

Aircraft noise and health effects: Recent findings

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Chapter 1

Introduction

- 1.1 The report aims to provide an update to the Environmental and Research Consultancy Department Report 0907 entitled Environmental Noise and Health Effects. Published in 2009, that report examined the evidence to date relating to transportation noise, in particular aircraft noise and the resulting impacts on various health endpoints. These included cardiovascular disease, night-time effects on sleep disturbance, children's cognition, psychological effects, performance and annoyance.
- 1.2 Aircraft noise and health effects is a rapidly growing area of research worldwide, and there have been many important findings published in recent years. Of particular importance has been the European Network of Noise and Health (ENNAH), which has connected researchers in the field throughout Europe to critically assess the current evidence base and identify gaps in the knowledge as well as suggesting directions for future research. The World Health Organisation (WHO) published their Burden of Disease from Environmental Noise report, which has enabled the calculation of healthy life years lost due to environmental noise which is very important for decisions on policy making. The European Environment Agency published their good practice guide on noise exposure and potential health effects which included important exposure-response relationships and thresholds for health endpoints and the Health and Safety Laboratory, through a Defra contract, produced their work on quantifying the links between environmental noise related hypertension and health effects.
- 1.3 In 2015 a review of aircraft noise and health effects by Charlotte Clark was published alongside the Airports Commission's final report on increasing airport capacity in the UK. The review was focused on the current state of knowledge concerning the effects of aircraft noise on a range of health outcomes, and the subsequent potential effects on exposed populations for three different expansion options. The review concluded that there is increasing evidence to support preventive measures such as insulation, policy, guidelines and limit values. Priorities for minimising the effects of aircraft noise should be focused on reducing annoyance, improving school environments for children and aiming to lower cardiovascular risk factors.
- 1.4 In addition to these key publications there have been many more studies into aviation noise and health effects since 2009. This report will review the main findings between 2009 and 2015 and will highlight areas that are considered important for future research.

- 1.5 The scope of this paper will focus around the cardiovascular impacts, sleep disturbance and children's learning with other areas such as performance and psychological effects being included. Although annoyance is often considered a health effect, for the purpose of this paper it will not be included as a single end point health effect, but of course it is appreciated that annoyance may be an important mediator in the relationship between aircraft noise, stress and various health endpoints such as cardiovascular disease. A dedicated CAP report on the current knowledge on aircraft noise and annoyance is planned.

Chapter 2

Cardiovascular effects

- 2.1 A Swedish study (Eriksson et al 2010) examined the cumulative gender-specific effects of aircraft noise on hypertension in a population of over 4000 adults residing close to Stockholm Arlanda airport. The study followed the participants over a period of 8-10 years and in addition to the main aim of investigating any potential differences between genders, the study also looked at the presence of sensitive sub-groups within the cohort. The study was part of a larger study on diabetes Type 2 risk factors and prevention measures, and therefore half of the sample in the aircraft noise study had a family history of diabetes. Baseline health measurements were taken at the beginning of the study, such as weight, waist and hip circumference and blood pressure. Participants also answered an extensive questionnaire on lifestyle factors and treatment for hypertension if there was any. After 8-10 years a follow-up questionnaire was administered with the same questions as in the baseline study, only with additional questions pertaining to aircraft noise and annoyance, noise sensitivity and diagnosis of hypertension.
- 2.2 Participants who were taking medication for hypertension at baseline were excluded from the aircraft noise study at follow up, along with those people displaying high blood pressure and/or those with missing data. During the study period the introduction of quieter aircraft resulted in a continuous decrease of the noise levels overall around the airport and interestingly a third runway was also built. This changed the flight paths which meant that most people experienced a decrease in noise levels, but as expected some areas experienced the opposite. Aircraft noise levels ranging from 50-65 dBA L_{den} were provided by Swedish Airports and Air Navigation Services, and were estimated with 1 dB(A) resolution using the Integrated Noise Model (INM). Exposure assessment was performed by Geographical Information Systems (GIS) and based on residential history during the period of the follow-up.
- 2.3 The results suggested that men and women exposed to 50 dBA L_{den} or above had a lower socioeconomic status and were more likely to work shifts than those exposed to below 50 dBA L_{den} . Alcohol consumption was lower for both sexes in the higher noise group also. Age and body mass index was associated with hypertension in males and females. Annoyance was strongly related to noise exposure, with 80% of people expressing annoyance when exposed to aircraft noise of 60 dBA L_{den} or above. Interestingly, males were more annoyed from 50 dBA L_{den} or above than females (36% and 29% respectively).

