Health Canada Santé Canada

Noise from Civilian Aircraft in the Vicinity of Airports – Implications for Human Health

I. Noise, Stress and Cardiovascular Disease

Canada

Noise from Civilian Aircraft in the Vicinity of Airports – Implications for Human Health

I. Noise, Stress and Cardiovascular Disease

Our mission is to help the people of Canada
maintain and improve their health.

Health Canada

Consumer and Clinical Radiation Protection Bureau
Healthy Environments and Consumer Safety Branch

Published by authority of the
Minister of Health

Également disponible en français sous le titre
Le bruit des avions civils à proximité des aéroports – effets sur la santé humaine
I. Bruit, stress et maladies cardio-vasculaires

This publication can be made available in/on (computer diskette/large print/audio-cassette/braille) upon request.

Copies of this report can be obtained from:
Consumer and Clinical Radiation Protection Bureau
Health Canada
Address locator: 6302C
775 Brookfield Road
Ottawa, Ontario K1A 1C1
Fax: (613) 952-7584
or from our Web site :
http://www.hc-sc.gc.ca/rpb

Cat. H46-2/01-256E
ISBN 0-662-30975-8

01-HECS-256

Table of Contents

Acknowledgements

This review was prepared by the following staff at Health Canada: Stephen Bly (coordinator and noise exposure reviewer), Dr. Slavica Vlahovich (health effects reviewer), Jack Mclean (stress and biological effects reviewer) and Sabit Cakmak (statistical analysis reviewer). Health Canada reviewers of this document were Bernard Choi and James McNamee. Tom Lowrey reviewed the document for Transport Canada.

1. Introduction

The purpose of this report is to provide detailed arguments and conclusions on the potential for high levels of aircraft noise to be linked to stress and cardiovascular disease. The analysis was prompted by several factors.

1. A link between cardiovascular effects and high levels of aircraft noise could not be excluded. There were significant discrepancies between several comprehensive reviews and policy statements on the subject. For example, reports of the Health Council of the Netherlands (Passchier-Vermeer, 1993; HCN, 1994; HCN, 1999) indicated that there was sufficient evidence of a causal relationship between ischemic heart disease and noise and between hypertension and noise, for 24 hour time-averaged noise levels exceeding 70 dBA outdoors. The report of the Institute for Environmental Health (IEH, 1997) indicated that there was sufficient evidence of a causal relationship between only ischemic heart disease at levels exceeding 70 dBA outdoors but the effect was not noted as being particularly important. The recent World Health Organization (WHO) guidelines for community noise (WHO, 1999) concluded that cardiovascular effects are associated with long-term exposure to 24 hour time-averaged noise levels above 65 dBA but that the associations are weak.

2. Chronic noise exposure has the potential for important public health consequences. Population exposure to transportation noise and the prevalence of cardiovascular disease in Canada suggest that this type of noise exposure has the potential to be a significant public health problem. It is estimated that about 2 million Canadians live in areas where road traffic noise exceeds 24 hour time-averaged outdoor levels of 65 dBA and as many as 50,000 live in areas where air traffic noise levels exceeds 65 dBA. Given that high blood pressure and heart disease are the 2nd and 6th most prevalent chronic diseases in Canada, respectively, there is the potential for transportation noise to have important consequences to public health.

3. Aircraft noise is a more highly annoying source of noise than road traffic noise and community groups in Canada have expressed significant concerns about the potential health effects of aircraft noise in the vicinity of airports. Of particular concern has been a recent longitudinal scientific study in Munich on the potential for physiological indications of chronic stress, particularly those relating to increases in blood pressure and stress hormone levels, in elementary school children living in areas exposed to aircraft noise. Theoretically, blood pressure and stress hormone effects could relate to long term effects on cardiovascular health.

4. Airport noise management committees and Transport Canada's Aircraft Noise and Emissions Committee require timely and reliable information on the health effects of noise so that they can be factored into decisions concerning airport and aircraft operation and regulation, as well as international aircraft noise policies affecting the Canadian air industry and air safety.

2. Methods

To examine the most relevant studies, optimize resources and provide a timely review of the subject, this analysis was limited to the evidence for cardiovascular biological and health effects of community aircraft noise around major civilian airports. As a result of the arguments described in the Introduction section, endpoints considered in the analysis for childhood studies were resting blood pressure and stress hormone levels. In adult populations, the endpoints were hypertension and ischemic heart disease. Other endpoints of interest such as sleep disturbance, annoyance, mental health and potential effects on learning in children will be dealt with in future "It's Your Health" publications and any required reports.

The report was based on examination of relevant English language reviews, journal and conference papers, published from 1975 to 1999. Peer reviewed papers were given more weight in formulating the conclusions. Results from a limited number of traffic noise studies and one aircraft noise paper around a military airport were also included because of the limited data on the potential for adverse cardiovascular effects from aircraft noise and because of the public health importance of traffic noise.

Noise from low flying military training flights was excluded because of the considerably different character of the noise compared to civilian airport flights.

Occupational studies were excluded from the review because they could only be used to assess the plausibility of cardiovascular disease hypotheses for environmental noise research, but not to draw conclusions about the effects. Most occupational noise studies do find increases in blood pressure, but this effect often disappears when adjustments are made for confounding factors. Unfortunately, many studies do not adequately correct for potential confounding factors present in the work environment (Stansfeld and Haines, 1997; Thompson, 1997). These results make it difficult to extrapolate the findings of occupational noise exposures to conclusions about the effects of environmental noise exposure.

3. Discussion and Conclusions

3.1 Stress

There is evidence that acute noise exposure can cause temporary elevations in heart rate, as well as increases in peripheral vasoconstriction and blood pressure (WHO, 1999; Passchier-Vermeer, 1993; HCN, 1994; Berglund and Lindvall, 1995; IEH, 1997). The evidence for effects of chronic noise exposure, however, is not consistent across studies. Studies of laboratory animals do suggest persistent elevation of blood pressure, but human laboratory studies are less consistent (Stansfeld and Haines, 1997).

