

Medical Research Council

Institute for Environment and Health

IEH report on

THE NON-AUDITORY EFFECTS OF
NOISE

1997

REPORT R10

The Institute for Environment and Health was established by the Medical Research Council at the University of Leicester in 1993. The Institute is partly funded by the Department of the Environment, Transport and the Regions, the Department of Health and other Government Departments and Agencies by way of specific research and consultancy contracts.

This report incorporates the output of a workshop held in May 1997, and has been prepared by the Institute for Environment and Health under a Department of Health contract. The workshop was held in collaboration with the Department of the Environment, Transport and the Regions. The Institute gratefully acknowledges the contribution of all those who attended the workshop and provided material for inclusion in the Report, but assumes no endorsement from these scientists for the conclusions and recommendations contained here.

The views expressed here do not necessarily represent those of any Government Department or Agency.

Published by the Institute for Environment and Health, 1997

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Printed by Page Bros, Norwich

ISBN 1 899110 14 3

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Definitions

dB	decibel: unit of sound level, or relative sound level, calculated as ten times the log (base 10) of a sound energy ratio
dB(A)	levels on a decibel scale of noise measured using a frequency dependent weighting which approximates the characteristics of human hearing. Referred to as A-weighted sound levels, these are widely used for noise assessment purposes.
L_{Aeq}	a measure of long-term average noise level expressed in dB(A). It is the level of a steady sound which, if heard continuously over the same period of time, would contain the same total sound energy as the actual varying sound. L_{Aeq} and L_{eq} are usually used interchangeably.

Executive summary

It has long been recognised that, as well as having detrimental effects on hearing, noise may be a cause of more subtle non-auditory health effects. Environmental noise, caused by traffic and industrial and recreational activities, is one of Europe's main local environmental problems.

Despite general agreement that environmental noise can be a source of annoyance, there is little consensus about the levels of noise responsible for other effects on health and well-being. The possible causal associations between adverse health effects and exposure to noise from environmental sources need to be rigorously investigated and well understood in order to facilitate the establishment of the best possible noise control policies, while at the same time maintaining an appropriate balance between benefit and cost.

This report is based on review papers prepared for a workshop on the non-auditory effects of noise, hosted by the Institute for Environment and Health on behalf of the Department of Health and the Department of the Environment, Transport and the Regions, in May 1997. It also includes a summary of the discussions at the workshop on the evidence for the non-auditory effects of exposure to noise from environmental sources, and recommendations for future research.

While a number of uncertainties remain about the non-auditory effects of noise, some conclusions have nonetheless been reached. The evidence for effects of environmental noise on health is strongest for annoyance, sleep disturbance (onset, latency, awakening during the night and premature awakening in the morning, subjective sleep quality, mood next day), ischaemic heart disease and performance by school children. The available data on other possible health consequences, such as low birthweight and psychiatric disorders, are inconclusive.

Further research to clarify the effects of environmental noise on non-auditory health should include both better characterisation of noise exposure and source, and better measurement of health outcomes.

EXECUTIVE SUMMARY

Types of studies to be encouraged include longitudinal studies, concentrating particularly on susceptible groups. Studies of ‘natural experiments’ (such as changes in the siting of an airport) are likely to be particularly informative, as are intervention studies. Further investigations of chronic health effects and the identification of particularly sensitive individuals are also important. In epidemiological studies, careful consideration needs to be given to confounding factors and effect modifiers in the association between noise and health. Emphasis should be given to studies combining other environmental stressors with noise.

In general, field studies are expected to be more informative than laboratory studies, although laboratory studies will be necessary to clarify specific causal hypotheses and to define exposure–response relationships. Field studies are likely to be expensive, though studies on children in school can be particularly cost-effective.

1 General introduction

Environmental noise, caused by traffic and industrial and recreational activities, is one of the main local environmental problems in Europe and the source of an increasing number of complaints from the public (Commission of the European Communities, 1996). The European Commission has estimated that “...close on 80 million people across Europe suffer from noise levels that scientists and health experts consider to be unacceptable, where most people become annoyed, where sleep is disturbed and where adverse health effects are to be feared”.

Most of this noise is generated by people travelling to and from work, by the transport of goods from place to place, and by the legitimate use of domestic appliances and leisure equipment. Considerable progress has been made over the last 30 years in noise technology, but surveys have shown that average noise exposure over the UK as a whole has hardly changed (Flindell, 1997). This is largely because traffic volume has tended to increase broadly in line with the reductions in noise levels generated by individual vehicles and because people are possibly becoming less considerate about the effects of household noise on their neighbours.

It seems unlikely that anything other than small reductions in the noise generated by individual vehicles will be achieved in the foreseeable future; it therefore follows that to achieve significant reductions in the numbers of people currently exposed to levels of noise deemed to be unacceptably high, the amounts of noise-generating activity will have to be curtailed. Any such constraints will have to be carefully balanced, in terms of their effects on the public, against the likely benefits to be achieved, and further information is required to allow these benefits to be precisely quantified.

There is a general consensus on the levels of noise which cause annoyance, but there is little agreement on those levels that contribute to other non-auditory health effects. In view of this, the Department of Health commissioned Dr Stephen Stansfeld, from the Department of Epidemiology and Public Health at University College London Medical School, to prepare a review of the published literature on the non-auditory effects of noise, and the Department of the Environment, Transport and the Regions and the Department of Health jointly

INTRODUCTION

requested the Institute for Environment and Health to convene a workshop of international experts to evaluate the current state of knowledge. A steering group was established to develop the programme for the workshop and to identify the experts to be invited. The workshop was held in Leicester, UK on 15 and 16 May 1997; the list of participants is given at the end of the report.

As well as the extensive critical review of the literature by Stephen Stansfeld, which is reproduced in Section 2 of this report, other invited experts were asked to prepare brief summary reviews on specific health effects that have been reported to be associated with environmental noise. These are presented in Section 3 of this report and were used as background papers to facilitate the discussions at the workshop.

The aims of the of the workshop were:

- to assess the strength of the evidence for any causal association between adverse health effects (other than auditory effects) and exposure to noise from environmental sources; and
- to identify gaps in knowledge and make recommendations for future research, taking into account the balance between the likely benefit and the research effort that will be required.

The assessment of the evidence for non-auditory adverse health effects arising from exposure to noise from environmental sources is presented in Section 4. This has been used to draw up recommendations for future research as outlined in Section 5. The overall conclusions are presented in Section 6.

It is hoped that this report will aid Government departments and other agencies to make an informed response to national and international concerns about the health issues and public perception of harm arising from environmental noise, as well as the need for research in this area.

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2 Environmental noise and health: A review of non- auditory effects

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* We gratefully acknowledge the considerable help provided by Louise Price in preparing this section

2.1 OVERVIEW

BACKGROUND

One of the ubiquitous features of urban and, increasingly, rural environments is exposure to environmental noise. Noise, defined as ‘unwanted sound’, usually predominantly aircraft, road traffic, and neighbours’ noise, is a type of stressor which might be expected to have a deleterious effect on health. It is generally suggested that noise will cause disturbances of activities and communication as well as annoyance and that these will lead to stress responses, and hence to symptoms and, possibly, overt illness (Van Dijk *et al.*, 1987).

Non-auditory effects of noise can be defined as ‘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)’ (Smith & Broadbent, 1992). Such effects include performance effects, physiological responses and health outcomes, annoyance and sleep disturbance.

The response to noise may partly depend on various characteristics of sound. These include intensity, frequency, complexity of sound, duration (whether intermittent or continuous) and the meaning of the noise. Studies of health effects of noise differ in how much these aspects of noise exposure are measured, perhaps because they often rely on long-term or retrospective evaluation of noise. This increases the difficulty of comparison between studies.

This section describes and critically evaluates, in turn, the research literature on noise exposure and sleep, performance (including memory and social behaviour), cardiovascular disease, the fetus, endocrine responses, psychiatric disorder, annoyance, vulnerable groups such as children, and combined effects of noise and other stressors on health. Putative mechanisms for noise effects are discussed and opportunities for further research are proposed where appropriate. Details are presented in subsequent sections (2.2 to 2.10).

SLEEP

Noise exposure during sleep may cause awakenings and change in sleep stages. Road traffic noise may also increase the time taken to fall asleep. Performance and mood may both be lowered following sleep disturbance by noise. There are differences in susceptibility to noise-induced sleep disturbance in the elderly and in noise sensitive subjects. Recent community studies suggest that there is little evidence of noise-induced sleep disturbance among residents exposed over the long term to aircraft noise below 82 dB(A). Further studies should examine, longitudinally, both the impact of changes in noise exposure on sleep disturbance in the community, including other factors likely to influence sleep, and performance following sleep disturbed by noise.

PERFORMANCE

There is evidence that noise impairs performance. In general, it seems that noise increases arousal and decreases attention through distraction and decreased focusing on stimuli peripheral to the task, as well as by altering choice of task strategy. Even relatively low levels of noise may have subtle ill effects, and in this respect, the state of the person at the time of performance may be as important as the noise itself. Individuals' perception of their degree of control over noise may also influence whether it impairs memory.

Noise may also affect social performance:

- ❑ as a stressor causing unwanted aversive changes in affective state;
- ❑ by masking speech and impairing communication; and
- ❑ by distracting attention from relevant cues in the immediate social environment.

It may be that people whose performance strategies are already limited for other reasons (e.g. through high anxiety), and who are faced with multiple tasks, may be more vulnerable to the masking and distracting effects of noise. Future research should continue to examine the effects of irrelevant speech on performance, to specify in more detail which work tasks are impaired by noise and to carry out longer-term studies of the effects of noise on performance in occupational settings.

CARDIOVASCULAR DISEASE

Acute physiological reactions to noise include an increase in heart rate and an increase in peripheral vasoconstriction and blood pressure; such responses to noise exposure habituate quickly. There is less consistent evidence on the impact of long-term exposure to noise, although studies on the effects of noise on heart rate during sleep suggest habituation does not occur. Laboratory studies of animals do suggest persistent elevation of blood pressure following noise exposure. Human laboratory studies have been less consistent. Most studies of occupational noise exposure and blood pressure find increases in blood pressure, although these are not always confirmed after adjustment for confounding in multivariate analyses. The findings across studies tend to be inconsistent. The strongest finding has been a dose–response relationship between noise exposure and hypertension in female Chinese textile mill employees, adjusting for age, years of work, salt intake, and family history of hypertension. Many studies do not fully adjust for potential confounding factors. In particular, other characteristics of the work environment, apart from noise exposure, are not measured well enough. Many studies are cross-sectional, with consequent difficulties in establishing both direction of causation and accurate estimation of noise exposure dose. The odds ratio (OR) for prevalence of hypertension varies from 1.03–1.77.

Noise annoyance is related to increased systolic and diastolic blood pressure after adjustment for age, body mass index, sex, education, smoking and occupational risk factors. It is not clear whether noise annoyance is just a proxy for noise exposure, or whether it is also related to other factors, such as socioeconomic status, which may be independent risk factors for high blood pressure. Recent community studies do not show convincing relationships between road traffic noise exposure and risk factors for coronary heart disease.

In the laboratory, short-term exposure to noise increases heart rate. Studies in occupational settings have been inconsistent, some finding higher baseline heart rate and others a higher mean resting heart rate only after exposure to noise at work. Low altitude military flights do not precipitate cardiac dysrhythmias or cause changes in heart rate. Future studies of noise and the cardiovascular system need to be longitudinal with careful measurement of length and intensity of noise exposure and more comprehensive adjustment for potential confounding factors.

THE FETUS

Preterm birth (less than 37 weeks pregnancy) has been significantly related to retrospective self-reporting of high occupational noise exposure. Birthweight was not related to noise exposure during pregnancy in 200 Taiwanese women, after adjustment for social class, smoking, alcohol use, maternal weight gain in pregnancy, and infant sex and gestational age. Future studies need to include better measures of noise exposure.

ENDOCRINE RESPONSES

The pattern of endocrine responses to noise is in keeping with noise exciting acute physiological responses, in the same way as other acute stressors. The most convincing evidence suggests that the adrenal medullary hormones, adrenaline and noradrenaline, are raised in working populations exposed to high levels of noise, though this has not been confirmed in all studies. Future studies need to examine endocrine responses to noise, longitudinally, and measure more potential confounding factors between noise and endocrine response.

PSYCHIATRIC DISORDER

The evidence that noise causes psychiatric disorder is equivocal. It causes some acute psychological symptoms, and is related to increased psychiatric hospital admission rates in some studies, but not in others. Exposure to aircraft noise was not related to increased rates of psychiatric disorder in a community study in West London. Small associations have been found between levels of road traffic noise and mental health symptoms and anxiety scores. Higher levels of aircraft noise have been associated with an increased tendency to take sleeping tablets in two studies. Although annoyance (see below) is related to psychiatric disorder, there is no evidence that noise leads to annoyance which leads to subsequent psychiatric disorder; rather both noise and psychiatric disorder have independent effects on increasing annoyance. Self-reported sensitivity to noise is a risk factor for psychiatric disorder. However, no interaction has been found between noise exposure and noise sensitivity in increasing vulnerability to psychiatric disorder. Further research should include longitudinal studies, measuring the effect of change in noise exposure on psychiatric disorder, and should include more comprehensive measures of psychiatric disorder.

ANNOYANCE

The most widespread and documented response to noise is annoyance, which may include fear and mild anger. In both traffic and aircraft noise studies, dose–response relationships have consistently been found between noise level and annoyance. Noise annoyance is often related to disturbances, by the noise of activities such as conversation, watching television or listening to the radio . There are both acoustic and non-acoustic predictors of noise annoyance. Important non-acoustic predictors of annoyance are noise sensitivity, fear of the noise source and attitudes to the noise source. A substantial amount of research has been carried out on annoyance.

NON-AUDITORY HEALTH EFFECTS IN CHILDREN

There is evidence that different groups within the population may differ in their susceptibility to the effects of noise. These groups include the elderly, people with existing physical or mental illness, and children. Most relevant work has been carried out among children. In children, aircraft noise exposure affects tasks which involve central processing and language comprehension, such as reading attention, problem solving and long-term memory. Other impairments relating to noise include deficits in sustained visual attention, difficulties in concentrating, poorer motivation, and poorer auditory discrimination and speech perception. Seven out of nine studies also report an elevation of resting blood pressure among children with long-term exposure to road traffic and aircraft noise. Additionally, raised levels of catecholamines have been found in children with long-term exposure to aircraft noise. Future research is needed to test proposed mechanisms of the effects of noise on cognitive impairment, and to examine factors that may moderate or confound the effect of noise on children’s health. Longitudinal studies will be needed to demonstrate whether noise effects persist over time.

COMBINED EFFECTS ON HEALTH OF NOISE EXPOSURE AND OTHER STRESSORS

Noise exposure may act together with other environmental stressors, either synergistically, antagonistically, or independently. Many studies have been carried out in this area but few have been able to measure well both noise exposure and other stressors. Because different populations have been studied in very different environments, the results have not been especially consistent. Combined effects of

noise and other stressors have been found that are of relevance to the issue of vulnerable groups. For example, having a cold infection interacted with exposure to noise to produce a reduction in simple reaction time. Future research should concentrate on field studies, with better stressor measurement and better measurement of confounding factors, to test hypotheses linked to specific health outcomes.

2.2 SLEEP

2.2.1 SLEEP DISTURBANCE

There is evidence, both objective and subjective, for sleep disturbance by noise (Öhrström, 1982; Öhrström *et al.*, 1988a; Lambert & Vallet, 1994). Although noise effects on sleep may habituate over time (Vallet & Francois, 1982), small sleep deficits may persist for years (Globus *et al.*, 1973), with unknown effects on health.

Mechanisms describing how noise exposure may affect sleep are manifold and not straightforward. Daytime exposure to noise preceding sleep may lead to a reduction in rapid eye movement (REM) sleep (Blois *et al.*, 1978). It has been argued that this implies an increased demand for central nervous system recovery during sleep and thus deep sleep is augmented. Exposure to noise during sleep has caused sleep disturbance proportional to the amount of noise experienced, in terms of an increased rate of changes in sleep stages and in number of awakenings. It is not clear whether this represents changes in the amount of sleep in each stage or is an effect of REM latency (Nakagawa, 1987).

