating and contracting is used to determine the relative activity in the sympathetic and parasympathetic nervous system, which control these actions. This test, like the MSLT, is sensitive to physiological variables and may not always accurately reflect subjective tiredness, which seems to follow a different schedule of diurnal variation than the objective measures (Danker-Hopfe, Kraemer, Dorn, Schmidt, Ehlert, Herrmann, 2001).

### **2.3.3 Performance and Memory Tasks and Learning**

Effects of long-term exposure to noise on learning may be measured through the use of standardized reading tests, see, for example, Stansfeld et al. (2005),. This allows researchers to determine if repeated exposure to noise, which may cause a mixture of increased stress or potentially disrupted learning, has in fact had any significant impact in terms of children’s actual progress in reading.

Other tasks might be used to measure memory of information (for example, a story) initially presented in the presence of noise. Students might then be asked to recall certain aspects of the story or asked whether certain events occurred. This would allow researchers to determine students’ ability to recall information presented in noise at a later time and recognize information initially presented.

Effects of noise on performance may be measured by examining performance on tasks in the absence and presence of noise. For instance, noise may increase arousal which may lead to increased performance on simpler tasks, or may increase performance of people who are tired or fatigued. However, on more complicated tasks arousal may diminish performance by leading subjects to overly focus on a few facts or ideas to the exclusion of other important information (Proctor and Zandt 1994).

Performance may also be affected by noise via communication disruption (Proctor and Zandt, 1994). Speech interference may be measured via the Articulation Index (ANSI, 1967) or its revision the Speech Intelligibility Index (ANSI, 1997). In this procedure the intensity levels of noise and speech are measured in either 20 or 15 divisions of frequency regions. Average differences between the speech and noise levels in the bands are weighted and normalized giving values between 0 and 1, 1 signifying perfect intelligibility. Hearing thresholds can also be input into the SII calculation. Simpler speech interference measures, often used in engineering applications, are the Speech Interference Level (SIL) or the Preferred Speech Interference Level (PSIL) which are averages of the 500, 1000, 2000 and 4000 Hz octave-band levels (in dB), which is the region where most speech energy occurs (ANSI, 2006).

#### **2.3.4 Blood Pressure Measures**

Blood pressure measurements have frequently been used in various studies conducted in laboratory settings. In many of these studies, blood pressure is measured only once. Blood pressure has also been measured at multiple times throughout the day in both the work and home environments using ambulatory blood pressure monitoring methods. Laboratory or doctor's office measurements of blood pressure often involve a mercury sphygmomanometer (blood pressure meter). This instrument includes a cuff which restricts blood flow and a pressure meter that measures the amount of pressure in the cuff and thus being applied to the artery. The cuff is typically placed on the arm at the same elevation as the heart and a stethoscope is used to listen to the artery 'downstream' of where the cuff is applied to see if blood flow has effectively ceased. This is done by listening to the turbulent noise resulting from the restricted flow. The turbulent noise begins to be audible when the artery begins to flow while partially constricted and ends when the flow is blocked completely or when the constriction is removed and normal flow restored. Measurement performed in the clinic or doctor's office may elicit an artificially increased blood pressure possibly resulting from stress associated with a doctor's visit. This effect is known as "white coat hypertension" and may be alleviated by using ambulatory blood pressure.

In ambulatory blood pressure measurement (ABP or ABPM), a monitoring device is worn by the subject which measures blood pressure at regular time intervals. Due to the absence of the potentially stressful laboratory or doctor's office measurement context, the likelihood of a "white coat" effect is diminished and measurements more characteristic of the subject's typical blood pressure may be obtained. Also this method allows for the monitoring of blood pressure effects that may be of short duration (e.g. increases elicited by noise exposure) or that occur over an extended time period or as part of a circadian pattern (such as the nocturnal decline in blood pressure). The degree of nocturnal decline in blood pressure has some importance as a predictor of cardio- and cerebro-vascular outcomes making the 24-hour monitoring ability of ambulatory blood pressure an especially valuable tool.

#### **2.3.5 Glucose Management/Diabetes Measures**

Glucose management is often measured by using some sort of glucose tolerance test. This involves a subject being given a quantity of glucose appropriate for their body mass (often taken orally) and taking samples of blood to find out how effective their body has been at reducing the surplus blood glucose levels down to normal levels through the action of insulin. Other measures may focus on reaction to insulin. One important example in this category is the hyperinsulinemic euglycemic clamp. In this test insulin is administered intravenously over a period of time. Intravenous glucose is also administered in an attempt to keep blood glucose levels within a normal range. The amount of glucose that must be given to compensate for the body's reaction to the additional insulin is used to determine the subject's sensitivity to insulin. Other methods of examining glycemic control may look at the presence of other markers which co-vary with glucose levels such as hemoglobin A1c levels (HbA<sub>1c</sub>), which are considered to be a stable marker of glucose levels in the body. Thus, by looking at HbA<sub>1c</sub> levels researchers can determine typical values for blood glucose levels. See, for example, (Knutson, Ryden, Mander and Van Cauter, 2006) for more information.

### **2.3.6 Measures of Sympathetic Tone**

Sympathetic tone, the amount of action in the sympathetic nervous system, is measured in a variety of ways. It may be measured directly by using micro-neurography, where nervous system electrical activity is invasively monitored and recorded for analysis. Sympathetic tone may be measured indirectly via signal processing of the electrocardiogram waveform which it influences. The balance of high and low frequencies in the waveform and the inter-beat period variability give researchers clues about the balance of neural activity in the sympathetic nervous system vs. the vagus nerve. This is called sympathovagal balance. Multiple references on factors affecting sympathetic tone are included in Section 3.3.1 of this report.

### **2.3.7 Indicators of Heart Disease**

The existence of heart disease has been determined in studies by using medical record searches, examination of death certificates, or interviews where a doctor had previously diagnosed myocardial infarction. Emergency admittance for a heart attack, changes in electrocardiogram (ECG) activity indicative of heart disease after reported chest pain, or other doctor diagnoses were all criteria considered in various studies. A review of methods used in various noise health studies is given in (Babisch, 2006a).

### **2.3.8 Noise Sensitivity**

Noise sensitivity has often been measured in health studies via a set of survey questions inquiring about a subject's feelings toward or reactions to noise, see, for example, the Noise Sensitivity Questionnaire (NoiSeQ) (Schutte, Sandrock, Griefahn, 2007) or Weinstein Noise Sensitivity Questionnaire (Kishikawa, Matsui, Uchiyama, Miyakawa, Hiramatsu, Stansfeld, 2006). Questions remain concerning possible relationships between sensitivity to awakenings while asleep and noise sensitivity while awake and results from the DLR (German Aerospace Center) suggest that these two variables may not be interchangeable (Basner, Samel, Isermann, 2006).

## **2.4 Some Concluding Comments**

From the above, it is clear that taking a comprehensive set of noise, physiological and psychological measurements to study the health impacts of noise on populations is challenging. Simpler or averaged measurements may fail to capture the information that is required to fully understand the cause and the biological mechanisms leading to health effects. More complicated measurements may lead to an overwhelming amount of data to analyze and are typically quite expensive to obtain. The measurement techniques and the environments in which they are made can both affect research outcomes. Another issue is the measurement (or prediction) of total noise exposure (and other exposures that lead to the same health outcomes) over long periods of time, so that health outcomes can be correctly attributed to a particular or multiple sources.

### **3 NOISE, AROUSAL AND SLEEP DISTURBANCE**

Normal sleep provides mental and physical renewal. The disruption of sleep may result in feelings of frustration as well as provide a path for more serious physical consequences. Noise from aircraft may disrupt sleep leading to delays in the initiation of sleep, awakenings during the night, or premature final awakening. Noise may also lead to disruptions in the structure of sleep leading to a composition increasingly dominated by lighter sleep stages. Maintaining sleep in the presence of intrusive noises may also exact a biological cost in maintaining sleep. Acute reactions to noise while sleeping may include short-term increases in blood pressure and heart rate. There is some concern about the possible effect of nighttime noise on the nocturnal dip in blood pressure, and indeed noise has been shown to affect factors which seem to influence dipping likelihood. Decreases in dip amplitude may have prognostic significance for future heart disease and stroke. Cardiovascular arousals, similar to the type described for comparatively small amplitudes of noise during sleep, have been observed in workers exposed to much greater amplitude sounds associated with their work in industrial settings. Sound exposure during both waking and sleeping may result in acute cardiovascular arousal and it has been hypothesized that this can contribute to longer term impacts. Noise during sleep, especially if accompanied by remembered awakening, may also result in annoyance.

#### **3.1 Sleep Disruption: Fragmentation/Reduction/Structure Changes and Tiredness**

Sleep is a state of diminished activity that plays an important role in the maintenance of health. According to the WHO Guidelines for Community Noise, “Uninterrupted sleep is known to be a prerequisite for good physiological and mental functioning of healthy persons (Hobson, 1989); sleep disturbance, on the other hand, is considered to be a major environmental noise effect.” Noise may potentially disrupt normal sleep by either disrupting its continuity, changing its structure (prevalence of various sleep stages) or reducing the absolute amount of time spent asleep or otherwise diminishing its restorative value by creating an excited condition without visible change in sleep structure (such as in an autonomic arousal, a transient increase in sympathetic tone—the activity level in the sympathetic nervous system (Griefahn, Brode, Marks, Basner, 2008) or by producing stress in an organism as it expends additional resources to maintain the sleep condition (Basner, 2008). To understand the potential health effects when sleep is disrupted, it is important to first understand some of the functions sleep appears to serve, what types of sleep disruption aircraft may produce, and how they are measured. The further question arises: what groups may be most susceptible to the proposed effects that might occur with increased noise exposure. The answers to these questions will then help inform later parts of the discussion where health effects beyond the more immediate potential outcomes of tiredness are discussed as potential outcomes of sleep disruption.

##### **3.1.1 Sleep disruption through sleep disorders as an introductory and limiting case**

Noise is known to disrupt sleep see, for example, (Basner, 2008; Basner et al., 2008), and sleep disruption may have negative consequence on health, see, for example, (Wu, Wang, Koh, Stanczyk, Lee, Yu, 2008; Gangwisch, Malaspina, Boden-Albala, Heymsfield, 2005; Spiegel, Leproult, Van Cauter, 1999; Tasali, Leproult, Ehrmann, Van Cauter, 2008; Knutson, Ryden, Mander and Van Cauter, 2006; Gottlieb, Redline, Nieto, Baldwin, Newman, Resnick, Punjabi, 2006). Research focusing on the adverse consequences of sleep disordered breathing (obstructive sleep apnea particularly, in which an individual’s breathing is periodically prevented by the closure of their airway, generating arousal and disrupting sleep) has been found to be associated with increased risks of hypertension, heart disease, diabetes, and other problems (Shaw, Punjabi, Wilding, Alberti, Zimet, 2008; Resnick, Redline, Shahar, Gilpin, Newman, Walter, Ewy, Howard, Punjabi, 2003). However, apnea carries with it the additional risk factor of exposure to hypoxemia, a state in which the blood oxygen content decreases to levels that may lead to other effects not related purely to arousal. Thus, while sleep apnea produces severe sleep disruption and may give us a limiting case in considering what potential health effects to look for via a sleep disturbance pathway, its usefulness as a model purely of sleep disruption is limited by the additional hypoxic (deprived of adequate oxygen) effects. However, similar patterns of associated illnesses such as hypertension (Bixler, Vgontzas, Lin, Ten Have, Leiby, Vela-Bueno, Kales, 2000) and diabetes (Phillips, Young, Finn, Asher, Hening, Purvis, 2000) are also

seen in other sleep disorders such as restless leg syndrome (in which individuals move limbs during sleep or feel irritation in their limbs that generates arousal), and also in those who experience short sleep and disrupted sleep in absence of a particular sleep disorder (Gottlieb, Redline, Nieto, Baldwin, Newman, Resnick, Punjabi, 2006).

### **3.1.2 Some brief background information on sleep**

In considering the types of disruption of sleep that may occur as a result of (presumably) nighttime aircraft noise, it is useful to have a working knowledge of some of the basic physiological phenomenon associated with sleep. Sleep is often classified into six stages: Awake, Stage-1 non-REM, Stage-2 non-REM, Stage-3 non-REM, Stage-4 non-REM, and REM.

REM stands for rapid eye movement and refers to the stage of sleep in which dreaming most often occurs. The name refers to the rapid movements of the eyes observed in this stage. During REM sleep the muscles of most individuals are effectively paralyzed and this helps prevent injuries to self and others while dreaming.

In Stage-1 of non-REM sleep, individual's brain activity transitions from the alpha waves (characteristic of a relaxed awake person with eyes closed) to theta waves (characteristic of sleep or certain types of memory tasks as well as spatial navigation). Because of the transitional character of this stage it is not always viewed as being strictly part of sleep. For example, in his Markov state transition sleep stage model Basner (2006b) considers the beginning of sleep from the initiation of Stage-2 non-REM. In some sleep metrics, for example, the sleep fragmentation index, Stage-1 non-REM sleep and Awake stages are combined into the one category.

Stage-2 non-REM is recognized by the presence of sleep spindles and K-complexes (particular EEG patterns) and unconsciousness is considered to have occurred by this point.

Stages-3&4 of non-REM sleep are collectively termed "deep sleep" and are marked by the presence of delta waves (a particular EEG patterns). The main difference between Stage-3 and Stage-4 is the prevalence of the delta waves in the EEG signals. For this reason, some models have opted to consider them together as a single parameter.

The remaining stage—waking—is sometimes considered as a condition of increased activity when individuals experience increased energy expenditure, seek food, and seek to reproduce.

Most individuals will experience greater concentrations of slow-wave sleep (non-REM stages 3&4) towards the beginning of the sleep period and greater concentrations of REM sleep towards the end. During slow-wave sleep, individuals are at their most difficult to awaken. This is often interpreted as a physiological protection of slow-wave sleep indicating that it is the most biologically necessary stage of sleep. As a further support of this, individuals who have undergone sleep deprivation often experience a "rebound" of slow-wave sleep; their sleep on the first night after sleep deprivation is dominated by the greater-than-usual prevalence of slow-wave sleep and a reduction of other stages. In later nights there is a rebound of REM sleep, before returning to the typical distribution of sleep stages. Also, if either slow-wave sleep or REM is selectively prevented then the body seems to preferentially seek to spend time in the missing stage. For example, if someone is prevented from entering REM for a time, when sleep is no longer restricted, REM sleep will occupy a larger than normal portion of their total sleep period, and this will occur for a length of time afterwards (after the REM deprivation).

Certain important physiological processes occur during sleep including the release of human growth hormone, changes in glucose levels and regulation. Also levels of activity in the sympathetic nervous system (associated with the arousal condition) generally decline in uninterrupted sleep (Tasali, Leproult, Ehrmann and Van Cauter, 2008). Normal length of sleep also appears to be important for the maintenance of proper levels of leptin and ghrelin, hunger regulating hormones (Gangwisch, Malaspina, Boden-Albala, Heymsfield, 2005). Another potentially important effect is the decrease in blood pressure typically seen in normal sleep. Blood pressure typically dips to its lowest daily point during sleep, and researchers in some studies have

shown that the size of the dip or the minimum reached is a potentially relevant predictor of cardiac health (Stolarz, Staessen, O'Brien, 2002), or cerebrovascular (O'Brien, Sheridan, O'Malley, 1988). A diminished dip is seen as a troubling indicator for both cardiovascular and cerebrovascular outcomes, while a blood pressure dip fulfilling expectations (of between 10 and 20 percent nocturnal reduction) is considered a positive indication. Dipping appears to be especially linked to having sufficient amounts of deep sleep, particularly Stage-4 non-REM sleep (Loredo, Nelesen, Ancoli-Israel, Dimsdale, 2004).

### **3.1.3 Types of sleep disruption that may occur with aircraft noise**

Disruptions of sleep that can result from noise may be considered in several main groups: disruption which makes falling asleep initially difficult (delayed onset of sleep); awakening or arousal from a deeper stage of sleep (these may result in decreased sleep continuity); or difficulty reinitiating sleep after an awakening (for instance a person is awakened by noise shortly before their normal time of arising and cannot return to sleep until the subsequent night). Additionally, arousal of the cardiovascular system may occur with or without a shift in stage although cardiovascular arousals are typically less severe if no shift in stage occurs (Griefahn, Brode, Marks, Basner, 2008).

Ohrstrom (1995) discusses the first group of sleep disruption mentioned: **difficulty initiating sleep**, as well as the second. In those exposed to 16, 32, 64, and 128 nighttime noise events at 45, 50, and 60 dBA he finds a significantly increased time required to initiate sleep beginning at 64 noise events per night—increases in the time required to get to sleep at this number of interruptions were on the order of 14-22 minutes, although Flindell, Bullmore, Robertson, Wright, Turner, Birch, Jiggins, Berry, Davidson and Dix (2000) found neither delays in sleep onset nor premature awakenings in the noise exposed condition of their aircraft noise exposure study. Also interestingly, Ohrstrom observed no significant effect with level of the noise stimuli on sleep initiation, and similar effects on sleep initiation were noted for 45, 50 and 60 dBA. His conclusions are that when trying to get to sleep, events in this range of noise levels appear to be roughly equal in their disruptive effect on sleep (Ohrstrom, 1995).

With regards to **awakenings**, however, Ohrstrom found that level and the frequency with which (how often) the stimuli occurred both played a significant role. The awakenings in this study were recorded via questionnaire in the morning, a measurement that is susceptible to errors due to sleep-state misperception (where subjects believe themselves to be awake when they are not) and unremembered awakenings.

Not all disruptions of sleep involve awakenings—remembered or otherwise. Disruptive events during sleep may lead to **changes in the prevalence of the various sleep stages** that may then lead to changes in physiological variables without awakening of the subjects (Tasali, Leproult, Ehrmann, Van Cauter, 2008). Sleep disrupted by acoustic stimuli may not contain as much of the deeper slow-wave stages (Philip, Stoohs, Guilleminault, 1994), which are seen as being most physiologically restorative; also REM may be decreased. Instead of these, increased prevalence of lighter non-REM stages is seen.

Also, in order to have restorative value, as measured by multiple sleep latency test (MSLT) scores, periods of sleep need to extend beyond a certain minimum length. This brings us to the problem of **fragmentation**. Sleep disrupted or fragmented by noise is associated with increased sympathetic nervous system arousal (Griefahn, Brode, Marks, Basner, 2008). Fragmented sleep may also be predictive of a nondipping cardiovascular profile (Matthews, Kamarck, Hall, Strollo, Owens, Buysse, Lee, Reis, 2008). Total sleep time is also important in order for sleep's restorative function to be completely fulfilled. The exact length needed individually may vary. However, habitually **shortened sleep** correlates with changes in glucose management as well as leptin and ghrelin regulation and blood pressure increases in population-based studies, as well as the more obvious resulting tiredness. Fragmented sleep has been found to result in many of the same consequences (Bonnet, Arand, 2003).

It appears that awakenings, more than sleep-state changes or other types of arousal effects, are generally what is focused on in policy decisions, perhaps because remembered awakenings attributed to noise are more likely to generate annoyance. According to Basner, Samel and Isermann (2006) awakenings induced by noise tend to last longer depending on the noise level that elicited them. Spontaneous awakenings are typically brief as are those caused by sounds with a maximum level of 65 dBA or less (measured outside of

the dwelling) and both result in a resumption of sleep within, generally, 1.5 minutes. Those awakenings elicited by maximum levels of 70 dBA or higher (outdoors) were associated with longer awakenings. This question of how much sleep is effectively lost throughout a night of noise may be important in gauging the magnitude of effect that may be expected via sleep loss.

One interesting idea in tracing the potential pathway from sleep disruption by noise to health is to investigate the **increased sleepiness** due to disruption by noise. Shortened sleep duration has been shown to be related to important health outcomes and objective sleepiness may be an effective measure of short sleep and its health consequences, although this remains to be studied. As mentioned earlier in Chapter 2, sleepiness has been measured by several methods historically, including through use of a questionnaire, by using multiple sleep latency testing (MSLT), or also, as in a recent study by Basner (2008), by using the pupillographic sleepiness test (PST).

Basner's 2008 study, where he compared sleepiness of normal subjects, with that of untreated sleep apnea patients, and that of the normal subjects after being subjected to noise during sleep, may be of significant interest because it is an approach that might help fill an important gap in our knowledge of potential health effect magnitudes. By comparing the magnitude of the objectively measured sleepiness in the normal patients (using PST) due to sleep disrupted by noise to that due to sleep loss in general as described in other studies, it may be possible to estimate the increased risk to health as a result of sleep disrupted by noise. It should be noted that one important effect of sleep apnea, in addition to sleep disruption, is oxygen restriction, and so health outcomes for apnea patients is more complicated than what might be expected from short or fragmented sleep alone. Basner found that those subjects experiencing noise-interrupted sleep had mean PST scores greater than the normal subjects who were not exposed to noise, but the mean scores for the noise-exposed group were much closer to those of the normal, not-noise-exposed subjects than to those of the apneic subjects. On this basis, they concluded that sleepiness due to the aircraft noise events may not necessarily reach "pathological" levels, i.e., the levels observed in apnea patients.

The questions that arise are: can PST be used as an indirect measure of sleep fragmentation or short sleep, and are the physiological consequences of sleepiness the same as the physiological consequences of short sleep? Basner found in this same study (2008) that changes in sleep stage structure were comparatively minor, raising the question of whether sleep stage changes are the main operative principle in health effects of sleep fragmentation or whether some other mechanism is at work. He comments that the additional resources required to maintain sleep under conditions of noise may in fact have a physiological cost (increased tiredness) as supported by the above study.

In other studies, however, seemingly contradictory results have been found. For example, in a 1999 UK study of aircraft noise exposure effects on EEG measured sleep (Flindell, Bullmore, Robertson, Wright, Turner, Birch, Jiggins, Berry Davidson, Dix, 2000) the researchers found no change in MSLT scores in the lab for those in the noise exposed condition. Subjects in the noise exposed condition also slept on average ten minutes longer. However, the sample size was not large in this study, at most 18 subjects depending on which portion of the study. Bonnet and Arand (2003b) comment on the results of their study where it was found that arousal did not apparently decrease the restorative effects of sleep measured using the multiple sleep latency test. In that study, however, the arousal was produced via chemical stimulants rather than noise which could lead to important differences in physiological reaction. Stepanski, Lamphere, Roehrs, Zorick and Roth (1987) also observed fragmentation of sleep that occurred without significant changes in sleep stage composition; this supports the possibility of increased sleepiness resulting from fragmentation itself, similar to the result of Basner (2008).

The clear result from Basner's study (2008) that nighttime aircraft noise may lead to increases in objectively measured sleepiness (PST), is an important result because subjective measures of sleepiness such as questionnaires are vulnerable to possible confounding by individual attitudes towards awakenings with noise and this measure avoids that complication. Perceived awakenings have been known to cause decreased perceived sleep quality and perceived sleep quantity (Basner, Samel, Isermann, 2006). One important limitation noted in the sleepiness study (Basner, 2008) was that it was conducted in the lab and not in the

field and other work conducted at the DLR (German Aerospace Center) showed important and systematic differences between reactions in the two environments (Basner et al., 2004).

#### **3.1.4 Susceptible Groups**

If changes in sleep are a significant avenue for the genesis of a proposed health effect, then an important question is: “who might be most likely to experience decreased quality, quantity, or restorative functioning of sleep as a result of aircraft noise during the night?” Based on a reanalysis of Fidell’s data, Anderson and Miller (2007) suggest the possible need to include a parametric individual sensitivity to awakening. Sensitivity to annoyance by noise while awake has been studied in multiple contexts (see section 5.1 on noise sensitivity for multiple examples), but examining individual differences in sensitivity to awakening as a result of noise seems to be a comparatively recent subject of investigation. If stimulated-awakening sensitivity does indeed have a strong trait-stable component (independent of tiredness, current stress levels and other temporary matters) then those with higher trait stimulated-awakening sensitivity would seem to be more likely to suffer health effects which might result from sleep loss or disruption. Expanding this idea, both sensitivities to arousal in individual sleep stages short of awakening and drive to obtain individual sleep stages may also vary inter-individually, and so individual differences in capacity to obtain necessary stages of sleep or ability to return to these stages after interruption may potentiate or impotentiate the related health effects. As an example of this phenomenon, one of the subjects in the study of Tasali, Leproult, Ehrmann and Van Cauter (2008), on selective slow-wave sleep deprivation by using acoustic stimuli, was found to require far more numerous stimuli in order to prevent entry into slow-wave sleep. Thus, individual tendency to obtain a particular stage of sleep may be of non-trivial importance in determining whether a given night of noise is likely to affect a particular person in ways potentially relevant to their health. Thus, individuals may vary in their susceptibility to the effects of nighttime aircraft noise based on their physiological ability to maintain the structure of sleep with the additional stressor or arousing influence of noise. Some groups of people have been identified as having more sleep-related difficulties to begin with. For example, elderly populations have been identified as having (though varying somewhat by gender) decreased slow-wave sleep, decreased sleep time, diminished efficiency of sleep, and decreased objective and perceived sleep quality. Thus, the elderly may constitute a susceptible population (Unruh, Redline, An, Buysse, Nieto, Yeh, Newman, 2008).

There is another possibility, that illustrated by Basner (2008) in the pupillographic study discussed earlier. That is, that it may be possible to experience increased objectively measured sleepiness (as measured by using PST) in response to a night of noise despite lack of significant changes in sleep structure in response to the noise. To quote his conclusion, “Hence, maintenance of sleep macrostructure was associated with a biological cost in terms of sleep recuperation”. Thus, further study may be necessary in order to determine the long term costs to individuals of maintaining sleep macrostructure even in those who do not show overt changes in their sleep; since these too appear to experience potentially detrimental effects.