Noise can be one of many environmental stressors. It does not elicit a unique stress response. The stress response is an adaptation or coping mechanism that occurs when the brain perceives experiences or challenges as threats. It is associated with secretion of the stress hormones, such as epinephrine, norepinephrine and cortisol, and changes in heart rate and blood pressure level. Normally, these return to baseline levels when the individual adapts or the experience(s) end (McEwen, 1998). These physiological changes are widely accepted as 'biomarkers' of stress (Frankenhauser, 1986; Scheuch, 1986) and represent a generalized response to any non-specific stressor, such as noise.

Two factors largely govern individual stress responses: (i) the magnitude of the perceived threat or challenge; and (ii) the individual's general state of physical health, which largely depends on genetic factors and one's developmental history, experiences, and behavioural and lifestyle choices (Lazarus and Folkman, 1984; McEwen, 1998).

It has been hypothesized that stress hormone levels and blood pressure may remain elevated as a result of frequent or excessive stress in susceptible individuals. The source of such stress can range from daily hassles to traumatic life events (McEwen, 1993, 1998; Rosmond, *et al*., 1998).

If the release of the stress hormones is sustained or excessive, the functional integrity of many organs and tissues can be compromised in susceptible individuals (Chrousos and Gold, 1998). Sustained release of cortisol has been associated with elevated blood pressure, depression, osteoporosis, immunosuppression, insulin resistance, visceral obesity, and the excessive stimulation of the amygdala, the fear center in the brain (Chrousos and Gold, 1998; McEwen, 1998; Tsigos and Chrousos, 1996; Friedman, *et al*., 1996). High levels of cortisol can also damage neurons in the hippocampus, an integral part of a negative feedback system that is responsible for returning cortisol levels to normal (McEwen, 1998).

Chronic stress can also have adverse effects on health if the behavioural response to perceived challenges or threats leads to harmful behaviours such as social isolation, aggression, and resorting to the excessive consumption of alcohol, tobacco, food and drugs (McEwen, 1998).

3.1.1 Stress – Conclusions

Noise can act as a short term stressor and has the potential, in susceptible individuals, to cause chronic physiological effects such as elevated blood pressure and stress hormone levels. There is evidence to suggest that physiological effects arising from chronic stress, as well as harmful behaviours, may exacerbate a variety of mental and physical adverse health effects such as cardiovascular disease, depression, osteoporosis, susceptibility to infections and diabetes (via insulin resistance).

3.2 Stress Related Physiological Effects in Children

Several epidemiological studies have examined whether stress related physiological effects in children were associated with exposure to aircraft noise. The endpoints studied were resting blood pressure and stress hormone levels. The detailed reviews and conclusions are provided in sections 3.2.1 to 3.2.4 below. The overall findings can be summarized as follows. The recent Munich airport study (Evans, *et al.*, 1995; Evans, *et al.*, 1998; Hygge, *et al.*, 1998), particularly because of its longitudinal design, has provided the strongest evidence for an association between aircraft noise and physiological effects, especially an increase in epinephrine and norepinephrine (catecholamine) levels. However, there are too few studies to

provide conclusive evidence of a cause and effect relationship between aircraft noise and physiological effects. Also, for the few studies that have been done: (i) the characterization of the noise exposure was sometimes difficult to interpret; (ii) associations were not consistently found; and (iii) there was a lack of controls for potentially important confounders. These findings cast doubt as to whether some factor other than aircraft noise was responsible for the observed differences between exposed and control populations.

3.2.1 Noise-induced Blood Pressure Effects

The systolic and diastolic blood pressures in children living in high noise areas around the Los Angeles (Cohen, *et al.*, 1980; Cohen, *et al.*, 1981), Munich (Evans, *et al.*, 1995; Evans, *et al.*, 1998; Hygge, *et al.*, 1998,) and Sydney airports (Morrell, *et al.*, 1998) were compared to those of children living in low aircraft noise areas.

The Los Angeles airport study used a matched group design, where matching was statistically successful for grade level and socioeconomic status. There were 262 subjects, 142 in the noisy area and 120 in the quiet control area. The study was longitudinal in design but only initial findings and 1year follow-up results were published. Additional statistical controls were applied using regression techniques for the confounding factors of racial distribution, known to have a significant effect on blood pressure, mobility (amount of time lived in the area prior to the endpoint measurements), and ponderosity (ratio of weight to height). This study utilized audiometric screening and controlled environments for the blood pressure measurements. The noise was described as yielding peak sound level readings in the school of 95 dBA in an air corridor with over 300 flights per day. Noise levels were not stated for the control group.

A statistically significant increase of 3 mm Hg in both systolic and diastolic blood pressure was observed in the noise-exposed group initially (Cohen, *et al.*, 1980). The probability was less than 0.03 that this increase occurred by chance. Closer examination of the increase indicated that a statistically significant effect occurred for African-Americans in the study, but not for Caucasians. The authors also reported that a rise in systolic blood pressure for Caucasian noise-exposed school children disappeared as length of enrollment increased. The probability that the rise occurred by chance was less than 0.07. The LA study was unable to find a statistically significant association between blood pressure and aircraft noise at the 1 year follow-up. The authors ascribed this to relocation of susceptible individuals from the study area, but this was not verified (Cohen, *et al.*, 1981).

The authors reported that the Los Angeles study suggested a link between aircraft noise and increases in blood pressure in chronically exposed schoolchildren. However, the suggested link is weakened by the inconsistency of the results as described above.

The Sydney study, which was cross-sectional in design, showed no effect of aircraft noise on blood pressure. Systolic and diastolic blood pressure levels were measured for 1,230 Year 3 schoolchildren from a random sample of primary schools within a 20 km radius of the Sydney airport. Response rates for the study were about 80% of schools approached and 40% of children in Year 3 from the participating schools. The authors stated that this was adequate because the outcome was a physical measurement. The accuracy of the blood pressure measurements was reported as ± 2 mm Hg.

Aircraft noise exposure was reported as monthly energy averaged noise levels accurate to single Australian Noise Energy Index (ANEI) units. They were geocoded to individual school and residential addresses of each participant. A level was assigned to each survey participant. The levels ranged from 15 to 45 ANEI.