Habituation occurs with an increased number of sound exposures per night and during several nights. The probability of awakening seems to increase with the number of noise stimuli in the night, but does seem to level off. Sleep awakenings can be measured either behaviourally or by the electroencephalogram (EEG). The behavioural measure of sleep disturbance can be assessed by the amount of body movement experienced during sleep. 'Large' body movements are associated with the number of awakenings. Öhrström (1989) showed there was no habituation during 14 nights of exposure to noise at maximum noise level exposure in the laboratory. However, there seemed to be some habituation to the effect of the

number of noise events. Both noise events and background noise level need to be taken into account in assessing potential for sleep disturbance, and the number of noise events exceeding a certain level is a useful measure. Objective sleep disturbance is likely to occur if there are more than 50 noise events per night with a maximum level of 50 dB(A) indoors or more. In fact there is a weak association between outdoor noise levels and sleep disturbance.

The Civil Aviation Authority study (1980) around Heathrow and Gatwick airports, suggested that the relative proportion of total sleep disturbance attributable to noise increases in noisy areas, but the level of total sleep disturbance is not increased. In effect, the work suggested a symptom-reporting or symptom-attribution effect rather than real noise effects. In an actigraphy study around four UK airports, sleep disturbance was studied in relation to a wide range of aircraft noise exposure over 15 consecutive nights (Horne *et al.*, 1994). Although there was a strong association between sleep EEGs and actigram-measured awakenings and self-reported sleep disturbance, no aircraft noise events were associated with awakenings detected by actigram, and the chance of sleep disturbance with aircraft noise exposure of <82 dB was insignificant. Although it is likely that the population studied was one which was adapted to aircraft noise exposure, this study is likely to be closer to real life than laboratory studies using subjects newly exposed to noise.

Road traffic noise at 50–60 dB(A) maximum increases the time taken to fall asleep. In particular, the number of noise events seems important (Öhrström & Rylander, 1990). The first third of the night is the time most vulnerable to sleep disturbance.

In laboratory studies, the combination of traffic noise and vibration was shown to be more disturbing, with reduced REM sleep and poorer rated sleep quality, than noise alone (Arnberg *et al.*, 1990).

Does noise disturbance differ across demographic groups of the population? The best evidence suggests that older people are more affected than younger people by noise during sleep (Eberhardt, 1982). Sleep disturbance by noise was reported exclusively by an institutionalised sample of elderly people (55–96 years) and not by an age-matched non-institutionalised control group (Middelkoop *et al.*, 1994). Children seem less susceptible to noise disturbance during sleep although they show a larger amplitude heart rate response to noise during sleep (Muzet & Eberhardt, 1980). Some studies suggest that women are more vulnerable than men to sleep disturbance by noise, but the results are not consistent. Studies on noise

abatement show that by reducing indoor noise level the amount of REM sleep and slow wave sleep can be increased (Vallet *et al.*, 1983). It does seem that although there may be some adaptation to sleep disturbance by noise, complete habituation does not occur; this is particularly so for heart rate during sleep.

As far as physiological effects are concerned, noise exposure during sleep may increase blood pressure, heart rate and finger pulse amplitude, as well as body movements (Muzet & Eberhardt, 1980). Not only may noise affect sleep but it may have after-effects during the day following disturbed sleep. In a community study of exposure to road traffic noise, perceived sleep quality, mood and performance (in terms of reaction time) were all decreased following sleep disturbed by road traffic noise (Öhrström, 1982). It was argued that this might have longer-term effects on psychosocial well-being. This could be a possible mechanism through which noise might affect mood.

What are the implications for chronic health effects resulting from sleep disturbance? Some studies have found that sleep disturbance may predict coronary heart disease. In a community study, people reporting noise-induced sleep disturbance had an increased risk of reporting angina pectoris (relative risk, RR = 1.86) and hypertension (RR = 2.32; Ising & Rebertisch, 1993). It is not clear from this study whether the sleep disturbance or the illness came first. This can only be examined further in a longitudinal study.

2.2.2 POSSIBLE MECHANISMS FOR NOISE EFFECTS ON SLEEP

Noise exposure during sleep is considered to increase awakening or to cause shifts from deeper to lighter sleep stages. Laboratory studies have shown more powerful effects than community studies, where some adaptation may have occurred. However, little habituation is shown in heart rate responses to noise during sleep, and protective mechanisms may be absent or less effective during sleep. If noise disturbs sleep then there may be after-effects such as lowered mood and diminished performance the next day. These effects are presumably secondary to lack of adequate sleep.

2.2.3 SCOPE FOR FURTHER RESEARCH

Research on noise and sleep disturbance should be combined with research on other health outcomes. There is scope for more assessment of effects on mood and performance after sleep. Ideally such studies should be carried out in the field and should incorporate the effect of changes in noise exposure, with several waves of data collection in the same subjects, involving both self-reported and physiological measures (e.g. heart rate), to assess whether adaptation to noise occurs and whether this differs for self-reported and physiological measures.

2.3 PERFORMANCE

2.3.1 EFFECTS ON PERFORMANCE

There is good evidence, largely from laboratory studies, that noise exposure does impair performance (Loeb, 1986). It has been shown in several studies that performance may be impaired if speech is played while a subject reads and remembers verbal material, although this effect is not found with non-speech noise (Salame & Baddeley, 1982). The effects of irrelevant speech are independent of the intensity and meaning of the speech. It has been suggested that the effect of speech is on memory rather than perception (Smith & Broadbent, 1992). The susceptibility of complex mental tasks to disruption by irrelevant speech suggests that reading, with its reliance on memory, may also be impaired. Jones (1990) has reviewed experiments on the effects of irrelevant speech on proof reading. In these studies it seems that the meaning of the speech was important, with meaningful speech being more disruptive than meaningless speech.

Some studies have examined the subjects' perceived control over noise and have found this and the predictability of noise to be important in determining effects and after-effects of noise exposure. Glass & Singer (1972) found that tasks performed during noise exposure were unimpaired, but tasks that were performed after the noise had been switched off were impaired. However, this impairment was reduced when subjects were given perceived control over the noise. Indeed, anticipation of a loud noise exposure, even in the absence of real exposure, may impair performance, and expectation of control counters this effect (Cohen & Spacapan, 1984). Willner and Neiva (1986) examined the effects of exposure to

brief uncontrollable noise on recall of information from memory. They found that uncontrollable loud noise increased the recall of negative trait words and suggested that noise induces a state similar to that found in depressed patients. However, other studies (Jones *et al.*, 1982) have suggested that perceived control may not be important in all experimental situations and that the previous task situation is most important in determining whether after-effects occur or not. Exerting control over a threatening stimulus may only reduce anxiety when control is easy to exercise. Indeed, the effort required to exercise control may result in physiological arousal similar to that which occurs when there is no assumption of control (Solomon *et al.*, 1980).

After-effects of noise exposure have also been studied following sleep; for instance, Wilkinson and Campbell (1984) reported that reducing noise exposure during sleep by the use of double glazing led to improved simple reaction time the next day.

Smith and Stansfeld (1986) compared self-reports of everyday errors (failures of attention, memory and action) given by subjects who lived in an area with a high level of aircraft noise with those in a similar group who lived in an area with a low level of aircraft noise. The high aircraft noise group reported a higher frequency of everyday errors, as did the noise-sensitive subjects. However, there was no interaction between noise sensitivity and the level of aircraft noise. Longer-term memory of work and mental arithmetic were both impaired in noise-sensitive subjects compared with less sensitive subjects, under noisy conditions (55 dB(A), 75 dB(A)), but not in quiet conditions (Belojevic *et al.*, 1992). There is some concern that there may be some confounding by neuroticism in these findings, and in fact studies of the effects of noise on cognitive tasks do suggest that neuroticism and anxiety are important determinants of individual differences in response to noise. For instance, Von Wright and Vauras (1980) showed that intermittent noise impaired retrieval for semantic memory among neurotic subjects, but had little effect on semantic memory of stable subjects.

Because arousal varies according to time of day, it has been suggested that the effects of noise may also vary with time of day; in practice this has not consistently been found (Smith, 1989). The possibility of an interaction between noise exposure and working at night has been examined in occupational studies. Effects on performance were found for both noise and night work, but these effects were independent and varied according to the nature of the task being performed (Smith & Broadbent, 1992).

2.3.2 MEMORY

There have been several studies suggesting that noise slows rehearsal in memory (Mohindra & Wilding, 1983). Overall, the results examining memory suggest that even moderate intensity noise may have an effect on verbal memory tasks. There is also evidence that noise may influence processes of selectivity in memory and attention. Hockey and Hamilton (1970) found that 80 dB noise impaired recall of task-irrelevant information, but improved recall of relevant information. However, the effects of selectivity have not consistently been found in experiments on noise. As Smith and Broadbent (1992) comment:

‘recent studies have shown that moderate intensity of noise does influence performance’, but ‘they also reveal the inadequacy of most theories that suggest that performance is shifted by noise in an invariant or mechanical fashion. Changes in the difficulty of the task, subject to prior experience and changes in other task parameters may abolish or even reverse certain effects. Many of the tasks affected by this level of noise have used verbal materials and this made it initially attractive to think in terms for an effect of noise on internal speech. However, verbal tasks often present subjects with several ways of doing a task and noise may change the relative efficiency of performing in one way rather than another.’

Thus, choice of strategy in performance in noisy conditions can act as a buffer against the effects of noise (Smith, 1985). Noise may tend to reinforce the use of a dominant strategy for task performance. It may influence allocation of resources to tasks according to priority, alter the efficiency of control processes, and make subjects more inflexible by impairing switching between cognitive strategies (Smith & Broadbent, 1992). It is interesting that voluntary effort may be used to compensate for potentially deleterious effects of noise on human performance. For instance, Tafalla *et al.* (1988) tested this in a study of the effects of noise on a complex mental arithmetic task. Reaction time for the task increased during exposure to noise, but only in low effort conditions. Noise had no effect on performance under higher effort conditions, but systolic and diastolic blood pressure did increase during the high effort conditions with noise exposure.

2.3.3 SOCIAL BEHAVIOUR

This is a very important area of noise research because many human activities involve social interaction which may relate not only to communication but also to health. Many of the effects of noise on social life reflect direct effects of noise on

communication. There is evidence that noise may reduce helping behaviour, increase aggression and reduce the processing of social cues seen as irrelevant to task performance.

Jones *et al.* (1981) have summarised the effects of noise on social performance. Firstly, it may act as a stressor, causing unwanted aversive changes in affective state; secondly, it may mask speech, interfere with communication and might logically lead to social isolation; and thirdly, noise may give rise to attentional changes away from social cues. Siegel and Steele (1980) found that noise affects the ability to assess and integrate information and leads to a choice of more extreme judgements. Evidently, in noisy conditions some involuntary choices are made and the range of possible task strategies is reduced.

Smith and Broadbent (1992) argue that the detrimental effects of noise on performance are unlikely to be due to auditory effects such as masking because the same noise exposure gives rise to detrimental effects in some conditions or experiments but not others, and after-effects of noise exposure are observed when subjects are being tested in quiet conditions.

2.3.4 SCOPE FOR FUTURE RESEARCH

Smith and Broadbent (1992) argue that the effects of noise are complicated, influenced by many different factors and are still only partially known. They say that 'noise has a definite effect on performance but that the precise nature of the effect depends on the type of noise and the task being performed'. They particularly argue for the importance of research on the effect of irrelevant speech on performance and on the selection of strategies to cope with performance in noisy conditions. There is also further scope for research on the combined effects of noise and other stressors, both in occupational and laboratory settings.

2.4 CARDIOVASCULAR DISEASE

2.4.1 PHYSIOLOGICAL RESPONSES TO NOISE EXPOSURE

Noise exposure causes a number of predictable acute physiological responses mediated through the autonomic nervous system. These have generally been measured as changes in blood pressure, heart rate, depth and rate of respiration, pupil size, skin conductance, muscle tension and endocrine outputs. Exposure to noise may cause physiological activation, including increase in heart rate, increase in blood pressure and peripheral vasoconstriction, and thus increased peripheral vascular resistance. The orienting reflex is the usual physiological response to noise (Sokolov, 1963) unless the noise is loud enough to produce a defence/startle reflex (Turpin & Siddle, 1983). The orienting reflex occurs in response to a stimulus that is either novel or indicates conflict or has learned significance. Defence/startle responses are evoked either by novel sounds or sounds which imply threat. There is rapid habituation to brief noise exposure, but habituation to prolonged noise, especially heart rate response to noise exposure during sleep, is less certain (Vallet *et al.*, 1983).

Studies of noise and cardiovascular disease have tended to concentrate on coronary heart disease, the most prevalent form of coronary disease, and have measured the most accessible indicators of cardiac function, such as blood pressure and heart rate. Raised blood pressure and heart rate may be the results of stressor exposure or indicators of increased predisposition to coronary heart disease.

2.4.2 LABORATORY STUDIES ON CARDIOVASCULAR DISEASE

Long-term exposure of animals, such as monkeys, to noise leads to persistent elevation of blood pressure (Petersen *et al.*, 1981). Animal studies have prompted researchers to examine the hypertensive effects of noise among human volunteers in laboratory studies. However, the results of these studies have not been consistent. Among policemen exposed to traffic noise (60 dB L_{eq}), the mean systolic and diastolic blood pressure rose in some individuals but fell in others (Ising, 1983). It may be that some groups are more vulnerable to raised blood pressure in noisy conditions, especially those with a predisposition to hypertension.

2.4.3 OCCUPATIONAL STUDIES ON CARDIOVASCULAR DISEASE

HIGH BLOOD PRESSURE

The strongest evidence for the effect of noise on the cardiovascular system comes from studies of blood pressure in occupational settings (Thompson, 1996). Nevertheless, positive effects have been demonstrated only in some of these studies. This is partly due to study design. Many studies have been cross-sectional, with inherent difficulties in establishing both direction of causation and accurate estimation of noise exposure. In order for noise to have a persistent effect in raising blood pressure it may have to be of a certain intensity and be present for a certain length of time. Many occupational studies have suggested that individuals exposed over the long term to continuous noise of 85 dB or more had higher blood pressures than those not so exposed (Parvizpoor, 1976; Zhao *et al.*, 1991; Lang *et al.*, 1992). In many of these studies noise exposure has also been an indicator of exposure to other factors, both physical and psychosocial, which are also associated with high blood pressure. Unless these other risk factors are controlled, spurious associations between noise and blood pressure may arise. Few studies have taken into account other potential confounding factors such as smoking and antihypertensive medication.

The major studies in this area are described below and summarised in Table 2.4.1.

Workers who had been exposed to noise for 20 years or more had a systolic blood pressure 16 mm higher and a diastolic blood pressure 7 mm higher than workers exposed for less than 10 years (Verbeek *et al.*, 1987). These results have been corrected for age, but not for body mass index, and it is possible that increased blood pressure was due to other adverse working conditions. Indeed, in a further study in a different population (Van Dijk *et al.*, 1987), these results were not confirmed.

In the cross-sectional study on blood pressure, carried out in Luebeck among 1046 men aged 30–69 years, traffic noise (measured by self-report questionnaire) was associated with a higher prevalence of hypertension (systolic blood pressure ≥ 160 mm, diastolic blood pressure ≥ 95 mm Hg) after stratification for age, body mass index, alcohol use (g/day), education, duration of residence and employment status (Herbold *et al.*, 1989). The overall OR for prevalence of hypertension was 1.32 (90% CI 1.03–1.69) and the ORs remained statistically significant after stratification for the above mentioned covariables separately, apart from body

Table 2.4.1 Occupational cross-sectional studies of noise exposure and blood pressure

Sample (n)	Noise intensity	Measures of hypertension	Hypertension risk factors controlled for	Findings	Reference
Community sample of men 30-69 y, Luebeck (1046)	Self reported road traffic noise	SBP \geq 160mm Hg DBP \geq 95 mm Hg	Age, BMI, alcohol consumption	Stratified results suggest noise relates to hypertension. Not confirmed in multivariate analysis	Herbold <i>et al.</i> , 1989
Israeli male industrial workers (191)	74-102 dB(A)		Age, involvement in physical work, smoking, BMI, hearing loss, use of hearing protectors	SBP, DBP raised in younger but not older workers	Green <i>et al.</i> , 1991
Female Chinese textile mill employees (1101)	75-104 dB(A)	SBP \geq 160mm Hg DBP \geq 95 mm Hg	Age, years of work, salt intake, family history of hypertension	Dose-response relationships in SBP and DBP	Zhao <i>et al.</i> , 1991
Parisian workers (7679)	\geq 85 dB(A)/8 hr day		Age, BMI, alcohol consumption, occupational category	SBP, DBP related to noise. Not confirmed in multivariate analysis	Lang <i>et al.</i> , 1992
Study of white South African miners (2197)		SBP \geq 140mm Hg DBP \geq 90mm Hg	Age, BMI	No noise effects on blood pressure	Hessel & Sluiss-Cremer, 1994
Workers in a metallurgical factory, Italy (8811)	\leq 80 dB (n = 8078) vs $>$ 80 dB (n = 733)	DBP \geq 95mm Hg	Age, BMI, duration of employment	Heart rate, DBP not different, SBP higher in noise	Fogari <i>et al.</i> , 1994
Blue collar workers from 21 Israeli industrial plants 60% response rate (3105)	80 dB(A)	Means only used	Age, smoking, coffee & cholesterol, industrial sector, physical work load	Noise exposure correlates with resting heart rate (significant in men) and DBP only in women. Intensity of noise exposure significantly associated with resting heart rate in women	Kristal-Boneh <i>et al.</i> , 1995

BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure

mass index. In logistic regression analysis, including age, body mass index, and alcohol consumption, the effect of traffic noise was no longer significant. Any effect of traffic noise on hypertension was quite small and may have been obscured by the imprecise measure of noise exposure and the lack of occupational noise exposure.

