BEL TECHNICAL REPORT. BEL 2008 - 001.
Version 2.0. March 3rd 2008

TECHNICAL REPORT

EFFECT OF NOISE ON PHYSICAL HEALTH RISK IN LONDON

REPORT ON PHASE 1 – REVIEW OF THE TOPIC

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VERSION 2.0 MARCH 3rd 2008

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ABSTRACT

The potential effects of noise on cardiovascular health have been studied for many years, and possible causal mechanisms, and "models" have been proposed.

In this report, the evidence for such effects, and possible "exposure-response relationships" have been reviewed systematically - starting from the basis of a number of previous reviews published between 1998 and 2006 by acknowledged experts in the field – but also including more recent specific research studies.

This report forms the basis of the next phase of the project, in which such an exposure-response relationship will be applied to estimate of the numbers of people at cardiovascular and related health risk, from road traffic and airport noise in London.

CONTENTS

EXECUTIVE SUMMARY

Berry Environmental Ltd [BEL] was commissioned, in December 2007, by the Noise Strategy Team at the Greater London Authority [GLA] to;

- Provide guidance on the robustness of published cardiovascular risk factors arising from ambient/environmental noise, using the latest available World Health Organisation (WHO) reviews, and other reviews of evidence/criteria, and to suggest best estimates of the factors from the range of published values.

- Use derived factors to generate estimates of the numbers of people at cardiovascular and related health risk, from road and air traffic noise, in London, from available population exposure data.

This Report covers the initial review phase of the project.

The potential effects of noise on cardiovascular health have been studied for many years, and possible causal mechanisms, and "models" have been proposed. Section 2 of this Report defines "*cardiovascular effects*" and gives a brief outline of some of these proposed "mechanisms" and "models".

This section then presents a Summary Table [Section 2.3] in which the key points from previous reviews are highlighted. This enables an overview of how the evidence has developed over recent years, to the extent that it can be summarised as ;

Biochemical effects:	*limited evidence*
Hypertension:	*limited or sufficient evidence*
Ischemic Heart Disease	*sufficient evidence*

A detailed overview of these previous published reviews of the topic is then given in Section 2.4, which looks at the evidence for such effects, and possible "exposure-response relationships", starting from the basis of a number of previous reviews published between 1998 and 2006 by acknowledged experts in the field. [Section 2.4]

More recent studies involving Road Traffic Noise and Aircraft Noise have also been identified and summarised [Section 3]. These more recent studies have examined a range of issues including the role of Air Pollution, the effects of **changes** in noise environment, specific night-time noise factors, the role of annoyance, effects on children, and a wider range of possible confounding factors such as social welfare status etc. Such studies have also introduced new research techniques in which, for example, the health risk is mapped as standardized morbidity ratio in space and time, allowing, the (changing) spatial pattern around an airport to be visualized and possible relationships with environmental quality quantified.

Such studies are helping to improve our knowledge – but further more detailed examination of these studies, and how their results can be applied, are required.

For Road Traffic Noise, an "exposure-response relationship" has been proposed, between noise level and risk of Myocardial infarction MI, and in fact this has been applied by Wolfgang Babisch to estimate the potential number of people at risk of cardiovascular effects from road traffic noise in Germany.

For Aircraft noise, there have as yet been an insufficient number of high quality studies on which to base such an exposure-response relationship for Myocardial infarction, but it has been argued by Babisch, that the relationship developed for Road Traffic Noise should be used as an “approximation”.

Account has been taken of a very recently issued Draft Chapter on Cardiovascular effects, by Babisch and Irene van Kamp - Version 2 Draft January 2008, for a new Report being prepared for the World Health Organisation [WHO] Working Group - Aircraft noise and health. [Section 4]

In the next phase of the project, these relationships can be applied to the specific “scenarios” of London Heathrow Airport, London City Airport and road traffic noise [using London Noise Maps etc]. A second report will present the results of these calculations to estimate of the numbers of people at cardiovascular and related health risk

The field is a very “active” one, with new research studies in progress and in preparation, and with a number of other organisations, such as WHO conducting further reviews. It is clear that there is considerable scope for further “monitoring” of the subject, and additional review/analysis.

1. INTRODUCTION - AIMS ETC

Berry Environmental Ltd [BEL] was commissioned, in December 2007, by the Noise Strategy Team at the Greater London Authority [GLA] to:

- Provide guidance on the robustness of published cardiovascular risk factors arising from ambient/environmental noise, using the latest available World Health Organisation (WHO) reviews, and other reviews of evidence/criteria, and to suggest best estimates of the factors from the range of published values.
- Use derived factors to generate estimates of the numbers of people at cardiovascular and related health risk, from road and air traffic noise, in London, from available population exposure data.

For convenience, the full Brief is given at Annex 1 to this Report.

The stated Aims are;
"1. Briefly review and list the latest evidence and published policies and/or guidance (adopted or proposed) of WHO and public bodies or authorities in other countries on the link between noise and physiological health impacts, providing a view on the robustness of the risk factors they use or currently propose and suggest best estimates of the risk factors and probable margins of error, and summarising plausible causal pathways to assist in popular interpretation of estimates. As far as possible, this should apply a standard of evidence similar to that applied to equivalent risk factors for air quality impacts.
2. Apply the suggested risk factors to available London population noise exposure data derived from maps for road traffic noise, Heathrow and London City Airports to determine overall impacts.
3. Make summary comparisons between the best estimate risk factors and outcomes for two other health impacts from road traffic in London (e.g. air pollution impacts and road traffic accidents), to place the noise outcomes in context.
4. Clearly explain the methodology used to derive the risk factors and to apply these to the population exposure data to derive overall numbers of premature deaths, QALY/DALYS and any other outcomes analysed, so that the client can derive and apply revised factors in future as scientific understanding advances and/or new population exposure data become available."

This Report covers the initial review phase of the project.

The present Report is arranged in 4 further Sections.

- Section 2 defines "*cardiovascular effects*" and gives a brief outline of some of the proposed "mechanisms" and "models" underlying the effects of noise on the human cardiovascular system. This section then presents a Summary Table in which the key points from previous reviews are highlighted. A detailed overview of these previous published reviews of the topic is then given in Section 2.4. Where possible URLs for these reports and papers are given.
- Section 3 outlines a number of more recent specific research studies.
- Section 4 deals with the very recent, as yet unpublished Chapter on *Cardiovascular effects of Aircraft Noise,* by Wolfgang Babisch and Irene van Kamp for the new WHO WG on Aircraft Noise and Health [see also Annex 3].

- Section 5 provides a **Summary and overall conclusions** on the available evidence, as a basis for subsequent calculations in the next phase of the project.
- Section 6 lists the relevant reference documents.

Annex 2 lists studies and reviews known to be in progress but as yet unpublished. This is to ensure such work is documented to allow for possible updating of the present report.

Annex 3 provides the full text of the WHO - Working Group – Aircraft noise and health - Chapter by Babisch and van Kamp - Version 2 Draft January 2008

Annex 4 provides a Glossary of terms.

Where possible, copies of all the key reference publications are being provided to the GLA – but many are available online.

NOTES

1. The Final Report will cover the issue of *"guidance (adopted or proposed) of WHO and public bodies or authorities in other countries on the link between noise and physiological health impacts"*

2. The Final Report will also include examples of calculations of "burden of disease" due to noise.

2. PREVIOUS REVIEWS

2.1 GENERAL

In the past ten years or so, there have been a number of important reviews of the state of our knowledge and the "strength of evidence" on possible effects of noise on the human cardiovascular system. These will be summarised below, but, as a first step, in order to put such evidence in context, it is useful to consider possible "mechanisms" or "models" underlying the effects of noise on people and on the human cardiovascular system in particular.

2.2 CARDIOVASCULAR EFFECTS OF NOISE - POSSIBLE MECHANISMS

The general term *"cardiovascular effects* " is taken to range from short-term acute physiological effects such as raised blood pressure, heart rate changes and the release of stress hormones, through to more chronic effects such as hypertension [chronically elevated blood pressure] , and ischaemic heart disease. The latter includes clinical symptoms of angina pectoris (chest pain), myocardial infarction (heart muscle damage), or electrocardiogram (ECG) abnormalities.

The complexity of the overall situation, and the extent of the inter-relationships between many factors which influence health, is clear from the model proposed by the Australian researcher, Soames Job in 1996 [1]. See Figure 1 below.

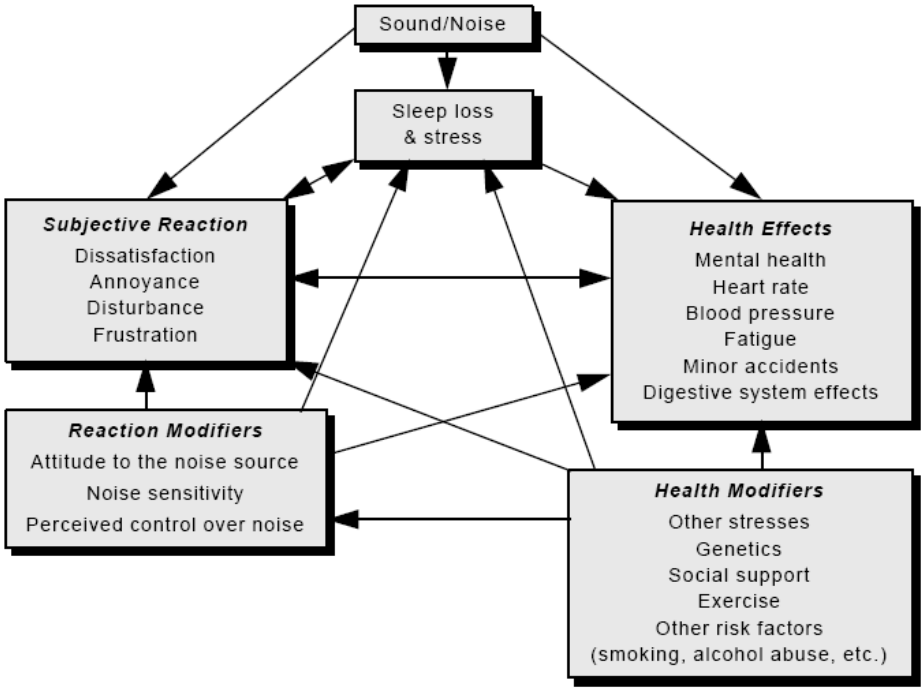

Figure 1. From Job 1996

In a report published by the National Institute for Public Health [RIVM] in the Netherlands [2], the authors outlined possible mechanisms as follows

"Long term noise exposure is associated with a number of effects on health and well-being. These include community responses such as annoyance, sleep disturbance, disturbance of daily activities and performance, and physiological effects such as

hearing loss, hypertension and ischemic heart disease (Berglund et al., 1999). Although there is much discussion about how noise can affect human health, it is hypothesised that stress plays an important role. A model of the Dutch Health Council points out the complexity between noise and health (HCN, 1999) (Figure 2). The model assumes that health status is determined by a combination of endogenous and exogenous factors such as the physical and social environment and life style. Noise exposure is only one of these exogenous factors. This process may be modified by personal characteristics such as attitude and coping style. Noise exposure induces disturbance of sleep and daily activities, annoyance and stress, which may lead to all sorts of intermediate responses, such as hypertension. In turn, these may affect the risk on cardiovascular disease or psychiatric disorders.

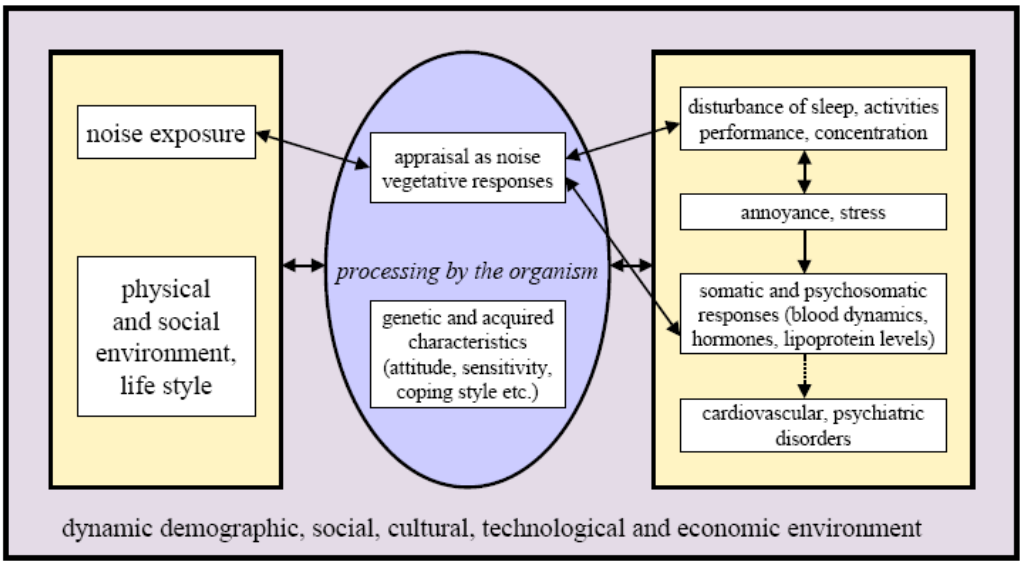

Figure 2. From RIVM 2005

To focus more specifically on cardiovascular effects, it should be noted that, in his comprehensive review of the subject published in 2006 [3], Wolfgang Babisch summarised the possible mechanisms as follows;

"Noise is an unspecific stressor that arouses the autonomous nervous system and the endocrine system. The generalised psycho-physiological concept given by Henry and Stephens can be applied directly to noise-induced stress reaction (Henry 1992). The stress-mechanism as such is genetically determined. It may be modified by experience and environmental factors. Its biological function is to prepare the organism to cope with a demanding stressor. Any arousal of the sympathetic and endocrine system is associated with changes in physiological functions and the metabolism of the organism, including blood pressure, cardiac output, blood lipids (cholesterol, triglycerides, free fatty acids, phosphatides), carbohydrates (glucose), electrolytes (magnesium, calcium), blood clotting factors (thrombocyte aggregation, blood viscosity), leukocyte count and others (Cohen et al. 1995; Friedman and Rosenman 1975; Lundberg 1999). In the long term functional changes and dysregulation due to changes of physiological set points may occur, thus increasing the risk of manifest diseases. Since many of the mentioned factors are known to be classical cardiovascular risk factors, the hypothesis has emerged that chronic noise exposure increases the risk of hypertension, arteriosclerosis and ischaemic heart disease."

Figure 3 shows a “reaction schema”, or model, proposed by Babisch.

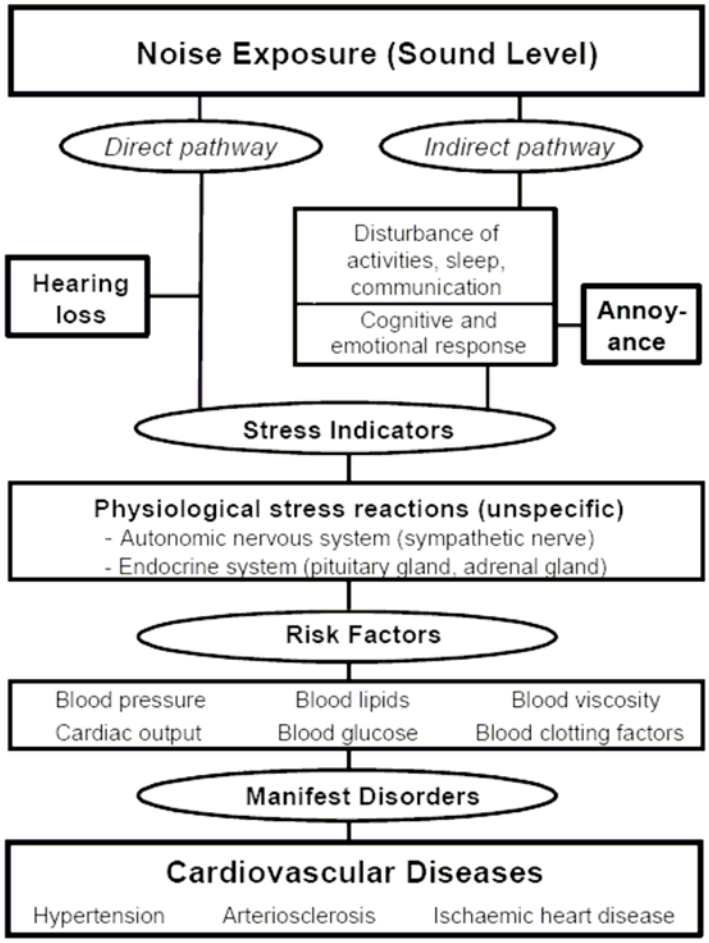

Figure 3. From Babisch [Reference 3]

Babisch commented that *“It simplifies the cause-effect chain i.e.: sound - annoyance (noise) -physiological arousal (stress indicators) - (biological) risk factors - disease - and mortality (the latter is not explicitly considered in the graph). The mechanism works 'directly' through synaptic nervous interactions and 'indirectly' through the emotional and the cognitive perception of the sound. It should be noted that the 'direct' pathway is relevant even at low sound levels particularly during sleep, when the organism is at its nadir of arousal. The objective noise exposure (sound level) and the subjective noise exposure (annoyance) may serve independently as exposure variables in the statistical analyses of the relationship between noise and health endpoints.”*

2.3 SUMMARY TABLE OF PREVIOUS REVIEWS.
The following Section, 2.4, provides a detailed overview of several previous reviews.