Another issue that needs exploration is whether people with habitually short sleep experience the possible detrimental effects of noise on sleep in the same way as those who sleep for a *normal* amount of time? On the one hand those with short sleep might be more susceptible to the health effects due to already shortened duration of sleep. On the other hand, habitually sleep deprived individuals might have an increased biological drive to maintain sleep due to the sleep debt accumulated through their shortened hours. Whether habitually short sleepers would be more or less affected by possible sleep-related health effects of aircraft noise is an important subject for future study as shorter hours of sleep become increasingly common in current society. Thus, future studies need to address the question of the importance of trait ease of awakening and trait ease of inter-stage arousal as well as the biological costs of sleep structure maintenance and possibly resulting health effects. Also, the potentially modifying effect of habitual sleep length and sleep debt on objectively measured tiredness (PST) should be studied.

### **3.2 Cardiovascular Arousal During Waking and Sleep (Blood Pressure and Heart Rate Effects)**

Short term increases in blood pressure due to noise are well documented in both laboratory and industrial settings and have led researchers to wonder whether long term exposure might lead to more permanent increases or other effects. Increased incidence of high blood pressure associated with long term exposure to industrial noise has also been documented. High blood pressure is a well-known risk factor for cardiovascular disease and particularly heart attack (myocardial infarction). Findings relating noise to blood pressure changes and especially to the development of hypertension are therefore of significant interest because transportation noise studies have exposed a possible link between noise exposure and myocardial infarction, for which noise-induced hypertension may be a pathway.

#### **3.2.1 Transient Blood Pressure in Waking**

Significant effects on ambulatory blood pressure due to exposure to loud sounds have been found in several studies (as discussed below) performed both in the laboratory and in the work place, and the results seem consistent between the two environments. Those in the workplace involved measuring ambulatory blood pressure together with exposure to noise (subjects wore a personal noise dosimeter) and examining the resulting trends in blood pressure response. Significant results were consistently found for increases in blood pressure in response to sound:

- ❑ Lusk, Hagerty, Gillespie and Ziemba (2004) found an increase of 2-mmHg systolic blood pressure (SBP) for an increase in average noise level of 10 dBA or a difference between average and peak noise levels of 5 dB. They also found a 2-mmHg diastolic blood pressure increase for an increase in average level of 13 dB or an increase in average to peak difference of 6 dBA.
- ❑ In a 2001 study by Fogari, Corradi, Marasi, Vanasia and Zanchetti, no significant finding was identified for clinical measures of blood pressure (where someone sits down and the doctor puts the cuff on and listens with a stethoscope, etc), however, when ambulatory methods were used, significant differences of +3.6 mmHg systolic and +2.3 mmHg diastolic blood pressure were found between a group of highly noise exposed (>85 dBA) and unexposed (<80 dBA) industrial workers. These differences grew to +5.6 mmHg and +3.5 mmHg when only the working part of the day (when exposure was taking place) was considered.
- ❑ In a 2003 study using ambulatory blood pressure monitoring, Chang, Jain, Wang and Chan (2003) found only a marginally significant transient effect of a 1 mmHg/1 dBA increase in blood pressure per 1 dB increase in level of occupational noise at a lag time of one hour from noise exposure. In this same study a significant difference was identified in sleep-time blood pressure of  $16 \pm 6$  mmHg between a group exposed to  $85 \pm 8$  dBA and one exposed to  $59 \pm 4$  dBA levels during the workday. This sleep-time result is of particular interest because it seems to display a longer term effect (that during the night as compared with only an hour later) than the transient cardiovascular arousals that have been more frequently examined. Nighttime blood pressure or the dip in nighttime blood pressure may have significant predictive value for heart disease beyond that of normal blood pressure so increases in nighttime blood pressure may be a non-benign effect (Stolarz, Staessen, O'Brien, 2002).

#### **3.2.2 Autonomic and Cardiovascular Arousal During Sleep**

Additional effects take place when exposure to noise occurs **during sleep** itself. Noise exposure during sleep (including transportation noise) results in short-term increases in blood pressure. In a subset of the HYENA study population, Haralabidis et al. (2008), found a significant increase in nighttime ambulatory blood pressure (6.2 mmHg systolic blood pressure and 7.4 mmHg diastolic blood pressure) in the 15-min time intervals in which an aircraft noise event occurred. Carter, Henderson, Hart, Booth, and Hunyor (2002) found that blood pressure increase appeared to be correlated with the sudden onset of the sounds rather than the level of the sounds. While awakening (either as a result of the noise, or when subjects were coincidentally already awake when the noise occurred) was found to increase the magnitude of the blood pressure effect, awakening was not a necessary condition for its occurrence.

An important observation common to these studies is a lack of a habituation effect. Carter et al. (2002) found no habituation effect for blood pressure across the three days of laboratory noise exposure in their experiment. In the HYENA study a subset of subjects who had lived in their houses for a sufficiently long time to habituate still had noticeable acute blood pressure changes in the presence of a noise event. Thus, neither short- nor long-term habituation was observed for this effect. The home setting of the HYENA study also makes a laboratory effect an unlikely explanation. Although differing in the parameters examined (sleep stage architecture versus blood pressure or heart rate arousals), this lack of a habituation effect parallels the findings of Kuroiwa, Xin, Suzuki, Sasazawa, and Kawada (2002) who showed that while subjective measures of sleep may exhibit habituation to repeat exposure to nighttime noise, polygraphic parameters (quantities of S1, S2, S3+S4, and REM) show little or no evidence of habituation across study nights. Thus, while some (especially subjective) effects of nighttime noise may diminish over time in an environment, others may not. Blood pressure and heart rate seem to be in this latter category.

### **3.2.3 Heart Rate Changes**

Significant changes in heart rate contemporary with noise events have also been noted in some studies.

Raggam, Cik, Holdrich, Fallast, Gallasch, Fend, Lackner and Marth (2007) measured responses to playback of binaurally recorded road noise in the laboratory and found increases in heart rate for all intervals in which subjects were noise exposed compared to intervals in which they were non-exposed. Results for changes in heart rate were less clear among the studies examined and were frequently only marginally significant. In a study by Lusk and Hagerty (2004) a 10% increase in number of minutes exposed to peak noise led to a significant 3 beat per minute (bpm) increase in heart rate. Fogari et al. (2001) report a significant increase in heart rate in a high noise exposure group (>85 dBA) vs. a low exposure group (<80 dBA) of 2.8 bpm during a working day, 2.2 bpm during a nonworking day and 3.7 bpm during the actual period of exposure, while during nighttime, significant differences in heart rate were not noted between the two groups. Additionally, in the Haralabidis et al. (2008) study a non-significant increase in heart rate of 5.4 bpm was observed in the presence of a noise event.

There are several obvious difficulties in measuring heart rate during the day. There are many transitory influences on heart rate including, but not limited to, concurrent emotional or physical stress or arousal, dietary intake, presence of stimulants (such as caffeine) etc. Thus, most studies have examined patients during sleep. Raggam, Cik, Holdrich, Fallast, Gallasch, Fend, Lackner, and Marth (2007) examined patients during waking-hours exposed to traffic noise in a laboratory setting. They noted increased heart rate in all noise affected segments relative to quiet segments. Increased heart rate has been linked in several studies to increased cardiovascular risks (Gillum, Makuc, Feldman, 1990; Gillman, Kannel Belanger, D'Agostino, 1993; Palatini and Julius, 1997). It has also been hypothesized that transient arousals may lead to more permanent elevations (Chang, Su, Lin, Jain, and Chan, 2007; Davies, Teschke, Kennedy, Hodgson, Hertzman, Demers, 2005; Carter Henderson, Lal, Hart, Booth, Hunyor, 2002; Morrell, Finn, Kim, Peppard, Badr, Young, 2000). Thus, repeated transient arousal of the cardiovascular system may be one mechanism by which noise may lead to more serious cardiovascular consequences.

Thus, acute noise exposure, whether during waking or sleep, may result in cardiovascular arousals including increases in blood pressure and heart rate. Repeated cardiac arousal in response to noise has been seen as a potential heart disease risk.

### **3.3 Sympathetic Tone**

An increase in sympathetic tone has been noted in a number of sleep studies and is believed to lead to several of the adverse outcomes of sleep disturbance. Decreases in either the length of the quality of sleep have been linked with increases in sympathetic tone. Increases in sympathetic activity may lead to increases in blood pressure, obesity, and insulin resistance, the individual components of the metabolic syndrome; a possible analogue of dysregulation as described in (Glei, Goldmann, Chuang, Weinstein, 2007.).

### **3.3.1 Relationship of sleep disturbance and sympathetic tone**

**Restriction or fragmentation of sleep appears to lead to increased sympathetic tone**—meaning the amount of activity or traffic in the sympathetic nervous system. The sympathetic nervous system is involved in the body's fight or flight mechanism and is responsible for priming the body for action. It is thus activated along with other systems when we experience stressors (Tsatsoulis and Fountoulakis, 2006). Its activity characteristically decreases during sleep, thus sleep restriction generally increases the amount of time in which it is active. Supporting this, in a study on restricted sleep, Spiegel, Leproult, and Van Cauter (1999) found an increased quantity of sympathetic nervous system activity in subjects in the restricted sleep condition (4 hrs in bed centered around 3 a.m.) versus the rested condition. In an investigation of the impact of individual sleep stages on the activity of the sympathetic nervous system using microneurography, Somers, Dyken, Mark, and Abboud (1993) determined that the sympathetic nervous system reaches its nadir (or lowest point) during non-REM stage four of sleep, which is the deepest sleep stage. They also found that arousal stimuli (acoustic stimuli such as a knock on the door) during stage 2 of non-REM sleep were associated with K-complexes, high-amplitude deflections on the EEG, variations often associated with sympathetic-nervous activation. Griefahn, Brode, Marks, and Basner (2008) found that sympathetic reactions elicited by awakening from various sleep stages differed by sleep stage with awakening from stage four of non-REM sleep (slow wave sleep) eliciting the strongest sympathetic reaction and awakening from REM sleep eliciting the weakest. They also found that among acoustic stimuli that did not result in awakening, heart rate increases were significantly influenced by the sleep stage prior to the stimulus, being least in slow wave sleep and greatest in REM sleep.

It appears that this increased activation during sleep may carry over into daytime sympathetic increases as well. Those with sleep apnea or sleep restriction have been noted to experience increased levels of sympathetic activity during the daytime, see, for example, (Wolk, R., Somers, V. K., 2006; Spiegel Knutson, Tasali, Leproult, Van Cauter, 2005, for example). Similar to the case for sleep disordered breathing, restless leg sufferers have been reported to experience increased sympathetic activity (Sforza, Pichot, Barthelemy, Haba-Rubio, Roche, 2005). Spiegel, Knutson, Leproult, Tasali, and Van Cauter (2005) propose, based on other studies, that sleep fragmentation may also lead to increased sympathetic nervous system output. These increases are viewed as a potential mediator of other associated health outcomes such as hypertension, see, for example, (Wolk and Somers, 2006) or diabetes/insulin resistance, see, for example, (Punjabi, et al., 2004).

### **3.3.2 Possible connection of sympathetic tone with hypertension**

**Sympathetic tone is believed to lead to hypertension.** It appears to have effects on each of peripheral blood flow, elevations of heart rate, breathing, and blood pressure (Griefahn et al, 2008). It is seen as a potential link in a number of the metabolic syndrome components leading to hypertension. Gangwisch, Heymsfield, Boden-Albala, Buijs, Kreier, Pickering, Rundle, Zammit, and Malaspina (2006) mention increased sympathetic activity in their study of hypertension and sleep duration in the National Health and Nutrition Examination Survey (NHANES) as a potential mediator of an effect of sleep duration on hypertension development. Yanai, Tomono, Ito, Furutani, Yoshida, and Tada, (2008) suggest that sympathetic activation may provide a mechanism for obesity-related hypertension via a stimulatory effect of increased leptin in obese individuals on sympathetic activity (i.e. leptin may stimulate the sympathetic nervous system and thus contribute to hypertension). Reaven, Lithell, and Landsberg (1996), analyzing the results of a study by Ward, Sparrow, Landsberg, Young, and Weiss (1993) propose that the sympathetic nervous system may be the link between insulin level and blood pressure. This likelihood was strengthened in their estimation by the fact that in response to a high fat diet insulin resistance first develops, followed by hypertension. They also observe that non-pharmacological interventions in patients with insulin resistance such as fasting, exercise, and weight loss reduce sympathetic nervous system activity and hypertension in parallel. These were seen as consistent with a role of sympathetic tone in primary hypertension. Esler, Rumantir, Wiesner, Kaye, Hastings and Lambert, (2001) discuss possible mediating pathways for the well-documented effects of sympathetic activity in essential hypertension. Possible mechanisms discussed include: increases in sympathetic innervations associated with nerve growth factor expression; increased sympathetic nerve firing rates; adrenaline cotransmission; and noradrenaline transporter malfunction (failure

of reuptake mechanism makes effects bigger than they otherwise would be). Whatever the mechanism or mechanisms may be, the key point for the purpose of this review (how aircraft noise might affect health) is that increased sympathetic activity (a possible outcome of noise exposure) is associated with hypertension although a complete picture of all the mechanisms at play is the subject of ongoing investigations.

Increased sympathetic tone activity has also been proposed as a mediator in **noise-related vascular changes**. Chang, Su, Lin, Jain, and Chan (2007) felt that their results showing sustained vascular property changes in noise exposed workers provided empirical evidence for the possible causative influence that noise has on blood pressure through sympathetic tone induced endothelial lesion.

### **3.3.3 Possible connection of sympathetic tone with glucose mismanagement**

**Sympathetic tone** has also been implicated as a potential pathway in the development of **glucose mismanagement**. In a 2008 study by Tasali, Leproult, Ehrmann, and Van Cauter, subjects were allowed a normal amount of total sleep time, but their slow-wave sleep (stages 3 & 4 of non-rapid eye movement sleep) was restricted using stimulation with (primarily) acoustic tones. In this study they observed an increase in sympathetic tone (activity in the sympathetic nervous system) and sympathovagal balance (the ratio of activity in the sympathetic nervous system to that in the vagus nerve, which helps regulate the heart). They measured this from a spectral analysis of heart rate variability and the electrocardiogram. The ratio of high versus low frequency content in the signal can be used to determine relative magnitudes of the sympathetic and vagal components. The observed increase in sympathetic activity was seen as being a potential mediator of the decreased regulation of glucose seen in sleep deprived people. In their experiment, particularly, they note that “pancreatic insulin release is inhibited by increased sympathetic versus parasympathetic tone” and “insulin resistance can also occur secondary to increased sympathetic activity”. Spiegel, Knutson, Leproult, Tasali, and Van Cauter (2005) similarly remark that “pancreatic beta-cell function is influenced by autonomic nervous system activity, with sympathetic activation inhibiting and parasympathetic activation stimulating insulin release.” Spiegel, Leproult, and Van Cauter (1999) observed a decrease in acute insulin response, which they stated could be a result of the increased sympathovagal balance. Thus, increased sympathetic tone may lead to both a decreased secretion of insulin and a lessened response to its signaling.

Effects of sympathetic and vagal activity on leptin and ghrelin signaling have also been proposed (Rayner and Trayhurn, 2001; Spiegel, Tasali, Penev, Van Cauter, 2004) and so may additionally lead to the eventual development of obesity as discussed below. If this is indeed the case, the activity of the sympathetic nervous system may be somewhat of a center point in the genesis of health effects of noise mediated by either sleep disruption or stress. As such, it justifies a place in future research seeking to determine the impacts of noise exposure during both the day and night, serving possibly both as an indicator of relevant exposure and a mediator of future pathological change. Future research should examine whether individuals exposed to nighttime noise generally have increased sympathetic activity, and determine if a night of noise may be equated to an equivalent amount of sleep loss in its effects on sympathetic nervous system activity. In one study, where sympathetic activity was assessed after a night of noise interrupted sleep and after a normal night’s sleep, Tasali, Leproult, Ehrmann and van Cauter (2008) found that when slow wave sleep was selectively prohibited using noise stimuli there were changes in glucose management. While this may be seen as a sympathetic nervous system response to noise exposure, the selective prevention of particular stages of sleep may be somewhat more specific than the less deliberately timed effect of aircraft noise disruption (aircraft noise might occur while someone was in various sleep stages while this intervention selectively suppressed slow-wave sleep). Particularly, the selective prevention of deep sleep might have an effect on the sympathetic nervous system beyond what might be produced by more general and less specifically timed exposure. For instance, sympathetic activity generally reaches a low point during slow-wave sleep so that restricting or perhaps even interrupting slow wave sleep might tend to increase sympathetic activity more than at other times. Further research is needed on this topic.

### 3.4 Leptin, Ghrelin and Hunger

The appetite regulating hormones leptin and ghrelin are widely suspected of playing a role in the relationship between short sleep duration and obesity via the regulation of appetite. Leptin leads to feelings of satiety (feeling not hungry or full) and correlates with both adiposity and longer-term sleep restriction (i.e. it is produced in adipose tissue and decreases with long term sleep restriction). Ghrelin, on the other hand, correlates more with both acute hunger and acute sleep restriction so that acute sleep restriction leads to increases in ghrelin levels (Taheri, Lin, Austin, Young, Emmanuel, 2004). Leptin levels fall and ghrelin levels rise in sleep restricted individuals potentially leading to intensified appetite and overeating.

A paper by Spiegel, Tasali, Penev and Van Cauter (2004) gives a good summary of both the effects of leptin and ghrelin and the effect of sleep restriction on their regulation, and in this section their reasoning is outlined. Leptin is an anorexigenic hormone meaning that it reduces hunger and produces feelings of satiety (feeling full). It is produced in the adipose (fatty) tissue and is thought to have a role in regulating energy intake and expenditure. Ghrelin on the other hand is an orexigenic hormone, meaning that it is hunger producing. It is produced primarily in cells lining the fundus of the stomach and in the pancreas. In the experiment of Spiegel et al. (2004), which involved 12 people and employed a cross-over design, subjects were involved in two studies six weeks apart. They were subjected to either an increased or decreased length of time in bed and queried as to their total hunger as well as their appetite for particular types of foods. During certain study nights, blood samples were drawn and leptin and ghrelin concentrations measured. During the 4-hour restricted sleep condition subjects had mean leptin levels 18% lower (for comparison they mention that three days of underfeeding by 900 calories per day results in a comparable decrease of 22%), and 28% higher ghrelin levels than in the 10-hour extended condition. Subjects also indicated a 24% increase in hunger ratings on a 10-cm visual scale in the sleep restricted condition. In their analysis, they also noted that 70% of the observed increase in appetite with sleep restriction could be accounted for by the change in the proportional concentrations of leptin and ghrelin. Thus, the role of these two hormones in regulating appetite should not be underestimated. Leptin and Ghrelin additionally form a possible link between the action of the sympathetic nervous system and obesity. Leptin secretion is inhibited by the sympathetic nervous system (Rayner and Trayhurn, 2001), while ghrelin secretion is inhibited by the action of the vagus nerve (Spiegel et al., 2004), thus sleep restriction leads to increased sympathovagal balance, which leads to increased leptin to ghrelin ratio, which in turn leads to increased hunger with an increased appetite for sweet salty foods with high carbohydrate content, and this leads finally (according to this hypothesis) to people becoming obese.

In a large observational study involving 1024 volunteers from the Wisconsin Sleep Cohort Study, subjects underwent polysomnography (recording of their sleep patterns with relevant physiological variables), as well as measures of hormone levels including leptin and ghrelin, and standard biometric information such as Body Mass Index (BMI). In this study population, participants experiencing shorter sleep (as part of their typical life, this was not an intervention study) had reduced leptin and elevated ghrelin similar to the trend seen in the smaller short-term study of Spiegel et al. (2004) just discussed. A reduction in sleep from 8-hours to 5-hours of sleep was associated with a predicted 15.5% decrease in leptin levels; this same reduction in sleep time results in a 14.9% increase in ghrelin levels. A trend of increasing body mass with shortened sleep was also observed and this was seen as possibly being a consequence of increased hunger and appetite associated with sleep related changes in leptin and ghrelin signaling (Taheri, Lin Austin, Young Mignot, 2004). The results of this study help generalize some aspects of the results of the previously mentioned study in that it shows that similar effects take place even if people are sleeping the amount of time to which they are accustomed and are not interfered with as to the length of their sleep. It maintains the involvement of the laboratory sleeping environment so this remains a potential factor, though the fact that the Spiegel et al. (2004) experiment found differences in the lab between those sleep restricted and those not diminishes the likelihood of this being purely a 'sleeping in the lab' effect. The fact that sleep duration was not regulated in the Taheri et al. (2004) experiment suggests a non-habituating aspect to the effect since it occurred in those experiencing their own typical sleep time (Taheri, Lin, Austin, Young and Emmanuel, 2004).

Further steps in the future might include a study in which samples are drawn in the home environment so as to conclusively exclude a laboratory effect. Also, for the purposes of this review it would be of substantial interest to see to what extent leptin and ghrelin are susceptible to change due to sleep disruption by nighttime noise. This would make it possible to gauge whether and to what extent sleep disruption by nighttime noise might lead to possible changes in leptin and ghrelin secretion and to resulting changes in obesity. To summarize leptin and ghrelin are used by the body in the regulation of appetite. The balance of these hormones changes with decreased sleep time in a way that favors increased appetite and may lead to increased obesity, a result demonstrated in longitudinal studies, discussed at greater length in the section on obesity below.

### **3.5 Immune Effects of Sleep Loss**

Some recent research has focused on the effects of sleep loss on the immune system. Weakening of the immune system may be considered a serious outcome on its own and can also lead to other health problems such as myocardial infarction (Nieminen, Mattila, Valtonen, 1993). Much of the study of immune effects of sleep disturbance has been performed in a laboratory and has involved some sort of sleep restriction paradigm. Chronic sleep deprivation in rats leads to death by sepsis (Majde, Krueger, 2005), however sleep deprivation in other animal models leads to more mixed results including improvements in some markers of immune function. In humans, after a sleep restriction of subjects between 10 p.m. and 3 a.m. subjects were found to have reduced natural killer cell activity, as well as reduced natural killer cell activity per number of natural killer cells, as well as reduced lymphokine activity killer cell activity (Irwin, McClintick, Costlow, Fortner, White, Gillin, 1996). The authors of the study mention, however, that other studies in humans have found increases in immune activity with sleep restriction. Additionally, while changes in sleep may affect the immune system the reverse is also true. The common link seems to be the action of cytokines, signaling chemicals used by the immune system, some of which also play a role in the regulation of sleep. Understanding of any bearing that noise may have on immune function will likely have to wait for future research to more fully delineate the roles that sleep plays in immune function. For reviews of these issues see, for example, (Motivala and Irwin, 2007); and (Majde and Krueger, 2005).

### **3.6 Summary**

In this section, research related to the influence of sleep and changes in sleep on blood pressure, heart rate, sympathetic tone, appetite controlling hormones (leptin and ghrelin) have been described. In addition, a summary of some research related to immune effects due to sleep loss was given. In the next section diseases that may be an outcome of such changes are discussed and research examining the connection between the changes and the health outcomes is described.

## 4 CARDIOVASCULAR AND METABOLIC CONSEQUENCES

Recent studies on noise health effects have linked noise exposure at night and during the day with the potential development of hypertension (Jarup, Babisch, Houthuijs, et al, 2008) and heart disease (Babisch, 2006). These works contained indications that the effects seen may be a result of the disruptive effects of noise on sleep. In other studies not dealing with noise, groups experiencing decreased sleep quality and quantity show increases in prevalence of and likelihood of developing obesity, Type-2 diabetes, hypertension and heart disease. This clustering of symptoms is similar to that seen in the metabolic syndrome. Although these studies did not examine noise, their findings for hypertension and heart disease may be illustrative of a potential mechanism for the proposed effects of nighttime noise on these variables, and justify further research to determine whether nighttime noise may be capable of sufficiently influencing the development of obesity and diabetes as well as the two more studied effects—hypertension and heart disease.

This should take place by first considering studies relating noise exposure to amounts and types of sleep disruption and then by considering the effect sizes seen in studies relating sleep disruption of various sizes to obesity and diabetes. This then would allow estimation of the likely magnitude of the effects of the less or un-studied effects. If the effect predicted in this manner is of sufficient magnitude, reexamination of previous studies on noise and health effects where these variables (obesity or diabetes) may have been included as confounders may reveal whether these possible effects do indeed occur. Such a finding would suggest the need for further well designed studies to ascertain the specifics of such a relationship. Further research is also needed to determine the relative contributions of daytime and nighttime exposure to noise related cardiovascular (or possibly metabolic) outcomes since stress (a second possible noise pathway) may also lead to obesity and insulin resistance (Tsatsoulis and Fountoulakis, 2006). For a summary of the proposed health effects and pathways, see Figure 4, below.

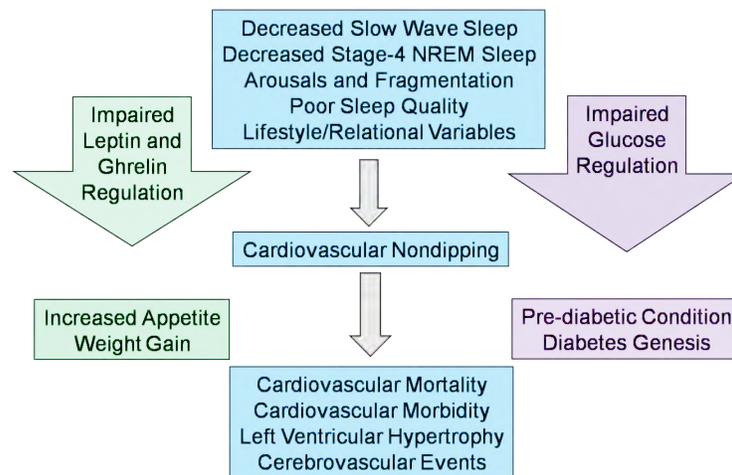


Figure 4: Relationships between sleep and metabolic and cardiovascular variables.