In a study of 769 Parisians, length of noise exposure (≥ 85 dB(A) for 8h/day; 432 subjects exposed) was statistically significantly related to both systolic and diastolic blood pressure (Lang *et al.*, 1992). However, after adjustment for age, body mass index, alcohol consumption and occupational category this effect was no longer statistically significant. In linear regression analysis, noise exposure (≥ 25 years) was significantly associated with both higher systolic and diastolic pressure, adjusting for age, body mass index and alcohol consumption. In this study, the selection of subjects was non-random, blood pressure was measured on a single occasion, and it was reported to be difficult to assess with accuracy the sound pressure levels to which a worker was exposed throughout 8 hours per day. It is reasonable to suppose that workers who are exposed to high noise may be different from non-exposed workers in terms of lifestyle characteristics and age, body mass index, alcohol intake, occupational characteristics and working conditions, such as job demands and shift work.

In a cross-sectional study of 8811 metallurgical workers in Italy, the groups exposed to high noise levels ($n = 733$; >80 dB) had the highest mean systolic blood pressure both in the overall sample and across four age groups (18–30, 31–40, 41–50, 51–60). The high exposure group also had a higher prevalence of hypertension (11.9% vs 7.5%; $p < 0.001$) which was significantly different across all ages and most pronounced in the older age groups (i.e. 51–60 years). However there were no differences in mean diastolic blood pressure or heart rate (Fogari *et al.*, 1994). The noise-related increase in prevalence of hypertension remained after stratification for body mass index. There were no significant differences in the distribution of family history of hypertension among the hypertensive subjects by noise. In a case-control comparison of 242 matched pairs of subjects, both systolic and diastolic blood pressure were raised in the high noise exposed group compared to the low (<80 dB(A)) noise exposed (16.1% vs 9.1%; $p = 0.0278$), and rate of elevation of blood pressure with age did not differ between the two groups. As Thompson (1996) notes ‘in these case-control data the OR for hypertension was 1.77, slightly higher than the overall prevalence ratio of 1.59 in the cross-sectional data’. The authors suggest that the better control of confounding factors is the reason for the significant mean difference in diastolic blood pressure observed in the case-control comparison but not in the unmatched cross-sectional analysis. The study has some drawbacks because smoking, temperature and the effect of other working

conditions were not controlled for. In a further analysis however, which examined predictors of hypertension, including age, total cholesterol, body mass index and work type, noise exposure still had a significant effect (Fogari *et al.*, 1995).

In the CORDIS Study, 2202 men and 904 women, from 21 industrial plants across Israel (involving six different sectors — metalwork, textile, light industry, electronics, foodstuffs, plywood), underwent physical examination and blood pressure measurement (Kristal-Boneh *et al.*, 1995). There was no association between noise and systolic blood pressure, and diastolic blood pressure was only related to noise in women. There was no evidence of a step-wise increase in blood pressure with increasing work-related noise exposure. No interactions with the source or type of noise (intermittent or continuous) were found. No significant effects were found for use of ear protection or for noise exposure at home. The authors attribute their lack of a noise effect on blood pressure partly to low noise levels (<95 dB(A)). They adjusted for more confounding factors than in many studies, but in fact the lack of blood pressure results was apparent before adjustment for confounding. There was no evidence that control or lack of control over the noise source influenced blood pressure or that noise at home influenced the association between work noise and blood pressure.

An extensive retrospective cohort study of 2197 South African miners (Hessel & Sluis-Cremer, 1994) found no associations between noise exposure and blood pressure; in addition, changes in noise level over time were not consistently related to changes in blood pressure. This study attempted to use routinely collected information from three yearly medical check-ups to assess the chronic effects of mixed noise exposure. Such an approach does have methodological problems. Blood pressure was not measured under standardised conditions and noise exposure was only indirectly measured by expert retrospective assessment. Smoking and job type were not taken into account and information from many of the original subjects could not be included because of missing data.

Measurement of ambulatory blood pressure in men in a single Israeli industrial plant yielded conflicting results (Green *et al.*, 1991). In 191 subjects exposed to noise levels ranging from 74–102 dB(A) (average 81 dB(A) in the lower noise group and 89 dB(A) in the higher noise group), systolic and diastolic blood pressure were raised in response to noise in the younger age group (25–44 years). This was reversed for systolic pressure in the older age group in which noise exposure had no effect on diastolic pressure despite adjustment for age, involvement in physical work, smoking, body mass index, hearing loss and use of ear protectors.

Matthews *et al.* (1987) showed that many stressful work conditions (e.g. 'few opportunities for promotion and participating in decisions at work, job insecurity, lack of social support at work and overall dissatisfaction with the job') were associated with elevated blood pressure. Smith and Broadbent (1992) suggest that this indicates that some of the effects of noise on blood pressure might reflect workers' general reaction to the working environment. Moreover, some studies suggest that there may be an interaction between mental load and noise exposure in causing effects on diastolic blood pressure.

Theorell (1990) demonstrated that noise at work was the only significant predictor of a rise in systolic blood pressure between home and work time after adjusting for age and demands/decision latitude at work. The effect of noise on systolic blood pressure elevation took place only in those with a family history of hypertension. However, the automatic method of blood pressure estimation did not include measurement of diastolic blood pressure, and the response rate for this section of the study was low.

One of the most convincing cross-sectional studies of noise exposure and hypertension was carried out on 1101 female workers in a Beijing textile mill (Zhao *et al.*, 1991). The sound pressure level in the workshops of the mill ranged from 75 dB(A) to 104 dB(A). All women had been employed in the mill for a least five years, all of them working in a single workshop for their entire working life. Prevalence of hypertension (systolic blood pressure ≥ 160 mm Hg or diastolic blood pressure ≥ 95 mm Hg or both) increased in a dose-dependent fashion from 5.1% in the group exposed to 75–80 dB(A) to 15.2% in the group exposed to 104 dB(A). In logistic regression analysis, sound pressure level still had a significant association with hypertension ($p < 0.047$) after adjusting for age, working years, salt intake and family history of hypertension; the adjusted OR for sound pressure level was 1.031. Standardised regression coefficients suggest that the effect of sound pressure level on hypertension is about half the magnitude of the effects of either family history of hypertension or high salt intake. 'Overall the logistic model estimates that a 30 dB(A) increase in sound pressure level roughly doubles the risk of hypertension, independent of the other risk factors measured.' Cumulative noise exposure did not predict increased risk of hypertension in this study, either because hypertension occurred during the five year noise exposure period before the start of the study or because of the difficulty in distinguishing the effects of age and years worked. This study is impressive because of the intensity and consistency of the noise exposure, the demonstration of a dose-response relationship and the adjustment for appropriate confounding factors (except body mass index).

HEARING LOSS AND BLOOD PRESSURE

One method of assessing the impact of occupational noise on blood pressure has been to use hearing loss attributed to occupational noise exposure as a proxy measure of noise exposure. In addition, some researchers have suggested that hearing loss may be a mediating step between noise and hypertension. Higher levels of systolic blood pressure and diastolic blood pressure have been associated with hearing loss in several studies (Johsson & Hansson, 1977; Manninen & Aro, 1979; Talbott *et al.*, 1985), largely in older workers. Talbott *et al.* (1985) carefully adjusted for potential confounding factors, although a follow-up study among retired workers from the same plant failed to show a relationship between hearing loss and blood pressure. There are, however, potential problems with this approach; in particular, hearing loss may be associated with elevated blood pressure and coronary heart disease independently of noise.

NOISE ANNOYANCE AND BLOOD PRESSURE

One possibility is that the effects of noise on blood pressure are mediated through an intermediate psychological response such as noise annoyance. Lercher *et al.* (1993) found that occupational noise annoyance, in a community based cross-sectional study, was linked with 2.1 (95% CI -3.0, 7.3) mm Hg increase in systolic blood pressure and 3.5 (95% CI 0.3, 7.4) mm Hg increase in diastolic blood pressure, after adjustment for age, body mass index, sex, education, smoking and other occupational risk factors. However, the combined effect of noise annoyance and low work satisfaction was twice as large on blood pressure (systolic blood pressure 7.5mm Hg (0, 15.0) diastolic blood pressure 6.3 mm Hg (0.6, 12.4)). There was a similarly large effect for the combination of night shift work and noise annoyance.

The interpretation of the results of this interesting study are not straightforward. It is not clear whether noise annoyance is a good proxy measure of occupational noise exposure or whether it is also a proxy for other factors, such as socioeconomic status (either via noise exposure or independently), which may have a powerful independent influence on blood pressure. The literature on noise, annoyance and mental ill-health suggests that, although annoyance levels and mental ill-health are strongly associated, and noise and annoyance are strongly associated, annoyance is not the mediating factor between noise and ill-health; it may be the same for blood pressure. The combination of work satisfaction and noise annoyance might amplify such a social class effect. In contrast, despite small

numbers and a response rate of 68%, these results have been adjusted for education and thus it is possible they are not confounded; further longitudinal studies are needed which include measurement of noise exposure.

2.4.4 COMMUNITY STUDIES ON CARDIOVASCULAR DISEASE

In a community survey of 6000 people, high aircraft noise exposure was found to be related to increased medical treatment for heart trouble and hypertension, more cardiovascular drug use and higher blood pressure (Knipschild, 1977). These results could not be explained by age, sex, smoking, height, weight or socioeconomic differences.

The results from the Caerphilly Study on road traffic noise are not very consistent in terms of the effect of noise on risk factors for coronary heart disease. There were statistically significant effects of noise on systolic but not diastolic blood pressure, total cholesterol, total triglyceride, blood viscosity, platelet count and glucose level (Babisch *et al.*, 1988; 1989).

In a case-control study of 693 men aged 31–70 years, high noise exposure was related to increased risk of myocardial infarction, with a population attributable risk of 33%, but the response rate in the study seemed low, reporting of noise exposure was subjective, and there was no adjustment for possible confounding factors like work characteristics (Ising *et al.*, 1996).

In summary, there is little evidence from community studies that environmental noise is related to hypertension, but there is some evidence that environmental noise may be a risk factor for coronary heart disease.

2.4.5 HEART RATE

In a laboratory study, subjects exposed to 75 dB(A) sound for 15 minutes exhibited higher heart rates after noise exposure than before (Parrot *et al.*, 1992). Heart rate responses to noise were greater in men than women but no greater effects were found in anxious subjects. In these experiments there was also brief peripheral vasodilation, not vasoconstriction as might be expected, in response to noise (Petiot *et al.*, 1992). However novel noise exposure in the laboratory is a somewhat artificial testing situation.

In an occupational setting, in males, only resting heart rate correlated positively and significantly with noise exposure, particularly above 80 dB(A); however, the effect was abolished by control for confounders. Although there was no difference in baseline heart rate, mean resting heart rate of noise exposed workers after approximately four hours exposure to noise was higher than in those not so exposed, suggesting an acute rather than a chronic effect of noise (Kristal-Boneh *et al.*, 1995).

A sudden intense exposure to noise may stimulate catecholamine secretion and precipitate cardiac dysrhythmias. However, both studies in coronary care units of the effect of speech noise and studies on the effects of noise from low altitude military flights on 68 cardiac patients on continuous cardiac monitoring failed to show changes in heart rate or the induction of ventricular extrasystoles (Brenner *et al.*, 1993).

2.4.6 POSSIBLE MECHANISMS FOR NOISE EFFECTS ON THE CARDIOVASCULAR SYSTEM

The effect of sound on the auditory system is transmitted to the reticular activating system and thus to the hypothalamus where further neuronal stimulation is activated and the hypothalamic pituitary-adrenal axis may be stimulated. In addition, Ising and colleagues (1985) suggest that noise may stimulate secretion of adrenal medullary hormones, including noradrenaline and adrenaline. They postulate that familiar noise may induce secretion of noradrenaline and unfamiliar noise, adrenaline. The effects of these two hormones are to raise peripheral resistance and increase blood pressure and heart rate. It is unclear, however, whether these effects, observed in laboratory conditions, will also occur during long-term noise exposure; also the extent of adaptation during long-term exposure is not known.

Jansen (1984) suggests that environmental factors may influence 'secondary' risk factors such as social status in the aetiology of coronary heart disease. Such secondary factors in turn influence 'primary' risk factors such as blood pressure, blood lipids and blood coagulation, through a mediating stage of emotional disturbance, such as discontent, anxiety and aggression. This model, quoted in Passchier-Vermeer (1993) is complex. Although both primary and secondary influences are risk factors for chronic heart disease, and there is good evidence for the potential emotional mediators also being risk factors for the disease, there is no conclusive evidence that the primary risk factors are linked to the secondary risk factors through these mediating emotional disturbances.

It has been suggested recently (Van Dijk, 1987; Lercher *et al.*, 1993) that noise effects on blood pressure may be mediated through noise annoyance. However, it may be that other factors associated with high blood pressure (e.g. social class, working conditions) are also independently associated with noise annoyance.

Noise exposure has been linked to hearing loss and high blood pressure. It has been postulated that hearing loss related to noise may be an index of noise effects on blood pressure. It is possible that noise induced hearing loss is a marker for noise exposure. It is also possible that hearing loss is related to changes in blood lipids or coagulation, which are also independently related to blood pressure (or make the ear more vulnerable to noise induced changes).

2.4.7 SCOPE FOR FURTHER RESEARCH

Many studies on noise and blood pressure have now been carried out in adult working populations. It is not clear whether more studies will yield any further useful information. However, there is room for improvement in the methodology of many of these studies. Many are cross-sectional and there is scope for longitudinal studies with more emphasis on better measurement of noise exposure, with the opportunity for assessing dose–response effects, and careful measurement of blood pressure and other cardiac risk factors (e.g. fibrinogen, lipids). In addition, there needs to be an assessment of other stressors at work (in particular, working conditions, decision latitude, job demands, social support, effort–reward imbalance) and thorough measurement of potential confounding factors, including age, sex, body mass index, smoking, alcohol use and family history of hypertension. One way to refine further studies would be to concentrate on a potentially vulnerable group such as people with a family history of hypertension.

2.5 THE FETUS

In a case–control study of 210 American mothers and 1260 controls, preterm birth (<37 weeks pregnancy) was significantly related to self-reported high occupational noise exposure (Luke *et al.*, 1995). However, noise exposure does not seem to have been included in the final model, the response rate was low and the study was based wholly on retrospective reporting. In a study of 200 Taiwanese

women, noise exposure, measured by 24-hour personal dosimetry (52.4–86.8 dB(A) L_{eq}) on three occasions in pregnancy, was not predictive of infant birthweight (Wu *et al.*, 1996). Furthermore, occupational noise exposure, road traffic noise exposure and listening to amplified music during pregnancy were not related to birthweight. This study improved on earlier studies by adjusting for social class, smoking and alcohol use, maternal weight gain in pregnancy, infants' sex and gestational age. Passchier-Vermeer (1993) summarises the effects of ten studies examining the effect of noise on the unborn child and finds no conclusive evidence for low birthweight or congenital defects related to noise exposure.

Future studies need to develop better measures of noise exposure during pregnancy and to continue to control for factors which may confound the association between noise and low birthweight.

