

Environmental Research and Consultancy Department



## **ERCD REPORT 1208**

# **Aircraft Noise, Sleep Disturbance and Health Effects: A Review**

**K Jones  
D P Rhodes**

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### **Summary**

This literature review was prepared for the Department for Transport as part of their consultation on the night flights regime to apply at Heathrow, Gatwick, and Stansted airports from 26<sup>th</sup> October 2014. The review aims to provide an overview of the main findings within environmental noise and health research, and includes the effects of sleep disturbance due to aircraft noise. The cost-benefit analysis of night flights is discussed in terms of previous methodology and proposals for future evaluation of the aircraft movements at night are put forward.

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ISBN 978 0 11792 795 7

Enquiries regarding the content of this publication should be addressed to:  
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Published by TSO (The Stationery Office) on behalf of the UK Civil Aviation Authority.

Printed copy available from:  
TSO, PO Box 29, Norwich NR3 1GN  
Telephone orders/General enquiries: 0870 600 5522  
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## Glossary of Terms

**A-weighting** A frequency weighting that is applied to the electrical signal within a noise-measuring instrument as a way of simulating the way the human ear responds to a range of acoustic frequencies.

**Adrenaline** Also referred to as Epinephrine. A hormone and neurotransmitter and member of the catecholamine family, which, when released increases the response of the sympathetic division of the Autonomic Nervous System.

**Alpha waves** Electromagnetic oscillations in the frequency range of 8–12 Hz observed in the brain during periods of waking relaxation with eyes closed.

**ANE** Aircraft noise event

**BCA** Behaviourally confirmed awakening

### Catecholamine

Hormones that are released by the adrenal glands in situations of stress such as psychological stress or low blood sugar levels. They include adrenaline, noradrenaline and dopamine.

**CBBN** Continuous broadband noise

**Cortisol** Hormone produced by the adrenal gland that is associated with stress responses, increasing blood pressure and blood sugar and reducing immune responses.

**dB** Decibel units describing sound level or changes of sound level.

**dBA** Levels on a decibel scale of noise measured using a frequency dependent weighting, which approximates the characteristics of human hearing. These are referred to as A-weighted sound levels.

**EEG** Electroencephalogram - used to measure brain activity during sleep.

**ECG** Electrocardiogram – used to measure heart rate.

**EMG** Electromyogram – measures facial muscle tone during sleep to identify REM.

**Endocrine** Typical endocrine glands are the pituitary, thyroid, and adrenal glands. Features of endocrine glands are, in general, their ductless nature, their vascularity, and usually the presence of intracellular vacuoles or granules storing their hormones.

**EOG** Electro-oculogram – measures movement of the eyes during sleep to help identify REM sleep.

**Ergotropic** Those mechanisms and the functional status of the nervous system that favour an organism's capacity to expend energy, as distinguished from the trophotropic mechanisms promoting rest and reconstitution of energy stores.

**IBBN** Intermittent broadband noise

**K-complex** An EEG waveform that occurs during stage 2 sleep. They occur randomly throughout stage 2 sleep, but may also occur in response to auditory stimuli.

**L<sub>A</sub>** The A-weighted sound level (in dBA).

**L<sub>Amax</sub>** The maximum A-weighted sound level (in dBA) measured during an aircraft fly-by.

**L<sub>eq</sub>** Equivalent sound level of aircraft noise, often called equivalent continuous sound level. L<sub>eq</sub> is most often measured on the A-weighted scale, giving the abbreviation L<sub>Aeq</sub>.

**L<sub>night</sub>** Equivalent sound level of aircraft noise in dBA for the 8-hour annual night (2300-0700).

**L<sub>den</sub>** Equivalent sound level of aircraft noise in dBA for the 24-hour annual day, evening, and night where the evening movements are weighted by 5 dB and night movements are weighted by 10 dB.

### **Noradrenaline**

Also known as Norepinephrine. Part of the catecholamine family, with dual roles as a hormone and neurotransmitter. A stress hormone, along with adrenaline, noradrenaline also underlies the fight-or-flight response, directly increasing heart rate, triggering the release of glucose from energy stores, and increasing blood flow to skeletal muscle.

**PNdB** Perceived Noise Decibels.

### **Polysomnography (PSG)**

A comprehensive recording of the biophysiological changes that occur during sleep. The PSG monitors many body functions including brain (EEG), eye movements (EOG), muscle activity or skeletal muscle activation (EMG) and heart rhythm (ECG).

**REM** Rapid Eye Movement sleep. A stage of sleep characterized by rapid movements of the eyes, low muscle tone and a rapid, low voltage EEG signal.

**SEL** Sound Exposure Level in dBA, a measure of noise event level, which accounts for both the duration and intensity of noise.

### **Sleep Efficiency Index**

The proportion of sleep in the episode potentially filled by sleep (i.e., the ratio of total sleep time to time in bed)

**Sleep latency** The length of time that it takes to accomplish the transition from full wakefulness to sleep, normally to the lightest sleep stage

**SPL** Sound Pressure Level.

**SWS** Slow wave sleep, characterised by low frequency, high amplitude waves on the EEG and comprised of Stages 3 and 4 sleep.

**Trophotropic** The movement of cells in relation to food or nutritive matter. Energy expending.



**TST** Total sleep time

**Vasoconstriction**

Narrowing (constriction) of blood vessels. When blood vessels constrict, the flow of blood is restricted or slowed

**VPC** Ventricular premature contraction

**WASO** Wake time after sleep onset

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# 1 Introduction

## 1.1 Background

- 1.1.1 Sleep is ubiquitous across species, and is fundamental to health and wellbeing, providing a regular resting period and preventing fatigue. Functions of sleep range from restoration at the cellular level, to neuronal repair, and it even plays a role in memory consolidation. Although most people would appreciate that sleep is necessary for survival and normal functioning, it is difficult to study the functions of sleep as it actually occurs. More often, it is the effects of sleep deprivation, fragmentation and manipulation of the sleep-wake cycle that are prolifically studied to examine the functions of sleep. Sleep researchers have been aware for quite some time that noise disturbs the sleep cycle and can cause alterations in sleep architecture, changes in sleep stage, body movements, decreased sleep quality and even awakenings during the sleep period. Next-day effects also exist, including increased fatigue, decreased performance levels and a resulting negative effect on mood. Noise also acts as a stressor on the body and can produce autonomic responses in the sleeping person, such as elevated cortisol, adrenaline and noradrenaline levels, which are implicated in long-term health effects on the cardiovascular system.
- 1.1.2 The most obvious source of noise at night comes from transportation, such as aircraft flying overhead, rail noise and road traffic. Due to its intermittent nature, aircraft noise is deemed to be the most annoying of transportation noise, with road noise being the least likely to annoy. It is the aim of this review to examine the work specifically produced on the effects of aircraft noise on sleep disturbance and other health effects, to provide an overview of the area, past and current undertakings and potential methodologies for evaluating the cost-benefits of night flights in terms of health impacts.
- 1.1.3 It is acknowledged that uninterrupted sleep is a prerequisite for good physiological and mental well being. The WHO Guidelines conclude that sleep disturbance is a major effect of environmental noise and that exposure to environmental noise may cause primary effects during sleep (e.g. awakening), and secondary effects that can be assessed after night-time noise exposure (e.g. next day tiredness). WHO identify the elderly, newborn, shift workers and persons with physical or mental disorders as being particularly vulnerable to sleep disturbance.
- 1.1.4 A report (Porter, 2000) prepared for the UK Department of Transport by National Air Traffic Services Ltd, considered the potentially adverse effects of night-time aircraft noise on people and reviewed available evidence. Porter's review is summarised below and provides the basis for the summary of the scientific literature presented here; it is supplemented by findings published since 2000 and the conclusions of various other reviews.
- 1.1.5 Porter categorised the potential effects of night-time aircraft noise as:
- Acute Responses: immediate or direct disturbances such as sleep disturbance (e.g. awakenings, sleep stage changes), other physiological changes that coincide with the noise events (e.g. increase in heart rate or blood pressure, or immune system effects) or acute annoyance.
  - Total Night Effects: aggregations of acute responses over a total night, such as sleep loss or frequent disturbances breaking up the general sleep pattern.

- Next Day Effects: short term effects of the acute responses and total night effects (e.g. next day tiredness, degradation of task performance, short-term annoyance).
- Chronic Effects: pervasive long-term consequences of continuing acute responses and next day effects. These are the same potential effects as discussed above in general terms (e.g. annoyance, cardiovascular and physiological effects, and mental health effects.)

1.1.6 The review will cover a wide range of noise effects on sleep, from the effect on the microstructure of the electroencephalogram (EEG); to obvious changes in sleep architecture and their implications for sleep quality, mood and performance. Field and laboratory studies will be compared, and the use of actigraphy versus polysomnography as a means of measuring sleep disturbance in large populations around airports will be discussed. The physiological implications of noise-induced sleep disturbance will be looked at, including the main stress hormone concentrations, heart rate and cardiovascular responses to noise. This highlights the importance of the neuroendocrine system in the recovery element of sleep function and is important to consider in terms of long-term health effects of noise disturbance. Due to aircraft flight exhibiting a high proportion of low-frequency noise, this is also included for further insight into the specific effects of aircraft noise on sleep disturbance. Suggestions for further work and a summary of current research into this area will be given.

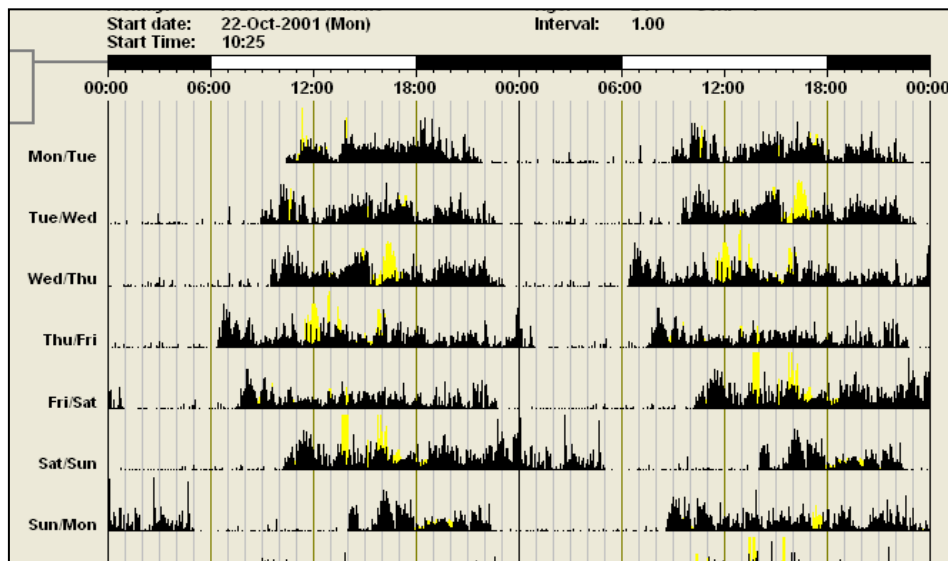
## 1.2 Sleep measurement

1.2.1 The most common and effective means of measuring sleep is by the Electroencephalogram (EEG). The scalp is “mapped” into specific sites and electrodes are attached accordingly, to measure changes in electrical activity in the brain as the subject sleeps. This provides a highly detailed record of the sleep period and charts progression through the sleep stages, changes within state, arousals and awakenings at the exact time at which they occur. Whilst providing the most accurate and detailed method of sleep measurement, it is usually easier to conduct whilst in a laboratory setting where the traces can be observed and electrodes can be replaced or reattached if necessary. It is also a relatively expensive and time-consuming method of sleep monitoring, and therefore is difficult to obtain results from large study samples.

1.2.2 A common non-invasive way of enabling sleep to be monitored in large samples is by actigraphy. The subject wears a small wristwatch sized monitor (actiwatch) on their wrist and is able to continue with their normal sleep/wake routine in their own home with no disruption. The actiwatches log movement at pre-prescribed intervals and produce a chart of activity (actigraph) and rest periods over the number of days in the study. However, because the actigraph gives an output of movement, and not brain activity, it is not always possible to correlate periods of rest with actual sleep. To corroborate actigraphy results it is common to ask subjects to keep a sleep diary throughout the study with details such as bed time, wake time, estimated sleep latency (time taken to fall asleep) and number and time of awakenings. The subjective sleep diary results, along with actigraphy software, can be used to calculate estimated sleep parameters such as sleep efficiency, fragmentation index, total sleep time, percentage time spent asleep etc. **Figure 1** shows an example of an actigraph.

1.2.3 Both methods of measuring sleep have been used in research into aircraft noise-induced sleep disturbance, and it is useful to separate these into field and laboratory studies.

**Figure 1:** Example of an actigraph. Dark bars represent activity, flat lines mean little or no activity, and yellow bars represent light exposure.



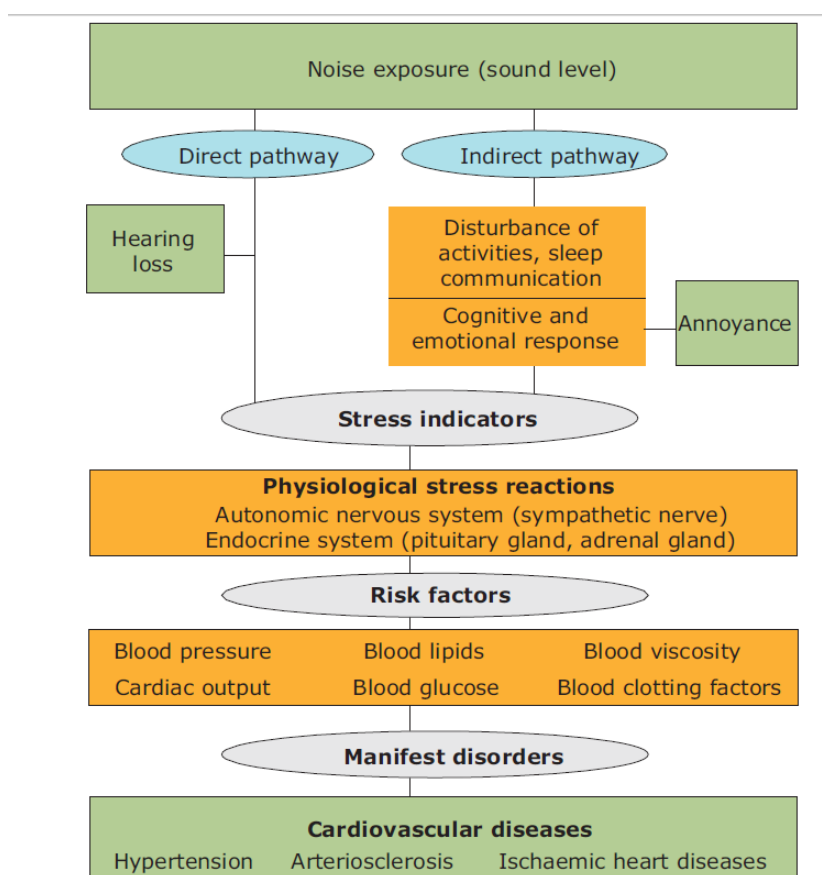
1.2.4 **Figure 2**, taken from Babisch (2002) summarises the effects of noise on the body.

1.2.5 Babisch (2002) explains that noise either directly or indirectly affects the autonomous nervous system and the endocrine system, which in turn affects the metabolic homeostasis (physiological balance) of the organism, including biological risk factors, and thus increasing the risk for manifest disorders in the long run. Indirect, in this respect, means that the subjective perception of sound, its cognitive interpretation and the available coping abilities play a role in physiological reaction. Direct, on the other hand, means that the activation of the regulatory system is determined by direct interaction of the acoustic nerve with other parts of the central nervous system (e.g. hypothalamus, amygdala). This is particularly relevant during sleep, where autonomous responses to single noise events, including changes in blood pressure and heart rate, have been shown in subjects who were subjectively not sleep disturbed.

1.2.6 Section 2 of this report reviews sleep disturbance research up to 1990, whilst section 3 reviews research after 1990. That year marks an approximate step change in magnitude and complexity of research studies into aircraft noise and sleep disturbance. Studies began to grow to include more subjects, more nights of data and record more information, including stress and cardiac indicators.

1.2.7 Section 4 summarises and reviews the health effects associated with sleep disturbance, including stress and cardiovascular risk and the effects on children. Section five discusses noise levels at which effects are considered to occur, including levels proposed for the protection of public health. Section six reviews research into monetising sleep disturbance and finally section seven provides an overall summary of the report.

**Figure 2: Effects of noise on the human body**



## 2 A summary of early studies into aircraft noise and sleep disturbance (1963 – 1990)

### 2.1 Introduction

- 2.1.1 In 1963, a report entitled “Noise”, written by the committee on the problem of noise and commonly referred to as “The Wilson Report” after Sir Alan Wilson, Chairman of the committee, referred to the World Health Organisation’s definition of health:

*“Health is a state of complete physical, mental and social well-being, and not merely an absence of disease and infirmity”*

- 2.1.2 The authors of the Wilson Report state that as people’s well being is diminished by noise; there can be no doubt that noise affects health.
- 2.1.3 Even as early as 1963 the authors heard evidence presented to them, which highlighted the problem of aircraft-induced sleep disturbance. In a social survey conducted on people living near London Airport at this time 22% said that they were sometimes kept from falling asleep by the noise of aircraft, and the proportion rose to 50% with very high levels of noise. Results indicated that a higher proportion, also increasing with noise intensity, complained that they were sometimes awakened by noise. The authors noted that it is important to limit noise during the earlier part of the night, when people are falling asleep, due to the decrease in likelihood of awakening during deeper phases of sleep later on in the sleep period.
- 2.1.4 Work carried out by NASA in the early 1970s (LeVere *et al*; 1972) looked at the effects of the timing of subsonic aircraft flight over various stages of the night, and their effect on sleep. EEG recordings were used to establish the relative change in brain activity when exposed to aircraft noise at different times of the night compared to baseline, or quiet nights. Seven of the fourteen study nights, excluding the first three baseline nights, were selected at random to be the noise conditions. Each recorded jet flyover was played back to reach a loudness of approximately 80 dBA, with an approximate duration of 20 seconds, according to a predetermined random schedule over six hours of sleep. Changes in the EEG recordings were obtained for each third of the night and analysed to obtain the degree of response to the jet aircraft noise. The results indicated that the response to jet noise stimuli were significant for each portion of the night, and outlasted the length of the flyover by a considerable amount. Interestingly, the effects were more pronounced in the first and last thirds of the sleep period, with the mean change in brain activity being significantly lower in the middle two hours of the sleep duration (difference between early and middle  $p = 0.047$ ; difference between middle and late  $p = 0.016$ ). It is worthy to note that specific sleep stages or awakenings were not examined, rather a mean value of cortical arousal for each of the three epochs, with the early and late periods being those that are more likely to correspond to the times that subjects are more likely to be trying to fall asleep, and beginning to wake up. The authors concluded that this result in particular indicated that further investigation into the timing of scheduled aircraft noise would be worthwhile.
- 2.1.5 Follow-up work at NASA (LeVere and Davis; 1977) found that a 15 dBA reduction in aircraft flyover noise results in less sleep disturbance but only during fast-wave EEG activity. Slow-wave sleep, the highest proportion of which occurs in the first half of the night and is characterised by low frequency, high amplitude delta waves, was unaffected by this reduction in overall noise. Furthermore, although the effects of the 15 dBA reduction were noticeable on the EEG, it was not subjectively noticed by subjects in terms of self-assessed sleep quality. This finding questions whether simply

reducing the noise level is beneficial to sleep; and still suggests that the timing of noise-exposure is likely to be pertinent.

- 2.1.6 Lucas also investigated the effects of aircraft noise on human sleep (Lucas, 1972) in terms of the response of sleeping subjects to the stimulus of simulated sonic booms and subsonic jet activated noise. The results suggested that children were relatively non-responsive to the stimuli, and in general the likelihood of awakening increased with age. The responses to the two types of stimuli did not differ, and the intensity of stimulus had little, if any effect on the frequency of arousal.
- 2.1.7 Cardiovascular effects of aircraft noise were also investigated around this time. Griefahn studied the effects of sonic booms on changes in pulse rate during sleep in 1975 (Griefahn, 1975). The sound level of the sonic booms were 83.5 dBA on average and were applied alternately either twice or four times per night for thirty nights. The booms were presented between 2200 and 0300. Following ten more noise-free nights, four nights of eight and sixteen booms alternately were presented. The interval between noises was 40 minutes in nights with two booms, 20 minutes in nights with four booms and in the nights with eight and sixteen booms, eight and four minutes respectively. The timing of the first boom was applied when a subject entered the deepest sleep stage. Pulse rate initially increased in frequency with a maximum in the fourth second, and then decreased below the level prior to the noise and then slowly increased to baseline level once more. No correlation was found between the intensity of the boom and the pulse reaction, or between the stage of sleep and the reaction. However, a highly significant correlation was found between the maximum post-boom increase of pulse rate and the rate prior to the boom, with the reaction becoming smaller as the pulse rate increased.
- 2.1.8 In 1977 the Minister for Companies, Aviation and Shipping commissioned a study into aircraft noise-related sleep disturbance (DORA Report 8008). The Directorate of Operational Research and Analysis (DORA) of the Civil Aviation Authority (CAA) conducted the study, and the three main aims were:
- To establish the nature and scale of all sleep disturbance from all causes around Heathrow and Gatwick airports
  - To assess the significance of aircraft noise in causing sleep disturbance
  - To investigate the relationship between exposure to aircraft noise and the degree of sleep disturbance.
- 2.1.9 Surveys were administered by post and face-to-face interview to a wide range of inhabitants living around Heathrow and Gatwick, together with an accompanying noise measurement programme and examination of the pattern of movements by aircraft at night. The main findings were:
- a) Disturbance, such as difficulty in falling asleep, awakening during the night and tiredness on waking occurred frequently irrespective of aircraft noise. For example, on the designated night, at sites where little or no aircraft noise was heard, typically about a quarter of the population sampled reported difficulty in getting to sleep, while in response to a question on awakenings, a third of the sample said they awoke more frequently than once a week.
  - b) The researchers concluded that the measure  $L_{Aeq}$  'Equivalent Continuous Sound Level', corresponding to the total noise energy produced by aircraft during the period 2300–0700, was a satisfactory measure of aircraft noise exposure i.e. it correlated well with sleep disturbance.



- c) The total disturbance of sleep, irrespective of attributed cause, showed a slight increase at higher  $L_{Aeq}$  levels. For example, the proportion of people who claimed to wake more than once a week increased from 30% for  $L_{Aeq}$  of around 40 dB, to 40% at the noisiest sites with  $L_{Aeq}$  values of about 65 dB.
- d) The disturbance attributed by respondents to aircraft noise increased more substantially as  $L_{Aeq}$  values increased i.e. the increase was greater than the corresponding increase in total reported disturbance. When asked about awakening, about half the respondents at the noisiest sites (65 dB  $L_{Aeq}$ ) gave aircraft noise as a main cause compared with a tenth at the sites with least aircraft noise (40 dB  $L_{Aeq}$ ).
- e) Although total disturbance was similar at Heathrow and Gatwick, respondents tended to attribute their disturbance to aircraft noise to a greater extent at Gatwick than those at Heathrow.
- f) The proportion of people who indicated difficulty falling asleep was higher at those sites where there was greater exposure to aircraft noise between 2200 and 2400.

2.1.10 The CAA/DORA study looked at subjective sleep disturbance with respect to aircraft noise, but valuable contributions into the effects of road traffic noise on sleep changes were also being made at this time, that could also be applied to aircraft noise. The long term effect of sleep disturbance due to traffic noise was investigated in people living near a main road and who had been exposed to noise for more than four years (Vallet *et al*, 1982). The findings indicated that young people show decreases mainly in sleep stages 3 and 4, and REM sleep deficits are seen in older people. In terms of cardiac responses, both maximum levels and average were important, with threshold levels of 37 dB  $L_{Aeq}$  and 45 dB  $L_{Aeq}$  at which a decrement in sleep quality is observed.