- 2.4 The hypertension results indicated that males were more likely to develop hypertension in areas exposed to 50 dB(A) L_{den} or above than females. For men the Relative Risk (RR) was 1.02 (95% CI 0.92-1.29) compared to women having a RR of 0.92 (95% CI 0.76-1.11). This was not a statistically significant difference among genders and as an overall sample there was no increased risk of hypertension following long-term aircraft noise exposure. However, when the cohort was restricted to those people not smoking at the time of the blood pressure measurements, a significant increase in risk per 5 dB was found in males RR 1.21 (1.05–1.39) but not in females RR 0.97(0.83–1.13). The authors explain that this may be due to nicotine having short-term effects on blood pressure and can therefore possibly skew the measurements. When both sexes were combined there was an increased risk for aircraft-noise related hypertension among those people that had reported annoyance to aircraft noise RR 1.42(1.11–1.82).
- 2.5 The authors suggest that it is possible that subjects with perceived noise annoyance represent a subgroup that is under greater risk of developing hypertension related to noise exposure. A possible explanation is that if noise-related hypertension is mediated through annoyance, this could contribute to explaining the observed gender difference in this study since men were more prone than women to report aircraft noise annoyance (36% and 29%, respectively). Observed annoyance levels were higher than expected, and this may be due to the increased awareness of aircraft noise with the opening of the third runway during the study period. An important point to consider when interpreting the difference in hypertension risk between the sexes is that on average women tend to develop hypertension when they are about ten years older than men, and this may be a contributing factor to the results observed in this study. It is also possible that the differences found between sexes may be attributable to confounding factors that are not accounted for. Finally, the sample used had a higher percentage of family history of diabetes (50%) compared to the standard proportion of 20-25% in the general population of corresponding age group which may predispose some of the population to a higher risk of cardiovascular disease.
- 2.6 Greiser et al (2011) published research concerning the risk increase of cardiovascular diseases and impact of aircraft noise in the Cologne-Bonn airport study. Previously, research had shown that there was an increase in the amount of cardiac medication prescribed with increasing aircraft noise exposure (2007). Aircraft, road and rail noise data were linked to hospital discharge diagnoses of just over one million people living in the study area. Confounders included age, environmental noise, prevalence of social welfare recipients of residential quarters and interaction of aircraft noise with age. The results showed that as age increased, the risk of cardiovascular disease decreased. Risk is more marked in females than males. For night-time aircraft noise of 50 dB L_{night} at

aged 50, the odds ratio for cardiovascular disease in men was 1.22 and in women 1.54, for myocardial infarction it was 1.18 in men and 1.54 in women, for heart failure in men 1.52 and 1.59 in women, stroke in men 1.36 and for women 1.36 also. The lack of difference between males and females for heart failure risk and stroke contradicts the hypertension findings with respect to gender in the Swedish study, although this study uses the L_{night} metric rather than L_{den} , which may be a factor.

- 2.7 Floud et al (2011) reported on medication use in relation to aircraft noise of populations surrounding six European airports, as part of the HYENA study. Differences were found between countries in terms of the effect of aircraft noise on antihypertensive use. For night-time aircraft noise a 10 dB increase was associated with an odds ratio of 1.34 (95% CI 1.14 to 1.57) for the UK and 1.19 (CI 1.02 to 1.38) for the Netherlands but no significant associations were found for other countries. There was also an association between aircraft noise and anxiolytic (anti-anxiety) medication, OR 1.28 (CI 1.04 to 1.57) for daytime and OR 1.27 (CI 1.01 to 1.59) for night-time. It should be noted that these confidence intervals are considerable in variation. This effect was found across countries. The authors concluded that although results suggested a possible effect of aircraft noise on the use of antihypertensive medication, the effect did not hold for all countries. The data was more consistent for anxiolytics in relation to aircraft noise across countries.
- 2.8 Harding et al (2011) on behalf of the Health and Safety Laboratory published a report on the quantification of noise related hypertension and the related health effects. The aims of the study were to identify the potential health outcomes associated with hypertension, to prioritise the health outcomes and quantify the links between noise and selected hypertension associated health outcomes. The second half of the report covered a methodology to allow a monetary value to be placed on the links between hypertension and health outcomes.
- 2.9 The base dose-response function for noise and hypertension used by Harding comes from Babisch and van Kamp (2009) who found an odds ratio for hypertension of 1.13 per 10 dBA increase in L_{den} in the range 45 to 70 dBA. Harding goes on to note that because the prevalence of hypertension in the population is greater than ten percent, that the odds ratio must be converted into relative risk in order to quantify the effect on the population.
- 2.10 Previously, the Interdepartmental Group on Cost Benefit analysis of noise IGCB(N) and WHO have considered that there is insufficient certainty from which to quantify the health outcomes from hypertension. However, Harding et al, after in depth review, found the following health outcomes from hypertension could be quantified.
- 2.11 The report concluded that there is substantial evidence for hypertension and blood pressure being an independent risk for cardiovascular disease (CVD).

Many studies investigating hypertension or blood pressure as an independent causal factor for CVD have used separate analyses for stroke and Ischaemic Heart Disease (IHD). It has been suggested that systolic blood pressure may be a better indicator of CVD risk than diastolic blood pressure.

- 2.12 The report discusses evidence of blood pressure being linked to all types of stroke, ischaemic (resulting from a clot) and haemorrhagic (rupturing of blood vessels within the brain). Hypertension is a known risk factor for strokes. There is strong evidence for a link between blood pressure and the incidence and mortality of IHD. IHD is due to the build up of plaque deposits on the artery walls and therefore leads to hardening of the arteries. When the plaque comes away from the walls, blockages can occur in the arteries which can cause a lack of oxygen (ischaemia) in the heart muscle. When the rupture of plaque on the coronary arteries occurs a clot can form, which can subsequently cause a rapid slowing or stop of blood flow and then the classic heart attack (myocardial infarction). There is evidence that lowering blood pressure can help prevent heart attacks.
- 2.13 The report discusses the evidence linking hypertension and dementia, or cognitive decline. The evidence is less strong than for cardiovascular disease, and is complicated by the ethical issues involved in studying long-term hypertension without treatment and also because by the time dementia manifests, hypertension can decrease as a result of weight loss or metabolic changes. There have also been findings that link cognitive decline with blood pressure in subjects aged 59-71 years.
- 2.14 The report also discussed the links between hypertension and end stage kidney disease, pregnancy, eye conditions and sexual function, but it was decided that based on the strength of the evidence and impact on the population that three health outcomes would be given priority in terms of quantification of links between noise and hypertension. These were Acute Myocardial Infarction (AMI), stroke and dementia. It should be noted that this study was designed to assess the risk of noise-related hypertension on the subsequent likelihood of hypertension resulting in the above health outcomes; it is not reporting that noise itself directly causes stroke and dementia.
- 2.15 Paunović et al (2011) published a critical review of studies into road and aircraft noise and children's blood pressure. The aim of the review was to compare the methodologies used to assess blood pressure across the thirteen studies included. Of the seven studies on aircraft noise and children's blood pressure, three were cross-sectional; four were longitudinal with a follow-up of between one and three years. The children were of similar age, between 8-12 years old, with weight and height measured and controlled for in most of the studies as well as ethnicity and in most cases, family history of hypertension. Noise exposure did vary across the studies, with some measurements being taken at schools,

