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Review and Synthesis of Epidemiological Studies

Dose-effect Curve and Risk Estimation

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by

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Federal Environmental Agency

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Transportation Noise and Cardiovascular Risk

Review and Synthesis of Epidemiological Studies, Dose-effect Curve and Risk Estimation

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Summary

The auditory system is continuously analysing acoustic information, which is filtered and interpreted by different cortical and sub-cortical brain structures. According to the general stress concept, repeated autonomic and endocrine responses can result in permanent functional and metabolic changes of the organism in chronically exposed subjects. Epidemiological studies suggest a higher risk of cardiovascular diseases, including high blood pressure and myocardial infarction, in subjects chronically exposed to high levels of road or air traffic noise. Sixty-one epidemiological noise studies were evaluated regarding the relationship between transportation noise and cardiovascular outcomes. A meta-analysis was conducted using strict inclusion/exclusion criteria for the studies. As a result, a common risk curve is derived for the relationship between road traffic noise 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 (in reference to $\leq$60 dB(A)). Using data from the national health statistics and estimates of the "Umweltbundesamt" regarding the traffic noise exposure, population attributable risk percentages are calculated for Germany. According to the results, approx. 4,000 MI cases per year (calculations were made for the year 1999) are attributed to the road traffic noise. If the risk curve is universally applied to all ischaemic heart diseases (IHD), the number would be approx. 27,000 IHD cases per year.

Verkehrslärm und kardiovaskuläres Risiko

Überblick und Synthese epidemiologischer Studien, Dosis-Wirkungs-Kurve und Risikoabschätzung

Wolfgang Babisch
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Zusammenfassung

Das Gehör analysiert fortwährend akustische Informationen, die von verschiedenen kortikalen und subkortikalen Strukturen gefiltert und analysiert werden. Dem allgemeinen Stressmodell zufolge können wiederholt auftretende autonome und endokrine Reaktionen bei chronisch exponierten Personen dauerhafte funktionelle und Stoffwechselveränderungen im Organismus bewirken. Epidemiologische Studien deuten auf ein erhöhtes Herz-Kreislaufrisiko bei chronisch verkehrslärmbelasteten Personengruppen (z. B. erhöhter Blutdruck oder Herzinfarkt). Einundsechzig epidemiologische Lärmstudien wurden hinsichtlich eines Zusammenhangs zwischen Verkehrslärm und kardiovaskulären Endpunkten evaluiert. Unter Berücksichtigung strikter Ein- und Ausschlusskriterien wurde eine Meta-Analyse der Studien durchgeführt. Als Ergebnis wurde eine Dosis-Wirkungskurve für den Zusammenhang zwischen Straßenverkehrslärm und der Inzidenz von Myokardinfarkt (MI) abgeleitet. Unterhalb von Straßenverkehrslärmpegeln von 60 dB(A) tagsüber (L_{day}: 6-22 h) konnte keine nennenswerte Erhöhung des Risikos festgestellt werden. Bei Verkehrslärmpegeln über 60 dB(A) zeigte sich ein kontinuierlicher Anstieg des MI-Risikos mit relativen Risiken (Odds ratios) von 1,1 bis 1,5 über den Schallpegelbereich von 61-80 dB(A) in Referenz zu ≤60 dB(A). Unter Verwendung von Daten der Gesundheitsberichtserstattung und Schätzungen des Umweltbundesamtes zur Verkehrslärmbelastung in Deutschland wurden bevölkerungsattributable Risikoanteile berechnet. Den Ergebnissen zufolge ist von jährlich ca. 4.000 MI-Fällen auszugehen (Berechnungen für das Jahr 1999), die dem Straßenverkehrslärm zuzuschreiben sind. Bei Anwendung der Risikokurve gleichermaßen auf alle ischämischen Herzkrankheiten (IHK) wären jährlich ca. 27.000 IHK-Fälle anzunehmen.

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1. Introduction

In section 1, chapter 6 of the Agenda 21 of the global action plan of the United Nations' Conference held in Rio de Janeiro in 1992 (UN 1993), five health-related target areas were addressed. Three of these could be directly applied to community noise. These are: the reduction of health risks related to the environment, the health problems in cities and the protection of sensitive groups (Schwenk 2000). Furthermore, it was stated explicitly in the protocol, that "Nationally determined action programmes, with international assistance, support and coordination, where necessary, in this area should include: Develop criteria for maximum permitted safe noise exposure levels, and promote noise assessment and control as part of environmental health programmes". Transportation noise was addressed as a major factor of concern in this respect in the Green Paper of the European Commission on future noise policy and at the 3rd European Ministerial Conference held in London in 1999 (European Commission 1996; WHO 1999). The issue of action plans to reduce harmful effects of noise exposure is addressed in the European Directive relating to the assessment and management of environmental noise (Directive 2002/49/EC 2002). However, the criteria for a quantitative risk assessment are not yet established.

It is a common experience that noise is unpleasant and affects the quality of life. It disturbs and interferes with activities of the individual including concentration, communication, relaxation and sleep (Schwela 2000; WHO 2000; WHO Regional Office for Europe 2000b). Besides the psychosocial effects of community noise, there is concern about the impact of noise on public health, particularly regarding cardiovascular outcomes (Passchier-Vermeer and Passchier 2000; Stansfeld et al. 2000a; Suter 1992). Non-auditory health effects of noise have been studied in humans for a couple of decades using laboratory and empirical methods. Biological reaction models have been derived, which are based on the general stress concept (Henry and Stephens 1977; Ising et al. 1980; Lercher 1996; Selye 1956).

Amongst other non-auditory health endpoints, short-term changes in circulation including blood pressure, heart rate, cardiac output and vasoconstriction as well as stress hormones (epinephrine, norepinephrine and corticosteroids) have been studied in experimental settings for many years (Babisch 2003b; Berglund and Lindvall 1995). However, not all biologically notifiable effects are of clinical relevance. Classical biological risk factors have been shown to be elevated in subjects that were exposed to high levels of traffic noise (Algers et al. 1978; Arguelles et al. 1970; Babisch and Gallacher 1990; Babisch et al. 1990; Dugué et al. 1994; Eiff et al. 1974; Eiff et al. 1981a; Goto and Kaneko 2002; Knipschild and Sallé 1979; Lercher and Kofler 1993; Manninen and Aro 1979; Marth et al. 1988; Rai et al. 1981; Schulte and Otten

1993a; Verdun di Cantogno et al. 1976; Yoshida et al. 1997). From this, the hypothesis emerged that persistent noise stress increases the risk of cardiovascular disorders including high blood pressure (hypertension) and ischaemic heart disease:

- Sound/noise is a psychosocial stressor that activates the sympathetic and endocrine system.
- Acute noise effects do not only occur at high sound levels in occupational settings, but also at relatively low environmental sound levels when, more importantly, certain activities such as concentration, relaxation or sleep are disturbed.

The questions that need to be answered are:

- Do these changes observed in the laboratory habituate or do they persist under chronic noise exposure?
- If they habituate, what are the physiological costs? If they persist, what are the long-term health effects?

Laboratory experiments on humans, as far as ethically acceptable, help us understand the effect mechanisms and can reveal individual reaction thresholds as the aftermath of acute noise exposure. However, they only offer provisional information on the long-term effects of chronic exposure and the possible genesis of ill-health. Aspects of adaptation, habituation and physical exhaustion (in the sense of the stress model) remain to a large extent unconsidered. In addition to this, the laboratory scenario influences the results of the study because of a changed acceptance of noise stimulation by the test person. Subjective experience of the noise situation, with all the consequences of annoyance, irritation and the disturbance of activities, plays an essential part in terms of physical reactions to noise.

For the same reasons, animal experiments used to find the answers to toxicological questions offer no effective alternative. This particularly applies to low-dose environmental levels. For the sake of efficiency, physiological effects are usually provoked by very high levels of exposure in animal experiments. This may cause general irritations in the organism, played out nonspecifically and independently of the actual exposure factor. Since the noise reactions themselves represent nonspecific stress reactions, the causal effects cannot always be completely separated from each other. In addition to this, the fundamental problem of transferring effect models and thresholds, derived from animal experiments to humans still exists. For this reason, quantitative derivations with reference to humans are only conditionally possible.

Just as uncertain is the extrapolation of noise effects in the environmental range (low dose range) from the results of epidemiological studies with industrial noise (high dose range). Not only the sound intensity influences the noise effects, but also the time structure and the frequency spectrum of the noise, the activity being carried out at the time, the time and place of the noise effects and attributes from the source of the sound itself. For this reason it is hardly surprising if, for example, an average of 85 dB(A) at the workplace induces less bodily reaction than 40 dB(A) during sleep at home.

Epidemiological research provides the possibility of an integral risk estimation based directly on empirical data gained under genuine conditions of exposure, taking into account any factors which may amplify or attenuate the noise effects. Determination of such effect modifications and identification of the groups at risk is an important assignment of future noise effects research (Thompson 1996). Exposure-effect relationships derived from epidemiological data offer a reliable basis for the determination of environmental standards (Adami and Trichopoulos 1999; Hertz-Picciotto 1995; Pearce 1999; Savitz et al. 1999; Soskolne 1999). It can be used for the derivation of “no/lowest observed adverse effect levels (NOAEL/LOAEL)” (Samet et al. 1998), which are important determinants in public health policy.

Large-scale epidemiological studies have been carried out for a long time (Babisch 2000). The studies suggest, that transportation noise is associated with adverse cardiovascular effects, in particular ischaemic heart disease. The epidemiological evidence is constantly increasing (Babisch 2002; Babisch 2004a). Other important health endpoints that have been intensively investigated in relation to chronic noise exposure are disrupted sleep (Ouis 1999; Passchier-Vermeer 2003a; Passchier-Vermeer 2003b), mental health (Stansfeld et al. 2000b), and effects on the endocrine system (Babisch 2003b; Ising and Braun 2000).

Decision-making and risk management rely on a quantitative risk assessment. Since many of the stress indicators and risk factors that have been investigated in relation to noise, impose a higher risk of cardiovascular diseases for noise exposed subjects, the focus in noise epidemiology is on cardiovascular health, including mean blood pressure, hypertension and ischaemic heart diseases. Furthermore, its relevance for public health comes from the high prevalence of cardiovascular diseases in developed and industrialized countries. Ischemic heart diseases are one of the major causes of premature death in modern societies (Doll 1992; WHO Regional Office for Europe 1999). The biological plausibility of the association derives from the numerous noise experiments that have been carried out in the laboratory.

2. Noise and stress - reaction model

The auditory system is continuously analysing acoustic information, which is filtered and interpreted by different cortical and sub-cortical brain structures. The limbic system, including the hippocampus and the amygdala, plays an important role in the emotional processing pathways (Spreng 2000). It has a close connection to the hypothalamus that controls the autonomic nervous system and the hormonal balance of the body. In laboratory studies, changes in blood flow, blood pressure and heart rate were found in subjects exposed to noise, as well as increases in the release of stress hormones including the catecholamines adrenaline and noradrenaline, and the corticosteroid cortisol (Babisch 2003b; Berglund and Lindvall 1995; Maschke et al. 2000). Such changes also occur during sleep without the involvement of cortical structures. The amygdalae has the capacity to learn due to it's plasticity, particularly with respect to the meaning of sound stimuli (e.g. danger of an approaching lorry) (Spreng 2000; Spreng 2004).

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 1 shows a reaction schema used in epidemiological noise research for hypothesis testing (Babisch 2002). 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.

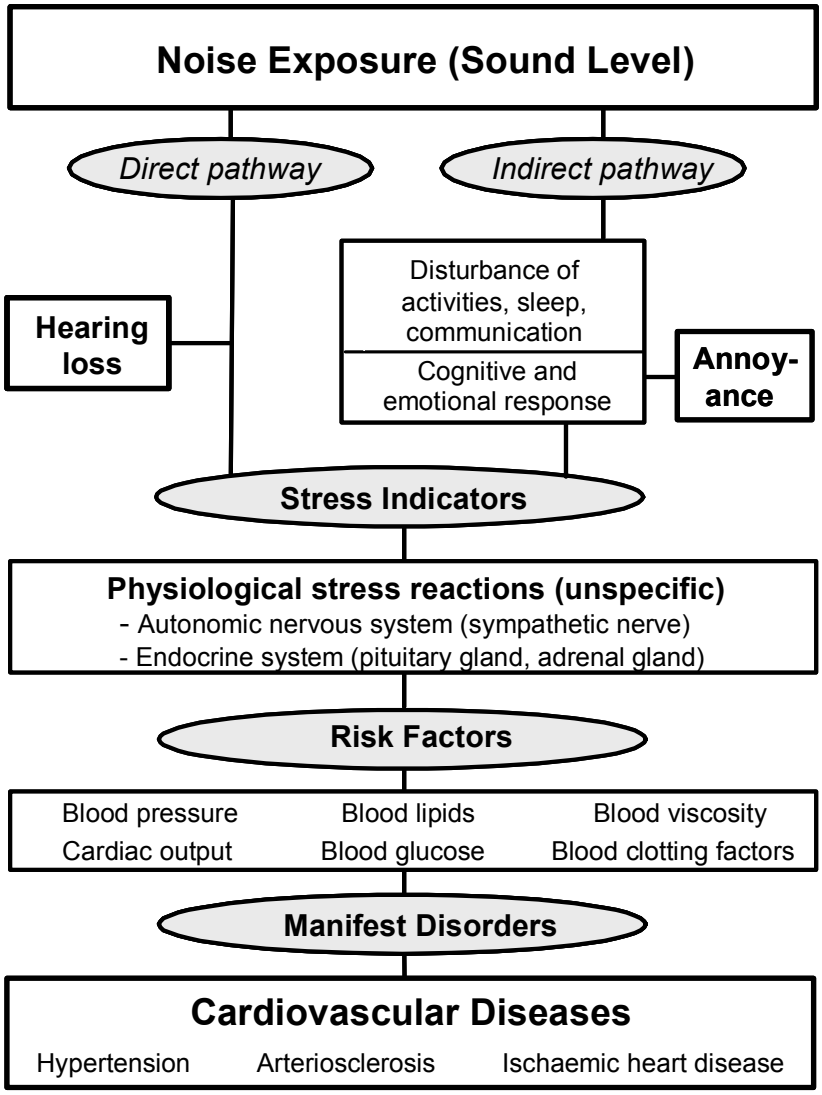


Figure 1. Noise effects reaction scheme (Babisch 2002)

Principally, the effects of environmental noise cannot be extrapolated from results of occupational noise studies. The two noise environments cannot be merged into one sound energy-related exposure-effect model (e. g., a simple 24 hour average noise level measured with a dose-meter). Noise effects are not only depending on the sound intensity but also on the frequency spectrum, the time pattern of the sound and the individuals' activities, which are disturbed. Therefore, epidemiological studies carried out under real-life conditions can provide the basis for a quantitative risk assessment. Other noise sources might act as confounders

and/or effect modifiers on the association of interest. It was shown that the effects of road traffic noise (at home) were stronger in subjects that were also exposed to high noise levels at work (Babisch et al. 1990).