Multiple linear regression was used to determine, simultaneously, the magnitude and statistical significance of the effect of aircraft noise and potentially confounding variables. The potential confounding factors included body size, child activity levels, use of salt on food, family history of high blood pressure, whether the child ate breakfast before school, ambient temperature, rail and road traffic noise. A correction for cluster sampling was made in the statistical analysis. All data were obtained between March 11, 1994 and May 6, 1995. The new runway at the Sydney airport opened in the middle of the study, October, 1994.

The study found that blood pressure was not associated with noise exposure. Diastolic blood pressure decreased with time after the opening of the new runway, and systolic blood pressure decreased if the house was insulated. No association was found with road or rail noise. Statistically significant confounders were weight, pulse rate, not eating before school (systolic), using salt on food (diastolic), non-English speaking background (systolic).

The authors noted the potential difficulty of finding an effect because blood pressure is normally highly variable, both between and within individuals. The estimation of the statistical power of the study is not stated in the 1998 Sydney conference paper. Therefore the possibility of a Type II error being committed is not addressed. (A Type II error occurs if the study finds no statistically significant association between outcome and exposure when, in fact, an association exists.) Any possibility that aircraft noise has an effect on childhood blood pressure can only be confirmed or disproved with longitudinal follow-up.

In the recent Munich airport study, there were two experimental groups, each with a less exposed control group, matched for sociodemographic characteristics. The first experimental group was exposed to the noise of the old Munich airport. The second experimental group was not initially exposed to aircraft noise, only after the opening of the new Munich airport (in a new location). The study was longitudinal because there were three testing times during the span of two years (wave 1: occurred 6 months prior to the change over of airports; wave 2: one year later; and wave 3: two years after wave 1).

In the first experimental group around the old Munich airport (Evans, *et al.,* 1995), a 3 mm increase in systolic blood pressure was found to be associated with aircraft noise. The authors concluded that the result was statistically significant because their analysis indicated that the probability (p-value) was less than 0.08 that the observed increase was due to chance. (Most scientists and statisticians would consider a p-value less than 0.05 to indicate statistical significance. Some scientists and statisticians describe p-values less than a number between 0.06 and 0.10 as being indicative of marginal or borderline significance. This latter description is somewhat controversial and some epidemiologists would state that p-values this large would likely be due to chance.) As socioeconomic status can be a confounding factor for blood pressure, it was important that the authors of the article showed that households in the noise-exposed and control areas did not differ in socioeconomic status. However, there was insufficient detail in the reporting of the statistical analysis to assess its validity. For example, for the endpoints of interest, the standard deviations were not reported, so that applicability of the t-test could not be verified.

For the second experimental group around the new Munich airport, over the 3 waves of the study, the increase in systolic and diastolic blood pressures for the noise affected community was 3.4 mm greater than for its matched control group (Evans, *et al.*, 1998; Hygge, *et al.,* 1998). Repeated measures statistics indicated that the probability was less than 0.05 that the difference in systolic blood pressure could be due to chance. (Most scientists and statisticians would consider it unlikely that the observed difference was due to chance). The rise in systolic blood pressure associated with aircraft noise was small compared to normal physiological variations in either population and was essentially the same as the difference in blood pressure level between the two populations at the beginning of the study. The observed rise in average diastolic blood pressure was assessed to have a probability of less than 0.06 of occurring by chance.

The exposure data makes it somewhat difficult to interpret the observed associations. The values for the 24 hour time-averaged sound levels, in A-weighted decibels (dBA), at the new airport in the noise-exposed and control groups (Hygge, private communication) are given in Table 1 below.

Table 1.
Time-averaged sound levels (24 hr.), Leq (dBA) new airport

Subject Group	Wave 1	Wave 2	Wave 3
Noise	53	66	62
Control	53	61	55

These data were obtained only outside the school that the children attended and only during the 24 hour periods in which the children underwent the physiological and psychological tests used in the study. Therefore it is difficult to tell how representative these sound levels were of the chronic exposure of the children. This difficulty is increased by the fairly large variations that were found in the time-averaged sound levels. For example, at Wave 2, the exposure level for the control neighbourhood was essentially the same as for the noise neighbourhood at Wave 3. This appears to weaken support for the hypothesis that aircraft noise significantly elevates blood pressure among children.

Except for socioeconomic status and ponderosity, the ratio of weight to height, the Munich study did not appear to control for confounding factors which have a bearing on blood pressure in childhood and adolescence . These factors include: differences in diet, such as salt intake (Elliott, 1991), body mass index, height, weight (the correlation coefficient for age 10 is about 0.4 for weight as a predictor of blood pressure (De Swiet, *et al.,* 1992)), levels of physical activity and age (De Swiet, *et al.,* 1992; Law, *et al.,* 1993; Task Force, 1987). As a result, the study's conclusions of an association between chronic noise exposure and increased blood pressure may not be valid.

Even if the epidemiological studies had reliably demonstrated an effect of chronic aircraft noise exposure on blood pressure, the observed elevations were probably not clinically significant. Throughout the Munich study, blood pressures measured in the noise-impacted and control groups were both around the 50th percentile range of a U.S. and U.K. population, according to the standards developed by the Second Task Force on Blood Pressure Control in Children (Task Force, 1987).

The only cause for clinical concern would be if the observed elevation in children could lead to elevated blood pressure in adulthood. This stems from evidence that suggests that a lower blood pressure will be associated with a lower risk for cardiovascular disease (MacMahon, *et al*., 1990). Conversely, any increase in blood pressure could be considered as representative of a higher risk. Although, there is some evidence that blood pressure in children can be correlated with blood pressure later in adulthood (Ingelfinger, 1994), the correlation is weak for 10 year old children (De Swiet, *et al.,* 1992), the age group in the Munich study. Therefore, it is unlikely that the observed elevation in blood pressure in children would lead to raised blood pressure in adulthood and a subsequent increased risk of cardiovascular disease.

3.2.1.1 Noise Induced Blood Pressure Effects – Conclusions

There were inconsistent findings between and within studies as to whether observed differences in blood pressure between controls and noise-exposed groups were due to chance. In addition, characterization of the noise exposure was difficult to interpret in the Munich study, casting some doubt as to whether observed differences were due to noise exposure. Furthermore, lack of control for some potentially confounding

factors in the Munich study further weakens support for the hypothesis that noise exposure alone was responsible for the observed differences in blood pressure.