### 4.1 Obesity

Studies have shown an increased risk of obesity in those receiving shorter sleep and fragmented sleep. No studies deliberately studying a relationship between noise exposure and prevalence of obesity were found. As was discussed in section 3, a plausible outcome of leptin and ghrelin appetite hormone disregulation may

be increased appetite and resultant weight gain. Thus, sleep restriction has been hypothesized to be a risk factor for the development of obesity in habitually sleep-deprived people. Accordingly, this hypothesis has been tested using both cross-sectional and longitudinal study designs with somewhat consistent results for short sleepers. Note: the expression “short sleeper” is often defined as 6 or fewer hours though exact definition varies by study and age group examined with younger individuals generally needing greater amounts of sleep. In longitudinal studies, researchers have tended to find more frequent increases in average BMI in those receiving shorter self- or other-reported sleep duration, however these results for adult populations are often not statistically significant, and so a measure of caution is required in their interpretation. However, several strong trends have emerged predicting prevalence of obesity in children by sleep behavior. Cross-sectional studies have resulted in consistently strong results although sometimes these have been restricted to a particular age group. This does however appear to be consistent with the theory because age-related changes in sleeping would tend to dilute the effects—i.e. since older people don't receive as much sleep typically, the effect of habitual restriction of it in an older population will be more diluted and is less likely to have a noticeable effect. Obesity is a potential risk factor for heart disease and is influenced by sleep wellness, which may be impacted by noise. Thus, its potential role as a waypoint in the development of hypothesized noise-induced heart disease makes it a subject of interest.

In a study of children involved in the Avon Longitudinal Study (Reilly, Armstrong, Dorosty, Emmett, Ness, Rogers, Steer and Sheriff, 2005) it was found that short sleep duration at three years of age (defined for this age group as less than 10.5 hours) was one of eight significant risk factors (Odds Ratio=1.45, 95% C. I. 1.10 to 1.89) predicting the development of early childhood obesity. Compare this to the also significant relationship found for watching television 8 or more hours per week at age three noted in the same study, which had an odds ratio of (OR=1.55, 95% C. I. 1.13 to 2.12). In both cases children are nearly half again more likely to develop childhood obesity. In another study dealing with children, Sekine, Yamagama, Handa, Saito, Nanri, Kawaminami, Yoshida and Kagamimori (2002) found a significant relationship between sleep time and the presence of childhood obesity. For children receiving 9-10 hours a night of sleep the odds ratio was (OR=1.49, 95% CI 1.08 to 2.14); for those receiving 8-9 hours of sleep a night the odds ratio was (OR=1.89, 95% CI 1.34 to 2.73) and for those receiving less than 8, the odds ratio was (OR=2.87, 95% CI 1.61–5.05) when compared to those receiving more than 10 hours of sleep. The trend here appears to be monotonic a common thread among childhood studies contrasting the mixed monotonic and U-shaped results often found in studies of adults.

Hasler, Buysse, Klaghofer, Gamma, Ajdcic, Eich, Rossler and Angst (2004) found a negative association between sleep duration and body mass index in young adults which diminished after 34 years of age, as well as an association between short sleep duration (defined as less than 6 hours) and obesity. Significant associations were observed between sleep duration and current, as well as previous obesity, highlighting the possibility that the causality is bidirectional. Taheri, Lin, Austin, Young, and Mignot (2004) found a U-shaped association between body-mass index and sleep duration in a sample of subjects between ages 30 and 60 (average age 53.1 years of age). They believed that this association was mediated through the appetite regulating properties of leptin and ghrelin, which they also measured in their study and found to be reduced and elevated (respectively) in those receiving lessened sleep. In a cross-sectional study of patient medical records and survey data (ages 18-91 mean age 48), Vorona, Winn, Babineau, Eng, Feldman, and Ware (2005) found that overweight and obese patients slept less than patients with a normal body-mass index although the trend was reversed in extremely obese patients. This appears to coincide with the U-shaped trend that has been noted in other studies. In a study of Gangwisch, Malaspina, Boden-Albala, and Heymsfield (2005) subjects between 32 and 49 years of age who reported getting less than 6 hours were at increased risk of developing obesity. Risk increased as length of sleep decreased in short sleepers. Those getting 2 to 4, 5, and 6 hours of sleep per night were respectively 135% (OR =2.35, 95% CI 1.36-4.05), 60% (OR = 1.60, 95% CI 1.12-2.29), and 27% (OR = 1.27, 95% CI 1.01-1.60) more likely to be obese (after adjusting for potential confounding variables) than subjects who reported getting 7 hours of sleep per night.

It is clear why shorter sleep durations (<6 hrs in most adult studies) can lead to obesity through its effect on hunger regulating hormones. What is less clear is why longer (>9 hrs) sleep duration may also lead to obesity. It has, however, been suggested that decreases in energy usage due to increased time in bed

(including by exercising less) may overbalance the effect of lessened food intake in these subjects (Taheri, Lin, Austin, Young, Mignot, 2004). Researchers examining prevalence of obesity with sleep duration have accordingly encountered mixed results when studying the results of long sleep duration. In longitudinal studies non-significant trends towards lessening weight gain with longer sleep have been observed so that longer sleepers could conceivably be at lower risk (Gangwisch, Malaspina, Boden-Albala, Heymsfield, 2005). On the other hand, in some cross-sectional studies a U-shaped relationship between sleep duration and obesity prevalence has been observed. This may be due to the development of sleep disorders such as apnea in obese persons, a fairly well attested phenomenon (see, for example, de Sousa, A. G. P., Cerato, C., Mancini, M. C., Halpern, A., 2008), and the resulting decrease in efficiency of disrupted sleep, but further research is needed to determine whether this is in fact the cause of the U-shaped relationship sometimes observed or whether there is another cause such as decreased daily energy expenditure. Additionally, the U-shaped relationship has not appeared in studies dealing with children's sleep, where the observed relationship between short sleep and obesity appears to be a causal and monotonic relationship, lending further support to the idea that shortened sleep may be a cause, rather than merely being a symptom, of obesity.

In summary, recent well-designed studies have shown an increase in the prevalence of obesity in subjects experiencing sleep durations that are short for their age-group. These trends have been hypothesized to result from changes in leptin and ghrelin signaling and resulting increases in appetite, especially for carbohydrate rich foods. Future studies should examine whether sleep affected by noise results in similar changes in these variables.

#### **4.2 Glucose Regulation and Diabetes**

Results of studies on acute and long term sleep restriction as well as low sleep quality (fragmentation) have shown a relationship between decreased sleep duration & quality and increased development or prevalence of diabetes. If either of sleep disruption or restriction is a major avenue for the development of heart disease and hypertension as a result of noise (Babisch, 2006, Jarup et al., 2008) diabetes may be another outcomes, and may also contribute to the incidence of heart disease. It may also have an increased likelihood of occurring together with the studied outcomes (hypertension and myocardial infarction) in studies of noise and heart disease. Thus, its inclusion as a confounder or the potential exclusion of those manifesting it may be problematic in statistical analyses though perhaps necessary because of the increased intensity of related health outcomes that results from this condition.

Increased diabetes risk with sleep disturbance has recently been identified in a number of studies dealing with length, quality, or stage structure of sleep. Studies of sleep restriction in the laboratory have shown decreased glucose tolerance and increased activity in the sympathetic nervous system (a portion of the nervous system associated with arousal) and have observed that sleep curtailment increases risk factors for diabetes (Spiegel, Leproult, Van Cauter, 1999; Tasali, Leproult, Ehrmann, Van Cauter, 2008). Supporting evidence for a longer term effect has come from a cross-sectional study by Knutson, Ryden, Mander and Van Cauter (2006) who found in a group of diabetic patients that sleep duration and quality were predictive of HbA<sub>1c</sub> levels, a key marker of glycemic control and thus diabetes severity. Like that between sleep and obesity, the connection between diabetes and disturbed sleep is a complicated one, since diabetes is also believed to contribute to sleep difficulties (Resnick, Redline, Shahar, Gilpin, Newman, Walter, Ewy, Howard, Punjabi, 2003; Spiegel, Knutson, Leproult, Tasali, Van Cauter, 2005). However, the causal connection is fairly well supported by longitudinal studies that show increased risk of diabetes for those free of diabetes at baseline but experiencing sleep disturbance defined as difficulty falling asleep (initiation) or maintaining sleep (Kawakami, Takatsuka, Shimizu, 2004; Mallon, Broman, Hetta, 2005; Meisinger, Meier, Loewel, 2005; Nilsson, Roost, Engstrom, Hedblad, Berglund, 2004), and those experiencing short sleep duration (Ayas, White Al-Delaimy, Manson, Stamofor, Speizer, Patel, Hu, 2003; Mallon et al, 2005; Yaggi, Araujo, McKinlay, 2006). In one case (Ayas et al., 2003) the effect appeared to diminish when adjusting for obesity, however, in light of the previously mentioned effects of sleep duration on obesity, this is not entirely surprising. It is possible that obesity is one mechanism through which sleep duration can affect the development of diabetes, as obesity is also a diabetes risk factor.

While obesity may be one mechanism through which the development of diabetes is influenced by sleep, there appears to be strong evidential support for a more direct effect on glucose management than that which may be mediated through obesity. A number of studies have implicated increased sympathetic nervous system activity as the mechanism whereby sleep disturbance effects glucose regulation (for example Tasali, Leproult, Ehrmann, van Cauter, 2008; Kawakami, Takatsuka, Shimizu, 2004; Mallon, Broman, Hetta, 2005; Meisinger, Heier, Loewel, 2005;). This is a link worthy of further study, especially since increased sympathetic nervous activity may also provide a pathway for the development of obesity via its influence on leptin and ghrelin signaling. (Spiegel, Tasali, Penev, van Cauter, 2004)

The magnitude of these effects is important because the proposed effect of noise on sleep duration and continuity is in most cases comparatively small, e.g. after a study at the German Aerospace Institute researchers concluded that on average people living near an airport could be kept (on average) from experiencing more than one additional awakening per night (Basner et al., 2006). An effect was found in the studies where diabetes was examined as a function of sleep duration, the effect was present when sleep was restricted as part of an experimental protocol and also when it was restricted as part of a modern schedule. Restriction to 4 hours of sleep per night for six days was investigated in a study by Spiegel, Leproult, and Van Cauter (1999) in which they found impaired glucose management of a degree and type typically seen in those much older (~30 years older for some effects) and those experiencing noninsulin-dependent diabetes. This finding of an acute effect on glucose tolerance points to the risks that may exist when sleep deprivation voluntary or otherwise persists over a long period of time. In this same study, sleep debt was also found to have significant negative effects on glucose management and be a potential metabolic health risk. In order to gauge the magnitude of some of these effects they are organized in tabular form in Table 1.

As mentioned earlier, Tasali, Leproult, Ehrmann, and Van Cauter (2008) restricted the slow-wave sleep of subjects using EEG to determine sleep stage and delivering acoustic stimuli in order to move subjects into lighter non-REM (non-random eye movement) sleep whenever they showed evidence of slow-wave activity. By selectively varying the duration, frequency and intensity of the stimulus, and only administering it when delta waves (the slow-waves spoken of) were present, they were able to selectively inhibit slow-wave sleep while keeping the total sleep duration constant and avoiding awakenings. This experimental protocol yielded several results:

First, insulin sensitivity was substantially reduced (25% reduction) in the slow-wave sleep restricted condition and was similar to what would be expected in a group with 8-13 kg greater body mass than the actual group studied or at high risk for diabetes. Also, no compensatory increase in insulin was observed, similar to findings in several prospective studies.

Second, there was no significant effect noted on nighttime cortisol secretions when slow-wave sleep was suppressed as described. In the slow-wave sleep mediated aspects of glucose management, it seems justifiable to say that cortisol the cause or is at least unnecessary for its occurrence.

Third, the number of microarousals was not significantly correlated with measures of glucose tolerance in all but one of the subjects so that sleep continuity did not seem to be the functional mechanism in the diminished glucose management. The actual diminishment of slow wave (stage 3 & 4) sleep appears to be the cause. Although different in some ways from the problem at hand (responses to aircraft noise) these results may give us an indication of a possible causal mechanism by which sleep fragmentation influences the development of Type 2 diabetes. Fragmented sleep has been noted to decrease slow-wave sleep in favor of lighter sleep stages; thus, stage changes may be a mechanism through which fragmentation influences glucose management, though whether it is the only one remains unknown—other impacts of sleep fragmentation may, e.g., lead to shortening of REM sleep, but the possible impact of this was not studied. Experiments fragmenting sleep, but carefully maintaining slow-wave sleep (possibly by fragmenting later parts of the sleep period) might be a way of making this determination.

Fourth, they observed a shift towards higher levels of sympathovagal balance which they interpreted as being indicative of a general shift towards greater general sympathetic activity. Since “over-activity of the

sympathetic nervous system results in insulin resistance” the observed change in sympathetic nervous activity may be a main avenue through which sleep disturbance may affect glucose management and lead to the genesis of diabetes. The changes in both sympathetic tone and insulin sensitivity are also characteristic of increases seen in aging.

Fifth, the source of disruption in this study was noise. While possibly differing in its characteristics from aircraft noise in many respects, and being specifically targeted in its timing to disrupt slow-wave sleep, it is an interesting demonstration, in principle, of the possibility of metabolic effects arising from noise exposure during sleep. This might be of special concern during the early night hours when slow wave sleep predominates. However, subjects are generally more resistant to awakening during slow-wave sleep than at other times. Some researchers have suggested that this time may be ideal for increased flight traffic because of this decreased probability of awakening (Anderson and Miller, 2007). A promising suggestion for its potential advantages, further research should take place on the potential impacts on metabolic variables of increased air traffic noise at these times before implementing such a policy.

Table 1: Studies and results relating sleep disruption and diabetes

First Author	Year Published	Follow-up time (years)	Age Range or Mean ± Std. Dev. (years)	Odds Ratios or Relative Risks for Developing Diabetes for Various Sleep Patterns		
				an average sleep time of 5 hours or less.	difficulty beginning sleep	fragmentation (difficulties maintaining sleep)
Kawakami	2004	8	not given	not assessed	2.98 (1.36-6.53)	2.23 (1.08-4.61)
Nilsson	2004	14.8±2.4 (mean ± st.dev.)	44.5± 4	not assessed	1.52 (1.05-2.2)**	not assessed
Mallon	2005	12	45-65	2.8 (1.1-7.3)	not assessed	4.8 (1.9-12.5)
Meisinger	2005	7.5	25-74	not assessed	non-significant results	1.60 (1.05-2.45) men 1.98 (1.20-3.29) women
Ayas	2003	10		Unadjusted 1.57 (1.28-1.92) Women adjusted 1.18 (.96-1.44) Symptomatic cases in women 1.34 (1.04-1.72)	not assessed	not assessed
Yaggi	2006	15	40-70	Unadjusted 2.59 (1.28-5.23) Not significant after adjustment 1.95 (.95-4.01) Also reported unadjusted results for 6 hours average sleep 1.91 (1.05-3.48)	not assessed	not assessed

To summarize, researchers studying the development of diabetes have found increased prevalence of diabetes among those experiencing shortened, and disrupted sleep. Evidence for a causal connection also comes from laboratory studies finding that acute sleep restriction negatively impacts glucose management. The action of the sympathetic nervous system seems to be a highly recurrent theme in the sleep-disturbance-related health effects and has been additionally linked to the action of leptin and ghrelin, the hunger regulating hormones believed to be important mediators in the development of sleep-disturbance-related obesity. Thus, the effects of noise on the action of the sympathetic nervous system during sleep

seem to be an important avenue for future research. The possible effect of a night of aircraft noise on metabolic variables should be a topic of future research.

### **4.3 Cardiovascular Nondipping**

Noise may affect health by acting to diminish the normal circadian decrease in blood pressure during nighttime. Possible intermediate mechanisms in such a relationship include stress and sleep disruption. Noise has been shown to lead to arousals, stage changes and fragmentation of sleep. These in turn may result in an impairment of the typical nighttime dip in blood pressure. Impairment of the nocturnal blood pressure decrease is predictive of cardiovascular and cerebrovascular mortality and morbidity. Some human relationships have been shown to influence dipping occurrence as well and perhaps ought to be included as confounders in future studies and analyzed for any modifying effects on outcomes.

#### **4.3.1 Nondipping and cardiovascular mortality and morbidity**

In most individuals, blood pressure declines between 10% and 20% during nighttime rest. Those who experience a dip larger than 20% are termed “extreme dippers”, while those experiencing decreases between 10 and 20% are termed “dippers” and those with blood pressures diminishing less than 10% are termed “nondippers”. More rarely, blood pressure may rise during nocturnal rest leading to an “inverted dipper” pattern. Cardiovascular nondipping has been seen as a predictor of future cardiovascular problems. These may include target organ damage (Kikuya, Ohkubo, Asayama, Metoki, Obara, Saito, Hashimoto, Totsumi, Hoshi, Satoh, Imai, 2004), left ventricular hypertrophy and cardiovascular mortality and morbidity.

Several recent studies have shown increased risks in those with a diminished nocturnal dip in blood pressure. For example: Ohkubo, et al. (2002) found a 20% increase in risk of cardiovascular mortality for each 5% decrease in nocturnal blood pressure decline. This was seen as independent of effects of blood pressure in general so that 24-h blood pressure and nocturnal decline in pressure emerged in the researchers’ analyses as distinct effects. Staessen et al. (1999) conducted a study of 808 patients with untreated hypertension randomizing some to treatment groups and others to placebo groups and conducting 24-h ambulatory blood pressure measurements. They found that nighttime blood pressure levels were better than daytime blood pressure levels as predictors of future cardiovascular mortality. They also found that a decreased day to night ratio of blood pressure increased cardiovascular risk; with a 10% increase in night-to-day ratio associated with a hazard rate for all cardiovascular end points of 1.41 (95% CI, 1.03-1.94), a 41% increase in risk for a 10% magnitude reduction in the nighttime dip. This result would seem to closely mirror the Ohkubo, et al. (2002) result (compare a 20% increase in risk for each 5% magnitude reduction in the nocturnal dip). The negative predictive effects of nondipping appear to be independent of hypertension as such. Verdecchia, et al. (1994) followed 1187 subjects for as many as 7.5 years. Their subjects included those with essential hypertension as well as normal control subjects. These subjects were further classified as either “dipping” or “nondipping” based on ambulatory blood pressure measurement. Increased risks were found in both nondippers and hypertensives with greatest risks seen in those experiencing both conditions with a relative risk of 6.26; (95% C.I. 1.92-20.32).

#### **4.3.2 The relationship between sleep structure and cardiovascular dipping**

Disruption of sleep can interfere with the normal nocturnal dip in blood pressure. Blood pressure measurements taken during temporary waking states or lighter stages of sleep are frequently higher than during sleep. Changes in physical activity patterns may also alter dipping/nondipping classification. Nevertheless, nondipping has been seen as a reliable measure. The lowest values of nocturnal blood pressure are generally encountered during stage-4 of non-REM sleep, corresponding to the deepest sleep of the night. Self-rated (using the Pittsburg Sleep Quality Index) poor sleep was also linked with decreased cardiovascular dipping. Frequent arousals as measured by the arousal index (average number of arousals per hour) can lead to a diminished nocturnal dip as can a decreased quantity of deeper sleep stages. Thus lighter and more disrupted sleep may diminish the nocturnal decline in blood pressure, an impairment (as discussed above) linked in epidemiological studies with increased negative health outcomes.

Lenz and Martinez (2007) studied ambulatory blood pressure in a group of 36 patients with suspected sleep apnea. The patients were monitored with polysomnography in order to determine actual sleep status during the blood pressure measurements. Blood pressure readings (both systolic and diastolic) were significantly increased during chance awakenings compared to during sleep (121±12 mmHg systolic blood pressure and 73±9 mmHg diastolic blood pressure during wake vs. 116±13 mmHg systolic blood pressure and 68±9 mmHg diastolic blood pressure during sleep). Thus, when measurements were taken during actual sleep the diagnoses of nocturnal hypertension or nondipping was significantly less likely than if measurements were taken during a random period (12 patients during sleep vs. 22 patients during random periods). Without further research, the prognostic implications of adjusting the ambulatory blood pressure for awakenings is somewhat unclear—would the nocturnal blood pressure with or without the awakenings or perhaps some weighted combination of the two constitute the “true” or more predictive value. Epidemiological studies into this question would be valuable in determining whether noise might have a relevant long-term effect on nocturnal blood pressure dipping and thus health. A similar question is addressed by Dimsdale, von Kanel, Profant, Melesen, Ancoli-Israel and Ziegler (2000). They examined the reliability of nocturnal blood pressure measurements taken on 17 subjects in 3 24-hr periods. They found that the results for dipping were significantly correlated across study nights and that this was true for multiple definitions of nighttime versus daytime. For instance they evaluated dipping as measured during the ‘clocktime’ defined nighttime period of 10 p.m. to 6 a.m. and compared that with ‘bedtime’, defined as when the subject reported being in bed and actual polysomnographically determined ‘sleeptime’, in which the subject was demonstrated to be physically asleep. They found that all three definitions were adequately reliable with some slight advantages to ‘bedtime’ or ‘sleeptime’ with no substantial differences between these two definitions due to low sleep latencies (subjects fell asleep within 8 minutes on average, which may be indicative of residual sleep debt, see, for example (Dement, 2000)).

Smolensky, Hermida, Catriotta and Portaluppi (2007) reviewed factors relating to cardiovascular dipping. They mention that the “deepest sleep stages, 3 and 4, coincide with the lowest blood pressure levels; whereas, the shallower sleep stages—1, 2 and REM sleep—correspond to relatively higher BP levels, although these remain lower than awake-state blood pressure levels.” Thus, preserving the structure of sleep—particularly its deeper stages—would seem to be important to the preserving of the nocturnal dip in blood pressure. Loredo, Ancoli-Israel and Dimsdale (2001) studied the relationship between sleep quality (determined through polysomnography) and nocturnal blood pressure dipping in 44 untreated sleep apnea patients. They found that the arousal index and monitored percentage of slow wave sleep each predicted about 10% of the variance in nocturnal blood pressure; results confirming the importance of sleep depth and continuity to the nocturnal decline in blood pressure. Loredo, Nelesen, Ancoli-Israel and Dimsdale (2004) studied the association between sleep quality and cardiovascular dipping in a sample of 62 self-designated normal subjects who had been screened for apnea and found well. Subjects were examined using polysomnography and ambulatory blood pressure measurement and divided into groups of dippers and nondippers. Sleep variables from the polysomnography were compared within the groups. The dippers experienced significantly more time in stage-4 slow-wave sleep and had a significantly lower arousal index. This finding for stage-4 sleep was particularly interesting in light of the relatively small amount of time spent in this stage in either group—14.3 minutes in the dipper group and 7.8 minutes in the nondipper group. This result appears to suggest the importance of preserving this deepest stage of sleep, particularly as it relates to normal blood pressure regulation. Matthews et al. (2008) studied the relationship between sleep/wake blood pressure ratio and sleep disturbance using two nights of polysomnography and ambulatory blood pressure as well as a longer term (9 day) sleep journal. Studied were 186 Caucasian and African-American men and women without known relevant cardiovascular, metabolic or sleep disorders. They found that increased sleep/wake blood pressure ratio was associated with more fragmented sleep, lighter sleep and more frequent arousals. No indications of differences in psychosocial stress were found between groups of dippers and nondippers. Although they did find that increased sleep/wake blood pressure ratio was associated with lighter sleep, it is not clear whether stage-4 sleep assumed the same prominence as in the Loredo et al. (2004) study because Matthews et al. combined stages-3 and -4 into a single deep sleep category. However, when Loredo et al. (2004) combined stages-3 and -4 in their study and examined the correlation with sleep/wake blood pressure ratios, the result was nonsignificant as well. This illustrates the

importance of a deep understanding and careful examination of the relevant sleep variables when studying the nondipping phenomenon.

Mansoor (2002) studied blood pressure dipping in its relationship with physical activity measured via wrist actigraphy. His population consisted of 52 hypertensive patients concurrently monitored for both blood pressure and activity and free of known sleep disorders. The nondippers displayed greater amounts of activity during their period in bed. Sleep latency was also longer in nondippers. O'Shea and Murphy (2000) studied the relationship between varying levels of daytime activity (measured by actigraphy) and dipping/nondipping status (measured with an ambulatory blood pressure measurement device). They found that subjects were more likely to demonstrate a dipping blood pressure profile when they had engaged in greater physical exertion during the day thus increasing their blood pressure during the active daytime period. The prognostic implication of such changes is somewhat unclear since the actual nighttime values were relatively unaffected but only the daytime values with which they were contrasted.

Yilmaz et al. (2007) evaluated 75 patients using ambulatory blood pressure and the Pittsburgh Sleep Quality Index (PSQI); these patients had been referred as having stage-1 hypertension. Scores greater than 5 on the PSQI (indicative of having poor sleep quality) were associated with a relative risk for having nondipping a nondipping blood pressure profile of 2.955 (95% C.I. 1.127-7.747). Thus, the risk of nondipping was essentially tripled among those with PSQI-measured poor sleep (a self-rating scale). Additionally, Backhaus, Junghans, Broocks, Riemann and Hohagen (2002) found significant correlations between the PSQI and sleep variables derived using polysomnography. A substantial reduction in physiological data nevertheless occurs leaving polysomnography with some advantages for sleep analyses. While polysomnography yields a large amount of detailed data, the Yilmaz et al. (2007) study nevertheless illustrates that nondipping may be predicted reasonably well by using the PSQI at much lower cost.

Thus, disruptions to sleep that increase arousals or fragmentation, or tend to promote lighter stages of sleep may lead to a nondipping blood pressure profile. If a nondipping blood pressure profile is responsible for the increases in mortality and morbidity seen in epidemiological studies, these may indicate a risk for long-term health associated with phenomena that disrupt sleep and thus lead to diminishment of the nocturnal blood pressure dip.