3. Previous reviews on environmental noise and cardiovascular risk

Classical, systematic and quantitative reviews have been published in the past, summarizing the results of studies that were carried out up to the end of the last century. The obstacles of such reviews were discussed in the respective literature (Dickersin 2002). Expert groups have assessed the evidence of the relationship between community noise and cardiovascular disease outcomes (Babisch 2000; Berglund and Lindvall 1995; Health Council of the Netherlands 1994; Health Council of the Netherlands 1999; Health Council of the Netherlands 2004; IEH 1997; Morrell et al. 1997; Passchier-Vermeer and Passchier 2000; Porter et al. 1998). Included was a classical review and synthesis report by Babisch (Babisch 2000) and a systematic review (meta-analysis) by v. Kempen et al. (Kempen et al. 2002). The status of evidence of the relationship between transportation noise and cardiovascular health as concluded in the literature was summarized as follows (Babisch 2002; Babisch 2004a).

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

The highest degree of evidence was for the association between community noise and ischaemic heart disease. Regarding hypertension the ratings were extremely heterogeneous.

With regard to hypertension, the relative risk found in four significantly positive studies was in the range between 1.5 and 3.3 for subjects who live in areas with a daytime average sound pressure level (L_{day}) in the range of 60-70 dB(A) or more (Babisch 2004a). However, significantly negative associations were also found. Across all studies no consistent pattern was seen for the relationship between transportation noise level and prevalence of hypertension. Exposure-effect relationships, which may support a causal interpretation of the findings, were rarely studied. When subjective ratings of noise or disturbances due to traffic noise were considered, the relative risks ranged from 0.8 to 2.3.

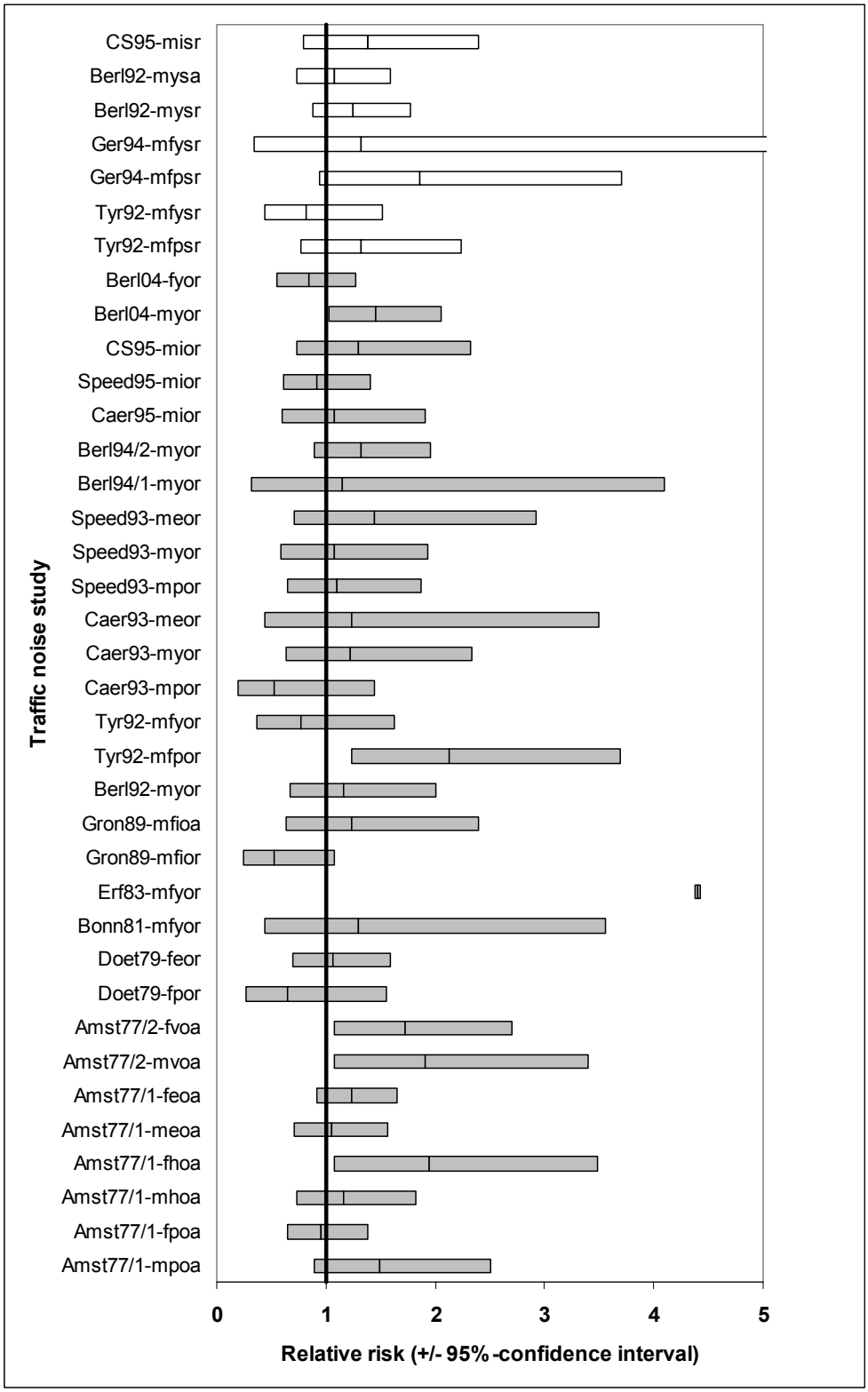
In a meta-analysis by v. Kempen et al. (Kempen et al. 2002), it was concluded that the relative risk of hypertension due to aircraft noise was 1.26 per increase of 5 dB(A) (95% CI: 1.14-1.39, L_{day} = 55 to 72 dB(A)). But only one study (Knipschild 1977a) was considered in the meta-analysis for the risk estimate. The exposure assessment as described in the reference of this study suggested two clusters rather than a continuous distribution of noise levels, which raises some concern when fitting the data using a continuous regression model (increase in risk per decibel). With respect to 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 (Kempen et al. 2002). Two cross-sectional studies were considered in this calculation (Knipschild et al. 1984; Knipschild and Sallé 1979).

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. This is illustrated in *Figure 2*. The entries are relative risks with 95% confidence intervals for dichotomic comparisons of noise exposure (extreme groups or high vs. low). The dark-shaded bars in the diagram refer to studies where the noise exposure was determined objectively (noise levels), the light-shaded bars where it was determined subjectively (annoyance). Road traffic and aircraft noise studies are here viewed together. No corresponding results are available for rail traffic studies. If different subgroups of the population (males/females) or different health endpoints were taken into account, specific studies appear more than once in the illustration. If a series of studies from a particular area under investigation were published in the same year, this is indicated by a serial number behind the year (e. g. “Amst77/1-mpoa” means Amsterdam, 1977, Study 1, males, angina pectoris, objective exposure, aircraft noise).

Some studies permit reflections on exposure-effect relationships. These mostly prospective studies suggest an increase in risk for outdoor noise levels above 65-70 dB(A) during the daytime, the relative risks ranging from 1.1 to 1.5 (Babisch 2004a). Noise effects were larger when mediating factors like years in residence, room orientation and window opening habits were considered in the analyses.

In the meta-analysis by v. Kempen et al. (Kempen et al. 2002) where two cross-sectional studies were considered (Babisch et al. 1993a), 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)) (Kempen et al. 2002). The meta analysis though did not consider a possible threshold of effect. 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)).



Captions: sex*:* f female, m male; noise measurement*:* o objective (sound level), dark-shaded beam; s subjective (annoyance), light-shaded beam; type of noise*:* a aircraft noise; r road traffic noise; ischemic heart disease*:* e ECG-ischemic signs, h heart complaints, i ischemic heart disease, p Angina pectoris, v cardiovascular complaints in general, y heart attack

Figure 2. Results of epidemiological studies on the association between traffic noise and ischaemic heart disease (original figure (Babisch 2002) has been modified according to the results of (Babisch 2004b))

New studies have appeared in the meantime, which are included in the present updated review (Babisch et al. 2005; Belojevic and Saric-Tanaskovic 2002; Bluhm et al. 2001; Evans et al. 2001; Franssen et al. 2004; Goto and Kaneko 2002; Lercher et al. 2002; Maschke 2003; Matsui et al. 2001; Matsui et al. 2004; Niemann and Maschke 2004; Rosenlund et al. 2001). Others are on their way or have not yet been finalized or fully published, e.g. the pan-European projects "Hyena" (Jarup et al. 2003) and "Ranch" (Haines et al. 2003; Kempen et al. 2003; Stansfeld et al. 2005).

4. Epidemiological studies

Sixty-one epidemiological studies were recognized as having either objectively or subjectively assessed the relationship between transportation noise and cardiovascular endpoints. The identification of studies was based on the author's expert-knowledge of the topic and the respective literature.

Table A1 lists epidemiological noise studies where cardiovascular effects were studied in relation to community noise levels, mainly road traffic noise and aircraft noise. Only one study refers to railway noise. The studies with their characteristics are given in chronological order and numbered **(# number)** for reference in the text and other tables. In *Table A1* the location (town and country where the study was carried out), the reference (first author and year of publication), the type of the study, the study subjects, sample size, exposure, outcome and control variables (covariates) are given. A classification of the statistical control of covariates in the analyses is given (0 = no control, 1 = group comparison, 2 = stratification/standardisation, 3 = model adjustment, 4 = matching). Also, an indication is given as to whether exposure and outcome were assessed on a subjective or objective basis ("S", "O").

All data presented in the *Tables in the Appendix* were obtained from the quoted literature, with the review laying no claim to completeness. In particular, full technical reports containing further information may not have been considered. In general, the scientific community is confronted with the problem of publication bias, which means that often studies with non-significant results remain unpublished. If not given in the references, adjusted estimates for the relative risk (odds ratio, risk ratio, proportional morbidity ratio) set out in the tables were recalculated for the purpose of this review on the basis of the data provided there in, with the least traffic noise exposed group of subjects as the reference group. If not explicitly given in the publication, test-based 95%-confidence intervals (Hennekens and Buring 1987) were estimated on the basis of the available information, if possible (software: Epi 6, Episheet, Depid).

In most of the *Tables in the Appendix* the results are grouped according to 5 dB(A)-categories for the daytime (L_{day}: 6-22 h) outdoor average A-weighted sound pressure level, which was considered in most studies. Information on night-time exposure (L_{night}: 22-6 hr or 23-7 hr) was seldom available. Newer studies used non-weighted or weighted averages of the 24 h exposure (L_{eq}, L_{dn}, L_{den}) (Directive 2002/49/EC 2002). Some aircraft noise studies used national calculation methods (e.g. Dutch Kosten Units). Sound levels were converted on the basis of best guess approximations to L_{day} (Bite and Bite 2004; Franssen et al. 2004; Matschat and Müller 1984; Passchier-Vermeer 1993). It should be noted in this context that decibel level

behave very robust to changes of traffic volume. Doubling/halving of road traffic volume results in a (only) 3 dB(A) higher/lower average sound pressure level. In the ambient environment common noise levels (L_{day}, L_{den}) range between approx. 45 and 75 dB(A). In urban settings, night-time average noise levels (22-6 h) for road traffic tend to be approx. 7-10 dB(A) lower than daytime average noise levels, relatively independent (no freeways) of the traffic volume of the street (Evans et al. 2001; Ullrich 1998; Utley 1985). 24h noise levels of road traffic are usually 1 to 3 dB(A) lower than daytime noise levels (Rylander et al. 1986). Such empirical factors are considered in calculations of weighted averages. According to the European directive on the assessment and management of environmental noise, penalties of 5 dB(A) and 10 dB(A) are considered for the evening period and the night period, respectively, for the calculation of the weighted noise indicator L_{den} (Directive 2002/49/EC 2002). Therefore, in epidemiological studies in which the relative effects of road traffic noise are studied, the sound emission during the daytime can as well be viewed as an approximate indicator of the sound exposure during the night (approx. 10 dB(A) lower), if no freeways are considered and where the day/night difference is less. Not all studies allow dose-response reflections because some of them considered very broad exposure categories.

Besides objective noise measurements, subjective measurements of exposure have been used in some epidemiological noise studies, which is in accordance with the noise-stress model. Type of road (e.g. busy street, side street etc.), disturbances and annoyance were rated by the study subjects from given scales. In the related following tables the results of these studies were grouped into four ordinal categories, depending on the items in the questionnaires: 1 = "never", "not at all", "dead end street" or "not affected"; 2 = "seldom", "a little" or "side street"; 3 = "sometimes", "moderate" or "busy road"; 4 = "often + always", "much + very much", "strongly", "major trunk road" or "affected".