The differences in blood pressure between control and exposed populations of 3 mm Hg would not be clinically significant in the subject population even if they had been reliably demonstrated by the epidemiological studies reviewed here.

3.2.2 Noise-induced Stress Hormone Effects

The Munich airport study is the only one around civilian airports to test for stress hormone levels in children. Measurements were made of the resting levels of the catecholamine (epinephrine and norepinephrine) and cortisol stress hormones (Evans, *et al.*, 1998; Hygge, *et al.*, 1998). The results showed evidence of elevated catecholamines but no change in cortisol associated with aircraft noise.

The design of the study and noise exposure values have been described above in Section 3.2.1. The results for the catecholamines are shown in Tables 2 and 3 across the 3 waves of measurement.

Table 2.
Changes in epinephrine levels

	Epinephrine ng/hr	Epinephrine ng/hr	Epinephrine ng/hr
Subject Group	Wave One	Wave Two	Wave Three
Aircraft Noise-impacted	229.2	328.1	341.9
Quiet Community	251.8	280.9	246.2

At Wave One, before the opening of the new airport, the levels of both catecholamines in the Quiet Community were higher than those in the community that was to be noise-affected by the new airport. However, at Waves Two and Three, the levels of both catecholamines in the Noisy Community increased much more than in the Quiet Community. The authors concluded that these results indicated a statistically significant association of catecholamine level with aircraft noise.

Table 3.
Changes in norepinephrine levels

Subject Group	Wave One Norepine-phrine (ng/hr)	Wave Two Norepine-phrine (ng/hr)	Wave Three Norepine-phrine (ng/hr)
Aircraft Noise-impacted	610.7	1,228.5	1,556.3
Quiet Community	660.0	879.7	950.7

As noted above in Section 3.2.1 the difficulties concerning the measured noise levels reduces the confidence that the observed stress hormone response arises from aircraft noise, as opposed to other factors associated with the development of the airport.

Also, although all groups were from the third and fourth grade at the start of the study, some age confounding cannot be ruled out. There is a fairly strong effect of age on urinary epinephrine output. For adults and children over the age of 10 years, the upper limit of normal for urinary epinephrine output is about 20 µg/day while for children under 10 years it is 14 µg/day (Behrman, *et al.*, 1987). For norepinephrine, this limit is about 100 µg/day for adults, 80 µg/day limit for children over 10 years of age and 65 µg/day for those under 10 years of age (Behrman, *et al.*, 1987). Therefore, over a two year study involving 9-11 year old children there would be a naturally occurring increase in epinephrine and norepinephrine output as the children's catecholamine output approaches adult values. The differences might be more pronounced if the children in the 2 groups were not matched for age.

It has been hypothesized that chronic and excessive elevations of the catecholamines can have adverse impacts on the cardiovascular and immune systems later in life. In the Munich study, the average excretion rates of epinephrine and norepinephrine for both Noisy and Quiet Communities were well within the normal limits of, on average, an output of 20.8 – 833 ng/h for epinephrine and 625 – 3333 ng/h for norepinephrine. (Normal values for 24h urinary output of epinephrine and norepinephrine for children greater than 10 years old are listed as 0.5 – 20 µg/d and 15 – 80 µg/d respectively (Behrman, *et al.*, 1987).)

3.2.2.1 Noise-induced Stress Hormone Effects – Conclusions

The results showed evidence of elevated catecholamines between control and exposed populations, but difficulties in interpreting the noise exposure and potential confounding factors due to age cast some doubt on how much of the observed difference in catecholamine levels was due to noise. Furthermore, the lack of a corroborating change in cortisol levels does not support the conclusion that any observed changes in catecholamines was a sign of chronic stress. Independent longitudinal studies would be needed to assess whether chronic exposure to aircraft noise leads to a chronic increase in stress hormone levels.

3.3 Cardiovascular Disease in Adults

The evidence was reviewed as to whether aircraft noise may be a risk factor for cardiovascular disease, taking into account criteria discussed in Appendix 1 for guiding the determination of a causal relationship in environmental studies. There are very few studies of environmental aircraft noise, in the vicinity of airports, dealing with cardiovascular disease. The main ones are the study by Altena, *et al.*, (1989) (as cited

in Passchier Vermeer, 1993 and Pulles, *et al.*, (1990)) and the studies done by Knipschild (Knipschild, 1977a; Knipschild, 1977b; Knipschild, 1977c). Therefore, in an effort to assess the risk of cardiovascular disease, traffic noise studies have also been considered in this report.

Of the traffic noise studies, the Caerphilly and Speedwell study (Babisch, *et al.*, 1993; Babisch, *et al.*, 1999) of the effects of noise on cardiovascular disease, and its risk factors, is the most persuasive because it has a longitudinal and prospective design, with a follow-up of 10 years. The study also has reasonably well defined health outcomes and exposure levels and more controls for confounding factors than other studies.

3.3.1 Hypertension

The study by Altena, *et al*., (1988), (Pulles, *et al*., 1990), was a cross-sectional study which examined 830 persons exposed to military aircraft noise and road traffic noise. The study population was divided among six exposure intervals. Prior to adjusting for confounding factors, regression analysis showed a statistically significant increase in systolic blood pressure with aircraft noise exposure. However, there was no significant relation between noise and blood pressure level after adjustments had been made for known risk factors such as age, sex, relative body mass, etc.

Knipschild studied the consequences of aircraft exposure around Schipold airport. His investigation consisted of three parts: a prevalence study of cardiovascular disease (Knipschild, 1977a), a survey of general practitioners for attendance for cardiovascular disease (Knipschild, 1977b), and a survey of purchases of hypertensive and other cardiovascular medications by pharmacies (Knipschild, 1977c). Respectively, these studies reported increases in: (i) prevalence of hypertension; (ii) attendance at the general practitioners office for cardiovascular disease; and (iii) purchases by pharmacists of cardiovascular medication, particularly antihypertensives, with increased levels of aircraft noise.

