Summary

The review covers the main findings of research into the effects of aircraft noise exposure on sleep, and includes the effects on electroencephalogram (EEG) and changes in sleep architecture, and their implications for sleep quality, mood and performance. Field and laboratory studies are compared, and the use of actigraphy versus polysomnography as a means of measuring sleep disturbance in large populations around airports is discussed. The physiological implications of noise-induced sleep disturbance are examined, including the main stress hormone concentrations, heart rate and cardiovascular responses to noise during sleep.

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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.

$L_A$  The A-weighted sound level (in dBA).

$L_{A_{max}}$  The maximum A-weighted sound level (in dBA) measured during an aircraft fly-by.

$L_{eq}$  Equivalent sound level of aircraft noise, often called equivalent continuous sound level. $L_{eq}$ is most often measured on the A-weighted scale, giving the abbreviation $L_{Aeq}$.

$L_{night}$  Equivalent sound level of aircraft noise in dBA for the 8-hour annual night (2300-0700).

$L_{den}$  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 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 altitude waves on the EEG and comprised of Stages 3 and 4 sleep.

Trophotopic  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
Intentionally Blank
1 Introduction

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.

1.2 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, to provide an overview of the area, past and current undertakings and potential ideas for future work in this domain.

1.3 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.


2.1 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”

2.2 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.

2.3 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.

2.4 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.

2.5 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.

2.6 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.
2.7 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.

2.8 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.

2.9 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 $L_{eq}$ '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 $L_{eq}$ levels. For example, the proportion of people who claimed to wake more than once a week increased from 30% for $L_{eq}$ of around 40 dBA, to 40% at the noisiest sites with $L_{eq}$ values of about 65 dBA.

d) The disturbance attributed by respondents to aircraft noise increased more substantially as $L_{eq}$ 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 dBA $L_{eq}$) gave
aircraft noise as a main cause compared with a tenth at the sites with least aircraft noise (40 Leq).

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.

2.10 The CAA/DORA study looked at subjective sleep disturbance with respect to aircraft noise, but valuable contributions into the effects of traffic noise on sleep changes that could also be applied to aircraft noise were also being made at this time. 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, 1882). The findings indicated that young people show decreases mainly in stages 3 and 4, and REM sleep deficits are seen in older people. In terms of cardiac responses, both peak levels and average were important, with threshold levels of 37 dBA Leq and 45 dBA Leq at which a decrement in sleep quality is observed.

2.11 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.

2.12 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.

2.13 This section has summarised the main contributions to the effect of aircraft noise-induced sleep disturbance understanding prior to 1990. The following sections of the report will group the findings of work since 1990 into two main areas of noise effects on sleep:

i. Disturbance to include changes in sleep architecture and structure as measured by EEG recordings, subjective ratings of sleep quality and awakenings, and ensuing effects on mood, alertness and performance following exposure to aircraft noise.

ii. Noise-induced cardiovascular and biochemical effects during sleep, including heart rate, and stress hormones.
3. The effects of aircraft noise on sleep structure, alertness, mood and performance

3.1 Sleep measurement

3.1.1 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.

3.1.2 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.

3.1.3 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.
3.2 Field Studies into Aircraft Noise and Sleep

3.2.1 In 1992 the findings of a study into aircraft noise and sleep disturbance, commissioned by the Department for Transport from the Department of Safety, Environment and Engineering at the CAA, were published (Ollerhead et al., 1992). The objectives of the study were to determine:

a) The relationships between outdoor aircraft noise levels and the probability of sleep disturbance.

b) The variation of these relationships with time of night

3.2.2 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.

3.2.3 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.

3.2.4 The main study used 8 sites; two around Heathrow, Gatwick, Stansted and Manchester Airports, and were selected for a range of SEL, $L_{eq}$ 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.

3.2.5 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.

3.2.6 The results suggested that below outdoor event levels of 90 dBA SEL (about 80 dBA $L_{max}$), 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.

3.2.7 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.

3.2.8 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.

3.2.9 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. $L_{max} > 80$ dB, outdoors) was still very low on average. 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.

3.2.10 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 responsivity 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 \( (r = -0.57; \text{df} = 17, p < 0.01) \), 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 \( (r = -0.67, \text{df} = 23, p < 0.01) \), with surges in SWS coinciding with troughs in spontaneous movement.