2.1.11 A different laboratory study examining the effects of traffic noise (Öhrstrom and Rylander, 1982) involved exposing subjects to intermittent and continuous noise during the night, finding a dose-response relationship between intermittent noise and subjective sleep quality. Similarly, this was also the case for body movements immediately following noise peaks during the nights with intermittent noise, and performance and mood were both decreased after this condition, but not following continuous noise nights.

2.1.12 Noise and social survey data were used from 673 respondents to develop a model of aircraft noise annoyance, including sleep disturbance, in the vicinity of Toronto International Airport (Taylor, 1982). The strongest direct effects were found for speech interference, attitudes toward aircraft operations, sleep interruption and personal sensitivity to noise.

2.1.13 This section has summarised the main contributions to the effect of aircraft noise-induced sleep disturbance understanding prior to 1990.

### **3 The effects of aircraft noise on sleep structure, alertness, mood and performance**

#### **3.1 Field Studies into Aircraft Noise and Sleep**

- 3.1.1 In 1992 the findings of a study into aircraft noise and sleep disturbance, commissioned by the Department of Transport from the Department of Safety, Environment and Engineering at the CAA, were published (Ollerhead *et al*, 1992).
- 3.1.2 The objectives of the study were to determine:
- The relationships between outdoor aircraft noise levels and the probability of sleep disturbance.
  - The variation of these relationships with time of night
- 3.1.3 Non-acoustical factors were also examined, such as age, sex, personal characteristics, and views of the neighbourhood, perceptions of sleep quality and the ways in which this might be affected by aircraft noise.
- 3.1.4 This study predominantly used social survey methods, with actigraphy and EEG recordings on a sub-group of participants, to enable validation of the actigraphy with respect to aircraft noise-induced sleep disturbance. The pilot study, conducted in 1990 involved a single site near Manchester Airport, and concluded that although actigraphy was a suitable measurement of sleep disturbance, additional sleep EEGs would be required to calibrate the results in the main study. It was found that the link between noise exposure and sleep disturbance was relatively weak and other factors (e.g. psychological) were identified as having an important role and required further investigation. In order to gain statistical significance, 50 subjects would need to be monitored for at least two weeks in the main study.
- 3.1.5 The main study used eight sites; two around Heathrow, Gatwick, Stansted and Manchester Airports, and were selected for a range of SEL,  $L_{Aeq}$  and N combinations. 200 subjects completed social surveys, with 50 of the subjects also completing 15 nights of actigraphy, sleep logs and daytime sleepiness questionnaires. From these 50 subjects, six had their EEG recorded simultaneously with the actigraphy for four consecutive nights at each site. In total almost 6,000 subject nights of data were collected, making it, at the time, the largest field study of aircraft noise and sleep disturbance undertaken.
- 3.1.6 The main conclusions to be drawn from the study were that actigraphy was a cost-effective, useful method of measuring sleep arousals in subjects participating in their own home, and that aircraft noise was a relatively minor cause of such arousals. Actigraphy was able to detect around 90% of awakenings of 10-15 seconds or more and can detect a large number of minor arousals, including brief awakenings, some sleep stage changes, and minor body movements. However, it should be noted that all of these characteristics occur naturally during normal sleep. Those subjects who reported awakenings often did not state a cause (26%) and of those who did, aircraft noise was found to be one of the minor causes, with less than one quarter of all subjects attributing this factor, on average about once every five nights.
- 3.1.7 The results suggested that below outdoor event levels of 90 dBA SEL (about 80 dB  $L_{Amax}$ ), Aircraft Noise Events (ANEs) are most unlikely to cause any increase in measured sleep disturbance from that which occurs naturally during normal sleep. For

those ANEs above this level, the average arousal rate was about 1 in 30, corresponding to a wakening rate of about 1 in 75.

- 3.1.8 Indications from the results measured in 15-minute periods showed that sleep arousals increase as a function of time throughout the night, which is consistent with the 90-minute duration of the sleep cycle. This finding suggested that people might show increased sensitivity to noise at certain times of the night. The authors concluded that sensitivity to aircraft noise is low during the first part of sleep, and increases until 0300-0400, and then decreases to a low level at the end of the night again, but it is important to remember that measurement is by actigraphy rather than EEG and so cannot detect all subtle changes in sleep structure. In general, males were found to be 15% more susceptible to disturbance (with or without aircraft noise), and other factors such as time of night, and the incidence of disturbance in the period preceding the ANE also have a bearing on the relationship between aircraft noise and sleep disturbance.
- 3.1.9 Horne, a co-author on this study, also published these findings in 1994 (Horne *et al*, 1994). It is important to consider that there are individual differences in terms of arousals in normal sleep, and so this is also the case in relation to aircraft noise. He reported small age and gender effects, which became apparent at about 180 minutes into sleep and increased towards the end of sleep, with males exhibiting more sleep disturbance than women, in general and as a result of aircraft noise. In terms of age for both genders, younger people (20–34yrs) moved around more during sleep, which is somewhat unexpected.
- 3.1.10 The findings from this field study suggest that the extent to which people experience sleep disturbance due to aircraft noise is much less pronounced in field studies where they are sleeping in their own home, compared to laboratory studies, where subjects are sleeping in unfamiliar surroundings and beds etc. The sleep of most subjects was largely unaffected by ANEs. The louder the ANE, the greater likelihood of an effect on sleep, but the response to louder ANEs (e.g.  $L_{Amax} > 80$  dB, outdoors) was still very low on average (1 in 75). In this study, the most disturbing factors were given as young children, illness, needing to go to the toilet and bed partner, and aircraft noise ranked relatively low as a cause of sleep disruption.
- 3.1.11 A further publication (Horne *et al* 1995) arising from this important study, examined the patterns of spontaneous and evoked body movements during sleep in the actigraphy and EEG data. In addition to the above conclusions, the authors also reported that although movement increased over sleep, the likelihood of an ANE-evoked response did not, and they both differ in rhythmicity. Analysis of the EEG data in more depth revealed that the responsiveness to aircraft noise specifically, seemed to be lower during Rapid Eye Movement (REM) sleep, whereby surges in REM were associated with depressions in aircraft noise-induced movement, after the first hour of sleep and for the next 4.5 hours ( $r = -0.57$ ;  $df = 17$ ,  $p < 0.01$ ), after which the association stops. The number of spontaneous movements was highly negatively correlated with Slow Wave Sleep (SWS). As SWS decreased the incidence of spontaneous movement (aircraft related or not) increased markedly ( $r = -0.67$ ,  $df = 23$ ,  $p < 0.01$ ), with surges in SWS coinciding with troughs in spontaneous movement.
- 3.1.12 The low rate of awakening found by the study (1 in 75) has been strongly criticised. However, DETR (1998) used this value, together with the number of flights and number of people exposed by each flight between 2300 and 0700 to estimate that between 7,000 and 9,000 awakenings occur nightly at Heathrow airport.

- 3.1.13 Griefahn *et al* (2000) reported the results of a study investigating physiological, subjective, and behavioural responses to noise from rail and road. Participants were studied using social survey ( $n = 1,600$ ) in eight areas exposed to road or rail noise, and actigraphy ( $n = 377$ ) for two periods, each consisting of five nights. Subjects gave information on whether the windows had been open or close during the monitoring periods, and qualitative and quantitative aspects of sleep together with the results of a reaction time performance test were also collected. The only significant association was between the windows being closed and those people likely to live in areas exposed to road noise. No other difference was recorded in terms of performance, body movements and subjective assessment of sleep parameters. The authors suggest that varying the sound pressure levels in future research may be useful.
- 3.1.14 The suggestion that there is a circadian pattern of sensitivity to aircraft noise, as found by Horne *et al* was echoed by Hume *et al* (2003) who looked at the complaints caused by aircraft operations, in terms of noise level and time of day.
- 3.1.15 The authors looked at the data on complaints, noise monitoring, aircraft flight paths and movements to assess annoyance due to time of day at Manchester airport. The louder the noise the more complaints were generated, with twice the complaints at 110-114 PNdB (approx. 97-101 dB  $L_{Amax}$ ) compared to at 74-79 PNdB (approx. 61-66 dB  $L_{Amax}$ ). The hourly pattern in flight frequency and complaints were distinct, and complaints per aircraft movement for each hour showed a 24-hour pattern with the night flights causing on average nearly 5 times more than the rest of the day. Greatest propensity to complain was at 0100-0200 and the lowest at 0800-0900, which suggests a circadian pattern in sensitivity to aircraft noise.
- 3.1.16 Diamond *et al* (2000) undertook a study (by interview and questionnaires) of the perceptions of aircraft noise, sleep and health around major UK airports. They found that:
- Sleep disturbance attributed to aircraft noise was associated with greater health problems.
  - Where night noise is relatively high, it causes annoyance to local residents and at two of the airports studied annoyance due to night noise exceeds that due to day time noise.
  - Where noise is relatively high, between 10% and 20% of respondents reported having difficulty getting to sleep at night and being woken up in the morning.
  - Very few people reported that their health was “extremely affected” by aircraft noise at night. However, between 30% and 60% of respondents at the various sites perceived their health to be “somewhat affected”.
  - Respondents who reported long term or recent physical or mental problems, or stress in their job or in their life generally, were more likely to report their health was affected by aircraft noise at night.
- 3.1.17 Field studies have been used to assess sleep by actigraphy, but also to obtain large samples of questionnaire data relating to general health and medication in relation to aircraft noise exposure (Franssen, 2004).
- 3.1.18 A cross sectional design was employed to obtain survey responses from 11812 subjects living within a 25km radius of Schiphol airport. Associations were significant for all health indicators per 10 dBA increase in  $L_{den}$ , except for use of prescribed and frequent use of sleep medication or sedatives. None of the health indicators were associated with aircraft noise exposure during the night, but use of non-prescribed sleep medication or sedatives was associated with aircraft noise exposure during the

late evening. Health complaints such as vitality, headache and tiredness were related with aircraft noise exposure, whereas other physical health complaints were not. The results suggested an association between community exposure to aircraft noise, and the health indicators “poor general health status”, “use of sleep medication”, and “use of medication for cardiovascular diseases”. The effect of aircraft noise on the cardiovascular system during sleep will be discussed in more detail in Section 4.

3.1.19 Michaud *et al* (2007) published a review of field studies of aircraft noise-induced sleep disturbance to examine the prevalence of disturbance. The effects of noise on sleep are mediated by many factors such as sound level, number, duration, time of occurrence, short- and long-term intermittency and consistency of distributions of aircraft noise intrusions into sleeping quarters. He looked at findings between 1990 and 2003, with regards to the ability of aircraft to:

- Interfere with the ability to fall asleep
- Curtail sleep duration
- Lessen the perceived quality of sleep
- Awaken people from sleep
- Increase bodily movements during sleep

3.1.20 Alongside work that has already been referred to (Ollerhead 1992; Hume 2003), Michaud *et al* (2007) describe the work done by Fidell (1995a, 1995b) which was a field study of 1-month duration on 27 people living near the main runway of a military airfield, and 35 subjects living near Los Angeles International Airport. A further 23 people living in neighbourhoods without appreciable noise exposure were controls. Subjects were asked to press a button on an awakening from sleep, for any reason. No actimetric or EEG measurements were made in this study, but questionnaires for subjective sleep quality, recalled awakenings, sleep latency and subjective tiredness were completed. Fidell *et al* attributed 16% of awakenings to noise events, and like Ollerhead found that the likelihood of awakening due to noise increased with time throughout the night. The subjective reports of tiredness in the evening were related to awakenings by noise events the previous night.

3.1.21 The mean indoor SEL for awakening was 81 dBA, and mean SEL that failed to awaken was 74 dBA. Taking into account a typical 15 dB for outdoor to indoor attenuation, these levels correspond to 96 and 89 dBA, very similar to the findings of Ollerhead *et al* (1992). Although greater SEL values were associated with a greater likelihood of awakening to aircraft noise, the slope of the relationship was not steep i.e. increase of 10 dB in SEL was only associated with a 1.7% increase in awakenings. Cumulative noise exposure throughout the night did not predict sleep disturbance and hence the study did not support adoption of  $L_{\text{night}}$  as a useful predictor.

3.1.22 Michaud summarises his review as follows:

“The literature review of recent field studies of aircraft noise-induced sleep disturbance finds that reliable generalisation of findings to population-level effects is complicated by individual differences among subjects, methodological and analytic differences among studies, and predictive relationships that account for only a small fraction of the variance in the relationship between noise exposure and sleep disturbance. It is nonetheless apparent in the studied circumstances of residential exposure that sleep disturbance effects of night-time aircraft noise intrusions are not dramatic on a per-event basis, and that linkages between outdoor aircraft noise exposure and sleep disturbance are tenuous. It is also

apparent that aircraft noise-induced sleep disturbance occurs more often during the later part of the night; that indoor sound levels are more loosely associated with sleep disturbance than outdoor measures; and that spontaneous awakenings, or awakenings attributable to non-aircraft indoor noises, occur more often than awakenings attributed to aircraft noise.”

- 3.1.23 Another study by Fidell (2000) used Behaviourally Confirmed Awakenings (BCA) and motility as indices of sleep disturbance, in Denver, Colorado. The study observed the sleep behaviour of subjects living near the airport, during a time when aircraft noise was reduced due to anticipated closure of Stapleton International Airport, coupled with an increase in aircraft noise for the residents living near to the new Denver International Airport, prior to opening. The age range of subjects was from young adults to the elderly and evenly distributed by gender. Morningness-Eveningness questionnaires were administered to assess diurnal preference i.e. whether people are morning or evening types, and actimetric and behavioural awakening measurements of sleep disturbance were made in 30-second epochs during 3 night-time periods: 0100-0130, 0300-0330, and 0500-0530. The percentage noise-induced behavioural awakenings (BCA) increased 0.25% per 1 dB increase in indoor SEL. For each increase of 1 dB in ambient  $L_{Aeq}$  levels, the actimetric and BCA responses due to noise events fell by 2-6%. Noise events were more likely to awaken men than women. Once the airport had opened, a statistically significant decrease in BCA was found, despite a large increase in indoor noise events. Prior = 1.71, after = 1.13. After closing of the DEN airport, BCA were not significantly different from each other, probably due to the levels of indoor noise events not changing notably (1.8 vs 1.64), although outdoor levels decreased from 58-46 dBA.
- 3.1.24 A further study by Fidell *et al* (2000) looked at sleep disturbance in 22 subjects with respect to anticipated increase in traffic prior to, and following the Atlanta Olympic Games. The number of noise events between 76 and 80 dB  $L_{Amax}$  increased slightly during the games. BCA were greatest prior to the games and fell from 1.8 to 1.2 per night during the games, and 1.0 afterwards. The indoor SEL predicted actimetrically monitored arousals, while outdoor SEL predicted BCA. Even at high noise levels most people were not awakened by aircraft overflights.
- 3.1.25 Passchier-Vermeer *et al* (2002) examined sleep disturbance in the vicinity of Schiphol airport in 418 subjects aged between 18-81 years, from 2200-0900 within bedrooms and at outdoor locations over 11 days. Sleep quality questionnaires were completed in the morning and evening, as were recalled awakenings due to aircraft noise, annoyance due to aircraft noise and motility. Actiwatch event markers were also used, whereby subjects pressed a button on the activity monitor to indicate they had been awakened. Subjective sleepiness ratings were taken five times a day in designated periods, and performance was measured by reaction time on a task to assess the effects of sleep loss on performance.
- 3.1.26 Aircraft noise effects were assessed on “instantaneous”, 24-hour and long-term effects. Instantaneous effects included motility was defined as movement occurring within any 15-second interval of an aircraft noise event, and aircraft noise-induced onset motility as movement within a 15-second epoch immediately following an interval in which movement had not occurred directly before. The 24-hour scale included sleep period, subjective measures such as sleep quality and BCAs. Long-term effects looked at the mean motility over the 11 nights, questionnaire responses, and indoor and outdoor noise metrics.
- 3.1.27 The results can be summarised as the following:

Instantaneous effects: ANEs increased the probability of motility and the onset of motility. Instantaneous measures were influenced by the average equivalent indoor ambient sound level assessed over the 11 sleep episodes. When this was low, the probability of motility due to aircraft noise was higher, especially at the higher  $L_{Amax}$  levels. Motility probability also increased as a function of time after sleep onset. I.e. was higher at the end than at the beginning of the night. In terms of age, motility peaked at in those subjects at 46 years of age. The study concluded that the probability of motility and the onset of motility had threshold levels of 32 dB  $L_{Amax, indoors}$  and 38 and 40 dBA  $SEL_{indoors}$  respectively. Outdoor to indoor attenuation was 21 dB. Average thresholds were found to be about 15 dBA lower than by Ollerhead et al (1992).

24-hour effects: There was a significant increase in mean motility during sleep, number of BCA, and number of recalled awakenings due to aircraft noise as a function of indoor equivalent aircraft sound level, and number of aircraft during the sleep period time. Mean motility over the night increased when:

- Average noise within the bedroom not due to aircraft increased
- When the transmission loss from outdoors to indoors was low
- When subjects indicated a difficulty falling asleep due to aircraft noise
- And in those subjects who attributed awakenings to aircraft noise exposure

When aircraft noise was given as cause for trouble falling asleep, sleep latency was about 15 minutes. Perceived sleep quality reduced as motility increased but indoor aircraft sound levels and numbers of aircraft were not related to perceived sleep quality. Perceived difficulty in falling asleep had a stronger influence on perceived sleep quality, fatigue, the number of subjectively recalled awakenings, and the number of BCA. Aircraft exposure at night appeared to have no impact on reaction time as a measure of performance.

Long term effects: When the average sound level within the bedroom over the 11 days increased, mean motility was also higher and sleep latency increased. Mean motility also related to frequency of recalled awakenings, BCA, sleeping medication use, sleep quality, general sleep complaints, and number of health complaints.

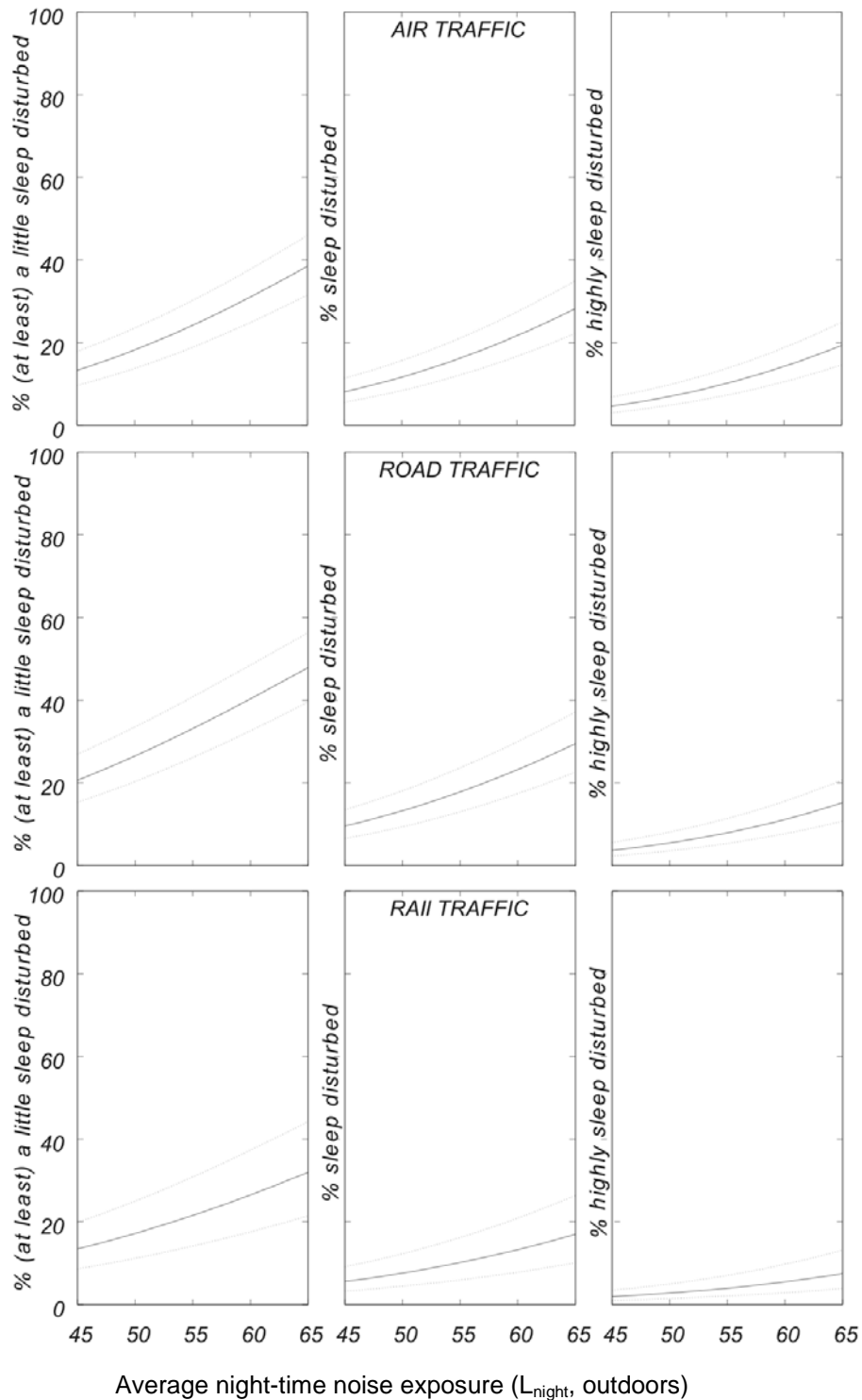
- 3.1.28 Michaud (2007) explains that the findings of the studies are not conclusive in terms of the effects of aircraft noise on changes in sleep states that do not result in awakenings. Neither behavioural awakenings nor motility measurements are capable of detecting more subtle interference with sleep quality, e.g. brief changes in stage or “microarousals” that might also reflect a state of disrupted sleep. He suggests that there is some agreement in terms of spontaneous awakenings being more common than aircraft noise-induced awakenings in airport neighbourhoods; a small percentage of people are awakened by aircraft noise, and although the propensity for noise-induced awakening increases with time spent in bed this is confounded by the fact that sleep is more easily disrupted with time anyway, so noise events in the latter half of the night are therefore more likely to wake people than in the earlier half anyway.
- 3.1.29 Öhrström *et al* (2006) studied the effects of road traffic noise on sleep in children and adults in Sweden. Although this paper did not measure the effects of aircraft noise on sleep, it is useful to investigate the differences between adults and children. 160 children between the ages of 9 and 12, and 160 parents were interviewed. Half of the families were measured with actigraphy and sleep logs. In the parents, a significant exposure-effect relationship was found between road noise levels and sleep quality, awakenings, keeping windows closed at night, and perceived interference with traffic noise. For children a significant exposure-effect relationship existed between road

traffic noise and sleep quality, and also daytime sleepiness. Children had a better-perceived sleep quality and fewer awakenings than parents; however actigraphy records indicated that the parents actually experienced better sleep.

- 3.1.30 Miedema and Vos (2007) have performed a meta-analysis of 28 datasets from 24 field studies into transport (aircraft, road and rail) noise and sleep disturbance. Re-analysis of existing data was performed because functions based on individual studies used different noise-exposure metrics and sleep disturbance variables, thereby making results difficult to compare. Contrary to previous studies finding that sleep disturbance correlated best with individual aircraft noise events, outdoor  $L_{\text{night}}$  was used for this analysis due to it being more widely available from existing study data. It was assumed that the outdoor-indoor differences and noise exposures at different sides of the building were treated as random factors. The data was translated to a scale of 0-100, and grouped into percentage (at least) a little sleep disturbed, percentage sleep disturbance, and percentage highly sleep disturbed (**Figure 3**). The confidence intervals illustrate that at the same average night time exposure levels, aircraft noise is associated with more self-reported sleep disturbance than road traffic noise, and road traffic noise is associated with more sleep disturbance than railway noise. The functions may be useful for evaluating night time noise exposures of a population (this analysis is not suitable for predicting individual reactions). At a given night time exposure level, self-reported sleep disturbance is maximal in people in their 50s, with road traffic and railway noise at age 50 years and for aircraft noise at age 56 years. This work has contributed to the debate on threshold levels for the protection of public health.  $L_{\text{night}}$  is also the night-time indicator required for mapping of major transport noise within the EU every five years, beginning in 2006.