4.1 Mean blood pressure

Table A2 lists the major findings of epidemiological traffic noise studies in which mean blood pressure was considered as the outcome. It indicates mean systolic and diastolic blood pressure differences as obtained from extreme group comparisons of noise exposure. The effects in children and in adults are discussed separately.

4.1.1 Children

4.1.1.1 Aircraft noise

Very crude data regarding more blood pressure abnormalities in children living in the vicinity of Russian airports were reported in the late Sixties [#03]. No detailed information is available in the international literature.

The results from a cross-sectional study on schoolchildren from schools and homes around Los Angeles airport exposed to different levels of air traffic noise support this finding [#10]. In this study blood pressure differences of 3 to 7 mmHg were found between the groups, depending on the years enrolled in school. A decreasing trend was found with increasing years of enrolment; the overall difference between the groups was 3 mmHg for systolic and diastolic pressure. However, the results may be confounded by incomplete control of ethnicity (Morrell et al. 1998). The blood pressure measurements were taken under quiet conditions in the schools. The longitudinal approach of analysis (1 yr follow-up) failed to show a relationship between noise exposure at the schools and change in blood pressure of the schoolchildren probably due to selective migration of the children's families [#11].

A cross-sectional study carried out around the old Munich airport revealed 2 mmHg higher systolic blood pressure readings in schoolchildren from noise exposed areas ($L_{eq,\ 24hr}$ = 68 dB(A) as compared to unexposed ($L_{eq,\ 24hr}$ = 59 dBA) [#36]. This difference was borderline significant. No noise effect was found with regard to diastolic blood pressure. In a longitudinal approach, blood pressure readings were analysed in schoolchildren before and after the opening of the new Munich airport in a noise-impacted and an unaffected control area [#39]. In the noise-impacted communities the 24-hr average sound pressure level (L_{eq}) was 53 dB(A) before the opening as compared to 62 dB(A) after the start of operation of the airport. In the control area the before and after noise levels were 53 dB(A) and 55 dB(A), respectively. Children from the noisy area showed a 2 to 4 mmHg higher increase in blood pressure readings than their counterparts from the quiet areas. However, 18 months after the opening, no difference in blood pressure readings was found between the well-matched children from the both areas. The higher change in blood pressure was due to lower values at the beginning of the follow-up.

The cross-sectional comparison of systolic and diastolic blood pressure readings in primary schoolchildren living in the vicinity of the Sydney airport revealed non-significant regression coefficients for the relationship with aircraft noise (range: 15 to 45 ANEI (Australian Noise Energy Index) at school of r = -0.017 (systolic) and r = -0.043 (diastolic) [#40]. This corresponds

to mean blood pressure differences of -1 mmHg across the whole noise range. The aircraft noise level at home was also not associated with the blood pressure (r = -0.010 and r = +0.010), nor was the road/rail traffic noise level at school. The longitudinal results regarding the change of blood pressure over time did not show an association with the noise level [#41]. The elapsed time since a reduction of noise exposure due to the opening of a new runway, however, was significantly negative correlated with diastolic blood pressure [#40]. This was interpreted as responses to changes in aircraft noise level being reversible over time.

Studies were carried out in Germany, regarding noise from low-flying military aircrafts. At that time particular areas were identified for pilot training. A pre-study revealed higher readings in children of up to 9 mmHg in systolic blood pressure, particularly, in extreme low-flying areas (75m) where sound levels were raised to L_{max} = 125 dB(A) [#21]. The effect was found in girls, but not in boys. However, these findings were not confirmed in the main study [#22] and another area [#20], where mock attack areas were largely excluded. It is reasonable to assume that the combination of noise and fear was the driving force. Other studies on low-altitude jet noise also did not show higher blood pressure readings in children [#26].

A very speculative interpretation was given with respect to a study that compared the blood pressure of deaf-mute children and children with normal hearing [#31]. The deaf-mute group had lower blood pressure readings, which was discussed with respect to the perception of the acoustic environment. However, the effect diminished with increasing age of the children.

4.1.1.2 Road traffic noise

In an early study with schoolchildren, from schools in the German town of Halle, exposed to different levels of road traffic noise, blood pressure readings were more than 10 mmHg higher in the group with the highest exposure [#01]. Blood pressure was probably measured under acute noise conditions in the classrooms. A exposure-effect relationship was found. Confounding factors such as social class were not assessed, but children with clinical manifestations of blood pressure related diseases were excluded from the analysis.

In the Tyrol study, children from 7 villages exposed to road traffic noise from transit routes were compared with children from 6 control villages with low traffic [#29]. Slightly lower, non-significant, mean blood pressure readings were found in the exposed group. Another study carried out years later in the same region in the Inn Valley revealed only a marginal and borderline significant higher systolic blood pressure in children, who were exposed to high noise levels (L_{dn} > 60 dB(A)) from road and railway noise, compared to less exposed children [#53].

In the city of Bratislava pre-school children attending kindergartens in different road traffic noise exposed districts were examined [#37]. Blood pressure measurements were taken in the kindergartens. Children from homes and/or kindergartens exposed to more traffic noise (≥70 dB(A)) showed systolic and diastolic blood pressure readings 2 to 5 mmHg higher than those from less exposed areas (≤60 dB(A)). This was statistically significant. Noise at the kindergartens had a higher impact on the blood pressure than the noise at home. A dose response relationship was found.

4.1.1.3 Discussion

The findings in children are difficult to interpret with regard to possible health risks in their later life. The effect may be of a temporary nature and may not be relevant to permanent health damage. There is evidence during childhood (Gillman et al. 1992), adolescence (Yong et al. 1993) and adulthood (Tate et al. 1995) that the blood pressure level at an early age is an important predictor of the blood pressure level at a later age. Studies over the full age range are missing (tracking). Growth and body weight are important factors for blood pressure development. The impact of body size was not adequately considered in some of the studies. A crude hint regarding reversible effects on blood pressure came from one study (Morrell et al. 2000). Results of the Munich intervention study on the effects of a reduction of aircraft noise have only been reported regarding cognitive performance but not with respect to change of blood pressure (Hygge et al. 2002). It was concluded from the available data on the length of exposure that children do not seem to adapt to high levels of road traffic noise but to some extent to aircraft noise (Bistrup et al. 2001; Passchier-Vermeer 2000). However, the data base appears to be too poor to draw final conclusions. Aircraft noise studies focussed on the exposure at school, while road traffic noise studies mostly considered the noise exposure at home. Different mechanisms (disturbed learning/concentration vs. disturbed relaxation/sleep) may be involved.

The conclusions given by Evans and Lepore seem still to hold true (Evans and Lepore 1993): “We know essentially nothing about the long-term consequences of early noise exposure on developing cardiovascular systems. The degree of blood pressure elevations is small. The clinical significance of such changes in childhood blood pressure is difficult to determine. The ranges of blood pressure among noise-exposed children are within the normal levels and do not suggest hypertension. The extent of BP elevations found from chronic exposure are probably not significant for children during their youth, but could portend elevations later in life that might be health damaging."

4.1.2 Adults

4.1.2.1 Aircraft noise

In the Munich aircraft noise study around the old Munich airport [#04], men and women from the noisiest areas had the highest blood pressure readings with a mean difference of approx. 3 mmHg (diastolic) as compared to the least exposed group. There, a "u"-shaped association was found across noise categories. A Japanese study compared the blood pressure of females that lived in different aircraft noise zones of Fukuoka airport with a control group. In the cross-sectional part of the study, a 4 mmHg higher systolic blood pressure was found in the higher exposed group (L_{dn} ≥ 70 dB(A)) compared to the reference group (L_{dn} < 60 dB(A)) [#56]. This finding was statistically not significant. In the follow-up study no differences in the change of systolic blood pressure were found. The control group showed a 4 mmHg higher increase of diastolic blood pressure than the exposed group [#57]. Only crude information was given in the reference about the study design.

The effects of military low flying aircraft noise were studied in two regions in Germany [#24, #25]. Neither in the 150 m nor in the 75 m altitude flight areas for aircraft operation were higher mean blood pressure readings for the adult population found, compared with subjects from control areas.

4.1.2.2 Road traffic noise

A Dutch cross-sectional study looked at the association between road and military aircraft noise and blood pressure [#17]. No clear blood pressure pattern was observed. While there was a significant positive trend of an increase in systolic blood pressure of 0.12 mmHg per noise category (6 categories) for aircraft noise after adjustment for covariates, a non-significant inverse trend of -0.03 mmHg per category was found with regard to road traffic noise. The diastolic blood pressure showed similar but non-significant trends across noise categories. When the two highest, and the two lowest aircraft noise categories were combined (>50 KE versus ≤40 KE, KE = Dutch aircraft noise measure), mean group differences in systolic and diastolic blood pressure of 5 mmHg and 2 mmHg, respectively, were found for this extreme group comparison (due to curvi-linear association across categories). Subjects with prevalent hypertension due to renal disease or chronic diseases which can cause hypertension or influence IHD, such as diabetes mellitus, congenital heart disease, heart valve disease, were not included in the sample subjected to medical examinations. Furthermore, participants who were receiving medication or dietary treatment for hypertension were excluded from the statistical analyses, which suggests the possibility of over-controlling. This applies also to the

clinical blood pressure measurements of the Bonn road traffic noise study, which refers to normotensive subjects [#09]. No remarkable blood pressure differences were found between subjects from the high noise (L_{day} > 65 dB(A)) and the low noise area (L_{day} < 60 dB(A)). An attempt to conduct a prospective study failed due to a high and probably selective migration rate amongst the young subjects under study, particularly, in the noisy areas [#16].

A "u"-shaped relationship similar to the Munich aircraft noise study [#04] was found in the Caerphilly study, with a mean systolic blood pressure difference of only 1 mmHg between subjects of the extreme groups of road traffic noise exposure (66-70 dBA versus 51-55 dBA) [#27]. However, the twin study carried out in Speedwell revealed an inverse relationship - the subjects in the highest noise category showing the lowest blood pressure readings [#28]. In a sub-sample, an effect modifying impact of work noise exposure on systolic blood pressure was demonstrated (Babisch et al. 1990).

A Dutch study on road traffic noise carried out in Amsterdam revealed a trend towards lower blood pressure readings in subjects exposed to higher traffic noise levels [#14], as did an Austrian cross-sectional study carried out in five villages in the state of Tyrol [#30]. This was both across noise level categories and annoyance categories. These negative findings were significant. A later study carried out in the same region did not show an association with mean blood pressure readings with any of the various noise level indicators that were considered [#52]. However, distance to the highway and distance to the rail track (in the valley) were meaningful predictors of the blood pressure (higher readings in subjects that lived closer to the traffic artery). When the results were stratified with respect to annoyance ratings, only in the "not at all" annoyed, was there a tendency towards higher readings for subjects exposed to higher noise levels. In the extreme group comparison, the clinical data of the Luebeck blood pressure study showed an increase of 2 mmHg (diastolic) in readings in male subjects exposed to high road traffic noise levels (>65 dB(A)), but not in females [#15]. Across noise level categories a non-linear association was found. Significantly higher systolic and diastolic blood pressure readings were found for men in the intermediate noise category 61-65 dB(A) (+4/+2 mm Hg). When the subjective description of the type of road was used to classify exposure (given by the subjects in a questionnaire), the noise effect proved to be more pronounced.

4.1.2.3 Discussion

Regarding mean blood pressure, no consistent findings in the relationship between traffic noise level and mean systolic or diastolic blood pressure can be seen in adults across the studies. In longitudinal studies, problems arose from migration of subjects, which had a considerable impact on sample size. The latter problem also applies to cross-sectional studies in general.

Sensitive subjects may tend to move out of the polluted areas, which dilutes the effect of interest. Medication due to high blood pressure may affect the blood pressure readings. However, the exclusion of subjects with hypertension or hypertension treatment, dilutes the true effect on blood pressure differences, if the hypothesis (noise causes high blood pressure) is true. In principle, hypotension - a fall in blood pressure - can also be a stress reaction. All this makes it more reasonable to look at manifest hypertension (defined by a cut off criterion) as a clinical outcome rather than at mean blood pressure readings (Ising 1983; Winkleby et al. 1988). To date, there is no evidence from epidemiological data, that community noise increases mean blood pressure readings in the adult population. However, this does not discard the noise hypothesis as such. Studies suffered from insufficient power, narrow exposure range or other difficulties in the study design.

4.2 Hypertension

Table A3 shows the results of epidemiological traffic noise studies for the relationship between community noise level and the prevalence or incidence of hypertension. Hypertension in these studies was either defined by WHO criteria (Guideline Subcommittee 1999), or similar criteria based on measurements of systolic and diastolic blood pressure, or from information which was obtained from a clinical interview, or a social survey questionnaire about doctor diagnosed hypertension. Most studies refer to road traffic noise. However, in recent years some new aircraft noise studies have been put into the database. The subjects studied were the adult male and female population, sometimes restricted to certain age groups.

4.2.1 Aircraft noise

An early and often cited study is not considered in *Table A3* because no detailed information regarding study design was given in the reference [#02]. There it is reported that adult subjects who lived near to an airport showed 2-4 times higher prevalence rates of cardiovascular (hypertension, hypotension, etc.) and other diseases, than those subjects who lived further away. In children, higher rates of blood pressure abnormalities and autonomic vascular changes were found [#02].

The well-known cross-sectional study carried out in the vicinity of the Amsterdam airport in the Seventies (response rate 42%) suggests relative risks of 1.5 (clinical interview) and 1.7 (blood pressure measurement), respectively, for noise levels of KE > 40 (Dutch “Kosten units”) compared with subjects who lived in areas where the noise levels were lower [#05]. The data were analysed dichotomously, because the noise data showed a clustered pattern (due to the

selection of communities). However, the study was re-analysed using a continuous logistic regression approach, resulting in a relative risk for hypertension of 1.26 (95% CI: 1.14-1.39) per 5 dB(A) increase in noise level, within the measurement range from approx. $L_{Aeq,\ 7\text{-}19h}$ = 55-72 dB(A) (Franssen et al. 2002; Kempen et al. 2002).