and some at residences. In terms of noise metrics, some studies used daytime levels; others used 24 hour noise levels, or monthly averaged exposure.

- 2.16 The measurement of blood pressure was fairly standard across the studies and used automatic measurement techniques, although there were discrepancies in terms of place, time and number of measurements taken and the degree of control for confounding factors. Despite these variations within the methodology, the authors conclude that there is a tendency toward positive association with noise exposure and an increase in children's blood pressure. It is recommended that more precise guidelines for measuring blood pressure in field studies are put in place. Some suggestions for such standardised protocols are summarised:
- Children's blood pressure should be measured at approximately the same time, either morning or afternoon.
 - The place where measurement occurs should be familiar, well lit, quiet and comfortable.
 - At least three measurements should be taken a few minutes apart, with the first reading being discarded.
 - Measurements should be repeated the following day in the same setting if possible.
 - Instructions should be given verbally and in written form.
 - Measurements should be taken after breakfast, intense physical activity, coffee or energy drinks, and psychoactive substances such as nicotine.
 - The child's emotions should be asked about and measurements avoided if there is expression of anxiety, fear anger or discomfort.
 - Measurements should be avoided if the child has a headache or fever or is receiving any medical treatment at the time of the study.
- 2.17 Chang et al (2012) investigated the effects of environmental noise on 24 hour ambulatory vascular properties in adults. Vascular properties include resistance, which is the resistance to flow that is needed to be overcome in order for blood to be pushed through the circulatory system. Vascular compliance and distensibility (elasticity) is the ability of a blood vessel wall to expand and contract passively with an increase of pressure and cardiac relaxation and pulsation. Early changes in vascular properties can be pre-cursors to identified conditions such as increased blood pressure, ventricular hypertrophy (muscle wastage) and arteriosclerosis (thickening and hardening of the artery walls).
- 2.18 The aim of this study was to monitor personal environmental noise dose, in this case mainly traffic noise as participants lived at least 10km from an airport, using a personal noise dosimeter (50 dBA to 120 dBA), and changes in vascular

properties using ambulatory non-invasive monitors worn on the wrist. Sixty six participants aged 18-32 years were recruited for the study from China Medical University in 2007. Participants answered a comprehensive screening questionnaire and refrained from smoking, caffeine, exercise, alcohol and listening to music for the duration of the study. The mean noise exposure for the daytime (0800-2300) was 61 dBA and for night-time (2300-0800) it was 48 dBA, with a 24 hour average of 56 dBA.

- 2.19 The results indicated that environmental noise exposure had temporary and sustained effects on vascular properties. There was an unexpected increase in arterial compliance during night-time, and a decrease in resistance during both daytime and night-time. Such changes were induced by present, 30 minute and 60 minute time-lagged noise exposures and contributed to the overall changes in vascular properties over a 24 hour period. The authors discuss possible theories as to why these changes occur, including as a reaction to stress-induced increased blood pressure, and interestingly as a possible response to noise-induced sleep disturbance. There was a lower arterial resistance at night-time compared with those measurements during the daytime among all subjects, which could explain the importance of noise disturbed sleep in cardiovascular diseases as sleep is an important modulator of cardiovascular function.
- 2.20 This is an important study as it is the first of its kind to provide evidence that environmental noise might affect structural changes in vascular properties that are related to hypertension. Although this study is not aircraft-noise specific it is possible that similar results may be observed as a response to aircraft noise and should be investigated in the future.
- 2.21 It is proposed that the physiological mechanism that is occurring may be that noise exposure causes the sympathetic and endocrine system to increase blood pressure. This then activates the muscular responses in arteries to allow for the surge of blood flow and to stabilise capillary pressure. It is these responses which produce the increased arterial compliance and decreased arterial resistance that subsequently prevent immediate damage within vessels.
- 2.22 In 2013 Babisch published a meta-analysis of noise and exposure-response curves between transportation noise and cardiovascular diseases. When considering epidemiological research approaches, Babisch stresses the importance of having a biological model for of how the noise exposure could affect health and the need for different research methods to be used to assess the impact rather than using the same methodology and therefore the same error, each time. He also discusses the possibility of a threshold of effect, which may arise due to biological reasons, or possibly due to imprecision in data and small sample sizes. There is a need for the magnitude of effect to have implications for public health, and only then if all of these factors are accounted

for should a quantitative risk assessment including cost-benefit analysis should be employed to influence any decision-making processes.