Data for the study of cardiovascular disease (Knipschild, 1977a) was collected by inviting members of a community consisting of eight villages to undergo medical examination. The medical screening included the collection of medical history data, measurement of blood pressure, x-ray of the heart, and ECG. There were approximately 6000 people medically screened, at a response rate of about 40%. Respondents were separated into high and low noise exposure groups. The high noise exposure group began at about a day-night sound level (Ldn) of about 62 dBA. (The day-night sound level is the time-averaged sound level obtained by averaging the sound exposure from 0700 one day to 0700 the next with the sound level being increased by 10 dB between 2200 and 0700 hours. The Ldn value of 62 dBA was estimated from the Dutch exposure units reported in Knipschild's work using a conversion factor in section 2.1 of Passchier-Vermeer (1993)).

The percent of individuals with measured blood pressure above 175/100 was greater in the high-noise group (Relative Risk =1.8, $p < 0.05$). Also, the percent of participants undergoing medical treatment for hypertension was greater in the high noise group (Relative Risk = 1.5, $p < 0.05$). This is traditionally considered statistically significant. The influence of age and sex was taken into account in the analysis. The influence of other confounding factors was not explicitly shown in the study. However, the author indicated that smoking, body mass, and village size had been taken into account, where possible and this information was collected as part of the medical screening. The author noted that there were some indications that the socio-economic status of the high-noise group may be lower, but there is no indication that this was accounted for in the study. Lack of control for this last confounding factor has been a recurring criticism of this study in several reviews (Cohen, *et al.*, 1986; Thompson, *et al.*, 1989; Berglund and Lindvall, 1995).

The survey of general practitioners took place in three villages around the airport over a one-week period in which 19 general practitioners recorded the age, sex, address, reason for visit (diagnosis) and medication used for all of their patients. The contact rate for cardiovascular diseases was reported as being greatest in the village with the highest noise exposure. Similarly, the usage of antihypertensive medication was higher, especially in women. As in the previous study, the effects of socio-economic status were not taken into account. The author noted that the control population, with the lowest noise exposure, had a higher-socioeconomic status and a greater proportion of white-collar workers.

The drug survey (Knipschild, 1977c) was conducted in 2 villages around Schipold airport, the high exposure and control villages of the general practitioner survey (Knipschild, 1977b). Over the period 1967-1974, drug purchases by the village pharmacies, per adult per year were used as an indicator of the consumption of medications in the subject populations. The village designated as the high noise exposure area experienced a change over time in the noise exposure levels; from little prior to 1969, to high noise exposure from 1969-1973 and only daytime noise during 1973-74. By contrast, the noise level in the control area remained constant. In the exposed area, a gradual increase was reported for purchases of cardiovascular drugs by the village pharmacies. The final value was up to two times the initial rate, the largest contribution being for antihypertensive drugs. This was not affected by the reduction in nighttime noise level in 1973. In the control area there was no change over time.

These studies suggest that there may be an association between hypertension and aircraft noise at Ldn values greater than about 62 dBA. However, the evidence for this association is not convincing because of the lack of controls for socioeconomic status in the first two studies and the lack of a statistical analysis in the third.

As there are so few studies on aircraft noise and cardiovascular effects in adults, it was necessary to broaden the review to include traffic noise studies. Babisch has recently

completed a comprehensive review of this subject (Babisch, 2000). In this paper, Babisch notes that dose assessments in most of the traffic noise studies were crude, usually based on noise maps of the region. Studies usually only had two exposure groups – low and high. Also, subjective estimates of exposure were sometimes used. For example, Herbold (1989) based his estimates on self-reporting of the type of road adjacent to the participant's home. The noise levels for these types of roads were then simply grouped as "low" and "high," depending on the type of road. Neus, *et al.,* (1983a, 1983b) based their estimates of noise exposure on traffic volume.

From the review by Babisch (2000), it is also clear that, using 95% confidence intervals, associations were not consistently found in independent studies. Only 4 of 10 studies reviewed by Babisch yielded associations between traffic noise and hypertension. Of these 4 studies, Babisch noted that 2 would meet modern standards of control for confounding factors. These were both cross-sectional studies. Babisch (2000) concluded that there was little epidemiological evidence of an increased risk of hypertension in subjects exposed to traffic noise.

3.3.1.1 Hypertension – Conclusions

The review of studies that investigated the potential link between hypertension and either aircraft or traffic noise exposure, indicated that the available evidence does not appear to convincingly demonstrate an association between aircraft noise and hypertension.

3.3.2 Ischemic Heart Disease

Ischemic heart disease is characterized by insufficient perfusion of oxygen to the heart muscle. For the study by Altena, *et al*., (Pulles, *et al.,* 1990; Altena, *et al.,* 1988), ischemic heart disease was assessed by clinical symptoms of angina pectoris (chest pain), myocardial infarction (heart muscle damage), or electrocardiogram (ECG) abnormalities as defined by criteria of the World Health Organization (WHO). This study, described in the previous section, did not show any increase in the prevalence of ischemic heart disease with increasing exposure to aircraft or traffic noise. There is a possibility that the negative finding may have been due to selection bias since those with hypertension were excluded from the study and this condition is a known risk factor for ischemic heart disease.

Details of the review of the Knipschild studies are provided above. The prevalence of cardiovascular disease was determined by a questionnaire and clinical examination (Knipschild, 1977a). The following data were recorded for each participant: clinical symptoms of angina pectoris (according to a standard WHO questionnaire), medical treatment for heart trouble and hypertension, usage of cardiovascular drugs, ECG abnormalities, heart shape and blood pressure measurements. The results indicated that, in the noise exposed population, there was a statistically significant increase in all but two of these endpoints, compared to the control group. The Relative Risks and p values were: (i) 1.4, $p < 0.05$ for medical treatment for heart trouble; (ii) 1.4, $p < 0.01$ for usage of cardiovascular drugs; and (iii) 1.6, $p < 0.05$ for pathological heart shape. No statistically significant differences were found for angina pectoris and ECG abnormalities, which are important indicators of ischemic heart disease, respectively.

The general practice survey showed an increase in contacts with the physician for cardiovascular disease in the noise-exposed area. The drug survey also showed an increase in cardiovascular drug use over time in a noise-exposed area. However, neither of these studies provides sufficient information to adequately assess their relevance to ischemic heart disease.