But**,** in order to summarise the various conclusions in these previous reviews over the last 9 or 10 years, and to see how the status of evidence has developed and "evolved ", the Key Points from each review are given in the Table below – in chronological order.

TABLE 1. SUMMARY OF PREVIOUS REVIEWS

REVIEW	KEY POINTS
Porter, Berry and Flindell 1998. Health effects-based noise assessment methods; a review and feasibility study. NPL Report CMAM 16.	…**weak evidence** that environmental noise exposure may contribute to non-auditory health effects such as cardio-vascular disease. …**evidence** for real effects contributing to increased morbidity such as cardiovascular effects is **not convincing** at this time, although it seems scientifically plausible that a minority of the population exposed at the highest noise levels might be susceptible to some increased risk.
W Babisch. 2000. *Traffic noise and cardiovascular disease: epidemiological review and synthesis.* Noise Health 2000; 2(8):9-32.	only a limited number of epidemiological studies available on the relationship between traffic noise and cardiovascular diseases. **no epidemiological evidence** of a relationship between noise exposure and mean blood pressure readings in adults. - noise-related increases in blood pressure are consistently seen in children. **hypertension** as a clinical outcome - there is **little evidence** that exposure to high traffic noise levels is associated with an increased risk. **ischaemic heart disease** - there is **some evidence** in the literature of an increased risk in subjects who live in noisy areas with outdoor noise levels of greater than 65-70 dB (A).
Health Canada 2001 Noise from Civilian Aircraft in the Vicinity of Airports – Implications for Human Health. I. Noise, Stress and Cardiovascular Disease.	..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 dB (A).
Stansfeld et al., 2001 Rapid Review of noise and Health in London Report to GLA April 2001	**..aircraft noise** exposure was associated with increased medical treatment, hypertension, and increased use of cardiovascular drugs [1977 study] ..the association between **road traffic noise** and risk factors was *"inconsistent and may be confined to groups annoyed by noise"*. The overall conclusion was that the risk of coronary heart disease associated with **aircraft and road traffic** noise was **small,** especially compared to other factors such as smoking
E. E. M. M. van Kempen, H Kruize, H C. Boshuizen, C B. Ameling, B A. M. Staatsen, and A E.M. de Hollander *The Association between*	..significant association for air traffic noise exposure and **hypertension**: [1977 study] Air traffic noise exposure **positively associated** with the consultation of a general practitioner or specialist, the use of

Noise Exposure and Blood Pressure and Ischemic Heart Disease: A Meta-analysis. Environmental Health Perspectives • VOLUME 110 | NUMBER 3 | March 2002 307	cardiovascular medicines, and angina pectoris. In cross-sectional studies, road traffic noise exposure increases the risk of myocardial infarction and total ischemic heart disease. ..the **evidence** for a relation between noise exposure and **ischaemic heart disease** is **still inconclusive** because of the limitations in exposure characterization, adjustment for important confounders, and the occurrence of publication bias.
E. E. M. M. van Kempen, B. Staatsen, I. van Kamp ***Selection and evaluation of exposure-effect relationships for health impact assessment in the field of noise and health.*** **RIVM report 630400001/2005**	… in the period between 2000 and 2005, results of new noise studies investigating the effects of road traffic, air traffic and rail traffic noise on cardiovascular disease had come out. They suggest that the conclusions from these newer studies did not really differ from what is already found in the published reviews on this topic.
W. Babisch 2006. ***Transportation Noise and Cardiovascular Risk. Review and Synthesis of Epidemiological Studies. Dose-effect Curve and Risk Estimation*** **Federal Environment Agency UBA . Berlin 2006**	**Hypertension** With regard to the association between community noise and hypertension, the picture is heterogeneous. • **aircraft noise and hypertension-** studies consistently show higher risks in higher exposed areas. The evidence has improved since the previous review. • **Road traffic noise,** the picture remains unclear. Across all studies no consistent pattern of the relationship between community noise and prevalence of hypertension can be seen. **IHD [MI]** With regard to IHD, the evidence of an association between community noise and IHD risk has increased since a previous review. ***Exposure-response curve now proposed –*** L_{day} ***and MI*** **Overall** With regard to hypertension and ischaemic heart disease, new studies with improved control for confounding factors, point in the direction of a positive association between community noise and CVD endpoints in adults. **Biochemical effects: limited evidence** **Hypertension: limited or sufficient evidence** **IHD: sufficient evidence**
H Miedema.2007 ***Exposure-response relationships for environmental noise*** **Proc Internoise 2007 Paper 07-179 CDROM**	Uses the 2006 Babisch report to propose Exposure-response relationship between L_{den} **and MI.** Part of a "set" of such curves – also Annoyance, Sleep etc

2.4 PUBLISHED REVIEWS

2.4.1

N D Porter, I H Flindell, B F Berry 1998.
***Health effects-based noise assessment methods; a review and feasibility study*. NPL Report CMAM 16.**
Although there were earlier reviews, such as the 1994 and 1997 publication of the Netherlands Health Council [4, 5], we will take as a starting point the review conducted at NPL for the then DETR. [6]

Basically this "assimilated" a number of earlier reviews which had all considered the complete range of health effects including;
Annoyance
Sleep
Performance
Mental Health
Stress
Cardiovascular
These reviews had assessed the "strength of evidence" in terms of in terms of the categories proposed by the International Agency for Research on Cancer (IARC) as 'sufficient', 'limited', 'inadequate' or 'lacking'.

Sufficient: a relationship has been observed between noise exposure and a specific health effect. Chance, bias and confounding factors can be ruled out with reasonable confidence.
Limited: an association has been observed between noise exposure and a specific health effect. Chance, bias and confounding factors cannot be ruled out with reasonable confidence
Inadequate: the available studies are of insufficient quality, lack the consistency or statistical power to permit a conclusion regarding the presence or absence of a causal relationship.
Lacking: several adequate studies are mutually consistent in not showing a positive association between exposure and health effect

The NPL Report concluded that:

- *There is some weak evidence that environmental noise exposure may contribute to non-auditory health effects such as cardio-vascular disease.*
- *The evidence for real effects contributing to increased morbidity such as cardiovascular effects is not convincing at this time, although it seems scientifically plausible that a minority of the population exposed at the highest noise levels might be susceptible to some increased risk.*

2.4.2

W Babisch. 2000. *Traffic noise and cardiovascular disease: epidemiological review and synthesis.* Noise Health 2000; 2(8): 9-32.
http://www.noiseandhealth.org/article.asp?issn=1463-1741;year=2000;volume=2;issue=8;spage=9;epage=32;aulast=Babisch;type=0

The abstract of this early specific review by Babisch [Reference 7] reads as follows:

"Compared to other environmental issues, only a limited number of epidemiological studies is available on the relationship between traffic noise and cardiovascular

diseases. The available literature provides no epidemiological evidence of a relationship between noise exposure and mean blood pressure readings in adults. However, noise-related increases in blood pressure are consistently seen in children. As far as hypertension as a clinical outcome is concerned, there is little evidence that exposure to high traffic noise levels is associated with an increased risk. With regard to ischaemic heart disease there is some evidence in the literature of an increased risk in subjects who live in noisy areas with outdoor noise levels of greater than 65-70 dBA."

2.4.3
Health Canada 2001
Noise from Civilian Aircraft in the Vicinity of Airports – Implications for Human Health. I. Noise, Stress and Cardiovascular Disease
http://www.hc-sc.gc.ca/ewh-semt/pubs/noise-bruit/01hecs-secs256/index_e.html
In 2001 the Canadian Government department Health Canada published this review – specifically on cardiovascular effects [8]. The review concluded as follows:

"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."

2.4.4
Stansfeld et al., 2001
Rapid Review of Noise and Health in London
Report to GLA, April 2001

This review [9] aimed to provide an accessible evidence base to inform the development of a Health Impact Assessment as part of the Mayor of London's Ambient Noise Strategy. It aimed to answer a range of key questions.
Effects considered were – Annoyance and Quality of Life, Sleep disturbance, Mental Health, and Cardiovascular disease. A separate section covered Children's health.

On Cardiovascular disease, having noted that much of the evidence comes from studies of exposure to noise in industrial occupational situations, the review considered a small number of "community noise "studies. These were the aircraft noise studies in the Netherlands reported by Knipschild et al in 1977 and the Caerphilly Collaborative Heart Disease study on road traffic noise as reported by Babisch in 1988 and 1999.

The review noted that, in the case of the Netherlands studies, although aircraft noise exposure was associated with increased medical treatment, hypertension, and increased use of cardiovascular drugs, it was difficult to rule out population selection effects. On the Caerphilly study it was noted that, whilst a small increase in "coronary events" could be detected in relation to road traffic noise, the association between noise and risk factors was *"inconsistent and may be confined to groups annoyed by*

noise". [Note - This aspect is taken up later in more recent papers by Babisch, see Section 3]
The overall conclusion was that the risk of coronary heart disease associated with aircraft and road traffic noise was small, especially compared to other factors such as smoking

2.4.5
E. E. M. M. van Kempen, H Kruize, H C. Boshuizen, C B. Ameling, B A. M. Staatsen, and A. E.M. de Hollander.
The Association between Noise Exposure and Blood Pressure and Ischemic Heart Disease: A Meta-analysis.
Environmental Health Perspectives • VOLUME 110 | NUMBER 3 | March 2002 307
http://www.ehponline.org/members/2002/110p307-317vankempen/EHP110p307PDF.PDF

To investigate the relationship between noise exposure (both occupational and community noise) and blood pressure and/or ischaemic heart disease, this detailed meta-analysis of 43 studies, carried out between 1970 and 1999, was completed and published in 2002 [Reference 10] . It covered a wide range of effects, varying from blood pressure changes to myocardial infarction.

The authors' summary was given as follows:

"Our meta-analysis showed a significant association for air traffic noise exposure and hypertension: We estimated relative risks per 5 dB (A) noise increase of 1.26 (1.14–1.39). Air traffic noise exposure was positively associated with the consultation of a general practitioner or specialist, the use of cardiovascular medicines, and angina pectoris.
In cross-sectional studies, road traffic noise exposure increases the risk of myocardial infarction and total ischemic heart disease.
Although we can conclude that noise exposure can contribute to the prevalence of cardiovascular disease, the evidence for a relation between noise exposure and ischemic heart disease is still inconclusive because of the limitations in exposure characterization, adjustment for important confounders, and the occurrence of publication bias.

The 2002 RIVM meta-analysis was itself reviewed and summarised by Babisch in his later 2006 review [reference 3].

He wrote as follows [minor edits added];

*"In this meta-analysis it was concluded that the relative risk of **hypertension** due to **aircraft noise** was 1.26 per increase of 5 dB (A) (95% CI: 1.14-1.39, L_{day} = 55 to 72 dB (A)). But in fact only one study was considered in the meta-analysis for the risk estimate. When one looks into the noise exposure assessment as described in the bibliographic reference of this study, this suggests two clusters rather than a continuous distribution of noise levels. This raises some concern when fitting the data using a continuous regression model.*

*For **road traffic noise and hypertension**, a pooled estimate of the relative risk of 0.95 per 5 dB (A) (95% CI: 0.84-1.08, L_{day} = <55 to 80 dB (A)) was calculated.*
Two cross-sectional studies were considered in this calculation.

*With regard to **ischaemic heart disease** across the studies, there was not much indication of an increased risk for subjects who lived in areas with a daytime average sound pressure level of less than 60 dB(A). For higher noise categories, higher risks were relatively consistently found amongst the studies. However, statistical significance was rarely achieved.*

*In this meta-analysis, where two cross-sectional studies were considered (Babisch et al. 1993), it was concluded that the relative risk of **ischaemic heart disease** was 1.09 per 5 dB (A) of the road traffic noise level (95% CI: 1.05-1.13, L_{day} = 51-70 dB (A)). However, the pooled estimate of the relative risk of two prospective studies (Babisch et al. 1999) was calculated to be 0.97 per 5 dB (A) (95% CI: 0.90-1.04, L_{day} = 51-70 dB (A).*
*When the diagnosis of **ischaemic heart disease** was limited to **myocardial infarction,** three studies were considered in this meta analysis (Babisch et al. 1999; Babisch et al. 1994). The linear effect estimate of the relative risk was 1.03 per 5 dB (A) increase in **road traffic noise** level (95% CI: 0.99-1.09, L_{day} = 51-80 dB (A)).*

2.4.6
E. E. M. M. van Kempen, B. Staatsen, I van Kamp. 2005.
Selection and evaluation of exposure-effect relationships for health impact assessment in the field of noise and health.
RIVM report 630400001/2005
http://www.rivm.nl/bibliotheek/rapporten/630400001.html

This report is a "*background document that can be used to assess the health impact attributable to noise in the Netherlands*" [Reference 2].
Available exposure-effect-relationships in the field of noise and health are reviewed and evaluated, using data published in the epidemiological literature as well as previous reviews. Their applicability for assessment in the Netherlands is demonstrated in case-studies.

In the part on "Effects on the Cardiovascular system", the RIVM report initially reviews earlier studies and meta-analyses – primarily the 2002 meta-analysis by van Kempen – summarised in 2.3.4 above.

The RIVM authors go on to point out that, in the period between 2000 and 2005, results of new noise studies investigating the effects of road traffic, air traffic and rail traffic noise on cardiovascular disease had come out. They suggest that the conclusions from these newer studies did not really differ from what is already found in the published reviews on this topic.

They note that the effect of night-time noise exposure had now been investigated and that the effects of air pollution were also taken into account.
The results of a German survey showed that night-time noise exposure was more strongly associated with medical treatment for hypertension than day-time noise exposure (reference 11. Maschke, 2003).

A time-series analysis of hospital admission data in Madrid in the period 1995-1997 showed a clear association between emergency admissions for all and specific (circulatory, respiratory) causes and environmental noise levels (61-72 dB (A)). Other explanatory factors such as air pollution levels were controlled for in the models. About 5% of all emergency admissions could be attributed to high noise levels (12. Tobias et al., 2001).

2.4.7
W. Babisch 2006.
Transportation Noise and Cardiovascular Risk. Review and Synthesis of Epidemiological Studies. Dose-effect Curve and Risk Estimation
Federal Environment Agency UBA. Berlin 2006
http://www.umweltdaten.de/publikationen/fpdf-l/2997.pdf

See also Babisch, W., *Transportation noise and cardiovascular risk: Updated review and synthesis of epidemiological studies indicate that the evidence has increased.* Noise and Health, 2006. 8: p. 1-29.
http://www.noiseandhealth.org/temp/NoiseHealth8301_095053.pdf

To date, these publications represent **the most comprehensive analytical review and summary of the evidence** [Reference 2 and 13].

Babisch himself first summarises earlier reviews, including those by the Netherlands Health Council, [4], Porter et al [6], van Kempen [10] etc., and his own previous reviews [7]
He notes that the status of evidence of the relationship between transportation noise and cardiovascular health as concluded in the earlier literature could be summarized as follows

- ***Biochemical effects: limited evidence***
- ***Hypertension: inadequate or limited or sufficient evidence***
- ***Ischaemic heart disease: limited or sufficient evidence***

Babisch went on to identify 61 epidemiological studies which had investigated the relationship between transportation noise and cardiovascular health endpoints, assessed either objectively or by self-reports. Overall, 20 of these studies refer to commercial aircraft noise, and 32 to road traffic noise.