**Figure 3:** Taken from Miedema (2007). The functions that specify three sleep disturbance measures (solid lines) in relation to the average night time noise exposure outside, and their 95% confidence intervals (broken lines) for air traffic, road traffic, and railway



## 3.2 Polysomnographic studies

- 3.2.1 EEG recordings allow detailed examination of fluctuations in brain activity as a response to noise, or any other stimulus. Changes in sleep stages, microarousals and the presence of alpha wave activity (8-12Hz) can mean that the quality of sleep is compromised; despite subjects being unaware that this is occurring.
- 3.2.2 Griefahn (2002) describes the primary effects of noise on sleep beginning with subtle changes in the EEG such as the presence of K complexes, followed by an increase in brain activity often accompanied with body movements and autonomous responses.
- 3.2.3 The effects of noise accumulate over the entire sleep period and increase the total time spent in shallow sleep. The secondary effects of noise are impaired subjective sleep quality, mood and performance.
- 3.2.4 It is not always possible to assume sufficient habituation has occurred in laboratory studies, as in the field subjects often woke less often, spent more time in deep/REM sleep, rated sleep quality as better and performed better after sound attenuation.
- 3.2.5 Griefahn (2002) explains that sleep disturbances increase with age and with self estimated sensitivity to noise, also personality traits, and diurnal preference (morningness-eveningness), with critical noise loads for continuous noises appearing to be between equivalent sounds levels of 37 dBA and 40 dBA.
- 3.2.6 Previous work conducted by Griefahn *et al* in 1976 was used to establish noise-polluted areas in Germany, and concluded that the night-time wake-up thresholds of aircraft noise was 60 dBA. However, Maschke *et al* (2004) re-evaluated this data and concluded that maximum noise levels of 48 dBA was a more accurate figure for defining waking thresholds at ear level in sleeping subjects.
- 3.2.7 Passchier-Vermeer (2003) carried out an analysis of data from seven studies (including those of Ollerhead, Fidell and Passchier-Vermeer identified earlier) into behavioural awakening as a result of exposure to commercial aircraft noise exposure to populations. She developed a method to convert onset of motility or EEG awakening to behavioural awakening. Her analysis concludes that the onset of behavioural awakening due to exposure to aircraft noise is 54 dBA SEL (indoor).
- 3.2.8 Raschke (2004) explains that the type of noise, frequency spectrum, information content, duration of interval in repeated episodes, sequential number of sleep cycles passed through, exposure time in relation to the phase length of the circadian system, and age all have important roles in terms of the reaction to auditory stimulation. All functions have a modifying effect on the arousal threshold. Reaction to noise stimuli is multilayer, e.g. in response to sounds of 100Hz and 0.5-second duration, applied in the range of 43 to 80 dB with a 15cm distance, reactions can be seen in the EEG, momentary heart rate, continuously recorded non-invasive blood pressure, integrated sympathetic activity and tidal volume. All show short-time responses to the stimulus. Previous research suggests that the arousal reaction from sleep is mediated via the locus coeruleus and the raphe nuclei in the brain, where Orexin (the hormone concerned with energy metabolism and food ingestion) plays an important role as hormonal transmitter for intact sleep-wake regulation functioning.
- 3.2.9 Raschke argues that micro-arousals are non-applicable as indicators of sleep disturbances and noise disturbance in noise effects research since they are valued at between 10 and 20 per hour in healthy persons anyway, and can be considered as

normal in this range. This makes it difficult to separate out normal arousal during sleep, from those specifically induced by noise exposure.

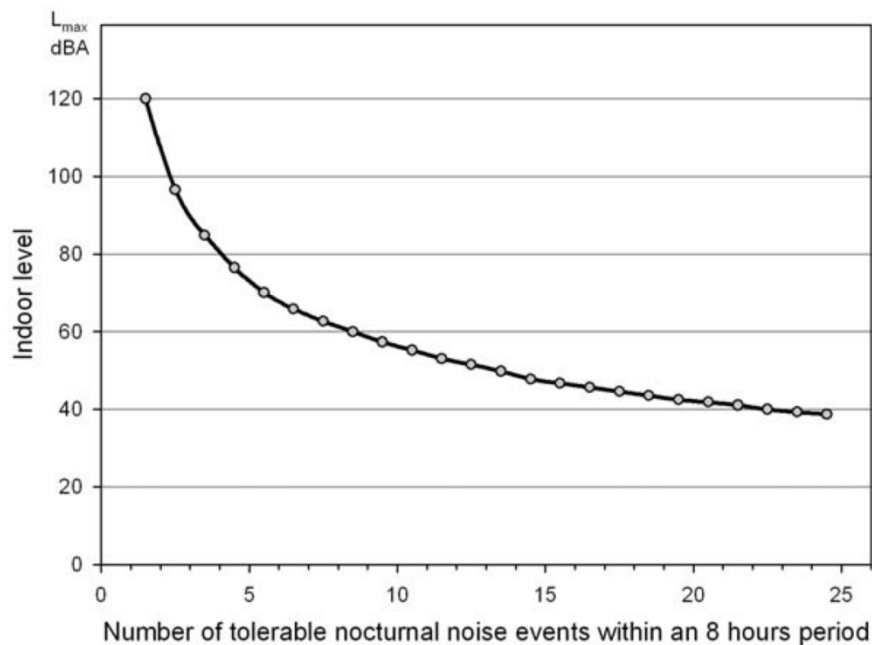
- 3.2.10 The effects of low frequency noise on sleep (as exhibited by aircraft) were studied by Persson Waye (2004). Low frequency noise (20-200Hz) typically propagates with little attenuation through walls and windows, therefore making many people exposed to such noise in their homes. Sleep disturbance is commonly reported in studies into low frequency noise.
- 3.2.11 The review gives indications that sleep disturbance due to low frequency noise warrants further concern. Amongst other studies it was found that in a cross sectional study performed on 279 people, no significant differences were detected in reported sleep among people exposed in their homes to flat frequency noise as compared to low frequency noise from ventilation/heat pumps (Persson Waye and Rylander 2001). It was found that fatigue, difficulty in falling asleep; feeling languid and tensed in the morning was reported to a higher degree among those annoyed by low frequency noise. Furthermore a significant dose-response relationship was found between reported annoyance and disturbed rest and degree of low frequency noise.
- 3.2.12 Those living in low-frequency areas have also reported higher incidences of chronic sleep disturbance, and depression compared to matched pairs not living in an area of low frequency. (Mirowska, 1998).
- 3.2.13 Although studies into aircraft noise are in the main performed on human subjects, sometimes it can be useful to investigate the effects on animals to gain insight into the processes behind the reaction. Rabat (2004) looked at the deleterious effects of an environmental noise on sleep and contribution of its physical components in a rat model.
- 3.2.14 The aim of this study was to confirm the effects of noise on sleep in a rat model and to determine the most deleterious physical component of noise regarding sleep structure.
- 3.2.15 Rats were exposed during 24-hours to environmental noise (EN) or artificial broadband noises (either continuous broad-band noise CBBN or intermittent broad-band noise IBBN). There have been conflicting findings in human fields studies as to the effects of one, or both CBBN and IBBN on REM and SWS, showing an effect on REM, SWS, none or both. The discrepancies may be down to individual variability in psychological sensitivity to noise, socioeconomic situation, differential cognitive processing of noise, or the use of pure tones.
- 3.2.16 All noises decreased both SWS and REM during the first hours of exposure. CBBN acted indirectly on REM through a reduction of SWS bout duration, whereas IBBN and EN disturbed directly and more strongly both SWS and REM. EN fragmented SWS and decreased the REM amount during the dark period, whereas IBBN only fragments REM sleep. Two physical factors are implicated, the intermittent and the frequency spectrum of the noise events, which both induce long-lasting sleep disturbances. An additive effect of frequency to intermittency tends to eliminate all possible adaptations to EN exposure, which could potentially lead to cognitive deficits. This may be worth considering when investigating the effect of noise on cognitive performance.
- 3.2.17 Basner and Samel (2004) at the DLR Institute for Aerospace in Germany conducted a large-scale, multi-stage study that aimed to investigate the acute effects of nocturnal aircraft noise on human sleep.

- 3.2.18 The authors reported that there have been conflicting findings in terms of assigning a threshold over which sleep disturbance is more likely to occur. Jansen (1995) assumed that the first changes in sleep depth induced by noise events are at a maximum level of 55 dBA, and awakenings at more than 60 dBA. However, these were individual observations without statistical evidence. Therefore the 60 dBA was assumed to be a theoretical benchmark from which to work. However in 1976 Greifahn *et al* tried to find an average value at which awakening was most likely and this figure came to around 60 dBA also, (SD 7 dB). Maschke *et al* did not agree with this and their calculation in 2001 gave a range of between 0 dBA and 48 dBA. These authors conclude from their new calculation that awakening is to be anticipated at 48 dBA with a probability of 95%. These newly calculated results contradict those derived by Maschke himself in 1992 where he deduced that the lower threshold for sleep stage changes should be set at a  $L_{Aeq}$  level of 36 dB, becoming particularly noticeable at  $L_{Aeq}$  50-56 dB. These results were taken from a sample size of  $n=40$  over 5 nights, but with no control group. A sub-sample of eight participants were exposed to sound over ten nights in order to examine catecholamine secretion (a measure of stress) in overnight urine samples. The results indicated a higher adrenaline secretion at 65 dBA than at 75 dBA. However, this was a small sample size and therefore it is difficult to attribute cause and effect.
- 3.2.19 The DLR study used a double blind crossover design. ANEs with differing distributions of  $L_{Amax}$  and frequency of occurrence were played back in pre-calibrated sleeping rooms while the physiological reactions were recorded. 128 subjects were investigated in the lab and 64 in the field, with an equal distribution of age, gender and prior exposure to aircraft noise.
- 3.2.20 Nights one and two were familiarisation and baseline nights, then subjects were exposed to 9 nights of aircraft noise with a varying distribution of  $L_{Amax}$  and rate of occurrence. Noise was played at regular intervals between 11.15pm and 6.45am. Eight subjects were exposed to the same pattern and level of noise per noisy night. The maximum level of an individual noise was between 50 and 80 dBA at the ear of the sleeper and the number of events per night ranged between 4 and 128 (i.e. intervals of between 3 minutes and 2 hours between noise events). These combinations were distributed over the 9 noise nights randomly and lead to continuous sound levels  $L_{Aeq}$  between 31.2 and 52.6 dB. The last two nights of the study were kept free of aircraft noise for comparative purposes.
- 3.2.21 EEG, Electro-oculogram (EOG), electromyogram (EMG), electrocardiogram (ECG), and finger pulse and respiration rates were all recorded. A test battery of memory and search tasks, reaction time, and a tracking task was also administered, along with questionnaires on mood, stress and recuperation, fatigue and flight-noise. The noise level indoors and outdoors was synchronised with the electrophysiological parameters to establish any relationship between aircraft noise and physiological reactions.
- 3.2.22 The difference in baseline and noise nights included a significant 9-minute reduction of SWS and an increase of stage 1 by 3.8 minutes. Therefore although total sleep time was not reduced significantly, the sleep architecture was considerably altered as a result of aircraft noise.
- 3.2.23 The percentage probability of awakening increased with  $L_{Amax}$  when the number of events was kept constant at 32 (2000 noise events were analysed in total). For a constant  $L_{Amax}$  level of 65 dB, the probability of awakening decreased with the number of noise events per night i.e. the more frequent the noise the less chance it will lead to an awakening.

3.2.24 As Griefahn and Spreng (2004) report, sleep disturbance from noise characteristically begins with a K-complex (a biphasic EEG wave formation accompanied by altered autonomic function such as increase in heart rate, constricted peripheral blood vessels), and also by body movements. Depending on the nature and intensity of the sound, this initial reaction is followed by a more or less long lasting desynchronisation of cortical activity that reach from a flattening of sleep up to awakening, thereby causing more or less extended partial sleep deprivations.

3.2.25 The authors developed two models that allowed the calculation of noise and number combinations that cause the same predefined risk with respect to intermittent noise (Griefahn 1992, Spreng 2002). The physiological model proposed by Spreng (**Figure 4**) refers to the admissible noise-induced release of cortisol in the normal range and its results match almost perfectly the noise and number relation determined for awakenings reported in the DLR study by Basner and Samel (2004).

**Figure 4:** The relation between the indoor maximum levels  $L_{Amax}$  and the number of tolerable noise events within an 8-hour period during the night. (Spreng 2002)



3.2.26 Based on this model, indoor evaluation limits were derived for intermittent noise as shown in Table 1 (Griefahn et al 2004) and applies to aircraft noise, which, concerning transportation noise, annoys the most and is true for Night-time Annoyance as well (Health council of the Netherlands 1999).

**Table 1** Indoor evaluation limits derived for intermittent noise, taken from Griefahn et al (2004)

Maximum level	Equivalent noise level	
Critical Limit:	$L_{\max, 22-6\text{ h}} = 6 \times 60 \text{ dBA}^*$	$L_{\text{eq}, 22-6\text{ h}} = 40 \text{ dBA}$
Protection Guide:	$L_{\max, 22-1\text{ h}} = 8 \times 56 \text{ dBA}$	$L_{\text{eq}, 22-1\text{ h}} = 35 \text{ dBA}$
	$L_{\max, 1-6\text{ h}} = 5 \times 53 \text{ dBA}$	$L_{\text{eq}, 1-6\text{ h}} = 32 \text{ dBA}$
Threshold Value:	$L_{\max, 22-6\text{ h}} = 23 \times 40 \text{ dBA}$	$L_{\text{eq}, 22-6\text{ h}} = 30 \text{ dBA}$

\* Levels must not be exceeded

#### Unweighted distribution of air traffic over the entire night

	Maximum level	Equivalent noise level
Critical Limit:	$L_{\max} = 6 \times 60 \text{ dBA}^*$	$L_{\text{eq}, 8\text{ h}} = 40 \text{ dBA}$
Protection Guide:	$L_{\max} = 13 \times 53 \text{ dBA}$	$L_{\text{eq}, 8\text{ h}} = 35 \text{ dBA}$
Threshold Value:	$L_{\max} = 23 \times 40 \text{ dBA}$	$L_{\text{eq}, 8\text{ h}} = 30 \text{ dBA}$

\* Levels must not be exceeded

*Critical Limits* indicate noise loads that shall be tolerated only exceptionally during a limited time, health hazards and/or health impairments are no longer excluded. *Protection guides*, below which health hazards are unlikely should not be exceeded. Excessions give reason for counter measures. *Threshold values* indicate physiological responses to noise.

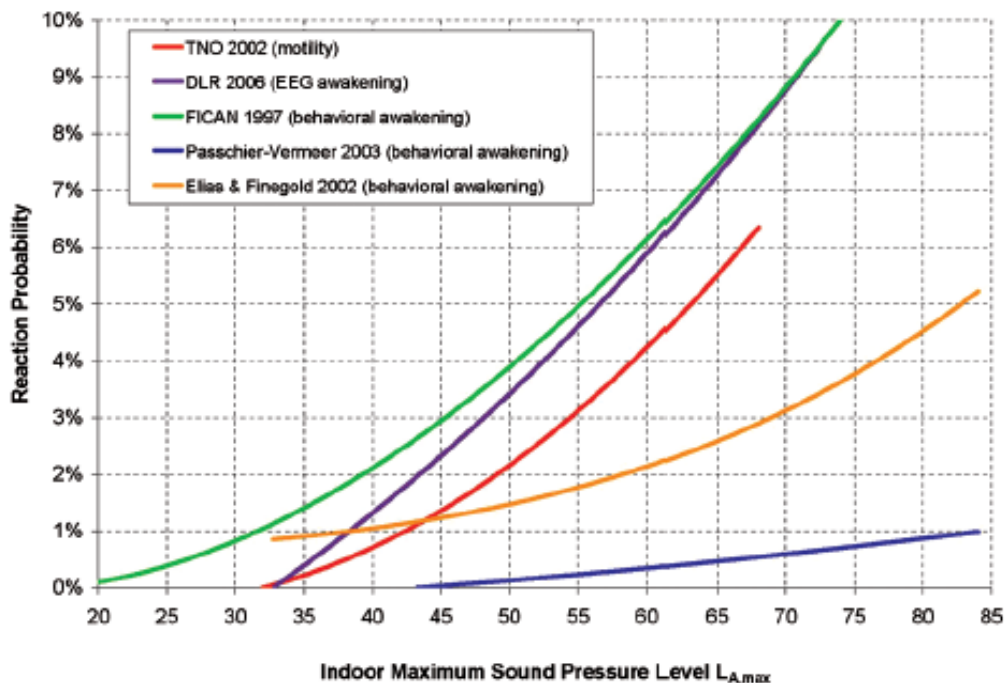
- 3.2.27 The result of sleep fragmentation, as is often caused by the response to aircraft noise can often mean impaired performance the following day, even if subjects are largely unaware that their sleep has been disturbed. Studies into the deleterious effects of aircraft noise on performance are rare, but could be important in our understanding of the way in which noise disturbance affects the brain. Schapkin *et al* (2006) looked at executive brain functions following exposure to nocturnal traffic noise. The term “executive” refers to those processes that are governed by the frontal lobes and pre-frontal cortex in the brain, and are considered to be complex, such as planning, decision-making, execution and inhibition of an action and are known to be particularly sensitive to sleep disturbance (Jones and Harrison 2001).
- 3.2.28 Impairments of neuronal mechanisms underlying overt performance after sleep disturbance were investigated using event-related potentials (ERPs). When the awake, subject has to detect rare stimuli, a large positive brain response with a 300ms peak latency (“target” P3) and with the parietal maximum as well as a P3 of smaller amplitude over the frontal sites are registered.
- 3.2.29 Fragmented sleep or sleep deprivation reduces the amplitude and/or lengthens the latency of the “frontal” P3. These data suggest impairments in executive functioning probably due to deactivation of frontal brain areas after sleep disturbance.
- 3.2.30 It was proposed that normal people who were exposed to nocturnal noise might also have moderate lengthening of the P3 latency, and/or reduction of its amplitude. The authors also proposed that the components related to inhibitory control (Nogo-N2 and Nogo-P3) will be more affected by noise-induced sleep disturbance than those related to target categorisation (Go-P3) and this effect will be stronger with increasing task difficulty. Dose-dependent after-effects were expected on performance and/or on ERP. Aircraft noise was applied during the four study nights with 3 equivalent noise

- levels ( $L_{Aeq}$ ) of 39, 44, and 50 dB and maximum values ( $L_{Amax}$ ) varied between 50 and 74 dB.
- 3.2.31 20 subjects were grouped into good or bad sleepers. The performance and inhibition related components (N2, P3) were smaller and latencies more prolonged in the difficult task, compared to the easy one. This effect was more pronounced for Nogo than for Go trials. Nogo-P3 amplitude was smaller in Noise than in “quiet” conditions in the difficult task only.
- 3.2.32 In the difficult task, the Nogo-P3 latency was prolonged in bad sleepers compared to good sleepers. The Nogo-P3 amplitude was reduced in Noise as compared to “Quiet” conditions in bad sleepers only. Sleep quality in bad sleepers worsened steadily with increasing noise levels. No effects of noise or subjective sleep quality on performance were found. Inhibitory processes appear to be selectively impaired after nocturnal noise exposure. The task difficulty and perceived sleep quality are important factors modulating noise effects. The results suggest that nocturnal traffic noise increase physiological costs for inhibitory functioning on the day even if no overt performance decrement is observed.
- 3.2.33 Basner et al (2006) published the results of their polysomnographic field study carried out between 1999 and 2004, investigating the effects of aircraft noise on mood and performance. Participants were between 19 and 61 years, free from sleep disorders and had normal hearing thresholds for their age. EEG, EOG, EMG, ECG, respiratory movements, finger pulse amplitude, position in bed and actigraphy were sampled.
- 3.2.34 Noise levels and actual sounds were recorded in the subjects’ bedrooms at the sleeper’s ear, and outside at a distance of 2m in front of the window. The beginning and end of each event were marked, and continuous monitoring of the subject in line with the ANEs allowed for a direct comparison of reactions to the noise.
- 3.2.35 Awakenings increased with the maximum level of an ANE. Awakenings induced by ANEs larger than 65 dB  $L_{Amax}$  were relatively short. Those awakenings induced by ANEs larger than 70 dB  $L_{Amax}$  were longer than spontaneous awakenings, and those below 65 dB  $L_{Amax}$ .
- 3.2.36 The authors concluded that there should be on average less than one additional awakening induced by aircraft noise a night. Noise induced awakenings recalled in the morning should be prevented as much as possible, and no relevant impairments of the process of falling asleep again should occur.
- 3.2.37 Griefahn *et al* (2006) found a difference in reactions to road, rail and aircraft noise in a sample size of 32 who slept with weekly changes between the noise conditions.
- 3.2.38 Comparison between the quiet nights of the control group and the noisy nights of the experimental group showed a difference between SWS latency, TST and a decrease of SWS during the first sleep cycle.
- 3.2.39 Sleep efficiency index was lower for all noise conditions, as was time spent in SWS, and REM sleep, and wakefulness after sleep onset (WASO) was higher than quiet nights for all conditions.
- 3.2.40 Most physiological variables showed strongest impairment under the impact of rail noise and smallest under the impact of traffic noise, with significance only reached on SWS latency, total time spent in SWS as well as for Stage 1 and wake, and SWS

during the first sleep cycle. Sleep quality was significantly reduced and fatigue increased, irrespective of noise type.

- 3.2.41 Sleep quality decreased with increasing sleep latency, latency to SWS and increasing WASO, with decreasing TST, and increasing amount of wake and stage 1, and decreasing amount of time in REM.
- 3.2.42 Executive, frontal tasks were used and a decrement in performance was found following noisy nights (switch and non-switch tests) and this increased with noise load. The correlation between RT and time spent in SWS suggests a model in which work speed is causally related via shortened SWS to the impact of noise during sleep.
- 3.2.43 **Figure 5** is included is taken from (Griefahn 2006), which summarises five field study dose-response curves for a single ANE, and reactions of the sleeper (for example, EEG awakenings, body movements, behavioural awakening)

**Figure 5: Comparison of five dose-response curves for a single ANE**



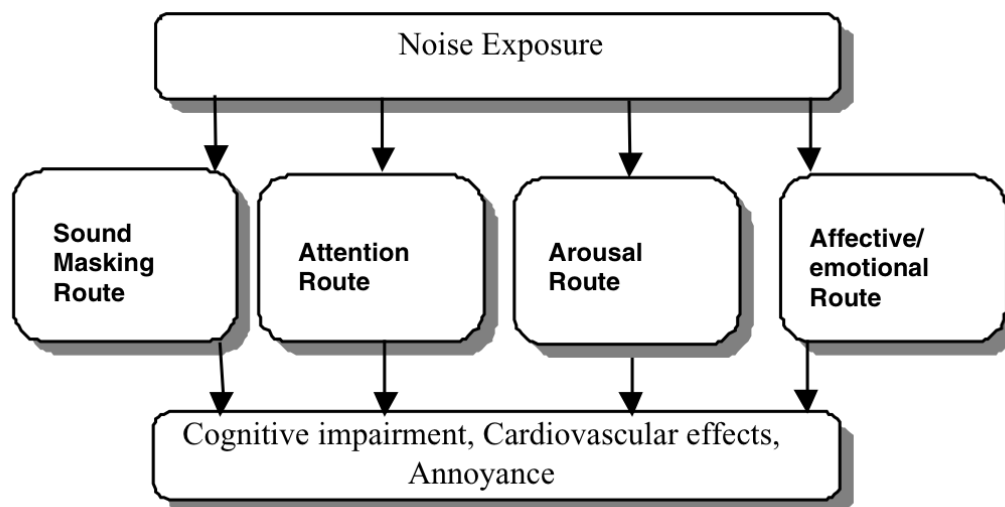
- 3.2.44 The FICAN curve (Federal Interagency Committee on Aviation Noise) is interpreted as predicting the 'maximum percent of the exposed population expected to be behaviourally awakened'. The heart rate and blood pressure of subjects was not habituated in the field, and the variance in awakening behaviour was also due to noise sensitivity, age, gender, current sleep stage, elapsed sleep time etc.