The shortcomings of the Knipschild studies have been described in the discussion above on hypertension. These shortcomings also apply to the ischemic heart disease endpoints. Furthermore, given the lack of statistically significant associations between noise level and two important indicators of ischemic heart disease in these studies, the available aircraft noise studies do not provide convincing evidence of an association between ischemic heart disease and environmental aircraft noise exposure.

The remaining studies on environmental noise and ischemic heart disease are traffic noise studies. These include a retrospective study of myocardial infarction in the city of Erfurt (Babisch, 2000) and 2 prospective case control studies of myocardial infarction in Berlin, Germany (Babisch, *et al*., 1994). In addition, prospective 10 year longitudinal studies of cardiovascular risk were done in the cities of Caerphilly and Speedwell in Wales and England, respectively (Babisch, *et al.,* 1993; Babisch, *et al.,* 1999). In Babisch's review of these studies (Babisch 2000), noise levels were reported as outdoor time-averaged traffic noise levels (06:00-22:00).

As reported by Babisch (2000), a high and significant proportional morbidity ratio in the Erfurt study was determined for areas with noise levels between 71-75 dBA compared to areas with noise levels of 61-65 dBA. However, methodological issues about the validity of the results have been raised by Babisch (2000). (This study is only available in German and has not been reviewed by the authors).

In the Berlin pre-study and main study (Babisch, *et al*., 1994), increase in incidence of myocardial infarction was assessed relative to populations living in areas with noise levels less than 60 dBA. Increases were observed but they were not statistically significant at the 95% confidence level. The lower limit of the 95% confidence interval was less than 1.0 for all odds ratios determined in these studies. The reported values of the odds ratios in the pre-study were 1.5 and 1.2 in the 61-65 dBA and 66-70 dBA noise level range, respectively. The 95% confidence intervals were 0.6-3.9 and 0.5-2.9, respectively. The corresponding odds ratios in the main study were 1.2 and 0.9 in the 61-65 dBA and 66-70 dBA noise level range, respectively. The corresponding 95% confidence intervals were 0.8-1.7 and 0.6-1.4. Even the reported mean values of the odds ratios (relative risks) showed no consistent trend with noise level under 70 dBA. The small sample size of the

pre-study led to only one case being available in the highest range of noise levels, precluding any further conclusions from being drawn from that study. In the main study, above 70 dBA, the mean value of the relative risk increased from 1.1 to 1.5 as the range of noise levels increased from 71-75 dBA to 76-80 dBA. The 95% confidence intervals were 0.7-1.7 and 0.8-2.8, respectively.

Further analysis of the Berlin study data was carried out by Babisch for the two highest ranges of noise levels. First, to ensure that the subjects had undergone sufficient exposure to the noise, analysis was restricted to subjects who had lived in the study areas for more than 15 years. Also, to improve the statistical power, the data was grouped into a single high noise exposure level of 71-80 dBA. The relative risk was then found to be 1.3 with a 95% confidence interval of 0.9-2.0. Babisch considered this result to be borderline significant ($p < 0.10$). The results of this study are not sufficient to demonstrate an association of traffic noise level with incidence of myocardial infarction. However, they suggest that further research is needed in areas with high populations exposed to high noise levels.

In the Caerphilly and Speedwell studies, the increase in risk in noise-exposed areas was assessed relative to populations in areas where the noise levels were less than 55 dBA. The Caerphilly and Speedwell studies are a series of investigations in which two cohort studies were done on the effects of traffic noise. They were part of a larger study to examine the predictive power of known and new risk factors for ischemic heart disease. These cohorts were studied over a ten year period. Combined analysis is available for a 6 year period.

This Caerphilly and Speedwell study has advantages over many others in that it is prospective in design. Exposure assessment is based on noise level measurement. Disease outcome is determined by hospital records according to well defined criteria. More confounders have been taken into account in this analysis than in any other study.

In the Caerphilly 10 year follow-up there was a slightly higher relative risk of ischemic heart disease in the 56-60 dBA and the 66-70 dBA subgroups, but this was only marginal and non-significant. In the Speedwell 10 year follow-up there was no increase in ischemic heart disease in any of the groups. However, Babisch, *et al.,* (1999) also provided an analysis of the data, pooling the populations in a 6 year follow-up. For this pooled data, in the highest noise-exposed group, 66-70 dBA, the adjusted odds ratio increased from 1.07 to 1.59 as further refinements were made to the exposure classification of the subjects. The 95% confidence interval also varied with these changes from 0.70-1.65 to 0.85-2.97. This included examining a subsample in residence not less than 15 years and taking into account window orientation, and window-opening practices.

Babisch, *et al*., (1999) also analyzed the data using an alternative model in which the noise exposure was set equal to the product of noise level with years of residence. Using this analysis, there is an increase in the odds ratio per year in residence from 1.007 to 1.017 in the highest noise category as the exposure assessment is refined accounting for window orientation and window opening practices. The 95% confidence intervals ranged from 0.992-1.023 to 0.998-1.036, respectively. The odds ratio of 1.017 was considered by Babisch, *et al.,* (1999) to be borderline significant, being greater than unity at a p value < 0.10.

The results of the Caerphilly and Speedwell study appear to be equivocal. The trend of increasing odds ratios, with improvements to the exposure assessment and when time of residence was considered, suggests there may be a slight increase in ischemic heart disease among those exposed to chronic high levels (> 66 dBA) of environmental noise. Nevertheless, there is considerable overlap of the 95% confidence intervals of these odds ratios suggesting that the increase may only have been due to chance. Also, the odds ratio of 1.017 per year in residence was only considered to be borderline significant by Babisch, *et al.,* (1999), again suggesting that the change in odds ratio with increasing time of residence may also have been due to chance.

3.3.2.1 Ischemic Heart Disease – Conclusions

There is no convincing evidence for a causal relationship between environmental noise and ischemic heart disease. At traditional 95% confidence levels used to assess statistical significance, dose response relationships have not been demonstrated. Also, potential trends with improved exposure assessment procedures and increasing years in residence may have been due to chance. Furthermore, the strength of the associations is typically relatively weak, with observed relative risk ratios or odds ratios ranging from 1.3 to 1.6, at most in the Berlin and the Caerphilly and Speedwell studies. In these studies, important confounding factors were taken into account and efforts had been made to reduce bias, including the effort of determining exposures by measurement.