- 2.23 For long-term noise exposure, Babisch updated his 2002 diagram representing the possible pathways that lead to health outcomes as a result of noise. In view of the experimental findings indicating that people do not physiologically habituate to noise exposure, even after being exposed for many years and even when they do not consciously report any disturbance during sleep for example, his updated model considers two pathways. The first is a non-conscious pathway via direct interactions of the acoustic nerve with the central nervous system, and the second is a conscious pathway via indirect physiological activation due to the emotional and cognitive reaction towards the noise. The theory is that both pathways result in changes in the autonomic and endocrine systems, resulting in unbalanced physiological and metabolic function, which may then result in cardiovascular disease in the long term. Babisch suggests that the indirect pathway may be dominant in people who are awake, and the direct pathway becomes dominant during sleep, and at much lower sound levels. This theory is represented in Figure 1.
- 2.24 Babisch produced a meta-analysis of results from road traffic and aircraft noise studies. Pooled effect estimates were derived from other meta-analyses on road noise and hypertension (24 studies, van Kempen and Babisch, 2012), road traffic and myocardial infarction (5 studies, Babisch, 2008), and aircraft noise and hypertension (5 studies, Babisch and van Kamp, 2009). Road traffic noise and stroke (Sørensen et al, 2011), and aircraft noise and myocardial infarction (Huss et al, 2011) each only contained one study, but were included in the analysis.
- 2.25 The exposure-response relationships are shown in Figure 2, and represent estimated relative risk with increasing sound level. The curves indicate that there is a higher risk of approximately 20-40% for those people where the weighted average outdoor level at the façade of their houses exceeds 65 dBA. Babisch suggests that if the difference between day and night noise levels is considered to be approximately 7-11 dBA, the findings can be converted to a night time noise level of 55 dBA. It should be acknowledged that there are wide variations between the onset of the exposure-response relationships, from L_{den} of 40 dBA to 60 dBA. It is of interest that that aircraft noise MI is such a higher risk than road traffic MI, yet the situation is reversed for hypertension, which is a known risk factor of MI. One critical factor in this research is that the very low aircraft noise exposure presented is an underestimate or was in the presence of other noise sources that were not accounted for.

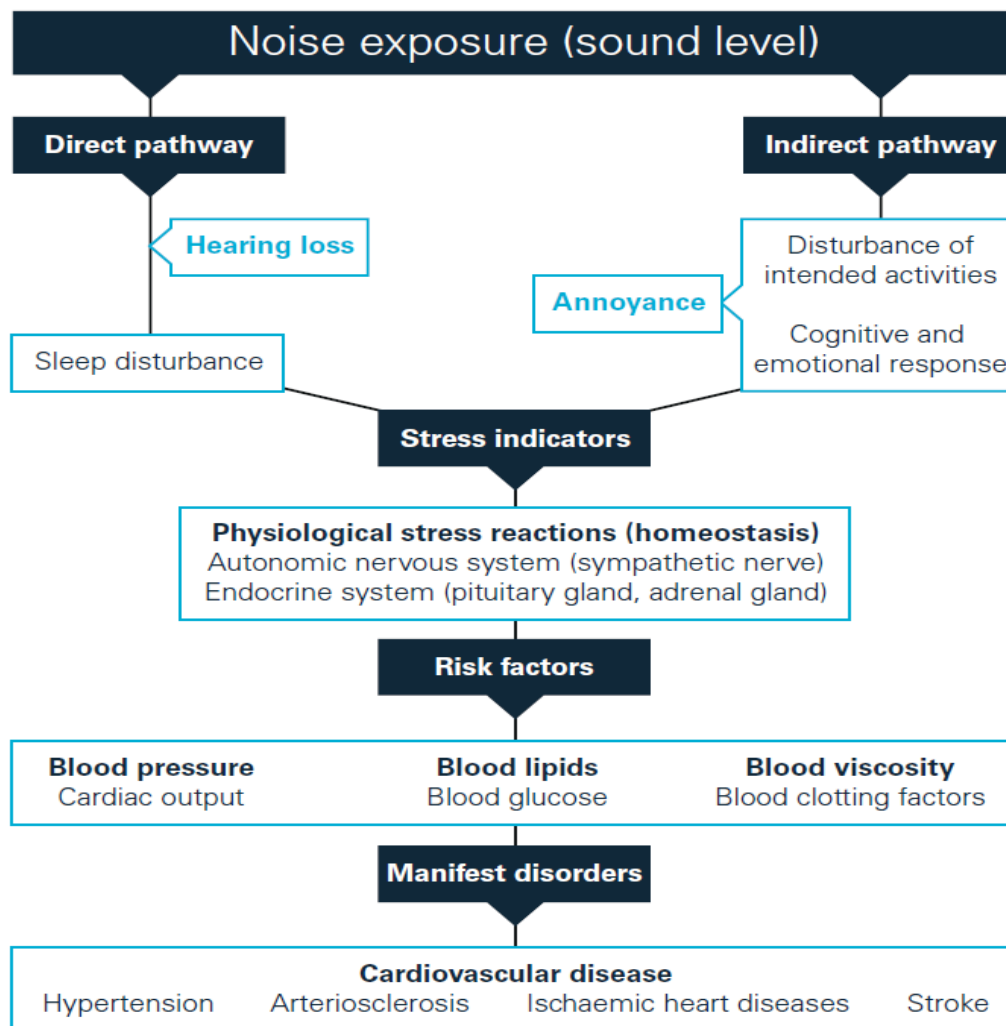


Figure 1: Noise reaction chart, updated version. Taken from Babisch, 2013.