2.6 ENDOCRINE RESPONSES

2.6.1 HORMONE SECRETION

The most convincing evidence relating exposure to noise and increased hormone secretion concerns noradrenaline and adrenaline levels. Buczynski & Kedziora (1983) found that 'impulse noise' caused increases in noradrenaline in men during exercise while 'pulse noise' lead to an increase in secretion of adrenaline. Cavatorta *et al.* (1987) found that glass workers exposed to 96 dB(A) noise had raised noradrenaline levels and adrenaline levels, which were 70% higher than those in a control group working in the machine shop. They also measured cortisol but found no increase. It is not clear that the effects of other job characteristics were fully controlled in this study; this might also have contributed to the elevated hormone levels.

Urinary catecholamine levels were raised in 50 female workers exposed to machinery noise (93–100 dB(A)) compared with 25 female workers in quieter working environments (71–75 dB(A); Sudo *et al.*, 1996). Cortisol levels shared the same trend. On the second day of testing, when the noise exposed workers wore earplugs, their catecholamine excretion decreased and they reported less fatigue. However, these findings of increased catecholamines across studies have not necessarily been consistent. For example, Follenius *et al.* (1980) found no effect on

noradrenaline, adrenaline, dopamine, growth hormone or adrenocorticotrophic hormone, after exposure to intermittent noise between 45 and 99 dB(A). They did however find that noise exposure seemed to halt the circadian decline in cortisol measured in the urine between 10:00 and 12:00 in the morning. Other studies have also found a significant rise in cortisol in relation to noise (Brandenberger *et al.*, 1980) There have been few studies in children but Hygge (1993) found increased adrenaline and noradrenaline in children with long-term exposure to aircraft noise around Munich airport.

Other hormones, apart from catecholamines and the hypothalamic-pituitary-adrenal axis, have also been studied. Beardwood (1982) found that more than half a normal subject population exposed to monotone stimulation had increased gonadotropin output and suggested that this might result from a failure of habituation because of attention paid to the noise signal. There is a suggestion here that the meaning of the noise may be important in determining response.

The general pattern of endocrine responses to noise is commensurate with noise exciting acute physiological responses, in the same way as other acute stressors.

2.6.2 SCOPE FOR FURTHER RESEARCH

Further research on the endocrine responses to noise should be carried out, as for the assessment of sleep disturbance effects, in collaboration with assessment of other health effects. Assessment of endocrine responsivity to noise is appropriate in occupational studies of noise exposure and in studies assessing performance in noisy conditions. There needs to be a replication of the Munich studies of children showing that noradrenaline and adrenaline are raised in response to aircraft noise. These measurements also need to be repeated longitudinally to assess reliability.

In addition, a new area of noise effects on health should be considered, namely that of immunosuppressive effects of noise. There needs to be more laboratory research to determine suitable indices of immune function for field studies (Bly *et al.*, 1988).

2.7 PSYCHIATRIC DISORDER

The association between noise and mental health has been examined using a variety of outcomes, including (at the simplest level) individual symptoms, as well as psychiatric hospital admission rates, use of health services, and community surveys.

2.7.1 PSYCHOLOGICAL SYMPTOMS

Symptoms reported among industrial workers regularly exposed to high noise levels in settings such as weaving mills (Granati *et al.*, 1959), jet aircraft test beds (Bugard *et al.*, 1953), schools (Crook & Langdon, 1974), and factories (Melamed *et al.*, 1988) include nausea, headaches, argumentativeness, changes in mood, anxiety, and sexual impotence. More self-reported illness and illness-related absenteeism (Cameron *et al.*, 1972), social conflicts at work and home (Jansen, 1961), and actual absenteeism (Cohen, 1976) have been found in noisy rather than quiet industries. Many of these industrial studies are difficult to interpret, however, because workers were exposed to other stressors, such as physical danger and heavy work demands, in addition to excessive noise, and these may be more potent than noise in causing symptoms. There may also be differential selection of individuals working in noisy areas. For instance, jobs in noisy areas may be less desirable, may be more difficult to fill, and hence may attract individuals with health problems which have prevented them from attaining more desirable jobs. In contrast health factors may operate in the selection of personnel for jobs in high noise-exposure areas which may be dangerous, demanding toughness and resilience not required for jobs in quieter areas (e.g. few symptoms were found among men working in high noise on aircraft carriers; Davis, 1958). Also the choice of coping strategies by individuals may influence whether aircraft noise actually causes symptoms. Evasive coping strategies were related to higher scores on the Hopkins Symptom Check List in areas of high exposure to aircraft noise (Passchier-Vermeer, 1993).

2.7.2 COMMUNITY STUDIES OF PSYCHOLOGICAL SYMPTOMS

Environmental noise experienced outside work settings, though less intense, tends to be more difficult for the ordinary citizen to avoid. Community surveys have

found that high percentages of people reported headaches, restless nights, and being tense and edgy in high-noise areas (OPCS, 1971; Kokokushka, 1973; Finke *et al.*, 1974; Öhrström, 1989). An explicit link between aircraft noise and symptoms, which emerges from such studies, raises the possibility of a bias towards over-reporting of symptoms (Barker & Tarnopolsky, 1978). Notably, a study around three Swiss airports (Grandjean *et al.*, 1973) did not mention that it was concerned with aircraft noise and did not find any association between the level of exposure to aircraft noise and symptoms. In the West London Survey, some symptoms, such as tinnitus, burns, cuts and minor accidents, and ear and skin problems, were more common in areas of high noise exposure (Tarnopolsky *et al.*, 1980). Acute symptoms (e.g. depression, irritability, difficulty getting to sleep, night waking, skin troubles, swollen ankles, and burns, cuts and minor accidents) were particularly common. However, apart from 'ear problems' and 'tinnitus', 20 out of 23 chronic symptoms were more common in low noise environments. Symptoms did not increase with increasing levels of noise. This is possibly related both to more social disadvantage and associated ill-health among residents in low aircraft noise exposure areas and to the possible unwillingness of chronically unhealthy individuals to move into potentially stressful high noise exposure areas. Nevertheless, it would not exclude an effect of noise in causing some acute psychological symptoms, and many of the effects of noise in industrial and teaching settings may in fact be related primarily to disturbances in communication.

2.7.3 MENTAL HOSPITAL ADMISSION RATES

Much of the concern with the possible effects of noise on mental health began with studies of admissions to psychiatric hospitals from noisy areas. Early studies found associations between the level of aircraft noise and psychiatric hospital admissions, both in London (Abey-Wickrama *et al.*, 1969) and Los Angeles (Meecham & Smith, 1977; Meecham & Shaw, 1979). These results have been criticised on methodological grounds (Chowns, 1970; Frerichs *et al.*, 1980), and a replication study of Abey Wickrama's study by Gattoni & Tarnopolsky (1973) failed to confirm earlier findings. Jenkins *et al.* (1979) found that age-standardised admission rates to a London psychiatric hospital, over four years, were higher as the level of noise in an area decreased, but lower noise areas were also central urban districts, where high admission rates would be expected. In a further extensive study (Jenkins *et al.*, 1981), high aircraft noise was associated with higher admission rates in two of three hospitals, but in all three hospitals, admission rates seemed to follow non-noise factors more closely. The effect of

noise, if any, could only be moderating, but not overriding, that of other causal variables. Kryter (1990), in a re-analysis of the data, found ‘a more consistently positive relation between level of exposure to aircraft noise and admissions rates’. In conclusion, it appears that hospital admission is influenced by many psychosocial variables which are more potent than exposure to noise. Therefore, whether or not noise causes psychiatric disorder would be better answered by studying a community sample.

2.7.4 PSYCHIATRIC MORBIDITY IN THE COMMUNITY

In a community pilot study carried out in west London, Tarnopolsky *et al.* (1978) found no association between aircraft noise exposure and either general health questionnaire (GHQ) scores (dichotomised 4/5, low scorers/high scorers; Goldberg, 1972) or estimated psychiatric cases (Goldberg *et al.*, 1970). This was so even when exposure to road traffic noise was controlled, except in three subgroups: people aged 15–44 of high education, women aged 15–44, and those in professional or managerial occupations. These authors expressed the guarded opinion that noise might have an effect in causing morbidity within certain vulnerable subgroups.

In the subsequent West London Survey of Psychiatric Morbidity (Tarnopolsky & Morton-Williams, 1980), 5885 adults were randomly selected from within four aircraft noise zones according to the Noise & Number Index. No overall relationship was found between aircraft noise and the prevalence of psychiatric morbidity either for GHQ scores or for estimated numbers of psychiatric cases, using various indices of noise exposure. However, there was an association between noise and psychiatric morbidity in two subgroups: those who finished full time education at 19 years or older, and professionals. When combined, these two categories, which had a strong association with each other, showed a significant association between noise and psychiatric morbidity ($\chi^2 = 8.18$, $df\ 3$, $p < 0.05$), as assessed by GHQ scores only. Tarnopolsky & Morton-Williams (1980) concluded that their results ‘show so far that noise *per se* in the community at large, does not seem to be a frequent, severe, pathogenic factor in causing mental illness but that it is associated with symptomatic response in selected subgroups of the population’.

The possible relationship between noise and psychiatric disorder was pursued further in a population unlikely to have been selected by noise exposure (which may be the case around a well-established airport such as Heathrow) by examining the association between road traffic noise exposure, noise sensitivity and

psychiatric disorder, in a study of the small town of Caerphilly, South Wales. In the longitudinal study, no association was found between the initial level of road traffic noise and minor psychiatric disorder, even after adjustment for sociodemographic factors and baseline psychiatric disorder (Stansfeld *et al.*, 1996). However, there was a small non-linear association of noise with increased anxiety scores (Stansfeld *et al.*, 1996).

Psychosocial well-being has been shown to be reduced in areas exposed to high traffic noise, but the results have not been especially consistent and may be mediated through disruptive effects on sleep (Öhrström, 1989, 1993). A questionnaire study of 1053 residents around Kadena airport in Japan found no relationship between noise and mental ill-health between 75–94 WECPNL (the power average of the maximum perceived noise level in dB(A)), but a dose–response relationship between noise exposure (75–95+WECPNL and above) and depression and nervousness (Ito *et al.*, 1994). Among 7540 people in a British road traffic noise study, the ‘noise level in dB(A) exceeded for 10% of the time’ was weakly associated with a five item mental health symptoms scale (Halpern, 1995). This association remained after adjustment for age, sex, income and length of residence. This scale included some clear mental health items, but also some that were less obviously related to mental health. Weaker associations between traffic density and the mental health symptom scale may relate to the skewed distribution of the traffic density variable. It also seems that traffic noise level was more important than traffic flow. There was no interaction between noise exposure and noise sensitivity in determining symptoms, as has also been reported in other studies (Stansfeld *et al.*, 1996). It is not clear that the reported association between noise level and mental health symptoms was actually due to noise exposure. Adjustment for the amount of ‘noise heard’ reduced the association very little, suggesting no causal association with noise, although ‘noise heard’ may not be well measured.

The use of health services has also been taken as a measure of the relationship between noise and psychiatric disorder. Grandjean (1973) reported that the proportion of the Swiss population taking drugs was higher in areas with high levels of aircraft noise, and Knipschild and Oudshoorn (1977) found that the purchase of sleeping pills, antacids, sedatives and antihypertensive drugs all increased in a village newly exposed to aircraft noise, but not in a control village where the noise level remained unchanged. In both studies, there was also an association between the rate of contact with general practitioners and level of noise exposure. In a study of five rural Austrian communities exposed to road traffic noise, exposure to a high noise level (> 55 dB(A)) was associated with an

increased risk of taking sleeping tablets (OR, 2.22; CI 1.13–4.38) and overall prescriptions (OR, 3.65; CI 2.13–6.26; Lercher, 1996) relative to exposure to low (≤ 55 dB(A)) noise levels. In the Heathrow study (Watkins *et al.*, 1981), various health care indicators were used (use of drugs, particularly psychiatric or self-prescribed, visits to the general practitioner, attendance at hospital, and contact with various community services), but none of these showed any clear trend in relation to levels of noise. This may be due to the differing distribution of ill-health in areas with different noise exposure, such that low noise areas have more people with chronic ill-health than do high noise areas.

2.7.5 NOISE ANNOYANCE, SYMPTOMS AND PSYCHIATRIC MORBIDITY

Noise annoyance is associated both with noise level and with symptoms and psychiatric disorder. Although there is a strong link between noise and annoyance, and those who are highly annoyed show the greatest number of symptoms, symptoms are not more common in high-noise compared with low-noise areas (Tarnopolsky *et al.*, 1980). This apparent paradox might be explained by the ‘vulnerability hypothesis’ (Tarnopolsky *et al.*, 1980). According to this explanation, noise is not directly pathogenic, but sorts individuals into annoyance categories according to their vulnerability to stress. Tarnopolsky *et al.* (1978) found that noise and minor psychiatric disorder were the strongest predictors of annoyance and that having psychiatric morbidity led to annoyance, rather than vice versa. Moreover, annoyance does not seem to act as an intervening variable between noise and morbidity. At any particular level of exposure, there is wide individual variation in the degree of annoyance that is expressed. Individual variance in annoyance can be explained largely in terms of noise sensitivity and attitudes to the source of the noise (Evans & Tafalla, 1987; Langdon, 1987; Job, 1988).

2.7.6 NOISE SENSITIVITY AND VULNERABILITY TO PSYCHIATRIC DISORDER

Noise sensitivity, based on attitudes to noise in general (Anderson, 1971; Stansfeld, 1992), is an intervening variable that explains much of the variance between exposure and individual annoyance responses (Langdon, 1976; Weinstein, 1978; Langdon *et al.*, 1981; Öhrström *et al.* 1988; Fields, 1994). Individuals who are noise-sensitive are also likely to be sensitive to other aspects

of the environment, such as temperature, brightness and unpleasant smells (Anderson, 1971; Broadbent 1972; Weinstein, 1978; Thomas & Jones, 1982; Stansfeld *et al.*, 1985; Öhrström *et al.*, 1988b). This raises the question whether noise-sensitive individuals are simply those who complain more about their environment. Certainly there is an association between noise sensitivity and neuroticism (Anderson, 1971; Thomas & Jones, 1982; Öhrström, *et al.*, 1988b; Jelinkova, 1988), although it has not been found in all studies (Broadbent, 1972). However, Weinstein (1980) suggested another view, that noise sensitivity is part of a critical-uncritical dimension, showing the same association as noise sensitivity to measures of noise, privacy, air pollution, and neighbourhood reactions. He suggested that the most critical subjects, among whom noise-sensitive people would be grouped, are not uniformly negative about their environment, but more discriminating than the uncritical group.

Noise sensitivity has also been related to current psychiatric disorder (Bennett, 1945; Tarnopolsky & Morton-Williams, 1980; Iwata, 1984; Poenaru *et al.*, 1987). Stansfeld *et al.* (1985) found that high noise sensitivity was particularly associated with phobic disorders and neurotic depression measured by the 'present state examination' (Wing *et al.*, 1974). Noise sensitivity has also been linked to a coping style based on avoidance which may have adverse health consequences (Pulles *et al.*, 1988). Noise sensitivity may be partly secondary to psychiatric disorder. Depressed patients followed up over four months became less noise-sensitive as they recovered. These 'subjective' psychological measurements were complemented by 'objective' psychophysiological laboratory investigation of reactions to noise, in a subsample of depressed patients. Noise-sensitive people tended to have higher levels of tonic physiological arousal, more phobic and defence/startle responses and slower habituation to noise (Stansfeld, 1992). Thus, noise-sensitive people attend more to noises, discriminate more between noises, find noises more threatening and out of their control, and react to, and adapt to noises more slowly than people who are less sensitive in this way. Through its association with greater perception of environmental threat and its links with negative affectivity and physiological arousal to noise, noise sensitivity may be an indicator of vulnerability to minor psychiatric disorder (Stansfeld, 1992).

In the Caerphilly Study, noise sensitivity did predict psychiatric disorder at follow-up, after adjusting for baseline psychiatric disorder, but did not interact with the noise level, suggesting that noise sensitivity does not specifically moderate the effect of noise on psychiatric disorder (Stansfeld *et al.*, 1996). After adjusting for trait anxiety at baseline, the effect of noise sensitivity was no longer statistically significant. This suggests that much of the association between noise sensitivity

and psychiatric disorder may be accounted for by the confounding association with trait anxiety. Thus, constitutionally anxious people may be both more aware of threatening aspects of their environment and more prone to future psychiatric disorder. It seems possible that these might be linked.