Thirty-seven of those 61 studies had assessed the prevalence or incidence of manifest diseases, including hypertension and ischemic heart diseases (angina pectoris, myocardial infarction, ECG abnormalities).

The various studies and their characteristics are listed in a number of tables, covering each of the health outcomes or endpoints, e.g. Blood Pressure, Hypertension, Ischemic Heart Disease, Drug use etc

The tables set out risk estimates [OR etc] derived from the individual
Studies, in 5 dB (A) categories of the average A-weighted sound pressure level during the day.

For this present review report, it is convenient to concentrate on the overall picture, firstly for; **Hypertension,** and then for **Ischemic Heart Disease IHD [including Myocardial infarction MI].**

Aircraft noise studies and Road Traffic studies are dealt with separately.

Hypertension

18 studies were considered and analysed by Babisch in this 2006 review. Of these 5 involved Aircraft Noise, but the majority related to road traffic noise. With regard to the association between community noise and hypertension, the picture was said to be *"heterogeneous*".

Hypertension - Aircraft noise

With respect to **aircraft noise and hypertension,** studies consistently show higher risks in higher exposed areas. The evidence was said to have improved since the previous review in 2000 [reference 7], but no common risk curve or exposure response relationship could be derived.

The relative risks found in four studies showing significant positive associations ranging between 1.4 and 2.1 for subjects who live in high exposed areas, with approximate daytime average sound pressure level in the range of 60-70 dB (A) or more. Swedish studies found a relative risk of 1.6 at even lower levels (>55 dB (A)).

Babisch went on to show the results of three peer-reviewed studies [references 14, 15, 16] that had been evaluated in another recent review for a 2005 Conference [17].

See Figure 4 below.
He commented, *"...regarding **aircraft noise** the available information is still limited. Although positive associations have been shown in studies, no common risk curve can be derived."*

[see HYENA paper December 2007 later]

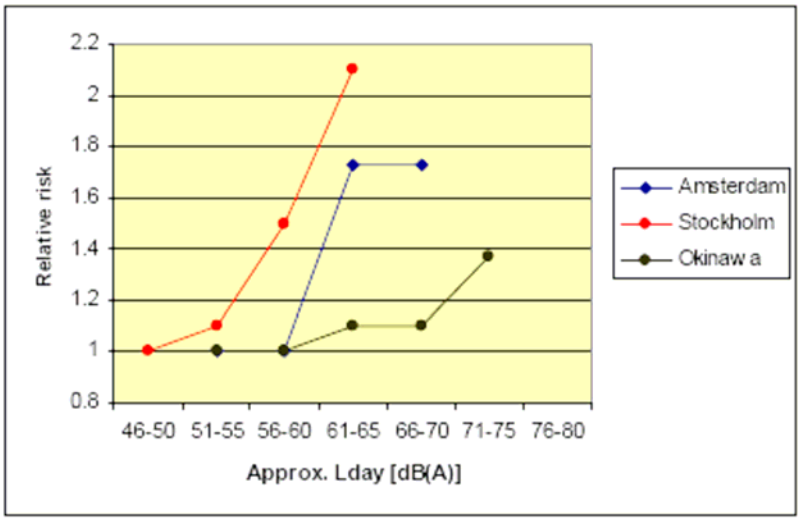

Figure 1: Association between aircraft noise level and the prevalence of hypertension.

Figure 4 – from Babisch 2005 [reference 17].

Hypertension - Road Traffic noise

With respect to **road traffic noise**, the picture was said to remain unclear. New studies, more likely than older studies, tend to suggest a higher risk of **hypertension** in subjects exposed to high levels of road traffic noise, showing relative risks between 1.5 and 3.0. However, the earlier studies cannot be neglected in the overall judgement process. Across all studies no consistent pattern of the relationship between community noise and prevalence of **hypertension** can be seen.

Ischemic Heart Disease IHD

Note – this includes:

- Clinical symptoms of angina pectoris, myocardial infarction (MI), ECG abnormalities as defined by WHO criteria, self-reported questionnaires regarding doctor-diagnosed heart attack – as reported in cross-sectional studies, and,
- Clinical myocardial infarction as obtained from hospital records, ECG measurements or clinical interviews – from longitudinal studies.

The majority of studies of IHD in this 2006 Babisch review refer to road traffic noise, but he discusses a few Aircraft noise studies as follows.

IHD - Aircraft Noise

Babisch notes;

Netherlands

"The calculation of standardized morbidity ratios (SMR) in an ecological study of 62 municipalities around Amsterdam's airport Schiphol, using aggregated data from the health registries recording the hospital admissions due to cardiovascular diseases (myocardial infarction, hypertension, ischaemic heart diseases and cerebrovascular diseases), did not show any apparent clustering in areas close to the airport.

A lot of information came from the Amsterdam aircraft noise studies that were carried out in the 1980's. Significant prevalence ratios of between 1.0 and 1.9 were calculated - depending on which IHD endpoint was looked at. The subjects lived in areas exposed to more than approx. 60 dB (A) outdoor noise level. The response rate of the "community cardiovascular survey" was approx. 42%. The "general practice survey" can be considered as an ecological study on contact rates for specific diseases, with general practitioners. Aggregated data of populations, not individuals, were analysed statistically. Multiple consultations were not excluded. The study provides information on the prevalence of cardiovascular disease, which must be viewed as a combination of hypertension and ischaemic heart diseases. In the study carried out in the four Dutch cities of Groningen, Twenthe, Leeuwarden and Amsterdam, regarding aircraft traffic noise, prevalence ratios greater than 1.0 were found for noise level categories greater than approx. 55 dB (A). However, no dose response relationship was found across the categories, and the relative risk for subjects in the highest noise category was 0.9. The response rate of approx. 43% refers to the subjects that participated in a previous psychological questionnaire survey (response rate there approx. 32%). Subjects that were identified in the questionnaire screening phase as being treated for hypertension were not included in the statistical analysis. This could be a matter of concern regarding selection bias in the study because high blood pressure is a major risk factor for IHD.

Germany

The Spandau Health Survey (response rate > 80%), which was primarily conducted with respect to road traffic noise, was also analysed with respect to aircraft noise [#58]. In the noise zone (according to the German Aircraft noise Act) of Leq(4) > 62 dB(A) the period prevalence (during the past 2 years) with respect to self-reported doctor's diagnosed angina pectoris was 1.6, and was not significant. However, with respect to the prevalence of myocardial infarction, a lower risk was found in the exposed group (relative risk = 0.4). The preliminary results of an ongoing study around the Stockholm airport showed the opposite [#60]: a higher risk of MI (relative risk = 2.6) in subjects exposed to FBM > 55 dB (A) (the Swedish calculation method of aircraft noise) and a lower risk for angina pectoris (relative risk = 0.9)."

IHD - Road traffic
With regard to IHD, the evidence of an association between community noise and IHD risk was said to have increased since the previous Babisch review in 2000 [Reference 7]. There was said to be not much indication of a higher IHD risk for subjects who live in areas with a daytime average sound pressure level of less than 60 dB (A) across the studies. For higher noise categories, a higher IHD risk was relatively consistently found amongst the studies. Statistical significance was rarely achieved. Some studies permit what Babisch describes as "reflections" on exposure-effect relationships. These studies suggest an increase in IHD risk for noise levels above 65-70 dB (A), the relative risks ranging from1.1 to 1.5 when the higher exposure categories were grouped together.

By way of **overall summary** of the "evidence", Babisch notes, on page 48 of the UBA report:

"*The evidence for a causal relationship between community or transportation noise and cardiovascular risk, appears to have increased throughout the recent years due to new studies that complement the data base. Compared with earlier conclusions [the 2000 review] this refers, in particular, to* ***hypertension and ischaemic heart diseases."***

Thus

- ***Biochemical effects: limited evidence***
- ***Hypertension: inadequate or limited or sufficient evidence***
- ***Ischaemic heart disease: limited or sufficient evidence***

…now becomes***….***

- **Biochemical effects: limited evidence**
- **Hypertension: ~~inadequate or~~ limited or sufficient evidence**
- **Ischaemic heart disease: ~~limited or~~ sufficient evidence**

Babisch meta-analysis and exposure-effect relationships

In Section 6 of the 2006 UBA report, on the basis of stringent criteria, five analytic and two descriptive studies were selected that could be used for deriving the relationship **between road traffic noise** and the risk of **myocardial infarction MI**. It was found that for noise levels above 60 dB (A), the risk of myocardial infarction increases monotonically.

Based on this, a common risk curve is derived for the relationship between road traffic noise – expressed in L_{day} - and the incidence of myocardial infarction (MI).

Below 60 dB (A) for the road traffic noise level during the day (L_{day}: 6-22 hr), no notifiable increase in MI risk could be detected. For noise levels greater than 60 dB (A), the MI risk increases continuously, with relative risks (odds ratios) ranging from 1.1 to 1.5, with reference to ≤60 dB (A).

A polynomial function was fitted to the pooled data points, and Linear, quadratic and cubic terms were considered. The results and the coefficients of the equations are shown in below [Babisch Figures 9 and 10], for the non-weighted and weighted (number of subjects) data.

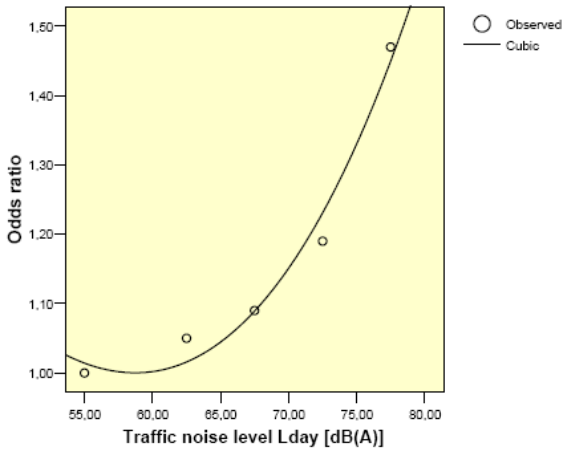

Figure 9. Polynomial curve fit (non-weighted data points) of the association between road traffic noise and incidence of myocardial infarction.
OR = 2.210093 – 0.001052*Noise^2 + 0.00001194531314644*Noise^3 ; R^2 = 0.98
(no significant linear term in the equation)

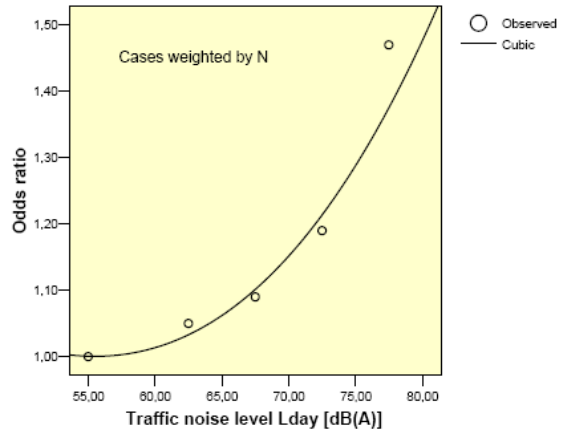

Figure 10. Polynomial curve fit (N-weighted data points) of the association between road traffic noise and incidence of myocardial infarction.
OR = 1.629657 – 0.000613*Noise^2 + 0.000007356734623455*Noise^3 ; R^2 = 0.96
(no significant linear term in the equation)

Figure 5. From Babisch 2006 – L_{day} and MI

In Section 8, Conclusions, of his 2006 Report, Babisch notes:

"*Aircraft noise has been less intensively studied in noise epidemiology. The studies focused on high blood pressure. Dose-response assessment was hardly considered. A large European study on the association between aircraft noise and road traffic noise, and blood pressure is currently being conducted (HYENA Jarup et al. 2003). Regarding aircraft noise - and particularly the ongoing debate on night-flight restrictions in the vicinity of busy airports - no other alternative exists at present than*

to take the MI risk curves derived from road traffic noise studies as an approximation for aircraft noise. Since aircraft noise acts on all sides of a building, i.e. different to road traffic noise, the suspicion exists that the effects induced by aircraft noise could be greater than those induced by road traffic (Babisch 2004a; Ortscheid and Wende 2000). This may be due to the lack of evasive possibilities within the home, and the greater annoyance reactions to aircraft noise, which are usually expressed in social surveys (Miedema and Vos 1998). More research is needed regarding the association between aircraft noise and cardiovascular endpoints."

NOTE – the same point about the applicability of the Road Traffic Noise curve to Aircraft is made in the more recent WHO Chapter – see Section 4 of this report.

2.4.8
H Miedema.2007
Exposure-response relationships for environmental noise
Proc Internoise 2007 Paper 07-179 CDROM

This paper presents an overview of exposure-response relationships that can be used for assessing the impact of environmental noise. It covers - Annoyance [transportation noise, stationary industrial noise and combined sources], Sleep disturbance, and myocardial infarction MI.

Miedema used the data in the above Babisch review of 2006 to arrive at the relationship for MI. See Figure 6 below

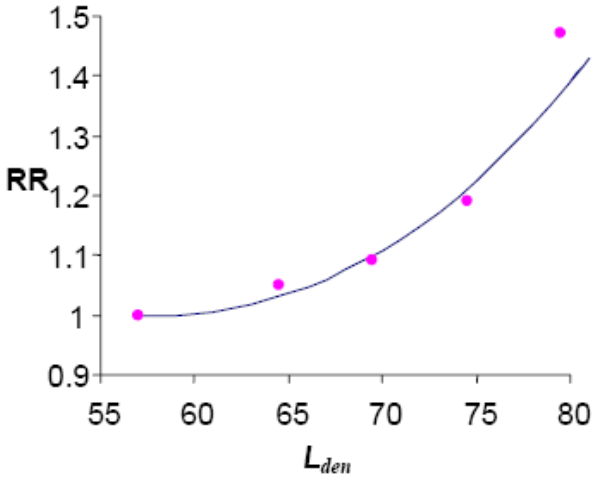

Figure 6 - from Miedema 2007. [The points that indicate the relative risk (RR) of myocardial infarction for different noise exposure classes, and a line fitted through these points - based on Babisch - assuming that in this case odds ratios can be equated to relative risks and that for road traffic *Lden* = $L_{06\text{-}22}$ + 2.]

3. RECENT SPECIFIC RESEARCH STUDIES [late 2006 to 2008]

Since the publication of the extensive Babisch review of 2006 [Reference 3] a number of newer papers have been published. These are briefly summarised below, in some cases using the published Abstracts etc.

3.1 Road Traffic

G. Bluhm, N Berglind, E Nordling, M Rosenlund
Road traffic noise and hypertension
Occup Environ Med 2007; 64:122–126. doi: 10.1136/oem.2005.025866

Background: It has been suggested that noise exposure increases the risk of hypertension. Road traffic is the dominant source of community noise exposure.
Objective: To study the association between exposure to residential road traffic noise and hypertension in an urban municipality.
Methods: The study population comprised randomly selected subjects aged 19–80 years. A postal questionnaire provided information on individual characteristics, including diagnosis of hypertension. The response rate was 77%, resulting in a study population of 667 subjects. The outdoor equivalent traffic noise level (Leq 24 h) at the residence of each individual was determined using noise-dispersion models and manual noise assessments. The individual noise exposure was classified in units of 5 dB (A), from 45 dB (A) to 65 dB (A).
Results: The odds ratio (OR) for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% confidence interval (CI) 1.06 to 1.80) per 5 dB (A) increase in noise exposure. The association seemed stronger among women (OR 1.71; 95% CI 1.17 to 2.50) and among those who had lived at the address for .10 years (OR 1.93; 95% CI 1.29 to 2.83). Analyses of categorical exposure variables suggested an exposure–response relationship. The strongest association between exposure to traffic noise and hypertension was found among those with the least expected misclassification of true individual exposure, as indicated by not having triple-glazed windows, living in an old house and having the bedroom window facing a street (OR 2.47; 95% CI 1.38 to 4.43).
Conclusion: The results of our study suggest an association between exposure to residential road traffic noise and hypertension.

NOTE

1. The "measure" of hypertension is self-reported - a fact which the authors themselves note as a possible bias - although it does refer to a history of specific diagnosis, rather than self-report of symptoms.