However, the available studies provide some evidence to suggest that there may be a slight increase in the risk of ischemic heart disease in people residing in areas with daily averaged traffic noise levels greater than 65 dBA. This indicates that more research on this subject is needed. Also, there needs to be continued assessment of future research on the potential for cardiovascular risks from aircraft noise. This follows from the relative consistency of elevated risk among the exposure groups with daily averaged sound levels greater than 65 dBA. It also follows from the temporal effect suggested by the increasing odds ratios with years of residence in the Caerphilly and Speedwell study. The need for more research in this area is also consistent with the suggested trend of increasing odds ratios with improved exposure assessment.

4. Recommendations

The available research does not support the contention that there is a significant risk of chronic stress and/or cardiovascular disease arising from long term exposure to outdoor daily aircraft noise levels above 65 dBA. This corresponds to Noise Exposure Forecast levels of about NEF = 33. (The NEF is used in Canada to characterize aircraft noise in an area.) However, the available studies indicate that more research is needed. Also, there needs to be continued assessment of future research on the potential for chronic stress and cardiovascular risks from aircraft noise. This will ensure that timely and accurate advice can be presented to the public and regulatory authorities to enable them to exercise their responsibility of managing the health risks of environmental aircraft noise.

References

Altena, K., W. Biesiot, N.E. van Brederode, I. van Kamp, T.R. Knottnerus, J.V. Lako, M.P.J. Pulles and T.E. Stewart. Environmental noise and health, description of data, models and methods used and the results of the epidemiological survey. Report No. GA-DR-0e-01. The Hague: Ministry of Housing, Physical Planning and the Environment, 1988.

Babisch, W., H. Ising, B. Kruppa and D. Wiens. The incidence of myocardial infarction and its relation to road traffic noise – the Berlin case-control studies. Environment International, 20: 469-474, 1994.

Babisch, W. Traffic noise and cardiovascular disease: epidemiological review and synthesis. Noise & Health, 8: 9-32, 2000.

Babisch,W., H. Ising, J.E.J. Gallacher, P.M. Sweetnam and P.C. Elwood. Traffic noise and cardiovascular risk: the Caerphilly and Speedwell Studies, third phase – 10-year follow up. Archives of Environmental Health, 54: 210-216, 1999.

Babisch,W., H. Ising, P.C. Elwood, D.S. Sharp and D. Bainton. Traffic noise and cardiovascular risk: the Caerphilly and Speedwell Studies, second phase. Risk estimation, prevalence, and incidence of ischemic heart disease. Archives of Environmental Health, 48: 406-413, 1993.

Behrman, R.E., V.C. Vaughan III and W.E. Nelson. Nelson Textbook of Pediatrics, 13th ed. W.B. Saunders Co., Table 29-2 (Reference Ranges), pp 1539-1540, 1987.

Berglund, B. and T. Lindvall (Eds.). *Community Noise*. Archives of the center for sensory research. Stockholm, 1995.

Chrousos, G.P. and P.W. Gold. Editorial: A healthy body in a healthy mind – and *vice versa* – the damaging power of "uncontrollable" stress. J. Clin. Endocrin. and Metab., 83: 1842-1845, 1998.

Cohen, S., G.W. Evans, D.S. Krantz and D. Stokols. Physiological, motivational and cognitive effects of aircraft noise on children: moving from the laboratory to the field. Am Psychol., 35: 231-243, 1980.

Cohen, S., G.W. Evans, D.S., Krantz and D. Stokols. Aircraft noise and children: longitudinal and cross-sectional evidence on adaptation to noise and the effectiveness of noise abatement. J. Pers Soc Psychol., 40: 331-345, 1981.

Cohen, S., G.W. Evans, D. Stokols and D.S. Krantz. *Behavior, Health and Environment Stress*, New York, Plenum Press, Chapter 4, 1986.

De Swiet, M., P. Fayers and E.A. Shinebourne. Blood pressure in first 10 years of life: the Brompton study, BMJ, 304: 23-26, 1992.

Elliott, P. Observational studies of salt and blood pressure, Hypertension, **17** (suppl.1), 103-108, 1991.

Evans, G.W., S. Hygge and M. Bullinger. Chronic noise exposure and physiological response: a prospective study of children living under environmental stress. Psychological Science, 9: 75-77, 1998.

Evans, G.W., S. Hygge and M. Bullinger. Chronic noise and psychological stress. Psychological Science, 6: 333-338, 1995.

Frankenhauser, M. A Psychobiological Framework for Research on Human Stress and Coping. In: Appley, M.H. and Trumbull, R. (Eds.). *Dynamics of Stress: Physiological, Psychological and Social Perspectives*. Plenum Press. New York, pp. 101- 116, 1986.

Friedman, T.C., G. Mastorakos, T.D. Newman, *et al*. Carbohydrate and lipid metabolism in endogenous hypercortisolism: shared features with metabolsim syndrome X and noninsulin- dependent diabetes mellitus. Endocr. J., 43: 645-656, 1996.

Glynn, J. A question of attribution. Lancet, 342, pp 530-532, August 28, 1993.

HCN. *Noise and Health. Report of a committee of the Health Council of the Netherlands*. Report No. 1994/15E. The Hague, 15 September, 1994.

HCN. *Public health impact of large airports*. Health Council of the Netherlands. Report No. 1999/14E, The Hague, 2 September, 1999.

Herbold, M., H.-W. Hense and U. Keil. Effects of road traffic noise on prevalence of hypertension in men: results of the Luebeck blood pressure study. Soz Praeventivmed, 34: 19-23, 1989.

Hygge, S., G.W. Evans and M. Bullinger. The Munich airport noise study – effects of chronic aircraft noise on children's cognition and health. *7th International Congress on Noise as a Public Health Problem*, Sydney, Australia, 268-274, November 1998.

IEH. *IEH report on the non-auditory effects of noise*. Report R10. Medical Research Council. Institute for Environmental Health, Leicester, U.K., pg 90, 1997.