2.7.7 POSSIBLE MECHANISMS FOR NOISE EFFECTS ON MENTAL HEALTH

Noise exposure may have psychological, behavioural and somatic effects. It has been postulated that noise exposure creates annoyance, which then leads on to more serious psychological effects. This pathway remains unconfirmed, rather noise causes annoyance, and independently, mental ill-health probably increases annoyance. Nevertheless, as Van Dijk suggests, minor psychological disturbance (e.g. tension, irritability, difficulty concentrating), rather than psychiatric disorder, is likely to be a sequel of noise annoyance and to feed back to increase annoyance.

A more sophisticated model (Passchier-Vermeer, 1993) incorporates the interaction between the person and their environment. In this model the person readjusts their behaviour in noisy conditions to reduce exposure. An important addition is the inclusion of appraisal of noise (in terms of danger, loss of quality, meaning of the noise, challenges for environmental control etc.) and coping (ability to alter behaviour to deal with stressors). This model emphasises that dealing with noise is not a passive process. As with other mechanisms for noise effects there is always the possibility that effects are spurious and that noise is merely a marker of other hazardous environmental conditions. This does not rule out the possibility that noise acts through moderating other stressors or through its effects being moderated by other factors such as perceived control or coping strategies

2.7.8 SCOPE FOR FURTHER RESEARCH

Currently there is still uncertainty as to whether noise exposure may cause mental health symptoms and raised anxiety levels. Existing studies may be confounded either by prior selection of subjects out of (or into) noisy areas, related to prior noise exposure, or by confounding between noise exposure, socioeconomic status (and deprivation) and psychiatric disorder. Ideally, further studies investigating the association between noise and psychiatric disorder should be carried out longitudinally, either in populations in whom there is a change to lower noise exposure, or better still, where baseline measurements are carried out in low noise

conditions and follow-up measurements are made at a time when noise exposure has increased. In this way the population will not be preselected to include only those less affected by noise, and migration of subjects out of the study sample can be followed, to assess whether noise-sensitive subjects tend to move away as the noise increases. There should also be measurement of other contemporaneous environmental stressors, careful assessment of socioeconomic differences between areas of high and low noise exposure and assessment of potential moderating factors between noise exposure and psychiatric disorder.

There is also a need for improving the measurement of noise sensitivity and annoyance. At the same time the breadth of psychiatric outcomes should be enlarged to include well-being, hostility, depression, anxiety and phobias, while also measuring relevant aspects of personality, such as neuroticism and negative affectivity, which may influence reporting of symptoms. This needs to be combined with measurement of the appraisal of noise sources and evaluation of coping mechanisms

2.8 ANNOYANCE

2.8.1 ANNOYANCE REACTIONS

The most widespread and well documented subjective response to noise is annoyance, which may include fear and mild anger, related to a belief that the individual is being avoidably harmed (Cohen & Weinstein, 1981). Noise is also seen as intrusive into personal privacy (Wilson, 1963), while its meaning, for any individual, is important in determining whether that person will be annoyed by it (Gunn, 1987).

Annoyance reactions are often associated with the degree of interference that any noise causes in everyday activities; such perceived interference probably precedes and leads on to annoyance (Taylor, 1984; Hall *et al.*, 1985). In both traffic and aircraft noise studies, noise levels have been found to be associated with annoyance in a dose-dependent fashion (McKinnell, 1963; Griffiths & Langdon, 1968; Schultz, 1978; Tarnopolsky & Morton-Williams, 1980). There is continuing evidence of a dose-effect relationship for transportation noise (Fields, 1984). Annoyance is also dependent on the context in which the noise is heard. Overall, it seems that conversation, watching television or listening to the radio (all involving

speech communication) are the activities most disturbed by aircraft noise (Hall *et al.*, 1985), while traffic noise, if present at night, is most disturbing for sleep.

2.8.2 ACOUSTIC PREDICTORS OF NOISE ANNOYANCE IN COMMUNITY SURVEYS

One of the primary characteristics determining the undesirability of noise is its loudness or perceived intensity. Loudness is comprised of the intensity of sound, the tonal distribution of sound and its duration. Judgements of noisiness and loudness of a series of aircraft and community sounds have shown small but significant differences (Berglund *et al.*, 1976). The evidence is mixed on the importance of the duration and the frequency components of sound in determining annoyance. High frequency noise has been found to be more annoying than low frequency noise (Bjork, 1986). McKennell (1977) found that the short duration of Concorde flights over London appeared to offset somewhat the increased perceived loudness of Concorde compared with conventional jet aircraft. Correlations between noise and annoyance are lower for impulse than continuous noise (Job, 1988). This may be partly because of the smaller range of noise exposure in some studies, but is also likely to result from the higher correlation between attitude and annoyance in impulse noise studies (Job, 1988). Vibrations are perceived as a complement to loud noise in most community surveys of noise and are found to be important factors in determining annoyance, particularly because they are a commonly experienced through other senses as well as hearing (McKennell, 1977).

There are complex interactions between background noise exposure and number of noise events in determining annoyance (Fields, 1984). There is some evidence to suggest that after a certain number of flights a ceiling effect on annoyance does occur, although the very small number of subjects exposed to very high noise levels makes this difficult to assess accurately. There is no evidence that ambient noise has a significant effect on noise annoyance related to target noises such as aircraft, road traffic, railway or impulse noise (Fields, 1996). Generally sex differences in annoyance have not been found. Age differences may relate to other factors, such as having young children or cohort effects (Utley & Keighley, 1988). The relationship between social class and annoyance and noise annoyance is inconsistent. If anything, higher socioeconomic status is related to higher annoyance (Bradley & Jonah, 1979). This may relate to expectations of higher standards of environmental conditions in those of higher socioeconomic status. It may also be that such individuals are more likely to complain about noise.

In a path model analysis of aircraft and traffic noise annoyance, Taylor (1984) found that noise sensitivity had a stronger effect on annoyance than aircraft noise level and was the most important non-noise predictor of annoyance. Other predictors of annoyance include fear of the noise or noise source (Gunn *et al.*, 1981; Bullen & Hede, 1986) and attitudes to the noise source. These include the predictability and controllability of the noise, general dislike of the environment (Langdon, 1987) and attitudes to the noise source, including perception of misfeasance (Borsky, 1961, Jonah *et al.*, 1981). Less than 50% of the surveys on annoyance reviewed by Fields (1992) found that socioeconomic factors influenced annoyance responses, but over 50% of surveys found that, after controlling for noise level, noise annoyance increases with fear of danger from the noise source, sensitivity to noise, the belief that the authorities can control the noise, awareness of the non-noise impacts of the source and the belief that the noise source is not important. There is also some limited evidence that personality traits, such as extraversion, predict annoyance, but not all studies have found this (Raw & Griffiths, 1988). In three information processing tasks, highly rigid subjects (on a scale of inflexibility) were more annoyed by noise and performed the tasks less accurately during noise exposure than more flexible subjects (Brand *et al.*, 1995).

2.8.3 SCOPE FOR FURTHER RESEARCH

Current research strategies are not broad enough to encompass and differentiate between the wide range of variables related to noise dissatisfaction. In evaluating the influence of noise on annoyance, factors such as a general disposition to feel vulnerable in the face of events, specific harm and risk attributed to noise sources, and differences in individual perceptions of environmental issues all need further consideration. Also we need to know to what extent specific attributes of harm to noise are really attributable to noise or are a function of disposition to feel greatly distressed.

2.9 NON-AUDITORY EFFECTS IN CHILDREN

2.9.1 VULNERABLE POPULATION GROUPS

Individuals within populations exposed to noise may not be uniformly susceptible to the effects of noise on health (Stansfeld, 1992; Job, 1995). Thus to assess the effects of noise on health it may be most productive to focus on exposure to noise in high risk groups. Elderly people may be more susceptible to sleep disturbance in noisy environments and may also possibly be more vulnerable to the hypertensive effects of noise. However, the best evidence of increased vulnerability to noise comes from studies of children. Children may be more susceptible to environmental stress than adults for a variety of reasons, including a lower cognitive capacity to understand environmental issues and anticipate stressors, and a lack of well-developed coping repertoires (Cohen *et al.*, 1986; Evans *et al.*, 1991). Impairments of early childhood development and education by environmental pollutants, such as noise, may have life-long effects on the ability to achieve academic potential and on health (Evans *et al.*, 1991).

2.9.2 COGNITION

The most striking and widely researched effects of noise found in children are cognitive impairments (Cohen *et al.*, 1986; Evans *et al.*, 1991; Evans & Lepore, 1993). Several researchers (Cohen *et al.*, 1986; Evans & Lepore, 1993; Hygge, 1994; Evans *et al.*, 1995) have claimed that tasks that involve central processing and language comprehension, such as reading, attention, problem solving and memory, are most affected by exposure to noise.

For pre-school aged children, Wachs and Gruen (1982) have accumulated data from cross-sectional and longitudinal studies indicating a negative association between home noise levels and cognitive development in children from six months to five years of age (reviewed by Evans & Lepore, 1993). This present review will concentrate on the effects of long-term exposure to noise on the cognitive development of primary aged school children (5–12), because this is a critical period for learning acquisition, in which future learning patterns are established.

The effects of long-term exposure to noise at home and school have been examined in well designed studies, which have direct relevance to the health of children in the community (see Evans and Lepore's 1993 review for further discussion of acute noise effects).

In studies examining the effects of long-term aircraft, train and road traffic noise on cognitive performance among school children, the following results have been found in children exposed to high levels of environmental noise:

- ❑ deficits in sustained visual attention (Karsdorff & Klappach, 1968; Kyzar, 1977; Heft, 1979; Moch-Sibony, 1984; Hambrick-Dixon, 1986; 1988; Sanz *et al.*, 1993);
- ❑ teachers reporting that children in noisy schools have more difficulties in concentrating in comparison with children from quieter schools (Crook & Langdon, 1974; Ko, 1979; Kryter, 1985);
- ❑ poorer auditory discrimination and speech perception (Cohen *et al.*, 1973, 1980; Moch-Sibony, 1984; Cohen *et al.*, 1986; Evans *et al.*, 1995);
- ❑ poorer memory that requires high processing demands (Fenton *et al.*, 1974 (short-term noise); Hygge, 1994 (short-term noise); Evans *et al.*, 1995; Hygge *et al.*, 1996); and
- ❑ poorer reading ability (Michelson, 1968; Cohen *et al.*, 1973; Bronzaft & McCarthy, 1975; Maser *et al.*, 1978; Bronzaft, 1981; Lukas *et al.*, 1981; Green *et al.*, 1982; Evans *et al.*, 1995).

Some of the earlier studies examining the effects of noise in children have serious methodological flaws, limiting the conclusions that can be drawn from the data. Some of these flaws are outlined below.

- ❑ Data were not provided to indicate how well socioeconomically matched the noise-exposed children were to the control sample (Karsdorf & Klappach, 1968; Kyzar, 1977; Heft, 1979).
- ❑ The sample size was not large enough (all of the studies).

- ❑ There were not enough schools to rule out a school effect confounding the results (Cohen *et al.*, 1980, 1981, 1986; Sanz *et al.*, 1993).
- ❑ Statistical methods were not sensitive enough (Sanz *et al.*, 1993).
- ❑ Most studies were cross-sectional.

The results from two longitudinal field studies, which controlled for socioeconomic factors, showed that long-term exposure to aircraft noise was consistently and reliably associated with cognitive impairments in school children (Cohen *et al.*, 1986; Evans & Lepore, 1993; Hygge, 1994; Evans *et al.*, 1995).

In the 1980s, impaired performance on a difficult cognitive task was found in primary school children aged 8–9 years around Los Angeles Airport (cross-sectional results Cohen *et al.*, 1980; longitudinal results Cohen *et al.*, 1981). In the 1990s, similar noise-related cognitive effects have been found in experimental field research around Munich Airport, in older children with a mean age of 10.8 years (cross-sectional results Evans *et al.*, 1995; longitudinal results Hygge *et al.*, 1996). In 1992 the old Munich airport closed and a new airport was opened. The cross-sectional results indicate an association between high noise exposure and poor long-term memory and reading comprehension (Evans *et al.*, 1995). Longitudinal analyses, after three waves of testing, indicate improvements in long-term memory after closure of the old airport. Strikingly, these effects were paralleled by impairment of the same cognitive skills after the new airport opened (Hygge *et al.*, 1996). Further research is required to clarify these findings in a larger sample of school children, to examine causal mechanisms and to propose and test interventions.

2.9.3 MOTIVATION

Long-term exposure to aircraft noise has been associated with decreased motivation in school children (Cohen *et al.*, 1980, 1981, 1986; Evans *et al.*, 1995). In Los Angeles and Munich, motivation was measured as persistence with a difficult cognitive task. School children exposed in the long term to high levels of aircraft noise persisted less with an insoluble puzzle (Glass & Singer, 1972) than control groups of low noise-exposed children (Cohen *et al.*, 1980, 1981, 1986; Evans *et al.*, 1995). This motivation effect may either be independent or secondary to noise related cognitive impairments. The conceptualisation and measurement of motivation in noise research needs further development.

2.9.4 CARDIOVASCULAR EFFECTS

There is evidence that children are not only susceptible to cognitive impairment in noisy environments, but may also react physiologically to noise in terms of raised blood pressure. Seven out of nine studies (from 1968–1990) reviewed by Evans and Lepore (1993) report elevations of resting blood pressure among children with long-term exposure to aircraft and road traffic noise. Even though methodological problems (such as unreliable noise measurements, selective attrition, lack of control of sociodemographic factors) seriously limit the conclusions that can be drawn from some of these earlier studies (e.g. Karsdorf & Klappach, 1968; Karagodina *et al.*, 1969), the Los Angeles and Munich studies and two further studies in the 1990s (Wu *et al.*, 1993; Regecova & Kellerova, 1995) provide more reliable evidence on cardiovascular effects.

In Los Angeles, the results from cross-sectional studies indicate small but significant increases in systolic and diastolic blood pressure associated with long-term exposure to aircraft noise (Cohen *et al.*, 1980, 1986). This elevated blood pressure, found in 1980, does not appear to habituate with continued exposure (Cohen *et al.*, 1986). The ranges of blood pressure elevation in noise-exposed children in these studies are within normal levels and do not suggest hypertension. In Munich, there was a marginally significant relationship between noise exposure and baseline systolic blood pressure. A lower reactivity in systolic blood pressure was also observed when a cognitive task was carried out during short-term exposure to noise (Evans *et al.*, 1995). Diastolic blood pressure and reactivity were unrelated to noise exposure.

Regecova and Kellerova (1995) found that exposure, while at school, to high levels of urban road traffic noise (>60 dB(A)) was associated with higher systolic and diastolic blood pressure and mean heart rate in children aged 3–7 years. This cross-sectional study provides very strong evidence that noise may lead to elevated blood pressure in children because the sample size is large (1542 children), there were 30 schools examined, and controls for age, body weight, height, demographic and socioeconomic factors were used in the analysis.

BLOOD PRESSURE IN CHILDREN

One way to examine whether noise influences blood pressure is to study a group not significantly exposed to noise at all. Wu *et al.* (1993) report a comparison of 309 deaf mute children with 583 normal hearing children; both groups were at

school in an area with heavy traffic noise (60–75 dB(A)). Both systolic and diastolic blood pressure were lower in the deaf mute children than in the normal hearing children, after adjusting for age and body mass index. However, it seems premature to attribute this lower blood pressure to less noise exposure, as the cause of the deafness (e.g. rubella) may influence cardiovascular health, and the nature of school task performance, which may influence blood pressure, is likely to be different between the two groups of children.

In order to eliminate the effects of confounding factors in the relationship between noise and blood pressure, Regecova and Kellerova (1995) carried out a cross-sectional study of 1542 3–7 year old children in kindergartens exposed to three noise conditions (quiet ≤ 60 dB(A), noisy 61–69 dB(A), very noisy ≥ 70 dB(A)). They found significantly higher systolic and diastolic blood pressure among children in noisy or very noisy environments (both kindergarten and home noise) compared with quiet environments. These findings were repeated after stratification for age and were not affected by height or weight. This is an important study, although it is difficult to tell whether social class might have confounded the association between noise exposure and blood pressure. Girls aged 9–13 years in Franken, exposed to military flights at 75 metres, had higher systolic (9 mm) and diastolic (3.4 mm) blood pressures than girls exposed to overflights at 150 metres. This was not found in either boys or girls together from another area tested (Munsterland; Ising *et al.*, 1990). There was no adjustment for potential confounding factors and no discussion of factors other than noise that might have accounted for the findings in this study.