2. Overall the numbers of subjects with hypertension is small.

3. Because the numbers of subjects in the 3 highest noise exposure categories [55-60, 60-65 and >65] are small, they are combined into the category of >55. If any of the n=25 subjects in the categories of noise exposure >60, or >65 were those reporting hypertension [n=22] , this would skew the apparent link to noise levels being reported as in the category > 55.

Y. Kluizenaar,, R. T. Gansevoort, P. E. de Jong and H M.E. Miedema. 2007
Cardiovascular effects of road traffic noise with adjustment for air pollution.
Proceedings of Inter-noise 2007, Istanbul Paper 07-244 CDROM

This study investigates cardiovascular effects of road traffic noise, accounting for air pollution. Noise and particulate matter (PM10) exposure was assessed for the City of Groningen, sample (N = 40 856), and a selection of subjects that next visited the outpatient clinic (PREVEND cohort; N = 8 592). Questionnaires and, for the cohort, measurements (e.g. systolic and diastolic blood pressure, BMI, cholesterol) provided cardiovascular endpoints, risk factors and confounders. For individual exposure assessment detailed spatial data (e.g. traffic characteristics, buildings, screening objects) were used together with geographical information systems (GIS) and state-of-the-art modeling techniques. Road traffic noise was associated with antihypertensive medication use in the City of Groningen sample (unadjusted OR = 1.31 per 10 dB increase Lden). Adjusted odds ratios were significant for the 45-55 year age group in the full model adjusted for PM10 (OR =1.19), and adjusted odds ratios were significant for higher exposure (Lden > 55 dB; OR= 1.21; with adjustment for PM10 OR = 1.31). In the cohort the unadjusted odds ratio was 1.35 for hypertension. The adjusted odds ratio was again significant for the 45-55 yr age group.

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G Belojevic, B. Jakovljevic, V Stojanov, K. Paunovic, J Ilic
Urban road-traffic noise and blood pressure and heart rate in preschool children.
Environment International (2007), doi:10.1016/j. envint.2007.08.003

Abstract

Night time noise exposure has very rarely been used in previous studies on the relationship between community noise and children's blood pressure, although children spend a larger part of their night time sleeping at home than adults. For this reason, we focused on night time noise exposure at children's residences and daytime noise at kindergartens. The aim of this study was to investigate the effects of urban road-traffic noise on children's blood pressure and heart rate. A cross-sectional study was performed on 328 preschool children (174 boys and 154 girls) aged 3–7 years, who attended 10 public kindergartens in Belgrade. Equivalent noise levels (Leq) were measured during night in front of children's residences and during day in front of kindergartens. A residence was regarded noisy if Leq exceeded 45 dB (A) during night and quiet if the Leq was ≤45 dB (A).

Noisy and quiet kindergartens were those with daily LeqN60 dB (A) and ≤60 dB (A), respectively. Children's blood pressure was measured with mercury sphygmomanometer. Heart rate was counted by radial artery palpitation for 1 min. The prevalence of children with hypertensive values of blood pressure was 3.96% (13 children, including 8 boys and 5 girls) with higher prevalence in children from noisy residences (5.70%), compared to children from quiet residences (1.48%). The difference was borderline significant (p=0.054). Systolic pressure was significantly higher (5mmHg on average) among children from noisy residences and kindergartens, compared to children from both quiet environments (p<0.01). Heart rate was significantly higher (2 beats/min on average) in children from noisy residences, compared to children from quiet residences (p<0.05). Multiple regression, after allowing for possible confounders, showed a significant correlation between noise exposure and children's systolic pressure (B=1.056; p=0.009).

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3.2 Aircraft noise

I van Kamp, D Houthuijs, C van Wiechen, and O Breugelmans 2006
Environmental noise and cardiovascular diseases: evidence from 10 year Schiphol research
Proceedings of Internoise 2006. Paper No.: in07_132, 7 pages (on CDROM).

The authors themselves summarise the scope and outcome of this paper as follows;
"The monitoring programme around Schiphol Airport, aimed at describing changes in environmental quality and related health effects after opening of a new runway, included several measures for cardiovascular diseases. Before (2002) and after (2005) opening of the runway, information was collected by means of questionnaire surveys. Also two studies with data from medical registries were carried out. Results from the surveys show an inconsistent pattern in the association between aircraft noise and self reported high blood pressure and use of medication for hypertension. There were no indications from the 2005 survey that the change of aircraft noise level, in addition to the noise level itself, had an extra impact.
Medical registries data revealed that the dispensing of anti-hypertensives was related to air-traffic noise. There were no indications for an increase of hospital admissions for cardiovascular diseases in relation to aircraft noise. Based on these results and results from earlier studies around the airport, there is a growing evidence for an association between exposure to air traffic noise and the prevalence of high blood pressure around Schiphol Airport.

Examining the paper in more detail we note that the medical data were based on analysis of routinely collected health data from two sources:
 Ten year period data (1995-2004) on hospital admissions from the Dutch national medical registration;
 Data about the dispension of prescribed medication by public pharmacies over a five year period (2000-2004).

Registration data were analysed by means of so called 'small area health statistics'. The level of analysis is post code area (5,700 inhabitants on average) as opposed to the individual level in the surveys. The incidence and prevalence of the indicators of cardiovascular disease for each postal code area were first standardized using population data stratified by age and sex.

Noise exposure was based on modelled yearly averaged Lden and Lnight levels for aircraft noise provided by the National Aerospace Laboratory (NLR) for an area of 55 by 71 km around the airport. The mean aircraft noise at postcode level was used as exposure indicator.

After adjustment for potential confounding factors at the postal code level and taking into account spatial correlation, the health risk is mapped as standardized morbidity ratio in space and time. In this way, the (changing) spatial pattern around the airport can be visualized and a possible relation with environmental quality quantified.

In addition, surveys (postal questionnaires) of self-reported health were carried out among adults in one year before (2002) and two years (2005) after the opening of the new runway. Questions about health indicators as well as determinants (age, lifestyle, sex etc) were included.

Results of the questionnaire surveys showed that, between 2002 and 2005 no differences were found in the prevalence of self reported cardiovascular disease in the study area. Only self reported hypertension had increased, from 8.6% in 2001 to 9.9% in 2004, but it was noted that this trend is also found at a national level.

In 2005, a statistical significant effect of Lden was found on self reported hypertension. When the noise level increases by 3 dB (A) the odds ratio is 1.2. Also, people who report they are severely annoyed by aircraft noise report hypertension twice as often. In addition, the self reported use of medication for CVD is associated with Lden as well as Lnight although not statistically significant. These associations were not found in 2002.

Changes in Lden and Lnight between 2002 and 2005 did not result in significant changes, either in medication use, or self-reported hypertension

Results from medication registries showed an increase in the dispensing of anti-hypertensive drugs with an increase in aircraft noise. Such an association was not found for hospital admissions. However, the prevalence of dispension of medication for hypertension (13%) is much higher than the prevalence of hospital admissions for e.g. hypertension (0.2%). The authors suggest that a small effect of aircraft noise in the range of other risk factors for hypertension might not be detectable within the small group of hospital admissions.

Overall the findings in the monitoring programme were said to be consistent with previous estimates of about 1,500 extra persons with hypertension as a result of aircraft noise exposure, and that this could result in "*a few additional cases of myocardial infarcts, strokes, and angina".*

This study in itself could not used to provide any new numerical information on exposure-response relationships, to add to existing knowledge.

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E. van Kempen, I. van Kamp, P. Fischer, H. Davies, D. Houthuijs, R. Stellato, C. Clark, S. Stansfeld.
Noise exposure and children's blood pressure and heart rate: The RANCH-project.
Occup. Environ. Med. published online 25 May 2006
http://oem.bmj.com/cgi/content/abstract/63/9/632

This paper initially comments on the fact that, because of the complexity of the link between blood pressure elevation and stress, and the preponderant influence of lifestyle and genetic predisposition, conclusions from earlier studies investigating the effects of noise exposure on children's blood pressure are limited and inconsistent.

Methodological problems emerging from these earlier studies are noted – including small differences in noise levels between the exposure groups, potential selection bias, a lack of control for socio-economic status factors, differences in insulation and parental history of high blood pressure.

It is also noted that most studies usually only focus on exposure at school when investigating the effects of noise exposure on children. It is questionable whether the health effects could be exclusively attributed to the noise exposure in school. The effect of night-time exposure has been hardly investigated in children.

It was against this background that, in order to investigate the possible association between noise exposure and children's blood pressure and heart rate, data were

collected from children living around London Heathrow Airport and Schiphol Airport gathered in the framework of the EU-funded RANCH project.

Out of 118 primary schools available in the British study area, 30 were invited to participate and all but one agreed. In the Netherlands, out of 366 available schools in the selected areas, 77 schools were invited to participate, and 33 agreed. The parents or carers of 2,179 children were approached through the schools by letter and 2,012 children had permission to take part. In the Netherlands all the children who had permission to take part and who were available on the day of testing had their blood pressure measured (n = 730); in the United Kingdom every second participating child was selected from the class list for blood pressure measurement (n = 553).
Noise exposure was assessed for each child by linking home and/or school addresses to modelled equivalent aircraft and road traffic noise levels. These predict the average outdoor noise exposure during a specified time interval.
In both centres, aircraft noise levels (LAeq, 7-23 hr, and LAeq, 23-7hr) were obtained from nationally available noise contours for both the home and school situation

In both centres, road traffic noise levels (LAeq, 7-23 hr) were obtained for the school situation. Road traffic noise levels (LAeq, 7-23 hr) for the home situation were only available for the Dutch sample.

Blood pressure measurements were taken in the afternoon in a quiet room in the school building using automatic blood pressure meters, according to a standard protocol

Children were given a questionnaire to take home for their mother (preferably), or other carer to complete. The questionnaire provided information on potential confounding factors (e.g. socio-economic status, birth weight, country of birth and parental history of high blood pressure).

The effect of chronic aircraft noise on blood pressure differed somewhat between the samples: In the Dutch sample, chronic aircraft noise exposure at school was related to an increase in blood pressure. Statistically significant increases were estimated for systolic and diastolic blood pressure, respectively. In the British sample, aircraft noise exposure at school was related to small and statistically non-significant increases in blood pressure.

For the pooled data, after adjustment for socio-economic status, ethnicity, cuff-size, room temperature, birth weight, parental hypertension, and pre-maturity only Aircraft noise **exposure at home** was related to a statistically significant increase in blood pressure

Chronic aircraft noise exposure during the night (LAeq, 23-7hr) at home was positively associated with systolic blood pressure only.

Due to the difference in exposure metrics and adjustment for confounders, comparison of the results of the RANCH study with other studies was difficult, and it was concluded that "*for aircraft noise exposure no consistent findings can be seen*".

Negative associations were found between road traffic noise exposure and blood pressure, which could not be explained.

It was not possible to derive an exposure-effect relationship between Noise exposure and children's blood pressure.

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C. Eriksson, M. Rosenlund, G. Pershagen, A. Hilding, C-G Ostenson, and G Bluhm. 2007. *Aircraft Noise and Incidence of Hypertension.* Epidemiology 2007; 18: 716–721

Summary of study method etc.

This study investigated the influence of aircraft noise on the incidence of **hypertension.** A cohort of 2754 men, aged 45-66, in 4 municipalities around Stockholm Arlanda airport was followed between 1992–1994 and 2002–2004. The cohort was based on the Stockholm Diabetes Preventive Program; half of the study subjects had a family history of diabetes.

Residential aircraft noise **exposure** (expressed as time-weighted equal energy and maximal noise levels) was assessed by geographical information systems techniques among those living near the airport. Noise exposure categories in Lden were 50-55, 55-60, 60-65 and > 65 dB (A).

Incident cases of **hypertension** were identified by physical examinations, including blood pressure measurements, and questionnaires in which subjects reported treatment or diagnosis of hypertension and information on cardiovascular risk factors. Analyses were restricted to 2027 subjects who completed the follow-up examination, were not treated for hypertension, and had a blood pressure below 140/90 mm Hg at enrollment.

Results:

For subjects exposed to energy-averaged levels above 50 dB (A) the adjusted relative risk for hypertension was 1.19 (95% CI _1.03–1.37). Maximum aircraft noise levels presented similar results, with a relative risk of 1.20 (1.03–1.40) for those exposed above 70dB (A).

Stronger associations were suggested among older subjects, those with a normal glucose tolerance, nonsmokers, and subjects not annoyed by noise from other sources.

Conclusion: These findings suggest that long-term aircraft noise exposure may increase the risk for hypertension.

See Figure 7 - Relative risk for hypertension among men in Stockholm
according to different levels of energy-averaged aircraft noise exposure (bars indicating 95% CI), adjusted for age and BMI [Body Mass Index]

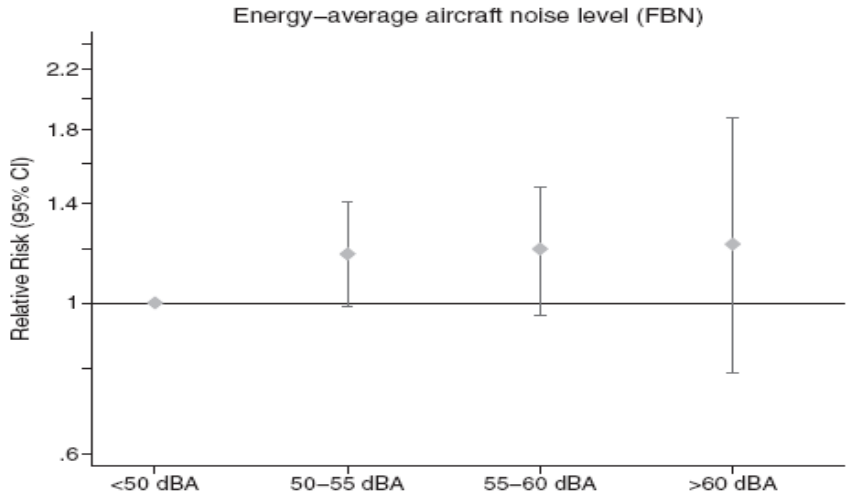

Figure 7 - From C. Eriksson, M. Rosenlund, G. Pershagen, A. Hilding, C-G Ostenson, and G Bluhm. 2007

NOTE – This study is now included in the Draft WHO chapter – see Section 4.

Greiser, E., C. Greiser, and K. Janhsen, 2007
***Night-time aircraft noise increases prevalence of prescriptions of anti-hypertensive and cardiovascular drugs irrespective of social class - the Cologne-Bonn Airport study*. J. Public Health, 2007. Doi 10.1007/s10389-007-0137-x.**

This paper reports an epidemiological study to investigate primarily the possible impact of night-time air traffic noise on prescription of antihypertensive and cardiovascular drugs in a general population residing in the vicinity of the airport, taking into account, among other potential confounders, social class.

The study region comprised the city of Cologne and two counties adjacent to the Cologne-Bonn Airport. Based on all individual flights from or to Cologne-Bonn Airport in 2004, time averaged spatial aircraft noise levels were calculated – essentially period LAeq. Noise levels were determined for the 6 months of 2004 with highest traffic density for four time periods: day (6.00 a.m.–10.00 p.m.), night (10.00 p.m.–6.00 a.m.) and two night periods (11.00 p. m.–1.00 a.m. and 3.00–5.00 a.m.).

Individual prescription data of 809,379 persons insured with compulsory sickness funds were linked to address-specific noise data (air traffic, road traffic, train traffic).

The main results discussed in detail in the paper are multivariate logistic analyses conducted in relation to a particular two-hour night-time aircraft noise period (3.00–5.00 a.m.). Adjustments were made in the statistical analysis for noise from other sources, age, density of nursing homes etc.

Particular focus was placed on stratifying the results in terms of prevalence of social welfare recipients.

In males there was an increase in the prescribing of anti-hypertensive drugs with increasing aircraft noise [in that two-hour night period] in all strata of social welfare prevalence. However, there were specific differences when comparing effects in different social welfare quartiles.

In females there were significant risk increases over all noise quartiles in all social welfare strata, but in three of four social welfare strata, odds ratios of the fourth noise quartile are lower than of the third. This is a kind of “saturation effect” at the highest noise levels, [2-hour LAeq category of 48 to 61 dBA]. This was discussed as possibly due to self-selection of people living in those regions with the highest noise.