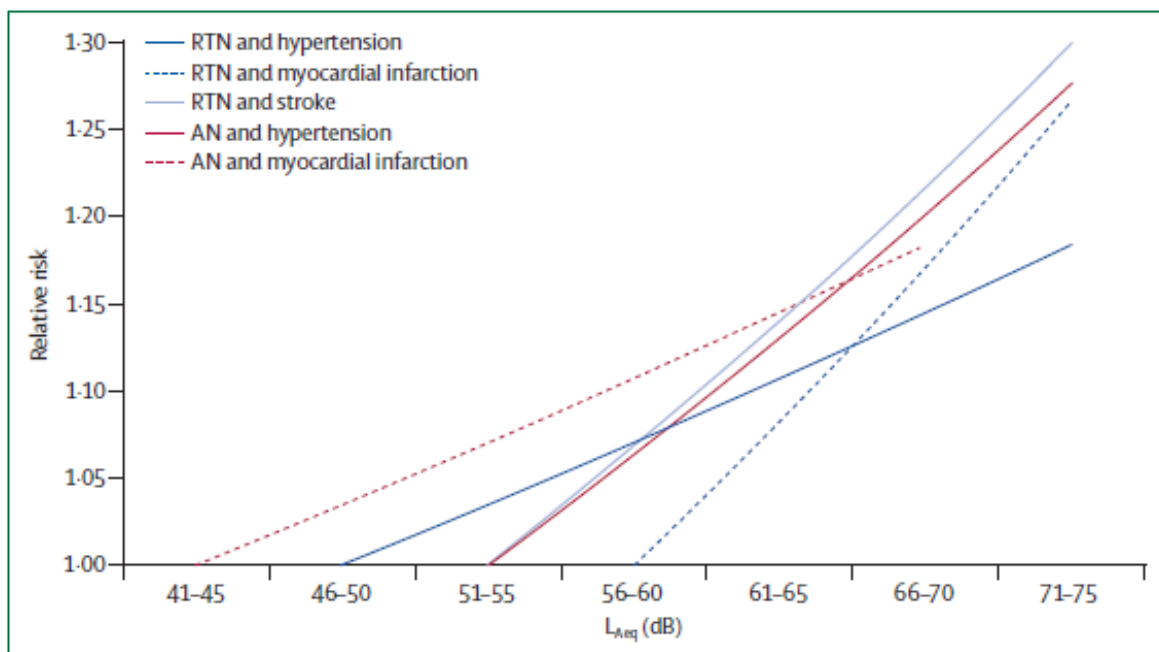


Figure 2: Exposure–response curves of road and aircraft noise and cardiovascular endpoints RTN and hypertension (24 studies, noise indicator LA_{eq} 16 h); RTN and myocardial infarction (five studies, noise indicator LA_{eq} 16 h); RTN and stroke (one study, noise indicator LDEN); AN and hypertension (five studies, noise indicator LDN); and AN and MI (one study, noise indicator LDN). RTN=road traffic noise. AN=aircraft noise.

- 2.26 Clearly, potential moderators and confounding variables need consideration in such research. These include location of rooms, windows being open or closed, length of residence, age, gender, and type of housing. Babisch suggests that future work should improve the noise assessment to consider secondary road networks and side streets, and quiet side dwellings should be included in the assessment. The issue of cumulative noise is important, i.e. it is critical to ensure that the dominant noise source is reflected in these types of studies. It is important that day-night differences should be investigated further, in relation to noise-induced sleep disturbance and development of cardiovascular diseases. Air pollution as a confounders or co-exposure also needs to be included in future work.
- 2.27 The findings from two UK studies focused around Heathrow airport were published in late 2013, and identified possible associations between aircraft-noise and health impacts on residents living in this vicinity.
- 2.28 The first was by Hansell et al (2013) from Imperial College, London which had the aim of investigating the association between aircraft noise and the risk of stroke, coronary heart disease and cardiovascular disease. The background to the research was that although there have been studies investigating cardiovascular effects of aircraft noise, the outcomes of those looking at stroke, coronary heart disease or cardiovascular disease are inconsistent. A possible reason for this may be due to a lack of statistical power because of the relatively small numbers of people exposed to high levels of aircraft noise.

- 2.29 This study examined comparisons between hospital admission rates for cardiovascular disease and mortality in neighbourhoods exposed to aircraft noise from Heathrow airport. Daytime (0700-2300) and night time (2300-0700) noise exposures were expressed as the average annual day LA_{eq} , 16 h and annual night LA_{eq} , 8 h respectively at a spatial resolution of 100 x 100 m, as estimated each year by the UK CAA and published by the Department for Transport. The study area included twelve London boroughs and nine districts to the west of London exposed to noise levels of at least 50dBA daytime (LA_{eq} , 16 h). For the twelve London boroughs data on air pollution in the form of particulate matter (PM10) at 20 x 20 m resolution, and road traffic noise at a spatial resolution of 10 x 10 m (LA_{eq} , 16 h) were also examined as potential confounding variables. Neighbourhoods were defined using the national census geographical units. The data on hospital admissions and deaths for 2001-2005 were obtained from the Office for National Statistics and Department of Health. The data for stroke, coronary heart disease and cardiovascular disease were then linked to postcode, geographic location and then noise exposure level. Confounders such as ethnicity, lung cancer (as a proxy for smoking) and deprivation were included.
- 2.30 Daytime aircraft noise and road noise was grouped into six categories from ≥ 51 to >63 dB in increments of 3 dB. For night time aircraft noise the increments were set at 5 dB intervals as less people were affected and categorised as ≤ 50 , >50 , and >55 dB. In order for a comparison between day and night time data to be made, daytime aircraft noise was also analysed using the same 5 dB groups. The study area covered 3.6 million people, only 2% living in the highest category of daytime or night time noise exposure.
- 2.31 The main findings on the hospital admissions with regard to stroke, coronary heart disease and cardiovascular disease are shown in Figure 3. With increased aircraft noise the risk of hospital admission also increased, with adjustment for ethnicity, deprivation and smoking included.