- 3.2.45 In terms of noise mitigation, the authors consider that traffic curfews should cover those times when most people are in bed trying to sleep. It is suggested that more information on the sleep habits of the population is required, and that shoulder hours may be need to be considered as an increase in traffic at these times could increase the effects on children, shiftworkers etc. It is concluded that in the future more research on noise mitigation measures is required, to assess their effectiveness in reducing noise induced sleep disturbance. The authors also suggest that future legislation should be based on both experimental studies of acute effects of noise exposure, as well as epidemiological studies on long-term health effects.



- 3.2.46 A recent study was conducted into the effects of aircraft noise on the macro- and microstructure of sleep, Basner *et al* (2007). 64 ANEs of maximum level of 45 dBA or 65 dBA were exposed to subjects over two nights, and compared to a baseline control night without noise. The authors found that the number of events per night increased in the order: awakenings, awakenings including changes to Stage 1 sleep, change to lighter sleep stage, and arousals, in that respective order, in control conditions as well as the two noise conditions. Arousals were four times as common as awakenings, irrespective of noise condition or control.
- 3.2.47 Miedema (2007) proposed a model of environmental noise disturbance as a stressor, impacting on behaviour (communication, concentration) and desired state (sleep and relaxation), with the ability to cope with such disturbance being important for health and well-being. The effects of noise depend on acoustical characteristics of the noise, such as loudness, time, pattern, and on aspects of the noise situation that may involve cognitive processing, such as expectations regarding the future development of the noise exposure, lack of short-term predictability, and a feeling of a lack of control over the source of the noise.
- 3.2.48 Miedema suggests that the model (**Figure 6**) involves four routes through which noise exerts its primary influence.

**Figure 6:** The four pathways through which the effects of noise are mediated. (Miedema 2007)



#### Sound masking Route:

This route reduces the comprehension of speech and masks speech, signals, music or natural sounds. International standard for the assessment of speech communication say that one-to-one conversation requires that the noise level does not exceed 41 dBA. At a distance of 4 m e.g. round a table or in a group, the noise must not exceed 29 dBA. These are very rarely achieved in urban areas and imply that the effects of environmental noise on communication are ubiquitous, especially in cities.

#### Attention Route:

Noise can negatively affect processes requiring attention. The effect of noise is probably most deleterious when impacting on working memory, and has been found

to depend on the priority and difficulty of the memory task, and type of sound. Millar (1979) indicated that it is the rehearsal of the items in working memory that is negatively affected by noise. If noise detracts from rehearsal it can have negative effects on the ability to derive implications and restructure information into more meaningful clusters.

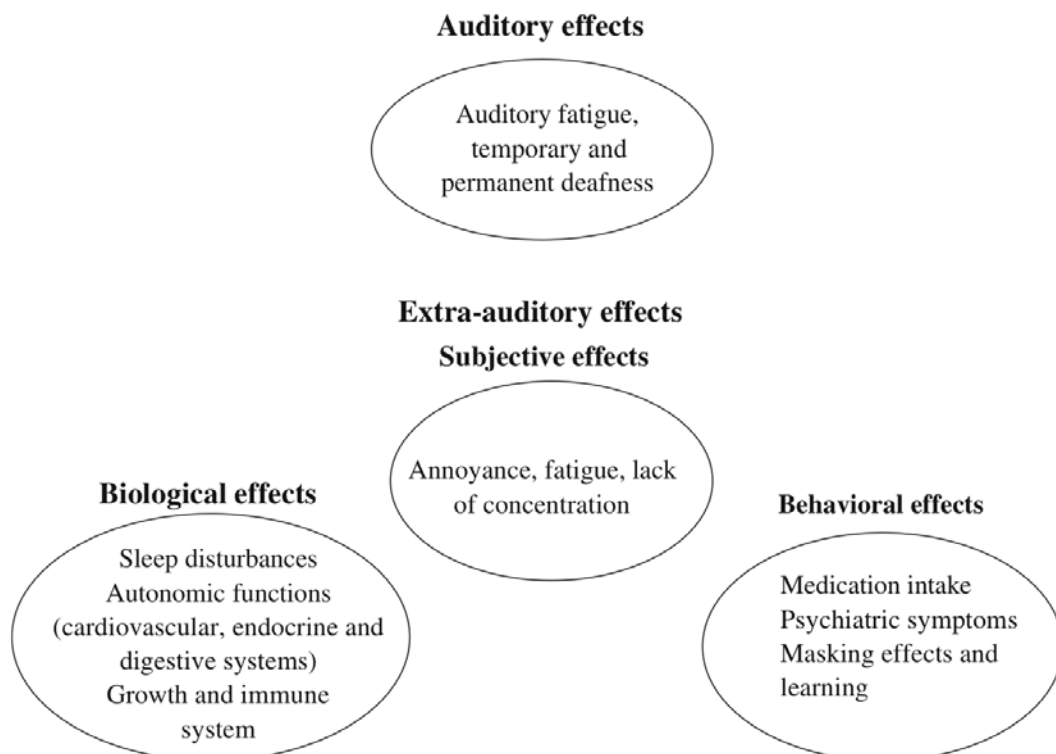
#### Arousal Route: Sleep

In field studies it has been found that the noise of a single event can cause instantaneous effects such as: extra motility, change in sleep state and EEG arousals, momentary changes in heart rate, and conscious awakening. The exposure-response relationship for conscious awakening has been assessed for civil aircraft (Passchier-Vermeer, 2003) Noise is described not by max sound level during the passage, but the total sound energy of the event (SEL). The effects of noise on sleep have low thresholds and the exposure-effect relationships increase monotonically. Noise is likely to be a dominant factor relating to sleep problems. More often it will cause a limited reduction in sleep quality that may not always be observed by the individual. Such noise-induced reductions of sleep quality may add to major causes of sleep problems that also appear to be mediated by increased arousal, such as social stress, medical stress, circadian stress and other environmental factors.

#### Affective-emotional route: fear and anger

As a result of noise affecting sleep, concentration, communication etc this frustration may lead to irritation or anger reactions. People high in trait anger may be more likely to show stronger emotional reactions when noise disturbs them. Fear can also be elicited with noise if it is associated with danger that threatens the individual. In this context it may be the worry of being in close proximity to an airport and therefore the concern over accidents that may induce fear, along with self-reported sensitivity to noise.

- 3.2.49 Miedema concludes that through masking, noise reduces comprehension, and through its effect on attention, noise affects the mental processing of information e.g. in reading. Through its effect on arousal, noise disturbs sleep, which may lead to fatigue, decreased performance, and depressed mood. Also, it may elicit emotional reactions when it interferes with behaviour or a desired state and may act as a stressor, or when it is associated with fear (aircraft noise). Such primary effects may in the long-term lead to annoyance, cognitive impairment, and/or cardiovascular effects. Chronic stress is also likely to be important in some long-term effects, in particular cardiovascular effects.
- 3.2.50 In the recent review on environmental noise, sleep and health Muzet (2007) explains the auditory and non-auditory effects of noise (**Figure 7**). Sleep disturbance is a non-auditory effect of noise. The input to the auditory area of the brain through the auditory pathways is prolonged by inputs reaching both the brain cortical area and the descending pathways of the autonomic functions. Therefore the sleeping body still responds to stimuli from the environment, although the noise sensitivity of the sleeper depends on several factors. These can be noise dependent e.g. type of noise, intensity, frequency, noise spectrum, interval, signification and the difference between the background noise level and the maximum amplitude of the occurring stimulus. Other factors are related to the sleeper, e.g. age, sex, personality and self-estimated sensitivity to noise.

**Figure 7:** Auditory and non-auditory effects of noise, taken from Muzet (2007)

3.2.51 The immediate effects of noise are seen as sleep disturbance, quantified by number and duration of nocturnal awakenings, number of sleep stage changes, and modifications in their amount. Also changes in the autonomic functions such as heart rate, blood pressure, vasoconstriction, and respiratory rate are observed.

3.2.52 Longer sleep latency and premature final awakening can reduce TST. Reports suggest that intermittent noises with maximum noise levels of 45 dBA and above can increase the time to fall asleep to 20 minutes. Combined with this, sleep pressure is reduced after the first 5 hours, therefore in the morning noise events are more likely to prevent the sleeper from going back to sleep.

3.2.53 Awakenings have a much higher threshold in deep sleep, e.g. SWS or REM, and a much lower threshold in lighter stages of sleep. The threshold depends on physical characteristics of the noisy environment (intermittent or sharp rising noise occurring above a low background noise will be particularly disturbing), as well as noise signification.

#### Sleep stage modifications

Nocturnal awakenings can be observed for an indoor  $L_{Amax}$  of 55 dB and above, and disturbance of normal sleep can be observed for maximum noise levels between 45 and 55 dBA. To protect noise-sensitive people, the WHO recommended a maximal level of 45 dB inside the bedroom, whereas for the same period the mean recommended level (integrated noise level over the 8 nocturnal hours:  $L_{night}$ ) was 30 dB. SWS is the most restorative sleep stage, whereas REM is important for memory consolidation. Carter (1996) reported that SWS might be reduced in young sleepers subjected to intermittent noise. Also, Muzet has previously reported that REM sleep rhythmicity could also be affected by environmental noise exposure. It is common to see a reduction in SWS and REM and an increase in shallower sleep stages, which

can become chronic and detrimental. Long-term studies of such reduced SWS are worth exploring and may prove to be important.

#### Autonomic responses

Heart rate changes and vasoconstrictions can be seen at much lower noise levels than are found to induce sleep disturbance and indicate that such disturbance can be felt when asleep even if there is no conscious memory of it the next day. The health effects of such responses can be cumulative, over a few thousand stimuli per night.

#### Secondary effects

Secondary effects include the subjective evaluation of sleep disturbance due to noise, such as complaints about sleep quality, delayed sleep onset, nocturnal awakenings, and early morning waking. They are often accompanied with increased sleepiness, tiredness and need for compensatory resting periods the following day.

Findings show that the subjective assessment of sleep quality does not accurately correspond to the objective measurement of sleep. When the number of noise events increase, the number of sleep modifications and/or awakenings also increases, but not proportionately. Porter (2000) found that noise heard at night was more intrusive and noticeable than noise heard during the day. This is due to reduced outside and inside background noise at night, and the circadian phase. It may also be a time of increased sensitivity to noise. Therefore it is wise to be cautionary when relying entirely on subjective reports of noise-related sleep disturbance due to their questionable validity.

Muzet (2007) reports that sleep disturbance occurring during the early part of the night and early morning prior to the natural time of awakening seem to be the most intrusive. This results in daytime sleepiness, fatigue and lower work capacity and increased accident rate. Fear of living under the flight path can also complicate the issue of accurately assessing subjective sleep quality as a result of noise, making the clarity of the relationship difficult to ascertain.

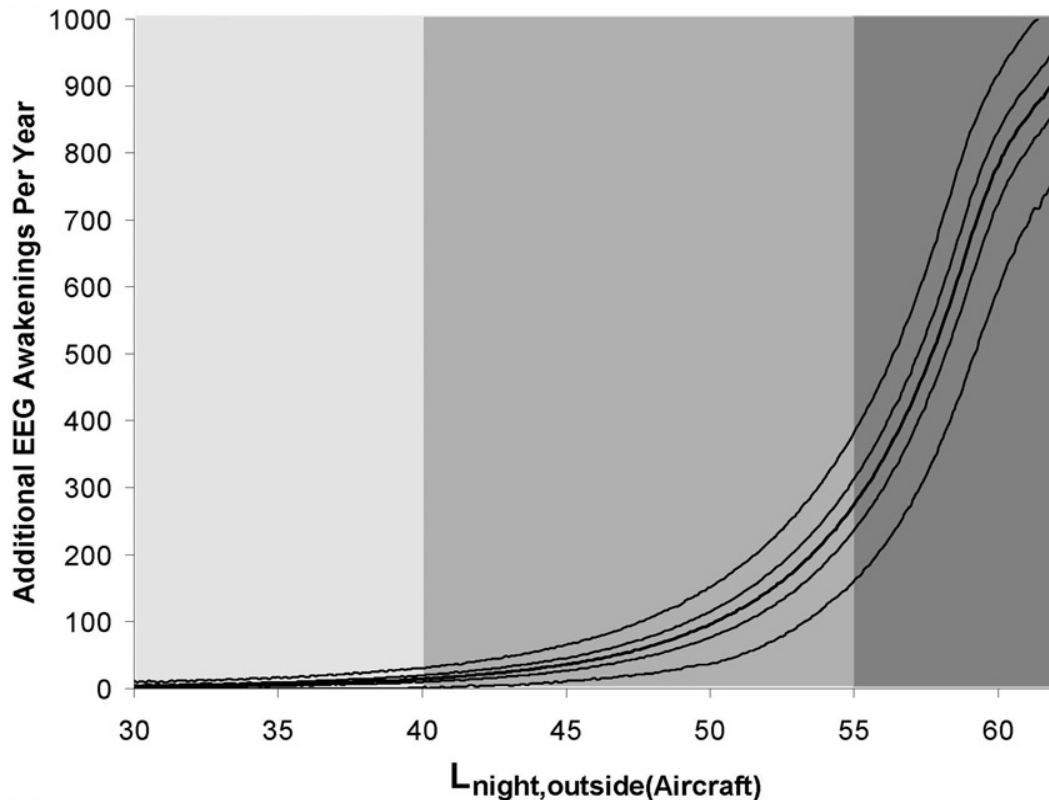
#### Other secondary effects

Stress hormones such as cortisol, noradrenaline and adrenaline are increased the following morning and there are also reports of cognitive impairment the next day.

- 3.2.54 Physiological sensitivity to noise can depend on the age of the sleeper. EEG changes and awakening thresholds are on average 10 dBA higher in children than in adults, however their cardiovascular sensitivity to noise is similar to older people.
- 3.2.55 In summary, there are conflicting findings, partly down to the difficulty in ascertaining a clear dose-effect relationship between noise and sleep disturbance, and the degree of interaction of confounding variables. The factors include noise characteristics, noise sensitivity, and the context of the environment.
- 3.2.56 Muzet (2007) suggests that future research should focus on the long-term effects of night-time noise exposure of different populations. A study of specific sub groups thought to be at risk, i.e. children, elderly, self-estimated sensitive people, insomniacs, sleep disorder patients, night and shift workers would be useful to assess differences between populations. Finally, the combined effects of noise exposure and other physical agents or stressors during sleep should be investigated to provide further understanding of the pathways in which noise disturbance effect sleep.

- 3.2.57 Brink (2009) produced a paper on determining awakening probabilities in night-time noise effects research. This paper was borne as a result of a German lawsuit involving Leipzig-Halle airport, which suspended its night-time curfew so it could be used as a freight-hub for a large logistics company. The surrounding controversy revealed that there was a lack of a common scientific standard for the probability of “noise-induced” awakenings. The aims included resolving the most problematic issues relating to the correct derivation of awakening reaction probability (as specified by EEG recordings) to noise events during sleep. It is explained that there is the need to know the probability of awakening spontaneously within a particular timeframe, as this information is required as well as the probability of awakening from noise. A time window is presented with representations of the probability of observed awakenings ( $P_{\text{observed}}$ ), spontaneous awakenings ( $P_{\text{spontaneous}}$ ) and additional i.e. awakenings that were not simply spontaneous ( $P_{\text{additional}}$ ).  $P_{\text{induced}}$  was given as the probability of awakening *independent* of spontaneous awakenings. The problem of interdependencies of reactions was raised. For forecasting awakening reactions for a particular night-noise scenario, it is important to know whether the total probability of awakenings can be expected to be always the same, independently of a particular noise distribution over the night. This is problematic because of the likelihood of awakening is dependent on sleep stage, and increases with the time spent asleep. The other issue is that a reaction to a noise event, (awakening or not) may influence the micro- and macrostructure of sleep and therefore can also alter the probabilities of awakening at future events. It was explained that additional variables such as total sleep time (TST) could be modelled for night-time noise scenarios by a process based on assumptions about transition probabilities of sleep state, duration of state, and effects of noise properties of the noise events on these variables.
- 3.2.58 The Defra report 2009 concluded that no single dose-response relationship could be recommended for sleep disturbance as part of a valuation methodology. It is suggested that investigation into the linkage between the transient effects of noise on sleep and potential long-term chronic health effects is required.
- 3.2.59 The HPA report discusses the difficulty in obtaining a dose-response relationship between environmental noise and sleep disturbance due to the differences in results between laboratory and field studies, and also the issue of habituation to noise.
- 3.2.60 Finegold (2010) has also published a recent paper on sleep disturbance and aircraft noise exposure. This includes an explanation that there is no single noise exposure metric that is agreed upon for use in sleep disturbance research, and there are conflicting perspectives, for example, the use of SEL versus  $L_{A_{\text{max}}}$ . Although the WHO NNG (2009) and END recommend  $L_{\text{night, outside}}$  to be used, in the USA SEL is still used as a metric for sleep disturbance with Finegold proposing a dose-response function based on the SEL of each event. The paper discusses the importance of the ‘meaning of sound’ as an important predictor of awakening, and highlights the current situation that there is little known about the long-term cumulative effects of intermittent sleep disturbance due to noise.
- 3.2.61 Basner et al (2010) discuss the mechanisms, mitigation and research needs of aircraft noise on sleep. This paper is also discussed in the health effects part of this report with reference to cardiovascular responses to aircraft noise at night. Sleep disturbance is examined, with data from the DLR field study on the effects of aircraft noise on sleep, being used to simulate single nights with 1 to 200 ANEs per night.  $L_{\text{night}}$  and number of additional awakenings from aircraft noise (based on the DLR 2006 exposure-response curve in Figure 5) were calculated and used to predict the degree of sleep fragmentation. These results (taken from Basner et al 2010) are shown in **Figure 8**.

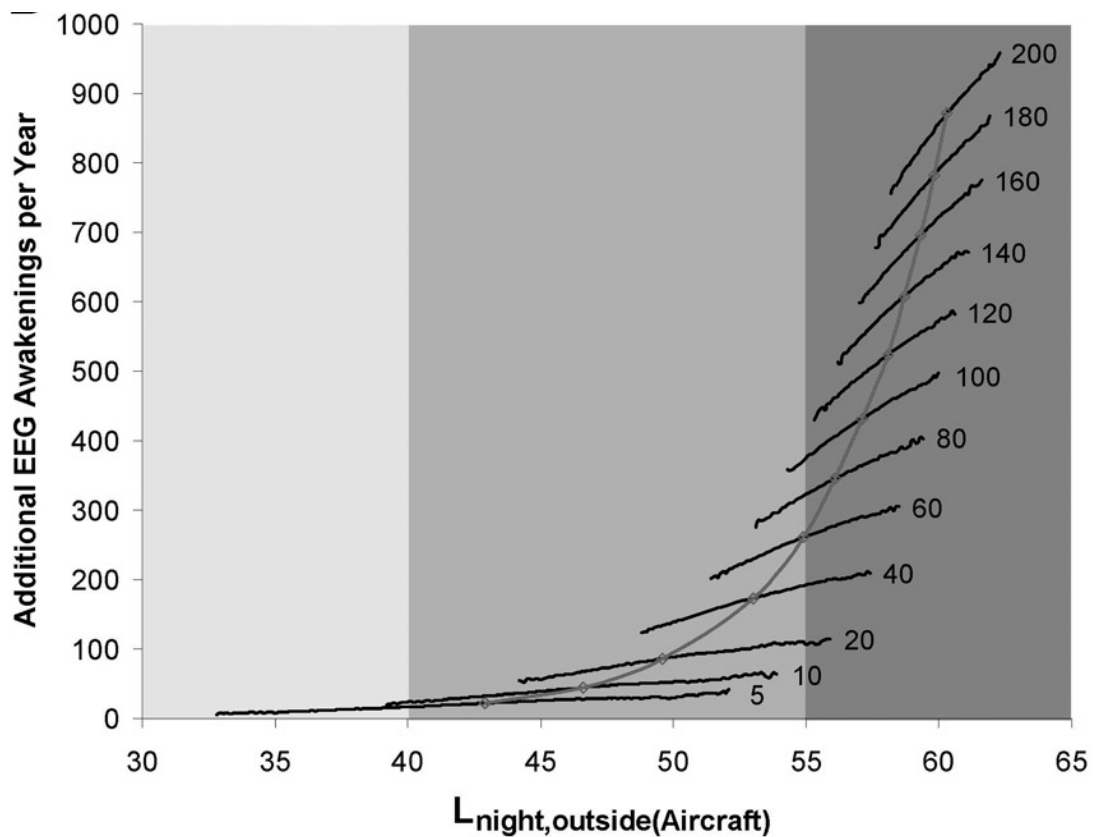
**Figure 8** The average number of awakenings additionally induced by aircraft noise per year is shown depending on  $L_{\text{night, outside}}$ . Altogether, 10 million eight-hour nights with 1 to 200 (1, 2, 3, ..., 200) noise events randomly drawn from the DLR field study were simulated. The lines represent (from below to above) 2.5, 25, 50, 75, and 97.5 percentiles. (Basner et al, 2010)



3.2.62 The shaded part of the graph represents the recommended target and interim noise limits as given in the WHO NNG (2009) of 40 and 55 dB  $L_{\text{night}}$  respectively. These limits are discussed in more detail in Section 5 of this report, but results from this study do seem to support the recommendations from WHO for the given limits.

3.2.63 The number of noise events was also studied in terms of sleep disturbance. Findings showed that there were differences in the degree of sleep fragmentation depending on the number of noise events that contributed to a particular  $L_{\text{night}}$  level. This is shown in **Figure 9**, for example at 55 dB  $L_{\text{night}}$  the number of awakenings varies between just over 100 (at 20 noise events) to nearly 400 (at 100 noise events).

**Figure 9** The average number of awakenings additionally induced by aircraft noise per year in relation to  $L_{night}$  and number of ANEs. The horizontal lines represent the median number of additional awakenings, with numbers on the right indicating the number of ANEs per night contributing to  $L_{night, outside}$ . The curve joins up the mid points for each number of events line.



3.2.64 The authors suggest that additional information on the number of noise events contributing to  $L_{night}$  would be useful in terms of allowing for a more precise prediction of the number of additional awakenings that could be expected.

3.2.65 A recent laboratory based study (Basner et al. 2011) examined the impacts of mixed transportation modes (air, road and rail) on sleep disturbance. 72 subjects were studied (32 male) for 11 consecutive nights with 0, 40, 80 and 120 noise events employed in a balanced design, in terms of number of noise events, maximum sound pressure level and equivalent noise load. The results showed that road traffic caused the most obvious changes in sleep structure and continuity whereas air and rail was considered more disturbing subjectively. This was attributed to road traffic noise events being too short to be consciously perceived by the subjects that had awoken in response to the event. The results also showed that while subjective annoyance was greater for aircraft noise, cortical and cardiac responses during sleep were lower for air compared to road and rail traffic. An interesting finding was that most (>90 %) of the noise induced awakenings merely replaced awakenings that would have occurred spontaneously, which helped to preserve sleep continuity and structure despite the noise. This suggests that within limits there is some homeostatic mechanism for internal monitoring and control of waking arousals (or maintaining sleep) that are allowed during each night's sleep.

3.2.66 Janssen (2011) investigated the number of aircraft events and motility during sleep. The background to this study was that both the WHO and EC advise on the use of the

$L_{\text{night}}$  metric as the primary indicator for sleep disturbance. The author explains, however, that an important question for noise policy is whether from a public health perspective it may be of interest/advantage to use the number of events in addition to  $L_{\text{night}}$ . For example, for some effects it may be preferable to reduce the number of events above a certain threshold than to lower the overall exposure level of events. This study used data from Passchier-Vermeer's 2002 study in the Netherlands, and looked at the association between objectively measured sleep disturbance and the number of aircraft noise events with respect to mean motility during the sleep period. The researchers wanted to know whether motility can be predicted more accurately taking the number of events into account. The results suggested that an increase in SEL contributes more to motility than an increase in the number of events. However, it was also found that the influence of the number of events increases with increasing levels of the event. Janssen suggested that to reduce motility, it may be better to prevent events with high maximum sound levels, than to reduce the overall number of events.