The analyses of health registration data with respect to the spatial distribution of the hospital admissions due to cardiovascular diseases (amongst which was hypertension), from 62 municipalities around Schiphol airport did not show a specific pattern of clustering in areas close to the airport [#48]. However, high blood pressure is not a particular reason for hospital admission. It is mostly treated by local general practitioners. A feasibility study was carried out around the Paris Roissy airport using the approach of a practice-based survey [#45]. The diagnoses of 7 doctors' practices from high and low aircraft noise exposed areas were analysed with respect to their patient's contacts over a week. No higher blood pressure was found in subjects exposed to high aircraft noise compared with less exposed subjects. However, subjects could have gone to other doctors outside the study area and vice versa. This problem of an unknown population at risk in practice-based epidemiology (e.g. sentinel practice systems) has been previously discussed in the literature (Schlaud et al. 1998).

The clinical examination of inhabitants (no response rate given) around a military air base on the island of Okinawa revealed a significantly higher prevalence (RR = 1.4) of hypertension in the group exposed to $L_{dn} \geq 70$ dB(A) [#49]. A study (postal questionnaire survey) carried out in Sweden around Stockholm's airport (response rate > 70%) showed a exposure-effect relationship with an increasing risk of hypertension starting at rather low ambient noise levels around FBN = 55 dB(A) (the Swedish weighted noise calculation method). For subjects exposed to noise levels >55 dB(A), a relative risk of 1.6 was found, which was significant [#50]. The preliminary results of another study carried out around this airport also give some first indications of a higher risk for aircraft noise exposed subjects (FBM > 55 dB(A)) of 1.6 [#60]. In the road traffic noise study carried out in the Berlin district of Spandau (response rate > 80%), aircraft noise was also assessed [#58]. The exposure assignment was based on old prognostic noise contours, which implies that there would be a problem of exposure misclassification. A steady increase in risk was found with increasing noise exposure. In the highest noise zone (according to the German Aircraft noise Act) of $L_{eq(4)}$ = 67-75 dB(A) the period prevalence (during the past 2 years) was 1.5. However, due to the small number of exposed subjects in the sample the confidence intervals were large. Since the subjects were taken from an ongoing health surveillance survey where subjects have voluntarily assigned themselves, the sample is then a highly selected one. Participating subjects could have a particular interest in a regular (free) clinical health check (subjects with health problems or health-aware subjects).

A telephone survey in Northern Germany [#23], as well as the clinical examinations [#24, #25] carried out on adults in different communities of military low-altitude flight zones in Germany, did not reveal any differences in the prevalence of high blood pressure (response rate 56%). The studies are not considered in *Table A3* because single event noise levels rather than average sound pressure levels were given. The clinical examinations carried out in Muensterland [#24] suffer from a very low response rate (6%). Non-significant prevalence ratios of 1.0 and 0.9 for clinically examined prevalence of hypertension were found in males and females respectively, for exposed areas compared to less exposed. The subjects were recruited from those participating in the telephone survey [#23]. The objective prevalence of hypertension was higher than the subjective prevalence of hypertension. The other study carried out in Franken (response rate 49%) revealed non-significant prevalence ratios of less than 1.0 in exposed subjects [#25].

4.2.2 Road traffic noise

The German road traffic noise study (response rate 60%) carried out in Bonn [#09] suggested a relative risk for hypertension of 1.5 for subjects who lived in areas where the traffic noise level exceeded L_{day} = 65 dB(A)). This finding was significant.

The study carried out in Erfurt [#12] is difficult to interpret. It appears to be a retrospective cohort study where disease frequencies in differently exposed groups (contact rates of patients with two medical centres) during the same period of time (1 year) were collected on an individual basis, but the data were analysed in terms of a proportional morbidity ratio. This means that the significantly higher risk of hypertension treatment in the exposed group may either be due to a higher incidence of hypertension (nominator) or to a lower incidence of treatment for other diseases (denominator) in the exposed group. A significant relative risk of 2.4 was found for subjects exposed to L_{day} = 75 dB(AI) compared to subjects that lived in a street where the noise level was L_{day} = 67 dB(AI). Even the control group was highly noise exposed.

The study carried out in Doetinchem [#08], and later studies carried out in the Eighties and early Nineties in Amsterdam [#14], Luebeck [#15], Berlin [#34] and Tyrol [#30] may be of higher validity as far as statistical control of possible confounding is concerned. They do not support the noise hypothesis, showing relative risks of between 0.5 and 1.0 for the group comparisons with regard to the road traffic noise level. The response rates obtained in these studies were approx. 74%, 70%, 75%, 64% and 62%, respectively. Also the results of another study that was carried out in the Inn Valley, with respect to road and railway noise (response rate: 51%) did not fall in the hypothesised direction [#52]. In the cross-sectional part of a before-after study carried

out in a village near Erfurt [#18], a significant relative risk of 2.4 was found for the period prevalence of hypertension in subjects that lived in a street where the noise level exceeded L_{day} = 75 dB(AI). The prevalence ratios were probably calculated as proportional morbidity ratios. The selection criteria of exposed and unexposed subjects are not clear and the possible impact of confounding factors remains unclear. The longitudinal approach of the study was concerned with the health benefit of a 10 dB(A) reduction in noise level in the exposed streets. Five years after this intervention, the recovery rate of patients with hypertension was markedly higher in the area previously subject to higher traffic noise levels than that of the control subjects [#19]. This suggests that primary essential hypertension due to stress-induced vasoconstrictive and cardiac mechanisms may have been more prevalent in the exposed group than in the control group before the intervention.

The picture changes a little, when new studies from more recent years are considered. While a Japanese study carried out in Tokyo also showed a negative finding (no association) with respect to prevalence of hypertension as assessed in a questionnaire survey [#38], two Swedish and one German study revealed significant results pointing in the direction of a higher risk in higher exposed subjects. As with the Swedish aircraft noise studies, higher risks were found at relatively low road traffic noise levels, $L_{eq,24hr}$ > 50 dB(A). Using geographical information about distances of houses from main roads and railway lines, the association between noise from road traffic and railway traffic and the prevalence of hypertension was studied in the Swedish town of Sollentuna [#46]. Medical diagnosis of hypertension was assessed with a self-administered questionnaire. The noise levels in the road traffic noise exposed group ranged from $L_{eq,24hr}$ 40 to 65 dB(A) and those for train noise from $L_{eq,24hr}$ 55 to 65 dBA. Response rates of approx. 76% were achieved. After adjustment for confounding factors, a significant relative risk of 1.8 for the total group was found in the road traffic noise exposed group when comparing groups exposed to $L_{eq,24hr}$ >50 dB(A) with $L_{eq,24hr}$ <50 dB(A). The effect was only seen in women though (relative risk of 3.3) and not in men (relative risk 1.0). A possible explanation could be that women spend more time at home. Regarding train noise, the opposite association was found. The subjects in the exposed area were at lower risk of hypertension than those in the control area. The relative risk of 0.8 was not significant. In contradiction to this, the prevalence of annoyance and sleep disturbance due to noise was highest within the railway noise exposed group as compared to the other groups. A re-analysis of the road noise sample using more definite exclusion criteria found a non-significant relative risk of 1.5 (men 1.4, women 1.8) in the total sample [#47]. In the sub-sample of subjects with at least 10 years in residence, the relative risk of 2.4 was larger and significant.

The Spandau Health Survey was already discussed with respect to aircraft noise. Its major emphasis was though on road traffic noise [#58]. The period prevalence (and the lifetime

prevalence) increased steadily with the road traffic noise level in the noise level range from L_{day} <55 to 70 dB(A) and L_{night} <50 to 65 dB(A). The relative risks were 1.5 (L_{day} >65 dB(A)) and 1.9 (L_{night} >55 dB(A)) depending on whether the exposure during the day of the living room or during the night of the bedroom was considered. The latter was significant. When subjects were analysed separately, for those who used to sleep with an open bedroom window, the relative risk was greater. However, due to a small sample size, this risk estimate cannot be interpreted in absolute terms (large confidence interval).

4.2.3 Annoyance

Table A4 shows the results of studies on the relationship between subjective ratings of traffic noise exposure and prevalence of hypertension. The cross-sectional studies from Amsterdam [#14] and Tyrol [#39] gave no indication of an increased risk of hypertension in subjects more annoyed/disturbed by traffic noise as compared to those less annoyed/disturbed. Based on prevalence of hypertension as reported on a self-administered questionnaire, a significant relative risk of 1.3 was found in subjects disturbed by heavy road traffic noise, in a cross-sectional study carried out in Berlin [#34]. Since exposure and disease were assessed on a subjective basis, these results are susceptible to recall bias due to over-reporting. This reservation is true for all cross-sectional studies where exposure and disease are assessed subjectively, and applies also to the prospective study carried out on a random sample of the German population [#35]. Although designed as a general population follow-up study on the incidence of various diseases in a pre-defined disease-free cohort, disturbance due to noise at home (presumable mainly traffic noise) and incidence of disease were assessed at the same time by questionnaire (during follow-up). With regard to noise at home, the study, therefore, must be viewed as cross-sectional (response rate approx. 79%). A relative risk of 0.9 (males: 1.2, females: 0.9) was found with regard to global disturbances ("affected" by traffic noise). However, a relative risk of hypertension of 2.3 was found with regard to reported sleep disturbances, which was significant.

In the Luebeck study [#15], a borderline significant relative risk of 1.3 was found in male subjects who described the street in which they lived as busy, as compared to those who described their residential streets as quiet. A exposure-effect relationship was found in the cross-sectional study carried out in Pancevo, Serbia (response rates 77% and 92% in non-exposed and exposed areas, respectively). Across annoyance categories a steady increase in risk of self-reported hypertension was found [#54]. The estimate of the relative risk of 1.8 for the highly annoyed subjects was significant. In the Spandau Health Survey no significantly higher risks were found in subjects that where annoyed by the noise [#58]. However, the relative risks of 1.2 (road traffic noise) and 1.3 (aircraft noise) were slightly higher for the

annoyance/disturbance during the night than the annoyance during the day (relative risks 1.0 and 1.2).

Results from the LARES study, which is a questionnaire survey that was carried out in 8 European cities using identical methods, showed in noise annoyed subjects a higher morbidity with respect to various self-reported health outcomes (as diagnosed by a doctor) than in not annoyed subjects [#62]. Amongst these was hypertension, which was significantly more prevalent in subjects strongly annoyed by general traffic noise (relative risk 1.6) and general neighbourhood noise (relative risk 1.7). Sleep disturbed subjects showed a similar relative risk of 1.5. The effects were not found in the elderly population (60 years and older).

4.2.4 Discussion

With regard to the association between community noise and hypertension, the picture is heterogeneous. With respect to aircraft noise and hypertension, studies consistently show higher risks in higher exposed areas. The evidence has improved since a previous review (Babisch 2000). The relative risks found in four studies showing significantly positive associations range 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)).

With respect to road traffic noise, the picture remains 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. Exposure-effect relationships were considered in new studies. Subjective ratings of noise or disturbances due to traffic noise seem to consistently show a positive association with prevalence of hypertension. The relative risks found here range from 0.8 to 2.3. These studies, however, are of lower validity due principally to methodological issues regarding over-reporting (Babisch et al. 2003b).

4.3 Ischaemic heart disease

Table A5 gives the results of cross-sectional epidemiological traffic noise studies on the relationship between noise level and prevalence of ischaemic heart disease (IHD); *Table A6* gives the results of case-control and cohort studies on the association between noise level and incidence of IHD. In cross-sectional studies, IHD prevalence was assessed by clinical symptoms of angina pectoris, myocardial infarction (MI), ECG abnormalities as defined by WHO criteria (Rose and Blackburn 1968), or from self-reported questionnaires regarding doctor-diagnosed heart attack. In longitudinal studies, IHD incidence was assessed by clinical myocardial infarction as obtained from hospital records, ECG measurements or clinical interviews. The majority of studies refer to road traffic noise.

4.3.1 Aircraft noise

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 [#48].

A lot of information came from the Amsterdam aircraft noise studies that were carried out in the 1980's [#05, #06]. 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" [#05] was approx. 42%. The "general practice survey" [#06] 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 [#17], 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.

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 $L_{eq(4)}$ > 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).

4.3.2 Road traffic noise

The non-significant results of the cross-sectional road traffic noise studies carried out in Bonn [#09], Caerphilly [#27], Speedwell [#28] and Berlin [#33], with response rates of approx. 60%, 89%, 92%, and 64%, consistently suggest relative IHD risks between 1.1 and 1.4 for outdoor noise levels of L_{day} >65 to 70 dB(A). The result of the Bonn study was not controlled for confounding factors because IHD was not the major interest. A very high significant relative risk of 4.9 was found in a study carried out in Tokyo, with respect to subjectively reported heart disease [#38]. However, the confidence intervals were also large due to the small sample size (response rate probably 93%). L_{24hr} 65 dB(A) was identified as a critical noise level above which the prevalence of ill health increased markedly. The Spandau Health Survey also revealed relatively high relative risks greater than 3 for road traffic noise levels L_{day} >60 dB(A) und L_{night} >50 dB(A), which were not significant [#58]. Again, the confidence intervals were large due to small numbers, which makes it difficult to interpret the data with respect to a exposure-effect relationship.

A study carried out in Tyrol [#30], revealed a significant relative risk of 2.1 with regard to angina pectoris, for subjects from areas of more than 60 dB(A), while a non-significant relationship - relative risk 0.8 - was found with regard to myocardial infarction. The response rate here was approx. 62%. The results of a Dutch study carried out in Doetinchem (response rate 74%) were also inconclusive and non-significant: a very small increase in risk at noise levels L_{day} > 65 dB(A) when clinical signs of ECG abnormalities were considered (relative risk 1.1), but a lower relative risk of 0.7 when angina pectoris was considered [#08]. No noise level related increase in IHD risk, as defined by the clinical interview and the ECG, was found in the study carried out

in the four Dutch cities of Groningen, Twenthe, Leeuwarden and Amsterdam [#17] regarding road traffic noise.