#### **4.3.3 Association between noise and sleep variables**

Noise has been shown to have a number of deleterious effects on sleep. These include difficulties falling asleep (Ohrstrom, 1995); see Flindell, Bullmore et al. (2000) for a contrary finding; disruption of sleep (see, for instance, Basner, Samel and Isermann, 2006 find that louder sounds may lead to longer term awakenings than quieter; changes in the structure of sleep including time spent in various sleep stages (Tasali, Leproult, Ehrmann, Van Cauter, 2008; Eberhardt and Axelsson, 1987); and possible decreases in the restorative qualities of sleep even in the absence of statistically significant stage changes (Basner, 2008; Bonnet and Arand 2003).

While most of these results are discussed in greater detail in the sleep section of the report, the results of Eberhardt and Axelsson (1987) are of special interest here. They studied changes in polysomnographic sleep parameters in subjects measured in their own home between nights with normal environmental noise and nights with window mounted sound insulation (resulting in a mean 8-dB decrease in levels). It was found that "the noise reductions caused an earlier onset and a prolonged duration of slow wave sleep" with stage 2-3 (the deepest of three subdivisions they made of stage-2 NREM sleep) reduced in the noise condition by 22% (5 minutes) and time spent in the combination of stages 3+4 decreased by 10% (8 minutes), this result just short of significance ( $p=0.06$ ) in their sample of seven males. In their population, which had been exposed to noise in their homes (where they were tested) for at least a year prior to the study, subject reactions to noise were seen which indicated that they had not yet habituated. The changed (decreased) noise levels affected the onset and duration of slow-wave sleep, showing improvements in these sleep parameters when the noise was reduced. Similar decreased magnitudes of slow-wave sleep were seen in non-dippers relative to dippers in the Loreda et al. (2004) study. A noise intervention study similar to this one using ambulatory blood pressure as well as polysomnography would enable us to understand whether

environmental noises such as those generated by aircraft indeed contribute to nondipping and help identify the role of sleep variables in mediating such a relationship.

While aircraft-noise induced nondipping has not been directly studied in an intervention paradigm as noise induced sleep changes have, acute effects of aircraft noise on sleep have been studied. Haralabidis et al. (2008) studied acute effects of aircraft noise on ambulatory blood pressure measured at 15-minuted intervals. They found that systolic and diastolic blood pressure both increased in 15-minute periods of time in which a noise event had occurred (+6.2 mmHg and +7.4 mmHg respectively).

A note on measurements of night blood pressure and the effects of these transient effects. It may be possible that a change in the level of such arousing stimuli via a change (intervention) in noise level would lead to a change in dipping behavior or at least dipping measurement. For instance, if a person had a blood pressure of 111-mmHg systolic blood pressure waking, then a (normal) 15% dip in blood pressure would give them a nocturnal systolic blood pressure of 94.35-mmHg. If, however, they experienced a noise event of the type encountered in the study, their systolic blood pressure would on average increase by 6.2-mmHg, giving them a nocturnal blood pressure of 100.55-mmHg. This would lead to a dip of 9.4%, which would be considered a nondipping systolic blood pressure profile. This elevation might typically continue for around 15-minutes values and would generally return to what they had been. Depending on when noise events and measurements occur this could lead to a determination of a nondipping blood pressure profile where a dipping profile may have otherwise occurred.

It is not immediately clear whether these short-term increases in nocturnal blood pressure are significant predictors of cardiovascular outcomes or whether they are less influential than the 'baseline' nocturnal blood pressure (the blood pressure as it might have been without the noise). However, some (including Haralabidis et al, 2008) have suggested that repeated arousals might lead to more permanent increases in blood pressure, or longer term acute elevation, as is seen in some waking industrial noise exposures. In the HYENA experiment both increases in short term blood pressure in response to acute noise exposure and longer-term blood pressure changes related to longer duration environmental noise exposure were observed thus somewhat supporting this hypothesis, though associations between the two were not reported.

#### **4.3.4 Effects of stress and social variables on dipping**

For some time researchers have investigated the association between human relationships and health outcomes. Supportive relationships have been seen as exerting a protective effect on cardiovascular health in longitudinal studies. Recently, researchers have investigated possible physiological pathways whereby relational variables may impact these health outcomes. A number of studies have employed ambulatory blood pressure in studying blood pressure effects of concurrent social interaction with individuals with whom the subject has an ongoing relationship. In these studies, event-dependent sampling was used to observe changes in blood pressure during social interaction and related them to the type and quality of the relationship to determine what significant associations may exist between cardiovascular arousal and relationship status and quality. Other researchers have looked at the association between relationship status or quality and nocturnal blood pressure dipping. Inasmuch as these variables lead to changes in patterns of cardiovascular dipping they may confer a protective or predispositive effect on a patient's risk associated with nondipping and its influence on cardiovascular outcomes.

Holt-Lundstad, Birmingham and Jones (2008) found a positive association between marital status and magnitude of cardiovascular dipping. Research by Holt-Lundstad, Jones and Brimingham (2009) similarly identified a favorable effect of relationship depth and low marital conflict on dipping. Linden, Klassen and Phillips (2008) found that healthy anger expression was associated with nocturnal blood pressure dipping. They found that nondippers may have a harder time letting go of angry feelings, and postulated that the way in which anger was processed influenced nocturnal blood pressure dipping. These researchers thus concluded that anger coping styles may be more important than current emotional state. Other researchers, somewhat unexpectedly, suggest that increases in level of stress may in fact also be associated with increased dipping (Fallo, Barzon, Rabbia, Navarrini, Conterno, Veglio, Cazzaro, Fava, Sonino, 2002). One might postulate that this is due perhaps to increased stress-related arousal during the day contrasting well

with a drop in pressure to levels that would still seem elevated by other standards. Additionally, those with blunted responses to daytime stimuli show an increased likelihood of nondipping blood pressure profile (Bishop, Pek, Ngau, 2006). While marriage has been found to be a protective factor for cardiovascular outcomes, stress is generally seen as a negative indication. Whether both of these factors' effects on blood pressure would have similar prognostic significance is somewhat unclear given the greatly contrasting nature of the stimuli (supportive versus possibly antagonistic). Of interest to us is whether these variables (stress and human relationships) might in some way affect sleep, sensitivity to aircraft-noise induced arousal, or in some other way buffer or enhance stress reactions to aircraft noise during the day. Current research, for example that of Holt-Lundstad et al. (2009) has employed the Pittsburg Sleep Quality Index (PSQI) in the context of relational cardiovascular dipping studies. The PSQI has been significantly related in previous studies to cardiovascular dipping as indicated above (Yilmaz et al., 2007) and to polysomnographically derived variables (Backhaus et al., 2002). However, associations between relational status and polysomnographically measured sleep variables remain to be studied. A potential interaction between relational variables and responses to noise from aircraft during nighttime sleep may be worth investigating and could be accomplished by partnering with researchers examining the relationship between sleep variables and relationships status or quality.

#### **4.4 Hypertension**

Hypertension is an important mediator in the development of heart disease and because of its prognostic significance it is a valued measure of health. Hypertension has been defined by the World Health Organization (WHO, 2003) as a systolic blood pressure  $\geq 140$ -mmHg or a diastolic blood pressure  $\geq 90$ -mmHg and this definition is generally used for its diagnoses, although an alternative definition of doctor-diagnosed hypertension is also considered acceptable in several studies if the patients are concurrently using a prescribed medication to treat their condition. Researchers in the HYENA study, for example, took this approach. Studies have shown correlations between noise exposure from aircraft (HYENA, Rosenlund et al. 2002; van Kempen et al, 2002), and hypertension, and sleep restriction (Lusardi et al. 1996; Lusardi et al. 1999; Gottlieb et al. 2006; Gangwisch et al. 2006) or quality (Morrell et al. 2000; Ekstedt et al. 2004) and hypertension. In the HYENA study, a correlation was found between nighttime aircraft noise as well as 24-hr road traffic noise and the prevalence of hypertension after adjusting for confounders. Thus, results of both studies supporting plausible biological pathways for hypertension genesis resulting from noise and environmental studies observing its apparent occurrence have appeared—thus, a well-supported case seems to exist for the occurrence of an effect of noise on hypertension development, possibly mediated by sleep disruption. Please note that, in this section, both shorter and longer term changes in blood pressure in response to stimuli are noted. Generally when we speak of hypertension, a longer-term condition is meant. However, a continuing series of shorter elevation may lead to outcomes similar to longer-term elevations and have been seen as a possible risk factor for longer term elevations (Chang, Su, Lin, Jain, and Chan, 2007).

Researchers studying normotensive and hypertensive subjects with affected sleep found increased blood pressure the following day. Lusardi, Megellini, Preti, Zoppi, Derosa, and Fogari (1996) restricted subjects to 5 hrs in bed and found a significantly increased blood pressure the morning following sleep restriction to 5 hrs a night in bed of +4-mmHg in systolic blood pressure as well as an increase of 4 beats per minute in heart rate. In a second study, this time with hypertensive patients Lusardi, Zoppi, Preti, Pesce, Piazzam and Fogari (1999) followed a similar protocol and found that both systolic and diastolic blood pressure were elevated the following day along with heart rate: +7.1 mmHg, +4 mmHg, and +5.5 bpm respectively.

Also, in the second study (on the hypertensive patients) it was found, when comparing an ambulatory blood pressure measurement during the sleep deprived day to that of the normal day, that all patients who had previously been “dippers” all became “nondippers” upon sleep deprivation, the nocturnal fall in blood pressure being approximately half of its normal magnitude. These results, evidence of an effect of acute sleep restriction on hypertension, resemble findings on habitual sleep duration in a cross sectional study by Gottlieb, Redline, Nieto, Baldwin, Newman, Resnick, Punjabi, (2006). In this study, part of the Sleep-Heart Health Study, habitual restriction to  $< 6$  hrs, or  $\geq 6$ , but  $< 7$  hrs of sleep was associated with odds ratios for

prevalence of hypertension of 1.66 (95% C.I. 1.35 2.04) and 1.19 (95% C.I. 1.02 1.39), Subject time in bed greater than 7-8 hrs was also associated with increased risks of hypertension (though of lesser magnitude than that associated with the <6 hrs of sleep condition). These results persisted even after adjusting for apnea-hypopnea index, which should effectively eliminate the possibility of confounding by breathing disorders. Additionally, the finding of an increased prevalence of hypertension with decreased sleep duration in the population is supported by a longitudinal study by Gangwisch, Heymsfield, Boden-Albala, Buijs, Kreier, Pickering, Rundle, Zammit, Malaspina, (2006). In this study sleep duration at baseline was associated with an increased risk for development of hypertension in the 8 to 10 year follow-up period of 2.10 (95% C.I. 1.58 2.79) for those receiving a self-reported  $\leq 5$  hrs of sleep per night. The increase in risk remained significant even after adjusting for obesity and diabetes although it was somewhat attenuated by the adjustment. This was seen as being consistent with the expectations that these variables may mediate some part of the risk. This attenuation is similar in some respects to the finding of Ayas et al. (2003) mentioned earlier concerning diabetes risk with reduced sleep when adjusting for obesity that was mentioned earlier. Gangwisch et al. (2006) mention further the potential role of sympathetic activation in mediating the relationship between sleep restriction and hypertension. The role of sympathetic activation seems to be a major theme in health effects relating to sleep disturbance; particularly those linked with the metabolic syndrome effects—obesity, diabetes, hypertension, etc.

Effects similar to those from sleep restriction were seen in the case of individuals experiencing **sleep fragmentation** or those frequently aroused during sleep. Ekstedt, Akerstedt, and Soderstrom (2004) found that number of arousals was the best predictor of both systolic and diastolic blood pressure as well as a number of other health-related factors. Further, in a study by Morrell, Finn, Kim, Peppard, Badr, and Young (2000) the relationship between sleep fragmentation index (the number of awakenings or shifts to stage one sleep per hour), and blood pressure was examined and a change in sleep fragmentation index of +1 (one extra arousal per hour) was found to be associated with an increase in blood pressure of 0.62-mmHg. This led to an increase of 3.1 mmHg for a two standard deviation increase (increase in sleep fragmentation index of +5) in sleep fragmentation index. The relationship was not observable in the population with an apnoea/hypopnea index greater than one, highlighting the importance of taking into account sleep disordered breathing in any study where either sleep length or sleep continuity is a dependent variable or a potential confounder. This may be especially true of any population-based blood pressure survey, where sleep-disordered breathing often leads to an increase in blood pressure and so may be a potential confounder. Questions about sleep length, continuity and snoring should also be included in any future studies dealing with blood pressure and noise.

In terms of waking effects of noise, Chang, Su, Lin, Jain, and Chan (2007) found increased systemic vascular resistance during working and sleep in highly noise exposed automobile workers when compared to those workers exposed to low levels of **industrial noise** as well as other changes. They interpreted their results as being indicative of changes in vascular structure resulting from repeated noise-related arousal that may lead to increased blood pressure. A key point made by Chang et al. paper is that repeated arousals may have sustained long-term effects beyond those of the somewhat more studied immediate (transient) cardiac arousal.

**Road Traffic noise** may also affect the development of high blood pressure. A study in children by Regecova and Kelleroval (1995) showed increased systolic blood pressure and diastolic blood pressure among children at schools exposed to higher levels of traffic noise. Also noted in the study was a decrease in heart rate among the high exposure group. An effect was also seen for levels at a child's home, but it was smaller than the effect seen for levels at school. This study was noted in the Babisch meta-analysis (2006) as one of the few significant studies on blood pressure in children amidst a number of studies with more heterogeneous results. de Kluizenaar, Gansevoort, Miedema, and de Jong (2007) studied the effects of road noise at home on hypertension within a population. Before adjusting for confounders they found a significant relationship between road traffic noise and the self reported use of antihypertensive drugs. However, after adjustment the result was attenuated and not significant. An interesting question might be what part of the noise exposure took place during the night because, if a large part, some attenuation when adjusting for confounders such as body mass index might be expected since the sleep related health effects (such as

obesity) may cluster in the way previously described. However, lacking that information, this is a purely speculative comment. Other significant results were found for road noise exposure in the age group 45-55, with odds ratios of ~1.3 after adjustment for relevant confounders including particulate matter, a class of air pollutant. Relationships seemed to strengthen at higher levels of noise exposure adding some support to the validity of the result. These two studies, together with that of Chang (2007) mentioned earlier, bring out an interesting point: that depending on the lifestyle and daily patterns of the group in question different exposure environments may be more or less relevant to the development of elevated blood pressure. If sleep is a key issue in a particular group then it may be anticipated that home exposure would be the most important whereas if high daytime levels are key then the work environment may be a more significant contributor, or possibly school and the educational setting. Although the questions of misidentification of exposure have been addressed in some studies, see, for example, Haralabidis et al. (2008) for aircraft vs. other sources, and van Kempen, Kruize, Boshuizen, Ameling, Staatsen and de Hollander, (2002), for work versus home exposure, it seems to remain an underexplored issue, and one of key importance when trying to establish cause and effect relationships and the path between the two. The HEARTS (Health Effects and Risks of Transportation Systems) project seeks to address this issue by predicting the likely exposure of individuals to multiple risk factors in multiple microenvironments characteristic of their typical daily travels. They are thus able to generate a much more detailed exposure profile than is possible with a single number metric measurement at home or even a combination of several metric types. Using this approach in future health studies may help avoid misattribution of relevant sources and allow for more precisely assessed exposure in population based studies.

Rosenlund, Berglind, Pershagen, Jarup, and Bluhm (2001) studied the effects of aircraft noise on hypertension in a cross section of people in Stockholm Sweden. Some of the people were nearer the airport and so had higher exposure while others were more distant and so experienced lower noise exposure. After adjustment for various parameters, obtained from an associated survey, they found two significant associations carrying increased risk of hypertension: for those exposed to energy averaged levels of 55-dBA measured outdoors or more an odds ratio of 1.6 (95% CI 1-2.5) was associated with an increased risk of hypertension and for those exposed to maximum levels 72-dBA or more they found an odds ratio of 1.8 (95% CI 1.1-2.8). This raises the additional interesting question of which is the better noise exposure measure: averaged energy, which has been a traditional favorite, or maximum level, or some other measure such as number of events or some other parameter relating to the noise or the circumstances of exposure that has not been previously or fully considered. Van Kempen et al. (2002) examined a number of studies in their meta-analysis and estimated a relative risk of developing hypertension of 1.14 (1.01-1.29) for occupational noise and 1.26 (1.14-1.39) for aircraft noise per 5-dB increment in each. This disparity also brings out the need to determine to what extent the psychoacoustic characteristics of the noise—its sharpness, roughness, and others, as well as its level and whether indoor or outdoor levels should be considered—are relevant factors in the development of health effects.

As mentioned earlier, nighttime aircraft and twenty-four hour road noise have also been identified as risk factors for hypertension in the HYENA study. In this large multicenter study of hypertension incidence surrounding six major European airports, 4861 subjects aged 45 to 70 that had lived in an area for five or more years — important because of the presumed induction effect (an induction effect is where it takes a certain amount of time for a particular cause to begin to have notable effects) — were examined to determine hypertension occurrence, as well as check for several potential hypertension risk factors. The standard World Health Organization criteria for hypertension of 140-mmHg systolic blood pressure or 90-mmHg diastolic blood pressure was used along with use of hypertension medication in the presence of diagnoses of hypertension by a physician — so either measured or diagnosed and treated hypertension. Exposure assessment was based on predictive models of exposure at an individual's home and the choice of subjects was designed to be stratified so that individuals were located near the center of their exposure groups. The measures include  $L_{day}$ , the A-weighted time-averaged sound pressure level measured between 7:00 and 23:00 or between 6:00 and 22:00, depending on the city, and  $L_{night}$  the A-weighted sound pressure level time-averaged between 23:00 and 7:00 or between 22:00 and 6:00. After adjusting for a number of confounders, two significant relationships were found between hypertension and noise: Nighttime aircraft

and 24-hr road noise  $LA_{eq}$ . For nighttime aircraft noise, a 10-dB increase was associated with a significant increase in odds ratio of 1.14 (95% CI 1.01 1.29); this dose-response relationship is illustrated in Figure 5.

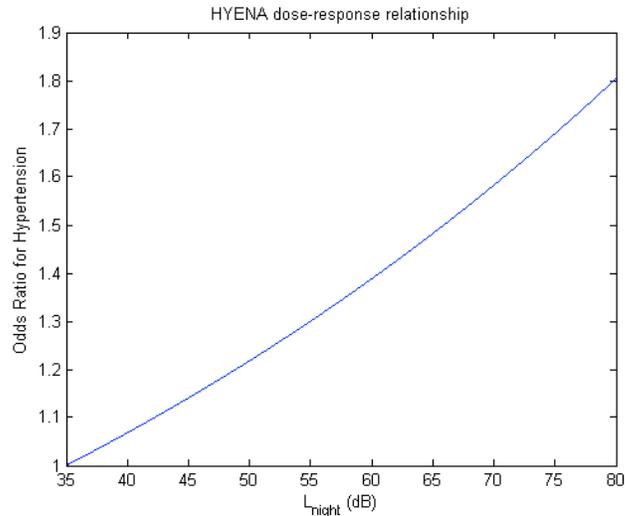
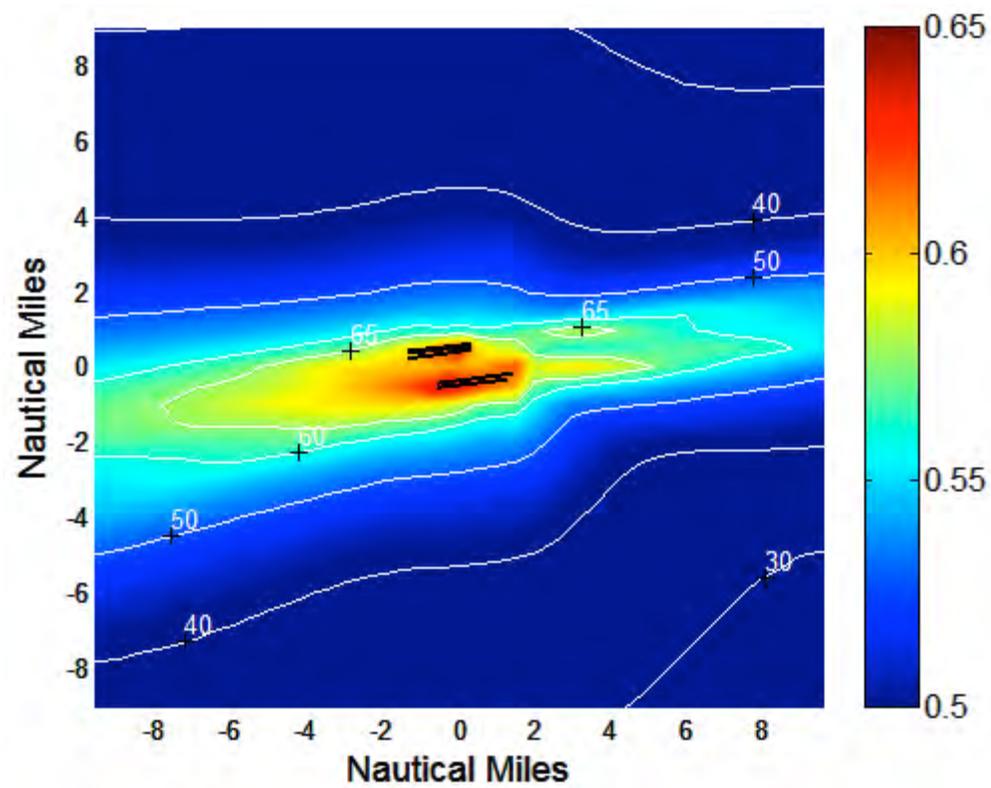
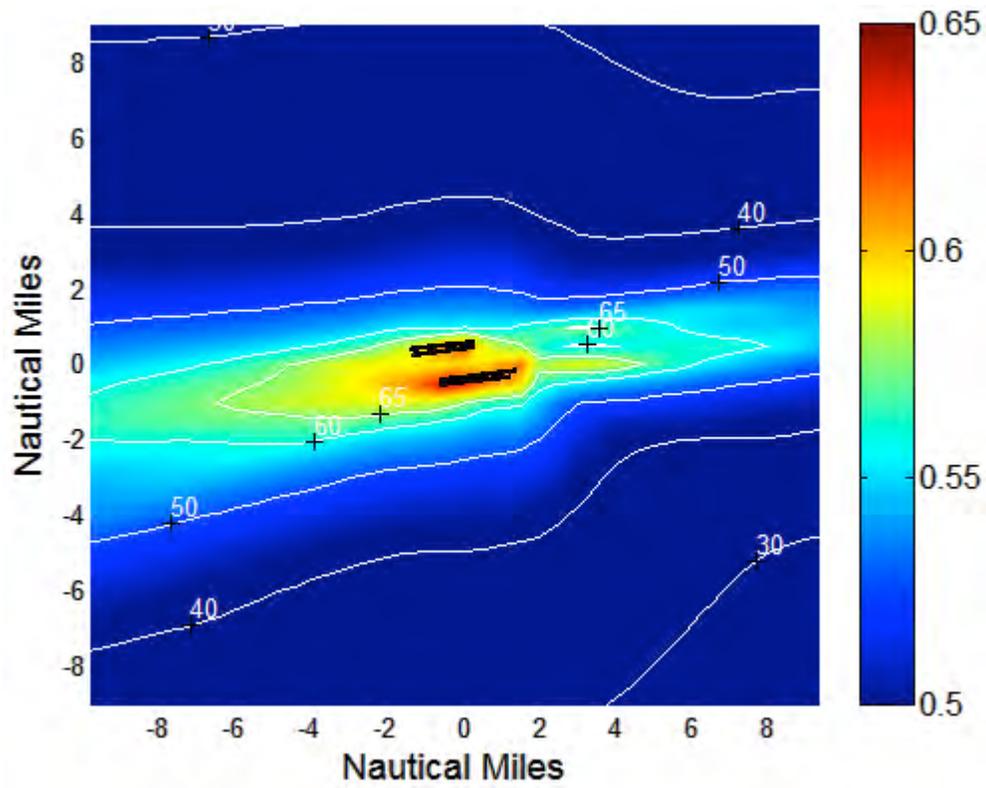


Figure 5: The dose response relationship from the HYENA project: a 10-dB increase leads to an elevated odds ratio of 1.14 (95% CI 1.01 1.29) and 35 dB(A) is assumed to be the baseline value. Subjects had lived in there > 5 years and ages of respondents were 45-70 years.

This relationship was used together with predictions of outdoor noise ( $L_{night}$ ) around Los Angeles International Airport to illustrate the potential impacts to produce a predicted hypertension prevalence map as shown in Figure 6. A further simulation was conducted to simulate the effect of doubling of nighttime noise. A third simulation was calculated simulating possible outcomes around Newark airport based on historical data. It should be noted that this recently identified model has not been validated through other studies. There are also questions as to whether the results are directly transferable from a collection of European populations to an American one. Hypertension occurrence for the population studied is assumed to be 50% and the baseline sound levels for  $L_{night}$  are assumed to be 35 dB(A).