3.2.67 Plante et al (2012) conducted a review of the evidence relating to aircraft noise and sleep disturbance. Studies were included based on quality and bias criteria and therefore many studies were not included due to methodological discrepancies or because they did not provide an objective measurement of noise levels. Nine studies met the inclusion criteria, eight of which were experimental, three were cross-sectional and one was an ecological study. The review summarised the design for each of the studies, noise events, measurements of sleep outcomes and findings. The authors concluded that aircraft noise exposure does impact on sleep disturbance and the deterioration of sleep outcomes based on the findings from moderate to high quality studies. As the sound levels increase, the probability of awakening increased and awakening times last for longer periods. In addition, individuals exposed to higher levels of noise have been found to have shorter periods of SWS, and sleep medication increased when aircraft noise events occurred in the evening. Gaps in the field were also identified, with the suggestion that research attention is given to the over 65s, people with chronic illness and pre-existing sleep disorders.

### 3.3 Summary

- 3.3.1 The majority of research into noise and sleep disturbance has concentrated on the relationship between individual aircraft noise event levels and the two principle characteristics of an ANE, the maximum level,  $L_{A_{\text{max}}}$  and the sound exposure level, SEL. Researchers have sometimes concentrated on indoor rather than outdoor levels, but appear to have ignored the fact that public policy has little control over outdoor-indoor attenuation levels, because, in all the but the highest noise areas, residents are free to open windows.
- 3.3.2 The focus has also been to identify the lowest observable threshold ( $L_{A_{\text{max}}}$ , SEL) at which to avoid effects.
- 3.3.3 Whilst SEL is a pre-requisite in the calculation of exposure metrics such as  $L_{\text{night, outdoors}}$ , it is seldom provided in addition to  $L_{\text{night, outdoors}}$  as it varies with location and aircraft type. As a result, there has been a shift, at least within Europe, towards linking sleep disturbance to the more readily available  $L_{\text{night, outdoors}}$  metric.



## 4 Health effects

### 4.1 Introduction

4.1.1 The World Health Organisation (WHO, 1968) defines health as follows:

“Health is not merely the absence of disease or infirmity but is a positive state of physical, mental and social well-being.”

4.1.2 This broad definition has been taken as the basis for including a review of various effects within this section.

4.1.3 It is universally accepted that exposure to high noise levels can induce hearing impairment, however at the levels of environmental noise exposure around civilian airports hearing loss is unlikely. This report therefore focuses on the non-auditory health effects of environmental noise, that is:

“All those effects on health and well-being that are caused by exposure to noise, with the exclusion of effects on the hearing organ and the effects which are due to the masking of auditory information (i.e. communication problems)”

4.1.4 This section presents a summary of the scientific knowledge of noise and health under the following categories:

- Cardiovascular and Physiological Effects
  - Myocardial infarction
  - Hypertension
  - Ischemic heart disease
  - Stress
- Next day effects
- Noise and Children
- Night time specific effects

4.1.5 Noise can elicit a stress response in the body in the same way as other stressors. The normal stress response is a coping mechanism that occurs when the brain perceives a threat. Acute noise exposures activate the autonomic and hormonal systems, leading to temporary changes such as increased blood pressure, increased heart rate and secretion of stress hormones. Normally, these return to baseline levels when the noise ends or the person adapts. However, prolonged exposure to noise may have the potential, in susceptible individuals, to cause chronic physiological effects such as hypertension, ischaemic heart disease (IHD) and elevated stress hormone levels. Sustained elevated hormone levels may affect the functional integrity of bodily organs and tissues.

4.1.6 With regard to cardiovascular effects, the WHO Guidelines conclude that epidemiological studies show that these occur after long-term exposure to noise (aircraft and road traffic) with values of 65 to 70 dB  $L_{Aeq24hour}$  – however the associations are weak. The association is somewhat stronger for IHD than for hypertension. The WHO identify that although the risks of noise having a negative impact on cardiovascular function are small, they are important because a large number of people are likely to be exposed to such noise levels.

4.1.7 The WHO NNG concludes that more research is needed regarding the association between aircraft noise and cardiovascular endpoints.

- 4.1.8 A literature review was undertaken of the scientific knowledge on the subject of 'environmental noise and health', with particular reference to aircraft noise. The World Health Organisation Guidelines for Community Noise ('WHO Guidelines' 1999) were taken as the basis for the review, and a literature search was carried out for key papers published after the WHO Guidelines and for review papers published since the late 1990s.
- 4.1.9 A number of review papers are referred to repeatedly throughout this section, these are:
- Health Council of the Netherlands (1999). Public Health Impact of Large Airports. ('HNC Review')
  - Health Canada (2002). Noise from Civilian Aircraft in the Vicinity of Airports, or Human Health - Noise, Stress and Cardiovascular disease. ('HC review')
  - Health Council Australia (2004). The Health Effects of Environmental Noise - Other than Hearing Loss. ('ECA Review')
  - Various reviews undertaken by Stansfeld and co-workers
- 4.1.10 Two papers have more recently been published in this area; the first was commissioned by the Department for Environment, Food and Rural Affairs (Defra) on behalf of their Interdepartmental Group on Cost and Benefit (IGCB) into an estimation of the dose-response relationship between noise exposure and health effects; the second is a Health Protection Agency (HPA) report entitled Environmental noise and health in the UK.
- 4.1.11 The Defra publication (2009) is authored by Bernard Berry and Ian Flindell, and comprises four main aims:
- To identify a comprehensive list of potential adverse health impacts from noise and review the current state of evidence for each of the impacts;
  - Where a robust evidence base exists, to recommend quantitative links (dose-response functions) for the impacts of noise on health which could be applied in the UK;
  - Identify any emerging adverse health impacts that should be kept under review for future consideration in evaluation; and
  - Identify any structural challenges to developing and maintaining strong quantitative links between noise and health outcomes
- 4.1.12 The HPA report (2009) was produced in response to increasing public concern about possible adverse effects of noise on health. It was prepared by an ad hoc group of experts at the request of the Department of Health and funded by the Defra. As before, this report is available on the HPA website. This report will be referred to where relevant.
- 4.1.13 The WHO Night Noise Guidelines for Europe (NNG) were published in October 2009. This document was presented as an extension to the WHO Guidelines for Community Noise document from 1999. The aim of the Night Noise Guidelines (2009) was to present conclusions from the WHO working group responsible for preparing guidelines to exposure to noise during sleep. These guidelines use both direct evidence concerning the effects of night noise and health, and also indirect evidence relating to the effects of noise on sleep and the relationship between sleep and health, as their basis.

- 4.1.14 The WHO Guidelines (1999, 2009) note that vulnerable people (e.g. people that are ill, old, depressed, foetuses, babies and young children, shift workers) may be less able to cope with the impacts of noise exposure and they may be at greater risk of harmful effects. Generally, there is little scientific research focused on these vulnerable groups. An exception to this is the research of the effects of environmental noise on children; a body of scientific literature specifically on the effects of aircraft noise on children is emerging. The limited evidence on foetal effects presented in various reviews is also summarised in this section.
- 4.1.15 The literature on the non-auditory health effects of environmental noise is extensive; this review does not aim to give an in-depth assessment of the nuances of the scientific work in this field, but to provide a succinct overview of the current research in this area.

## 4.2 Myocardial Infarction (MI) and Hypertension

- 4.2.1 Di Nisi *et al* (1990) investigated the cardiovascular responses to noise during wake and sleep in two groups of 40 males and females each grouped according to self reported sensitivity to noise being high or low. Subjects were exposed to common noises such as aircraft at 67 dBA, trucks at 61.9 dB, motorcycles at 52.7 dB, trains at 68.2 dB and telephones at 62 dB all  $L_{Aeq}$ , with a maximum intensity of the aircraft noise at 86 dB  $L_{Aeq}$  occurred in the morning and afternoon. Heart rate and finger-pulse responses were compared to sensitivity, gender and time of day.
- 4.2.2 Heart rate (HR) responses showed differences between the sensitivity groups, but not type of noise, whereas the opposite was found for finger-pulse (FP) results, with no significant difference in sensitivity but clear differences between noises.
- 4.2.3 Ten subjects from each group were selected and exposed to the same noises at night whilst being recorded. Both HR and FP were greater during the sleep period for both groups, compared to waking, and did not differ between gender or sensitivity. Both responses showed differences in noise types, which were based on their noise-equivalent level value.
- 4.2.4 The relationship between road traffic and blood pressure and heart rate in preschool children was examined during the night at children's residences, and during the day at Kindergartens (Belojevic *et al*, 2007). A cross-sectional study was performed on 328 preschool children ages 3-7years, who attended 10 public kindergartens in Belgrade.  $L_{Aeq}$  was measured during the night in front of the children's homes and during the day in front of the kindergartens. A home was classified as noisy if the  $L_{eq}$  exceeded 45 dBA during the night and quiet if the  $L_{Aeq}$  was  $\leq 45$  dB. Noisy and quiet kindergartens were those with daily  $L_{Aeq} > 60$  dB and  $\leq 60$  dB respectively. The prevalence of children with hypertensive values of blood pressure was 3.9%, with a higher prevalence in children from noisy residences (5.7%), compared to children from quiet residences (1.48%). Systolic pressure was significantly higher (5mmHg on average) among children from noisy residences, compared to children from both quiet environments. Heart rate was significantly higher (2 beats/min on average) in children from noisy residences. The authors stressed, however, that it was not known if these effects were of a temporary nature and whether they could be reversed upon cessation of the noise exposure.
- 4.2.5 A cross-sectional study of environmental noise and community health was conducted in neighbourhoods around Sydney Airport, with high exposure to aircraft noise and in a matched control suburb unaffected by aircraft noise (Black *et al*, 2007). The relationships between health-related quality of life and aircraft noise, and long-term

exposure to aircraft noise and adult high blood pressure levels were examined using social surveys. Noise measurements were undertaken that lead to the development of a novel metric – the noise gap index, NGI that includes considerations of background environmental noise. The NGI was developed as an index that is easy to understand by the layperson, and that also quantifies relevant aspects of the potential impacts of aircraft noise. It was found that subjects living in high and medium background environmental noise areas were more likely to be annoyed by the same aircraft noise exposure level than subjects living in low background environmental noise areas. The research concluded that:

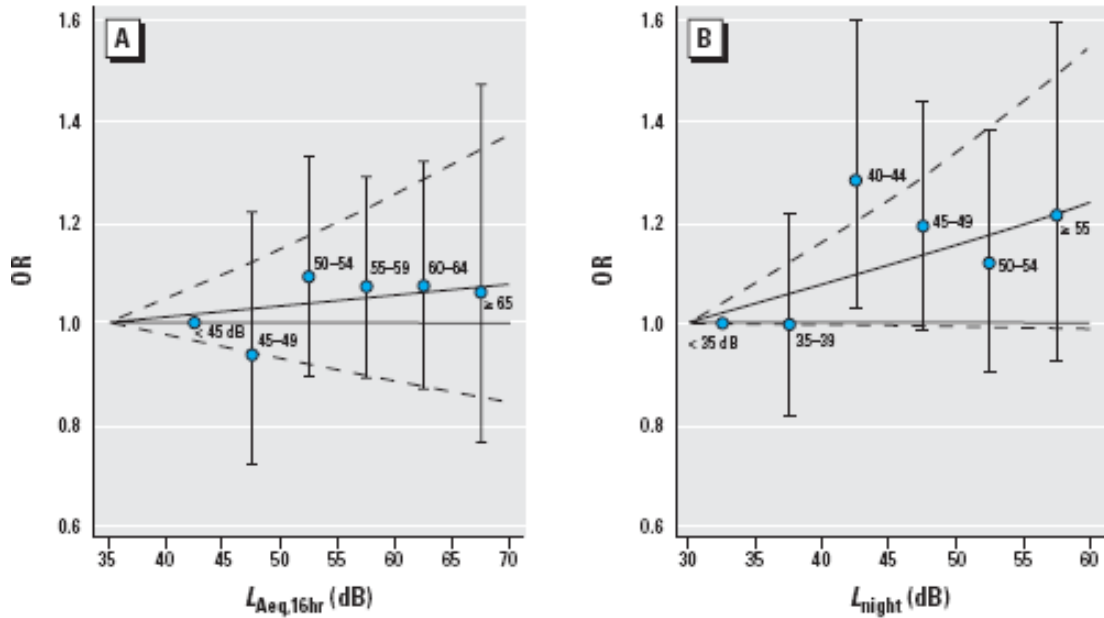
- Long-term aircraft noise exposure was significantly associated with chronic noise stress
- Chronic noise stress was significantly associated with prevalence of hypertension

4.2.6 Perhaps the most publicised study to examine the effects of aircraft noise on hypertension in recent years is the HYENA study (Hypertension and Exposure to Noise near Airports) (Larup et al, 2007). A total of 4861 people participated in the study, in an age range of 45-70 years old, with a minimum length of residence of five years, living near one of six major European airports (London Heathrow, Berlin Tegel, Amsterdam Schiphol, Stockholm Arlanda, Milan Malpensa and Athens Elephterios Venizelos airport). The selection process created exposure contrast to aircraft noise and road traffic noise within countries, ensuring that sufficient numbers of inhabitants in the appropriate age range had expected exposures  $> 60$  dBA and  $< 50$  dBA. Participants were interviewed by specially trained staff, and their blood pressure measured on three occasions; at the beginning of the interview, after five minutes' rest, and then again after a further one minute's rest and finally after the interview as a validity control. The mean of the first two readings was used to define blood pressure for the subsequent analyses.

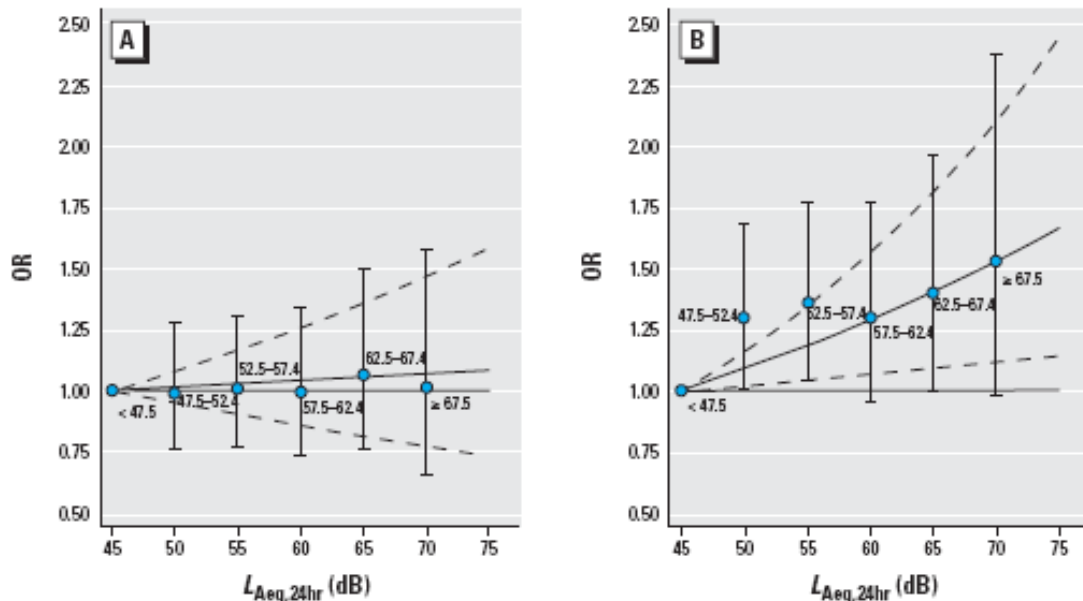
4.2.7 **Figure 10** shows the odds ratios for hypertension in relation to aircraft noise during the day ( $L_{Aeq,16h}$ ) and during the night ( $L_{night}$ ). A rise in odds ratio with increasing exposure is indicated primarily for night-time noise, with no differences found between males and females.

4.2.8 **Figure 11** shows the odds ratios for hypertension in men and women in relation to average road traffic noise exposure ( $L_{Aeq, 24h}$ ) An increase in risk for men with increasing exposure was reported, but this was not found in women.

**Figure 10** Odds ratios of hypertension in relation to aircraft noise (5 dB categories).  $L_{Aeq, 16h}$  (A) and  $L_{night}$  (B) were included separately in the model. Adjusted for country, age, sex, BMI, alcohol intake, education, and exercise. Error bars denote 95% confidence intervals for the categorical (5 dB) analysis. The unbroken and broken curves show the ORs and corresponding 95% CIs for the continuous analysis. Taken from Jarup et al, 2008.



**Figure 11** ORs in women (A) and men (B) in relation to road traffic noise ( $L_{Aeq, 24h}$ , 5 dB categories) separately included in the model. Adjusted for country, age, sex, BMI, education, and exercise. Error bars denote 95% confidence intervals for the categorical (5 dB) analysis. The unbroken and broken curves show the ORs and corresponding 95% CIs for the continuous analysis. Taken from Jarup et al, 2008.



4.2.9 The results from the HYENA study indicated that there were significant exposure response relationships between exposure to night-time aircraft noise exposure, daily

average road traffic noise and risk of hypertension. The authors highlighted that the higher risk for night-time noise may be a consequence of less misclassification of exposure during the night (i.e. participants are more likely to be home during the night). They suggest that the higher night-time risks may also be explained by acute physiological responses induced by night-time noise events that might affect restoration during sleep. The gender difference with relation to road traffic noise was an interesting finding and one that could be explored further. Overall, the conclusions from the HYENA study were that the increased risk of hypertension in relation to aircraft and road traffic noise near airports might contribute to the burden of cardiovascular disease. The authors suggested that preventative measures should be considered to reduce road traffic noise and night-time noise from aircraft.

4.2.10 As part of the framework of the HYENA study, the acute effects of night-time noise in relation to blood pressure were also reported in 140 subjects (Haralabidis et al, 2008). Measurements of blood pressure were taken every 15 minutes during the study night in participants' homes. Noise level equivalents for every second, every minute and for every 15-minute period in-between blood pressure measurements were calculated. Noise events were classified into four categories:

- Indoor
- Aircraft
- Road traffic
- Other outdoor

4.2.11 The results indicated that both systolic and diastolic blood pressure, as well as heart rate increased with higher noise levels during the preceding minutes, independently of the noise source. Significant increases in blood pressure was also seen when the source of the noise was taken into account. The effects of the source-specific noise were comparable for aircraft, traffic and indoor events and were similar to those of the total measured noise. The authors concluded that the absence of short-term habituation to the cardiovascular effects of noise, especially those during sleep, are likely to support a link between acute and long-term effects of noise exposure and hypertension and cardiovascular disease.

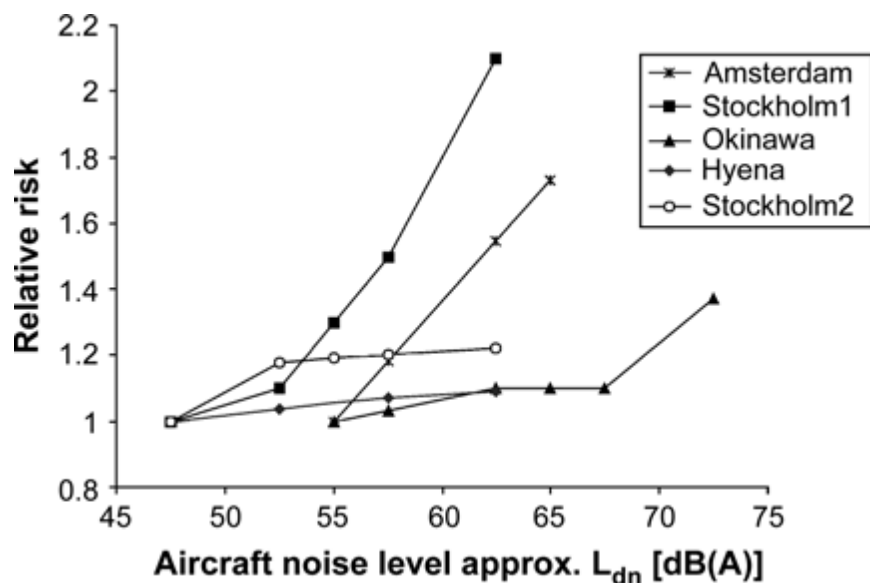
4.2.12 Greifahn *et al* (2008) analysed heart rate responses to traffic noise during sleep, and examined the effects of factors such as time of night, acoustic parameters and momentary sleep stage. Twenty-four subjects were required to sleep in the laboratory for four consecutive nights, for three consecutive weeks, with exposure to aircraft, road or rail noise in each of the weeks. One of the nights was a randomly assigned quiet night (32 dBA), and the noise exposure nights had maximum levels of 45-77 dBA. PSG and ECG were recorded throughout each of the nights, with participants being asked to sleep between 2300 and 0700. The results indicated that response patterns were mainly determined by the occurrence or absence of awakenings. When awakenings occurred, heart rate responses were monophasic and increased over more than one minute. These responses were not influenced by the acoustic parameters, with the strongest influence being the sleep stage at which the exposure occurred. The strongest response was found during REM sleep, with the weakest response occurring when subjects were in SWS.

4.2.13 When awakenings did not occur, the heart rate responses were biphasic. An initial acceleration with a maximum after four to eleven seconds was followed by a deceleration to a minimum below the baseline after 12 to 23 seconds, followed by a consecutive increase towards baseline values. In these instances, there was a significant influence of the type of noise, with railway noise causing the earliest and aircraft noise producing the latest increase in heart rate. The same pattern of

response was observed as before in terms of sleep stage, with the largest change seen in REM, and the smallest in SWS.

- 4.2.14 These responses did not decrease as a function of time throughout the night, and the authors suggest that therefore habituation is unlikely to occur. They suggest that this may be the main reason for potentially pathologic responses over time, and that these may play a significant part in promoting traffic noise induced cardiovascular disease, particularly in those responses accompanied by awakenings.
- 4.2.15 Basner *et al* (2008) reported data on the comparison between sleep disturbance responses measured by polysomnography, and single channel ECG with respect to aircraft noise, with the hypothesis being that cardiac activations can be used as estimates for EEG awakenings. Data from 129 subjects, 985 nights and 23855 ANEs were used. Subjects were required to sleep in a laboratory for 13 nights, with night 1 as an adaptation night, 2 as a baseline, and nights 3-11 involving ANEs with levels between 45 and 80 dB  $L_{Amax}$ . 30 different exposure patterns were used, to give a spread of values of SPL and number of ANEs across the study, and these were randomly assigned.
- 4.2.16 Both EEG awakenings and cardiac activations increased with increasing maximum SPLs. The two types of responses were highly correlated, with exposure-response curves for reactions induced by aircraft noise being almost identical for EEG and ECG responses. This suggests that the single channel ECG is a good estimate of EEG responses. It was therefore suggested that the ECG method might prove to be an effective way of collecting physiological data from large numbers of unsupervised participants, possibly alongside other low maintenance methods such as actigraphy in order to further validate results. The analysis of the ECG data is automatic and objective as it is analysed using an ECG algorithm, and therefore is also more reliable, faster and cheaper than PSG analysis. Basner stresses that further investigation and validation in the field is required, and that at present, polysomnography remains the gold standard for recording physiological response to nocturnal noise exposure.
- 4.2.17 Babisch and van Kamp (2009) evaluated the Exposure-response relationship of the association between aircraft noise and the risk of hypertension. There has been no clear association found between aircraft noise, ischemic heart disease, and myocardial infarction, possibly due to the absence of large scale quantitative studies. There is sufficient qualitative evidence, however, that aircraft noise increases the risk of hypertension in adults. The authors evaluated the literature for the WHO working group on "Aircraft Noise and Health". With respect to the needs of a quantitative risk assessment for burden of disease calculations, the authors attempted to derive an exposure-response relationship based on a meta-analysis. An in-depth discussion of the criteria for inclusion is given in the paper, with five studies being chosen as the basis for analysis. An approximate graphical representation of the results are given in **Figure 12**, but authors caution that no conclusions regarding possible threshold value or noise level related risks (in absolute terms) can be drawn.

**Figure 12** Association between aircraft noise level and the prevalence or incidence of hypertension



4.2.18 When linear trend coefficients of all the five studies are calculated and pooled afterwards ('regression approach') the pooled effect estimate of the relative risk is 1.13 (95% CI = 1.00-1.28) per 10 dBA. The authors caution that the limitations involving the pooling of studies due to methodological differences in the assessment of exposure and outcome between studies mean that the association must be viewed as preliminary. It is suggested to use  $L_{den} \leq 50$  or  $L_{den} \leq 55$  dBA as a reference category of the exposure-response relationship. The respective relative risks for subjects who live in areas where  $L_{den}$  is between 55 to 60 dBA and between 60 to 65 dBA would then approximate to 1.13 and 1.20, or 1.06 and 1.13, respectively.