Table A6 gives the results of epidemiological traffic noise studies, about the relationship between noise level and incidence of IHD. All these studies are concerned with road traffic noise. A high and significant proportional morbidity ratio of 4.4 was derived from the retrospective study carried out in Erfurt for subjects exposed to L_{day} 75 dB(AI) compared to subjects that lived in a street where the noise level was L_{day} 67 dB(AI) [#12]. Some methodological issues concerning the validity of the results were raised earlier. The other studies are prospective ones. In the Berlin hospital- and population-based case-control studies (pre- and main study), non-significant relative risks of 1.2-1.3 were observed for men where the outdoor noise levels were higher than 70 dB(A) for L_{day}, suggesting a threshold at about 70 dB(A) [#32, #33]. Response rates for cases/controls were approx. 90%/90% and 90%/64%, respectively. The risk increased in the main study, when only subjects were considered that had lived for at least 15 years in their residence. While the pre-study suffers from small numbers, the main study refers to a large sample size. In the 10-year follow-up cohort studies in Caerphilly and Speedwell (response rates > 90%), no noise effects were detected with regard to the (address-related) outdoor traffic noise level [#42, #43]. However, the 6-year follow-up analyses of the pooled reconstructed cohort (first follow-up survivors plus newly recruited subjects, response rate approx. 90%), in which exposure assessment accounted for residence time, room orientation and window opening habits, revealed non-significant relative risks of between 1.2 and 1.6 for subjects in the highest L_{day} 66-70 dBA category compared to the lowest (51-55 dB(A)) [#44]. Furthermore, only in this highest noise category was a positive relationship between IHD risk and years in residence found, showing relative risks of between 1.01 and 1.02 per year.

A similar approach for a hospital-based case-control study was carried out 10 years later in the “NaRoMI”-study [#61]. Males and females from the entire city of Berlin (including former Eastern political part) were considered (response rate: 86%). No higher risk was found in traffic noise exposed women. However, the earlier findings in men were confirmed [#33]. A exposure-effect relationship was found over the range from L_{day} <60 dB(A) to 75 dB(A). The relative risk was 1.3 for subjects in the highest noise category (>70 dB(A)) and increased to 1.8 when subjects were considered that had lived at least for 10 years at their residence.

Indirect support for the noise hypothesis comes from a large cohort study, which was originally not designed as a noise study, but for studying the effects of air pollution. The study considered all-cause mortality and specific mortality, including cardiopulmonary causes over a follow-up period of 8 years [#55]. After adjustment for confounders, the association between air pollutants

decreased and was not significant while the association between living near a major road (within 50 m of a major urban road or within 100 m of a freeway) and all cause mortality increased, and was significant. When indicator variables of air pollution and distance to major road were treated simultaneously in the model, the effect estimates for the single pollutant models decreased substantially, while distance to major road showed a strong and significant association (relative risk = 1.95 (1.09-3.51)). The authors concluded that unmeasured confounders were to some extent responsible for the association. It appears to be reasonable that road traffic noise could be an "unknown" confounder. This interpretation is further supported by the fact that non-cardiopulmonary death and death due to lung cancer were not associated with any of the air pollution variables in the study.

The approach of a time-series study is often applied in air pollution epidemiology to investigate the acute effects of changes in air pollutants. In a time-series study carried out in Madrid [#51], significantly higher rates of emergency admissions to a major hospital were found for all causes, circulatory and to a lesser extent for respiratory causes on days with higher background noise levels after controlling for the effect air pollutants. The variation of noise levels was small (L_{10}-$L_{90} \approx 4$ dB(A)) as one would expect from experience in noise measurement. An increase of 1 dB(A) was approximately equivalent to an increase of 25 µg/m^3 of air pollutants for the relative risk. The findings are difficult to interpret. Although acute and temporary autonomic responses to noise were frequently found in laboratory studies, the long-term and severe effects of chronic noise exposure – according to the noise hypothesis - are related to the development of cardiovascular disorders in the long run. Residual confounding can be an explanation for these acute effects associated with changes in noise.

4.3.3 Annoyance

Table A7 gives results of studies on the relationship between subjective ratings of road traffic noise exposure and prevalence or incidence of ischaemic heart diseases. The cross-sectional studies from Tyrol [#30], Berlin [#34], Pancevo [#54] and the noise related analyses carried out as part of a general population follow-up study of two random German population samples [#35], revealed relative risks of between 0.8 and 1.9 in subjects highly annoyed/disturbed or subjectively "affected" by traffic noise, in comparison with subjects who were less annoyed/disturbed/affected. Response rates were approx. 62%, 64%, 79% and 86%, respectively, in these studies (#35: of those who participated in a previous survey). The significant effect in the Pancevo study was only found for men (relative risk: 1.7) not for women.

The results of the LARES study carried out in 9 European cities, showed in noise annoyed subjects higher risks of heart attack than in non-annoyed subjects [#62]. The relative risks for

strongly annoyed of 1.4 (general traffic noise) and 2.0 (general neighbourhood noise) were not significant. In the Spandau Health Survey the numbers were too small for a reliable analysis of data [#58]. However, there was a tendency that relative risks of angina pectoris for highly annoyed subjects were higher with respect to the annoyance during the day than the night. This applies to annoyance due to road traffic noise as well as aircraft noise.

The prospective studies carried out in Caerphilly and Speedwell [#42, #43, #44] revealed pooled relative risks of IHD of between 1.0 and 1.4 only in subjects of the highest annoyance/disturbance category considered. A strong effect-modifying impact of pre-existing diseases on the relationship was found in the Caerphilly and Speedwell study. Relative risks were higher in healthy subjects, ranging from 1.7 to 2.7, but not in subjects with prevalent chronic diseases. This was discussed with respect to recall bias. The new case-control study carried out in Berlin ("NaRoMI"-study) revealed a significant relative risk (odds ratio) of 1.10 per category on a 5-point noise annoyance scale, with respect to annoyance due to road traffic noise during the night in males [#61]. This corresponds with a calculated risk of 1.3 for highly annoyed subjects. In females no such association was found. However, annoyance due to aircraft noise during the night was significantly associated with a higher MI risk in females (relative risk 2.1), which was not found in males. Annoyance due to noise during the day was not associated with MI risk.

4.3.4 Discussion

With regard to ischaemic heart disease (IHD), the evidence of an association between community noise and IHD risk has increased since a previous review (Babisch 2000). There is 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 reflections on exposure-effect relationships. These mostly prospective studies suggest an increase in IHD risk for noise levels above 65-70 dB(A), the relative risks ranging from 1.1 to 1.5 when the higher exposure categories were grouped together. Noise effects were larger when mediating factors like residence time, room orientation and window opening habits were considered in the analyses. This accounts for long induction periods (McCarron and Smith 2005; Rose 2005) and improves exposure assessment. The results appear as consistent when subjective responses of disturbances and annoyance are considered, showing relative risks ranging from 0.8 to 2.7 in highly annoyed/disturbed/affected subjects. However, these findings may be of lower validity due to methodological issues.

4.4 Medication and drug consumption

Table A8 gives the results of studies on the relationship between drug consumption and community noise. Medication was primarily investigated with respect to aircraft noise. A significant prevalence ratio for cardiovascular medication of 1.4 was found in the sample of the Amsterdam airport [#05]. The results of the "drug survey", where the annual data of the pharmacies regarding the purchase of cardio-vascular drugs were analysed (repeated cross-sectional survey) supported this finding. An increase in drug purchase with time was found in the exposed areas and not in the less exposed. This refers to the purchase of cardiovascular and antihypertensive drugs, as well as the purchase of hypnotics, sedatives and antacids. Furthermore a dependency with changes in night-flight regulations was found (decrease after reduction of night-flights). A large recent study around Amsterdam airport found only a slightly higher risk of self-reported medication with cardiovascular drugs, including antihypertensive drugs, (relative risk 1.2) in aircraft noise exposed subjects where the noise level L_{den} exceeded 50 dB(A) [#59]. Exposure-effect relationships across noise levels (L_{den} = <50 to 65 dB(A)) with respect to prescribed and non-prescribed sedatives/sleeping pills were found (relative risk 1.5 and 2.0, respectively) in the highest noise category of L_{den} = 61-65 dB(A). The preliminary results of an ongoing aircraft noise study from Sweden carried out around Stockholm's airport are in line with the Dutch studies [#60]. A significant relative risk of 1.6 for the use of antihypertensive drugs was found in male subjects, where the noise level according to the Swedish calculation standard exceeded FBM = 55 dB(A).

The road traffic noise studies, where medication/purchase of drugs was investigated also tend to show a higher use in higher exposed subjects [#09, #13, #30]. The relative risk for cardiovascular drugs was 1.3 in the Bonn study [#09] and 5.0 in the Erfurt study [#13]. The results for other drugs including sleeping pills, sedatives, tranquillizer and hypnotics ranged between 1.2 and 3.8 in these studies.

All in all, the studies on the relationship between the use of medication or purchase of drugs and community noise support the general hypothesis of an increase in sleep disturbance and cardiovascular risk in noise-exposed subjects.

5. Evaluation of individual studies

This section refers only to studies where the prevalence or the incidence of manifest cardiovascular diseases was considered as a potential health outcome of chronic exposure to environmental noise. The focus here is on a quantitative risk assessment with respect to manifest diseases. Furthermore, studies on the effects of low-altitude jet-fighter noise are also excluded, because this type of noise includes other dimensions of stress (e.g. fear). Thirty-seven studies had assessed the prevalence or incidence of manifest diseases, including hypertension and ischaemic heart diseases (angina pectoris, myocardial infarction, ECG abnormalities).

5.1 Criteria

Epidemiological reasoning is largely based on the magnitude of effect estimates, dose-response relationships, consistency of finding, biological plausibility of the effects and exclusion of possible bias (Hill 1965; Weed and Hursting 1998). The usefulness of the Hill criteria has been discussed critically (Morabia 1991; Rothman and Greenland 1998; Rothman and Greenland 2005; Thygesen et al. 2005; Weed 2000). However, internal (the role of chance) and external validity (absence of bias and confounding) are important issues in the evaluation of studies. Analytical studies (e. g. cohort or cases-control studies) are usually considered as having a higher validity and credibility than descriptive studies (e. g. cross-sectional or ecological studies) (Hennekens and Buring 1987), although many of the reservations about cross-sectional studies seem to be of minor importance when considering noise. For example, it does not appear to be very likely that diseased subjects tend to move differentially more often into exposed areas. Rather the opposite may be true, if noise stress is recognised as a potential cause of the individual's health problem. Thus, a cross-sectional study design may act conservatively on the results. The presence of a linear dose-response relationship is not a necessary criterion of causality. Non-linear relationships, including "u-" or "j-" shaped, saturation and threshold effects may reflect true associations (Calabrese and Baldwin 2003; Rockhill 2005). With respect to the derivation of guideline values in public health policy, the assessment of a exposure-effect relationship enables a quantitative risk assessment on the basis of continuous or semi-continuous (e.g. 5 dB(A) categories) exposure data. Dichotomous exposure data - on the other hand - that refer to a cut off criterion which splits the entire exposure range into two halves, can be used to evaluate the hypothesis of an association (qualitative interpretation), but not to make a quantitative assessment.

The objective or subjective assessment of exposure and/or health outcomes is an important issue when judging the validity of a study (Cartwright and Flindell 2000; Hatfield et al. 2001; Malmström et al. 1999). The objective prevalence of hypertension was found to be higher in a population sample than the subjective prevalence of hypertension (Schulte and Otten 1993b). In a telephone survey more than half of the hypertensives classified themselves as normotensive (sensitivity 40% for men and 46% for women) (Bowlin et al. 1993). In a representative health survey, the validity of the self-reported assessment of morbidity (subjective morbidity) was found to be "low" with respect to hypercholesterolaemia, "intermediate" with respect to angina pectoris, hypertension and stroke, and "high" with respect to myocardial infarction (Bormann et al. 1990). Myocardial infarction is a very definite and severe health outcome which subjects would clearly remember if they had experienced it. Its assessment by questionnaire tends to be more credible than that regarding hypertension. Test-retest reliability was found to be good with respect to "harder" outcomes, including high blood pressure and heart attack (Lipworth et al. 2001; Lundberg and Manderbacka 1996). Over-reporting, on the other hand, may be a source of potential bias, particularly, when both, exposure and outcome, are assessed on a subjective basis (Babisch et al. 2003b; Winkleby et al. 1988). The subjects may be more prone to blame their environment for their health problems, or may even tend to exaggerate adverse effects or exposure in order to influence noise policy. Therefore, a higher credibility and ranking was given to studies where exposure and outcome were assessed objectively (e.g. sound level versus subjective ratings, and measurement of blood pressure or a structured clinical interview versus self-reported hypertension in a self-administered questionnaire). This means that the sound level must have been measured or calculated on the basis of the traffic counts, and clinical interviews or measurements must have been carried out by medically trained personnel (no self-administered questionnaire data) to give a study a high ranking.

Studies categorised "0" or "1" (no control, group comparisons) in *Table A1*, regarding the assessment of confounding factors ("covariates") do not fulfil the criteria for an adequate treatment of confounding (stratification, model adjustment (regression), matching) in the analyses. Studies which have been adequately controlled for a reasonable set of confounding variables in the statistical analyses, besides age and sex, were given a high ranking.

5.2 Judgement

The judgement about the epidemiological studies was made with respect to the identification of good quality studies that can be feasibly considered for the derivation of guideline values. These studies can either be used for a statistical meta-analysis, for a combined interpretation (synthesis), or for individual interpretations. All of the studies listed in *Tables A3-A7* were

therefore evaluated with respect to the following criteria for the inclusion or exclusion in the synthesis process. Necessary criteria were: (1) peer-reviewed in the international literature, (2) reasonable control of possible confounding, (3) objective assessment of exposure and (4) objective assessment of outcome. Additional criteria for the ranking were: (5) type of study and (6) dose-response assessment.