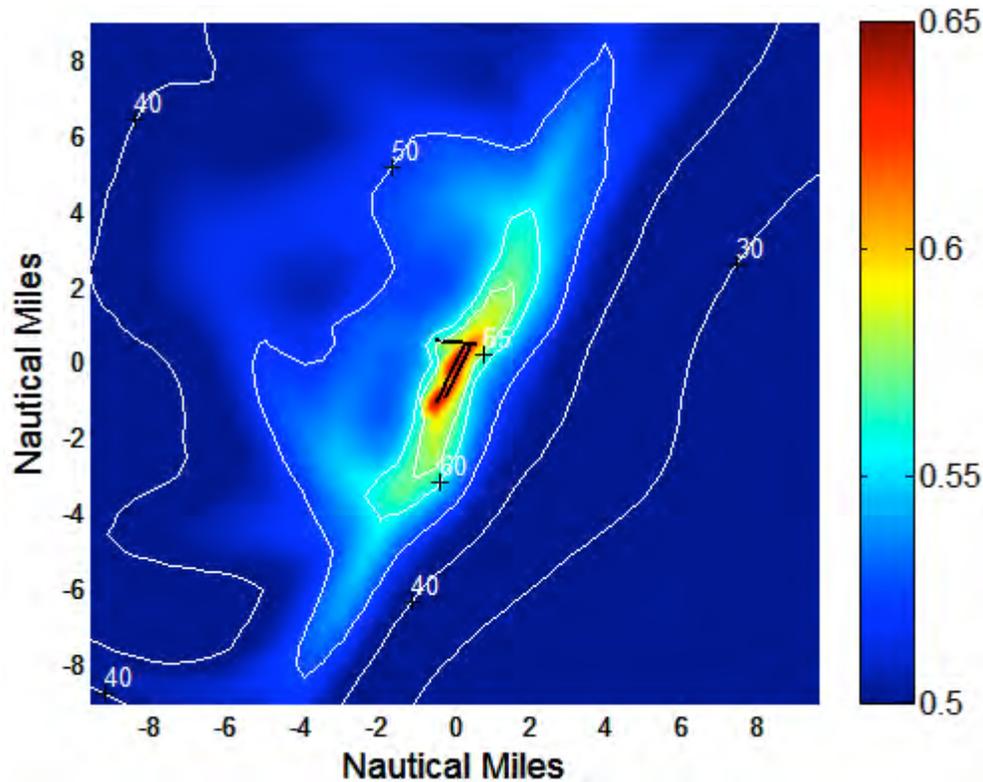


Figure 6: (Upper) An illustrative example of projected incidence rates per person of hypertension (for age group 45-70 years) around Los Angeles International Airport (LAX) based on the relationship in the HYENA report for  $L_{night}$  outdoors. Assumptions include a baseline incidence of hypertension of  $p = 0.5$  and a baseline (unexposed) level of sound of 35 dB(A)  $L_{night}$  outdoors. Also, there is an assumption in this analysis that a US population would respond in a manner similar to the Europeans in the HYENA study and that the dose-response relationship shown in Figure 5 is applicable, which may not be the case.  $DNL$  contours are also shown. Noise exposure was predicted by using INM and 1996 flight track data. (Middle) A second case using the same model and airport but simulating the effects of doubling the number of nighttime flights. (Lower) Using the same model but historical flightpath data from Newark International Airport.

For a 10-dB increase in exposure of men to road traffic noise an increase in odds ratio of 1.54 (95% CI: 0.99 - 2.40) was noted. Thus, effects were noted for both a condition where subjects were presumably sleeping much of the time and a condition where subjects were exposed in a wider variety of contexts. The night finding appears to support the view that arousal whether associated with sleep disruption, or, in absence of disruption, purely cardiovascular arousal during sleep may cause an excess risk of hypertension in a population. The road noise finding seems to support the view that repeated exposure throughout an entire 24-hr period may lead to longer term consequences possibly as a result of accumulated transient arousal effects as suggested by Chang et al. (2007) or similarly through stress as proposed by Tsatsoulis and Fountoulakis (2006). This may also illustrate a difference in the effects of exposure to different noise types, although this requires further investigation—in any event, the stratification of aircraft exposure seems to have allowed more effective differentiation between exposure types (aircraft versus road), an important HYENA advantage.

One particular advantage of more detailed noise exposure assessment seems then to be that it allows a more complete picture of the comparative magnitude of different causative factors in the progressive degradation of health. A main limitation in this study, which is common to many studies of this type, is, as

has been noted, that the noise was only assessed at subjects' homes, resulting in an incomplete picture of their exposure. Another possible weakness of the HYENA study, and other health effects of noise studies, is that it does not seem to have taken into account the potential confounding effects of habitual sleep duration or disorder. As has been illustrated previously, the sleep variables are important in the development of both obesity (a confounder in this study) and hypertension (the main measured variable). Thus, future studies should include questions on sleep variables as items in questionnaire or interview segments of the study. However, it should be noted that this omission would most likely have a conservative effect on results increasing the size of error bars and a correlation between sleep disruption by noise and obesity would act to systematically *reduce* the strength of observed relationships upon adjusting for this factor as a confounder. Thus, despite these shortcomings, the HYENA study in which significant relationships between 24-hr road noise and nighttime aircraft noise were in fact seen, is an important contribution and a major step forward in our understanding of the health effects of noise.

In conclusion, noise may possibly affect health either through exposure during the day or night. Transient arousals of the cardiovascular system during the day, such as those experienced in some industrial jobs as a result of noise, may lead to more permanent changes upon repeat exposure. Changes in the amount or continuity of nocturnal sleep, possibly acting through the arousal mechanisms of the sympathetic nervous system, may also lead to the development of hypertension. Future research needs to address further the relevance of differences in noise types and characteristics, explore more the question of the important exposure settings, and conclusively differentiate between the waking and sleeping effects of noise exposure. Further studies might also benefit from questionnaire items taking into account potential confounding by sleep disordered breathing as well as other nighttime noise exposure within the home.

#### **4.5 Heart Disease**

Recent studies have shown a possible connection between heart disease and noise. Notable among the research on this topic is the Babisch meta-analysis (2006). Although the combination of studies was not itself sufficient to produce a statistically significant dose-effect relationship due to large uncertainties, the evidence he presented, including the consequences of effect modification and other evidentiary matters, were deemed adequate to conclude that sufficient evidence now exists for a proposed relationship between noise exposure and ischemic heart disease. The non-significant dose-response relationship which he determined between these two variables in his meta-analysis — based on a number of studies with similar designs — is an important landmark in noise health effects research, and has proven influential. Babisch and others have used his dose-response curve to estimate the burden of noise-induced ischemic heart disease for Germany and Switzerland as well as Europe (WHO, 2005). Heart disease has also been significantly associated with noise exposure at home in populations (Babisch, 2003a), although not all studies have achieved either positive results or statistical significance.

Sleep disruption, which has been seen as an important health effect in its own right (Passchier-Vermeer and Passchier, 2000), and which may contribute to other outcomes, as discussed previously, may also be an avenue for the development of cardiovascular disease. This possibility is supported by the role of modifiers of nighttime noise exposure (Babisch 2006). This should not be surprising since short and/or fragmented sleep appear to lead to hypertension, obesity, and diabetes, several known risk factors for heart disease as well as, potentially, cardiovascular non-dipping. Increased incidence of heart disease has also been observed to be associated with short sleep in a population based study (Ayas et al, 2003). Similar to the risk of short sleep, fragmented sleep (as produced by restless leg syndrome and possibly by snoring) has also been associated with the development of heart disease (Winkelman et al, 2007; D'Alessandro et al, 1990). Those with a diminished nocturnal dip in blood pressure also experience an elevated risk of heart disease (Stolarz et al. 2002; Staessen et al, 1999; Verdecchia et al, 1994). Nondipping may also be associated with shortened sleep (Gangwisch, et al., 2006; Lusardi, et al., 1999; Matthews, et al., 2008) or fragmented sleep (Matthews, et al., 2008). Thus, a number of cardiovascular risk factors flank the path from noise exposure during sleep to ischemic heart disease. To summarize, these include especially obesity, diabetes, hypertension, and the potential diminishment of the amplitude of the nocturnal cardiovascular dip. Possible confounding with air pollutants is also discussed. Evidence also supports an effect of waking exposure to

high levels of noise in the workplace. Noise, in the waking context is thus seen as playing the role of a nonspecific stressor, leading to health outcomes through a variety of potential physiological and psychological pathways.

Hoffman, Moebus, Stang, Beck, Dragano, Mohlenkamp, Schmermund, Memmesheimer, Mann, Erbel, Jockel (2006) studied the relationship between nearness of residence to major roads (near defined as within 150 m) compared to those more distant. The study proceeded with an especial interest in the possible role of air pollutants in leading to cardiovascular disease—a potentially difficult confounder for noise studies as many of the same sources (e.g. cars and airplanes) produce both noise and chemical or material emissions. They did not however press their theoretical mechanism farther than their data justified. The extent of their conclusion was that there seemed to be an association between nearness to roads and coronary heart disease. The rough odds ratio for this was significant 1.62 (95% CI 1.12-2.34). After adjusting for cardiovascular risk factors this increased to an odds ratio of 1.85 (95% CI 1.21-2.84). Stronger effects were observed in men, with an odds ratio of 2.33 (95% CI 1.44-3.78), and those younger than sixty with an odds ratio of 2.67 (95% CI 1.24-5.74), and never-smokers odds ratio=2.72 (95% CI 1.40-5.29) probably a result of the fewer number of competing risk factors in these populations. Noise exposure was not assessed, and we can only assume that it was substantially elevated in the near-road subjects. However, because of the open question of the effects of pollution in this study and the cited sparsity of studies of this type with which to compare or with which to disambiguate the effects of particulate air pollution vs. noise pollution, interpretation of these results with regards to noise should be limited for now.

Tonne, Melly, Mittleman, Coull, Goldberg, and Schwartz (2007) likewise investigated the effects of long-term exposure to road traffic in their study as part of the Worcester Heart Attack Study. In this case-control study, cumulative traffic within 100m of a subject's home and distance from major roadways were used as the exposure variables and acute myocardial infarction as the response variable. In this study they found an increase in odds ratio for acute myocardial infarction of 5% (95% CI 3-6%) per kilometer nearer a major roadway. This study illustrates an effect of nearness to traffic increasing myocardial infarction risk essentially similar to that reported by Hoffman et al. (2006), and reminds us to reiterate the same point: that the twin variables of noise exposure and air pollution exposure need to be carefully disambiguated in order to properly estimate their respective effects on heart disease. This problem is mentioned and discussed in the Babisch meta-analysis (2006). He talks about the problem of confounding between noise and air pollution and finds that at least one study, that of Hoek, Brunekreef, Goldbohm, Fischer and van den Brandt (2002), who looked at both air pollution and road distance effects together, diminished the size of the effect attributable to the air pollution upon adjustment while the road distance per se remained relevant. An "unknown" confounder was named as the likely culprit, and Babisch pointed out that noise was the likely identity of that culprit. Nevertheless, this justifies additional careful study in order to disambiguate the effects attributable to noise and air pollution so that the correct mechanism may be addressed and the appropriate corrective action taken.

A significant increase in risk of myocardial infarction (heart attack) was also found from noise in an ecological study of Grazuleviciene, Lekaviciute, Mozgeris, Merkevicius, and Deikus (2004). Significant limitations existed within the study design, however, because many potential confounders were not assessed and noise was estimated by electoral district, which may allow large mismeasurement of individual exposure even at home. Additionally electoral district might realistically be associated with other lifestyle factors that might prove confounding. Thus, despite observing a significant increase in relative risk (RR) in the subgroup of men 55-64, RR=1.92 (95% CI 1.00-3.67), the strength of these results is considerably diminished by the important questions that remain unanswered.

In what amounts to a first of its kind study described by Selander, Nilsson, Bluhm, Rosenlund, Lindqvist, Nise and Pershagen (2009), the roles of exposure to road noise and air pollution were simultaneously analyzed. This study is an example of best research practices to date. They included annoyance variables from multiple sources including neighbor noise, a potential confounder of the transportation noise relationship. They also took into account noise exposure at work, although the quantification of this perhaps was not very detailed since it was evaluated using a survey. They took into account exposure occurring at multiple residential locations over time considering several different possible weighting schemes (i.e. time-

averaging the levels experienced at the residence vs. time-averaged energy at the residence). They observed that increased annoyance due to noise heard in the bedroom was a meaningful predictor of elevated risks. Job strain also appeared to have a modifying effect on myocardial infarction incidence consistent with a possible stress hypothesis. They found significantly increased odds ratios of 1.38 (95% CI 1.11-1.71) for myocardial infarction with road noise exposure in a subset not exposed to other noise types—this illustrates the potential importance of exact exposure classification, a feature lacking in many previous studies. They asked survey questions about neighbor noise but used predicted levels so improvement remains possible via more precise exposure assessment of neighborhood noise. They assessed exposure modifiers such as bedroom orientation using a survey questionnaire. This combination of best practices led to a significant result in the portion of the population that had the least exposure contamination from other noise sources. From this it is concluded that further advances in characterizing noise exposure precisely may reasonably produce further positive results. This study also provides a major evidentiary step forward in understanding the risk of cardiovascular effects due to transportation noise exposure.

In the Noise and Risk of Myocardial Infarction (NaRoMI) study a previous examination of heart disease with respect to noise in West Berlin showed a positive, though non-significant, association with heart disease in those living there 15 years or more. Accordingly, a further study with a larger population was initiated. Exposure was assessed through a mixture of modeling and measurement depending on the precise positioning and ease of prediction. Time-at-current-residence, an important potential modifier, was assessed, along with other suspected modifying and confounding factors. Positioning of houses within the 60 dBA contour of the airport was also noted to avoid source confounding. A questionnaire was administered assessing subjective noise exposure including exposure at work to adjust for possible confounding of exposure. Day averaged A-weighted sound pressure levels were used in their reporting of noise at home in lieu of more precise measures because day and night were found to be highly correlated and thus were treated interchangeably—this is frequently the case and so effects reported in terms of daytime levels may still be mediated through a nighttime effect, or vice-versa. Positive, but nonsignificant odds ratios were found in men in the highest noise category (>70 dBA) OR=1.27 (95% CI 0.88-1.84) versus men in the lowest ( $\leq$ 60 dBA), as well as men in the two highest noise categories (>65dBA) with OR=1.18 (95% CI 0.93-1.49). The finding was reversed in women and non-significant. In a sub-sample of men with  $\geq$ 10 years at present address, the odds ratio for men in the highest noise category was OR=1.81 (95% CI 1.02-3.21), which was statistically significant. Likewise when the upper two noise categories were combined in this subsample (>65dBA), the odds ratio was significant OR=1.45 (95% CI 1.03-2.05). When a sub-sample of those with 15-yrs of residence was examined the population size was too small to obtain statistical significance although the trend remained observable. Residence time seemed to increase the prevalence of effects in the higher noise categories but not do this in the lower noise categories. The idea of a lengthy induction time for effects to develop on the order of 10+ years is in keeping with the noise hypothesis (Babisch, 2006). Increased time in residence was seen to increase relationships between noise exposure and heart outcomes in the set of studies included in his meta-analysis (Caerphilly, Speedwell, and Berlin studies) (Babisch et al., 1999; Babisch et al., 1994).

Also of interest in this meta-analysis, road traffic noise annoyance at night was a significant factor for males OR=1.10 (95% CI 1.01-1.20) and aircraft noise annoyance at night was a significant factor for females OR=1.28 (95% CI 1.01-1.63). There seems to be a possibility that subject annoyance by transportation noise at night might be related to sleep disturbance related occurrences, but lacking further information this is speculative. Another important observation of the Babisch meta-analysis is the effects of nighttime noise. A number of findings mentioned in his compilation having to do with exposure modification support the hypothesis that sleep mediation plays a role in the genesis of cardiovascular health effects. For instance: the exposure in the bedroom during the night was found to be slightly more predictive of hypertension than the exposure of the living room during the night in the Spandau Health Survey (Maschke, 2003; Babisch, 2006). In that same study, sleeping with windows open was associated with a large increase in risk although the small sample size may limit the conclusiveness of the result. Similarly in the Tyrol study, which Babisch (2006) describes, window opening during the night but not the day was associated with higher blood pressure. This is especially interesting because daytime window opening not yielding an effect would seem unresponsive of a possible primary role of air-pollution, unless, of course, sleep is air-pollution sensitive in

some way. Additionally air-pollution might still play a main role if it were found to vary in a diurnal fashion leading to increased exposure at night. Additionally, in a second study conducted in Tyrol Austria of Lercher (1992) and summarized in Babisch (2006), switching the sleeping chamber from the bedroom to some less exposed room of the house led to decreases in blood pressure, pointing strongly to a role of sleep disturbance in the genesis of noise related blood pressure changes although issues of subjective control of the environment ought to also be investigated. Based on a collection of studies Babisch also found that subjective ratings of noise having to do with sleep disturbance and awakenings were more predictive of heart disease than subjective ratings that focused on non-sleep factors.

More overt evidence for effects on heart health through a sleep disturbance pathway have come from the study of sleep disorders such as obstructive sleep apnea syndrome and restless leg syndrome, and also from a population based study dealing with sleep duration and heart disease. In a study of restless leg suffering and cardiovascular disease, Winkelman, Shahar, Sharier, and Gottlieb (2007) found an increased incidence of cardiovascular disease with presence of restless leg symptoms at least 16 times per month for both cardiovascular disease OR=2.03 (95% CI 1.43-2.89) and coronary artery disease OR=1.98 (95% CI 1.36-2.88), which grew stronger with increasing severity of symptoms. This seems to follow the secondary symptoms trend set by sleep apnea, quite possibly the limiting model for all health effects attributable to or mediated through sleep. Because of the great severity of the sleep disruption effects associated with repeated obstruction of breathing during sleep, any health effect not seen in apnea patients is not likely to be associated with disrupted sleep—it thus serves as a limiting case health effects mediated by sleep disruption. As a representative model of noise interrupted sleep health effects, however, restless leg has a significant advantage over sleep apnea because, unlike apnea, restless leg does not carry risks of hypoxemia—lack of oxygen in the blood stream due to obstructed breathing. It does, however, like noise, lead to various types of arousal and sleep disruption. It may thus be possible to interpret this study of restless leg in terms of the effects of multiple arousals, and from this perspective it strengthens the case for an effect of sleep fragmentation on heart disease. The question of a nighttime noise related effect on heart disease then becomes a question of in what ways and to what extent does noise disrupt sleep or generate arousal.

The belief in a potential effect of sleep loss on heart disease genesis is similarly supported by a study by Ayas, White, Manson, Stampfer, Speizer, Malhotra, and Hu (2003). This study, carried out with women subjects as part of the Nurses' Health Study, followed a population of women ages 45-65 in the nursing profession for a period of ten years. Subjects filled out a mailed questionnaire beginning in 1986 and repeated biannually until 1996; thus, sleep duration was self-reported. Incidents of coronary heart disease in each sleep duration grouping were noted and compared at the end of the period of observation. Those with restricted sleep experienced an increased risk of heart disease compared to those receiving 8-hrs of sleep. Those receiving five or fewer hours of sleep had a significantly increased relative risk of 1.82 (95% CI 1.34-2.41). Those sleeping between 5 and 6-hrs had a relative risk of 1.30 (95% CI 1.08-1.57), which was significant as well. The elevation for 7 and 8 hours of sleep was very slight and nonsignificant. Sleep duration greater than 9 hours was also associated with significant increased risk of 1.57 (95% CI 1.18-1.26), which was a source of puzzlement to the authors since no biological mechanism for the increased risk in longer sleepers was apparent. The results for short sleepers appear to mirror results seen in previously mentioned laboratory and observational studies dealing with the effects of sleep duration and fragmentation on the development of obesity, diabetes, and hypertension—several important cardiovascular risk factors. Combined with the previously cited instances of modification of health effects by factors likely to affect sleep (open windows, room orientation, sleeping chamber location etc), both a proximate cause (noise related sleep disturbance) and apparent evidence of effects of nighttime noise seems to exist, and with them a plausible case for an effect on heart disease genesis mediated by nighttime noise through sleep disturbance.

Daytime noise appears to also play a potential role in the development of heart disease. Evidence for this comes from studies in an industrial or work setting. For instance, in a 2005 study of current and former blue-collar lumber mill workers in British Columbia with at least one year's employment, increased cardiovascular mortality risk was observed in both the whole cohort, and especially in a subgroup that did not employ

hearing protection, for which the relative risk was 1.5 (95% CI 1.1-2.2). The greatest increases in risk of cardiovascular mortality were seen in current employees and this together with the other findings was taken as supportive of both acute and longer-term effects of exposure to noise (Davies, Teschke, Kennedy, Hodgson, Hertzman, Demers, 2005).

In a case-control study of 395 myocardial infarction patients and 2148 controls, relative risk of myocardial infarction (heart attack) was found to increase significantly with increased noise exposure in the workplace. Noise exposure was measured in this study as a subjective comparison (by the subjects completing questionnaires) of the levels at work to those produced by several familiar noise sources representing differing exposure levels. The “population attributable risk” (the portion of illness in the sick population for which the workplace noise was responsible) was 27% after adjustment for social class, suggesting that 27% of the observed myocardial infarction in the sample was attributable to noise. This placed work noise as the second greatest factor for myocardial infarction (after smoking) and was seen by the authors as being unexpectedly high. Noise was believed to operate as a “nonspecific stressor” acting through various physiological channels to produce decreases in health (Ising, Babisch, Kruppa, Lindthammer, Wiens, 1997).

In conclusion, evidence from industrial studies supports a potential role of noise exposure in the genesis of some instances of heart disease. Researchers have also demonstrated what seems to be increased incidence of heart disease as a result of environmental noise exposure. Evidence by way of the modifiers of exposure in Babisch’s meta-analysis (Babisch, 2006) supports a role of nighttime noise in the genesis of these health effects. A number of studies support the hypothesis that disruption of sleep of various types may lead to increased incidence of both heart disease risk factors and heart disease itself. Although not ultimately significant in the statistical sense, Babisch’s exposure-response relationship has been used extensively to evaluate the burden of disease due to transportation noise (see, for example, WHO 2005). The recent report of Berry and Flindell (2009) is generally accepting of Babisch’s exposure-response relationship and concludes that his statistical meta-analysis relied on sound practices. They noted cardiovascular disease as one of two direct health effects of environmental noise, the other being hearing impairment, concluded that it was an objective endpoint. They concluded that the Babisch dose-response relationship provides “an adequate basis for a methodology to value health effects.”

As an example of the magnitude of effects that would be predicted by using Babisch’s myocardial infarction dose-response relationship consider Figures 7 and 8, below. The effects predicted by his relationship have been evaluated by first using an INM simulation to *predict*  $L_{day}$  for the Los Angeles International Airport (LAX) and then applying the formula from page 53 of Babisch (2006):

$$OR = 1.629657 - 0.000613 L_{day}^2 + 0.000007356734623455 L_{day}^3, \quad L_{day} > 55 \text{ dB(A)}, \quad (2)$$

and assuming a baseline prevalence of heart disease at 2% of the population

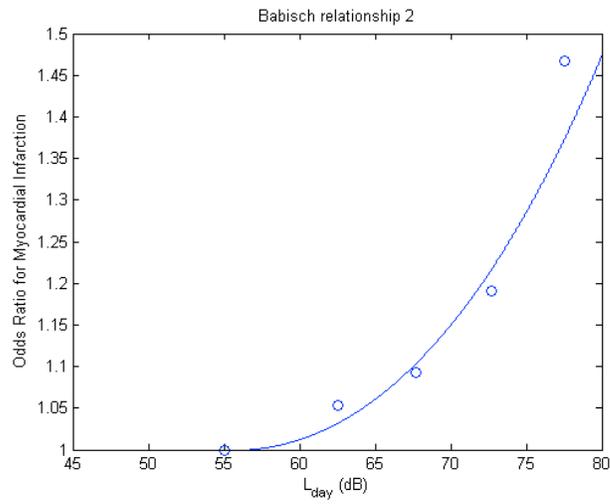
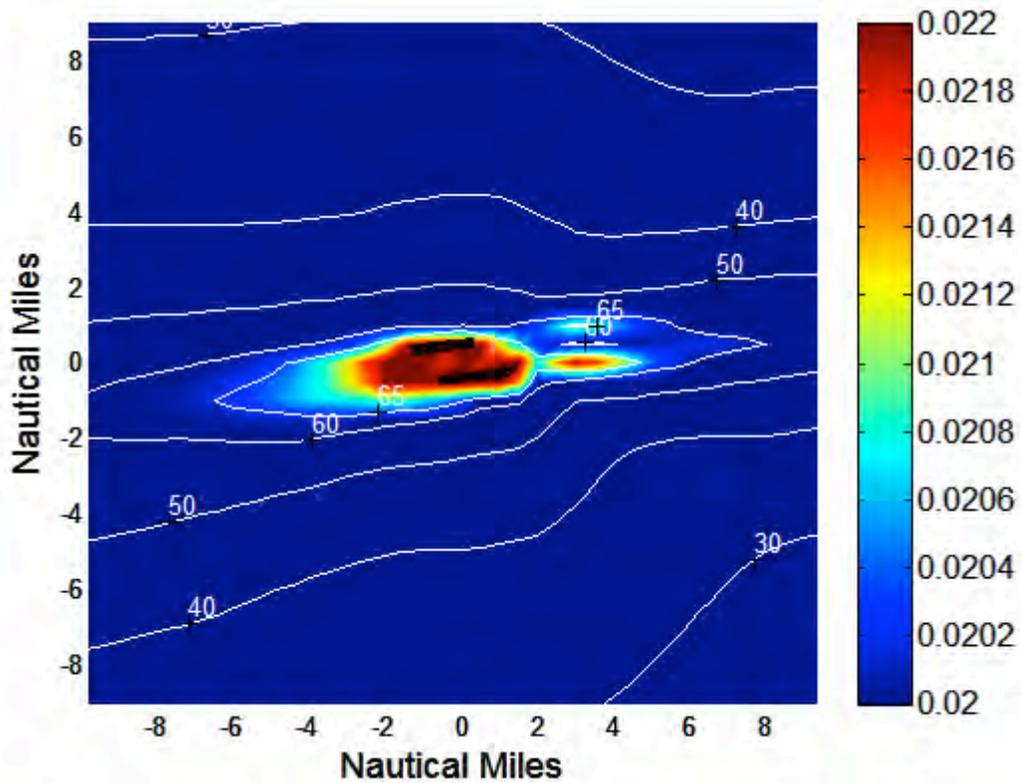
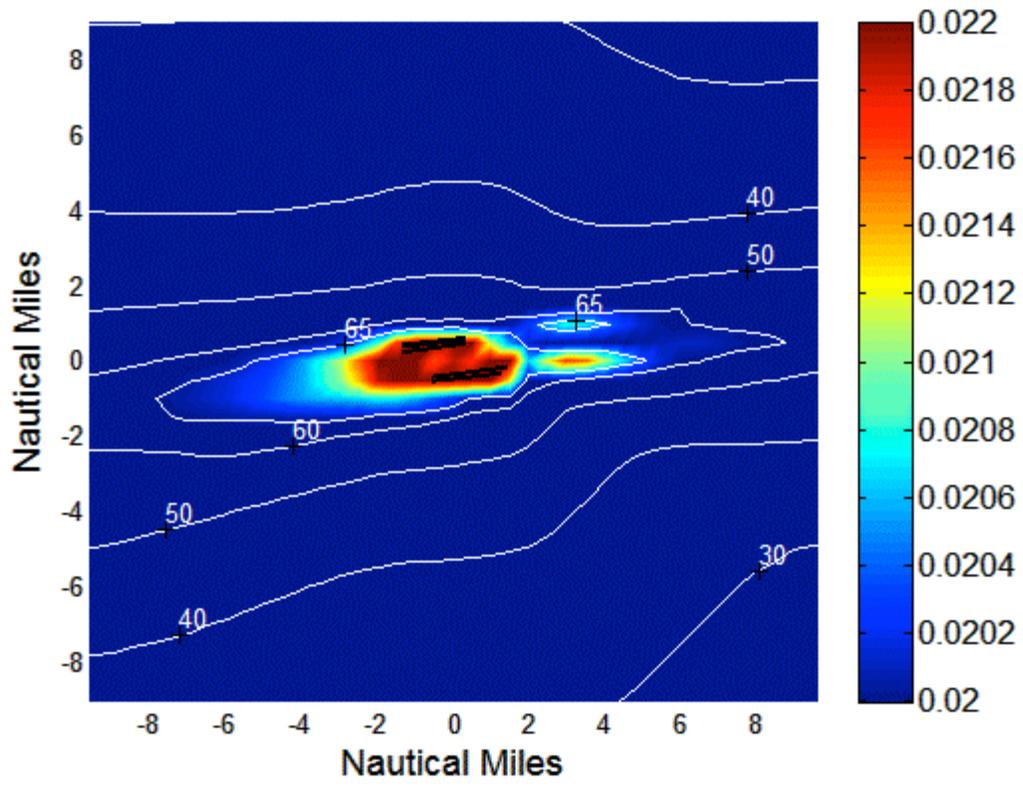


Figure 7: Polynomial curve fit based on Babisch's weighted meta-analysis dose-response relationship taken from pg. 53 of (Babisch, 2006). An odds ratio of one indicates no observed effect, the polynomial approximation starts at the baseline value of  $L_{day}$  of 55 dB(A). The data points that he was fitting to are also plotted.