Whilst the results are of general interest, because of the way the results are analysed in relation to such a specific night time period, and the way the noise data categories are presented in the publication, it is not possible at present, without additional information, to combine these results with others or use the results in any subsequent calculations.

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Babisch, W., et al., *Association between noise annoyance and high blood pressure. Preliminary results from the HYENA study*.
Proceedings of Inter-noise 2007, Istanbul, Paper No. in07-133 (10 pages).
The EU-funded HYENA study (HYENA = HYpertension and Exposure to Noise near Airports) is a large-scale multi-centered cross-sectional study carried out simultaneously in 6 European countries to assess the relationship between aircraft noise and road traffic noise on the one hand, and the prevalence of high blood pressure on the other.

The study population included 4,861 people (2,404 men and 2,467 women) aged between 45 and 70 years at the time of interview, and who had been living for at least 5 years, near one of the six major European airports (London Heathrow, Berlin Tegel, Amsterdam Schiphol, Stockholm Arlanda, Milan Malpensa) and Athens Elephterios Venizelos. In Stockholm, the population living near the City Airport (Bromma) was also included to increase the number of exposed subjects. Fieldwork was carried out during the years 2003-2005

This particular paper on the project, the most recent available does not deal with any noise exposure-effect relationship but covers mainly the complex issue of the relationship between annoyance [which the authors sometimes refer to as "subjective exposure"] and hypertension. [See later summary of the full peer-reviewed journal paper on the HYENA project published December 11 2007].

Noise annoyance was assessed using the non-verbal 11-point ICBEN scale, and items covered annoyance from air traffic, road traffic and other community noise or indoor noise sources (e.g. railway, motorcycles, industry, construction, neighbors and indoor installations). A distinction was made between the annoyance during the day and the annoyance during the night.

Objective blood pressure measurements were carried out in residents' homes by specially trained staff. Hypertension was assessed from these measurements using standard WHO clinical criteria.

Use of anti-hypertensive medication was assessed and in addition the subjects were asked about the prevalence of doctor-diagnosed hypertension.

A wide range of potential confounding factors were assessed including such as country, age, education, alcohol intake, body mass index, physical exercise and, sex.

Effect modifiers included in the analysis were - noise sensitivity, coping style, belief in authorities and attitude towards the airport. The frequency of usage of noise reducing remedies was assessed (e.g. ear plugs, closing windows, closing window shutters, etc).

The authors expressed the main conclusions on the complex association between Annoyance and hypertension [HT] as follows;
"HYENA subjects who were more annoyed due to aircraft and road traffic noise reported more often that a doctor had ever diagnosed HT, and were more often under medication for HT. This could indicate that chronic noise stress had raised their blood pressure. However, the association can also be interpreted vice-versa. Subjects who were aware of their high blood pressure reported higher noise annoyance. This could either be due to an increased sensitivity to noise as a consequence of their health problem, or due to over-reporting because subjects may tend to blame the environment as being a reason for their health problem. This latter interpretation is supported by the fact that the clinical blood pressure measurements which consider 'mild' (unknown/untreated) HT, did not show much of an association

with noise annoyance. More annoyed subjects could also have over-reported high blood pressure. In fact, noise sensitivity was higher in subjects who reported doctor diagnosed HT, but not in subjects where HT was assessed more objectively based on BP readings or medication intake."

NOTE – the full paper on HYENA follows, published December 11 2007

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Lars Jarup, Wolfgang Babisch, et al 2007.
Hypertension and Exposure to Noise near Airports - the HYENA study.
Environmental Health Perspectives.
http://www.ehponline.org/docs/2007/10775/abstract.html
Online 11 December 2007

Abstract
Background
An increasing number of people are exposed to aircraft and road traffic noise. Hypertension is an important risk factor for cardiovascular disease and even a small contribution in risk from environmental factors may have a major impact on public health. The HYENA study aimed to assess the relations between noise from aircraft or road traffic near airports and the risk of hypertension.
Methods. The study measured blood pressure and collected data on health, socio-economic and life-style factors, including diet and physical activity, via questionnaire at home visits for 4,861 persons aged 45 to 70, who had lived at least five years near any of six major European airports. Noise exposure was assessed using detailed models with a resolution of 1dB (5dB for UK road traffic noise), and a spatial resolution of 250 x 250m for aircraft and 10 x 10m for road traffic noise.
Results. We found significant exposure-response relationships between night-time aircraft as well as average daily road traffic noise exposure and risk of hypertension after adjustment for major confounders. For night-time aircraft noise, a 10dB increase in exposure was associated with an odds ratio of 1.14 (95% confidence interval: 1.01-1.29). The exposure-response relationships were similar for road traffic noise and stronger for men with an odds ratio of 1.54 (95% CI: 0.99-2.40) in the highest exposure category (>65dB) ;(ptrend = 0.008).

Conclusions. Our results indicate excess risks of hypertension related to long term noise exposure, primarily for night-time aircraft noise and daily average road traffic noise.

The key figures from this paper on the HYENA study are shown below.

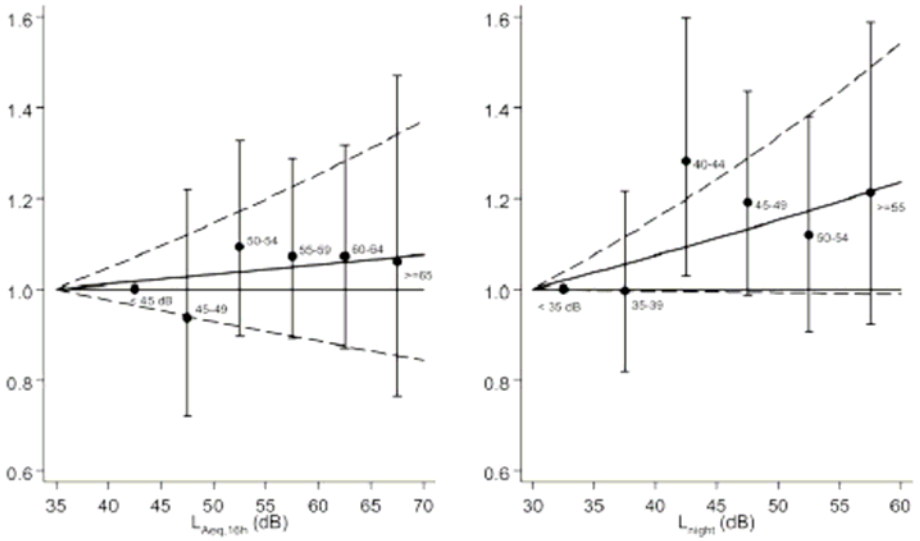

Figure 1. Odds ratios (OR) of hypertension in relation to aircraft noise (5dB categories). LAeq, 16h (left) and Lnight (right) separately included in the model. Adjusted for country, age, gender, BMI, alcohol intake, education and exercise. The error bars denote 95% confidence intervals for the categorical (5dB) analysis. The unbroken and broken curves show the OR and corresponding 95% confidence interval for the continuous analysis.

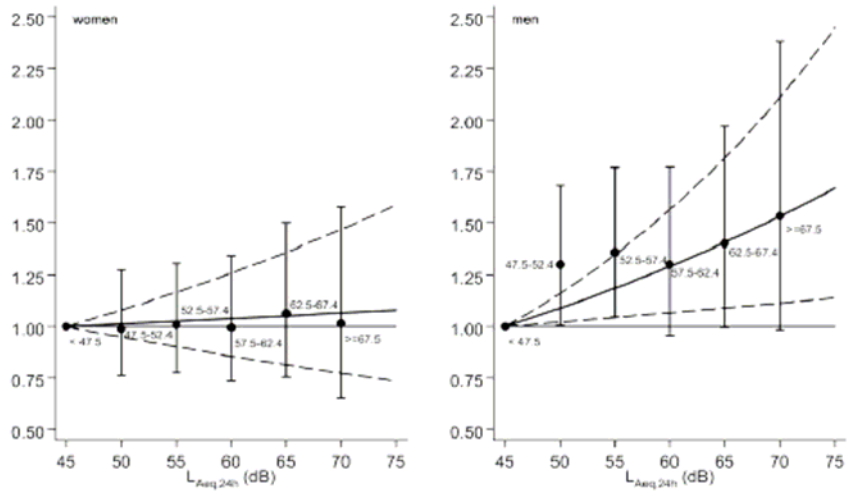

Figure 2. Odds ratios (OR) of hypertension in women (left) and men (right) in relation to road traffic noise (LAeq, 24h, 5dB categories) separately included in the model. Adjusted for country, age, BMI and alcohol intake, education and exercise. The error bars denote 95% confidence intervals for the categorical (5dB) analysis. The unbroken and broken curves show the OR and corresponding 95% confidence interval for the continuous analysis.

Figure 9. From Jarup, Babisch et al. HYENA, E H Perspectives. December 2007.

NOTE – THESE NEWLY PUBLISHED RESULTS FROM THE HYENA PROJECT HAVE JUST BEEN INCLUDED BY BABISCH IN THE 2nd DRAFT OF HIS CHAPTER FOR THE NEW 2008 WHO REVIEW

Working Paper = WHO Working Group: AIRCRAFT NOISE AND HEALTH. Cardiovascular effects of aircraft noise. **2st draft (January 2008).** *Wolfgang Babisch and Irene van Kamp*

SEE NEXT SECTION

4. WORLD HEALTH ORGANISATION [WHO] Working Group – Aircraft noise and health - Chapter on Cardiovascular effects, by Babisch and van Kamp - Version 2 Draft January 2008

The Working Group on Evidence Review of Aircraft Noise and Health was established by the Noise and Health programme of WHO/Europe European Centre for Environment and Health late in 2007.

For an updated review of scientific evidence on health impacts of aircraft noise, the epidemiologists, acoustic engineers, and other experts met at WHO/Europe European Centre for Environment and Health in Bonn, Germany, on 11-12 October 2007. The UN agency specialised in civil aviation, the International Civil Aviation Organization (ICAO) also participated at the meeting.

The meeting reviewed the Chapters on annoyance, sleep disturbance, cardiovascular disorders, physiological responses, psychological responses, exposure assessment and risk management, which had been drafted by the co-authors of the Working Group. Technical and policy comments were made by the participants, and the co-authors were requested to provide the revision for the final editorial process, beginning in mid-January 2008. The final WHO Report will be published as a WHO document on the web and in print – probably in the summer of 2008.

Wolfgang Babisch from the UBA in Berlin and Irene van Kamp from RIVM in Netherlands, have recently issued the 2nd Draft of their chapter.

Cardiovascular effects of aircraft noise
2st draft (December 2007) – issued January 10 2008

Wolfgang Babisch[1] and Irene van Kamp[2]

[1]Federal Environment Agency (UBA), Berlin, Germany
[2]The National Institute for Public Health and the Environment (RIVM), Bilthoven, The Netherlands

In view of the importance of this document**, the full text of this Report is given as Annex 3.** However, The Abstract, conclusions and a key Figure are given here for convenience. Important sections of the text are highlighted **in Bold.**

Abstract

Noise is a stressor that affects the autonomic nervous system and the endocrine system. Under conditions of chronic noise stress the cardiovascular system may adversely be affected. Epidemiological noise studies regarding the relationship between aircraft noise and cardiovascular effects have been carried out on adults and on children focussing on mean blood pressure, hypertension and ischemic heart diseases as cardiovascular endpoints. **While there is evidence that road traffic noise increases the risk of ischemic heart disease, including myocardial infarction, there is less such evidence for such an association with aircraft noise**. This is partly due to the fact that large scale clinical studies are missing. **There is sufficient evidence that aircraft noise increases the risk of hypertension.** The results regarding aircraft noise and children's blood pressure are still inconsistent. This may be due to methodological reasons.

Conclusions

The general conclusion is that there is **sufficient evidence** for a positive relationship between aircraft noise and high blood pressure and the use of cardiovascular medication**. However, no single common exposure-response relationship can be established for the association between aircraft noise and cardiovascular risk due to methodological differences between studies and the lack of continuous or semi-continuous (multi-**

categorical) noise data. For the same reason no answer can be given regarding possible effect thresholds.

Road traffic noise studies suggest that the cardiovascular risk increases when the outdoor noise level during the day exceeds 60-65 dB(A) and 50-55 dB(A) during the night, respectively. As to whether this information can be applied to aircraft noise remains unclear. However, this may be a conservative approach, considering on the basis of annoyance studies that aircraft noise effects may even be stronger than those of road traffic noise. Annoyance studies showed that aircraft noise is more annoying than road traffic noise of the same average noise level, which might partly be explained by less exposure misclassification (no shielding of aircraft noise, no unexposed rooms). New aircraft noise studies suggest that the risk may increase at even lower night noise levels. Depending on whether high blood pressure was assessed by a self-administered postal questionnaire or by clinical measurements, the magnitudes and the possible thresholds of effect varied between and within studies. Effects were more pronounced, when subjective measurements of high blood pressure were considered. This may raise questions regarding over-reporting. The validity of study results appears to be even more a problem when subjective noise annoyance was considered for exposure. The effect estimates tend to be larger but may be prone to over-reporting, particularly in cross-sectional studies where both, exposure and outcome, are assessed on a self-reported basis with the same questionnaire.

In the present summary, only those studies were considered in which aircraft noise was the explicit noise source. However, in a situation where information is lacking, the results of studies on the association between road traffic noise and myocardial infarction may also serve as an approximation for possible effects of aircraft noise [WHO reports 'Night Noise Guidelines' and 'Environmental Noise Burden of Disease', both available soon].

The available results do not allow for a distinction between the sexes. Males have been studied much more often than females. There is some indication that males may be more affected by road traffic noise. However, also contradictory results were found. The database is too weak for final conclusions regarding any gender differences. Due to the use of different noise indicators in aircraft noise studies only very crude comparisons can be made between studies on the basis of common noise indicators, e. g. L_{dn} or $L_{Aeq,6\text{-}22hr}$. Most aircraft noise studies did not distinguish between day and the night. A road traffic noise study and two aircraft noise studies suggest that noise during the night may be more harmful than during the day . However, no firm conclusions can be drawn about the relative contribution of day and night exposure because noise indices are usually highly correlated. One study suggests not only that noise during the night may be the primary source of adverse effects; it also shows that within the night period, effects due to noise in the early morning shoulder hours may be larger.

The contribution of noise on children's blood pressure is still not fully understood. Predisposi-tional and lifestyle factors seem to dominate and it is hard to study the influence of environmental noise separate from these. This might be one of the reasons why conclusions about the effect of noise exposure on children's blood pressure are limited and inconsistent. Methodological problems which arise are study size, insufficient contrast between noise levels, selection bias and insufficient adjustment for factors such as SES, parental history, noise insulation and ethnicity. Moreover, most studies on cardiovascular effects in children have focused on school exposure while at least the combination of day- and night time exposure and the related lack of restoration might be of importance in the development of cardiovascular disease due to early childhood blood pressure changes.
……..

Figure 1 from this new WHO Chapter, showing Ldn and Relative risk of Hypertension is given below as Figure 10 of this Report. For comparison purposes, the version from the 2005 Conference paper – Lday – is repeated [Figure 4 of this report]

The Authors note that;
"Only a very few epidemiological studies are available on adults, in which the association between aircraft noise and clinical states of cardiovascular diseases were assessed. Five studies appear reasonably valid for further consideration because minimum requirements regarding the validity of the assessment of exposure, outcome and the statistical control for confounding factors were fulfilled [34,47-49,65,66]. However, noise level related data pooling ('categorical approach') is difficult due to the fact that different (national) exposure indices were used. A graphical presentation of results using approximations with respect to the common noise indicator L_{dn} is shown in Figure 1. No conclusions regarding possible threshold values or noise level related risks (in absolute terms) can be made."