3.2.11 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 \( (n = 1600) \) 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.

3.2.12 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.

3.2.13 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 compared to at 74-79 PNdB. 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.

3.2.14 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).

3.2.15 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.

3.2.16 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

3.2.17 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.

3.2.18 The mean indoor SEL for awakening was 81 dB, and mean SEL that failed to awaken was 74 dB. 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.

3.2.19 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 (BA) increased 0.25% per 1 dB increase in indoor SEL. For each increase of 1 dB in ambient \( L_{\text{eq}} \) levels, the actimetric and BA 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 BA was found, despite a large increase in indoor noise events. Prior = 1.71, after = 1.13. After closing of the DEN airport, BA 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 dB.

3.2.20 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 $L_{\text{max}}$ 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 BA. Even at high noise levels most people were not awakened by aircraft overflights.

3.2.21 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 at 5 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.

3.2.22 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.

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 $L_{\text{max}}$ 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.

**24-h 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

3.2.23 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.
3.2.24 **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.

3.2.25 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.

3.2.26 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.

3.2.27 Recently, 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. Reanalysis 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. Outdoor \( L_{\text{night}} \) was used in this analysis and 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 2). 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.
Figure 2: 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.
3.3 Polysomnographic studies

3.3.1 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 activity (8-12Hz) can mean that the quality of sleep is compromised; despite subjects being unaware that this is occurring.

3.3.2 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.

3.3.3 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.

3.3.4 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.

3.3.5 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.

3.3.6 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.

3.3.7 Raschke (2004) explains that the type of noise, frequency spectrum, information consent, 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 lucus 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.

3.3.8 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.

3.3.9 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.
3.3.10 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.

3.3.11 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).

3.3.12 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.

3.3.13 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.

3.3.14 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.

3.3.15 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.

3.3.16 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.

3.3.17 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, where
the beginning of noise-induced awakenings is to be expected. 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 $L_{eq}$ level of 36 dBA, becoming particularly noticeable at
$L_{eq}$ 50-56 dBA. These results were taken from a sample size of $n=40$ over 5 nights,
but with no control group. A sub-sample of 8 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.

3.3.18 The DLR study used a double blind crossover design. ANEs with differing
distributions of $L_{A,s, \ max}$ 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.

3.3.19 Nights one and two were familiarization and baseline nights, then subjects were
exposed to 9 nights of aircraft noise with a varying distribution of $L_{A,s, \ max}$ 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 dB 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 $L_{eq}$ between 31.2 and 52.6 dBA. The last two nights of the
study were kept free of aircraft noise for comparative purposes.

3.3.20 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.

3.3.21 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.

3.3.22 The percentage probability of awakening increased with $L_{A,s, \ max}$ when the number of
events was kept constant at 32 (2000 noise events were analysed in total). For a
constant $L_{A,s, \ max}$ 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.

3.3.23 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.

3.3.24 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 3) 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 3:** taken from Spreng (2002) and shows the relation between the indoor maximum levels Lmax and the number of tolerable noise events within an 8-hour period during the night.

3.3.25 Based on this model, evaluation limits were derived for intermittent noise as shown in Table 1 (Griefahn et al 2002) 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).
3.3.26 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).

3.3.27 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.

3.3.28 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.

3.3.29 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

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### Table 1: Evaluation limits derived for intermittent noise, taken from Griefahn et al (2002)

<table>
<thead>
<tr>
<th>Maximum level</th>
<th>Equivalent noise level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Critical Limit:</td>
<td></td>
</tr>
<tr>
<td>$L_{\text{max}, 22-6 , \text{h}} = 6 \times 60 , \text{dBA}$</td>
<td>$L_{\text{eq}, 22-6 , \text{h}} = 40 , \text{dBA}$</td>
</tr>
<tr>
<td>Protection Guide:</td>
<td></td>
</tr>
<tr>
<td>$L_{\text{max}, 22-1 , \text{h}} = 8 \times 56 , \text{dBA}$</td>
<td>$L_{\text{eq}, 22-1 , \text{h}} = 35 , \text{dBA}$</td>
</tr>
<tr>
<td>$L_{\text{max}, 1-6 , \text{h}} = 5 \times 53 , \text{dBA}$</td>
<td>$L_{\text{eq}, 1-6 , \text{h}} = 32 , \text{dBA}$</td>
</tr>
<tr>
<td>Threshold Value:</td>
<td></td>
</tr>
<tr>
<td>$L_{\text{max}, 22-6 , \text{h}} = 23 \times 40 , \text{dBA}$</td>
<td>$L_{\text{eq}, 22-6 , \text{h}} = 30 , \text{dBA}$</td>
</tr>
</tbody>
</table>

* Levels must not be exceeded.
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 (Leq) of 39, 44, and 50 dBA and maximum values (Lmax) varied between 50 and 74 dBA.