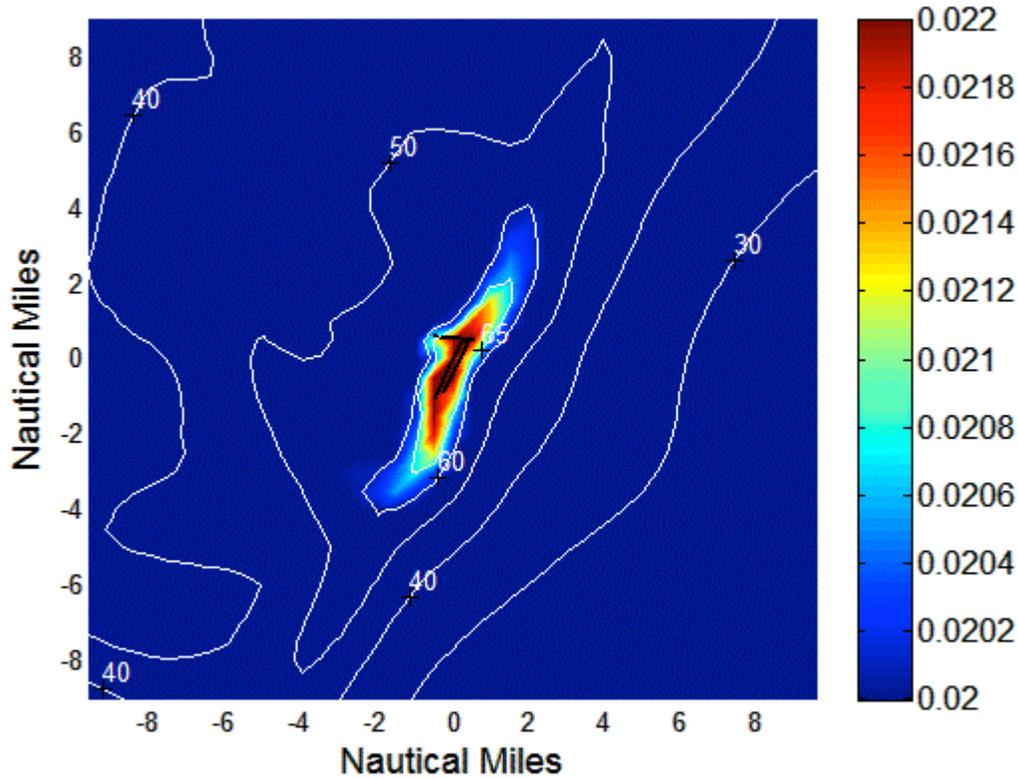


Figure 8: (Upper) Illustrative example of predicted incidence of myocardial infarction around LAX based on the Babisch (2006) meta-analysis dose-response relationship shown in Figure 7. Note that this relationship was developed primarily from road-traffic noise studies, and thus may not be appropriate for evaluating the impact of aircraft noise. Also, there is the assumption that health risk factors are distributed in the population around LAX in the same way that they were for the populations in the studies that produced the data on which the dose-response relationship was derived. The prevalence in the population without noise was assumed to be  $p=0.02$ . Contours shown are *DNL* contours. The noise exposure predictions are based on an INM simulation using 1996 flight track data. (Middle) A second case using the same model and airport but simulating the effects of doubling the number of nighttime flights. (Lower) Using the same model but historical flightpath data from Newark International Airport.

## **5 OTHER HEALTH & WELFARE EFFECTS OF NOISE / SLEEP DISRUPTION**

### **5.1 Annoyance and Noise Sensitivity**

Annoyance is one of the main effects historically examined in terms of population consequences of noise (Passchier-Vermeer and Passchier, 2000). Annoyance may be influenced by the qualities as well as the context of the sound in question. Annoyance responses may also be influenced by individual noise sensitivity. Noise sensitivity has generally been determined via survey questions. It has been assumed in some studies to be associated with awakening sensitivity, though this remains a questionable assumption. Remembered awakenings, however, have been seen as an important and much to be avoided source of annoyance and potential complaints. Noise sensitivity has further been seen as a potential modifier predicting reaction to noise in some studies, but in others has been viewed as a stable trait that affects individual reaction to noise independent of actual levels. This has led some researchers to inquire whether noise sensitive subjects selectively seek a quieter environment leaving a survivor population. Noise sensitivity also may be associated with trait anxiety and its related health consequences. Thus, caution should be exercised as noise sensitivity may co-vary with some health effects of interest independent of mediation by noise. A contrasting view is provided by the authors of the LARES study, an epidemiological study wherein relationships between noise annoyance and health outcomes were investigated. Annoyance has been covered extensively elsewhere and is not a primary focus of this review. For a review of this subject see Berglund and Lindvall (1995) or Passchier-Vermeer and Passchier (2000).

Annoyance by noise may be modified by attitude toward the sound sources and personal characteristics such as noise sensitivity (Miedema and Vos, 1999; Ouis, 2001). It may also be influenced by sound characteristics such as, e.g., loudness, sharpness, roughness or tonalness. The relative simplicity of most environmental noise assessment methods used in studies of annoyance do not allow for investigations of better noise quantification such as the sound characteristics listed above. However certain generalities do exist in what people find annoying. Speech and task interference, disruption of leisure activities or relaxation or sleep have been noted as potential sources of noise-related annoyance. However, strong contextual and individual differences also influence what constitutes an annoying sound to an individual. For instance a comparatively soft, non-sharp, stable sound such as HVAC noise may be seen as annoying when heard in company with sharper, louder, and significantly more variable musical instruments if it prevents a total silence from falling at a moment where that is an expected part of a musical experience. Thus, certain aspects of annoyance from noise may depend on non-acoustical factors (Fidell, Silvati, Haboly, 2002) and may be difficult to model.

One factor often believed to play a role in susceptibility to annoyance is “noise sensitivity”. Noise sensitivity has often been measured as a questionnaire item (Persson, Bjork, Ardo, Albin, Jakobsson, 2007), but has also been considered as a possible mediator in sleep disruption susceptibility. For instance, work done at the German Aerospace Institute (DLR) involving a polysomnographic study of sleep disturbance by aircraft noise in the laboratory and in the field, included a large population chosen to somewhat over-represent those with high questionnaire-determined noise sensitivity in order to allow a meaningful study of this potentially vulnerable group (Basner, Samel, Isermann, 2006). Increased awakening probability was found in the self-reported noise sensitive population during the laboratory portion of the study but not during the field portion of the study. Thus, there may be a relationship between noise sensitivity and awakening sensitivity, but in this study the relationship seems to depend on a modifying effect of environment, and does not appear in the more typical home setting. Thus, it is unclear whether noise sensitivity, as determined by a survey, is an adequate proxy for individual awakening probability. Subjects may therefore be sensitive to noise in one sense (annoyance) without necessarily being sensitive to it in others (awakening). An excessive individual sensitivity to noise at night resulting in increased awakenings, beyond that individual's natural tendency to awaken during the night for other causes, might constitute an increased risk for noise health effects. Thus, individual sensitivity to awakening by noise may be a modifier of the noise-health relationship,

whether or not it correlates with survey-measured noise sensitivity during the waking hours, and may also lead to increased annoyance if such awakenings are remembered. Recalled awakenings are a particular source of annoyance, and lead to diminished evaluations of sleep quality. Reductions of recalled awakening should therefore be addressed in noise policy decisions (Basner, Samel, Isermann, 2006).

In an attempt to verify a hypothesis that noise sensitivity mediated the annoyance response to noise, Van Kamp, Job, Hatfield, Haines, Stellato and Stansfeld (2004) investigated the relationship between exposure and response in those stratified into low, medium, and high noise sensitivity. In both high- and low-sensitivity groups, sensitivity appeared to act independent of noise level in determining annoyance as in an additive effect rather than a multiplicative effect of sensitivity. There was some correlation between noise exposure and annoyance in the middle group. The lack of correlation in the upper and lower groups and especially the upper group runs contrary to the idea of noise sensitivity influencing individual susceptibility to noise because of the unfulfilled expectation of stronger relationship between noise and annoyance in the highly sensitive group. Instead it seems to act independently of noise exposure to increase annoyance in all but the middle group. This also contrasts with the results of Miedema and Vos (2003) who found, in their meta-analysis, a modifying role of sensitivity beyond the additive effect seen here. Van Kamp et al. (2004) commenting on this finding observed that the difference may have been due to the single item appraisal of sensitivity used in the studies included in the meta-analysis.

One question which has been examined is whether noise sensitivity leads people to seek a quieter environment. This might lead to mostly noise insensitive people being left in a louder area after a prolonged period of time (a survivor population). This was investigated by Nijland, Hartemink, van Kamp and van Wee (2007) in a survey study of 300 households in a Dutch suburb chosen both for its noise stratification and fairly uniform socio-economic conditions. They found a correlation between high noise exposure and noise annoyance. No significant relationship between noise exposure and noise sensitivity was found. Thus, noise-sensitive people did not seem to selectively move to quieter areas. They gave as possible explanations for this the possibility that people are not aware of their own sensitivity, the possibility that noise sensitivity increases with residence time (although their study found no correlation between residence time and sensitivity), and the possibility that other pressures relating to obtaining housing (such as scarcity or cost) may have had greater weight in individuals housing decisions in the area studied. Additionally, noise-sensitive people were less satisfied with their dwellings and living environment regardless of whether they were in high or low-exposure areas and are almost twice as likely to want to move, however the effect does not appear to be modified by noise exposure.

In the Large Analysis and Review of European Housing and Health Status (LARES) study, noise annoyance from a variety of sources was measured via survey questionnaire along with a number of health endpoints. They pursued the view that noise annoyance might lead “through central nervous processes...to an inadequate neuro-endocrine reaction and finally to regulatory diseases.” Noise from air traffic was not the chief contributor and was lumped with other “traffic” noise including train and parking noise along with other automobile noise. In this study, focused as it was on housing-related factors and health, investigators looked at a number of potential confounders beyond those frequently examined. These included humidity and air quality in the residence, daylight in the residence, nearby green areas and temperature and heating during winter. Annoyance was measured by asking subjects how much, considering the previous twelve-month period, a given sound source has annoyed them while at home using the five-point International Congress on Biological Effects of Noise (ICBEN) scale from none to extremely. Increased risks were found with increased annoyance for many maladies. Included among these were respiratory, cardiovascular, emotional, and connective tissue diseases. Children seemed to experience primarily respiratory ailments in relation to noise annoyance and there was also a trend toward increased depression with increased annoyance. For non-elderly adults (18-59 years of age) there were significant associations between annoyance and multiple health endpoints. One unexpected result from this study was that in elderly people who were viewed as a potentially vulnerable group did not show many of the associations seen in younger subjects, instead showing significant increases with annoyance by traffic noise only for arthritis and stroke. One reason given for the absence of other expected associations was the increased baseline incidence of disease among the elderly population leading to a dilution effect. Another result of interest from this study was that

neighborhood noise was in many instances also a risk factor for disease. Neighborhood noise annoyance (which clearly cannot be easily predicted by tools designed to consider only air and ground traffic noise contributions) may be an important confounder to consider in future noise surveys. These results may be somewhat difficult to interpret inasmuch as there was not an actual noise measurement or prediction but only annoyance was recorded.

Using a trait anxiety scale included in a public survey with 2856 respondents, a correlation was found between trait anxiety in the study population and noise sensitivity. Thus, people who could be described as noise sensitive were more likely to also fit descriptions for neuroticism, trait anxiety, negative affectivity, or emotionality. Thus, it may be unwise to use annoyance as a proxy for exposure in health effects studies inasmuch as correlations may exist between annoyance and anxiety which may be more directly related to health matters (Persson, Bjork, Ardo, Albin, Jakobsson, 2007). For instance noise sensitivity was found to be significantly correlated with cardiovascular mortality in Finnish women but not Finnish men (Heinonen-Guzejev, Vuorinen, Mussalo-Rauhamaa, Heikkila, Koskenvuo, Kaprio, 2007). In a study of depressive symptoms in middle aged women Bromberger and Matthews (1996) found that women with trait anxiety had more depressive symptoms. Also they suggested that their results "raised the possibility of a reporting bias by neurotic women." They also mentioned the Bolger and Schilling (1991) finding that highly neurotic persons are more emotionally reactive to daily hassles. Thus, negative affectivity may very well precipitate "noise sensitivity", allowing annoyance to potentially be correlated with other health effects regardless of noise. Thus, care should be taken with its use in health effects of noise studies.

## **5.2 Possible Relationship Between Cardiovascular Outcomes and Annoyance**

A great deal of time has been spent addressing the health effect of noise as they may be mediated by noise exposure during sleep. The possibilities of exposure during waking hours will now be addressed. The concern with annoyance and the concern with health effects arising out of stress reactions in humans is in fact the dominant historical position in noise-health research. These stress reactions have often been seen as being related to a state of emotional annoyance at an intruding noise, though this is not always considered a prerequisite for the stress reaction.. For example, much of the explanation of potential pathways for noise to lead to cardiovascular disease given in (Babisch, 2006) relies on the concept of noise as a generalized stressor capable of affecting cardiovascular and hormonal conditions in the body. Implicit also in the focus of the LARES project is the assumption that noise→annoyance→health effects is a plausible pathway for health effects. In this same line of thinking, "noise sensitive" persons have been thought of by some as a potentially vulnerable group for annoyance-stress type effects. Babisch's comments in the Night Noise Guidelines for Europe chapter IV p. 64 (WHO,2009) support this noise to health effects via annoyance viewpoint: "It simplifies the cause-effect chain i.e.: sound - annoyance (noise) - physiological arousal (stress indicators) - (biological) risk factors - disease - and mortality." Thus, many authors see a stress reaction as the main mediator between noise and health effects.

Stress reactions involve a number of physiological pathways centering in the fight or flight response. For instance, Rozanski, Blumenthal and Kaplan (1999) while reviewing papers relating psychological factors to cardiovascular disease mention that, "Acute stress triggers myocardial ischemia, promotes arrhythmogenesis, stimulates platelet function, and increases blood viscosity through hemoconcentration." Cardiovascular to stress reactions may be elevated in some people and thus contribute to disease results. Stress may play a role in some sleep difficulties also making some of the connections in any proposed web of causality difficult to untangle. Additionally some psychological and interpersonal factors may play a role in mitigating health effects resulting through stress.

When an individual encounters a sufficiently stressful situation a number of things happen (primarily as reviewed by Tsatsoulis and Fountoulakis (2006)): increased secretion of glucocorticoids and catecholamines from the adrenal gland; the activation of the sympathetic nervous system; and resulting insulin resistance of the liver and skeletal muscles. Rozanski, Blumenthal and Kaplan (1999) review evidence relating coronary artery disease to psychosocial factors finding that "Acute stress triggers myocardial ischemia, promotes arrhythmogenesis, stimulates platelet function, and increases blood viscosity through hemoconcentration." While potentially advantageous in priming the body for physical action or hardship, many of these

adaptations become malicious when not followed by physical activity or in the absence of regular physical activity often seen in our current society (Tsatsoulis and Fountoulakis, 2006). Frequently repeated severe stress reactions are believed to lead to hypertension and heart disease. There is also believed to be some relationship between how quickly one recovers from an emotional stressor and resulting health outcomes.

Five main issues contemplated as modifiers of the relationship between stressors, stress and health are reactivity, recovery, anger, rumination and social support. The first of these, reactivity, refers to how strongly the body reacts to a stressor—i.e. how much heart rate, blood pressure, heart rate variability etc. are elevated. Small associations have been found between blood pressure reactivity to mental stress and increase in blood pressure over a five year period (Carroll, Ring, Hunt, Ford, Macintyre, 2003).

Cardiovascular reactivity refers to the degree or extent of reactions of the cardiovascular system to stress. These may take the form of increases in blood pressure, heart rate, variability of heart rate or changes in the high frequency content of the heart beat waveform. As the term is generally used it seems to indicate primarily the strength of an initial reaction to the stressor rather than the length or integrated magnitude of the reaction. Increased cardiovascular reactivity has been found to be modestly predictive of increases in blood pressure (Key, Campbell, Bacon, Gerin, 2008). For example, Carroll, Ring, Hunt, Ford and Macintyre (2003) reported a small but significant association between increased systolic blood pressure reactivity to a stress task and increased blood pressure at 5-yr follow-up. There may be some value in determining cardiovascular reactivity to a stress task in future prospective noise studies in order to determine if those with high cardiovascular reactivity to stress may represent a vulnerable population. It would be interesting to see how cardiovascular reactivity to a noise stimulus might interact with individual noise exposure in predicting potential prospective blood pressure changes.

Although initial reactions to a stressor may be similar among individuals this is no guarantee of similarity in either the duration or the degree of recovery among these same individuals. For instance recovery appears to be quickened among the physically fit whereas initial reactions may be of the same magnitude between fit and unfit subjects (Linden, Earle, Gerin, Christenfeld, 1997). Conditions of chronic stress may similarly increase reactivity and elongate recovery. Longer recovery times were observed in a group of subjects experiencing chronic stress as a result of crowded neighborhood conditions when exposed to a laboratory stress task. Increased blood pressure and heart rate reactivity were also seen in this group relative to a low chronic stress group in response to the task. In a similar research paradigm workers in a factory unwound more quickly from acute stress after their vacations relative to a measurement taken before their vacations; longer diastolic blood pressure recovery times in response to a cognitive challenge or physical exercise was also a significant predictor of hypertension development; and longer recovery was also seen in the obese (Linden, Earle, Gerin, Christenfeld, 1997). However causality is in question here—Tsatsoulis and Fountoulakis found that stress affects processes leading to the development of central adiposity. Thus, it may be the stress which caused the fat rather than vice versa (Linden, et al., 1997). Rumination and anger have also been suggested as exerting a possibly strong effect on recovery.

Rumination describes in humans the process of recalling and to some degree reliving past experiences and (particularly in this context) injuries. The word is used in both a trait sense—a somewhat constant tendency to thus recall and remember past events—and a state sense—being in a state of remembrance of past events at the time in question. Those with higher trait rumination have been found to be more likely to experience a future depressive episode and this trait has thus been suggested as a potential mediator in the relationship between stress and hypertension by acting to prolong the associated cardiovascular activation (Key, Campbell, Bacon, Gerin, 2008). Key, et al. (2008) studied trait and state rumination in undergraduate women and found that cardiovascular recovery (diastolic blood pressure and pulse variability returning to normal after an arousing (stressful) event) was delayed by state rumination in those who were low trait ruminators (not as likely to ruminate in general) when compared with low trait ruminators who were not actively thinking about stressful events (non-ruminators not currently ruminating). In high trait ruminators no difference in the response variables was seen between active and non-active rumination. High trait ruminators were found to be more likely than low trait ruminators to be ruminating five minutes into the recovery period but no significant association was seen ten minutes afterward. No association was found between trait rumination presence or absence and any of the reactivity variables. Significantly greater

diastolic blood pressure area under the curve (essentially an integration of diastolic blood pressure over a time interval which can ideally give a single-number-measure of total arousal occurring as a result of an event) was found in trait ruminators suggesting that they experienced inferior recovery relative to low trait ruminators. Rumination was linked to poorer heart-rate variability recovery in low trait ruminators indicating continuing sympathetic arousal. "Diastolic blood pressure reflects the flexibility of artery walls and has increased prognostic significance relative to systolic blood pressure in persons below the age of 45 (Malhorta and Townsend 2000)." Given the potential negative consequences of rumination on stressful events it has been proposed that distraction may provide an effective intervention. This may be especially true where the response elicits anger.

Subjects recover quickly from most laboratory stress tasks and return to baseline values within 1-2 minutes. Anger on the other hand elicits more lasting elevations lasting even as many as 10 minutes or longer (Linden, Earle, Gerin, Christenfeld, 1997). This may seem unexpected given that "based on the reactivity hypothesis, it would be expected that both positive and negative emotions would be equally related to hypertension, however only negative emotions have been systematically associated with sustained high blood pressure" (Key et al., 2008). Anger appears to be capable of producing a sustained cardiovascular elevation. Linden et al. (1997) further mention that, "Whenever cardiovascular recovery was slow there also was a magnified acute cortisol response and slow recovery suggesting anger-induced HPA-axis activity. Findings from these studies are entirely consistent with a massive body of epidemiological work suggesting that chronic anger and hostility may contribute to cardiovascular disease." Thus, sustained elevations of the cardiovascular system due to anger are a serious matter and a potential source of diminished health. To the extent the noise elicits anger responses in people it may be a threat to their health through these mechanisms. This perhaps gives additional weight and relevance to findings on annoyance that otherwise would seem to be an emotional rather than a physiological outcome.

One factor that seems to protect against stress mediated cardiovascular consequences is social support. Rodriguez, Burg, Meng, Pickering, Jin, Sacco, Boden-Albala, Homma, and Tullio (2008) found that social support was associated with decreased prevalence of cardiovascular nondipping. They described it as an important predictive effect. This may constitute a useful variable to account and adjust for in studies where cardiovascular outcomes are considered since it may eliminate yet another source of error in prediction. For instance high social support was associated with a 69% lower incidence of cardiovascular non-dipping. This may seem like a strange thing to mention in a health of effects of aircraft noise report, but the potentially high pay-off of strong social support in mitigating health-related outcomes makes it important to study. First, because it may be a potential confounding factor. Secondly, it may help explain differences in health outcomes in different situations. When attempting to study a health-influencing factor that may be of modest size compared to other factors influencing the same outcome, it is important to have detailed information on the other large-magnitude factors so that the researcher may be assured that any apparent association between the studied factor and the outcome is not, in fact, due to a non-random distribution of some other influential factor in the study population; the problem of confounding. Some potential social variables—education, income, amount and type of green spaces, cleanliness, etc; factors that influence how people interact and in what contexts—may vary strongly spatially and may have a significant correlation with exposure to environmental noise and influence the outcome variables. Controlling for such social variables thus becomes important in health effects of noise studies where nonrandom distribution of these influential factors may lead to spurious results.

In summary, stress has been seen as playing an important role in the genesis of cardiovascular disease and hypertension and is an important potential pathway in the relationship between noise and possible cardiovascular outcomes. The relationship between noise and stress may be mediated by factors such as cardiovascular reactivity, cardiovascular recovery, rumination on stressors, anger toward a potential stressor and social support. Thus, these variables should be accounted for in future noise studies.

### 5.3 Memory Decrements and Recall

While not a major focus of this particular report, a few words seem appropriate on the possible effects of noise from aircraft on memory formation and consolidation. Several possible questions with regards to memory are: How does noise affect an individual's ability to learn during instruction? How does a noisy learning environment affect later recall? Also, could disruption of sleep through aircraft noise affect sleep-mediated memory consolidation? For a good summation of the non-sleep-mediated part of the subject see the review by Passchier-Vermeer and Passchier (2000).

In a study of children exposed to road and aircraft noise in the school environment increases in sound exposure levels were associated with delays in reading comprehension of 1-2 months per 5-dB increment of noise with consistencies of effects but different magnitudes (different dose-response magnitude) by country. There was also a significant trend towards increasing difficulties in recognition memory with increased aircraft noise. Aircraft noise was not, on the other hand, associated with decreases in working memory, prospective memory, or sustained attention in the populations studied. An increased function of episodic memory was seen with regards to road noise, which was unexpected and was deemed to be of unknown origin (Stansfeld, Berglund, Lopez-Barrio, Fischer, Ohrstrom, Haines, Head, Hygge, van Kamp, Berry, 2005).