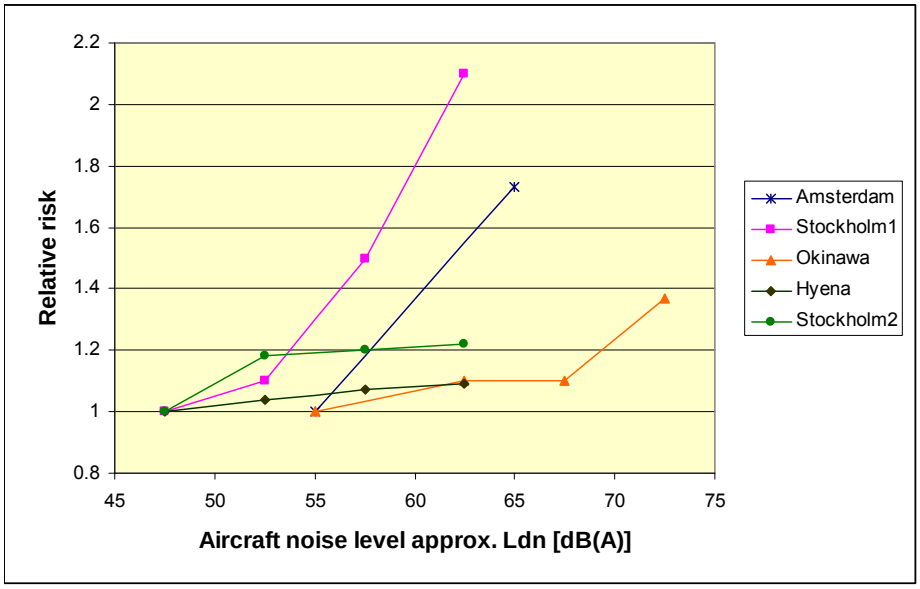

Figure 10. HYENA study 2007. Ldn and Relative risk of Hypertension

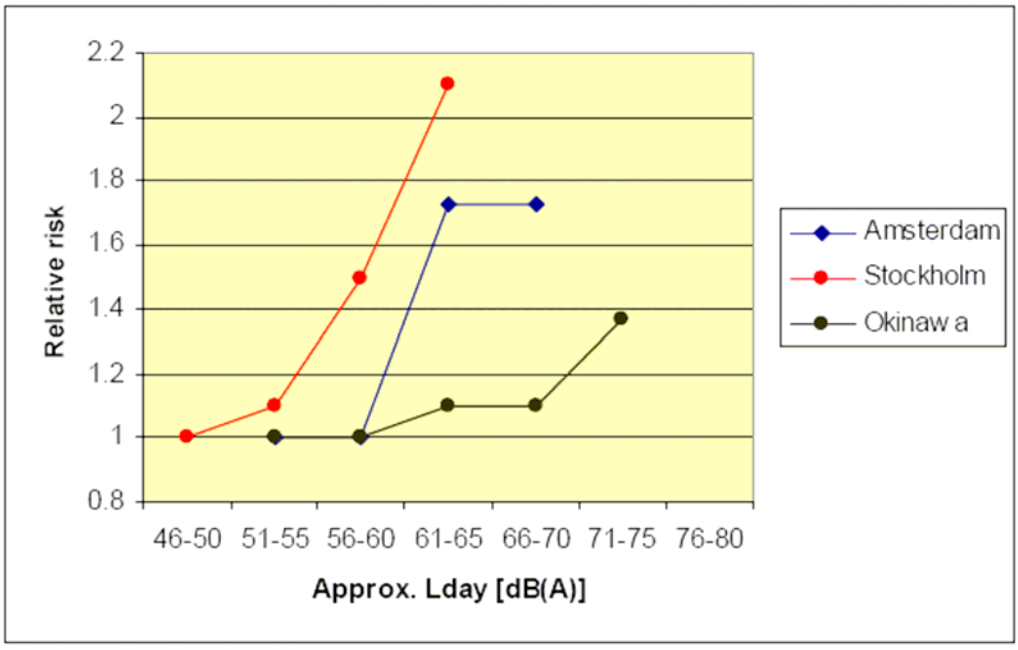

Figure 4 – repeated from page 13

5. SUMMARY AND OVERALL CONCLUSIONS.

- The potential effects of noise on cardiovascular health have been studied for many years, and possible causal mechanisms, and "models" have been proposed. Section 2 of this Report defines "*cardiovascular effects*" and gives a brief outline of some of these proposed "mechanisms" and "models".
- This section then presents a Summary Table [Section 2.3] in which the key points from previous reviews are highlighted. This enables an overview of how the evidence has developed over recent years, to the extent that it can be summarised as ;

Biochemical effects:	*limited evidence*
Hypertension:	*limited or sufficient evidence*
Ischemic Heart Disease	*sufficient evidence*

- A detailed overview of these previous published reviews of the topic is then given in Section 2.4, which looks at the evidence for such effects, and possible "exposure-response relationships", starting from the basis of a number of previous reviews published between 1998 and 2006 by acknowledged experts in the field.
- More recent studies involving Road Traffic Noise and Aircraft Noise have also been identified and summarised. These more recent studies have examined a range of issues including the role of Air Pollution, the effects of **changes** in noise environment, specific night-time noise factors, the role of annoyance, effects on children, and a wider range of possible confounding factors such as social welfare status etc. Such studies have also introduced new research techniques in which, for example, the health risk is mapped as standardized morbidity ratio in space and time, allowing the (changing) spatial pattern around an airport to be visualized and possible relationships with environmental quality quantified. [Section 3]
- Such studies are helping to improve our knowledge – but further more detailed examination of these studies, and how their results can be applied, are required.
- For Road Traffic Noise, an "exposure-response relationship" has been proposed, between noise level and risk of Myocardial infarction MI, and in fact this has been applied by Wolfgang Babisch to estimate the potential number of people at risk of cardiovascular effects from road traffic noise in Germany.
- For Aircraft noise, there have as yet been an insufficient number of high quality studies on which to base such an exposure-response relationship but it has been argued by Wolfgang Babisch, that the relationship developed for Road Traffic Noise should be used as an "approximation".
- Account has been taken of a very recently issued Draft Chapter on Cardiovascular effects, by Babisch and Irene van Kamp - Version 2 Draft January 2008, for a new Report being prepared for the World Health Organisation [WHO] Working Group - Aircraft noise and health. [Section 4]
- In the next phase of the project, these relationships can be applied to the specific "scenarios" of London Heathrow Airport, London City Airport and road traffic noise [using London Noise Maps etc].
- The field is a very "active" one, with new research studies in progress and in preparation, and with a number of other organisations, such as WHO, conducting further reviews. It is clear that there is considerable scope for further "monitoring" of the subject, and additional review/analysis.

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Annex 1. The Brief [as at December 6 2007]

BRIEF – EFFECT OF NOISE ON PHYSICAL HEALTH RISK IN LONDON

1. Summary

To provide guidance on the robustness of published cardiovascular risk factors arising from ambient/environmental noise, using latest available World Health Organisation (WHO) and other reviews of evidence/criteria, and suggest best estimates of the factors from the range of published values. Use derived factors to generate estimates of the numbers of people at cardiovascular and related health risk from road and air traffic noise in London from available population exposure data (for example, from Defra London Road Traffic Noise Map) on a basis as near-compatible as possible with existing air quality-related estimates.

2. Background

Ambient, or environmental, noise is popularly perceived as primarily a qualitative issue for health – in other words, one affecting health in its broadest definition (e.g. the WHO definition as not just lack of harm, but a positive sense of well-being) and hence in terms of disturbance, annoyance, quality of life etc. While these are important issues, there are indications that ambient noise may also have health impacts in the narrower, physiological sense. One of the main areas of study into potential health impacts has related to increased risk of heart disease (ischaemic heart disease and myocardial infarction) as distinct from the universally accepted impacts of higher levels of noise, such as that within some industrial premises, where the main effect is seen as damage to hearing. Public bodies in some countries in Europe (e.g. the Netherlands) appear to have accepted non-auditory physiological health impacts as of sufficient risk to have guidance to address them. Recently, significant publicity has been given to preliminary findings by a WHO group of experts looking into the burden of disease from environmental noise[1].

In the UK, this issue does not appear to have been given the same degree of attention and credence. While recognising the possibility of physiological impacts, a more cautious 'lack of absolute proof' viewpoint has tended to prevail. Reasons for differences in official recognition in different countries are not entirely clear.

In 1998 a UK government-sponsored project reviewed the evidence for physiological and other impacts from noise and concluded (on physiological impacts) that while there probably were such impacts, further research was needed to confirm these and their scale. Defra and the Department of Health (DH) then jointly commissioned some new scientific studies, though these mainly focussed on non-physiological effects. These were completed between 2000 and 2002 and are published on the DH website.

Following the foundation of the GLA, a 'Rapid Review on Noise and Health for London' was commissioned by the GLA in 2001, although this mainly reviewed similar evidence. Again, this review concluded that noise could have cardiovascular health effects, but did not quantify them, suggesting that the effect was small and that noise was not considered likely to be a major risk factor for heart disease. On this latter point, however, it should be noted that, given the large numbers dying each year from heart disease (in excess of 10,000 in London in 2001, according to the

[1] **Quantifying burden of disease from environmental noise: Second technical meeting report - Bern, Switzerland, 15 – 16 December 2005.**
http://www.euro.who.int/Document/NOH/Noise_EDB_2nd_mtg.pdf

London Health Observatory[1]), even a small additional risk from noise could equate to significant numbers of premature deaths.

Since the GLA review, a number of further studies have been published, in particular by WHO, and these seem to increase support for the existence of tangible physiological health impacts. A short study to update understanding of the strength of the latest evidence and to apply best available estimates of risk to population exposures to ambient noise in London is therefore appropriate.

3. Objective

This is intended to be a brief desktop study to enable the GLA to update its understanding of the issues and to make preliminary, although as robust as practicable, estimates of the impact of noise on the cardiovascular and related health risk of Londoners, based on available published risk factor estimates and population exposures to noise.

4. Aims

In summary, the aims of the project are to:

1. Briefly review and list the latest evidence and published policies and/or guidance (adopted or proposed) of WHO and public bodies or authorities in other countries on the link between noise and physiological health impacts, providing a view on the robustness of the risk factors they use or currently propose and suggest best estimates of the risk factors and probable margins of error, and summarising plausible causal pathways to assist in popular interpretation of estimates. As far as possible, this should apply a standard of evidence similar to that applied to equivalent risk factors for air quality impacts.
2. Apply the suggested risk factors to available London population noise exposure data derived from maps for road traffic noise, Heathrow and London City Airports to determine overall impacts.
3. Make summary comparisons between the best estimate risk factors and outcomes for two other health impacts from road traffic in London (e.g. air pollution impacts and road traffic accidents), to place the noise outcomes in context.
4. Clearly explain the methodology used to derive the risk factors and to apply these to the population exposure data to derive overall numbers of premature deaths, QALY/DALYS and any other outcomes analysed, so that the client can derive and apply revised factors in future as scientific understanding advances and/or new population exposure data become available.

5. Brief

The proposal is for a brief desktop study, with two reporting elements, to update the GLA's understanding of the evidence for the cardiovascular and related impacts of ambient noise and to quantify, as far as possible, implications for Londoners' health on the basis of the best available evidence.

Element one will review the latest evidence and the policies and/or guidance adopted or currently under consideration (e.g. in draft papers) by the WHO, and also the evidence utilised by public bodies or authorities in other countries that already accept a link between noise and physiological health, listing the bodies that accept a link and their reasons as far as practicable, including a summary of the hypothesised causal mechanisms. The assessment should apply, as far as practicable, a standard similar to that applied to the assessment of the strength of evidence for air quality health impacts. It will report the strength of evidence, including issues related to night time noise exposure and determine the best estimate for the factors and the range of the

[1] http://www.lho.org.uk/HIL/Disease_Groups/Cardiovascular.aspx

most likely values (including applying the risk factors used by public bodies or authorities in other countries) of standard metrics, such as the contribution to overall myocardial infarction mortality rate, number of premature deaths per year and QALYS/DALYS. Summary comparisons will also be made between these results and appropriate equivalent outcomes for up to two other health impacts from road traffic (e.g. impacts of air pollution from road traffic, deaths due to road traffic accidents), chosen in consultation with the client.

Element two will then apply these factors to London noise exposure data (e.g. Defra London Road Traffic Noise Map (LRTNM) and its population exposure data (to be supplied by the client) and, separately, to the published noise contours and population exposures for Heathrow and London City Airports, adapting the factors and/or the exposure data[1] as necessary, to determine the health impacts on those affected (including indication of the ranges of likely values). It should explain clearly how these values have been derived and the methodology used so that they can be applied by the client to future updated maps and to maps for other noise sources (such as railways) individually, and, if feasible, consolidated (multi-source) noise maps, as they become available. Uncertainties in the data and their sources should be clearly stated.

For aircraft noise impacts, in particular, the most exposed populations around Heathrow and London City airports may not be demographically similar to the London average (in terms of age, other health factors affecting cardiovascular disease risk, ethnicity etc.). Quantification of such equalities issues is not expected to be feasible within the resources available for this study. The report should discuss the principles, as far as is possible in the light of current knowledge, governing how demographic differences and associated differences in health profiles could affect risk associated with noise exposure, and indicate where or how far future equalities analysis might be feasible. Health profiles can be found on the London Health Observatory website at http://www.communityhealthprofiles.info/

6. Deliverables

1. A draft report on element one for discussion and agreement with the client, in electronic form.
2. A draft final report for discussion and agreement with the client, in electronic form.
3. A final report - both a printed version and electronic versions in both Word and PDF formats.
4. Provision should be made for a progress meeting following the draft report on element one and a meeting to discuss the draft final report, with the client at City Hall.

7. Timescale

The progress meeting will be held as soon as practicable after provision of the draft report on element one in late December 2007. The draft final report should be provided by 15 February 2008 at the latest, with a discussion meeting as soon as practicable afterwards. The final report should be provided by 14 March 2008 at the latest.

[1] For example, the preliminary WHO risk factors relate to $L_{Aeq,\ 16h}$ noise levels for the period 06.00 to 22.00 (see Footnote 1, especially: Annex 2, Table 2). The LRTNM population exposure data are available for L_{den}, $L_{Aeq,\ 12h}$, $L_{Aeq,\ 4h}$, and $L_{Aeq,\ 8h}$ only and the periods are divided at 07.00, 19.00 and 23.00. Although the WHO report suggests that L_{den} may be used as a proxy for $L_{Aeq,\ 16h}$, this is on the assumption that nighttime noise levels will be approximately 10 dBA lower than daytime ones, which is not generally true for London where noise levels typically fall by much less at night. It will be necessary therefore to use best estimates based on the exposures to the available indices, since fundamental recalculation of the map would be necessary to derive the 06.00 – 22.00 data and is beyond the scope of this study.

Annex 2. PROJECTS / PUBLICATIONS "IN PROGRESS"

Publications

The Effect of Transportation Noise on Health & Cognitive Development: a Review of Recent Evidence.
Charlotte Clark, Stephen A Stansfeld
Queen Mary, University of London
Prepared for International Journal of Comparative Psychology, submitted Nov 2007
[BEL have a copy]

UK Department of Health.
Ad-Hoc Advisory Group on Noise and Health – Report on "Environmental Noise and Health in the UK. In late stages of preparation.

WHO Night Noise Guidelines

WHO EBD

WHO Evidence Review on Aircraft Noise and Health – commenced October 2007, publication due October 2008 – see Annex 3.