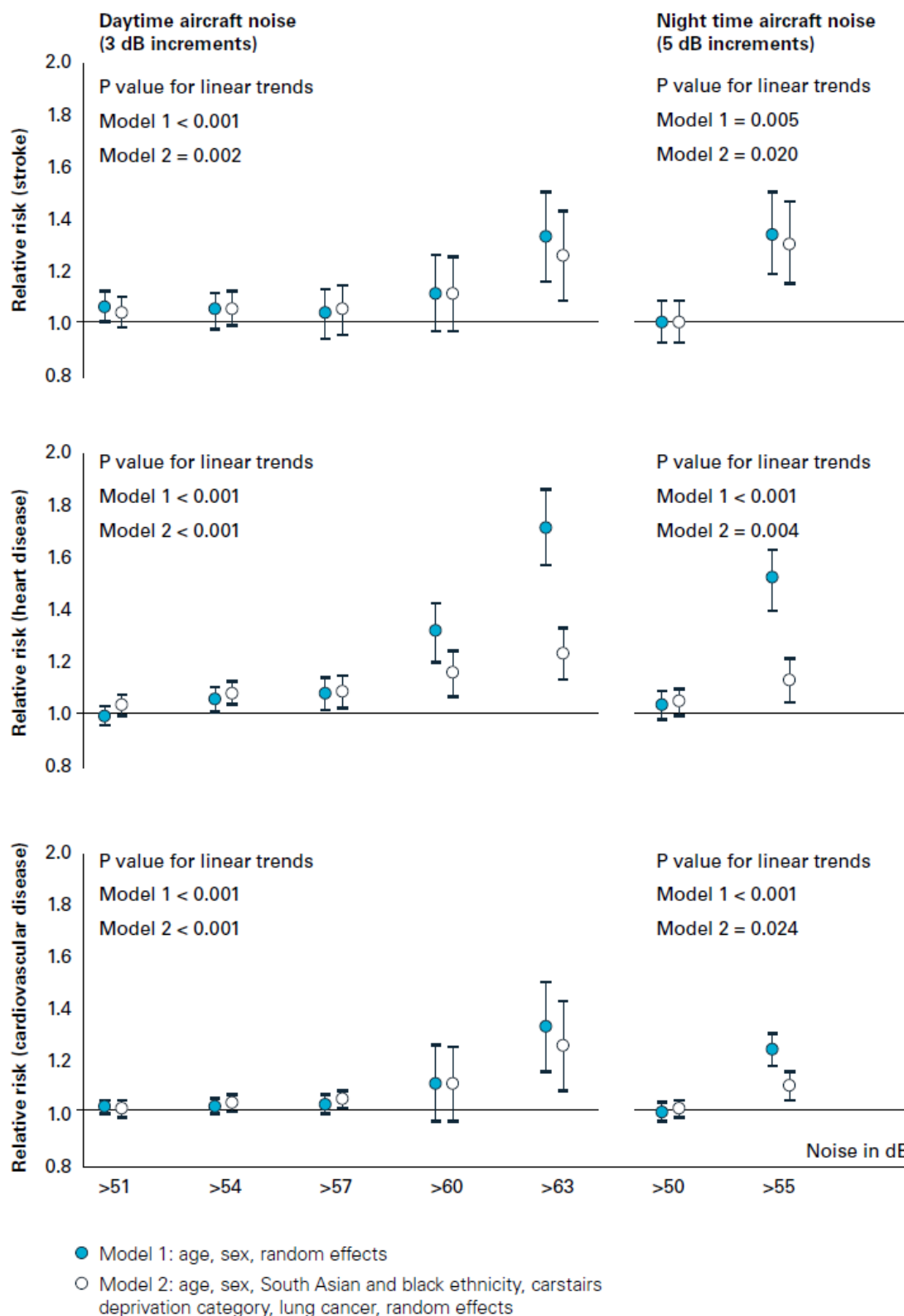


Figure 3: Relative risks for associations between hospital admissions for stroke coronary heart disease and cardiovascular disease between 2001 and 2005, and the annual weighted average daytime aircraft noise and night time aircraft noise in 2001 census output areas. Reproduced without permission from Hansell et al (2013).