Hygge (2003) discusses the effect of transportation and speech noise sources on recall and recognition in children observed during an experiment in public schools. Diminished recall and recognition of a passage read by subjects in the presence of noise were observed relative to a passage of comparable difficulty read by subjects in the absence of noise. A larger effect was seen on recall items than on recognition items and the absolute magnitude of the noise effect decreased with item difficulty. Aircraft noise was found to be more impairing than road noise, which was more impairing than train noise paralleling the oft-quoted annoyance results of Miedema and Vos (1998). Effort and attention were unaffected by noise in this study. Thus, the immediate ability to learn new information in a form that is recallable later may be impaired by noise from aircraft or road transportation.

In a Federal Interagency Committee on Aviation Noise (FICAN) report (Eagan, Anderson, Nicholas, Horonjeff, Tivnan, 2004), it was reported that improvements were seen on both verbal and math/science standardized tests in schools that had recently experienced reduced exposure to aircraft noise either through noise insulation or contemporary airport closure. Airports were chosen in states with publicly available standardized test results. Aircraft noise exposure was determined by using the FAA's Integrated Noise Model computed for time periods when school was in session with reductions estimated for school construction type and insulation. All schools chosen were public schools. Demographic variables were included for possible confounding. Significant decreases were seen in standardized test failure rates among high schools after noise reduction. Trends for middle schools and elementary, however, were not as strong and were not significant. Some limitations of the study include: representativeness of the study as well as a lack of indoor noise measurements to precisely determine exposure. An observation made in this study was that those students most likely to fail were those that seemed to benefit the most from the noise reduction.

Some researchers studying the relationship between **sleep** and memory have found what they interpret to be roles for sleep stages, particularly slow-wave sleep and REM, in the **consolidation and enhancement of procedural memory**. Stickgold is a proponent of this theory and finds that the product of REM in the last quarter of the night and slow wave sleep in the first quarter of the night correlates with improvement in a visual discrimination task (Stickgold, Whidbee, Schirmer, Patel, Hobson, 2000). However, it remains controversial and a subject of debate: for a supporting review see Stickgold (2005), and for a rebutting review see Vertes and Siegel (2005). The rebutting review points out both that only certain types of memory are believed to be affected (procedural particularly) and that the effect seen (and the magnitude likely seen in more practical applications) may be of small magnitude compared with a practice effect, wherein a subject improves performance on a task over multiple trials. The studies mentioned deal exclusively with length and time in each sleep stage but do not address continuity issues apart from overall sleep structure changes (changes in total composition). It is not clear whether a noticeable effect on memory consolidation due to aircraft noise would be produced since the changes in sleep structure may be smaller than those examined

by Stickgold et al. (2000). Also it is unclear whether disruption by fragmentation would affect the outcome as this was not studied in these experiments.

#### **5.4 Performance Decreases/Speech Interference/Task Interference**

Several varieties of performance changes are possible with noise exposure beyond those discussed above for the learning environment. Sleep loss, fragmentation and stage structure changes during sleep may lead to both subjective and objective tiredness as mentioned above in the section on sleep disruption. Speech interference may also be a problem but is not a major focus of this review. See, e.g., Berglund and Lindvall (1995) for a review. Task interference is discussed in the WHO Community Noise Guidelines (WHO, 2000?), which follows closely the earlier review of Berglund and Lindvall (1995). Accidents and other impairment due to tiredness resulting from sleep disruption due to aircraft noise may be a consideration, although the effect size is likely to be comparatively small.

**Tiredness** may be a risk factor for accidents in work and automobile operation as well as diminished quality of service. For instance, Muecke (2005) concluded that effects of fatigue due to rotating shifts may negatively affect the quality of patient care in nursing. Lyznicki, Doege, Davis and Williams (1998) discuss the role of sleepiness in automobile accidents in their review. Bonnet and Arand (2003) review several studies finding similar decrements in those with short or fragmented sleep and those partaking of alcohol when measured on a driving task. Individuals may experience handicaps similar in magnitude to intoxication even on the first day of total sleep deprivation. A .1% BAC content led to levels of performance decrease seen at 18-20 hrs without sleep. Approximately (they say) what happens when someone is kept up until 4 am. The magnitude of the effect that could be expected from aircraft related disruption would necessarily be much smaller, however. Predine, Chau, Lorentz, Predine, Legras, Benamghar, Pierson, Guillame, Aptel and Mergel (2002) found that tiredness measured by survey was associated with 9.5% of accidents at school in a group of young adolescents studied. The data was collected via questionnaires filled out by the school's nurses who dealt with accident victims. Ilhan, Durukan, Aras, Turkcuoglu and Aygun (2006) found an increased prevalence of needle stick injuries (self) in nurses working more than 8 hours. In a survey of young American workers (the National Longitudinal Survey of Youth) (Dembe, Erickson, Delbos, Banks, 2005), working jobs with overtime shifts was associated with a 61% increase in hazard rate of accidents; working at least 12 hrs per day with a hazard rate of 37%; and working at least 60 hrs per week with a hazard rate of 23%. The injury rate (injuries per hour per hundred) was also seen to increase with the number of hours worked in a dose-response type relationship. The tiredness or sleepiness may clearly lead to accidents of various types. However, it should be noted that many of the studies noted deal with issues which would involve both sleepiness and mental fatigue associated with work; thus, the results should be scrutinized in that light.

As mentioned above in the section on sleep, objective sleepiness as measured by pupillography showed increases after nights of aircraft noise exposure in the lab (Basner, 2008). However this result differs from the findings of Flindell, et al. (2000) who for the most part found no association between laboratory noise exposure and multiple sleep latency test scores, which are generally well correlated with pupillographic measures of sleepiness as well as actual sleep debt. Flindell et al. also measured performance after a night of noise and found mixed results with some tests results yielding increases in performance and others decreases in the noise-exposed condition. Their findings did not seem to suggest that noise induced sleep effects on performance were of a sufficient magnitude to cause problems for performance in most cases.

**Speech interference** due to noise has been addressed extensively elsewhere and may be a source of both stress and accidents; it is particularly addressed in a review by Berglund and Lindvall (1995) who also address **task interference**. In this report, noise is mentioned as possibly contributing to errors at work, increased arousal leading to increases in concentration on simple tasks and possible decreases in effectiveness on complex tasks, results also reported by Proctor and Van Zandt (1994). An apparent tradeoff was noted in noisy conditions where subjects experienced faster mental processing at the expense of less available memory capacity (Hockey, 1979).

Uncontrollability of noise may also be an issue in its effects. Coping strategies may lead to accompanying detriments associated with effort expended in coping — by dissociation etc. It is fairly clear that disrupted speech may produce annoyance. This may be especially true when the speech is in a context where one would normally expect a quiet environment or where one is desiring relaxed leisure. Annoyance due to speech interference thus cannot be easily separated from the context in which it occurs. For a further discussion of the effects noise on speech interference as well as some of the annoyance issues involved, the interested reader is referred to (Berglund and Lindvall, 1995; WHO, 1999)

In summary, noise effects on sleep may lead to tiredness, which may lead to accidents. However, in most cases the tiredness effect of sleep disrupted by environmental noise seems to be of very small magnitude. Noise may directly disrupt communication leading to errors or stress. It may also increase annoyance, especially when it conflicts with an expectation of quiet or where hearing may be impaired such as in the elderly (Berglund and Lindvall, 1995). Noise may act to increase arousal, which may increase performance on some measures and decrease performance on other tasks depending, at least in part, on the task complexity as increases in arousal may also lead to decreases in cognitive ability to consider a greater number of factors (Proctor and Van Zandt, 1994).

## 6 POTENTIAL FUTURE RESEARCH

A great deal of research remains to be done in the field of health effects of noise. For instance, serious questions remain as to the most relevant exposure types and environments and the best metrics with which to evaluate these risks. During the day and night people are exposed to many different sources of noise in a variety of contexts. Distinguishing between the exposure-contributions of multiple environments and sources could allow researchers in a prospective epidemiological study to determine which exposures and contexts are most relevant for health. This information could then be used by policy makers to regulate development in a way optimally suited to balance risks and benefits of noise producing infrastructure.

While some research findings concerning hypertension and heart disease seem to support a possible role of noise disturbance through sleep, other potential outcomes of sleep disrupted by aircraft such as obesity and diabetes have not been specifically investigated. So, one topic for future research would be the laboratory investigation of the effects of a night or multiple nights of aircraft noise-disrupted sleep on the sympathetic nervous system, leptin and ghrelin signaling, and glucose management, as well as a further examination the known transient cardiovascular effects. The use of obesity as a potential confounder for hypertension and heart disease studies is clearly necessary due to its known relationship. However, obesity may co-vary with lost or disrupted sleep. Thus, attempts to adjust it out of models may in fact reduce the strength of a real effect of sleep disruption on adiposity as well as the confounding effect of non-sleep-disruption-related obesity. A more sophisticated means of adjustment may be needed (if such is indeed possible) to remove effects of obesity not due to noise-related sleep disruption from data without removing those that are.

With so many unanswered questions and limited resources, it is desirable to make studies as cost-effective as possible. Accordingly, methods for conducting HYENA-like surveys in the United States via the possible use of hospital admission records, CDC records or other potentially available preexisting data sets, has been investigated. This seems at first like a very appealing option because of the large numbers involved (contributing statistical power) and the potentially reduced cost if the data already exists in a usable form containing the necessary information. However, this process entails several formidable challenges. In order to aid the achievement of significance, studies like HYENA assessed noise using predictive methods at subjects' homes. Address information is potentially identifying and is likely to be difficult to obtain under HIPAA (the Health Insurance Portability and Accountability Act of 1996). Without address data of at least the street level, exposure assessment would be impossible. It is possible that an arrangement could be reached to perform the computation of subjects' community sound exposure on a computer located in the hospital without requiring identifying data to leave the facility at any time. This would however require a high degree of cooperation, and might still involve a lengthy approval process and strong safeguards against potentially divulging protected medical information. Additionally, this supposes that the hospital or likely hospitals in question keep patient data in a standardized format. Currently this does not seem to be the case in the United States, although electronic medical records are becoming more common and some legislative steps in facilitating standardization have taken place and this seems to be an item of interest in national health care policy. Though the future benefits of such innovations are promising, large scale implementation and use of standardized electronic medical records may yet be a long way off. Research at this point using existing medical records of multiple institutions surrounding several airports would potentially have to consider records of various paper and electronic formats.

Further challenges ensue. Researchers working on HYENA also had access to variables such as bedroom orientation and window opening habits, which may significantly modify nocturnal exposure, as well as other significant lifestyle variables and other potential confounders. As an example of the potential impacts of variables modifying exposure, a study by Flindell, et al. (2006) found that while levels in the high- and low-noise exposure groups in their study were stratified by an average difference of 5.6 dB L<sub>max</sub> measured outdoors, these differences decreased to only 0.2 dB (almost certainly less than a just noticeable difference in amplitude and probably less than the magnitude of routine measurement errors) when measured indoors. Additionally, they found an average difference of almost five decibels between the windows open and the windows closed condition. There was some indication seen in their study population that subjects in the high noise area were more likely to sleep with windows closed, thus diminishing the significance of an outdoor

measure that subjects might be acting to compensate. Also they observed that houses in the high noise area seemed to be better insulated against noise. Thus, knowledge of these variables (window opening, house orientation, location, insulation and construction, or most ideally actual indoor measurements in the bedroom) that increase the accuracy of noise exposure assessment appear to be a key feature in obtaining significant results. Subject window opening habits would likely be most easily obtained from the subjects directly and any study that lacks this information would be flawed. Another necessary feature that likely could not be obtained from medical records is subject time in current residence. Additionally, other sources of noise near the home may contribute to a subject's relevant noise exposure. For instance, researchers working on the LARES study found that neighbor noise was the second most prominent source of noise annoyance (Niemann, Bonnefoy, Braubach, Hecht, Machke, Rodrigues, Robbel, 2006). This may impose a fundamental limitation on the accuracy of any proposed level of noise exposure not based on an actual measurement at the house or in the bedroom. Researchers examining cardiovascular health effects of noise have suggested a role of induction time in potentiating these effects (Babisch, 2006). Participation in HYENA, for instance, required 5 or more years of elapsed time in current residence. Again this information would be most easily obtained from subject interviews or questionnaires, but both of these methods require active subject participation. Thus, the use of hospital records would have severe limitations in accuracy of exposure assessment including—difficulties obtaining exact address data and safeguarding it from misuse, a lack of data concerning house orientation, construction and window opening, and a lack of data concerning time in residence—any of which limitations might possibly be enough to lead to an inconclusive result by itself. If these were to be included in the interviews or admission documents at the hospital it could potentially become a valuable resource, however this would seem to require subjects consensual participation and be essentially a new study as such rather than a mere examination of records with many of the attending difficulties.

A second approach would be to partner with one of the large prospective studies dealing with the recognized effects of short or disrupted sleep—obesity, diabetes, hypertension, and heart disease. Many of these studies already account for the necessary confounders and many have large sample sizes. A number of studies of this type have been mentioned in this report. With the addition of noise exposure assessment some of these studies have features that would give them advantages in some areas over some of the best-designed cardiovascular effects of noise studies to date. One such feature is polysomnography — this allows researchers to control for the possible impacts of sleep disordered breathing (which may introduce multiple nighttime arousals and mask the effects of nighttime noise in affected subjects) while also yielding information on sleep structure, total sleep time and continuity. Problems with sleep continuity, duration and structure may contribute to some of the same outcomes proposed for aircraft noise and accounting for these potentially confounding factors should be looked upon as a possible avenue of improvement in future studies. While the detailed sleep structure and continuity data of polysomnography was not collected by all the researchers whose works have been reviewed, other methods such as sleep journals or subject interviews were nevertheless effective in determining typical sleep time, and by using them it was possible to get an indication of possible sleep-disordered breathing by asking if subjects had been told they snored. Inasmuch as sleep is an important mediator in the path from noise to cardiovascular outcomes, knowledge of variables which gauge sleep (whether polysomnographic or not) would allow researchers to account for possible confounding or modification of the relationship between noise and these outcomes by sleep. This might also allow researchers to determine if various sleep characteristics are important indicators of a nighttime noise vulnerable population. Another positive feature is the opportunity to focus on some of the outcomes of sleep loss or disruption that have not been a primary focus in the noise studies—obesity and diabetes. A great deal could be gained by partnering with a prospective study that was looking at health effects of sleep loss over time.

Potential disadvantages of partnering with either existing or future studies might include increased difficulties in selecting the population. For instance a five-year time in residence might exclude many participants in a mobile society. However, this may also be an advantage as the possible effects on people of varying levels of noise exposure in multiple residences over a period of time is an area needing further study. This might help answer the question: How does previous environmental exposure modify or accumulate with the effects of current environmental exposure? In an increasingly mobile society this is an important consideration for

policy-makers when assessing the needs of, and impacts on, individuals who may not live in an area long enough to experience the proposed long-term effects. Other difficulties in partnering with other studies might include specialized non-representative populations being studied (e.g., only nurses), noise exposure that was not well stratified, locations not near airports (where aircraft noise might not be the dominant or even a significant contributor) or difficulties with sample size, among others. Nevertheless, the prospect of being able to study noise health effects with comparatively little additional effort is appealing, and many of these challenges could be overcome through a long-term strategy involving multiple research partners with similar research questions. While the idea presented here is to partner with future prospective studies so that the desired data may be collected from the beginning, it may also be possible to do this after the fact though this might be more difficult as it might represent a change in study protocol. Many of these studies already have address information in their possession and noise exposures could be paired with houses in a blinded manner (so as to prevent disclosure of any sensitive information), but the same exposure modifier problems would persist as mentioned in the discussion of hospital use data (above): unknown house orientation, effects of insulation, and window opening habits.

While less economical, large scale prospective studies may provide a much higher degrees of control over types and quality of the data collected and, with that, better statistical control over potential confounders. Future studies of the health effects of noise, especially those considering nighttime noise, should take into account habitual sleep duration, sleep quality, and potential sleep disorders in addition to those factors which have been accounted for in previous well-designed studies such as HYENA. Additionally statistical modeling of populations may play an important (and potentially very cost-effective) role in determining which health outcomes should receive first priority for future investigation (particularly in terms of those outcomes that have not yet received primary examination in a large noise study). Future large noise-health studies may be done somewhat differently than in the past. In Europe, the HEARTS (Health Effects and Risks of Transport Systems) methodology allows for the assessment of detailed individual exposure to multiple environmental effects occurring over entire modeled days of routine activity. An individual subject's daily routine could be modeled as they progress from home to work and eventually return and sleep. This type of detailed projected exposure information may allow comparison of the importance of different portions of an individual's noise exposure and thus answer the important question of relative importance of different exposure environments (home vs. work vs. leisure vs. sleep etc). A similar benefit might be gained from using subject routine tracking through GPS. In a study by Wiehe, Carroll, Liu, Haberkorn, Hoch, Wilson, and Fortenberry (2008), GPS enabled cell phones were used to track adolescent girls for a period of one week. Their study was designed to assess the feasibility of using this method to study the relationship between risky and health-detrimental behavior and location and context. The cell phones they used determined subject location every five minutes and transmitted it to a secure database. They found that those who participated not only did not object to the monitoring but were interested in participating in potential follow-up studies, in part because of the incentive power of the free and fairly sophisticated phone. It is unclear how well this set of motivations might map to adults who might potentially be more resistant to any apparent potential infringement of their private lives. One reasonable question concerning this issue might be whether such information would be protected from court authority or whether it could possibly be subpoenaed leading to potential embarrassment or culpability. In the study mentioned above a Certificate of Confidentiality was obtained from the Department of Health, which protects subjects from having the data obtained in the study disclosed in legal proceedings or otherwise except for rare cases where a life might be saved by its disclosure. As might be expected, strong data protection techniques were employed in this study including electronic and physical protection of data. Any similar study would have to carefully consider how to adequately protect individual information.

Possibly the limiting case of accurate exposure assessment might be obtained via 24-hr noise dosimetry. This would preferably occur using a device that could transmit noise exposure measurements similarly to what was done in the cell phone/GPS study and would need to be used for a long period of time. Though attributing the noise to particular sources might be challenging, the raw magnitude information could perhaps be coupled with location information to infer the exposure type as well as directly observe the magnitude. The power of such a study increases yet again when coupled with the use of ambulatory blood pressure monitoring. Many subjects and businesses, however, might find such monitoring of either noise or

blood pressure exposure overly intrusive and potentially unwelcome. Preliminary studies addressing what types of people would be willing to participate would be needed to eliminate the possibility of a non-representative population arising through selection biases having to do with willingness to be tracked.

A recent study described by Selander, Nilsson, Bluhm, Rosenlund, Lindqvist, Nise and Pershagen (2009), illustrates many of the best practices and improved techniques described in this section. This is one of the few studies wherein exposures to road, noise and air pollution were simultaneously analyzed. Annoyance variables from multiple sources were measured via questionnaire and included neighbor noise, identified as a potentially serious concern in the LARES study. Inability to take account of neighbor noise has been a major blind spot of studies conducted using noise prediction programs such as INM, which of course do not predict noise emission by neighbors, e.g. children playing, car and house doors slamming, lawnmowers, etc. Selander and coworkers took into account noise exposure at work, although perhaps not at the desired level of detail. They took into account exposure occurring at multiple residences over time with different possible weighting schemes. They also took into account other important potential confounders such as job strain, which also appeared to have a modifying effect consistent with a possible stress hypothesis. They found significantly increased odds ratios for myocardial infarction with road noise exposure (1.38; 95% CI 1.11-1.71) in a subset not exposed to other noise types. This illustrates the potential importance of exact exposure classification and seems to indicate it as a primary improvement that is possible in this field of research. Additionally neighbor noise was assessed in this study via survey questions and noise exposure was determined by prediction; more precise exposure could have been accomplished via actual measurement. They assessed exposure modifiers such as bedroom orientation using a survey questionnaire, an essential feature as indicated above. Overall, it is a very well-constructed study leading to a significant result.

Future research needs to build upon the foundation of increasingly careful and methodical research which has been conducted in investigating the health impacts of aircraft noise exposure through a prospective study. With accurate and thorough exposure-assessment techniques, researchers will be enabled to determine the most important exposure types and contexts in relation to the subsequent development of detrimental changes in health. This information could be used by policy makers to reduce risk and improve popular health.

One of the most important questions when investigating the possible health effects of noise is “once we know for sure what they are, what should we do with them?” How, in other words, should they be weighed in value judgments such as when deciding whether to put an airport in a particular location or to significantly expand traffic at an existing facility; instances where both benefits and detriments may occur? Systems of hedonic or hedonistic indicators such as the changes in property values in response to changes in consumer demand when such changes in infrastructure are put in place are one such approach. These have been seen as representative of subjective changes in quality of life. However, consumers may not easily react to that of which they are not aware. The cardiovascular health effects of noise are not widely known. Accordingly, the system of hedonic indicators may not be able to represent the potential cardiovascular health effects of noise at this time. Another method, whose use is more common in Europe, is the “DALY” system. In this approach to costing, the severity of a health effect of any cause is assessed by determining the number of Disability Adjusted Life Years (DALY) that an illness produces. A Disability Adjusted Life Year is the amount of life or quality of life deemed to be lost due to death (in which case the number of DALY assessed would equal their expected life span after their premature death) or the fraction of quality of life (always less than one for living individuals) that is diminished by a modifying health condition times the number of further years the individual lives with the condition. The weight for a specific condition is generally determined via consultation with experts in the field of interest who compare the severity of the features and difficulties associated with a condition to the severity of conditions for which the severity weight has already been determined. Thus, any new illness is costed by comparison of its features with previously evaluated illnesses. This method of assessing total decrease in quality of life summed over a population has the advantage of giving a total estimate of health effects that allows direct comparison with other causes. The single number comparison and the simplification made possible may thus be a policy-making advantage. Disadvantages of the method may include disagreement over what truly constitutes a health effect. For

instance, the 2005 World Health Organization Report on Quantifying the Burden of Disease from Environmental Noise (WHO, 2005) includes annoyance, tinnitus, and cognitive impairment, which some might not see as health effects so much as quality of life issues. Nevertheless the construct is valuable for dealing with quality of life changes as well as health so the question becomes one of what those using it see as relevant changes in quality of life that need to be considered in a potential valuation. Other difficulties involved with the process include uncertainty of the dose-response relationship. The method determines the number of people likely to experience an illness that would not have otherwise experienced it (the fraction attributable) and multiplies the severity weight by the number of people experiencing it as a result of exposure and sums over each health effect to obtain the population total. Another criticism of this method is that some people may have severity weights greater than one for multiple health outcomes and thus contribute more to the total burden of disease by remaining alive than by dying. However, in theory, severity weights are not permitted to exceed one. Additionally, there is some concern that the measure might not be sufficiently widely agreed upon for noise (Maurice and Lee (eds) 2009). Though such controversies exist, the use of DALY's for cost-benefit analysis appears promising, particularly because it allows one to directly compare health effects from different sources, e.g., noise and air quality.

## 7 CONCLUSIONS AND RECOMMENDATIONS

Potentially serious health outcomes have been identified in studies involving transportation noise exposure in a population. These include heart disease and hypertension and the observed effects seem to be related especially to nighttime noise exposure although similar daytime exposure effects have also been identified.

The Babisch meta-analysis reports an odds ratio for occurrence of myocardial infarction of 1.13 per 10 dB increase of  $L_{day}$  and the HYENA results found an increase odds ratio for hypertension in those exposed to nighttime aircraft noise of 1.14 per 10 dB increment of noise. The possible role of sleep disruption in the development of these health outcomes has been investigated in this report along with the potential role of stress reactions. Hypertension and heart disease have been identified as potential outcomes in studies examining the effects of sleep disruption on health outcomes. Thus, a potential pathway exists for noise exposure to lead to these ailments through the long-term disruption or shortening of sleep. However, the exact size of the effect on sleep due to aircraft noise is not perfectly clear and may be small in magnitude, although significant results have been obtained for objectively measured sleepiness resulting from aircraft noise in the laboratory. Additionally, two other outcomes—obesity and diabetes—also seem to correlate with reduced or disrupted sleep and thus should also be examined as potential noise outcomes.

As illustrated in Figure 9, results of past sleep-health research including their dose-response relationships could be used with sleep disturbance and noise prediction models to predict the likelihood of increases in these outcomes with increased noise. This should be done by first examining mathematically the documented effects due to sleep to see if amounts of sleep disruption commonly observed are likely to be sufficient to produce detrimental effects in a population through these other outcomes. If justified, this could be followed by a reanalysis of data from studies that took into account potential confounding from body mass index to see if an effect is notable or the addition of noise data to studies investigating obesity or diabetes. At the very least, great care should be taken in adjusting models of noise health effects for obesity and diabetes presence as they may be mediating variables in the relationship between noise and hypertension and heart disease.

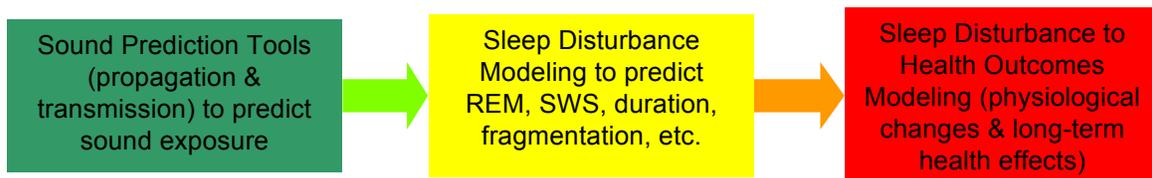


Figure 9: Depiction of a cascade of computational tools allowing quantitative estimates of health effects from noise predictions.

Noise may also affect health through exposure during hours when people are awake, often associated with a noisy working environment. In this case health effects seem to work through the medium of repeated arousals until long-term changes in vascular properties result. Both waking and sleeping arousal appear to be important and differentiating between the two sources of exposure in studies in order to determine the portion of disease attributable to one or the other is a challenging prospect possibly requiring either individual 24-hr noise monitoring or else elaborate exposure prediction schemes in order to find what portion of exposure was at work, what portion at home, and what people were exposed to while traveling. The HEARTS (Health Effects and Risks of Transport Systems) project methodology may be helpful in obtaining such exact and thorough exposure information within the context of a prospective study. The challenges

involved in discerning the most relevant exposure types and contexts seems to necessitate a prospective epidemiological study of these issues.