Ongoing Projects

Prof Hugh Davies, Univ of British Columbia Canada
See http://www.cher.ubc.ca/UBCBAQS/welcome.htm

Aim: The occurrence of cardiovascular disease among older adults will be correlated, spatially and temporally, with residential noise levels and residential air pollution levels.
Background: During the past two decades, epidemiological studies conducted worldwide have shown an increased risk for cardiovascular disease (CVD), particularly deaths, in relation to both short- and long-term exposure to ambient air pollution. At the same time, studies have demonstrated associations between exposure to road traffic noise and cardiovascular disease. Automobile traffic is a major source of both air and noise pollutants, and therefore the relationship between them and CVD may be complicated, resulting in confounding if study design does not account for this relation. No studies have yet looked at the potential interactions between these two exposures. We are proposing a study to examine the association of residential noise and cardiovascular disease and the interaction of noise and air pollution and cardiovascular disease among a cohort of adults living in the Greater Vancouver Regional District (GVRD).
Previous work by applicants: A cohort of adults has been enumerated who have lived in the Georgia Air Basin for the 5 years prior to 1999 (557,640 women and 512,415 men). For the follow-up period from 1999 through 2003 CVD hospitalizations and deaths among cohort members have been identified by age group and residential address. We have identified cases including acute coronary syndrome, or chronic coronary syndrome, or hypertensive disease. We have estimated air pollution exposure levels, and applied to cohort members using the spatial surfaces.
Aims of proposed study: (1) to develop a noise map for the GVRD; (2) to determine accurate estimates of noise levels at residential building facades, and assign noise exposure levels to cohort members; (3) to examine the relations

between noise exposure and CVD mortality and morbidity; and (4) to examine the interaction between noise and air pollution and CVD.
Methods: The GVRD noise map will be developed using CadnaA software, building on arterial road noise models completed in 2005 by the City. Noise levels will be assigned to individual building facades using GIS coordinates converted to postal codes. Internal comparisons, with people in the lowest noise exposure category as the base-line group, will be performed to examine the relationship between each disease category and indexes of exposure. Only the first hospital visit (during the follow-up period) for each cardiovascular disease category will be used. Follow-up will be censored after first diagnosis. Poisson regression analyses adjusting for sex, age, socio-economic factors, predisposing medical conditions, and other potential risk factors will be used. Combined effects of noise and air pollution will be examined using interaction terms in multivariable Poisson regression models. Lagging of exposure will be used to test for the existence of a latency period. As a sub-study, we will look at the prevalence of major CVD risk factors among members of the cohort who also participated in the Canadian Community Health Survey (CCHS) conducted by Statistics Canada in 2000 (n=6,317). These data will be used to compare the prevalence of smoking and other risk factors between different exposure groups to assess the direction and potential magnitude of confounding.
Relevance: Both air pollution and noise are believed to contribute considerably to the worldwide burden of CVD. Although the major urban source of the two pollutants is the same (motor vehicles) ours will be the first study to investigate them simultaneously and to elucidate the characteristics of their joint effects. This work will result in more precise risk estimates for effects of both pollutants, and better targeting of control measures.

Annex 3. WHO - Working Group – Aircraft noise and health - Babisch and van Kamp. Chapter on Cardiovascular Effects - Version 2 Draft January 2008

NOTE – this text is subject to editing by the WHO WG in the next few weeks, but is provided in full because of its importance to this GLA project.
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WHO Working Group: AIRCRAFT NOISE AND HEALTH

Cardiovascular effects of aircraft noise
2st draft (December 2007) issued to WG January 10 2008

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Abstract

Noise is a stressor that affects the autonomic nervous system and the endocrine system. Under conditions of chronic noise stress the cardiovascular system may adversely be affected. Epidemiological noise studies regarding the relationship between aircraft noise and cardiovascular effects have been carried out on adults and on children focussing on mean blood pressure, hypertension and ischemic heart diseases as cardiovascular endpoints. While there is evidence that road traffic noise increases the risk of ischemic heart disease, including myocardial infarction, there is less such evidence for such an association with aircraft noise. This is partly due to the fact that large scale clinical studies are missing. There is sufficient evidence that aircraft noise increases the risk of hypertension. The results regarding aircraft noise and children's blood pressure are still inconsistent. This may be due to methodological reasons.

Introduction

The auditory system is continuously analyzing acoustic information, which is filtered and interpreted by different brain structures. The hypothesis that long-term exposure to environmental noise – including aircraft noise – causes adverse health effects is based on three major findings and facts:

1) Laboratory studies show that exposure to acute noise affects the sympathetic and endocrine system, resulting in unspecific physiological responses (e.g. heart rate, blood pressure, vasoconstriction, stress hormones, EEG) [1-7].
2) Noise-induced instantaneous autonomic responses do not only occur in waking hours but also in sleeping subjects even when no EEG awakening is present [8-10] [Report NNGL, available soon]. They do not adapt on a long-term basis although a clear subjective habituation occurs after a few nights [11]. Repeated arousal from sleep is associated with a sustained increase in daytime blood pressure [12]. The cortical perception of the sound as well as sub-cortical reflections due to the direct nervous interactions of the acoustic nerve with hypothalamic structures stimulates the autonomous nervous system. From this the hypothesis emerged that long-term exposure to noise adversely affects the homeostasis of the human organism, including metabolic function and the cardiovascular system [13-17]. Persistent changes in endogenous risk factors due to noise-induced dysregulation promote the development of chronic disorders such as atherosclerosis, hypertension and ischemic heart diseases and others in the long run.

3) Although effects tend to be diluted in occupational studies due to the "healthy worker effect", epidemiological studies carried out in the occupational field have shown that employees working in high noise environments are at a higher risk for high blood pressure and myocardial infarction [18-23]. Similar effects may occur with respect to community noise.

The general stress theory referring to the sympathetic-adrenal-medullary system (SAM axis) and the pituitary-adrenal-cortical system (hypothalamic-pituitary-adrenal = HPA axis) is the rational for the non-auditory physiological effects of noise [24,25]. The biological plausibility derives from laboratory experiments on acute noise effects. Studies have been carried out assessing the relationship between road and aircraft noise on cardiovascular endpoints. This chapter is concerned with the epidemiological evidence of this association.

Descriptions of protocol for the review

The focus here is on epidemiological studies or surveys directly related to associations between aircraft noise and cardiovascular disease (CVD) outcomes. Many environmental noise studies refer to road traffic noise, serving as an approximation of effects of transportation noise, in general. In accordance with the reaction model, the endpoints considered in this review are primarily of cardiovascular nature. Noise research has been focusing on these endpoints for reasons of statistical power (high prevalence in the general population) and their impact on public health [26]. A distinction is made between the effects on adults and on children. Clinical manifestations of cardiovascular diseases are not very likely in young people. Therefore blood pressure reading is the major outcome that has been studied in children and adolescents. In adults, however, manifestations of high blood pressure (hypertension) and ischemic heart diseases (myocardial infarction, angina pectoris, ischemic signs in the ECG, heart failure) are major outcomes of interest. The diagnosis is either based on self-reported doctor-diagnosed occurrence/treatment of disease, hospital admission rates, drug medication intake, or on actual blood pressure measurements (taken in rest). The same applies to the assessment of exposure. It is either based on self-reported traffic volume (e. g. type of street) or subjective perception of the noise (disturbance/annoyance), or on modeled noise contours (noise maps, isophones) or noise measurements taken near the subjects' houses. Finally, the type of study (ecologic, descriptive (e.g. cross-sectional study), and analytic (e.g. case-control study, cohort study) is considered as a decision criterion.

Identification of relevant studies

The selection of relevant studies is made on comprehensive previous reviews [18,27,28] and the experts' knowledge about new publications and ongoing research in this field. In a recent review update [29,30] altogether 61 epidemiological studies were identified that addressed the association between transportation noise and cardiovascular endpoint; 20 of which referred to commercial aircraft noise [31-52], 8 to military aircraft noise, 32 to road traffic noise, and 13 to other environmental noise sources. The cardiovascular chapters of the WHO reports "Night Noise Guidelines" and "Environmental Noise Burden of Disease" refer to this review. Studies focusing on low flying jet-fighter noise showed higher blood pressure readings in children but not in adults [53-57]. The effects may largely be due to anxiety and fear rather than to the noise stress as such. These studies are therefore not considered in this present summary on the effects of aircraft noise. However, studies regarding noise from aircraft operations around airfields, which is comparable to commercial aircraft noise

(no steep level increases) are considered [47,58]. New aircraft noise studies are now available that were not considered in previous reviews [59-66].

Systematic assessment of the validity of identified studies

Evaluation criteria for the validity of studies with respect to possible exposure misclassification, confounding, selection bias, recall and observation bias were: objective (noise level) vs. subjective exposure assessment, objective (clinical) vs. subjective assessment of outcome, type of study, reasonable control of confounding factors, statistical methods of analyses, peer-reviewed reference.

Studies on adults
Some studies are not feasible for a synthesis or a meta-analysis, either because only sparse information is given with respect to the study design and selection criteria or confounding factors are insufficiently accounted for [31,50]. Some study results are only preliminary or not yet peer-reviewed [43,51,52,64,67]. However, in those cross-sectional studies - although mostly not significant - higher mean blood pressure readings or a higher prevalence of cardiovascular disorders or medication intake were found in exposed subjects compared with non-exposed, supporting the hypothesis as such (consistency) [68].

Repeated studies carried out around Schiphol airport in the Netherlands looking at aircraft noise and drug medication either on an individual level (self-reported medication intake) or on a spatial level (prescribed medication purchased by pharmacies) revealed higher relative risks of cardiovascular medication ranging between 1.2 and 1.4 for a noise level difference of approximately 10 dB(A) [34,44,60]. When comparing the noise exposure throughout the whole day (L_{den}) with the noise exposure during the night (L_{night}) effects were stronger with respect to L_{den}.
[see Explanatory Note 1 at end of this Annex]
In the most recent phase of the Schiphol environment and health monitoring programme a higher risk of approximately 1.8 was found for the same noise level difference [61,62]. In a longitudinal approach a decrease in the purchase of cardiovascular and antihypertensive drugs was found after a reduction of night flights [36]. A recent cross-sectional study carried out around Cologne airport in Germany demonstrated higher individual prescriptions of antihypertensive and cardiac drugs in subjects exposed to high levels of aircraft noise, particularly, during the night and the early morning hours (3-5 hrs) [59]. The study was unbiased with respect to the assessment of exposure and outcome because objective data were used (noise contours, health insurance records). However, no data regarding individual confounders were available, only spatially aggregated covariates could be considered. Higher risks were found for subjects where L_{night} exceeded 39 dB(A). Preliminary results from a Swedish follow-up study carried out around Stockholm's airport suggest a higher intake of antihypertensive medication in subjects exposed to noise levels ('FBN') of more than 55 dB(A) compared to less exposed (relative risk 1.6). The results are based on a small sub-sample of the total cohort [52].

Regarding the prevalence of hypertension and heart problems much information is derived from Dutch studies carried out around Schiphol airport [34,35,61,62,69,70]. The assessment of high blood pressure and ischemic heart problems was based on clinical measurements [34,35], medical interviews [34,35], hospital admission rates [61,62], and self-reported hypertension [61,62]. In the older studies, a non-significant increase in risk of heart disease was found ranging between 1.1 and 1.4 in people (males and females taken together) who were exposed to 'NNI' > 37 (approximately L_{dn} > 62 dB(A)) [34]. For hypertension a significantly higher risk of 1.7 (95% CI = 1.4-2.2) was found for this noise level difference of approximately 10 dB(A) [34]. Regarding the

prevalence of all cardiovascular diseases, including high blood pressure, a significant relative risk of 1.8 was found [35]. In the later studies, no noise effects were found with respect to hospital admissions for cardiovascular diseases [61,62]. However, a statistical significant effect of L_{den} was found on self-reported hypertension. When the noise increases by 3 dB (A) the odds ratio was 1.2, which corresponds with a relative risk of approximately 1.8 for a 10 dB (A) difference in noise level, confirming the earlier studies. In a new multi-centred study carried out around six European airports a significant increase in the risk of hypertension of 1.1 (95% CI = 1.0-1.3) for a 10 dB(A) difference of aircraft noise during the night (L_{night}) was found [65]. Across categories no clear exposure-response relationship was found. However, the large confidence intervals did not discard the assumption of a linear relationship. No such association was found with respect to the exposure during the day, possibly due to exposure misclassification (time spent outside home). Thus, a smaller relative risk was found for the 24 hr noise indicator L_{den} of 1.1 (95% CI = 0.9-1.3) per 20 dB(A).

A Swedish study carried out around Stockholm's major airport assessed the prevalence of (self-reported doctor-diagnosed) by postal questionnaire. An exposure-response association between aircraft noise and high blood pressure was found with relative risks ranging between 1.1 and 2.1 for noise levels between approximately 'FBN' = 53 to 63 dB(A) [49]. When noise categories were combined, the effect was significant for 'FBN' > 55 dB (A). The analysis of trend resulted in a relative risk of 1.3 (95% CI = 0.8-2.2) per 5 dB (A). Studies carried out around the Kadena military airfield on Okinawa in Japan also demonstrated an exposure-response relationship of an increasing prevalence of clinically assessed hypertension with increasing noise exposure [47]. However, the effects were found at higher noise levels than for civil airports ('WECPNL' > 75 dB, approximately L_{dn} > 60 dB (A)). This may be due to the fact that night- and weekend-flights were largely omitted. Only one prospective study assessing disease incidence is known. The study was carried out around Stockholm's major airport. The association between aircraft noise and high blood pressure was investigated. Subjects exposed to weighted energy-averaged levels ('FBN') above 50 dB(A) had a significant relative risk of 1.2 for the development of hypertension over the 10-year follow-up period compared with less exposed [66]. The increase in risk per 10 dB (A) was 1.2 (95% CI = 1.0-1.2).

Studies on children

Most evidence in relation to aircraft noise on children is derived from school studies carried out in Los Angeles [37,38], the Munich Airport study [39,40,71], the Sydney Airport study [41,42], and the RANCH study [72].

In studies around the Los Angeles airport blood pressure differences of 2 to 7 mmHg were found between groups of exposure depending on the years enrolled in school. The results may be confounded by incomplete control of ethnicity [42]. Blood pressure measures were taken during quiet periods in school, in order to exclude acute noise effects. Longitudinal measurements after a year failed to show a relationship between noise exposure at school and a change in blood pressure, probably due to selective migration of the schoolchildren. The cross-sectional study around the old Munich airport revealed a borderline significant effect of 2 mmHg higher systolic blood pressure readings in schoolchildren from noise exposed areas (Leq, 24hr = 68 dB(A)) as compared to unexposed children ($L_{eq, 24hr}$ = 59 dB(A)). No noise effect was found with regard to diastolic blood pressure [39]. Longitudinal studies carried out around the new airport showed a 2 to 4 mmHg larger increase in BP readings in exposed children than in their counterparts from the quiet areas 18 months after the opening of the new airport. However, the well-matched children from the exposed and the control group had the same absolute blood pressure. The higher change in blood pressure was due to lower values at the beginning of the follow-up. The cross-

sectional study around Sydney Airport revealed a non-significant relation between aircraft noise and diastolic and systolic blood pressure in children [42].

In a cross-sectional study carried out around Schiphol and Heathrow Airports on schoolchildren (RANCH) a non-significant relationship was found between aircraft exposure at school (LAeq, 7-23 hr) and measured systolic blood pressure, diastolic blood pressure and heart rate after adjustment for relevant confounders [72]. However, aircraft noise at home (expressed as LAeq, 7-23hr) was significantly related to higher systolic (0.10 mmHg/dB (A)) and diastolic (0.19 mmHg/dB (A)) blood pressure. Chronic aircraft noise exposure during the night (LAeq, 23-7hr) at home was also positively associated with blood pressure. This latter association was significant only for systolic blood pressure. In the pooled dataset an increase of 0.09 mmHg/dB (A) was found. Due to significant differences in noise effects between the two centres no univocal conclusions about the association between aircraft noise exposure and blood pressure in children could be drawn [72]. Explanations put forward concern differences in flight pattern variation, and aircraft fleet. Also differences in schooling systems and teachers' attitudes towards noise might have differential effects on the children's reactions to noise. None of these could be tested on the available data. Finally, even though the results were adjusted for ethnic differences and diet residual confounding due to these factors might explain the differences [73].

The use of meta-analysis

Different approaches have been used to assess pooled effect estimates and exposure-response relationships in order to carry out a quantitative risk assessment. Van Kempen et al. [18] calculated uniform regression coefficients across all noise categories within individual studies ('regression approach'). The regression coefficients were then pooled over all studies. Babisch [29] calculated pooled relative risks for individual noise categories from different noise studies, which were then considered for an exposure-response relationship ('category approach'). Both approaches have advantages and disadvantages. The regression approach has the advantage that regression coefficients can easily be pooled regardless of actual noise levels; only the slope (regression coefficient) of the exposure-response relationship is taken into account. For example, some studies showed high risks at relatively low noise levels [49], while others showed an increase of risk only at higher noise exposures [47]. The disadvantage is that the linear regression approach does not account for non-linear associations and possible thresholds of effects. The category approach is noise level oriented. Only relative risks from different studies referring to the same noise category are pooled to derive an exposure-response curve. This has the advantage that possible thresholds of effects can be determined. The approach also accounts for non-linear associations. It is less likely to obscure possible higher risks in higher noise categories in which the numbers of subjects is relatively small - which is the case in random population samples given the empirical noise distribution, and specifically around large airports. For example, in case of j-shaped or quadratic associations an overall regression coefficient underestimates the risks in higher noise categories, simply because the slope of the regression line is primarily determined by the larger numbers of subjects in the lower exposure categories, where effects may be smaller. The disadvantage of this approach is that it relies on relatively homogeneous and comparable noise indicators in order to pool the effect estimates from different studies within noise categories. One could think of studies where relationships within the studies reflect true associations, but the noise assessment in absolute terms may not be comparable due to methodological reasons (e. g. measurement vs. modeling, different calculation methods, different time periods, weighing factors, different reference points, different sides of the house, etc.).