4.2.19 A Swedish study (Rosenlund, 2001) found that the prevalence of hypertension was higher among people exposed to average noise levels of at least 55 dBA or maximum levels above 72 dBA, around Arlanda airport, Stockholm. However, the methodological approach of this study has been criticised.

4.2.20 Goto (2002) reported on a study to investigate the blood pressure levels in those living around an airport in Japan. Examination of study data from 469 women living around the airport, and exposed to varying levels of aircraft noise, found that blood pressure was not associated with aircraft noise level. In a questionnaire survey around Schiphol Airport, Franssen, (2004) found that the risks of poor self-rated health, and of medication use for cardiovascular diseases or increased blood pressure, increased with aircraft noise levels. Franssen concludes that exposure to aircraft noise may be a risk factor for cardiovascular disease.

4.2.21 It is not only the effects of aircraft noise on sleep during the night that has been studied. Carter *et al* (2002) examined the cardiovascular response to environmental noise during sleep in shift workers who were sleeping during the day in a sleep laboratory. Nine female permanent night duty nurses were exposed to noises from trucks, civilian aircraft, low altitude military aircraft and tones, presented at 55, 65 and 77 dB  $L_{Amax}$ . The authors reported that heart rate was responsive to noise levels, but not the noise type. Blood pressure increased primarily to the sudden onset of sounds, and noise-induced awakening and alpha wave EEG responses were related to blood pressure increases. Any increase in heart rate was greatest when subjects were awakened by noise, or were already awake. The authors concluded that over these range of noises, heart rate responds to noise level during sleep, and blood pressure



to sounds of a sudden onset. However, they recommend that due to the sensitivity of the spectral analysis of blood pressure, it should be studied in people sleeping in their own home.

- 4.2.22 Often, there is a discussion that sleep represents a trophotopic phase (energy storing), contrasting with an ergotropic (energy consuming) phase when we are awake (Maschke and Hecht 2004). Therefore, frequent, or long-awakening reactions endanger recovery and therefore health. Such frequent occurrences of arousal triggered by nocturnal noise can lead to a deformation of the circadian rhythm. Also, the deep SWS phases in the first part of the night are associated with a nadir of cortisol, and a maximum of growth hormone, both necessary for the physical wellbeing of the sleeper.
- 4.2.23 The link between hypertension and road traffic noise exposure was studied (de Kluizenaar et al, 2007). The study design was cross-sectional ( $n = 40,856$ ) and participants were inhabitants of Groningen, Netherlands. Before adjustment for confounding variables, road traffic noise exposure was associated with self-reported use of antihypertensive medication in the whole sample, however following adjustment the association persisted in subjects between 45 and 55 years old, and at exposure levels of  $L_{den} > 55$  dB. The authors suggested that exposure to high levels of road traffic noise may be associated with hypertension in subjects in this age range, and that the associations are stronger at higher noise levels.
- 4.2.24 Heart rate, blood pressure and noise perception in relation to aircraft noise was measured in residents around Frankfurt Airport (Aydin and Kaltenbach, 2007). Two areas were selected, in which aircraft noise was the predominant source of noise created by aircraft taking off but not landing. The responses of residents were measured over a twelve week period, with one area being exposed to air traffic noise for three quarters of the given time, and the other area only exposed for one quarter of the time. Blood pressure and heart rate was monitored in 53 subjects (aged 50-52  $\pm$  15 years) over three months, alongside subjective perception of noise and sleep quality. Thirty one subjects lived to the west of the airport, and were exposed to a nocturnal equivalent continuous air traffic noise level of  $L_{Aeq} = 50$  dB outside during departures from runway 25. Twenty-two subjects lived east of the airport and were exposed to  $L_{Aeq} = 50$  dB during departures from runway 07. During opposite flight directions, aircraft noise corresponded to  $L_{Aeq} = 40$  dB in both areas. The airport operated runway 25 for about 75% of the time, and runway 07 for 25% of the time. Average blood pressure was significantly higher in the West group with higher noise exposure. Morning systolic and diastolic blood pressure was higher in the west group. The East group exhibited a daily parallel between changes in noise and their subjective noise perception, which was not found in the west group. The authors suggested that this was a consequence of higher noise stress levels in the West group, and concluded that a nocturnal aircraft noise level of  $L_{Aeq} = 50$  dB can have negative effects on subjective noise perception and on objective parameters of circulation.
- 4.2.25 A paper by Basner, Griefahn and van den Berg (2010) focuses on an Anotec Consulting study in 2003, which examined 400,000 people that were exposed to a  $L_{night} > 45$  dB, around 53 major airports in the EU. The authors explain that this is likely to increase to 550,000 in 2015 and aimed to analyse noise-induced sleep disturbance by looking at:

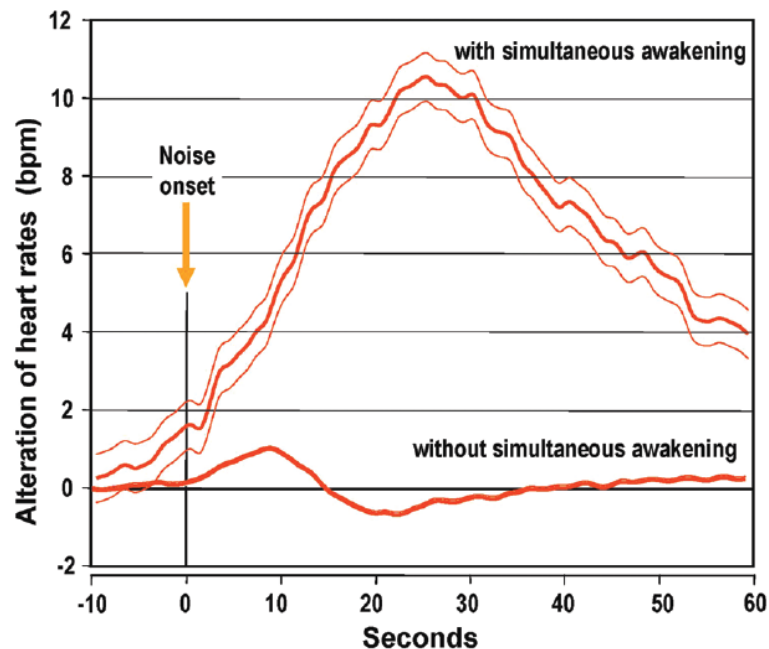
- Event-related analysis
- Whole night sleep parameters
- Dose-response relationships
- Mitigation of aircraft noise effects
- Vulnerable groups
- Research needs

4.2.26 Event –related analysis is discussed, which establishes a direct association between an ANE and the reaction of the subject, although because awakenings occur spontaneously as well as a response to aircraft noise this must be taken into account. EEG awakenings are most often used as predictor of long-term health effects because:

- Awakenings are strongest form of activation
- Specific
- Awakenings usually occur with increases in heart rate, which in turn can play a role in high blood pressure and cardiovascular disease

4.2.27 The following graph (**Figure 13**), taken from Griefahn (2006) shows the noise-induced alterations in heart rate with and without simultaneous EEG awakenings.

**Figure 13:** Noise induced changes in heart rate with and without EEG awakening



4.2.28 With EEG awakenings the maximum average heart rate increased by 10 b.p.m and did not reach baseline levels 60s after onset. Without EEG awakening the maximum average heart rate increased by 1 b.p.m and reached baseline levels 15 seconds after noise onset.

4.2.29 These data highlights the importance of the relationship between a noise stimulus and the autonomic cardiovascular responses should awakening as defined by changes in the EEG occur, and illustrates the need to keep additional awakenings induced by aircraft noise at night, to a minimum.

4.2.30 The analysis of whole night sleep parameters resulted in the following findings:

- Noise can result in an overall heightened state of arousal level that leads to a redistribution of time spent in different sleep stages
- An increase in wake and stage 1 sleep
- Decrease in REM and SWS
- Although overall changes are relatively small, these could be of clinical relevance in sensitive populations or chronic exposure situations in terms of short-term (e.g. daytime sleepiness) and long-term (hypertension) health effects

4.2.31 This detailed paper stresses the need for future large scale field studies on the effects of nocturnal aircraft noise on sleep. It is suggested that several groups of the population are included, such as children and chronically ill. Long-term studies are needed to investigate the future consequences of noise-induced sleep disturbance. Further recommendations include epidemiological case-control studies on the association of nocturnal aircraft noise exposure and cardiovascular disease.

4.2.32 Greiser et al (2011) published research concerning the risk increase of cardiovascular diseases and impact of aircraft noise in the Cologne-Bonn airport study. Previously, research had shown that there was an increase in the amount of cardiac medication prescribed with increasing aircraft noise exposure (2007). Aircraft, road and rail noise data were linked to hospital discharge diagnoses of 1,020,528 people living in the study area. Confounders included age, environmental noise, prevalence of social welfare recipients of residential quarters and interaction of aircraft noise with age. The results showed that as age increased, the risk of cardiovascular disease decreased. Risk is more marked in females than males. For night-time aircraft noise of 50 dB  $L_{\text{night}}$  at aged 50, the odds ratio for cardiovascular disease in men was 1.22 and in women 1.54, for myocardial infarction it was 1.18 in men and 1.54 in women, for heart failure in men 1.52 and 1.59 in women, stroke in men 1.36 and for women 1.36 also.

4.2.33 Floud et al (2011) reported on the medication use in relation to aircraft noise of populations surrounding six European airports, as part of the HYENA study. Differences were found between countries in terms of the effect of aircraft noise on antihypertensive use. For night-time aircraft noise a 10 dB increase was associated with an odds ratio of 1.34 (95% CI 1.14 to 1.57) for the UK and 1.19 (CI 1.02 to 1.38) for the Netherlands but no significant associations were found for other countries. There was also an association between aircraft noise and anxiolytic (anti-anxiety) medication, OR 1.28 (CI 1.04 to 1.57) for daytime and OR 1.27 (CI 1.01 to 1.59) for night-time. This effect was found across countries. The authors concluded that although results suggested a possible effect of aircraft noise on the use of antihypertensive medication, the effect did not hold for all countries. The data was more consistent for anxiolytics in relation to aircraft noise across countries.

### **4.3 Ischemic Heart Disease (IHD), including Myocardial Infarction (MI)**

4.3.1 Many studies investigating the cardiovascular effects of aircraft noise examine a range of health outcomes. Some of the studies mentioned in the previous section include references to IHD, however there are studies that specifically focus on this health measure. Examples of such research are given in this section.

4.3.2 Two studies (Babisch, 1999) 'Caerphilly & Speedwell Studies' were undertaken to investigate the hypothesis that prolonged exposure to traffic noise at home increases

the risk of IHD. The increase in risk in the noise-exposed areas was assessed relative to populations where the noise levels were less than 55 dBA. After the cohorts had been studied over a 10-year period, it was concluded that, solely on the basis of the Caerphilly and Speedwell studies it cannot be deduced that traffic noise increases the risk for IHD.

- 4.3.3 In 2000 Babisch published a comprehensive review of the literature on environmental noise and cardiovascular disease. Of the 10 studies reviewed by Babisch, four showed associations between traffic noise and hypertension. Of these Babisch considered that two met requirements in terms of controlling sufficiently for confounding factors. He concluded that there was little epidemiological evidence of an increased risk of hypertension in subjects exposed to traffic noise and some evidence regarding the association between transportation noise and IHD. In 2006 Babisch updated his review to incorporate new studies published since 2000. He concluded that:
- 4.3.4 There is no evidence from epidemiological data, that community noise increases (mean) blood pressure in the adult population. However, he notes that this lack of evidence does not discard the hypothesis that there may be a relationship between transportation noise and blood pressure but that the studies undertaken suffer from insufficient power and design difficulties.
- 4.3.5 With regard to aircraft noise and hypertension evidence has improved since the previous 2000 review – showing higher risks in higher exposed areas (approximate daytime average noise levels in the range 60 to 70 dBA). The findings for road traffic noise show no consistent pattern.
- 4.3.6 For IHD the evidence of association between community noise (review focused mainly on road traffic noise but did include some aircraft noise studies) has increased since the previous review. There is not much indication of a higher IHD risk for subjects who live in areas with daytime average noise levels of less than 60 dBA but across studies for higher noise categories, a higher IHD risk was relatively consistently found – however, statistical significance was rarely achieved.
- 4.3.7 The HC and ECA Reviews, and a review by Stansfeld (2000), concluded that the available evidence does not appear to convincingly demonstrate an association between aircraft noise and hypertension or IHD. However, they do conclude that the available studies provide some evidence to suggest that there may be a slight risk of IHD. All reviewers recommend that further research is needed to examine the impact of noise on cardiovascular health. The HCN Review considers that above exposures of 70 dB  $L_{Aeq,16h}$  there is sufficient evidence for noise-induced IHD and hypertension.
- 4.3.8 In an analysis of 43 epidemiological studies (published between 1970 and 1999 for both occupational and environmental exposure) that investigated the relationship between blood pressure and/or IHD disease, van Kempen (2002) concluded that the evidence on noise exposure, blood pressure and IHD is still limited. With respect to hypertension, results were contradictory, a significant association was found for air traffic noise and hypertension but there was little evidence of an increase in blood pressure in subjects exposed to road traffic noise. For IHD, only a few studies were available and the evidence for association between noise exposure and IHD was found to be inconclusive.
- 4.3.9 A study (Willich, 2006, Babisch, 2005) was undertaken in Berlin to determine the association between chronic exposure to road traffic noise and the risk of cardiovascular disease (specifically myocardial infarction). The data were analysed

using different approaches by two research groups, both groups conclude that chronic exposure to road traffic noise increases the risk for cardiovascular disease and that the level of risk appears to be related to gender; however, the level of risk determined varies between the two approaches.

- 4.3.10 The Defra report examined the effects of environmental noise and the risk of cardiovascular disease, and the main conclusion drawn was that current research suggests an increasing relative risk of myocardial infarction in people living in areas with road traffic sound levels measured outdoors above 65 dB  $L_{Aeq,16h}$  day, increasing up to about 1.4 to 1.5 in areas with road traffic sound levels measured outdoors above 75 dB  $L_{Aeq,16h}$  day.
- 4.3.11 Harding et al (2011) on behalf of the Health and Safety Laboratory published a report on the quantification of noise related hypertension and the related health effects. The aims of the study were to identify the potential health outcomes associated with hypertension, to prioritise the health outcomes and quantify the links between noise and selected hypertension associated health outcomes. The second half of the report covered a methodology to allow a monetary value to be placed on the links between hypertension and health outcomes. This half of the study will be covered in section 6 of this report.
- 4.3.12 The base dose-response function for noise and hypertension used by Harding comes from Babisch and van Kamp (2009) who found an odds ratio for hypertension of 1.13 per 10 dBA increase in  $L_{den}$  in the range 45 to 70 dBA. Harding goes on to note that because the prevalence of hypertension in the population is greater than ten percent, that the odds ratio must be converted into relative risk in order to quantify the effect on the population.
- 4.3.13 Previously, IGCB(N) and WHO have considered that there is insufficient certainty from which to quantify the health outcomes from hypertension. However, Harding et al, after extension review, found the following health outcomes from hypertension could be quantified:

#### Cardiovascular disease

- 4.3.14 The report concluded that there is substantial evidence for hypertension and blood pressure being an independent risk for cardiovascular disease (CVD). Many studies investigating hypertension or blood pressure as an independent causal factor for CVD have used separate analyses for stroke and IHD. It has been suggested that systolic blood pressure may be a better indicator of CVD risk than diastolic blood pressure.

#### Stroke

- 4.3.15 The report discusses evidence of blood pressure being linked to all types of stroke, ischaemic (resulting from a clot) and haemorrhagic (rupturing of blood vessels within the brain). Hypertension is a known risk factor for strokes.

#### Ischaemic Heart Disease (IHD)

- 4.3.16 There is strong evidence for a link between blood pressure and the incidence and mortality of IHD. IHD is due to the build up of plaque deposits on the artery walls and therefore leads to hardening of the arteries. When the plaque comes away from the walls, blockages can occur in the arteries which can cause a lack of oxygen (ischaemia) in the heart muscle. When the rupture of plaque on the coronary arteries occurs a clot can form, which can subsequently cause a rapid slowing or stop of blood

flow and then the classic heart attack (myocardial infarction). There is evidence that lowering blood pressure can help prevent heart attacks.

### Dementia

- 4.3.17 The report discusses the evidence linking hypertension and dementia, or cognitive decline. The evidence is less strong than for cardiovascular disease, and is complicated by the ethical issues involved in studying long-term hypertension without treatment and also because by the time dementia manifests, hypertension can decrease as a result of weight loss or metabolic changes. There have also been findings that link cognitive decline with blood pressure in subjects ages 59-71 years.
- 4.3.18 The report also discussed the links between hypertension and end stage kidney disease, pregnancy, eye conditions and sexual function, but it was decided that based on the strength of the evidence and impact on the population that three health outcomes would be given priority in terms of quantification of links between noise and hypertension. These were Acute Myocardial Infarction (AMI), stroke and dementia. The outcomes of the quantification process for these end points are outlined in Section 6 of this report.
- 4.3.19 It should be noted that this study was designed to assess the risk of noise-related hypertension on the subsequent likelihood of hypertension resulting in the above health outcomes; it is not reporting that noise itself directly causes stroke and dementia.

### Stress and mental health effects

- 4.3.20 Various reviews on environmental noise and health have concluded as follows:
- HCN (1999): the evidence for a causal effect between noise exposure and biochemical effects is limited.
  - HC (2002): the available research does not support the contention that there is a significant risk of chronic stress arising from long term exposure to outdoor daily aircraft noise levels above 65 dBA.
  - ECA (2004): internationally the evidence from epidemiological studies for an impact on long term stress is limited or suggestive only.

All reviews identify the need for further research in this area.

- 4.3.21 However, some recent studies have identified elevated levels of stress hormones in association with noise exposure at night-time and in children exposed to aircraft noise.
- 4.3.22 The contractility of the stomach was examined in relation to different types of noise (Castle et al, 2007). Subjects were exposed to hospital noise, traffic noise and conversation babble and their gastric myoelectrical activity was recorded. The results indicated that loud noise altered the electrical activity in the stomach particularly in younger people under the age of 50 years.
- 4.3.23 Black et al (2007) suggest that although there are often instances of increased pharmaceutical drugs for hypertension and stress around airports, no studies have applied cognitive behavioural therapy (CBT) as an intervention to alleviate stress experienced by residents from long-term exposure to aircraft noise living around commercial or military airports, and this may be a valuable tool in helping to decrease the stress-inducing effects of aircraft noise.

- 4.3.24 The published research findings on the impact of night-time environmental noise exposure on stress hormone levels are inconsistent. Maaß (2004) reports findings of a sleep laboratory study and associated field study investigating the effects of nocturnal aircraft noise; he found no significant influence of aircraft noise on excretions of stress hormones or electrolytes.
- 4.3.25 Maschke (2004) has observed that average stress hormone levels may be acutely raised by traffic noise at night. At the same time, the quality of the sleep experienced by the test persons and their feeling of well-being next morning is poorer. Exposure to 16 overhead flights with maximum levels of 55 dBA produced a significant increase in the secretion of stress hormones. He also notes that the general findings in relation to noise exposure at night and stress hormone levels in overnight urine samples are inconclusive, and show individuals with increases in stress hormone levels and others with decreased values.
- 4.3.26 In a study by Babisch (2001) of middle aged women living in Berlin, whose bedrooms or living rooms faced streets of varying traffic volume, significant associations were found between noise exposure and the nocturnal secretion of stress hormones in urine, with regard to exposure in the bedroom (but not in the living room). This indicated a higher chronic physiological stress response in noise exposed subjects as compared to the less exposed. Babisch concludes that, the fact that noise effects were only seen with regard to exposure of the bedroom and not the living room of the subjects, suggests that particularly night-time disturbances of sleep may be associated with adverse effects of traffic noise.
- 4.3.27 Based on a review of recent studies on the relationship between traffic noise disturbance at night and increases in stress hormones Ising (2004) concludes that:
- “...noise exposures over time periods of years may induce, in a certain percentage of exposed persons, permanent changes of stress hormone regulation, along with possible consequences in terms of functional and organic damages.”
- 4.3.28 In a review of the literature on environmental noise and mental health Stansfeld (2000) concluded that current evidence does seem to suggest that environmental noise exposure, especially at higher levels, is related to mental health symptoms (such as depression) and possibly raised anxiety and consumption of sedative medication, but there is little evidence of more severe health problems such as clinically definable psychiatric disorder. For example (examples taken from Stansfeld's Review):
- A questionnaire study of 1053 residents living around Kadena military airport in Japan found an association between the highest noise exposure group and higher scores of depressiveness and neurosis.
  - In a British study of 7540 people exposed to road traffic noise, it was found that the noise level was weakly associated with a mental health symptoms scale.
  - A study of the impact of traffic noise (undertaken in Caerphilly) found that there was no association between road traffic noise and minor psychiatric disorder. However, there was a small non-linear association of noise with increased anxiety scores.
  - A Health Impact Assessment around Schiphol Airport suggested that the use of non-prescribed sleep medication or sedatives was associated with aircraft noise exposure during the late evening, but not with exposure during the night. Vitality related health complaints such as tiredness and headache were associated with

aircraft noise, whereas most other physical complaints were not.

4.3.29 Meister (2000) reports on a questionnaire based survey (among 2001 respondents living in Minnesota, USA) to assess the impact of commercial aircraft noise on human health. Four of the neighbourhoods in the survey were exposed to aircraft noise and two non-exposed control communities were also included. Meister found:

- All general health measures were significantly worse for the neighbourhoods exposed to aircraft noise than for the controls – the greater the noise levels the worse the health measures were.
- Mental health scores in neighbourhoods exposed to noise were lower than the scores in the control neighbourhoods (higher score implies more positive health status).
- A sense of vitality reduced among those exposed to aircraft noise compared with those not exposed.
- Stress levels were higher among those exposed to aircraft noise; as stress increased mental health and a sense of vitality decreased.

4.3.30 Stansfeld (2000) reports that studies from the 1970s and 1980s found that a high percentage of people reported headaches, restless nights and being tense and edgy in high noise areas. However, an explicit link between aircraft noise and symptoms in these studies raises the possibility of a bias towards over-reporting, due to personal attitudes towards aircraft noise. A study around three Swiss airports, which did not mention that the study was related to aircraft noise, did not find any association between the level of aircraft noise exposure and symptoms.

4.3.31 Evidence that exposure to aircraft noise is associated with higher psychiatric admission rates is mixed. Early studies (in the 1970s) around Heathrow and Los Angeles Airports found weak associations between the level of aircraft noise and psychiatric hospital admissions in the general population. These studies have been criticised on methodological grounds and further comprehensive studies have found, at most, a moderating rather than a causal role for noise on hospital admission rates. However, Kryter (1990) found an association between aircraft noise and psychiatric hospital admission rates in a re-analysis of data accepting admissions from around Heathrow Airport.

4.3.32 Researchers suggest that it may be that certain groups are more vulnerable to noise in the mental health context – particularly, children, the elderly and people with pre-existing illness, especially depression.

4.3.33 The Defra and HPA reports did not conclude that there is sufficient evidence for a reliable dose-response relationship between environmental noise and psychological health, and therefore suggest that this is an area that requires further investigation before any conclusions can be drawn.

4.3.34 Catecholamines are chemical compounds that function as neurotransmitters or hormones, and can be measured in urine or blood. Examples of catecholamines include noradrenaline and dopamine, which act as neuromodulators in the central nervous system, and as hormones in the blood circulation.

4.3.35 Catecholamine levels can be measured as an indicator of stress, which can be induced from psychological reactions or environmental stressors such as increased sound levels, intense light, or low blood sugar levels. They cause general physiological changes that prepare the body for physical activity (fight or flight), and



typical effects are increases in heart rate, blood pressure, blood glucose levels, and a general reaction of the sympathetic nervous system.