- 2.32 The two sets of data illustrate the difference between the two types of adjustment for confounders. Model one represents adjustment for age, sex and random effects, and model two also includes ethnicity, deprivation and lung cancer. This separate analysis was chosen because the initial data highlighted that areas with a high proportion of South Asian and black ethnicity population were concentrated in the north eastern and eastern parts of the study area, which were also areas with higher deprivation and lung cancer risk.
- 2.33 Interestingly, adjustment for ethnicity, deprivation and lung cancer results in a much lesser degree of relative risk of hospital admissions particularly for coronary heart disease at noise exposure levels of more than 60 dB LA_{eq}, 16h. The same pattern is seen for cardiovascular disease, although to a lesser degree. It is important to consider the effect of ethnicity (in particular South Asian ethnicity, which is itself strongly associated with risk of coronary heart disease). The authors explained that when controlling for South Asian ethnicity in particular. It has a noticeable effect on these results, the effect due to noise exposure decreases quite dramatically. When comparing areas exposed to more than 63 dB LA_{eq}, 16 h to those exposed to 51 dB LA_{eq}, 16 h or less, the relative risk for hospital admissions due to stroke was 1.24 (1.08 to 1.43, 95% CI), for coronary heart disease was 1.21 (1.12 to 1.31, 95% CI) and for cardiovascular disease was 1.14 (1.08 to 1.20, 95% CI). The results for night time aircraft noise (>55 dB v ≤50 dB) were 1.29 (1.14 to 1.46, 95% CI), 1.12 (1.04 to 1.20, 95% CI) and 1.09 (1.04 to 1.14, 95% CI) respectively. When using the same categories for daytime and night time noise the results suggested higher relative risks for night time noise.
- 2.34 The corresponding results for relative risk of mortality were similar at the higher noise levels. In adjusted models for daytime aircraft noise (>63 dB v ≤51 dB) the relative risk for stroke mortality was 1.21 (95% confidence interval 0.98 to 1.49), for coronary heart disease was 1.15 (1.02 to 1.30), and for cardiovascular disease was 1.16 (1.04 to 1.29). The relative risks for night time aircraft noise (>55 dB v ≤50 dB) were 1.23 (1.02 to 1.26), 1.11 (0.99 to 1.24), and 1.14 (1.03 to 1.26) respectively. The results were unchanged with additional adjustment for PM10 and road traffic noise in the twelve boroughs of London. It was reported that the results obtained when using the same categories for daytime and night time aircraft noise indicated that the relative risks for mortality were higher for night time noise.
- 2.35 There are several issues to consider when interpreting the results from this study. Firstly, although road noise was included in the confounding variable analysis, rail noise was omitted which would have helped give a more representative group of noise confounders. Secondly, although the researchers have attempted to take into account the issue of confounding air pollution by including exposure to PM10, they did not include exposure to Nitrogen Dioxides (NO₂), possibly because NO₂ is primarily linked with respiratory disease rather

than cardiovascular disease. However, considering that NO₂ concentrations exceed EU Air Quality limits at a number of locations within the study area - including both factors would have enabled any confounding effects of air pollution to be more fully understood.

- 2.36 In terms of the noise categories, the increments ceased at 63 dB and above. It is unclear why this number was chosen as the cut-off point and levels such as 66 dB and 69 dB and above were all grouped together in this category and not analysed separately, even though there should have been sufficient population numbers in order to perform discrete analyses. It is possible, however, that this choice was due to statistical sampling issues, whereby there were not enough hospital admissions or mortality cases to be grouped into separate noise categories.
- 2.37 As mentioned briefly earlier, the differences in effect size between the two models is marked, especially so for relative risk of hospital admissions for all three outcomes but especially for coronary heart disease at exposure levels of more than 60 dB and more than 63 dB.
- 2.38 When looking at mortality risk, as opposed to risk of hospital admission, the relative risk actually decreases to less than 1.0, for the noise exposure between 57 and 60 dB LA_{eq}, 16h, for stroke and cardiovascular disease in both models, although this effect is more pronounced for stroke. This suggests the possibility of a further confounding variable that has not been taken into account. The results also suggest a higher risk of mortality from coronary heart disease than cardiovascular disease. This is counter intuitive given that cardiovascular disease encompasses all the diseases of the heart and circulation, including coronary heart disease and stroke along with heart failure and congenital heart disease. It would be expected that the largest effect would be seen for the category of cardiovascular disease, and stroke and coronary heart disease would show smaller effects, as they are subsets of this.
- 2.39 For the night noise data, the upper limit cut-off is noise exposure of at least 55 dB, but it is not explained as to why this is the case. This appears to encompass a large range of noise levels in just one category, for example the risk factor could occur at much higher levels such as 69 dB, yet there is no distinction to allow for this possibility within the analysis and it would benefit from the refinement of noise categories.
- 2.40 It is acknowledged within the paper that it was not possible to have access to individual level information on confounders such as smoking, so results at area levels may not be applicable to individuals. It was not possible for the study to distinguish between short and long term effects of noise and length of residency in this study, which would merit further research. A potential source of bias may be the lack of information concerning the migration in and out of the study areas.

- 2.41 The differences between night time noise and day time noise could not be distinguished due to their high degree of correlation. The authors suggested that further research is needed to assess whether night time noise affecting sleep may be contributing to the observed results. In addition to possible causal relationships between aircraft noise and cardiovascular outcomes, it is important to consider the potential for confounding and ecological bias in this study. An important area for further research would be to determine the relative contribution of night time noise compared with daytime noise to the respective health endpoint.
- 2.42 An independent review of this study was commissioned by the Department of Environment, Food and Rural Affairs (Defra) and conducted by Stansfeld et al (2014). The review concluded that the study added to the evidence supporting the link between aircraft noise, coronary heart disease, stroke and cardiovascular mortality yet the associations were inconsistent across all measures. The reviewers suggested that this may be due to the relatively small association between aircraft noise and cardiovascular risk and the various confounding issues that are inevitable found in studies of this nature.
- 2.43 Due to the fact that it was not possible to control for confounders on an individual level, it is important to note that the effect size reported may be subject to a degree of error. The reviewers recommended that the effect magnitudes reported in the study should not be used in subsequent economic analyses.
- 2.44 The second study that included health effects around Heathrow was by Floud et al (2013), again from Imperial College, London. This European study was an extension to the Hypertension and Environmental Noise near Airports (HYENA) study, using self-reported data on heart disease and stroke between 2004 and 2006 from 4,712 people living near six European airports. This study examined road traffic noise and aircraft noise around London Heathrow, Amsterdam Schiphol, Stockholm Arlanda and Bromma, Milan Malpensa, Berlin Tegel and Athens Elephtherios Venizelos with the aim of investigating whether there is an association between exposure to aircraft noise or road traffic noise and heart disease and stroke.
- 2.45 In the HYENA study residents around the given airport were exposed to ranges of noise levels between less than 50 dBA to more than 60 dBA $LA_{eq,16h}$. As part of the health questionnaire participants were asked to declare if they had ever been diagnosed with angina, myocardial infarction (MI) or stroke whilst at their current address. This represented the 'heart disease and stroke' factor within this study. Aircraft noise was estimated for annual average day time (0700-2300) $LA_{eq,16h}$ and night time (2300-0700) L_{night} and road traffic noise was estimated using the 24 hour metric $LA_{eq,24h}$. The lower limit cut-off levels were 35 dBA for daytime aircraft noise, 30 dBA for night time aircraft noise and 45 dBA for road traffic. The researchers appear to have chosen these very low noise exposures,