2.9.5 ENDOCRINE DISTURBANCE

The Munich study was the first study to examine neuroendocrine indices of chronic stress among people exposed to community noise. Overnight resting levels of urinary catecholamines (adrenaline and noradrenaline) were significantly higher in children exposed long-term to aircraft noise than in those not so exposed (Evans *et al.*, 1995). There was no significant association between long-term exposure to aircraft noise and cortisol level.

2.9.6 NOISE ANNOYANCE

Children have been found to be annoyed by long-term exposure to noise (Bronzaft & McCarthy, 1975; Evans *et al.*, 1995). In Munich, it was found that children

living in noisier areas were significantly more annoyed by noise in their community, as indexed by a calibrated community measure that adjusts for individual differences in rating criteria for annoyance judgments. Children living in the noisy environment had lower psychological well-being than children living in quieter environments (Evans *et al.*, 1995). It is important to recognise that even young children report disturbance by environmental noise. In many ways a child's annoyance response to noise may be less subject to bias because children are less affected by other factors that influence annoyance in adults, such as political attitudes and attitudes to environmental issues.

2.9.7 POSSIBLE MECHANISMS OF NOISE EFFECTS

Three main mechanisms have been proposed to account for the effects of noise exposure on cognition and motivation in children. The mechanism accounting for the effects of noise exposure on children's blood pressure, endocrine disturbance and annoyance is considered to be the same stress mechanism as that proposed to account for the effect of noise in adults. Most of the noise research among children has been exploratory and cross-sectional, which means that future research needs to examine the explanatory power of these cognitive and motivation mechanisms.

ATTENTION DIFFICULTIES: A MECHANISM FOR COGNITIVE EFFECTS

The main theory guiding research into noise related cognitive impairments has been that noise restricts attention to central cues during complex language related tasks (Cohen *et al.*, 1980; Heft, 1985). Children may adapt to noise interference during activities by filtering out the unwanted noise stimuli. This tuning out strategy may over generalise to all situations when noise is not present, such that children tune out stimuli indiscriminately. In turn, this may lead to noise-exposed children having a poorer ability to sustain attention in the classroom, which may continue over time to affect concentration and learning, even in the absence of noise exposure. This attention theory has been supported by experimental studies among adults (Smith & Jones, 1992) and children (Evans & Lepore, 1993).

TEACHER FRUSTRATION AND COMMUNICATION DIFFICULTIES: A MECHANISM FOR COGNITIVE AND MOTIVATION EFFECTS

Distraction and annoyance from noise interruptions may have indirect effects on cognitive development because of alterations in parents' and teachers' behaviour (Evans *et al.*, 1991). Noise exposure also affects communication in the classroom, making it more difficult for children to learn and teachers to teach; this may lead to frustration and interruption in speech and may reduce instruction time (Bronzaft & McCarthy, 1975; Crook & Langdon, 1974). Environmental noise may affect teachers' and parents' encouragement of children, and this may, potentially, contribute to the effects of noise on child cognitive development and motivation.

LEARNED HELPLESSNESS: A MECHANISM FOR MOTIVATION EFFECTS

Lack of persistence with a difficult task has been considered a manifestation of the helplessness state. It has been postulated that children feel that they have a 'lack of control' over their noisy environment and thus develop a learned helplessness state (Cohen *et al.*, 1980; Evans & Lepore, 1993; Evans *et al.*, 1995). Children may adapt to noise interference during activities by withdrawal strategies or by learning to be helpless in the presence of uncontrollable noise when performing tasks. This learned helplessness may become a generalised learned response, which becomes manifest as low motivation during performance of all tasks, regardless of the presence of noise.

2.9.8 SCOPE FOR FURTHER RESEARCH

These studies provide preliminary evidence suggesting that there are strong grounds for concern about the effect of long-term aircraft noise on cognition and blood pressure in children. The studies indicate that endocrine status, motivation and annoyance may be affected by noise exposure. The findings need to be clarified by further research to address the following critical issues:

- ❑ the testing of proposed mechanisms;
- ❑ the examination of factors that may potentially mediate, moderate and confound the effect of noise on child health; and

- ❑ the isolation of vulnerable groups within the child population.

Future research needs to be longitudinal, well-controlled, with a large sample of school children from many schools and with a wide range of noise exposure. Longitudinal research will be able to:

- ❑ demonstrate whether these noise effects persist over time;
- ❑ assess whether the size of the effect increases or decreases; and
- ❑ examine both the current effects of noise and the delayed effects of noise.

2.10 COMBINED EFFECTS ON HEALTH OF NOISE AND OTHER STRESSORS

2.10.1 NOISE AND OTHER STRESSORS

There is much debate over whether the effects of noise on health may be augmented by, or may augment in turn, the impact of other stressors on health. Stressors may act synergistically, antagonistically or may not interact at all. Stressors may include physical, chemical, and biological factors, as well as the structure of the work task itself, the formal conditions of employment and work relationships (Van Dormolen & Hertog, 1988). There has been much emphasis on laboratory studies, without considering that results of such studies may lack external validity.

Past research on combined effects has not considered common conditions and levels of stressors across studies, direct and indirect effects, long durations of exposure and complex tasks (Sandover & Porter, 1987). In a laboratory-based performance experiment, involving 134 18–34 year olds, an interaction was found between having a cold and noise exposure on simple reaction time (Smith *et al.*, 1993). There was little difference between healthy and cold subjects' performance

when tested in quiet, but for subjects tested in noisy conditions (70 dB(A)) performance was much slower for the subjects who had a cold. This result was fairly specific because other tests showed effects of noise (e.g. detection of repeated numbers task) or effects of colds, but no interaction.

In a factorial design experiment involving 60 healthy male subjects, noise and vibration had independent significant effects on diastolic blood pressure, while temperature, and noise had a combined effect on morning adrenaline secretion rate (Manninen, 1987), but these were only a few of the many effects tested. Several studies have considered the combined effects of noise and vibration in drivers. A higher degree of physiological activation was found, indexed by raised heart rate, in those having to cope with noise and vibration simultaneously (Cnockaert *et al.*, 1988).

Some studies do not separate out the combined effects. For instance, noise and vibration exposure in furniture industry workers caused joint pain, chest pain, stomach disorder, headaches, tremor and visual problems (Bielski, 1987).

Studies on night shift work and noise show conflicting results. Smith (1988) found that noise and night work had independent effects on performance testing, with generally no interactions with personality, except a small interaction with introversion. In contrast, Koller *et al.* (1988) found that night shift workers were more sensitive to noise, and this was related to more health problems. It could be that noise-sensitive shift workers are more disturbed by noise while sleeping during the day, or there might be synergism between the stressor of night work and occupational noise exposure, which is moderated by noise sensitivity.

2.10.2 POSSIBLE MECHANISMS OF NOISE EFFECTS

Noise may either act independently of other stressors, or in a synergistic or antagonistic manner. Statistical interactions between noise and other stressors may be difficult to interpret and may depend on the method of analysis. There is a lack of clearly defined conceptual models in this area of research, and the conflicting results are difficult to interpret. In addition, laboratory results may not be generally applicable to field conditions, where the pattern of stressors and their interpretation may be different.

2.10.3 SCOPE FOR FURTHER RESEARCH

There is a need for more field studies with defined objectives and outcomes, better measurement of stressor exposures and more attention paid to measurement of confounding factors. Nevertheless, the impact of combined stressors on health is likely to be complex, and individual appraisal of stressors and coping processes must also be taken into account if meaningful results are to be obtained.

2.11 CONCLUSIONS

The evidence for effects of environmental noise on health is strongest for annoyance, sleep and performance. Occupational noise exposure also shows an association with raised blood pressure. Dose–response relationships can be demonstrated for annoyance, and less consistently, for blood pressure.

Undoubtedly there is a need for further research to clarify this complex area. This should include better measurement of noise exposure and better measurement of health outcomes, and more attention should be given to consideration of confounding factors and effect modifiers in the association between noise and health. There should also be more emphasis on studies combining different environmental stressors with noise and including complementary health outcomes (e.g. endocrine and performance measures in occupational studies). Moreover, there should be a greater emphasis on field studies using longitudinal designs with careful choice of samples to avoid undue bias related to prior noise exposure. Studies on vulnerable groups within the population may yield the most important results.

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3 Summary reviews on noise exposure and specific health effects

3.1 THE EFFECTS OF NOISE ON SLEEP

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3.1.1 INTRODUCTION

Investigations of the effects of noise on sleep have focused to a great extent on primary effects during sleep, such as changes in electroencephalogram (EEG) and heart rate. In recent years, studies have also been performed on different after-effects, such as subjective sleep quality, performance and psychosocial well-being. Little attention has been paid, however, to effects of noise on the time needed to fall asleep.

3.1.2 EFFECTS OF NOISE ON TIME TO FALL ASLEEP

Difficulty in falling asleep is considered to be an important aspect of noise-induced sleep disturbances in man. Different studies on noise effects, with noise levels from 45 dB(A) peak level, show an increase of 7 to 15 minutes in the time to fall asleep. The number of events and the difference between background and peak level seem to be more important than the absolute peak level for these types of effects.

3.1.3 EFFECTS OF NOISE DURING SLEEP

Cardiac responses during sleep occur at very low peak noise levels (32 dB(A); Vallet *et al.*, 1988). For other acute reactions, such as body movements, recent studies show no difference in the reactions to 45, 50 or 60 dB(A) maximum noise levels. A threefold increase in the number of body movements at all three noise levels has been found at 16 noise exposure events per night, and a slightly lower

increase (twofold) at 64 events per night, when compared with quiet periods of the night. To minimise awakening effects, Griefahn (1990) proposed limits for both the number of noise events and maximum noise levels, for irregular noise during the night. According to Griefahn, no awakenings occur at levels below 54 L_{Amax} irrespective of the number of noise events. At higher L_{Amax} levels the number of awakenings increases with the number of noise events up to about 40 noise events per night.

3.1.4 AFTER-EFFECTS OF NOISE-DISTURBED SLEEP

More recent studies indicate reduced subjective sleep quality after exposure to 45 dB(A) maximum noise levels, among noise-sensitive persons (Öhrström, 1995). This group of 'rather or very noise-sensitive' individuals has been shown to include about one third of the general population. At 32 noise events per night, sleep quality was reduced by 9%, and at 128 noise events the reduction was 19%, when compared with quiet, control nights. A maximum noise level of 45 dB(A) also caused increased tiredness during the day. Further evidence is now available in the literature concerning more long-term effects of noise-disturbed sleep. These results indicate that noise disturbed sleep may also effect psychosocial well-being.

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3.2 NOISE AND PERFORMANCE

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Studies on the effects of noise on performance have been reviewed previously (Smith, 1993). The main points are as follows.

3.2.1 FIELD STUDIES

Field studies have examined the effects of noise on accidents, productivity, performance of office tasks and cognitive functioning in children. All of these areas show some evidence of detrimental effects of noise, but the data are really too sparse to allow a precise assessment of the crucial exposure parameters or magnitude of the effects.

3.2.2 LABORATORY STUDIES

Laboratory studies can be sub-divided into those which have examined the effects of continuous white noise and those which have concentrated on specific noise sources (e.g. intermittent noise, irrelevant speech, low frequency noise). The effects of continuous noise are generally small and complex. They often depend on the precise nature of the activity being conducted or on other contextual factors. In addition, short exposure to noise may often be arousing, probably due to increases in central noradrenaline. In contrast, the effects of intermittent noise are well-established, with impairments being related to changes in noise levels and to effects at encoding or response, depending on when the noise occurs. Similarly, low frequency noise can reduce alertness and prolonged exposure can lead to the same sort of impairments seen in other low arousal situations (e.g. momentary lapses of attention). Irrelevant speech effects have also been clearly demonstrated and models of the underlying mechanisms are now well-developed. The practical importance of such irrelevant speech effects is also clear, in that impairments can be observed with very low intensities.

3.2.3 COMBINED EFFECTS

One common feature of much of the noise research is that effects will depend on the combination of the type of noise, the task being carried out and characteristics of the person. Indeed, it is clearly important to identify subgroups who may be especially vulnerable or particular states which increase the negative impact of noise. For example, we have demonstrated that noise has no detrimental effect on the simple reaction time of subjects who are healthy, but can produce considerable slowing of responses when the subjects are suffering from upper respiratory tract illnesses.

3.2.4 FUTURE RESEARCH NEEDS

Crucial information on the effects of long-term noise exposure is still required. Similarly, a transactional approach is necessary, in order to allow an examination of the combination of factors which may mediate or moderate the effects of noise on performance. Finally, it is not enough just to describe effects of noise, rather it is necessary to develop the appropriate theoretical framework to make interpretation possible.

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3.3 CARDIOVASCULAR AND FETAL EFFECTS OF NOISE

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3.3.1 INTRODUCTION

This section addresses the evidence for adverse effects of environmental noise on the cardiovascular system and the fetus, from population-based (i.e. epidemiological) literature published in English since the 1980s. Studies show equivocal results. Controversies about the evidence centre around issues of study design, exposure characterisation and appropriate health end-points.

3.3.2 CARDIOVASCULAR EFFECTS

Studies in occupational settings, where noise levels are highest, give some guidance as to important exposure characteristics and personal factors which influence the observed outcomes of environmental noise exposure. A preponderance of this research shows a positive association between noise exposure above 85 dB(A) and hypertension, with prevalence ratios ranging from 1.3 to 3.6 (Idzior-Walus, 1987; Thompson, 1996). Most of the studies are cross-sectional and control for few confounders. They provide little information about exposure characterisation. Sound level measurements were made usually at the workstation, at intermittent times (Wu *et al.*, 1987; Lang *et al.*, 1992; Fogari *et al.*, 1994; Kristal-Boneh *et al.*, 1995); only one study used personal dosimeters (Van Dijk *et al.*, 1987), and in another experts used sound level data to classify jobs as to noise level (Hessel & Sluis-Cremer, 1994). The strongest risks were reported in workers exposed to continuous noise of more than 85 dB(A) for 20–25 years (Verbeek *et al.*, 1987; Lang *et al.*, 1992) or to exceptionally high noise levels (>95 dB(A)) for as few as five years (Zhao *et al.*, 1991). In most of the studies, the level of noise was dichotomized at 85 dB(A). Very few workers were exposed at levels above 90 dB(A), making determination of dose–response relationships difficult. Only one (Zhao *et al.*, 1991) of four studies (Van Dijk *et al.*, 1987; Hessel

& Sluis-Cremer, 1994; Kristal-Boneh *et al.*, 1995) reporting several levels of noise showed an effect on blood pressure. The level of noise at which blood pressure began to increase is difficult to interpret, but a hypertension OR of 1.7 was observed at 96 dB(A) exposure. The individual's response to noise may partially determine whether an adverse health effect is observed (Aro, 1984; Van Dijk *et al.*, 1987; Kristal-Boneh *et al.*, 1995).

Evidence for blood pressure effects from environmental noise exposure is less compelling. While the recent Luebeck study indicated a slightly higher risk of hypertension (OR 1.3) for men living in areas where traffic noise levels were 70 dB(A) or more compared with men from lower noise areas (Herbold *et al.*, 1989), the Bonn traffic noise study (Otten *et al.*, 1988), several studies in the Netherlands (Knipschild & Salle, 1979; Pulles *et al.*, 1988), and the prospective Speedwell and Caerphilly studies (Babisch & Gallacher, 1988) have shown no increase in blood pressure due to noise. Similarly, the IVEM research on aircraft noise has shown no increase in average systolic and diastolic blood pressure among residents closest to airports when interviewing variables were taken into account. (Passchier-Vermeer, 1993).

Findings from the Speedwell and Caerphilly studies show a marginal increase in risk for ischaemic heart disease. After four years, the relative risk for prevalent ischaemic heart disease, when adjusted for nine risk factors, was 1.2 for men exposed to traffic noise levels above 66–70 dB(A) *versus* men exposed at 51–55 dB(A); the predicted incident risk ratio was 1.1 (Babisch *et al.*, 1993a). Preliminary results of the ten year follow-up for the Speedwell sample supported this earlier finding (Babisch *et al.*, 1993b). The Berlin population-based case-control study of men exposed to somewhat higher traffic noise levels (71–80 dB(A) *versus* 51–60 dB(A)) had similar results; the OR for myocardial infarction was 1.3 (Babisch *et al.*, 1994).