For both approaches it is essential that critical decisions are made as to which studies are included in the meta-analyses and which are not. Studies that are not suitable with respect to issues of exposure misclassification, selection bias, observation bias, or confounding should be excluded from the meta-analyses. Only a very few epidemiological studies are available on adults, in which the association between aircraft noise and clinical states of cardiovascular diseases were assessed. Five studies appear reasonably valid for further consideration because minimum requirements regarding the validity of the assessment of exposure, outcome and the statistical control for confounding factors were fulfilled [34,47-49,65,66]. However, noise level related data pooling ('categorical approach') is difficult due to the fact that different (national) exposure indices were used. A graphical presentation of results using approximations with respect to the common noise indicator L_{dn} is shown in *Figure 1*. No conclusions regarding possible threshold values or noise level related risks (in absolute terms) can be made.

When linear trend coefficients of all the five studies are calculated and pooled afterwards ('regression approach') the pooled effect estimate of the relative risk is 1.13 (95% CI = 1.00-1.28) per 10 dB(A). The results are shown in *Table* 1. The pooled effect estimate is significant. No major difference between fixed and random effect models[1] found when the individual coefficients obtained from the six airports of the HYENA study are considered individually in the meta-analysis to better account for the heterogeneity between individual studies. (Note: If the pooled Hyena results are used instead as shown in Figure 1, significant fixed and random effect estimates of 1.12 and 1.29, respectively, are calculated.) The result is almost the same when either the 'Okinawa study' (military aircraft noise, out-dated noise data) or the 'Stockholm1 study' (subjective assessment of exposure) or both are excluded from the meta-analysis due to their low statistical weights (OR = 1.12, 95% CI = 0.98-1.28).

The calculations were made using the procedures 'Meta' and 'Metareg' of the statistical package Stata, Version 9. Individual odds ratios and confidence intervals were taken from summary reports [29] and the original publications for this purpose [65,66] to calculate regression coefficients of individual studies and odds ratios with respect to the weighted day/night noise indicator L_{dn}, which is supposed to be very similar to L_{den}. However, one has to have in mind that different criteria and assessment methods for hypertension were used. For example, some studies refer to the 'old' WHO criterion of 160/100 mmHg [34,48,49], others refer to the 'new' WHO criterion of 140/90 mmHg [65,66].

[1] See Glossary

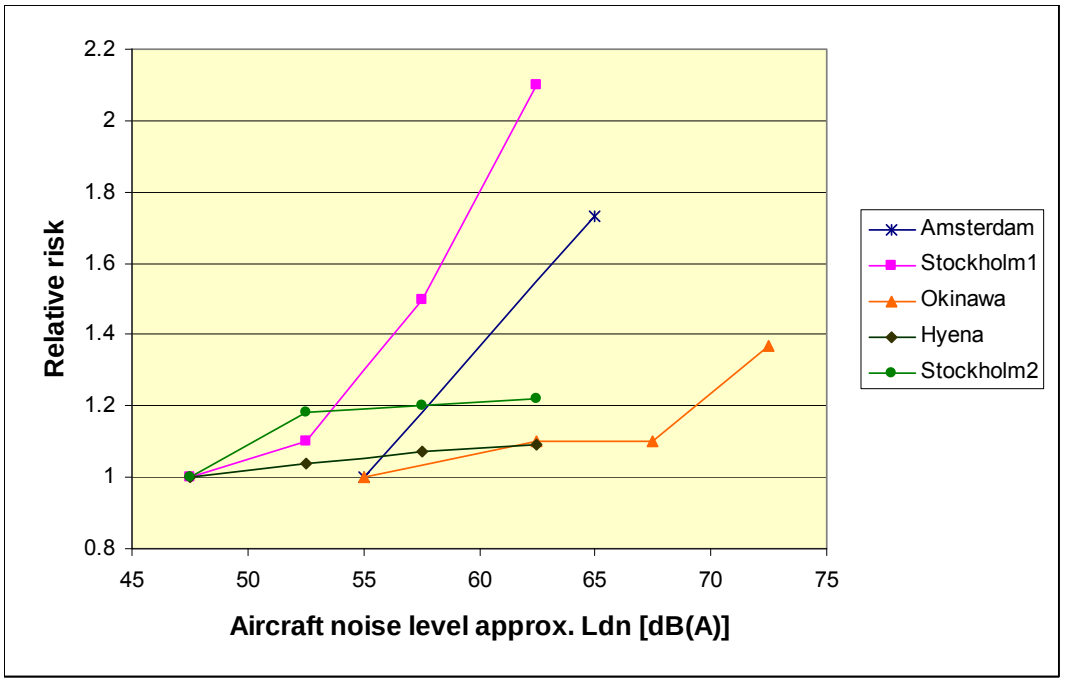

Figure 1. **Association between aircraft noise level and the prevalence of hypertension.**

***Table 1*. Meta analysis of epidemiological studies of the association between aircraft noise and hypertension (Note: Individual logistic regression coefficients are pooled. The studies differ with respect to study type, hypertension criteria and exposure indicators)**

Study	No. of subjects	Fixed weight	Random weight	Effect estimate per 10 dB(A)	95%-confidence interval	P-value
Amsterdam	5,828	76.55	28.05	1.73	1.38 - 2.16	
Stockholm 1	2,959	3.75	3.46	1.69	0.61 - 4.65	
Stockholm 2	2,392	140.37	33.65	1.21	1.03 - 1.43	
Okinawa	28,781	17.91	12.75	1.27	0.80 - 2.02	
Hyena-London	600	107.38	31.35	1.05	0.87 - 1.27	
Hyena-Berlin	972	209.93	36.56	1.18	1.03 - 1.35	
Hyena-Amsterdam	898	78.39	28.29	0.99	0.79 - 1.24	
Hyena-Stockholm	1,003	95.67	30.26	0.87	0.71 - 1.06	
Hyena-Athens	635	47.37	22.88	1.14	0.86 - 1.52	
Hyena-Milan	753	105.98	31.22	0.99	0.82 - 1.20	
Pooled fixed				1.13	1.06 - 1.20	0.000
Pooled random				1.13	1.00 - 1.28	0.044
Heterogeneity				Q = 26.13		p = 0.002

Conclusions

The general conclusion is that there is sufficient evidence for a positive relationship between aircraft noise and high blood pressure and the use of cardiovascular medication. However, no single common exposure-response relationship can be established for the association between aircraft noise and cardiovascular risk due to methodological differences between studies and the lack of continuous or semi-continuous (multi-categorical) noise data. For the same reason no answer can be

given regarding possible effect thresholds. Road traffic noise studies suggest that the cardiovascular risk increases when the outdoor noise level during the day exceeds 60-65 dB(A) and 50-55 dB(A) during the night, respectively. As to whether this information can be applied to aircraft noise remains unclear. However, this may be a conservative approach, considering on the basis of annoyance studies that aircraft noise effects may even be stronger than those of road traffic noise. Annoyance studies showed that aircraft noise is more annoying than road traffic noise of the same average noise level [74,75], which might partly be explained by less exposure misclassification (no shielding of aircraft noise, no unexposed rooms[1]). New aircraft noise studies suggest that the risk may increase at even lower night noise levels. Depending on whether high blood pressure was assessed by a self-administered postal questionnaire or by clinical measurements, the magnitudes and the possible thresholds of effect varied between and within studies [62,64]. Effects were more pronounced, when subjective measurements of high blood pressure were considered. This may raise questions regarding over-reporting [62,64,76]. The validity of study results appears to be even more a problem when subjective noise annoyance was considered for exposure [44,61,62,64]. The effect estimates tend to be larger but may be prone to over-reporting, particularly in cross-sectional studies where both, exposure and outcome, are assessed on a self-reported basis with the same questionnaire.

In the present summary, only those studies were considered in which aircraft noise was the explicit noise source. However, in a situation where information is lacking, the results of studies on the association between road traffic noise and myocardial infarction may also serve as an approximation for possible effects of aircraft noise [WHO reports 'Night Noise Guidelines' and 'Environmental Noise Burden of Disease', both available soon].

The available results do not allow for a distinction between the sexes. Males have been studied much more often than females. There is some indication that males may be more affected by road traffic noise [65,77-79]. However, also contradictory results were found [80]. The database is too weak for final conclusions regarding any gender differences.

NOTE: because there is not enough data, even to determine gender differences, then, although it would be interesting and useful to look at other factors for population sub-groups, e.g ethnicity, the problem of insufficient data is even more pronounced.

Due to the use of different noise indicators in aircraft noise studies only very crude comparisons can be made between studies on the basis of common noise indicators, e. g. L_{dn} or $L_{Aeq,6\text{-}22hr}$. Most aircraft noise studies did not distinguish between day and the night. A road traffic noise study and two aircraft noise studies suggest that noise during the night may be more harmful than during the day [59,65,81]. However, no firm conclusions can be drawn about the relative contribution of day and night exposure because noise indices are usually highly correlated. One study suggests not only that noise during the night may be the primary source of adverse effects; it also shows that within the night period, effects due to noise in the early morning shoulder hours may be larger [59].

The contribution of noise on children's blood pressure is still not fully understood. Predispositional and lifestyle factors seem to dominate and it is hard to study the influence of environmental noise separate from these. This might be one of the reasons why conclusions about the effect of noise exposure on children's blood

[1] Note – this could be due to the fact that the noise level of individual aircraft events is above that of traffic noise levels, at the same L_{Aeq}.

pressure are limited and inconsistent. Methodological problems which arise are study size, insufficient contrast between noise levels, selection bias and insufficient adjustment for factors such as SES, parental history, noise insulation and ethnicity. Moreover, most studies on cardiovascular effects in children have focused on school exposure while at least the combination of day- and night time exposure and the related lack of restoration might be of importance in the development of cardiovascular disease due to early childhood blood pressure changes.

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Explanatory Note 1

This WHO Working Group paper, in summarising and discussing recent work carried out around Schiphol airport in the Netherlands [see page 49] notes that

"When comparing the noise exposure throughout the whole day (L_{den}) with the noise exposure during the night (L_{night}) effects were stronger with respect to L_{den}."

In fact this comment refers only to the study by Franssen in 2004 [Reference 44 in the WHO Paper].

Franssen EAM, Wiechen CMAG, Nagelkerke NJD, Lebret E. Aircraft noise around a large international airport and its impact on general health and medication use. Occup. Environ. Med. 2004;61:405-413.

It is important to see the whole text on this issue – as follows – with Bold text where relevant – as well as Table 5 from the journal paper.

Table 5 presents the associations between health indicators and aircraft noise exposure measures. Associations with Lden are all positive and statistically significant, except for prescribed sleep medication or sedatives and its frequent use. **The health indicators do not appear to be related to noise exposure during the night (LAeq, 23–07 h). However, the use of nonprescribed sleep medication or sedatives is associated with aircraft noise exposure in the late evening (LAeq, 22–23 h) with an OR of 1.72.** Analyses of the separate VOEG items showed statistically significant relations of Lden with six health complaints (ORs for an increase of 10 dB(A)): shortness of breath (OR=1.29, 95% CI 1.09 to 1.53); feelings of tiredness (OR=1.34, 95% CI 1.17 to 1.53); headache (OR=1.16, 95% CI 1.01 to 1.34); tired sooner than considered normal (OR=1.47, 95% CI 1.26 to 1.70); listlessness (OR=1.17,95% CI 1.01 to 1.36); and tired and not fully rested in the morning (OR=1.20, 95% CI 1.03 to 1.41). For the remaining seven complaints the ORs were lower, ranging from 0.99 to 1.17, and not statistically significant. Table 6 shows the relation of the health indicators with Lden when this noise measure was categorised. The ORs tend to rise with increasing noise levels, but differences between the categories are not statistically significant.

Table 5 Odds ratios (OR) and 95% confidence intervals (CI) after multiple logistic regression of health indicators, in relation to various noise exposure measures per 10 dB(A) increase in noise levels, controlling for potential determinants

Health indicator	n_{total}	n_{effect}	Noise measure	OR	95% CI
Poor self-rated health (single question)	10412	1969	L_{den}	1.23	1.04 to 1.46
			$L_{Aeq,\ 23–07\ hrs}$	1.05	0.91 to 1.22
Poor self-rated health (VOEG score)	9887	1871	L_{den}	1.21	1.02 to 1.43
			$L_{Aeq,\ 23–07\ hrs}$	1.08	0.94 to 1.25
Medication for cardiovascular diseases/increased blood pressure	10105	1316	L_{den}	1.30	1.06 to 1.60
			$L_{Aeq,\ 23–07\ hrs}$	1.13	0.94 to 1.35
Prescribed sleep medication or sedatives	7240	516	L_{den}	1.25	0.93 to 1.68
			$L_{Aeq,\ 23–07\ hrs}$	0.91	0.70 to 1.18
			$L_{Aeq,\ 22–23\ hrs}$	1.26	0.99 to 1.60
Non-prescribed sleep medication or sedatives	7240	309	L_{den}	2.34	1.63 to 3.35
			$L_{Aeq,\ 23–07\ hrs}$	1.20	0.87 to 1.65
			$L_{Aeq,\ 22–23\ hrs}$	1.72	1.27 to 2.32
Frequent use of sleep medication or sedatives	7175	189	L_{den}	1.02	0.63 to 1.65
			$L_{Aeq,\ 23–07\ hrs}$	1.36	0.91 to 2.04
			$L_{Aeq,\ 22–23\ hrs}$	1.15	0.78 to 1.70

Annex 4. Glossary

In order of occurrence of terms – not alphabetical.

MEDICAL TERMINOLOGY

Cardiovascular disease
Disease of the heart and blood vessel system, such as coronary heart disease, heart attack, high blood pressure, stroke, angina and rheumatic heart disease.

Hypertension
Chronically elevated blood pressure

Ischaemic [or Ischemic]
Refers to the state of not having enough blood flow.

Ischaemic heart disease
Includes clinical symptoms of angina pectoris (chest pain), myocardial infarction (heart muscle damage), or electrocardiogram (ECG) abnormalities.

Angina pectoris
Chest pain or breathlessness caused by lack of blood flow to the heart.

Myocardial infarction
Heart attack.

STATISTICAL TERMINOLOGY

Exposure-response relationship
Mathematical relationship between the amount or level of a factor, such as noise to which a group or individual was exposed, and the response. It is a general term, used whether the "response" is annoyance, sleep disturbance, or other health effect, such as cardiovascular effects.

Meta-analysis
A statistical technique for combining and integrating the data derived from a number of experimental studies undertaken on a specific topic.

Odds Ratio [OR]
The ratio of the odds of an event in the experimental (intervention) group to the odds of an event in the control group. Odds are the ratio of the number of people in a group with an event to the number without an event. Thus, if a group of 100 people had an event rate of 0.20, the event happened to 20 people and did not happen to 80, and the odds would be 20/80 or 0.25.

Relative risk (RR, or risk ratio)
The ratio of the probability of developing, in a specified period of time, an outcome among those receiving the treatment of interest or exposed to a risk factor, compared with the probability of developing the outcome if the risk factor or intervention is not present (i.e. the ratio of risk in the treated group to the risk in the control group).

Fixed and random weighting
This refers to the use of fixed, or random effects models in multi-level statistical modelling, of the kind used in epidemiological studies of noise and health, and meta-analyses of such studies.

Fixed effects model
A statistical model that stipulates that the units under analysis (e.g. people in a trial or study in a meta-analysis) are the ones of interest, and thus constitute the entire population of units. Only within-study variation is taken to influence the uncertainty of results (as reflected in the confidence interval) of a meta-analysis using a fixed-effect model. Variation between the estimates of effect from each study (heterogeneity) does not affect the confidence interval in a fixed-effect model.

Random effects model
A statistical model sometimes used in meta-analysis, in which both within-study sampling error (variance) and between-studies variation are included in the assessment of the uncertainty (or confidence interval) of the results of a meta-analysis (see Fixed effect model). If there is significant heterogeneity among the results of the included studies, random-effects models will give wider confidence intervals than fixed-effects models.

Monotonical function
A function is monotonical if it only increases or only decreases.