All six criteria were fulfilled by the two prospective cohort studies carried out in Caerphilly and Speedwell [#42, #43, #44], the two prospective case-control studies carried out in the western part of Berlin ("Berlin I" and "Berlin II") [#32, #33], and the new prospective case-control study carried out in entire Berlin ("NaRoMI" = "Berlin III") [#61]. The studies refer to road traffic noise and the incidence of myocardial infarction. They were also the only ones considered in an earlier meta-analysis on this issue (Kempen et al. 2002), with the exception of the “NaRoMI” study, which was not available at that time. All these studies are observational analytic studies (Hennekens and Buring 1987).

If descriptive studies on individuals – namely cross-sectional studies - are allowed, another two studies from Caerphilly and Speedwell on the association between road traffic noise and the prevalence of ischaemic heart diseases (IHD), myocardial infarction (MI) and angina pectoris (AP) can be taken into account [#27, #28]. These studies were also considered in the meta-analysis by v. Kempen et al. (Kempen et al. 2002). However, the results of the Berlin study on MI prevalence [#33] - which was also considered in that meta-analysis - are not considered here, because the outcome was assessed subjectively with a self-administered questionnaire (an exclusion criterion). Regarding aircraft noise, the cross-sectional Okinawa study [#49] on the association between aircraft noise and hypertension fulfils the inclusion criteria.

For a quantitative assessment of the association between community noise and hypertension, only one study is available which fulfils the inclusion criteria. The cross-sectional study carried out around the Kadena airfield in Okinawa suggests a continuous increase in risk of hypertension with increasing aircraft noise level [#49]. However, only sparse information is given in the peer-reviewed reference. If studies are included which do not assess dose-response relationships but only compare dichotomous categories of exposure in the analyses, two more studies can be included on the list. The studies were carried out in the vicinity of the Amsterdam airport. They suggest a higher risk of cardiovascular diseases in general [#06], and – specifically - for hypertension and ischaemic heart diseases (angina pectoris, ECG-abnormalities, heart trouble) [#05] in subjects from areas exposed to high aircraft noise. These studies were considered in the meta-analysis by Kempen et al. (Kempen et al. 2002). However, they do not fulfil the strict criteria set here. In the peer-reviewed reference the focus was on the dichotomous analysis of the data [#05]. The selection of the study areas suggested clustered

data (high and low exposure). The general practice survey [#06] appears to be a survey of aggregated data with respect to contact rates, similar to the more recent study on hospital admission rates in 62 municipalities around Amsterdam's airport [#59]. No individual data for control of confounding were assessed in these studies. Both can be classified as ecological studies, which are more likely to raise hypotheses rather than to test them (Greenland 2001).

Finally, if the inclusion criteria are widened to include peer-reviewed studies that assessed exposure-effect relationships between objective indicators of exposure and the subjective (self-reported) prevalence of diseases, a further two studies can be considered. These are the cross-sectional study carried out in Stockholm regarding the association between aircraft noise and hypertension [#50], and the cross-sectional part of the study in Berlin regarding the association between road traffic noise and myocardial infarction [#33].

Figure 3 shows the results of the three aircraft noise studies carried out in Amsterdam, Okinawa and Stockholm [#05, #49, #50]. Approximate conversions for L_{day} from different noise indices were made (see section 4). The graph clearly indicates that the results are too heterogeneous to derive a pooled exposure-effect curve. However, all three studies show an increase in risk with increasing noise level (*Table A3*). The lower risks observed in the Okinawa study could be due to the fact that military aircrafts were not regularly operating during the night. In the highly exposed areas, average numbers of 1-3 flights/landings per night were calculated. For 2% of the days though, the flight activity was much higher (5-28 flights/landings per night), but for the majority of nights there was no/less flight noise (Matsui et al. 1998).

Studies that are not given a high ranking according to the above mentioned criteria, however, may serve as additional sources of information to support the evidence of the conclusions being made on the basis of this review. On the other hand, negative findings do not necessarily discount them.

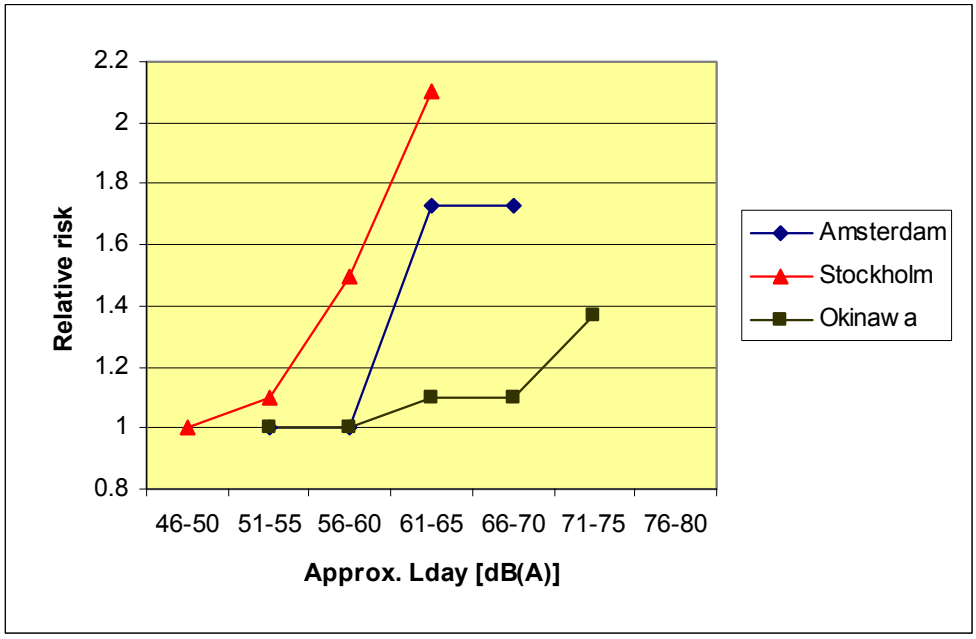


Figure 3. Association between aircraft noise level and the prevalence of hypertension

5.3 Exposure-effect relationship

In the previous section, the assessment of a exposure-effect relationship was an inclusion criterion for the consideration of a study for a meta-analysis. In this section, all studies that assessed a exposure-effect relationship are reviewed with respect to the actual presence of such a relationship. However, this evaluation is not based on statistical significance but on a rough estimation ("eye balling") of the effect estimates in different exposure categories and the consideration of the magnitude of the effect estimates and the confidence intervals. This process includes peer-reviewed studies and other studies.

Some of the studies suggest "u-shaped" or “j-shaped” curves of association [#06, #27, #28, #33]. Other studies suggest a continuous increase in risk with increasing noise exposure for noise levels [#38, #49, #50, #58, #61]. If only studies are included, that fulfil the inclusion criteria from section 5.2, then there is an indication of a threshold effect between 65 and 70 dB(A) from the older studies [#27, #28, #33]; whereas a new study suggests a continuous increase in risk with increasing noise level [#61].

5.4 Effect modification

5.4.1 Residence time

Support for any noise effect relationship may come from subgroup analyses that are in line with the noise hypothesis. This refers to effect modification with respect to residence time, window opening behaviour and other determinants that affect the noise exposure and cumulative noise dose.

In the Amsterdam aircraft noise studies, a steady increase in the purchase of cardiovascular and antihypertensive drugs at local pharmacies was found over the period of 8 years in a community newly exposed to aircraft noise. No such increase was found in a control community that was not exposed to aircraft noise. Positive associations between the prevalence of cardiovascular diseases and residence time in exposed areas (but not in unexposed) were also found in the road traffic noise studies carried out in Bonn with respect to hypertension [#09], and in Caerphilly and Speedwell with respect to the ischaemic heart disease [#44].

When the analyses of the road traffic noise studies carried out in Berlin, Caerphilly and Speedwell were restricted to subjects who had not moved within a retrospective period of 10 to 15 years, the effect estimates turned out to be larger than for the total samples of each study [#33, #44, #61]. This is illustrated in *Figures 4-6*. Similarly, when only subjects with long residence time were considered, a larger noise effect was found in the study in Sollentuna with respect to hypertension [#47]. No such an effect was found in the Luebeck study [#15].

The cross-sectional data of the study carried out in Los Angeles on children regarding mean blood pressure, indicated some habituation to aircraft noise [#10]. The longer the children were enrolled in the school, the smaller was the difference in blood pressure between exposed and non-exposed children. However, the follow-up study suggested that this may also be an effect of attrition [#11]. The longer the families experienced the noise, the more likely that they moved away from the exposed areas (selection bias). In general, effects on children due to noise exposure at school and effects on adults due to noise exposure at home reflect different kinds of disturbances (e.g. speech intelligibility vs. sleep). In contradiction to this, blood pressure differences between children exposed and not-exposed to road traffic noise increased with school-grade [#01].

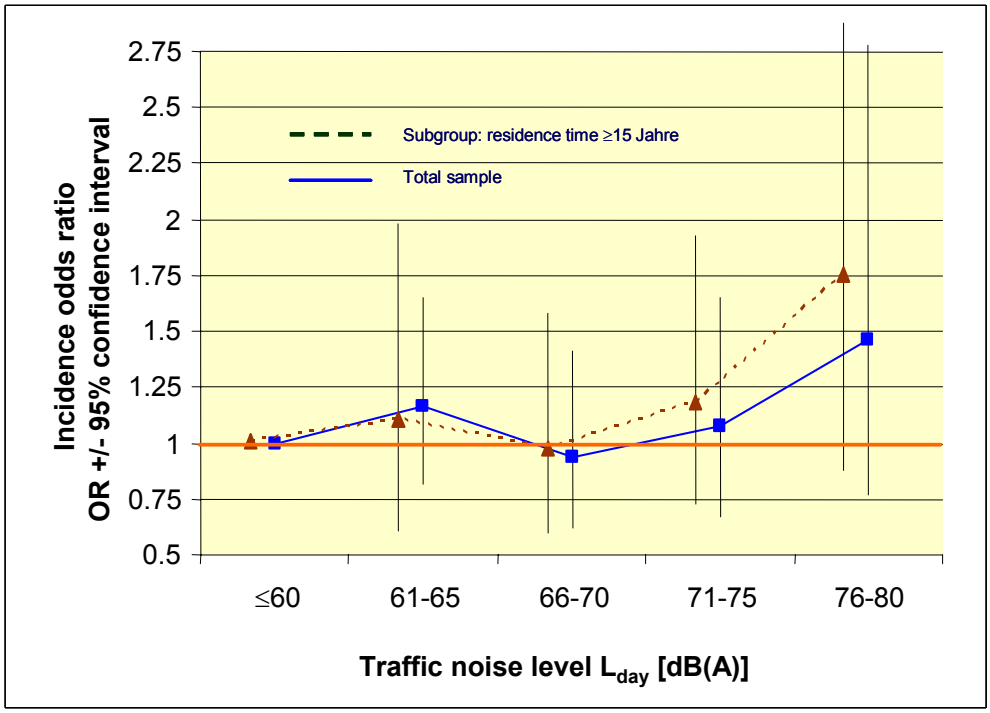


Figure 4. Berlin traffic noise studies (Babisch et al. 1999): Association between road traffic noise level and incidence of myocardial infarction. Sensitivity analyses: total sample vs. subgroup ≥15 yrs of residence time

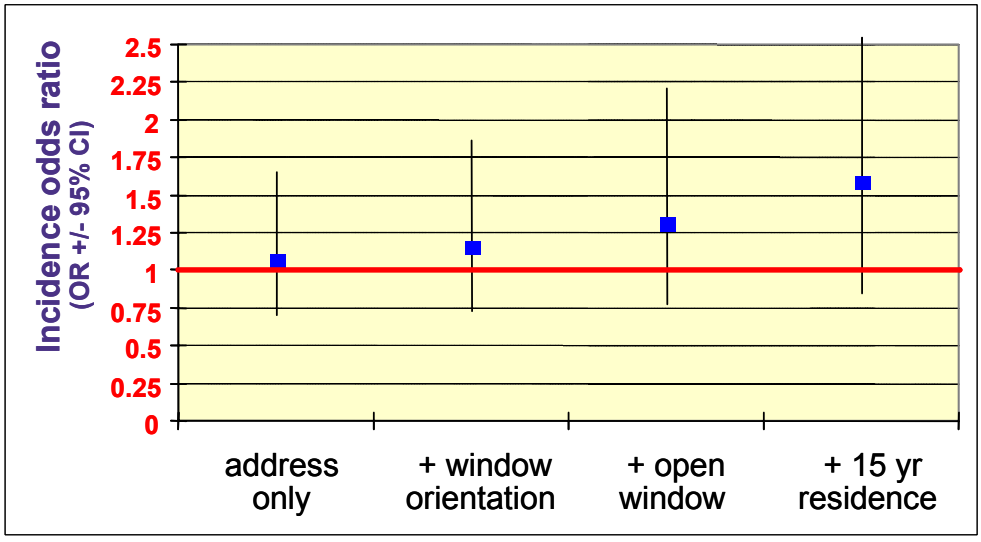


Figure 5. Caerphilly and Speedwell studies (Babisch et al. 1999): Association between road traffic noise level and incidence of major events of ischaemic heart disease (extreme group comparison: $L_{eq,day}$ = 66-70 vs. 51-55 dB(A)). Sensitivity analyses: total sample vs. subgroup windows facing the street, subgroup windows facing the street and windows open, subgroup windows facing the street and windows open and ≥15 yrs residence time