Overall, the evidence indicates that daytime traffic or aircraft noise levels greater than 65–70 dB(A) may be associated with a slightly increased risk for ischaemic heart disease. Future studies need to incorporate more precise measurement of exposure (such as room orientation, total time spent in the noisy area), disturbance from noise as well as actual noise level, and risk factors which may confound the associations. For larger magnitudes of effect, individuals exposed at noise levels greater than $L_{eq, day}$ of 70 dB(A) should be studied.

3.3.3 EFFECTS OF NOISE ON THE FETUS

Research on adverse fetal outcomes among pregnant women living or working in noisy environments remains limited. Early studies of anomalies among populations exposed to noise have not been replicated when specific defects of varying aetiologies have been considered and confounding variables have been controlled (Thompson, 1996).

It is not surprising that several airport and industrial noise studies have shown contradictory results when low birthweight was the outcome. Low birthweight includes two conditions, preterm births and intrauterine growth retardation, which are now believed to represent two different aetiologies. To separate these effects, it is necessary to know, with some precision, the timing and dose of exposure during the pregnancy. Since very few of the risk or causal factors are known for these two birthweight entities, confounding resulting from unrecognized factors remains a major problem. The suggested mechanism is that noise contributes to decreased utero-placental blood flow resulting in fetal hypoxia and increased secretion of maternal catecholamine. Recently, a prospective Finnish investigation, using industrial hygienists to assess noise levels of the jobs or industries reported by the pregnant women, revealed an association between noise levels of 85 dB $L_{Aeq, 8hr}$ or higher and intrauterine growth retardation, but not preterm birth (Nurminen & Kurppa, 1989). In contrast, no association was found between birthweight and personal noise exposure of less than 85 dB(A) $L_{eq, 24 hr}$ by Taiwanese researchers. Noise levels were determined with the use of personal dosimeters during the first, second and third trimesters of pregnancy, and ranged from 52.4 to 86.8 dB(A) $L_{eq, 24 hr}$ (Win *et al.*, 1996).

To conclude, evidence of fetal effects of noise remains equivocal. The effects of noise on intrauterine growth needs further study. In addition to the timing and dose of exposure, other stressors that may decrease utero-placental blood flow, and strong risk factors such as shift work must be considered in analyses.

3.3.4 CONCLUSIONS

Findings from occupational noise studies that should be considered in planning environmental noise studies include those outlined below.

- Daily (or continuous) exposures to high noise levels for 5 to 20–25 years duration may be required before blood pressure and other cardiovascular effects are manifested as clinical disease states.

- ❑ Because of changes (arising from regulatory activities) in the levels of noise to which individuals are exposed, populations may not be exposed at noise levels of sufficient intensity and duration to produce non-auditory effects detectable from other general stress effects.
- ❑ Populations exposed at the higher noise levels may not be of sufficient size to allow detection of the weak associations expected under a general stress hypothesis, in particular such populations may be too small to allow dose–response determinations.
- ❑ Confounding and mediating variables influence the results observed; the individual’s response (such as to the noise source) has the potential for being a strong mediating variable in the noise-cardiovascular relationship.
- ❑ More accurate characterisation (such as type, source, intermittency, frequency, duration) of the individual’s noise exposure is needed to reduce misclassification of exposure and strengthen associations that are observed.
- ❑ More information is needed about the impact on non-auditory health effects of removing the individual from the noise for periods of time each day (respite time either through leaving the noise area, or use of hearing protectors or other noise reduction devices).
- ❑ The longitudinal (prospective) design is the best for making causal inferences; to date, longitudinal studies tend to show weaker associations than cross-sectional (prevalence) studies.

Traffic and aircraft noise research indicates that the inferences listed above, drawn from occupational studies, can also be made from environmental noise studies and should be considered in planning future studies. In addition:

- ❑ sleep disturbance due to noise might influence physiologic effects of noise; sleep effects and cardiovascular and other chronic effects need to be studied concurrently; and
- ❑ subjects exposed to noise levels higher than 70 dB(A) $L_{eq, day}$ should be target populations for future study.

Future studies of fetal effects of noise need to:

- ❑ use personal dosimeters to measure the individual's noise exposure;
- ❑ measure exposure of pregnant woman several times throughout the pregnancy;
- ❑ focus on women exposed to very high noise levels, possibly levels greater than 90 dB(A) $L_{eq, day}$;
- ❑ control for other strong risk factors of outcome of interest; and
- ❑ focus on small for gestational age fetal growth, the outcome most likely to show effects, if any exist.

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3.4 NOISE AND MENTAL HEALTH

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Environmental noise causes annoyance and sleep disturbance and interferes with task performance, but does it go beyond this to cause psychiatric disorder? In the last 25 years, studies have related noise exposure, largely from aircraft and road traffic, to psychiatric disorder, measured as psychological symptoms, psychotropic drug use, psychiatric hospital admissions, and questionnaire measurement of psychiatric disorder in the community. Individual symptoms such as headaches, irritability, restless nights and being tense and edgy have been related to high levels of aircraft noise, although these studies may be subject to response bias and the effects of greater social disadvantage in noisy rather than quiet areas (Tarnopolsky *et al.*, 1980). Early studies of associations between aircraft noise and psychiatric hospital admissions have been criticised methodologically, and further comprehensive studies have found, at most, a moderating rather than a causal role for noise on admission rates (Jenkins *et al.*, 1981; Kryter, 1990). However, studies of psychotropic drug use have found that increases in levels of aircraft and traffic noise were accompanied by increases in sedative and sleeping pill use (Knipschild & Oudshoorn, 1977; Lercher, 1996). Noise level has not been associated with psychiatric disorder measured by questionnaire in cross-sectional population studies of aircraft noise (Tarnopolsky & Morton-Williams, 1980). Road traffic noise studies have shown mixed results: no association has been found with psychiatric disorder, but associations with mental health symptoms and a small association between noise and anxiety levels have been observed (Halpern, 1995; Stansfeld *et al.*, 1996). If there are few overall effects of environmental noise on the psychological health of the population, it may be that there are certain groups who are more vulnerable to noise, particularly children, the elderly and people with existing illness. Future studies need to take into account factors of vulnerability to noise and should study populations newly exposed to noise, using longitudinal designs and more detailed measures of psychiatric disorder. At the same time there needs to be better measurement of noise exposure in these studies. The effect of noise on health also needs to be considered in the context of other stressors. Perceived threat from noise and ways of coping with noise should also be measured.

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3.5 THE EFFECTS OF SHORT-TERM AND LONG-TERM NOISE EXPOSURE ON CHILDREN

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3.5.1 SHORT-TERM NOISE EXPOSURE

In a series of classroom experiments (Hygge, 1997), the effects of different noise sources at different noise levels on long-term recall and recognition in children aged 12–14 years have been studied. In the first four experiments, aircraft, road traffic, train or verbal noise were played back at 66 dB(A) (peak levels 76 dB(A) fast) to each class. In the next two experiments aircraft noise or road traffic noise were played back at 55 dB(A) L_{eq} (peak levels 66 dB(A) fast). In the last two experiments train and aircraft noise were combined into the same session, with one or the other as the more frequent source, and played back at 66 dB(A) L_{eq} .

At 66 dB(A) L_{eq} aircraft and road traffic noise impaired recall, but train noise and verbal noise did not. At 55 dB(A) L_{eq} aircraft noise again impaired recall but road traffic noise did not. For the two combinations of aircraft and train noise at 66 dB(A) L_{eq} , recall was impaired by the train dominated noise. The noise effects did not interact with learning ability and there were no general noise effects on long-term recognition.

3.5.2 LONG-TERM NOISE EXPOSURE

In parallel to the classroom studies, children around the new and old airports in Munich have been studied with a longitudinal, prospective design (Evans *et al.*, 1995; Hygge *et al.*, 1996). Psychophysiological, cognitive, motivational, and quality of life measures were taken once before and twice after the shutdown of the former Munich International Airport in May 1992 and the inauguration of the current one at the same time. One of the dependent measures in the Munich

study was a long-term recall test, mapped on the very same recall test in the classroom studies.

Children who had long-term exposure to aircraft noise at the old airport, before the shutdown, showed worse long-term recall than their sociodemographically matched counterparts from quieter areas. Two years later, after the shutdown of the airport, the relative disadvantage of the formerly aircraft noise-exposed group disappeared. At the new airport before the opening, there was no initial difference in recall between those who were about to experience exposure to aircraft noise and their matched controls. However, two years later, the group exposed to aircraft noise showed impaired recall relative to their controls.

Thus, for long-term recall, the aircraft noise effects at the old airport waned off when it was shut down. At the new airport the validity of the long-term recall measure was conceptually replicated by showing a progressive deterioration after the airport was brought into operation.

On a difficult word-list, the same pattern of effects as for long-term memory was found at both airports. This indicates that language acquisition and mastery are also adversely affected by noise effects.

The sensitivity and validity of the long-term recall measure both to short-term aircraft noise exposure in the classroom and to the termination and onset of long-term exposure to aircraft noise in Munich, together with the many different tests and response systems involved in the three measurement waves, sets an empirical platform for addressing some theoretically important issues. For example:

- are the noise effects stronger with some cognitive measures than others, and if so why; and
- is the effect of noise on cognition paralleled in other response systems, and if so is the connection causal?

Some aspects of these issues have been analysed in data arising from these studies; others remain to be tested statistically. Basic findings from the statistical analyses done so far are that cognitive tasks relying on central processing involving language understanding, reading and memory are more vulnerable to noise (cf Evans & Lepore, 1993), and that the time-pattern of such cognitive effects is paralleled by concomitant changes in stress hormones and blood pressure.

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3.6 THE HEALTH COUNCIL OF THE NETHERLANDS REPORT

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In 1994 an international committee of the Health Council of the Netherlands prepared a report in which the results of scientific research into the effects of environmental, occupational and recreational noise on health were reviewed (e.g. Smoorenburg *et al.*, 1996). The report evaluates the evidence for causal relationships between exposure to noise and specific effects on health. The committee rated, on the basis of the results of epidemiological surveys, the evidence of a causal relationship between noise exposure and an effect according to four categories: sufficient, limited, inadequate and lack of evidence. For the health effects with sufficient evidence of a causal relation with noise exposure, special emphasis was placed on exposure–effect relations and on observation thresholds. (The observation threshold is the lowest exposure value at which, for an average population, an effect from exposure to noise is observed in epidemiological surveys). Table 3.6.1 gives the results for environmental noise exposures.

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Table 3.6.1 Health Council of the Netherlands classification of the evidence for non-auditory effects of noise*

Effect	Classification of evidence	Observation threshold measure value in dB(A)	
Hearing loss	Sufficient	$L_{Aeq,24h}$	70 (indoors)
Hypertension	Sufficient	$L_{Aeq,06-22h}$	70
Ischaemic heart disease	Sufficient	$L_{Aeq,06-22h}$	70
Hormonal effects	Limited		
Immune effects	Limited		
Birthweight	Limited		
Congenital effects	Lack		
Psychiatric disorders	Limited		
Annoyance	Sufficient	$L_{Aeq,24h}$	40 (transportation noise)
Psychosocial well-being	Limited		
Performance schoolchildren	Sufficient	L_{school}	70
Sleep disturbance:			
Sleep pattern	Sufficient		?
Awakening	Sufficient	SEL	60 (indoors)
Sleep stages	Sufficient	SEL	35 (indoors)
Sleep quality	Sufficient	$L_{Aeq,night}$	40
Heart frequency	Sufficient	SEL	40 (indoors)
Hormones	Limited		
Immune system	Inadequate		
Mood next day	Sufficient	$L_{Aeq,night}$	<60
Performance next day	Limited		

*classification of long-term effects of exposure to environmental noise, with evidence of a causal relationship and data on the observation threshold (determined outdoors, unless otherwise specified)

4 Evaluation of the evidence for the non-auditory effects of noise exposure

4.1 EVALUATION

EXPOSURE TO ENVIRONMENTAL NOISE

The effects of environmental noise on health and well-being are considerably influenced by the source of the noise. There are many different potential sources, such as traffic noise, neighbour noise, noise from hobbies, noise in the home and occupational noise, which combine and contribute to the total noise exposure for a particular individual at a particular time. Human response to noise depends on the type of noise, the situation, background levels of noise, the general environment, and so on. Furthermore, the personal significance of the noise to an individual may have a greater influence than the amplitude (loudness) of the noise on the response of that individual. This may be particularly true of noise that disturbs sleep. In REM sleep an individual's response to noise is less affected by amplitude than by context, for example whispering a name of particular significance may have a bigger impact than clapping hands.

In health effects studies reported to date, characterisation of noise has sometimes been poor. Future research needs to be directed to better sound characterisation, as is done, for example, in laboratory-based acoustics research. Ideally sound characterisation should be carried out (preferably at the individual level) at the same time that health outcomes are investigated. Good sound characterisation includes not just noise level, but also details about the noise.

Establishing an objective indicator of annoyance caused by noise is difficult. Better characterisation of noise should help define what causes annoyance. An example would be annoyance due to traffic noise from different kinds of road surface, where the same level of noise from different surfaces can elicit quite different responses. Non-acoustical variables, which in the case of annoyance caused by aircraft, for example, might include fear of crashes and a perception of a lack of concern by operators, may also influence the degree of annoyance that is felt. The period of exposure, whether over months or years, may also play a part; though some habituation may occur, it is also possible that uneasiness increases as the noise exposure continues.

It is not possible to identify one single measure to quantify noise levels. An important subject for future research is investigation of the most appropriate

metric(s) against which to assess potential health effects; the appropriate measure may vary according to the response being evaluated.

Although outdoor noise level is probably one of the least important variables for response to noise, it is the exposure variable that has been most studied. This hampers any attempt to derive safe or unacceptable noise level limits. Outdoor noise levels are, nonetheless, important for policy making, as it is often only outdoor exposure that can be readily measured and controlled.

Noise itself may in fact be an important and useful proxy for other environmental problems. In some studies where noise level is an input variable it may not be the most important variable in the study. In the case of road traffic noise exposure, the most appropriate input variable may be distance from a main road, and this may affect air pollution, anxiety about children playing outside, and so on. Any observed effects cannot, therefore, be assumed to be due to differences in noise levels alone. It is very difficult, and may be impossible, to distinguish between such correlated input variables in practical research study designs. This is a major problem for future research.

STUDIES ON ADVERSE HEALTH EFFECTS

In general terms, attempts to evaluate the adverse health effects alleged to be associated with environmental noise are hampered by the fact that many of the effects reported are weak effects occurring after long-term exposure. Nonetheless, even a weak effect may be important in public health terms if it affects the whole population.

Environmental noise (which is low-level noise) does not cause direct damage to the tissues because the levels of physical energy are too low. Any adverse effects are therefore generally indirect, resulting from the way that the body responds to noise, rather than being directly caused by the noise itself. It is difficult to classify normal acute effects, such as elevated heart rate during acoustic startle or disturbance to sleep pattern without awakening, as being directly harmful unless they lead to chronic consequences.

The links between acute and chronic effects of environmental noise are not well understood. Some studies have investigated statistical associations without attempting to explain possible causative mechanisms. This is an important gap in the existing knowledge base.

As in many other areas, a distinction must be drawn between research conducted under controlled laboratory conditions and research conducted in the field. In experimental studies, confounding factors can be controlled and exposure can be exaggerated in order to develop dose–response relationships, even though such exposures and the resulting effects may not be observable in the field. Such laboratory studies are essential to establish mechanisms of effect, and as the knowledge of underlying mechanisms improves, the scientific basis for planning control strategies becomes more robust.

The most informative data on non-auditory effects of noise can be expected to come from longitudinal field studies investigating changes in environmental noise levels, such as, for example, the Munich airport study (see Section 3.5.2). Such opportunities occur only rarely and every effort should be made to conduct epidemiological studies when these situations do occur. One such possibility might be to study the impact of the Channel Tunnel rail link, in terms of the relative impacts of road traffic and railway noise. Other future airport developments will also provide good opportunities for study.

Cross-sectional studies are generally less informative than longitudinal studies because the choice of sample in the former may lead to only very specific information being gathered, which cannot be generalised satisfactorily to the population as a whole. Furthermore, depending on the numbers of areas studied and the often limited range of average exposure, cross-sectional studies may give only limited information on dose–response relationships.

SUSCEPTIBLE GROUPS

A number of approaches may be used to assess the impact of environmental noise on health. One is to concentrate on effects for which there is evidence for a causal association. This has been the approach of the Health Council of the Netherlands (see Section 3.6). Another possibility is to focus on susceptible groups, both because it may be easier to identify effects in such groups, and because they may represent the sector of the population that most needs to be protected (see below). Obvious candidate groups are the elderly and children.