Inasmuch as these effects exist, they are not known to the general population. Thus, the system of hedonic indicators may not be able adequately account for them at the present time. This could be solved in several ways: education programs could let the public know of the potential risks and allow individuals to make informed decisions. Presumably doing so would affect hedonic indicators as individuals began taking the information into account and thus the system could maintain usefulness, even with the added considerations, although this might take some time to adjust. (This of course assumes that individuals generally take potential health risks into account in determining their behavior in a way representative of the actual cost to them of their behavior, which may be incorrect). Alternatively, a costing system such as the Disability Adjusted Life Year (DALY) system could allow decision makers to take measure of the aggregated health effects in a single number representative of total loss of health and life, which could then be weighed against potential increases in welfare and quality of life resulting from proposed transportation infrastructure changes. Either of these methods could be used effectively to balance the positive and negative features of proposed growth leading to additional noise exposure provided that the above assumption of people realistically weighing the potential effects of individual exposure proves valid.

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## APPENDIX A: TABULATION OF STUDIES RELATED TO NOISE AND HEALTH OUTCOMES

Table A-1: Matrix of Studies Showing Health Risks for Various Sleep Variables

	Reduction of Duration/Deprivation of Sleep	Sleep Stage Changes	Fragmentation and Disturbance of Sleep	Cardiovascular Arousal/Nondipping
<b>Diabetes Risk Factors</b>	Ayas 2003 Gottlieb 2005 Knutson 2006 Mallon 2005 Spiegel 1999 Spiegel 2005 Yaggi 2006 Wolk 2006	Tasali 2008	Kawakami 2004 Knutson 2006 Mallon 2005 Meisinger 2005 Phillips 2000 Spiegel 2005 Nilsson 2004 Tasali 2008	
<b>Obesity</b>	Gangwisch 2005 Hasler 2004 Kalra 2003 (background) Reilly 2005 Sekine 2002 Spiegel 2003 Spiegel 2004 Taheri 2004 Vorona 2005 Yamagami 2002		Gupta 2002 Fogelholm 2007	
<b>Hypertension</b>	Gangwisch 2006 Gottlieb 2006 Lusardi 1996 Lusardi 1999		Appels 1987 Bixler 2000 Ekstedt 2004 Morrell 2000 Sullit 2006	
<b>Nondipping of Blood Pressure</b>			Lenz 2007 Loredo 2001 (against somewhat) Loredo 2004 Mansoor 2002 Matthews 2008 O'Shea 2000 (modifying) Pankow 1997 Portaluppi 1997	N/A
<b>Heart Disease</b>	Ayas 2003 Ikehara 2009 Kripke 2002		D'Alessandro 1990 Hla 2001 Newman 2001 Shahar 2001 Winkelman 2007	Kikuya 2004 O'Brien 1988 Ohkubo 1997 Ohkubo 2002 Smolensky 2007 Staessen 1999 Stolarz 2002 Verdecchia 1994 <b>(Heart-rate related hereafter)</b> Gillum 1990 Gillman 1993 Palatini 1997

<b>Continued...</b>	<b>Reduction of Duration/Deprivation of Sleep</b>	<b>Sleep Stage Changes</b>	<b>Fragmentation and Disturbance of Sleep</b>	<b>Cardiovascular Arousal/Nondipping</b>
<b>Tiredness, Memory Effects and Decreased Performance</b>	Fiorica 1968 Fischer 2002 Kollar 1966(against) Lyznicki 1998 Muecke 2005 Vertes 2005 (against)	Bonnet 1986b Bonnet 1986c Bonnet 2003 Ferrara 2000 Huber 2004 Marshall 2006 Stickgold 1998 Stickgold 2000 Stickgold 2000b Stickgold 2005 Vertes 2005	Bonnet1986b Bonnet 1986c Resta 2003 Stepanski 2002 Vertes 2005 Vgontzas 1997	
<b>Other Hormone Changes</b>	Dzaja 2004 Leproult 1997 Lusardi 1999 Meier-Ewert 2004 Mullington 2003 Redwine 2000 Spiegel 2003 Spiegel 2004 Taheri 2004 (against) Taheri 2007 Vgontzas 1997	Luboshitzky 2001 Redwine 2000 Tiemeier 2002	Ekstedt 2004 Carter 1994 (against) Luboshitzky 2001 Redwine 2000	
<b>Miscellaneous Mortality/Health</b>	Horne 1978 Kripke 1979 Kripke 2002 Passchier 2000 Patel 2004			Kikuya 2004 O'Brien 1988 Ohkubo 1997 Ohkubo 2002 Staessen 1999 Stolarz 2002 Verdecchia 1994

**Table A-2: Matrix of Studies Linking an Environmental Noise Stressor or Sleep Disorder to a Consequent Intermediate or Long-Term Health Outcome**

	Night-time noise	Day-time noise/general noise	Sleep Disordered Breathing
<b>Hypertension/Blood Pressure Alterations</b>	Babisch 2006 Bach 1991 Carter 2002 Griefahn 2008 Haralabidis 2008 Jarup 2008	Andren 1980 Aydin 2007 Babisch 2006 Bjork 2006 Chang 2003 Chang 2007 Eggerteson 1984 (acute) Fogari 2001 Fouriaud 1984 Goto 2002(no significant finding) Kluizenaar 2007 Lusk 2004 Rosenlund 2001 Regecova 1995 Talbot 1999 van Kempen 2002 Zhao 1991	Bixler 2000 Morrell 2000 Newman 2001 Pankow 1997
<b>Heart Attack (Myocardial Infarction)</b>	Babisch 2006	Babisch 2000 Babisch 2004 (NaRoMI) Babisch 2006 Davies 2005 Grazuleviciene 2004 Hoffman 2006 Ising 1997 Selander 2009 Tonne 2007 Van Kempen 2002	D'Alessandro 1990 Hla 2001 (Ischemia) Newman 2001 Shahar 2001 Winkelman 2007
<b>Nondipping</b>			Portaluppi 1997 Loredo 2001
<b>Miscellaneous Mortality/Health</b>		Franssen 2004 Melamed 1997 Fujino 2007 Evans 1998 Andren 1981 Persson Waye 2001	

**Table A-3: Matrix of Studies Showing Noise Leading to Acute Effects**

	<b>Aircraft</b>	<b>Other Traffic/Work</b>	<b>Synthetic Noises</b>
<b>Stage Arousals</b>	Basner 2008 (against) Basner 2008b Flindell 2000 Griefahn 2006	Bach 1991 Eberhardt 1987 Griefahn 2006 Marks 2008	Campbell 2005
<b>Autonomic Arousals and Transient Blood Pressure</b>	Carter 2002 Carter 1994 Haralabidis 2008	Bach 1991 Carter 1994 Carter 2002 Di Nisi 1990 Griefahn 2008 Kuroiwa 2002	Aaron 1996 Di Nisi 1990 Johnson 1968 Townsend 1973
<b>Awake Arousal and Transient Blood Pressure</b>		Andren 1980 Chang 2003 Chang 2007 Di Nisi 1990 Eggertson 1984 Fogari and Zoppi 2001 Hansson 1984 Lusk 2004 Raggam 2006	
<b>Generic Sleep Disturbance</b>	Basner 2006 Basner 2008 Fidell 1995 Flindell 2000 Griefahn 2006 Horne 1994 Michaud 2007	Griefahn 2006 Eberhardt 1987 Marks 2008 Ohrstrom 1995	Bonnet 1986 (background) Aaron 1996
<b>Habituation</b>	Kuroiwa 2002		Townsend 1973 Rabat 2005

## APPENDIX B: GLOSSARY

A-weighting	a filter applied to a noise monitoring device designed to mimic the response of the human ear for sounds at low level
ABPM	Ambulatory blood pressure monitoring
Acute	medically used, a term describing a condition that has either sudden onset or short duration; as used here it generally refers to short-term effects
Adiposity	a medical term for the degree of fatness of a subject
AHI	Apnea-Hypopnea Index, see below
Alpha waves	brain wave activity characteristic of a person awake with closed eyes
Ambulatory Blood Pressure Monitoring	(or ABPM) Method where blood pressure is repeatedly sampled at a desired interval of time using a device that the subject is attached to and carries if they are mobile. Values are recorded and analyzed later. Helps solve problem of “white coat hypertension” where subjects may experience increases in blood pressure in response to having a person measure them
Antihypertensive	a medicine or intervention that has the effect of lowering blood pressure
Anorexigenic	hunger diminishing
Apnea	short term stoppage of breathing, as used here a medical condition in which sleeping individuals temporarily stop breathing, often as a result of a blocked airway; it frequently results in arousal
Apnea-Hypopnea Index	(AHI) a measure of the degree of sleep-disordered breathing, it is given as the number of apneas (a respiratory event involving total stoppages of airflow) and hypopneas (a type of respiratory event involving large reduction in airflow volume) in a night divided by the total number of hours slept, thus the average number of events per night
Apnea Index (AI)	may be calculated using just the number of apneas divided by the total length of sleep time.
Arousal	depending on the usage it can refer to awakening from sleep, or an increase in readiness for action, or a transition from a deeper to a lighter stage of sleep, or the excitation of the cardiovascular system characterized by increases in blood pressure and heart rate
Articulation Index	a measure of speech intelligibility for use with stationary sounds. Speech Interference Index (SII) (ANSI, 1997), is a revision of this
Attributable	in this context responsible for a particular effect or, in a population, responsible for the prevalence of an effect
Beta Cells	pancreatic cells that make and release insulin

Bidirectional	going both ways, in this case it refers to a type of causality where either one of two effect may lead to the other
Biometric	having to do with the measurement of living things
Body mass index (BMI)	the weight of a subject in kilograms (kg) divided by the subject's height in meters (m) squared thus $\text{kg/m}^2$ ; it is considered predictive of some health outcomes in many cases but may run into difficulty with more muscular subjects
C-Peptide	a byproduct of insulin production that is released together with insulin and can be used to measure insulin levels indirectly
Cardiac Arousals	changes in heart rate, blood pressure and such in response to a stimulus during sleep, which may occur both with and without resulting changes in sleep stage or awakening in
Cardiovascular	the system of the body including the heart, lungs, and blood vessels, responsible for distributing nutrients and oxygen to the body
CaseControl Study	a study design in which cases (those manifesting a certain medical condition) are compared with controls (those not manifesting the condition examined) in order to determine what factors may lead to or be predictive of the genesis of the condition
Causal	in this context a causal relationship is one in which one circumstance leads to another that would not have occurred without it, or in a population would have occurred less frequently
Confounding	Confounding variables are two variables (explanatory or lurking variables) that are confounded when their effects on a response variable cannot be distinguished from each other.
Consolidation	referring to memory in this context, the solidifying of memory
Cortisol	a hormone that may become elevated in response to stress; it leads to decreases in immune function as well as increased blood pressure as well as changes in blood sugar levels
Cost (verb)	to evaluate the cost (noun) of something
Co-vary	to vary together
Cross-sectional Study	a study examining a portion of the population at a fixed time; thus, it is often compared with a snapshot; correlations between variable at a particular moment to identify possible relationships
DALY	Disability Adjusted Life-Year, the amount of life and quality of life lost as a result of a particular effect
Decrement	decrease in quality of quantity, the opposite of increment
Deep sleep	non REM stage 3 and 4 of sleep in which a subject is most difficult to awaken, delta waves are strongly present in brain activity, and important restorative processes are thought to occur
Deleterious	harmful

Delta waves	brain activity characteristic of a deep sleep state (non-REM stages 3&4)
Diabetes (Type 2)	a metabolic condition characterized by “insulin resistance”, wherein the body becomes somewhat insensitive to insulin and thus exhibits diminished glucose uptake. It is associated with increased cardiovascular risks.
Diastolic	low point in the arterial blood pressure waveform, the second number in the blood pressure “first over second” reading
Dipper	one who experiences a >10% dip in nocturnal blood pressure versus daytime values
Disambiguate	to make unambiguous or remove ambiguity, to clarify
Dissociation	in studies of noise effects in the learning environment, a proposed coping mechanism that may have detrimental effects
Dose-response	a relationship in which a certain amount of exposure to a stimulus leads to a certain amount of an effect
Dosimetry	the measurement of an individual's exposure to some effect, generally noise in this study
Environmental	relating to or having to do with the environment, in this case often used to refer to exposure to noise outside of work, so while driving, or at home
ECG	electrocardiography, see EKG
EEG	electroencephalogram, a measurement of the electrical activity of the brain
EKG	electrocardiography, an electrical recording of the heart
EMG	electromyography, a measurement of the electrical activity of muscles
Event	in this context a distinct occurrence of noise, such as an aircraft pass by
Excess risk	increased probability of some outcomes seen in response to some variable so probability with variable minus probability in the absence of that variable
Fasting	thought other definitions exist, for the purpose of this study refraining from partaking of food for a period of time
Fragmentation	in the context of this study, the frequent disruption of sleep possibly leading to awakening or changes in sleep's structure, may limit restorative capacity of affected sleep
Ghrelin	a hormone produced primarily in the stomach that regulates appetite. Orexigenic, it induces feeling of hunger
Glucose	a natural sugar which plays an important role in human metabolism

Glucose tolerance	the degree to which the body is able to effectively manage glucose levels keeping them within an acceptable range through appropriate uptake
Glucose Tolerance Test	a measure of bodily glucose uptake: the person being tested is administered an amount of glucose proportional to their body weight and blood samples are taken in order to determine how quickly the body is able to clear the excess glucose, impaired reaction (higher levels after a specified period of time) is characteristic of type 2 diabetes
Glycemic control	regulation of glucose within the body
Growth factor	naturally occurring substances whose presence promotes growth of tissues or cells
HEARTS	“Health Effects and Risks of Transportation Systems” a project undertaken by the World Health Organization that evaluates individual exposure to multiple environmental stressors in multiple microenvironments so as to generate a more comprehensive picture of individual exposure
Hedonic measures	refers to a set of measures of cost and benefit based on how environmental noise sources such as airports effect people’s quality of life in the area and thus such decisions as house buying
Hemoglobin A1c	a form of hemoglobin that forms in proportion to levels of glucose in the blood; as a result, it may be seen as an indicator of long-term glucose levels in the body, and may be looked upon as a marker of glycemic control
Hormone	chemicals used by the body to affect the actions of various systems of the body
Hyperinsulinemic euglycemic clamp	a technique for measuring insulin resistance, it involves administering a known dose of insulin and infusing glucose so as to keep levels within a certain range; the amount of glucose required to maintain that range is considered as an indication of how responsive the body is to insulin which signals glucose uptake
Hypertension	long-term high blood pressure; the World Health Organization defines hypertension as blood pressures consistently over 140 mmHg systolic blood pressure or 90 mmHg diastolic blood pressure; also included in this review are studies that included previously doctor diagnosed hypertension that was being currently treated with antihypertensive drugs
Hypertensive	having hypertension (high blood pressure)
Hypopnea	a respiratory event in which breathing is reduced, but not totally, especially during sleep in this study; it may lead to arousal or sleep fragmentation
Hypoxemia	decrease in blood oxygen levels
Immission	exposure, taking in from the environment
Incidence	frequency of occurrence

Induction effect	some diseases require a period of exposure to a stressor or “induction time” before that stressor begins to have a noticeable effect on the occurrence of the disease, thus the period of time required for a stressor to begin producing an outcome
INM	Integrated Noise Model, predicts outdoor noise metrics given airport operations.
Insulin dependent diabetes	type of diabetes requiring treatment with insulin to avoid unsafe levels of blood sugar leading to tissue damage
Insulin resistance	a condition in which the body does not respond in the normal manner to insulin (insulin normally triggers uptake of glucose in the body but with insulin resistance it does not)
Interquartile	in statistics the interquartile range is the portion of the data set lying within the middle two quarters of the distribution, so it includes everything except the highest quarter and the lowest quarter
Ischemic heart disease	heart disease characterized by reduced blood flow to the heart tissue
K-complexes	EEG spikes characteristic of the initiation of stage two of non-REM sleep, they also seem to indicate an arousal event
Laboratory Study	type of study design in which many variables are directly controlled in order to reduce confounding by unmeasured or uncontrolled factors; often conducted in the laboratory there is somewhat of a tradeoff between the benefits of a stable and predictable environment and the detriments of a less realistic and familiar one, which may lead to systematic differences in reaction in some types of study
Latency	how long you have to wait for something, the time between a signal and a response, one usage in this report concerns sleep latency, how long it takes an individual to initiate sleep given favorable conditions: lights out, quiet, etc
Leptin	a protein hormone with a role in regulation food intake and energy expenditure, it is anorexigenic, meaning it reduces hunger or signals satiety (feeling of being full).
$L_{max}$	the maximum A-weighted sound pressure level (measured on a fast or slow setting on a sound level meter) occurring in the course of a noise event
Longitudinal Study	type of study design in which a population is followed and measured in order to determine correlation between observed variables and to determine possible relationships
Loudness	is the psychophysical measure of sound that is most correlated with the magnitude of the sound
Markov model	the only Markov model mentioned in this paper is Basner’s (2008) which is a model that predicts the probability of moving to another specific sleep stage given the current stage, whether noise is occurring or has just occurred, and the number of epochs (time) since sleep onset.

Mediating	in this context a variable may be said to be mediating if a change in a first variable leads to a change in a second variable which then leads to a change in a third variable, the second would then be called a mediating variable in the relationship between the first and third variable
Meta-analysis	an analysis in which multiple studies, possibly not statistically significant on their own but preferably of similar design are pooled together (either the raw data or the results) and analyzed in order to obtain a more statistically powerful result or other synthesis result beyond that obtainable from the individual studies
Metabolic Syndrome	a grouping of medical conditions including insulin resistance (often diabetes), obesity (especially central), hypertension, dyslipidemia and heart disease
Microenvironments	concept used in the HEARTS project; a definable portion of a subjects environmental exposure with known exposure levels and types, e.g. home, work or transportation
Microneurography	invasive method of measuring activity in nerves electrically
Modifiers	variables which may change the way the other variables interact, for instance the noise level at a subjects house may be known, but the subjects window opening habits may change the noise exposure that they in fact experience
MSLT	Multiple Sleep Latency Test (see below).
Multiple Sleep Latency Test	(MSLT) The “gold standard” for objective sleepiness, the MSLT is a measure determined by having a subject attempt to nap in a darkened environment until they are effectively asleep as determined via polysomnography. Latency basically means how long it takes them to fall asleep. They take naps at multiple times of the day so that variations in circadian alerting may be taken into effect, hence the “multiple” part of the name. MSLT has been understood as reflecting the strength of the drive to obtain sleep as that drive is influenced by “sleep debt” the amount of sleep a person is behind on and possibly other variables
Myocardial infarction	also known as a heart attack, this occurs when blood flow to part of the heart is disrupted leading to cessation of function and possible death of heart muscle tissue, as well as possible death
NHANES	National Health and Nutrition Examination Survey
NMSIM	Noise Modeling Simulation, a program developed by Wyle to predict noise metrics at locations around airports. This is similar to INM, but includes more functionality
Non-dipper	one who does not experience the normal 10% dip in nocturnal blood pressure relative to daytime values or one who experiences a diminished (<10%) dip
Non-insulin dependent diabetes	type of diabetes in which the body becomes resistant to the action of insulin or produces somewhat less than is needed so that normal uptake of glucose in response to insulin may not occur

Non-REM	Sleep stages not involving rapid eye movement (REM) sleep.
Nonsignificant	the outcome of a statistical analysis that showed that it is likely that the result (the effect found) occurred by chance.
Obesity	a condition characterized by increase of weight to the point of potentially diminished health, often considers as BMI>30 kg/m <sup>2</sup>
Odds ratio	a number describing the ratio between the odds of an event occurring in a first group and that same event occurring in a second group; if <i>a</i> is the incidence in the first group and <i>b</i> the incidence in the second then the odds ratio is given by $\frac{a}{1-a} \div \frac{b}{1-b}$
Orexigenic	hunger producing
Parasympathetic	portion of the nervous system which acts to decrease arousal
Peripheral resistance	property or degree of fluid flow resistance in the peripheral blood vasculature
Pharmacological	“of drugs”; thus, a pharmacological intervention is an intervention using drugs
Pollution	the presence of contaminants in or their introduction into an environment possibly causing harm
Polysomnography	the use of the combination of readouts from an electroencephalogram (monitors brainwave patterns), an electrocardiogram (monitors heart rate), an electromyogram (monitors muscle activity), and an electro-oculogram (measures eye movements) to determine the presence of clinically defined sleep as well as the stage of sleep being experienced
Potentiate	to make powerful or effective
Procedural memory	in contrast to declarative memory which might contain a transmissible description of how to do things, more experiential memory of how to do things by doing them; for instance dancing or riding a bike, they are learned but can but cannot be learned purely from a textbook, they have to be experienced to be truly learned
Proinsulin	an insulin precursor made in the beta-cells
Prospective Study	a study which follows a group of people over a period of time monitoring their changes and differentiating them with regards to some variable
PST	Pupillographic Sleepiness Test, (see below).
Psychomotor tasks	a task involving the ability of a subject to quickly recognize and appropriately respond to a stimulus
Pupillographic Sleepiness Test	(PST) is a test of objective sleepiness which appears to measure the same underlying variables as the multiple sleep latency test (MSLT). It does this by observing the dilations and contractions of the pupil and extracting information about the degree and frequency of motion.

Recognition memory	type of memory allowing subject ability to recognize previously experienced stimuli
Relative risk	a measure of relative prevalence of some condition between groups: if prevalence in group one is "a" and that in group 2 is "b", and group 2 is taken as the standard group, then the relative risk of group a is a/b relative to group b
REM	Rapid Eye Movement, one of the five stages of sleep.
Restless leg syndrome	a condition characterized by irritation and desire to move the limbs as well as movement of the limbs during sleep, possibly with resulting arousal
Re-uptake	the re-absorption of some substance in the body, particularly of neurotransmitters; in this paper the removal of sympathetic nervous system neurotransmitters
Roughness	a psychoacoustic attribute describing modulation of a signal within a certain modulation frequency range
Satiety	the feeling of fullness
SDI	Sleep Disturbance Index, see below
SELA	A-weighted sound exposure level. The total sound energy occurring within 10 dB of the peak A-weighted sound pressure level divided by the duration of the within-10dB-of-peak-level part of the event.
Sensitivity	in this case refers to subjects' sensitivity to noise, it has been thought of as a potential modifier in noise related health effects
SFI	Sleep Fragmentation Index, see below
Sharpness	psychoacoustic parameter corresponding primarily to the relative weighting of low and high frequencies: increased relative quantities of high frequencies typically leads to increased sharpness
Short sleep	decreased duration of sleep, for adults typically 6 or fewer hours, though the amount depends on the subject's age
Significant (Statistically)	the outcome of a statistical analysis that showed that it is unlikely that the result (the effect found) occurred by chance.
SII	Speech Interference Index, see Articulation Index
Sleep Disturbance Index	(SDI) a measure of sleep disturbance calculated by dividing the total number of arousals and awakenings (likely determined via polysomnography) in a night by the total length of sleep; thus, it characterizes the average number of disturbance per hour
Sleep Fragmentation Index	(SFI) number of awakenings and shifts to stage one sleep in a night (likely as determined by polysomnography).
Sleep Stage	one of several common states comprising sleep; for full details see the section in the description of metrics and the section on sleep
Speech Interference Index	(SII) this is an update of the Articulation Index
Sphygmomanometer	device used to measure blood pressure

Stanford Sleepiness Scale	a survey measure used to assess aspects of subjective sleepiness
Subjective	as compared with objective; often refers to measures of opinion or subject feelings rather than measurement of physiological parameters; subjective evaluation often involves the use of a questionnaire
Subsample	a subgroup within a study population in which some particular question is investigated
Survey	the use of a written questionnaire or interview to gain information on a population
Survivor population	a population in which vulnerable people have been eliminated, for instance if some aspect of working in a coal mine causes respiratory problems then those most susceptible to respiratory problems are not likely to be long term employees; the remaining employees then are more resistant to the adverse conditions than the general population may be
Sympathetic nervous system	the portion of the nervous system responsible for increasing arousal and priming the body for action
Sympathovagal balance	the balance between the activity of the sympathetic nervous system and the vagus nerve responsible among other things for some heart regulation
Systolic	in this context, referring to the peak in the pressure of the cardiac pressure waveform, the first number in the blood pressure "first over second" reading
Testosterone	a steroid hormone produced in the testes or males and the ovaries of females, potentially important in maintaining cardiovascular health, release appears circadian
Theta waves	EEG pattern associated with initiation of sleep or quiet focus
Total Sleep Time	(TST) the total length of time a subject slept in a given night
Trait anxiety	a stable trait similar in some respects to neuroticism and negative affectivity also linked with noise sensitivity in some studies
Trait-stable	existing in a stable form in a person's biology or personality rather than something that is more situation dependent
Transient	short-term, passing with time, etc
TST	total sleep time, see above
Vagus nerve	a nerve responsible among other things for some heart regulation
Vascular	of the blood vessels
Visual scale	a scale used in subject testing in which the subject is asked to rate something using a scale, these may be able to display both ordinal properties as well as proportional values

White coat hypertension	short-term hypertension probably in response to medical examination leading in many instances to a false positive diagnoses of hypertension
WHO	World Health Organization, see below
Wisconsin sleep cohort study	a longitudinal study making use of both polysomnography and survey questionnaires and examining relationships between short sleep and body mass index
World Health Organization	(WHO) agency of the United Nations which deals with international public health issues; its stated goal according to their constitution is the attainment by all peoples of the highest possible level of health. <a href="http://www.who.int/topics/noise/en/">http://www.who.int/topics/noise/en/</a>
Wrist Actigraphy	the placing of a bracelet with accelerometers or similar sensors on the wrist of a person being monitored to determine when they are active or dormant (motility)