- 4.3.36 Carter *et al* (1994) studied catecholamines in urine, cardiac arrhythmia and arousals in sleep in response to environmental noise. Nine subjects who were already documented with cardiac arrhythmia over 4 nights were investigated in a sleep laboratory. Cardiac arrhythmia (CA) has prognostic significance in people with heart disease, and raised serum catecholamines may be related to increased blood pressure and risk of heart disease. CA is common in the adult population and the causes behind arrhythmic events such as ventricular premature contractions (VPCs) are not well understood.
- 4.3.37 Research suggests that heart rate is responsive to environmental noise events during sleep, the response consisting of an increase followed by a decrease. Concentrations of circulating catecholamines normally reach their nadir during sleeping hours. Because noise affects heart rate during sleep, it is conceivable that serum catecholamine levels are also increased by noise-induced arousal during sleep.
- 4.3.38 EEG and ECG were recorded throughout each night, with the first night used for familiarisation, then two counterbalanced nights of truck or aircraft noise and one quiet night. Sleep stage and noise were related to the probability of an arousal (in this case an alpha wave response), but there was no interaction between the two factors. The probability of an alpha wave response decreased from stages 1-4 and in REM was similar to in stage 2. Alpha wave latency was found to be shorter in noise than in quiet intervals. Noise and sleep stage at interval (noisy or quiet) onset were related to the number of sleep stage changes during the interval, with reliably more sleep stage changes in noisy than in quiet intervals. Four subjects showed frequent VPCs during the experiment, and were significantly related to sleep stage but not to noise events. The excretion of urinary catecholamines did not differ between noise and quiet nights.
- 4.3.39 Cortisol is also an important hormone that is associated with stress, and is released by the adrenal glands. Concentrations are typically highest first thing in the morning, on waking, and lowest during sleep. Spreng (2002) assessed cortical excitations, and cortisol excretion in relation to an estimation of tolerable nightly over-flights.
- 4.3.40 Noise induces cortisol excretion even below the awakening threshold. Repeated noise events such as over-flights during night time leads to an accumulation of the cortisol concentration in the blood, due to its time constant of exponential decrease being about 10 to 50 times larger than for adrenaline and noradrenaline. For example the time course for the metabolism of cortisol is 64 minutes, compared to adrenaline seconds to 3 minutes, and noradrenaline 7 to 12 minutes.
- 4.3.41 An attempt was made to calculate cortisol accumulation using an initial value of noise induced small cortisol increase at the nightly threshold of beginning vegetative overreaction around 53 dBA. The range of minimal and maximal normal cortisol values were used as a borderline and the relation between maximum sound pressure level and cortical excitation was taken into account and a formula developed to estimate tolerable events during night-time periods. An example of the results over 8hrs in the night was values of 11 events with 5 dBA indoor maximum level, or 5 events with 75 dBA indoor maximum level respectively.
- 4.3.42 Stress hormones also represent a link between noise and health impairment. The average concentration may be raised by traffic noise at night, with simultaneous deleterious effects on sleep quality and well-being the following morning.

- 4.3.43 Persson Wayne *et al* (2004) studied the cortisol response and subjective sleep disturbance following low-frequency noise, in a counterbalanced design with half of the subjects exposed to a sound pressure level of 40 dBA on their fourth night in the sleep laboratory, with a comparative reference night on the fifth night, and the opposite for the other half. Subjective sleep disturbances were recorded by questionnaires and cortisol response upon awakening was measured in saliva.
- 4.3.44 Subjects were more tired and less socially orientated in the morning after nights with low-frequency noise, and mood was negatively affected also in the evening after nights with low-frequency noise. There was no effect of noise condition on cortisol response, but there were effects of group and weekday, suggesting that more work needs to be done before cortisol response can accurately be used as an indicator of noise-disturbed sleep.
- 4.3.45 The WHO NNG (2009) concludes that evidence does suggest that environmental noise exposure at higher levels is related to mental health symptoms and possibly raised anxiety, but there is little evidence that it has more serious effects. There is not strong evidence for the association between noise exposure and mental ill health, except perhaps above 70 dB  $L_{Aeq}$ . The document highlights that as most studies have examined the effects of daytime noise on mental health, it cannot be ruled out that night-time noise may have effects on mental health at lower levels than daytime noise.

#### 4.4 Next day effects

- 4.4.1 The term 'next day effects' refers to the possible outcomes resulting from aircraft noise exposure that can be observed at a later stage. Generally this refers to cognitive performance and sleepiness or fatigue felt the following day.
- 4.4.2 Schapkin *et al* (2006) report that the scientific literature on whether noise-induced sleep disturbance affects the next day performance of adults is mixed. He notes that the scientific literature suggest that disturbed sleep affects performance in complex tasks, but that performance in simple psychomotor tasks can probably be prevented by individuals exerting additional effort. Schapkin investigated the impairment of neuronal mechanisms underlying performance after sleep disturbance by measuring event-related brain potentials (ERPs) – this is a new approach to investigating the impact of night-time noise. His results suggested that physiological costs to maintain performance are increased after noisy nights and that ERPs may be more sensitive indicators of moderate sleep disturbances caused by noise than performance measures.
- 4.4.3 The WHO Guidelines report that studies of both laboratory subjects and workers exposed to occupational noise, have found that noise adversely affects cognitive task performance. Such studies have shown that although noise induced arousal may produce better performance in simple tasks in the short term, cognitive performance substantially deteriorates for more complex tasks. Reading, attention, problem solving and memorisation are among the cognitive effects most strongly affected by noise.
- 4.4.4 There have been a number of field studies of school children, which have observed that noise impairs their cognitive performance, however, according to the WHO Guidelines there is no published research on whether environmental noise at home impairs cognitive performance in adults.

- 4.4.5 In agreement with the WHO Guidelines, other reviews report that there is good evidence from laboratory studies that noise exposure impairs performance in adults. The literature search and reviews considered have not identified any new research published since the WHO Guidelines, which contributes significantly to the understanding of the impact of aircraft noise on the performance in adults. However, reference has been found<sup>7</sup> to a paper published in 1986, which compared the self-reports of everyday errors (failures of attention, memory and action) by subjects living in an area of West London exposed to a high level of aircraft noise with those in a similar group who lived in an area with low level of aircraft noise. The high-aircraft noise group reported a higher frequency of everyday errors and so did noise-sensitive subjects. According to Stansfeld (2000), concern has been expressed that there may be some confounding by neuroticism in these findings, and studies of the effects of noise on cognitive tasks do suggest that neuroticism and anxiety are important in determining individual differences in response to noise.
- 4.4.6 Basner (2008) published a paper on the effects of nocturnal aircraft noise exposure and daytime sleepiness. The purpose of the study was to objectively assess daytime sleepiness following aircraft exposure at night, using rhythmic changes in pupil diameter that are regulated by the autonomic nervous system. These changes are referred to as fatigue waves, and the measurement is given as the Pupillary Unrest Index (PUI), which is high in sleepy subjects. The results showed that nocturnal aircraft noise resulted in increased objective daytime sleepiness. Sleepiness levels increased significantly with an increase in the number of aircraft noise events ( $p = 0.021$ ), maximum sound pressure levels ( $p = 0.028$ ) and also with an increase in  $L_{Aeq}$  ( $p = 0.013$ ). These levels were not sufficiently high to reach pathological levels, as observed in a study on obstructive sleep apnoea patients. Basner discussed, however, the importance of this finding in terms of objective measurements of sleepiness, and the need to investigate such objective methodology in the field also.
- 4.4.7 The HCN Review concludes that the evidence for causal relationship between environmental noise and decreased general performance is limited.

## 4.5 Children

- 4.5.1 Children are generally considered to be a vulnerable group, that may be less able to cope with the impacts of noise exposure and they may be at greater risk of harmful effects. In a review of the non-auditory effects of noise on health, Stansfeld (2003) explains that:
- “It is likely that children represent a group which is particularly vulnerable to the non-auditory effects of noise. They have less cognitive capacity to understand and anticipate stressors and lack well-developed coping strategies. Moreover, in view of the fact that children are still developing both physically and cognitively, there is a possible risk that exposure to an environmental stressor such as noise may have irreversible negative consequences for this group.”
- 4.5.2 Stansfeld (2000) also notes that some children in the population may be more vulnerable to noise effects than others. He concludes that there is limited evidence that children who have lower aptitude or other difficulties, such as learning difficulties and cerebral palsy, may be more vulnerable to harmful effects of noise on cognitive performance.
- 4.5.3 The WHO Guidelines provide a brief overview of the effects of environmental noise on children. They conclude that chronic exposure to aircraft noise during early childhood

appears to impair reading acquisition and reduces motivational capabilities (this is based on the studies of Los Angeles and Munich Airports – see below). It is also noted that of recent concern are the concomitant psychophysiological changes (blood pressure and stress hormone levels). The WHO Guidelines consider that the evidence on noise pollution and health is strong enough to warrant monitoring programmes at schools, and that schools should not be located near major noise sources, such as airports.

4.5.4 During and since the late 1990s there has been a significant amount of research published investigating the effects of aircraft noise on children (particularly focusing on cognitive effects). Substantial studies have been undertaken around European airports:

- The Munich Airport Study (Hygge, 1998) took advantage of a natural experiment created by the closing of an existing airport and the opening of a new airport. Before the change over of airports, children at both sites were recruited into experimental and control groups. One set of data were collected prior to the change over of the airports, the second set a year later and a third set two years later. The children were assessed on physiological, perceptual, cognitive, motivational and quality of life measures.
- The West London Schools Study (WLSS – Stansfeld, 2000) a cross-sectional study which was carried out in schools in the area surrounding Heathrow Airport, to determine the association of aircraft noise exposure with cognitive performance. A total of 236 children from 20 schools took part in the study, 10 high noise schools and 10 control low noise schools.
- The Schools Environment and Health Study (SEH) – Haines (2001) - a study around Heathrow airport to compare the school performance and health of children attending four schools in a high aircraft noise area, with those of children from four matched control schools in a low aircraft noise area.
- The RANCH study (Road Traffic and Aircraft Noise Exposure and Children's Cognition and Health; Effect Relationships and Combined Effects) – Stansfeld (2005) – a cross-sectional study that enrolled a total of 2,844 children from 89 schools around Schiphol (Netherlands), Heathrow and Barajas (Spain) Airports. This Study is the largest known epidemiological study undertaken of exposure and children's cognition and health.

4.5.5 A body of research available from a study undertaken around Los Angeles Airport by Cohen et al (1980, 1981) published in the early 1980s is also widely cited in the scientific literature. In the Los Angeles Study children in four schools exposed to high levels of noise were matched with children in three low noise schools, a first wave of measurements were followed up a year later.

4.5.6 The findings of these key studies are summarised below, along with pertinent findings from other recently published studies.

#### Cognition in children

4.5.7 Across the literature the evidence for the effects of noise exposure on child health is strongest for cognitive effects; however the effects of noise have not been found uniformly across all cognitive functions. Stansfeld (2003) summarises (this summary includes amongst others the findings of the Munich, Heathrow and Los Angeles studies described above) the effects that have been found for children exposed to high levels of environmental noise as:

- Deficits in sustained attention and visual attention.
- Difficulties in concentrating (based on teachers' reports).
- Poorer auditory discrimination and speech perception.
- Poorer memory requiring high processing demands.
- Poorer reading ability and school performance on national standardised tests.

4.5.8 More recent substantive findings on cognitive performance come from the RANCH Study. This study found that exposure to chronic aircraft noise could impair cognitive development in children, specifically reading comprehension. The results indicated a linear exposure-effect association between exposure to aircraft noise and impaired reading comprehension and recognition memory in children. The study found that aircraft noise exposure was not associated with recall, impairment in working memory, prospective memory or sustained attention. For road traffic noise the study found no association with reading comprehension, recognition, working memory, prospective memory or sustained attention and that exposure to road traffic noise improved recall; the RANCH team could find no definitive explanation for this latter finding. Stansfeld suggests that aircraft noise, because of its intensity, the location of the source and its variability and unpredictability is likely to have a greater effect on children's reading than road traffic noise, which might be of a more constant intensity.

4.5.9 Shield (2003) compared external noise levels at over 50 London schools (schools were not in areas exposed predominantly to aircraft noise) with the schools' scores in standardised assessment tests (SATs) of children aged 7 to 11. She found significant relationships between external noise levels and SATs scores, with environmental noise having a detrimental effect upon children's performance; the relationship being stronger for older children. A similar study was carried out at schools located around Heathrow airport, in this study no obvious strong consistent relationship was found between noise and SATs scores, although the results suggest that aircraft noise may have a negative effect upon SATs scores for reading.

4.5.10 The HCN Review considers the findings of the Munich, WLSS and Los Angeles studies and concludes that there is sufficient evidence for a causal relationship between aircraft noise and the performance of children in schools.

4.5.11 Stansfeld et al (2010) examined the effect of night-time aircraft noise exposure on the cognitive performance of children. This analysis was an extension of the RANCH study, and the Munich study in which 330 children were assessed on their cognitive performance in three waves, each a year apart, before and after the switch over of airports. Aircraft noise exposure and self-reported sleep quality measures were analysed across airports to examine whether changes in night-time noise exposure had any impact on reported sleep quality, and if this was then reflected in the pattern of change in cognitive performance. In the Munich study analysis of sleep quality questions showed no evidence of interactions between airport, noise and measurement wave, which suggests that poor sleep quality does not mediate the association between noise exposure and cognition. In the RANCH study, there was no evidence to suggest that night noise had any additional effect to daytime noise exposure. The authors explain that this investigation utilised secondary data and therefore was not specifically designed to investigate night time aircraft noise exposure on cognitive performance in children, but the results from both studies suggest that night time aircraft noise exposure does not appear to add any further deleterious effect to the cognitive performance decrement induced by daytime noise alone. They recommend that future research should be focussed around the school, for the protection of children against the effects of aircraft noise exposure on performance.

4.5.12 It is important to note that studies on children are mostly designed to focus on daytime noise exposure during learning; therefore there is limited or no information on night time specific effects. Children are included as part of the vulnerable groups, however, and therefore should be given due consideration in this way.

#### **4.6 Health Effects: Conclusions**

##### Hypertension, Ischemic Heart Disease and Myocardial Infarction

4.6.1 In terms of cardiovascular impact there are mixed conclusions from the various reviews and papers on the evidence for effects. Some reviewers consider that there is sufficient evidence, others that the evidence does not convincingly demonstrate an association. Based on existing evidence, it is possible that exposure to aircraft noise may be a risk factor for cardiovascular disease and all would agree that further research is needed to examine the impact of noise on cardiovascular health. For Myocardial Infarction, the WHO Environmental Burden of Disease report suggests that night time effects may be of the same magnitude as day time effects, and therefore proposes an Odds Ratio of 1.1 for 60-65 dBA  $L_{\text{night}}$  and an Odds Ratio of 1.2 for 65-70 dBA  $L_{\text{night}}$ .

##### Stress and Mental Health

4.6.2 Reviewers generally consider that the evidence for mental health effects is inconclusive or limited. There seems to be a trend emerging of some evidence for mental health symptoms (eg depression, anxiety) but not of more severe health problems such as clinically defined psychiatric disorder.

4.6.3 The scientific literature generally finds that the evidence for long term impact on stress hormone levels is inconclusive or limited.

##### Next day effects (adults)

4.6.4 There is a lack of data on the impact of environmental noise on the performance of adults and no firm conclusions can be drawn. Across the scientific literature it is agreed that above a certain threshold, environmental noise can cause awakening, and at levels significantly lower, it can also induce sleep stage changes. The threshold level above which effects are found remains a controversial point. There also seems to be general consensus that environmental noise can affect subjective sleep quality, mood the next day and has an acute impact on heart rate. However, as yet, there appears to be no strong/consistent scientific evidence of chronic objective effects (e.g. on stress hormone levels or immune system) or performance the next day.

##### Noise and Children

4.6.5 There is a growing body of literature on the impact of aircraft noise on children's health. Across the literature the evidence for the effects of noise exposure on child health is strongest for cognitive effects (particularly reading). Some studies have found that chronically noise exposed children have raised levels of stress, increased blood pressure and mental health effects; however there is still insufficient data to provide unequivocal evidence of such effects

## 5 Noise Levels at which Health Effects Occur

5.1.1 The WHO NNG (2009) included tables on the observed effect thresholds of noise. The threshold levels for sufficient and limited evidence were presented.

5.1.2 Sufficient evidence is defined as: A causal relation has been established between exposure to night noise and a health effect. In studies where coincidence, bias and distortion could reasonably be excluded, the relation could be observed. The biological plausibility of the noise leading to the health effect is also well established.

5.1.3 Limited evidence is defined as: A relation between the noise and the health effect has not been observed directly, but there is available evidence of good quality supporting the causal association. Indirect evidence is often abundant, linking noise exposure to an intermediate effect of physiological changes, which lead to the adverse health effect.

5.1.4 **Table 2** summarises the sufficient evidence for exposure to night noise and health effects as given in the WHO NNG (2009).

**Table 2** Summary of effects and threshold levels for effects where sufficient evidence is available (taken from WHO NNG, 2009)

	Effect	Indicator	Threshold, dB
Biological effects	Change in cardiovascular activity	*	*
	EEG awakening	L <sub>Amax,inside</sub>	35
	Motility, onset of motility	L <sub>Amax,inside</sub>	32
	Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep	L <sub>Amax,inside</sub>	35
Sleep quality	Waking up in the night and/or too early in the morning	L <sub>Amax,inside</sub>	42
	Prolongation of the sleep inception period, difficulty getting to sleep	*	*
	Sleep fragmentation, reduced sleeping time	*	*
Well-being	Increased average motility when sleeping	L <sub>night, outside</sub>	42
	Self-reported sleep disturbance	L <sub>night, outside</sub>	42
Medical conditions	Use of somnifacient drugs and sedatives	L <sub>night, outside</sub>	40
	Environmental insomnia	L <sub>night, outside</sub>	42

\* Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.

5.1.5 **Table 3** summarises the limited evidence for which there may be a health effect due to night noise.

**Table 3** Summary of effects and threshold levels for effects where limited evidence is available (taken from WHO NNG, 2009)

	Effect	Indicator	Threshold, dB
<b>Biological effects</b>	Changes in (stress) hormone levels	*	*
	Drowsiness/tiredness during the day/evening	*	*
<b>Well being</b>	Increased daytime irritability	*	*
	Impaired social contacts	*	*
	Complaints	Lnight, outside	35
	Impaired cognitive performance	*	*
	Insomnia	*	*
<b>Medical conditions</b>	Hypertension	Lnight, outside	50
	Obesity	*	*
	Depression (in women)	*	*
	Myocardial infarction	Lnight, outside	50
	Reduction in life expectancy	*	*
	Psychic disorders	Lnight, outside	60
	(Occupational) accidents	*	*

\* Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.

#### Griefahn and Scheuch Evaluation Criteria

5.1.6 Based on an extensive review of the literature Griefahn and Scheuch (2004) suggest 'evaluation criteria' specifically for aircraft noise exposure to protect those living in the vicinity of civil airports. The purpose of these criteria is to provide guidance on the noise levels at which control measures need to be introduced, to protect communities around airports from the potential adverse health effects of noise. Griefahn and Scheuch propose a three tier hierarchy of criteria:

- Critical limits – above these levels there is a risk of health effects and such levels should only be tolerated as an exception for a limited time. Above these levels noise it is imperative that noise control measures should be introduced.
- Protection Guides – Exposure below these levels should not induce adverse health effects in the average person, although sensitive groups may still be affected. These are the 'central assessment values' above which action should be taken to reduce noise exposure.



- Threshold Values – inform about measurable physiological and psychological reactions to noise exposure where long term adverse health effects are not expected. To increase quality of life these values constitute a long term goal.

5.1.7 Griefahn and Scheuch’s proposed Critical Limits, Protection Guides and Threshold Values for sleep disturbance, annoyance and cardiovascular disease are shown in **Table 4**. It can be seen that the proposed Threshold Values for annoyance and sleep disturbance are in alignment with the WHO threshold guideline levels. Griefahn notes that although the WHO Guideline Values and proposed Threshold Values provide a long-term goal, achieving them around airports is currently practically impossible without complete cessation of aircraft movements. The Protection Guides and Critical Limits provide more practical ‘tolerable limits’ for the avoidance of adverse health effects in those living in the communities around civil airports.

**Table 4** Griefahn and Scheuch’s proposed Critical Limits, Protection Guides and Threshold Values for Sleep Disturbance, Annoyance and Cardiovascular Disease

Effect	Evaluation Criteria	Measure	Value	Indoor/ Outdoor
Sleep Disturbance*	Critical Limit	dB L <sub>Amax</sub> 22-06 hour	6 events at 60 dBA	Indoor
	Critical Limit	L <sub>Aeq</sub> 22-06 hour	40	Indoor
	Protection Guide	dB L <sub>Amax</sub> 22-06 hour	13 events at 53 dBA	Indoor
	Protection Guide	dB L <sub>Amax</sub> 22-01 hour	8 events at 56 dBA	Indoor
	Protection Guide	dB L <sub>Amax</sub> 01-06 hour	5 events at 53 dBA	Indoor
	Protection Guide	dB L <sub>Aeq</sub> 22-06 hour	35	Indoor
	Protection Guide	dB L <sub>Aeq</sub> 22-01 hour	35	Indoor
	Protection Guide	dB L <sub>Aeq</sub> 01-06 hour	32	Indoor
	Threshold Value	dB L <sub>Amax</sub> 22-06 hour	23 events at 40 dBA	Indoor
	Threshold Value	dB L <sub>Aeq</sub> 22-06 hour	30	Indoor
High Annoyance**	Critical Limit	dB L <sub>Aeq</sub> 06-22 hour	65	Outdoor
	Protection Guide	dB L <sub>Aeq</sub> 06-22 hour	62	Outdoor
	Threshold Value	dB L <sub>Aeq</sub> 06-22 hour	55	Outdoor
Chronic Disease **	Critical Limit	dB L <sub>Amax</sub> 06-22 hour	19 events at 99 dBA	Outdoor
	Critical Limit	dB L <sub>Aeq</sub> 06-22 hour	70	Outdoor
	Protection Guide	dB L <sub>Amax</sub> 06-22 hour	25 events at 90 dBA	Outdoor
	Protection Guide	dB L <sub>Aeq</sub> 06-22 hour	65	Outdoor

\* Griefahn and Scheuch suggest that if it is not possible to have no aircraft movements during the night, then concentrating air traffic to the first part of the night is preferable, as people are less sensitive to noise during the 2200 to 0100 hours time period and disturbances during the early part of the night can be compensated for in the following quieter period. They therefore propose different Protection Guide levels for the earlier and later part of the night as shown above.

\*\* Griefahn and Scheuch found that the data were not strong enough to establish maximum level (L<sub>Amax</sub>) evaluation criteria for annoyance or Threshold Values for chronic disease.

5.1.8 The WHO NNG (2009) concluded that below 30 dB L<sub>night,outside</sub>, no effects on sleep are observed except for a slight increase in the frequency of body movements during sleep due to night noise. It was concluded that there is not sufficient evidence that the biological effects observed at the level below 40 dB L<sub>night,outside</sub> are harmful to health. The relationship between night noise exposure and health effects as summarised in the WHO NNG (2009) are presented in **Table 5**.

**Table 5** Effects of different levels of night noise on the population’s health (taken from the WHO NNG, 2009)

Average night noise level over a year $L_{\text{night, outside}}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{\text{night, outside}}$ of 30 dB is equivalent to the no observed effect level (NOEL) for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, and arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. $L_{\text{night, outside}}$ of 40 dB is equivalent to the lowest observed adverse effect level (LOAEL) for night noise.
40 to 55 dB	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

5.1.9 Table 5 highlights WHO’s view that above 55 dB  $L_{\text{night}}$  noise is a significant concern to public health. As a result it has set an interim target of **55 dB**  $L_{\text{night, outside}}$ . For the longer term it recommends that night noise exposure should be reduced below 40 dB  $L_{\text{night, outside}}$ . It is explained that the interim target is recommended in the situations where the achievement of the NNG is not feasible in the short-term for various reasons. The interim target is not a health-based limit value by itself and vulnerable groups cannot be protected at this level.