There is no real reason to suppose that children or old people in general are more sensitive to noise effects than the general population, though they may be more vulnerable because of factors such as lifestyle, which may affect exposure or response. Nonetheless it is plausible to consider that certain individuals may be

more sensitive than others, and these also should be investigated where they can be identified, for example people who are especially reactive to noise (preferably as assessed by some objective measure).

STUDIES ON SLEEP

It has been suggested (Pearsons *et al.*, 1990) that in general the relatively few field studies that have been conducted show a more consistent relationship between adverse health effects and noise than do the more numerous laboratory studies. In fact, field studies seem to demonstrate a smaller effect of noise than do experimental studies. It has been suggested that this may be a result of not comparing similar types of exposure in the two types of study, for example indoor exposure levels in a field study compared with outdoor exposure levels in an experimental study. However, when the data are corrected to take this into account, (e.g. Smoorenburg *et al.*, 1996), field studies still demonstrate less of an effect than laboratory studies. However, in real life situations habituation to factors that disturb sleep may occur, whereas experimental studies of one night's duration cannot take habituation into account.

Hearing is not suspended during sleep. Normal physiological responses to sound, such as increased heart rate, will therefore still occur during sleep and not habituate. This kind of effect is of no clinical importance.

The best measures of insufficient or disturbed sleep are sleepiness in the day time and the number of awakenings at night. Tiredness and sleepiness are not the same and should not be confused, especially when investigating sleep disturbances in depressed individuals. A feature of most depressive/anxiety disorders is sleep disturbance coupled with a feeling of tiredness during the day time. Depressed people may seek a cause, other than their illness, for their night time waking and daytime tiredness and may presume that the cause is noise exposure.

There is some debate as to whether disturbances that change sleep patterns (or sleep latency), in particular the amount of time spent in REM sleep (dreaming), have the greatest effect on sleep quality, or whether undisturbed total sleep is the most important aspect of good sleep quality.

PSYCHIATRIC DISORDERS

The available evidence suggests noise has only a slight impact on psychiatric disorders. When studying the impact of environmental noise, it may be easier to investigate the social perception of noise rather than monitoring effects such as anxiety.

ANNOYANCE

Annoyance caused by noise is a major source of complaint to local environmental health offices. That people are annoyed by a wide range of external noise events is beyond dispute. Many factors may contribute to annoyance from noise, including lack of control of the situation, frequency of the occurrence and time of day, a perception that the activity causing the noise is unnecessary, and neighbour noise perceived by the complainant to be inappropriate (e.g. loud music, dogs). Not only is annoyance a source of complaint in itself, there are also implied links between noise, leading to annoyance and stress, and cardiovascular and mental health effects (see Sections 2.4 and 2.7).

POLICIES FOR CONTROL OF ENVIRONMENTAL NOISE

Although environmental (but not occupational) standards for air pollution, for example, are often aimed at the protection of the more sensitive groups (such as those suffering from respiratory diseases), this has not usually been the situation when setting noise standards. In fact, identification of the appropriate susceptible groups to protect from environmental noise may be difficult. There is evidence to suggest that the psychiatrically disturbed may be one such group. Nonetheless, when establishing control policies, although it cannot be expected that the most sensitive individual can necessarily be protected, the goal should be to offer a reasonable level of protection to susceptible groups. Such an approach will automatically protect the 'normal' population. In order to institute effective control policies there is a need to establish rigorous scientific evidence for the prevalence of certain effects in noise exposed populations.

4.2 SUMMARY

Table 4.2.1 summarises the discussions at the workshop on the strength of the evidence for causal associations between environmental noise and each of the adverse health effects reviewed in Sections 2 and 3, and the table provides a basis for future debate. The general structure of the table is based on a similar table prepared by the Health Council of the Netherlands (see Section 3.6), which was prepared following an in-depth review. The evaluations presented here, though in general agreement with those of the Health Council of the Netherlands, are somewhat different. Where possible, an indication is given of the noise level at which observable effects might be expected, though as noted above (Section 4.1) it was considered that it is not possible to identify a single measure to quantify noise exposure. The table also indicates which subgroups of the population are most likely to be affected by the various health impacts described.

The evidence for effects of environmental noise is strongest for annoyance, sleep disturbance (onset, latency, awakening during the night and premature awakening in the morning, subjective sleep quality, mood next day), ischaemic heart disease and performance by schoolchildren. The available data on other possible health consequences, such as low birthweight and psychiatric disorders are currently inconclusive.

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Table 4.2.1 Chronic effects of exposure to environmental noise

Effect	Classification	Guideline value dB(A)	Population most affected	Importance of effect	Comments
Sleep onset/latency	S		Probably young adults		
Waking during night	S	60 (SEL : indoors)	Sensitive groups e.g. anxious, depressed		Character of noise is important, effect depends on which sleep stage reached when noise occurs
Waking prematurely in the morning	S	<60? (SEL), depends on background noise	Sensitive groups e.g. anxious, depressed	+	Not known how important noise is <i>per se</i>
Subjective sleep quality (reported)	S	40 ($L_{Aeq,night}$)		+	Character of noise is important
Mood next day (depression and irritability)	S	<60($L_{Aeq,night}$)		+	Character of noise is important
Sleepiness and performance next day	I			+	E.g. assessed by reaction time
Hypertension	I		Family history of hypertension	+	Hypertension is strong risk factor for heart disease
Ischaemic heart disease	S	70(L_{Aeq} : outdoors)			Night shift workers have elevated risk of heart disease
Birthweight	I			+	
Psychiatric disorders	I			+	
Annoyance	S	40($L_{Aeq,24h}$; transportation noise)	Self-reported noise-sensitive individuals	+	Depending on its character, even some low level noise may be a problem
Performance by schoolchildren	S	65($L_{-school}$)	Pupils with learning disabilities, hearing impairment, English as a second language	+	Could be large differences depending on the source, temporal variation may be more important than level

S, there is sufficient evidence for a causal association between noise exposure and the health end-point

I, the evidence for a causal association between noise exposure and the health end-point is inconclusive

SEL, sound exposure level

+ indicates a particularly important effect

5 Recommendations for future research

Developed from an original paper by B Berry, National Physics Laboratory, Teddington, Middlesex, and incorporating workshop discussions

5.1 INTRODUCTION

Taking into account the reviews of the various non-auditory health effects of environmental noise presented in Sections 2 and 3, and the general discussion of some of the areas of concern given in Section 4, it is clear that there are unresolved questions that should be addressed by additional research initiatives. These are summarised below (Section 5.2).

No attempt has been made at this time to set priorities for the new research suggested. Further consideration is needed before such priorities can be established. The highest priority should be given to areas of research which provide a sound scientific basis for national noise standards and which underpin locally developed targets for different areas. In particular research likely to provide information pertinent to the debate on the expected beneficial *versus* adverse impact of noise control should be encouraged.

A number of outstanding methodological issues, which will need to be addressed in any future research, are outlined below.

RECOMMENDATIONS

- ❑ The adverse health effects of noise other than annoyance tend to be quite weak. Either only a small minority of the population is affected (if at all) or the average effects over the population as a whole seem to be small. This affects the design of research studies.
- ❑ The precise biological mechanisms by which noise might contribute to chronic health effects are not clear. This causes difficulties in establishing cause and effect.
- ❑ In field studies, it is difficult to distinguish the effects of noise exposure per se from the effects of other correlated and confounding variables. It is relatively easy to resolve the issue of confounding variables in controlled laboratory studies, but it can be difficult to translate the results of such studies to real life situations.

For epidemiological studies investigating the chronic effects of noise exposure, a longitudinal approach is generally preferable. In all such studies an adequate characterisation of noise level, character and source, and duration of exposure is of paramount importance. Many of the studies proposed below could be linked with an assessment both of the effects of noise in relation to the effects of other stressors on one or more health end-points, and of the combined effects of noise and other stressors. Such studies will require good measures to quantify stressors other than noise and careful consideration of confounding factors.

5.2 RESEARCH REQUIREMENTS

MEASUREMENTS OF EXPOSURE AND EFFECT

Greater attention should be paid to the need for standardisation of measures of effects and measures of noise exposure. More work on noise characterisation could possibly be incorporated into ongoing studies such as the Speedwell and Caerphilly (see Sections 2.4.4 and 3.3.2) studies. A secondary analysis of noise exposure might also be possible in some ongoing studies of other factors disturbing sleep. Future research on noise exposure should be directed to:

- ❑ better sound characterisation (noise level, noise source, nature of noise, e.g. barking, music, traffic noise etc.), ideally carried out (preferably at the individual level) at the same time that health outcomes are investigated;
- ❑ investigation of the best measure(s) that should be used to quantify different kinds of noise associated with different health outcomes, taking into account the consideration that it is not possible to identify one single parameter for quantifying noise levels;
- ❑ studies on links between acute and chronic effects of noise exposure.

SLEEP

As noted above, longitudinal studies are far more informative than cross-sectional studies, and should, therefore, be encouraged. However, it is recognised that longitudinal studies are likely to be costly (e.g. £7–8 million for a comprehensive study). It would also be beneficial to combine studies of the effects of noise with those of other factors that disturb sleep in order to establish the relative importance of noise, although such studies would require large numbers of subjects. The following studies are particularly recommended:

- ❑ longitudinal studies of the impact of changes in noise exposure, paying particular attention to the possibility of investigating ‘natural experiments’ such as the re-siting of airports or the Channel Tunnel, or populations such as shift workers;
- ❑ studies on time taken to fall asleep, effects of noise during sleep, premature awakening and after-effects of noise-disturbed sleep such as performance next day, sleepiness the next day and psychosocial well-being;
- ❑ intervention studies on measures taken to counter the effects of noise;
- ❑ studies on susceptible groups and sensitive individuals (if they can be identified by some objective physiological measure, the establishment of which would require preliminary laboratory studies) — studies should concentrate on groups where there are a high percentage of complaints or high levels of exposure or both;

RECOMMENDATIONS

- ❑ studies on noise sources other than aircraft and on noise from combinations of sources.

PERFORMANCE

In assessing performance effects there is a need not only to describe the effects caused by noise, but also to develop a theoretical framework to enable the interpretation of different types of study. This will require precise descriptions of the performance tasks being assessed. The widest possible population range should be studied. Suggested studies are:

- ❑ investigation of the effects of irrelevant speech, in particular what type of work task is impaired;
- ❑ comparisons of laboratory and field studies;
- ❑ the effect of background music on performance;
- ❑ longer-term studies in occupational settings.

HYPERTENSION

As any effect would be expected to be very small, a very large study would be needed to investigate the influence of environmental noise on hypertension. It is therefore recommended that the effect of noise on hypertension should first be investigated by longitudinal occupational studies. Should studies of environmental noise then be justified, it would be easier to study, for example, road traffic noise than aircraft noise, as a larger population would be likely to be affected.

ISCHAEMIC HEART DISEASE

Again, though the high cost is likely to be a limiting factor, longitudinal studies are preferable, with careful measurement of duration and intensity of individual exposure, and a more comprehensive adjustment for potential confounding factors than has been the case in many studies reported to date. Although case-control studies of patients with ischaemic heart disease can be informative,

there will often be difficulties in characterising noise exposure. Cross-sectional studies are unlikely to be informative. Specific studies should investigate:

- ❑ people with a family history of hypertension;
- ❑ the role of annoyance and coping strategies, which could affect the risk for ischaemic heart disease.

FETAL EFFECTS

Characterisation of noise exposure, including level of noise and duration of exposure is particularly important and should be an essential feature of all future studies. All studies should give adequate consideration to confounding risk factors. The following studies are recommended:

- ❑ studies of intrauterine growth retardation specifically and separately from low birthweight and prematurity, with personal dosimetry throughout pregnancy if possible and control for other stressors and other strong risk factors;
- ❑ multi-centre studies of pregnancy outcome, with pregnant mothers stratified according to noise dosimetry;
- ❑ studies of pregnancy outcome in mothers exposed to $L_{Aeq} >70$ dB and $L_{Aeq} >90$ dB — these would necessarily have to be occupational studies;
- ❑ evaluation of the Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC) study to see if noise exposure during pregnancy can be estimated.

ENDOCRINE AND IMMUNE RESPONSES

Longitudinal studies with adequate control of potential confounding factors should be encouraged. The effects of noise on the hormonal and immune systems may best be studied by considering noise as one of the possible causative factors in ongoing studies investigating such health impacts. Other recommended studies include investigation of:

RECOMMENDATIONS

- ❑ changes (increases and decreases) in hormone levels in association with noise exposure and the associations between noise exposure, performance and hormone levels;
- ❑ the effects of noise on immune system function;
- ❑ hormone levels as markers of effect in studies of noise and the cardiovascular system.

PSYCHIATRIC DISORDERS

Longitudinal studies looking at changes in noise exposure (which should be well characterised) will be the most informative. There is also a need for the development of more detailed and comprehensive measures of psychiatric disorders, including a wider range of outcome measures than has been the case to date. Particularly informative would be studies to investigate the relative role of noise in comparison with other environmental stressors and coping strategies. Recommended studies are:

- ❑ an investigation of the effects on health of living in close proximity to a busy road, including an investigation of the relative influences of road traffic noise, safety, air pollution, road surface, coping strategies (such as closing windows, living to the rear of the house) etc;
- ❑ longitudinal studies of populations recently exposed to a new noise source;
- ❑ a case-control study of prescriptions issued for treatment of anxiety disorders, with a retrospective assessment of noise exposure;
- ❑ studies on the relative effects of noise and other factors, such as housing, in causing anxiety disorders.

ANNOYANCE

As already mentioned, sound characterisation is particularly important to enable the determination of what kinds of noise are particularly annoying. As studies of

systematic changes in noise character are difficult to conduct in the field, there will be a role here for laboratory studies. The following studies are recommended:

- ❑ studies on annoyance in combination with studies on coping strategies;
- ❑ studies on the link between annoyance and stress, including investigation of the extent to which behavioural impacts lead to physical health effects such as stress;
- ❑ additional laboratory studies on the effects of systematic changes in noises of different types;
- ❑ further investigations into how readily individuals can discriminate between different noise sources.

CHILDREN

As an adjunct to longitudinal studies on performance and physiological effects such as hypertension, laboratory studies could be used to investigate more specific effects such as the type of memory that is most affected by noise. Epidemiological studies should be used to investigate effects in large numbers of children, over a range of schools, and should include studies on particularly vulnerable groups within the child population. Experimental studies would be less expensive than epidemiological studies and would involve a far more homogeneous group than is normally the case in general population studies. In both epidemiological and laboratory studies effects should be investigated at different age levels. Recommended studies include:

- ❑ investigation of the effects of noise on blood pressure in children;
- ❑ studies of the effects of noise on speech perception;
- ❑ experimental studies of effects on memory, including intervention studies on counter measures such as insulating classrooms;
- ❑ an investigation to determine whether some cognitive measures are more affected by noise than others;

RECOMMENDATIONS

- studies to determine whether noise effects on cognition are paralleled by effects in other response systems, such as stress hormones and blood pressure, and if so whether there is a causal connection.

6 Overall conclusions

A number of uncertainties remain about the non-auditory effects of noise; nonetheless, some conclusions have been reached. The evidence for effects of environmental noise on health is strongest for annoyance, sleep disturbance (onset, latency, awakening during the night and premature awakening in the morning, subjective sleep quality, mood next day), ischaemic heart disease and performance by school children. The available data on other possible health consequences, such as low birthweight and psychiatric disorders are inconclusive.

Further research to clarify the effects of environmental noise on non-auditory health should include better characterisation of noise exposure and source, and better measurement of health outcomes.

The types of studies to be encouraged include longitudinal studies concentrating particularly on susceptible groups. Studies of natural experiments (such as changes in the siting of an airport) are likely to be particularly informative, as are intervention studies. Studies to investigate chronic health effects are also important, likewise the identification of particularly sensitive individuals. In epidemiological studies, careful consideration should be given to confounding factors and effect modifiers in the association between noise and health. Emphasis should be given to studies combining other environmental stressors with noise.

In general, field studies are expected to be more informative than laboratory studies, although laboratory studies will be necessary to clarify specific causal hypotheses and to define dose–response relationships. Field studies are likely to be expensive, though studies on children in school could be particularly cost-effective.

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Acknowledgements

Peer review of the final document was provided by S Benton, B Berry, J Crook, I Flindell, C Jones, R Maynard and S Stansfeld

Editing by L Shuker

Typesetting by P Forster (IEH)

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