because the information seemed to be available. Such low exposure data have not been validated and are typically associated with long-distance sound propagation with associated large uncertainty. Secondly, the aircraft noise values are from aircraft noise sources alone. However, overall ambient noise exposure levels in urban and suburban areas rarely drop below 40dBA, so the cut-off levels are likely to be below ambient noise exposure levels in much of the study areas.

- 2.46 In those study, as a possible confounder, nitrogen dioxide (NO₂) was estimated at participants' addresses using dispersion modelling in the UK, Netherlands and Sweden.
- 2.47 The results indicated that 5.9% of the study population responded with self-reported heart disease and stroke, with the UK having the highest proportion of 8%. Night time aircraft noise was associated with self-reported heart disease and stroke but this effect was no longer present when controlled for confounding variables such as age sex, body mass index, education and ethnicity. Importantly, when the length of residence was included in the analysis, there was a significant association for those people who had lived at their current address for 20 years or more (odds ratio 1.25, 95% confidence intervals of 1.03 to 1.51) per 10 dBA increase in noise exposure. However, in contrast to night time noise, daytime aircraft exposure had no significant association with heart disease and stroke before and after controlling for confounders.
- 2.48 For road noise there was an increase in proportion of self-reported heart disease and stroke that remained after controlling for confounding variables, and length of residence did not appear to display effect modification for this noise source. Weak correlations were found between aircraft noise and NO₂ levels, with moderate correlations found between road noise exposure and NO₂. For participants who had lived at the same address for 20 years or more the association between night time noise and heart disease and stroke was significant after adjustment for NO₂. When NO₂ levels were factored into the analysis for subsamples of 24 hour road noise exposure, the significant association was lost, which suggested that NO₂ is a confounding variable in this relationship.
- 2.49 There are important points to consider when interpreting the results from this study. Firstly, the data are self-reported, which may lend itself to over or under-reporting and therefore increasing bias within the sample. Secondly, the lack of statistical significance between daytime aircraft noise and heart disease and stroke is striking and should not be overlooked. It was in fact close to zero association. Clearly this may be due to participants being away at work during the day and therefore not being necessarily exposed to the noise dose that their house receives during the day.

- 2.50 The finding that night time aircraft noise was not significantly associated with self-reported heart disease and stroke after adjustment for confounders is of significance. However, given the association for those residents who had lived at the same address for 20 plus years, the results suggest that the relationship between aircraft noise exposure at night may be strengthened over time, and could be cumulative in nature.
- 2.51 This study found that associations between road noise and heart disease and stroke were confounded by air pollution, although the associations between aircraft noise and heart disease and stroke remained robust even after adjustment for NO₂. This is not unexpected, since road traffic is the predominant contributor to NO₂ pollution exposure. In addition the results suggested that for road traffic noise and heart disease and stroke, age may be a modifier as an association was found for those participants aged over 65 years. This probably needs to be investigated further however, in larger samples with increased power and the inclusion of air pollution as a co-exposure.
- 2.52 Although this study attempted to analyse air pollution as a confounding variable, the choice to use NO₂ alone does not fully represent the effects of air pollution, as particulate matter is also associated with transport emissions. Finally, although education level was controlled for in this study, socioeconomic status such as income or area-level deprivation was not taken into account and may also be a confounding factor.
- 2.53 This study provides a valuable insight into the associations between road traffic and aircraft noise and these particular health outcomes. Although the results suggest a possible long-term effect of night time aircraft noise (>20 years) on self-reported heart disease and stroke, the possibility of bias and further confounding issues should be considered carefully. In terms of road traffic noise and heart disease and stroke it is important to take into account the possible confounder of air pollution and age as an effect modifier before any firm conclusions can be drawn.
- 2.54 In addition to the two UK studies a US study was recently published by Correia et al (2013) from Boston School of Public Health and Harvard University, investigating aircraft noise exposure and hospital admission rates.
- 2.55 The aim was to investigate whether aircraft noise exposure is linked with hospital admissions due to cardiovascular disease in people of 65 years of age or older. The sample population was Medicare enrollees that lived close to 89 airports within the US. In total just over 6 million people aged 65 or more, enrolled in Medicare and residing in the 2,218 postcodes close to the 89 airports were studied. This sample size corresponds to approximately 15% of the entire US population of older people. The researchers used information from the Medicare insurance claims to analyse details such as when participants were admitted, length of stay, primary reason for admission, age, sex, ethnicity and postcode. In