3.3.30 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.

3.3.31 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.

3.3.32 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.

3.3.33 The Sound Pressure Levels (SPLs) 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.

3.3.34 Awakenings increased with the max SPL of an ANE. Awakenings induced by ANEs larger than 65 dB were relatively short. Those awakenings induced by ANEs larger than 70 dB were longer than spontaneous awakenings, and those below 65 dB.

3.3.35 The authors concluded that there should be on average less than 1 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.

3.3.36 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.

3.3.37 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.

3.3.38 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.
3.3.39 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.

3.3.40 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.

3.3.41 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.

3.3.42 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 sound pressure levels 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.

3.3.43 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.

3.3.44 Miedema suggests that the model (Figure 4) involves four routes through which noise exerts its primary influence.
3.3.45 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 4m 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.

3.3.46 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.

3.3.47 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-Vermee, 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

Figure 4: Taken from Miedema (2007) illustrating the four pathways through which the effects of noise are mediated.
problems that also appear to be mediated by increased arousal, such as social stress, medical stress, circadian stress and other environmental factors.

3.3.48 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.

3.3.49 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.

3.3.50 In the recent review on environmental noise, sleep and health Muzet (2007) explains the auditory and non-auditory effects of noise (Figure 5). Sleep disturbance is a non-auditory effect of noise. The input to the auditory area of the brain though 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, nose 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.

3.3.51 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.
3.3.52 Longer sleep latency and premature final awakening can reduce TST. Reports suggest that intermittent noises with peak 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.

3.3.53 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.

3.3.54 Sleep stage modifications
Nocturnal awakenings can be observed for an indoor $L_{\text{max}}$ of 55 dBA and above, and disturbance of normal sleep can be observed for peak 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: $L_{\text{night}}$) 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.
3.3.55 **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.

3.3.56 **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.

3.3.57 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.

3.3.58 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.

3.3.59 **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.

3.3.60 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.

3.3.61 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.

3.3.62 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.
4. Cardiovascular and biochemical effects of noise exposure during sleep

4.01 This section looks at the effects of noise exposure on hormonal and cardiovascular parameters during sleep.

4.02 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 dBA, motorcycles at 52.7 dBA, trains at 68.2 dBA and telephones at 62 dBA all L eq, with a maximum intensity of the aircraft noise at 86 dBA L eq occurred in the morning and afternoon. Heart rate and finger-pulse responses were compared to sensitivity, gender and time of day.

4.03 Heart rate responses showed differences between the sensitivity groups, but not type of noise, whereas the opposite was found for finger-pulse results, with no significant difference in sensitivity but clear differences between noises.

4.04 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.

4.05 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.

4.06 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.

4.07 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.

4.08 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.

4.09 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 response), but there was no interaction between the two factors. The
probability of an alpha response decreased from stages 1-4 and in REM was similar to in stage 2. Alpha 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 VPC’s 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.

4.10 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 corticol excitations, and cortisol excretion in relation to an estimation of tolerable nightly over-flights.

4.11 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 metabolisation of cortisol is 64 minutes, compared to adrenaline seconds to 3 minutes, and noradrenaline 7 to 12 minutes.

4.12 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 peak sounds 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 peak level, or 5 events with 75 dBA indoor peak level respectively.

4.13 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 dBA 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 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.

4.14 Often, the argument in 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.

4.15 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.
4.16 Persson Waye 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.

4.17 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.

4.18 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.

4.19 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.

4.20 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.

4.21 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 SPLs of between 45 and 80 dBA. 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.
4.22 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.

5. Summary

5.01 This review has summarised the main findings from research into aircraft noise-induced sleep disturbance. 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.

5.02 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.

5.03 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.
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