Ingelfinger, J.R. Pediatric Hypertension, Current Opinions in Pediatrics 6: 198-205, 1994.

Knipschild, P. Medical Effects of Aircraft Noise: Community Cardiovascular Survey. Int. Arch. Occup. Environ. Health, 40: 185-190, 1977a.

Knipschild, P. Medical effects of aircraft noise: general practice survey. Int Arch Occup Environ Health, 40: 191-196, 1977b.

Knipschild, P. and N. Oudshoorn. Medical effects of aircraft noise: drug survey. Int Arch Occup Environ Health, 40: 197-200, 1977c.

Law, C.M., M. De Swiet, C. Osmond, *et al*. Initiation of hypertension in utero and its amplification throughout life, BMJ, 306: 24-27, 1993.

Lazarus, R.S. and S. Folkman. *Stress, Appraisal and Coping*. New York: Springer-Verlag, 1984.

MacMahon, S., R. Peto, J. Cutler, *et al*. Blood pressure, stroke and coronary heart disease: part I, prolonged differences in blood pressure: prospective observational studies corrected for regression dilution bias. Lancet, **335**: 765-774, 1990.

McEwen, B.S. Protective and damaging effects of stress mediators. New England Journal of Medicine, 338: 171-179, 1998.

McEwen, B.S. Stress and the Individual, Arch Internal Med, 153: 2093-2101, 1993.

Morrell, S., R. Taylor, N. Carter, S. Job and P. Peploe. Cross-sectional relationship between blood pressure of school children and aircraft noise, *7th International Congress on Noise as a Public Health Problem*, Sydney, Australia, 275-279, November 1998.

Neus, H., H. Ruddel and W. Schulte. Traffic noise and hypertension: an epidemiological study on the role of subjective reactions. International Archives of Occupational and Environmental Health, 51: 223-229, 1983a.

Neus, H., H. Ruddel, W. Schulte and A.W. von Eiff. The long-term effect of noise on blood pressure. Journal of Hypertension, 1 (Supplement 2): 251-253, 1983b.

Passchier-Vermeer, W. *Noise and health*, The Hague: Health Council of the Netherlands, publication no. A93/02E, pp 79-83, 1993.

Pulles, T., W. Biesot and R. Stewart. Adverse effects of environmental noise on health: an interdisciplinary approach. Environment International, 16: 437-46, 1990.

Rosmond, R., M.F. Dallman and P. Bjorntorp. Stress-related cortisol secretion in men: Relationships with abdominal obesity, endocrine, metabolic and hemodynamic abnormalities. J. Clin. Endocrinol. Metab., 83: 1853-1859, 1998.

Scheuch, K. Theoretical and Empirical Considerations in the Theory of Stress from a Psychophysiological Point-of View. In: Appley, M.H. and Trumbull, R. 1986. (Eds.). *Dynamics of Stress: Physiological, Psychological and Social Perspectives*. Plenum Press. New York, pp. 117- 139, 1986.

Stansfeld, S. and M. Haines. Environmental noise and health: A review of non-auditory effects. In: *IEH report on the non-auditory effects of noise*. Report R10. Medical Research Council. Institute for Environmental Health, Leicester, U.K., pp 7-64, 1997.

Task Force on blood pressure control in children. Report of the second task force on blood pressure control in children – 1987, Pediatrics, 79: 1-25, 1987.

Thompson, S.J. Cardiovascular and fetal effects of noise. In: *IEH report on the nonauditory effects of noise*. Report R10. Medical Research Council. Institute for Environmental Health, Leicester, U.K., pp 70-75, 1997.

Thompson S., S. Fidell and B.G. Tabachnick. *Feasibility of Epidemiologic Research on Non-auditory Health Effects of Residential Aircraft Noise Exposure, Summary of Literature on Cardiovascular Effects on Noise Exposure*. Volume III. Human Systems Command, Brooks Air Force Base, TX, HSD-TR-89-007, Dec. 1989.

Traven, N.D., E.O. Talbott and E.K. Ishii. Association and Causation in Environmental Epidemiology. In: *Introduction to environmental epidemiology.* Talbott, E.O. and Craun, G.F. ed., Lewis Publishers, New York, pp 39-46, 1995.

Tsigos, C. and G.P. Chrousos. Differential diagnosis and management of Cushing's syndrome. Ann. Rev. Med., 47: 443-461, 1996.

WHO. *Guidelines for Community Noise*. Berglund, B., Lindvall, T. and Schwela, D.H. (Eds.). World Health Organization, Geneva. Available at http://www.who.int/peh/noise/noiseindex.html, pp 47-48, 59, 1999.

Appendix 1

There are several sets of criteria to assess the likely role of cause in an association observed in an epidemiological study. The criteria used for this report were adapted from the Bradford Hill criteria (Glynn, 1993; Traven, *et al.*, 1995). They are summarized below.

Strength of association. A relative risk greater than 3 provides good evidence that an association is likely to be causal. Important confounders can produce strong associations if not controlled for. A small relative risk, on the other hand, does not rule out a causal relationship – it only means that it is more difficult to exclude some other explanation.

Dose-response. If a dose response relationship is seen, it strengthens the likelihood of causality. Again confounding factors may lead to spurious dose-effect relationships. The absence of a dose-response relationship does not necessarily weaken an association, especially if there is a threshold effect.

Temporality. In its simplest form the causal factor must precede the onset of the disease in question. However, interactions between exposure to the agent and the body's biological system may influence the temporal relationship between exposure and disease.

Reversibility. Removal or reduction of exposure can provide convincing evidence of causality.

Consistency. An association that is shown repeatedly in different studies is unlikely to be due to chance. If conducted by different investigators in different places and times an association is unlikely to be due to a constant bias.

Plausibility. The existence of a suggested mechanism by which the agent causes a disease is reassuring, but will depend on the knowledge at the time. Mechanisms for an association between chronic aircraft noise exposure and cardiovascular disease have been proposed

Specificity. Since most diseases have more than one factor contributing to their onset, specificity of an observed association cannot be insisted upon. It is understood that there will not be a one-to-one relationship between aircraft noise and cardiovascular disease.