5.1.10 In terms of END thresholds, the WHO Night Noise guidelines give clear advice that from the health point of view the calculations of night time burden should start at 40 dB  $L_{\text{night}}$  and that action planning should at least contain actions to bring down the noise level to below 55 dB  $L_{\text{night}}$ . The EEA report suggests that lowering the actual threshold of  $L_{\text{night}} = 50$  dB to  $L_{\text{night}} = 40$  dB would give a better understanding of the magnitude of the problem, and consequently a better allocation of efforts.

## 5.2 Conclusion

- 5.2.1 Whilst agreement upon threshold noise levels that assure effective protection of the health of the population from night-time aircraft noise remains controversial, the evidence highlighted in sections three and four of this report illustrates the growing issue of night noise and health and in particular, the need to reduce the numbers of people exposed to levels above 55 dB  $L_{\text{night, outside}}$  in order to protect public health.

## 6 Economic cost of sleep disturbance

### 6.1 Introduction

- 6.1.1 For the purpose of future policy surrounding night flights within the UK, it is important to assess the both the economic benefits in terms of revenue, employment etc against the health dis-benefits, or costs to the population affected by aircraft noise at night. The following reports mentioned summarise the methodology that has previously been used to calculate such cost-benefits, and the resulting issues that arise.

### 6.2 Defra and HPA reports

- 6.2.1 Two papers have recently been published in this area; the first was commissioned by the Department for Environment, Food and Rural Affairs (Defra) on behalf of their Interdepartmental Group on Cost and Benefit (IGCB) into an estimation of the dose-response relationship between noise exposure and health effects; the second was a Health Protection Agency (HPA) report entitled Environmental noise and health in the UK. Both reports were published in 2009.

- 6.2.2 The aims of the Defra report were:

- To identify a comprehensive list of potential adverse health impacts from noise and review the current state of evidence for each of the impacts;
- Where a robust evidence base exists, to recommend quantitative links (dose-response functions) for the impacts of noise on health which could be applied in the UK;
- Identify any emerging adverse health impacts that should be kept under review for future consideration in evaluation; and
- Identify any structural challenges to developing and maintaining strong quantitative links between noise and health outcomes.

- 6.2.3 In terms of night noise, the Defra report concluded that no single dose-response relationship is recommended for sleep disturbance and noise as a monetary valuation method and that further research into acute, transient and long-term effects are required, however the report did also include the findings relating to daytime noise:

- Strong empirical evidence was identified linking noise to acute myocardial infarction (AMI) (heart attacks) and other cardiovascular illnesses.
- Some evidence was found between noise and other health effects, including annoyance, mental health, hypertension (high blood pressure), sleep disturbance, cognitive development in children and hearing impairment. However, evidence around the monetary valuation of these impacts found in these studies (e.g.

amenity) was not judged to be sufficiently robust to be directly used to monetise noise impacts.

- Structural barriers were suggested to explain why consensus around a single dose-response function for any of these of noise impacts based on health effects may be delayed or prevented.
- The review has also highlighted a number of non-health impacts that may arise from noise. For example, sleep disturbance/loss caused by excessive noise may have negative impacts on both productivity and amenity.

6.2.4 The HPA report included the following:

- Discussions about difficulties in dose-response curves for annoyance and aircraft noise e.g. Scatter and changes in annoyance reactions.
- No reliable relationship between environmental noise and psychological health was found.
- The difficulty with sleep research due to habituation and issues with lab versus field studies was highlighted.
- Recommended an advisory group is set up for future research needs.

### 6.3 European Environment Agency Report – Good Practice Guide on Noise Exposure and Potential Health Effects

6.3.1 The Expert Panel on Noise (EPoN), which is a working group that supports the European Environment Agency and European Commission with the implementation and development of an effective noise policy for Europe, produced this report in 2010.

6.3.2 The group aims to build upon tasks delivered by previous working groups, particularly regarding Directive 2002/49/EC relating to the assessment and management of environmental noise. This good practice guide is intended to assist policymakers, competent authorities and any other interested parties in understanding and fulfilling the requirements of the directive by making recommendations on linking action planning to recent evidence relating to the health impacts of environmental noise and, among others, the WHO Night Noise Guidelines for Europe.

6.3.3 With respect to risk assessment of noise impact, the document refers to the assessment of *attributive fraction* which describes the reduction in disease incidence that would be observed if the population were entirely unexposed, compared with its current (actual) exposure pattern.

$$AF = \left\{ \sum (P_i \cdot RR_i) - 1 \right\} / \sum (P_i \cdot RR_i)$$

where: AF = Attributive Fraction

$P_i$  = Proportion of the population in exposure category  $i$

$RR_i$  = relative risk at exposure category  $i$  compared to the reference level.

6.3.4 An example is presented using the German population exposed to road noise, but it would be possible to do the same with aircraft noise to obtain the percentage number of people exposed and the relative risk of Myocardial Infarction (or other variables) due to aircraft noise.

6.3.5 The paper also discusses the quality targets that should be aimed for within the member states and shows a comparison of the  $L_{den}$  planning values for residential areas between the states. It is noted that although most of the limits are close to the WHO noise and health recommendation of 50/55  $L_{Aeq,16h}$ , some are substantially

higher. For the night-time levels, the averages are  $L_{\text{night}} = 50$  dB for railway road noise, 46 dB for aircraft noise and 42 dB for industry.

## 6.4 WHO Burden of Disease due to Environmental Noise

6.4.1 In this report DALYs are expressed as:

$$\text{DALY} = \text{YLL} + \text{YLD}$$

6.4.2 Where YLL is the number of years of life lost and YLD is the number of years lived with disability.

6.4.3 The Environmental Burden of Disease (EBD) of each end-point was estimated using the following information and data:

- the distribution of environmental noise exposure within the population;
- the exposure–response relationships for the particular health end-point;
- the population-attributable fraction due to environmental noise exposure;
- a population-based estimate of the incidence or prevalence of the health end-point from surveys or routinely reported statistics; and
- the value of DW for each health end-point.

6.4.4 The percentage of “highly sleep disturbed” persons (HSD) due to aircraft noise exposure as a function  $L_{\text{night}}$  was calculated with the equation:

$$\text{HSD}(\%) = 18.147 - 0.956 \times L_{\text{night}} \times 0.01482 \times L_{\text{night}}^2$$

6.4.5 In this case the measure for HSD was based on a self-reported scale of 1-100 of sleep disturbance. A similar approach as taken for annoyance was adopted, with cut off values for HSD chosen as 50 and 72 respectively in order to determine the percentage of people highly sleep-disturbed by transportation noise.

6.4.6 WHO proposed two approaches to calculating EBD from the HSD data:

Exposure based assessment: The exposure-based approach estimates the prevalence of high sleep disturbance (reporting 72 or higher on a 100-point scale) due to noise by combining the exposure data with the exposure–response relationships for high sleep disturbance. One year of night-time exposure to road traffic noise is proposed as the duration causing high sleep disturbance, since people with a bedroom exposed to a road with a high level of night traffic are subject to more or less stationary noise levels at night. Therefore, it can be assumed that their sleep disturbance exists all year round.

DALYs for sleep disturbance were calculated using the road traffic noise exposure distribution in  $L_{\text{night}}$  as assessed in the Netherlands in 2000, the total population of the Netherlands in 2000 (15 864 000), the exposure–response relationships presented above for sleep disturbance due to road traffic noise (using the expected percentage of highly sleep-disturbed people at the midpoint of the category as a function of  $L_{\text{night}}$  in the range 45–65 dBA) and the Disability Weight (DW) of 0.089.

This calculation suggests that there are 24 669 DALYs lost in the Netherlands due to road traffic noise-induced sleep disturbance. Taking 0.04 and 0.10 as the extremes of the range for the weights, the credible range for the DALYs is from 14096 to 35242. This is a very conservative estimate, derived only for the exposure–response and

exposure data for road traffic noise and not including the impacts of aircraft and railway noise. However, although the impact at a given exposure level is expected to be higher for aircraft noise (but slightly lower for railway noise), far fewer people are exposed to aircraft (and railway) noise than to road traffic noise.

Conservative estimates applied to the calculation using exposure data from noise maps give a total of 900 000 DALYs lost from noise-induced sleep disturbance for the EU population living in towns of > 50 000 inhabitants.

Outcome based assessment: Uses survey data from the population to assess the relative contribution of various sources of environmental noise to overall self-reported sleep disturbance. This is measured on a scale of 1-10. The three highest points are considered to represent HSD. This approach allows individual sources to be counted more directly.

## 6.5 CE Delft Report

6.5.1 HACAN Clearskies commissioned CE Delft, an environment and consultancy agency based in the Netherlands to produce a report (published in January 2011) investigating the costs and benefits to the UK of a ban on night flights before 0600. The study used social cost benefit analysis to explore three possible outcomes of a ban:

- All flights and connections are rescheduled to daytime operations
- All flights are scheduled to daytime operations but connections are lost, leading to a decrease in the number of transfer passengers
- All flights currently arriving or departing during the night are cancelled

6.5.2 Social cost benefit analysis identified the direct, indirect and external effects of a night flight ban in monetary terms so that the net costs or benefits can be calculated. In this report the cost/benefits related to welfare effects. The methodology (to be explained in detail in the accompanying worked example report) utilises the correlation between 8 hour  $L_{\text{night}}$  noise exposure and the percentage Highly Sleep Disturbed (HSD) proposed by Miedema (2007). This self-reported sleep disturbance relationship was been assessed for aircraft, road traffic and railway noise by conducting a comprehensive analysis of the pooled original data from 24 studies containing 22771 cases for whom the night-time noise exposure and self-reported noise-induced sleep disturbance, are known.

6.5.3 The polynomial approximation for the percentage highly sleep disturbed (%HSD) is:

$$\text{HSD}(\%) = 18.147 - 0.956 \times L_{\text{night}} + 0.01482 \times L_{\text{night}}^2$$

6.5.4 Miedema explains that the above relationships can be applied in the range  $40 \leq L_{\text{night}} \leq 70$  dB(A). The relationships are based on data in the  $L_{\text{night}}$  range 45-65 dB(A) and are expected to give approximations also for lower exposures (40-45 dB(A)) and higher exposures (65-70 dB(A)). It should be noted that the author suggest that there is a need for improving the estimates of the functions that specify the self-reported sleep disturbance in relation to the night-time noise exposure for aircraft noise because the estimated individual variance was very high and the estimated study variance was not fully stable. The cause of this large individual variance is not understood.

- 6.5.5 The CE Delft report concluded that the impacts ranged from an increase of £860 million to a decrease of £35 million over a period of ten years (2013-2023). The loss would be as a result of all current night time passengers stopped travelling to Heathrow once a night flight ban was introduced. The benefit is explained in terms of the lack of noise-induced sleep disturbance that impacts welfare in the UK.
- 6.5.6 The analysis used the relationship between  $L_{\text{night}}$  contours and the odds ratio for hypertension, and relates this to DALYs to obtain a monetary value estimate of the health impact. This is an interesting approach to use, however it should be noted that the authors have compared the benefits of the night flights in the night quota period (2330 – 0600), which equates to 16 flights, with the disbenefits of the whole night period (2300 – 0700). In summer 2009, the average shoulder hour flights per night were 17 (2300-2300) and 52 (0600-0700), so there is a discrepancy of 69 flights per night by only considering the benefit of the flights in the quota period night. The summer 2009 average  $L_{\text{night}}$  traffic was 82 flights per 2300-0700 night. This considerable difference is an important detail and may invalidate some of the findings in the report.
- 6.5.7 Further examination of the methodology is required, with the possibility of replicating the analysis to obtain revised figures with respect to equal time period comparisons. It is considered that the social cost benefit approach may be useful for further studies of this nature.

## 6.6 Interdepartmental Group on Cost and Benefits of Noise (IGCB(N))

- 6.6.1 The Interdepartmental Group on Cost Benefits of Noise (IGCB (N)) have produced two reports on the valuation of noise impacts. The first, in 2008 examined the impact pathway as a central methodology for assessing noise, linking between the identification of the noise source, modelling and dispersion of noise and then the quantification and monetisation of the impacts. This report identified four groups of noise impacts, namely, health, amenity (annoyance), productivity and ecosystems. Health effects were deemed to be the most urgent area for further research, with the growth in the literature concerning noise and health effects contributing largely to this decision, along with the estimated costs of noise-induced health effects to be in the region of 2-3 billion pounds per year. It was shortly after this report that the IGCB(N) commissioned Bernard Berry and Ian Flindell to conduct an investigation into the links between noise and health. This report (Berry and Flindell, 2009) referred to in section 4 and 6 of this report, was then used by the IGCB(N) to investigate how the findings could be used for cost benefit appraisal methods.
- 6.6.2 IGCB(N) produced a second report in 2011, which attempted to value the human health impacts of environmental noise exposure. The main findings and recommendations included:
- Acute myocardial infarction (AMI) can be applied into monetary valuation of noise using the 2006 Babisch dose-response function. The IGCB(N) is recommending the use of the Babisch curve to assess the additional risk of AMI with rising noise levels and has generated a methodology which monetises this risk.
  - The use of the IGCB(N)'s indicative quantification of hypertension and sleep disturbance impacts to reflect the associated risks in these areas. Dose-response functions identified can be used for sensitivity analysis in policy appraisal, but evidence is not sufficiently developed to monetise these quantified effects. These impacts will instead be presented as the additional risk of incidences given marginal rises in environmental noise levels.

- Continued use of the Department for Transport's WebTAG monetary values for the amenity impacts of noise.
- Prioritising and monitoring policy-oriented research in areas where impacts are believed to be significant, but quantification not sufficiently developed to enable inclusion in the IGCB(N) methodology. Specifically, the IGCB(N) will monitor developments in monetising hypertension and sleep disturbance impacts, and reconciling confounding factors in dose-response functions such as air quality impacts and self-selections bias.

## 6.7 Health and Safety Laboratory Report

- 6.7.1 In 2011 Harding et al from the Health and Safety Laboratory published a report on quantifying the links between environmental related noise hypertension and health effects (referred to in Section 4.3 of this report). The aims were to identify the related health outcomes that follow on from hypertension, and to propose a methodology for valuing the links between environmental related hypertension and such health effects. The report focuses on three health outcomes resulting from hypertension; acute myocardial infarction (AMI), stroke and dementia. Calculation of risk was conducted by combining the risk of hypertension associated with environmental noise and the risk of each outcome associated with hypertension. The study investigated 23 urban agglomerations in England and a number of urban and non-urban agglomerations in Wales, representing 43% of the UK population.
- 6.7.2 The additional cases of AMI, stroke and dementia associated with environmental noise related hypertension in one year from road and railway noise levels  $L_{den} \geq 55$  dB(A) were estimated, and the Quality Adjusted Life Years (QALYs) were calculated accordingly (the value of one QALY being taken as £60,000). The QALYs lost to AMI, stroke and dementia due to road noise in the selected study areas were valued at £1,056 million (£286m for AMI, £310m for stroke and £460m for dementia) and for railway noise £43 million (£12m for AMI, £13m for stroke and £18m for dementia). This method, and the recommendations given by IGCB(N) can be used for calculating the associated hypertension and health effects from aircraft noise, which is explained in further detail in the report "Proposed methodology for Estimating the cost of sleep disturbance from aircraft noise".
- 6.7.3 Harding et al stress that the methodology is dependent on accurate values for the initial risk of hypertension due to environmental noise, and uncertainties in the literature and risk estimates may therefore affect the monetary valuation outcomes.

## 6.8 A US Perspective

- 6.8.1 Finegold (2010) highlights that in contrast to the WHO Night Noise Guidelines for Europe, there is no internationally agreed noise metric for estimating sleep disturbance. He notes that past research has highlighted that SEL is a better predictor than  $L_{Amax}$  for the number of awakenings, and was the approach taken by DfT (1998) to quantify the impact of night noise.
- 6.8.2 In order to monetise the loss of amenity resulting from aircraft noise-induced awakenings, a disability weighting is required. To date WHO has only recommended a disability weighting based on the %HSD derived from noise exposure calculated using  $L_{night}$ . Thus, whilst SEL is a much better predictor of sleep disturbance than  $L_{night}$  it is not possible to monetise the loss of amenity associated with noise induced awakenings.



## 6.9 Acute health effects

6.9.1 As noted in section 4, the WHO NNG (2009) considers that exposure above 55 dB  $L_{\text{night}}$  brings increased risk of myocardial infarctions:

- 55-60 dB  $L_{\text{night}}$  odds ratio: 1.1
- 60-65 dB  $L_{\text{night}}$  odds ratio: 1.2

6.9.2 Whilst there is still much debate, some, including the IGCB(N) consider this outcome should be monetised and added to that estimated from sleep disturbance, the rationale being that for moderate exposure levels sleep disturbance results in only temporary or short term effects, but at higher exposure levels the risk of acute health effects such as myocardial infarction exist.

6.9.3 Because myocardial infarctions are a well-established condition, baseline risk data is available. Secondly, there is an established disability weighting value for myocardial infarctions. Taken together, it is therefore possible to monetise the impact of additional myocardial infarctions due to noise exposure.

6.9.4 Whilst the second report of the IGCB(N) agreed that noise exposure increased the risk of hypertension, it concluded that, because of the number of health outcomes that can arise from hypertension, it was not possible monetise the effects of hypertension. However, a report by Harding et al (2011) prepared for the IGCB(N) concludes that exposure above 45 dB  $L_{\text{night}}$  brings increased risk of hypertension, and this leads to increased risk of hypertensive stroke and dementia that can be quantified and monetised. Whilst the IGCB(N) has not formally approved the findings it is likely to do so.

## 6.10 Conclusion

6.10.1 When trying to evaluate the potential cost benefit of aircraft night-time noise in terms of sleep and health effects, it is not possible to use the standard dose-response relationship methodology for all elements due to a lack of evidence of night time specific functions. There is, however, consensus that the percentage highly sleep disturbed dose-response function as proposed by Miedema (2007) and recommended by WHO may be used to monetise the effects of night-time sleep disturbance. It should, however, not be forgotten that this dose-response function is based on self-reported data which possesses a high degree of unexplained variance.

6.10.2 In terms of acute health effects, there is a lack of night-time specific dose-response functions, however, in some instances the WHO considers that the night-time specific risk is as great as the daytime or 24hr risk. What is clear is that is important that equivalent time periods are compared.

6.10.3 The Intergovernmental Group on Costs and Benefits of environmental noise, IGCB(N), has endorsed the WHO recommendations on monetising health impacts due to environmental noise and conducted their own peer review of the research using UK experts.

6.10.4 This review, like that of Berry and Flindell for Defra (2009) considers the work to have a sound methodological basis, and is conducted by credible researchers with strong track records in their fields. The IGCB(N) even comments on UK versus European research:

"It was felt that national policy appraisal development places unduly high weighting on domestic evidence at the expense of considering valuable external studies in formulating evaluation methodologies. The IGCB(N) does not exclude evidence from other geographic areas; however, it is true that additional weight may be given to national studies. The key reason for this is that as noise is subjective, national studies are seen to better reflect uniquely national characteristics of the domestic population's sensitivities and susceptibilities to noise. However, the IGCB(N) continues to monitor international evidence, and welcomes the formation of the European Network on Noise and Health (ENNAH), which will facilitate this work. At the present time, the IGCB(N) is considering how best it could contribute to this group."

- 6.10.5 Due to the nature of the methodologies involved in researching the impacts of environmental noise on health it is very difficult to eliminate confounding variables. For example when cross-sectional field studies are used to compare health effects in resident populations in different areas, there are inevitably other variables which could contribute to the observed effects. Similarly, although longitudinal studies that examine the existence of health effects in different population over time are statistically more powerful, the issue of confounding variables still remains. It is also difficult to provide theoretical hypotheses that are capable of explaining the biological pathways by which noise might contribute to adverse health effects and which would be capable of being tested in the field. An obvious example of a confounding variable is the presence of air pollution in conjunction with environmental noise. In terms of participants, the issue of self-selection bias would be considered a confounding variable, along with socio-economic status and lifestyle factors.
- 6.10.6 Confounding variables such as diet, lifestyle and genetic factors, mean that it is necessary to have large sample sizes in order to achieve the required statistical power. In some cases, existing noise exposed populations will not be large enough in total to provide sufficient statistical power for reliable detection of some of the weaker proposed relationships.
- 6.10.7 The causal pathways between noise and health outcomes are not yet fully understood and it can be difficult to establish definitive cause and effect. This can result in health outcomes being correlated with available noise indicators, e.g.  $L_{\text{day}}$ ,  $L_{\text{night}}$  and  $L_{\text{den}}$ , even though they may adequately represent the noise dose received.
- 6.10.8 Often, the relationships observed between the input and output variables can be statistically quite weak, even if significant relationships have been found. It is widely agreed within the research community that further work into noise and health is required, particularly in understanding the mechanisms by which long-term exposure to noise may influence health outcomes.
- 6.10.9 The IGCB(N) report and methodology has been a valuable development for the monetisation of health risks due to environmental noise. This coupled with the Health and Safety Laboratory report (Harding, 2011) allows for progress to be made in assessing the relative risks of primary and secondary health outcomes due to environmental noise. It is a combination of the above methodologies that enables the cost-benefit analysis of night flights to be evaluated. A full breakdown of this methodology can be found in the accompanying technical report "Proposed methodology for estimating the cost of sleep disturbance from aircraft noise".

## 7 Summary

- 7.1 This review has summarised the main findings from research into aircraft noise-induced sleep disturbance and health effects. The findings are not conclusive and are often contradictory, highlighting the practical difficulties in designing studies of this nature. It is often difficult to control for confounding variables such as individual sensitivity to noise, attitudes to aircraft noise, fear, habituation effects, age and gender. In order to obtain the sample sizes required, it is useful to use actigraphy as a means of measurement of sleep, combined with polysomnography where possible and cost allows. Laboratory studies into aircraft noise exposure provide a valuable contribution to the area, as they enable the real-time effects on sleep architecture to be measured, which are often not noticed by the sleeper, yet have follow-on implications for fatigue, daytime sleepiness, performance and mood.
- 7.2 Laboratory studies, however, often suffer from markedly increased effects, often attributed to a lack of habituation in unfamiliar surroundings. Field studies avoid this, but introduce other difficulties, such as noise intrusion from other sources.
- 7.3 Notwithstanding these issues, WHO considers that the onset of the effects of noise on sleep occurs at an aircraft noise event level of 32 dB  $L_{Amax,indoors}$ .
- 7.4 The work on cardiovascular and hormonal changes that occur during sleep as a result of noise highlight the importance for further work into the area, due to the potential for long-term health effects.
- 7.5 It would be useful to investigate these effects in larger sample sizes, perhaps alongside other health measures. A long-term study of sleep disturbance in a large sample of subjects, in various sites exposed to aircraft noise would be valuable so the effects over time could be compared within groups. Ideally, it would include subjective data, polysomnography in a selection of subjects at regular intervals, actigraphy, and a regular measurement of stress hormones, although it is appreciated that this is likely to be expensive and would require considerable planning in order to achieve meaningful results.
- 7.6 Analysis of the economic cost-benefits of night flights is limited to self-reported sleep disturbance and the increased risk of myocardial infarctions. WHO recommends that the percentage highly sleep disturbed is used, along with the disability weighting recommended by WHO in order to monetise the effects of sleep disturbance. The WHO proposes odds ratios for noise-induced myocardial infarctions can be used to estimate the number of additional myocardial infarctions and these can be monetised using established disability weightings from the health sector.

## Key findings

7.7 In conclusion, the following key findings must be considered when taking into account cost-benefit analysis of night flights.

- The WHO recommends an interim limit of 55dB  $L_{\text{night}}$  for the protection of residents against significant noise-induced adverse health effects.
- Percentage highly sleep disturbed (%HSD) can be used to monetise sleep disturbance based on night-time exposure,  $L_{\text{night}}$ . This measure is taken from self-reported estimates of sleep disturbance.
- Levels above 55 dB  $L_{\text{night}}$  result in increased risk of myocardial infarctions and these can be monetised using established methods.
- Levels above 45 dB  $L_{\text{night}}$  result in increased risk of hypertension, and this can lead to hypertensive strokes and dementia, which can be monetised using established methods.

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