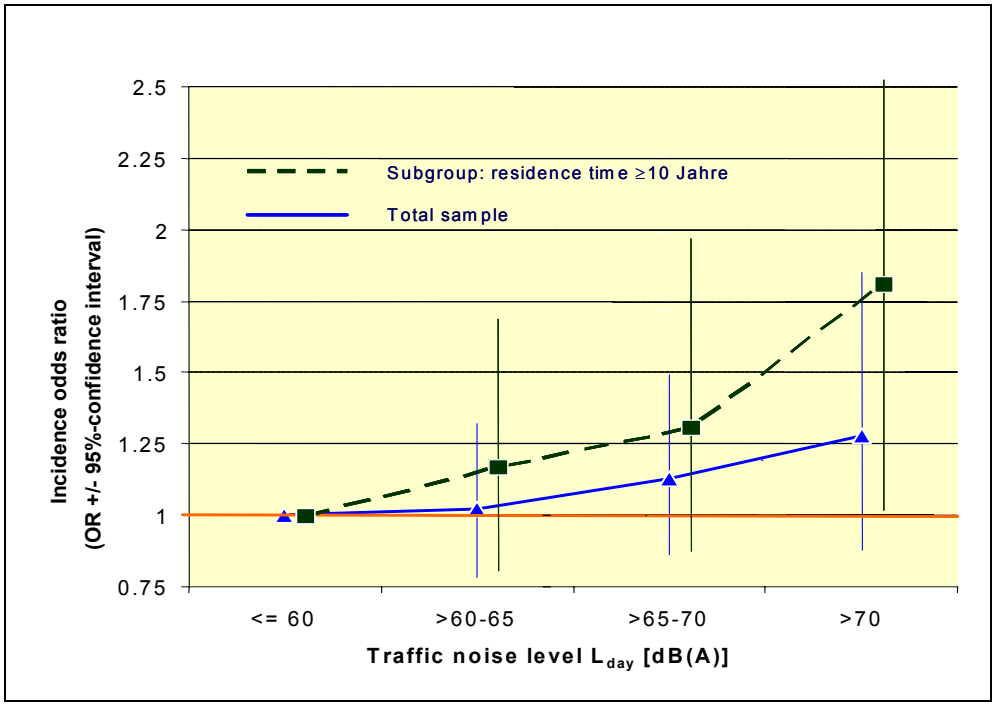


Figure 6. NaRoMI study (Babisch et al., 2005): Association between road traffic noise and incidence of myocardial infarction. Sensitivity analyses: total sample vs. subgroup ≥10 yrs of residence time

Intervention studies were conducted with respect to changes in blood pressure and changes in air traffic operation (e.g. opening/closing of airports or runways). In the Munich study, a larger increase in blood pressure was found in children from a noisy area [#39]. Other Studies suggested reversible effects on blood pressure when the exposure was lowered [#40, #19].

5.4.2 Room orientation and window opening

In the Tyrol study, significantly lower blood pressure readings were found in subjects who kept the windows closed throughout the night [#30]. When the subjects lived close to the highway (within a distance of approx. 500 m), the prevalence of hypertension was higher in subjects whose bedroom was facing the main road than in those, whose bedroom was not facing the main road. The orientation of rooms and window opening was also found to be an effect modifier of the association between road traffic noise and ischaemic heart disease in the Caerphilly and Speedwell studies [#44]. The relative risk with respect to the noise level was slightly higher in subjects with rooms facing the street and subjects keeping the windows usually open when spending time in the room (see *Figure 4*). A much greater relative risk of hypertension was found in subjects who slept with open bedroom windows in the Spandau Health Survey [#58].

5.4.3 Other modifiers

Hearing impairment was found to be an effect modifier on the association between aircraft noise and hypertension [#50]. Amongst the exposed subjects, a higher risk associated with the noise was only found in subjects without hearing loss.

5.5 Exposure during the night

Epidemiological noise research provides hardly any information regarding the particular impact of noise exposure during the night on cardiovascular health outcomes.

The Spandau Health Survey explicitly distinguished between the exposure of the living room (during the day) and the exposure of the bedroom (during the night). There, a slightly higher relative risk of hypertension was found with respect to the traffic noise level during the night (relative risk 1.9 vs. 1.5) compared with the noise level during the day [#58]. Furthermore, sleeping with open bedroom windows was associated with a large increase in risk. However due to the small sample size, the confidence intervals were very large.

In the drug survey of the Amsterdam aircraft noise studies, a steady increase in purchase of hypnotics (sleeping pills) and sedatives was found. This trend decreased considerably when night flights were largely banned. Such a decrease was not found regarding cardiovascular drugs for which the purchase also increased with time. However, this may partly be due to the fact that atherosclerotic manifestations of high blood pressure are less reversible (in contrast to vasoconstriction, which is related more to acute or semi-acute effects, e.g. in children).

It was mentioned in the previous section that closing the windows had a protective effect on blood pressure readings in the Tyrol study [#30]. This was only found regarding closing the windows during the night and not during the day. Furthermore, subjects who had switched the bedroom and the living room because of the noise, had significantly lower blood pressure than those who had not. The findings are discussed in a broader context under coping strategies (Lercher 1996).

When subjective responses to community noise were considered, higher relative risks of cardiovascular diseases were found for noise-related disturbances of sleep and relaxation, rather than for other disturbances or subjective descriptions of noise exposure, which did not refer to the night-time. This was found in the Caerphilly and Speedwell studies [#44], the

"NaRoMI" study [#61], the Spandau Health Survey [#58] and for a general population sample of Germany [#35]. The "LARES" study [#62], in which noise-induced sleep disturbance was assessed, did not show a higher relative risk compared to general annoyance.

5.6 Risk groups

5.6.1 Age and gender

Most epidemiological noise studies looked at cardiovascular effects of community noise in men. This may simply be due to the fact that the prevalence of cardiovascular diseases in middle-aged subjects is higher in men than in women (Hense et al. 2003). Statistical power is an important issue for the design of a study. Furthermore, in noise experiments, physiological reactions controlled by the autonomic nervous system were less pronounced in females than in males (Ising and Braun 2000; Neus et al. 1980). Improper control for possible differential effects of the intake of sex hormones including contraceptives, which may protect or promote adverse (noise-) stress effects (Petitti 2005), may act conservatively on the results (Cairns et al. 1985; Eiff 1993; Farley et al. 1998).

In the studies carried out in Luebeck [#15], Pancevo [#54], Berlin [#61], Stockholm [#50], a German population sample [#35], Bonn (when considering residence time) [#09], and in Amsterdam (when considering angina pectoris) [#05], higher prevalences of hypertension, ischaemic heart diseases and the use of cardiovascular drugs, were found in noise exposed men rather than in women. The opposite was found in the studies carried out in Bonn (when considering sound level) [#09], Sollentuna [#47], and in Amsterdam (heart trouble] [#05].

In the studies carried out in the Soviet Union, it was reported that noise effects on the cardiovascular system were more pronounced in young and middle-aged subjects [#02]. Similar results were found in Swedish noise studies [#47, #60] and the "LARES" study [#62]. The opposite (larger effects in elderly subjects) was reported from the Amsterdam study [#05] and the Stockholm study [#50].

5.6.2 Children

The available database on cardiovascular effects of noise in children is poor. No data is available that refers, in particular, to noise and sleep. The quantitative impact of transportation noise on the cardiovascular system is still a matter of research. A quantitative health risk assessment for children cannot be made at the moment.

Based on the available information from noise studies, it must be concluded that children do not appear to be a particular risk group with respect to cardiovascular outcomes, especially blood pressure. This does not mean that the literature does not suggest higher blood pressure readings in children. It only means, that the effect in children does not appear to be different than that in adults. However, children may be longer exposed to noise throughout their lifetime than the adults that have already been studied. No long-term follow-up studies are known that focus on noise exposure. Most studies on children considered noise in schools rather than noise at home, which implies different mechanisms about how noise could contribute to a rise in blood pressure (raised effort in learning/speech perception vs. disturbed relaxation/sleep).

5.6.3 Health impaired subjects

The prospective part of the Caerphilly and Speedwell studies gave a small hint that health status could be a modifying factor. In subjects with prevalent chronic diseases, road traffic noise was associated with a slightly larger increase in the incidence (new cases) of ischaemic heart diseases than in subjects without prevalent diseases – when the objective noise level was considered (Babisch et al. 2003b). Surprisingly, when annoyance and disturbances due to traffic noise were considered for exposure, the opposite was found. Noise effects were only seen in subjects without prevalent diseases. This was discussed with respect to reporting bias.

5.7 Statistical significance

One of the problems of environmental epidemiology is to statistically ascertain small effects, but on the other hand small relative risks may achieve relevance in environmental policy because of the larger number of exposed persons (Neus et al. 1995). Most of the evaluated noise studies did not achieve statistical significance with the results.

Errors of type-I (α-error, level of significance) are normally used for identifying the statistical uncertainty of effects estimators. Such an error describes the probability with which a test hypothesis (correlation detected) is erroneously accepted instead of a null hypothesis (no correlation). The null hypothesis is conventionally discarded in statistical analyses if the α-error is ≤5% (Sachs 1974). This decision criterion (test of significance), successfully applied in technical fields of quality assurance, is also used in health and environmental research. Errors of type-II (β-error), however, may not be ignored in terms of any possible erroneous decisions in the highly valued asset of health protection (Ortscheid 1995). This β-error refers to the

probability of zero-hypotheses being erroneously retained. The smaller the α-error chosen for the acceptance of a test hypothesis, the greater is the probability of not determining a true correlation.

Statistical test levels may in principle be variably handled, depending on the question raised (Hartung et al. 1995). In the epidemiological literature, time and again publications are found where α-errors of ≤10% are taken as a measure for decisions. This has certainly unleashed controversial discussion. However, it has to be borne in mind in the discussion of significance that usually "two-sided" statistical tests are calculated. This means that for distribution parameters in a test sample, deviations in both directions of the expected value are considered and tested. But if there exist justified assumptions of a change in direction of an effect parameter under exposure conditions, e.g. in experimental laboratory tests or for biological plausibility reasons, then the statistical test procedure may also be carried out "one-sided". (NB: The 5% criterion of the one-sided test equals the two-sided test for the 10%-level of significance).

In the specialist literature, the mechanistical application of the significance criterion is rejected, especially in the public health area (Burton et al. 1998; Rothman 1986; Woolson and Kleinman 1989). In environmental epidemiology where large sample tests are needed for statistical effect evidence, non-significant results are often caused by weak test power. "Non-significance", however, does not mean that no correlation exists (Morrell et al. 1997; Rothman 1986). Therefore, the statement of confidence intervals (for instance of the 95% confidence interval) is required for calculating the effect estimator (Hennekens and Buring 1987; Rothman and Greenland 1998). It is expressly pointed out that the objective of quoting confidence intervals for statistical characteristics is not one for reducing the information to trivial significance assessment, but to provide a quantitative assessment of the statistical safety of the effect estimator (Rothman et al. 1993). (NB: Inclusion of a relative risk of "1" in the 95% confidence interval is equivalent to an α-error of 5%). An unexpectedly high relative risk, for example, which is significant and comprises a large confidence interval, may yet be of little evidence compared to a low relative risk with a small confidence interval that only just fails statistical significance.

5.8 Evidence and causality

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 (see section 3) this refers, in particular, to hypertension and ischaemic heart diseases.

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

Causality in epidemiology can never be proven (Christoffel and Teret 1991; Parascandola and Weed 2001; Schlesselman 1987; Weed 2000). It is a gradual term for which evidence is increasing with the increasing number of facts. However, the magnitude of effect, presence of exposure-effect relationship, consistency with other studies in different populations and with different methodology and biological plausibility are commonly accepted arguments for a causal relationship (Evans 1976; Hill 1965; Morabia 1991; Weed and Hursting 1998).

We have to learn to live with uncertainties (Rose 1992; Scheuplein 1993). However, “no scientific evidence” does not mean “no effect” (Morrell et al. 1997). The precautionary principle can be the ground on which decisions can be made, given the small and weak database that we at the moment have to rely on (Ricci et al. 2003; WHO Regional Office for Europe 2000a). Horton stated the precautionary principle as: “We must act on facts, and on the most accurate interpretation of them, using the best scientific information. That does not mean that we must sit back until we have 100% evidence about everything. Where the state of the health of the people is at stake, the risks can be so high and the costs of corrective action so great, that prevention is better than cure. We must analyse the possible benefits and costs of interaction. Where there are significant risks of damage to the public health, we should be prepared to take action to diminish those risks, even when the scientific knowledge is not conclusive, if the balance of likely costs and benefits justifies it” (Horton 1998). Cost-benefit analyses and probabilistic approaches can help decision making (Ricci et al. 2003).

Decision makers have to make their decisions on rational grounds of limited resources, concurring risks and quality targets. They strongly rely on cost-effectiveness and cost-benefit considerations (Brown 1985; Cleland-Hamnett 1993; Moghissi 1993). However, the setting of environmental standards including limit values, guideline values and other standards is not a purely scientific task in this respect. It was pointed out by Rohrmann (Rohrmann 1993) that “critical limits for environmental stressors can not be derived from empirical sciences. They are

socio-political settings that depend on the weighing system of all groups involved. Limit values are a normative act as a result of complex considerations about benefits, risks and costs.”

6. Exposure - effect curve: meta analysis

The concept of meta-analysis was used to aggregate and summarize the findings of the different studies (Blettner et al. 1999; Olkin 1995). Considerations of the evidence on correlations between exposure factors and effects are generally not undertaken on the basis of individual study findings anyway, but on the basis of the entire available literature on the issue in question (qualitatively by graphical or quantitatively by mathematical methods) (Blettner et al. 1999). The reason for conducting a meta-analysis, for instance, may be that a number of studies methodically appraised as good with common design features, which when taken individually would perhaps not produce significant results. When the studies are pooled an overall effects estimate with a smaller confidence interval may be determined, which may in fact be significant in the conventional sense. On the other hand, any grounds for heterogeneity between studies should also be determined within the framework of a meta-analysis (Blair et al. 1995; Olkin 1995). Meta-analysis provides a more formal statistical approach to the criterion of consistency than the “rule of thumb” summarizing techniques. However, meta-analysis alone is not sufficient for making causal claims (Weed 2000).

For a quantitative risk assessment and the derivation of guidelines for public health noise policy a common exposure-effect (dose-response) curve is required. The risk estimates obtained from different noise studies can be summarized using the statistical approach of a meta-analysis. Based on the judgement criteria discussed in section 5.2, five analytic and two descriptive studies emerged that can be used to derive a common exposure-effect curve for the association between road traffic noise and the risk of myocardial infarction. Two separate meta analyses were made by considering the analytic studies that were carried out in Caerphilly and Speedwell (pooled 6 yrs follow-up data) [#44] and Berlin (case-control studies "Berlin I", "Berlin II", "Berlin III") on the one hand [#32, #33, #61], and the descriptive studies that were carried out in Caerphilly and Speedwell (cross-sectional studies) on the other hand [#27, #28]. It turned out as a result of the evaluation process, that all these studies referred to the road traffic noise during the day (L_{day}: 6-22 hr) and the incidence or prevalence of myocardial infarction as the outcome. Study subjects were men. In all analytic studies, the orientation of rooms was considered for the exposure assessment (facing the street or not). With respect to the Caerphilly and Speedwell studies, the 6 yrs pooled follow-up data provided the necessary information (see *Table A6*). In all descriptive studies the traffic noise level referred to the facades that were facing the street and did not consider the orientation of rooms/windows. All individual effect estimates were adjusted for the covariates considered in each of the studies. Different sets of covariates were considered in each study. However, this pragmatic approach accounts best for possible

confounding in each study and provides the most reliable effect estimates derived from each study.

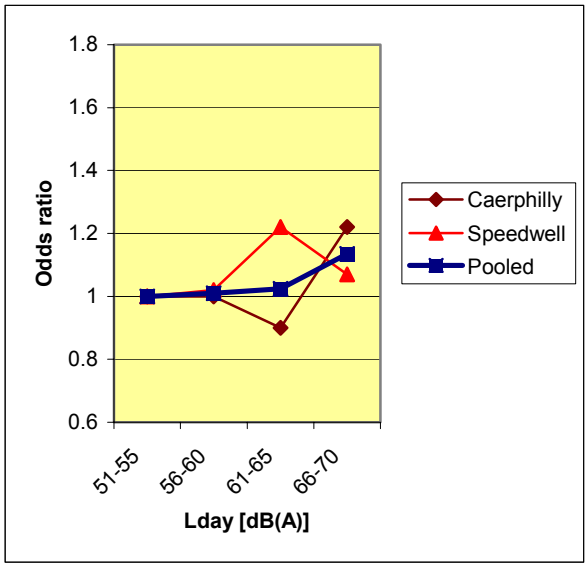
In a previous meta analysis, the regression coefficients for the whole range of noise levels from single studies were pooled (Kempen et al. 2002). Here, a different approach was considered, to account for non-linear relationships in studies (e.g. “j”-shaped curves). The effect estimates given for noise categories from different studies (5 dB(A) classes) were pooled across studies.

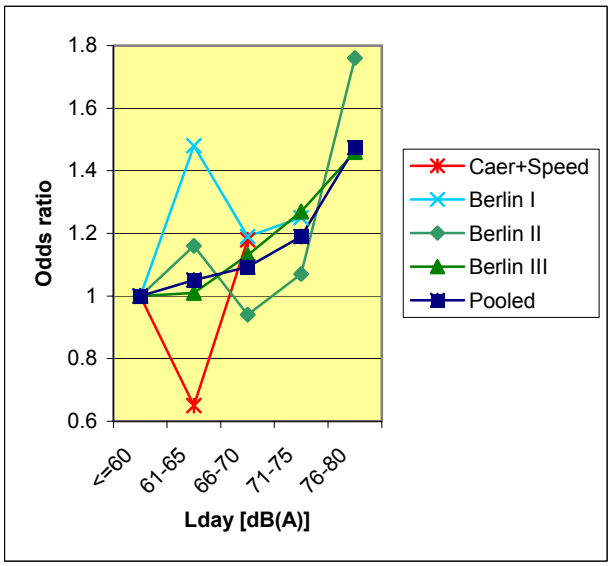
The programme "META" was downloaded from the STATA website for use in the statistical package STATA (version 8.0), and for calculating the pooled random effect estimates. *Table 1* shows individual and pooled effect estimates with confidence intervals (rounded brackets), statistical weights (square brackets) for the individual studies, and the Q-test of heterogeneity between studies (Takkouche et al. 1999). According to the Q-test, the nil-hypothesis of non-heterogeneity was never discarded. *Figures 7 and 8* show odds ratios of individual studies and the pooled estimates for the descriptive and analytic studies. *Figures 13 and 14* show pooled effect estimates together with their 95% confidence intervals.

Table 1. Single and pooled (meta analysis) effect estimates (odds ratios and 95% confidence intervals) for descriptive and analytic studies on the relationship between road traffic noise level (L_{day}) and the incidence/prevalence of myocardial infarction

	Road traffic noise level - L_{day} - [dB(A)]					
Descriptive studies	**51-55**	**56-60**	**61-65**	**66-70**		**N**
Caerphilly	1.00	1.00 (0.58-1.71), [13.29]	0.90 (0.56-1.44), [17.23]	1.22 (0.63-2.35), [8.98]		2512
Speedwell	1.00	1.02 (0.57-1.83), [11.19]	1.22 (0.70-2.12), [12.62]	1.07 (0.59-1.94), [10.94]		2348
Pooled	1.00	1.01 (0.68-1.50)	1.02 (0.72-1.47)	1.14 (0.73-1.76)		
Q-Test		p = 0.96	p = 0.41	p = 0.77		
Analytic studies	**<=60**	**61-65**	**66-70**	**71-75**	**76-80**	**N**
Caerphilly + Speedwell	1.00	0.65 (0.27-1.57), [4.95]	1.18 (0.74-1.89), [17.48]	---	---	3950
Berlin I	1.00	1.48 (0.57-3.85), [4.21]	1.19 (0.49-2.87), [4.94]	1.25 (0.41-3.81), [3.09]	1.76 (0.11-28.5), [0.50]	243
Berlin II	1.00	1.16 (0.82-1.65), [31.43]	0.94 (0.62-1.42), [22.76]	1.07 (0.68-1.68), [18.92]	1.46 (0.77-2.78), [9.27]	4035
Berlin III	1.00	1.01 (0.77-1.32), [54.42]	1.13 (0.86-1.49), [50.87]	1.27 (0.88-1.84), [28.24]	---	4115
Pooled	1.00	1.05 (0.86-1.29)	1.09 (0.90-1.34)	1.19 (0.90-1.57)	1.47 (0.79-2.76)	
Q-Test		p = 0.57	p = 0.87	p = 0.84	p = 0.90	

Numbers are odds ratios, 95% confidence intervals are given in round brackets (), statistical weights are given in square brackets []; Pooled = pooled estimates (meta analysis), p = probability of the Q-Test for heterogeneity, N = sample size





Figures 7 and 8. Single and pooled effect estimates (odds ratios) for the descriptive and analytic studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction

A polynomial function was fitted to the pooled data points of *Figures 8 and 14*. Linear, quadratic and cubic terms were considered. The results and the coefficients of the equations are shown in *Figures 9 and 10* for the non-weighted and weighted (number of subjects) data. The weights are: 8963 (≤60 dB(A)), 1063 (61-65 dB(A)), 843 (66-70 dB(A)), 346 (71-75 dB(A)), 59 (76-80 dB(A)). Mean category values of the decibel-axis were considered for the calculation. For the reference category "≤60 dB(A)" a value of 55 dB(A) was used, because this category includes also a large number of noise levels below 55 dB(A). Changing this value to others (e. g. 52.5 or 57.5) had only a very marginal impact on the coefficients and the fit statistics. More than 95% of the variance (R^2) of the pooled data points was explained by the polynomial functions. For quantitative risk calculations either the data referring to mean category values, or the fitted curve referring to the weighted data points, should be used.

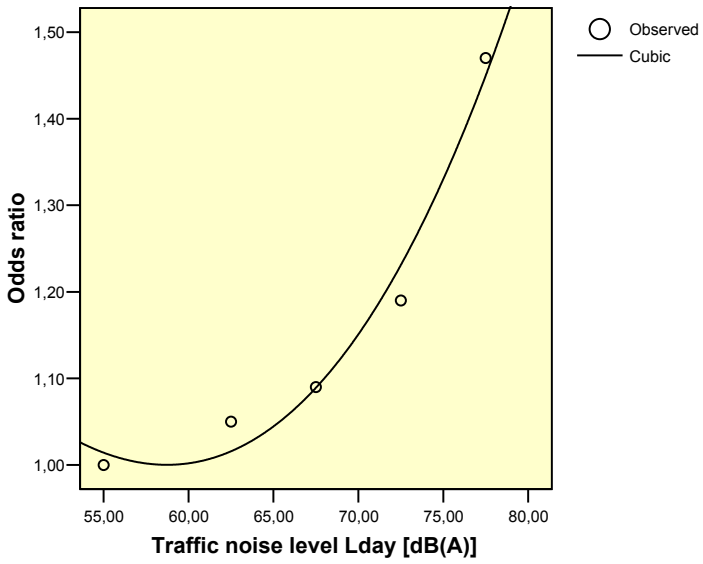


Figure 9. Polynomial curve fit (non-weighted data points) of the association between road traffic noise and incidence of myocardial infarction.
OR = 2.210093 – 0.001052*Noise^2 + 0.00001194531314644*Noise^3 ; R^2 = 0.98
(no significant linear term in the equation)

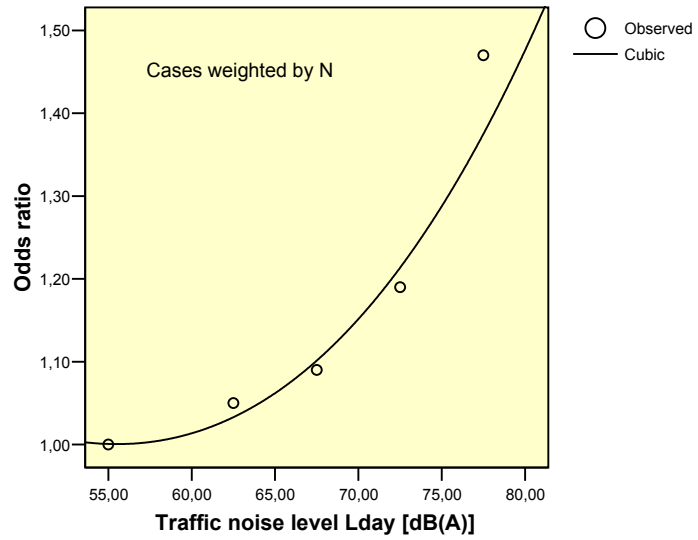


Figure 10. Polynomial curve fit (N-weighted data points) of the association between road traffic noise and incidence of myocardial infarction.
OR = 1.629657 – 0.000613*Noise^2 + 0.000007356734623455*Noise^3 ; R^2 = 0.96
(no significant linear term in the equation)

7. Risk evaluation

7.1 Conceptual framework

A conceptual framework for the regulation of environmental hazards was given by the US National Research Council (National Research Council 1983; Patton 1993). It is illustrated in *Figure 11* (Neus and Boikat 2000). The process of risk assessment (risk evaluation) comprises hazard identification ("Which health outcome is relevant for the exposure?"), exposure assessment ("How many are affected") and dose-response assessment ("Threshold of effect?"). This information is summarized in "risk characterization" ("health hazard characterization"). It involves the interpretation of the available evidence from the available data and other scientific disciplines, and is subject to discussion of the uncertainties (WHO Working Group 2000). These include chance, bias and validity of studies as well as transparency, replicability and comprehensiveness of reviews. As a result of the risk evaluation process, a quantitative estimate about the likelihood that the hazard will affect exposed people can be derived. Usually attributable risk percentages are calculated (Walter 1998). This serves as key information for any kind of risk management including regulatory options (Jasanoff 1993).

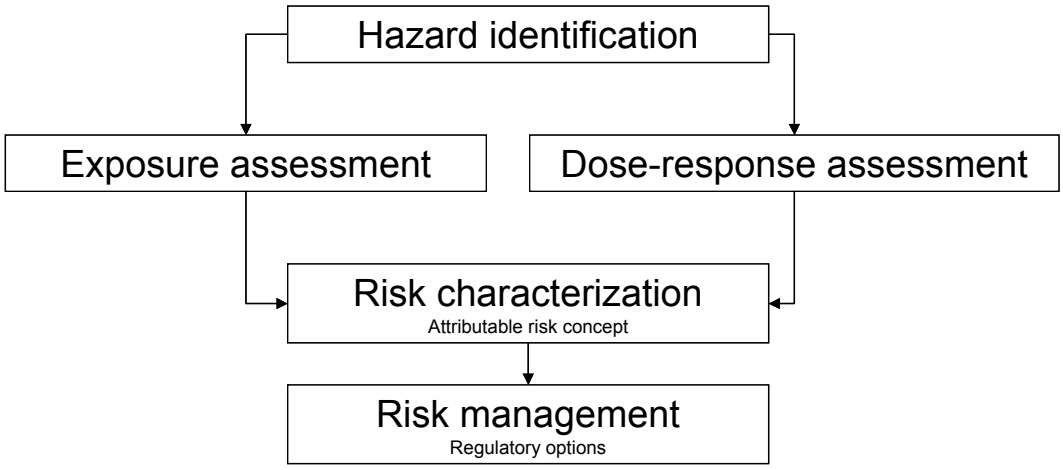


Figure 11. Process of risk evaluation

The term "adverse" is essential in this context of environmental standard setting. Risk management should ensure that "adverse" health effects do not occur. The World Health Organization defines an "adverse effect" as follows (WHO 1994): "Change in morphology, physiology, growth, development or life span of an organism, which results in impairment of the functional capacity to compensate for additional stress, or increase in susceptibility to the

harmful effect of other environmental influences". It is obvious that the relevance of noise effect increases with increasing severity and the high prevalence of the considered health outcome. The fact that an organism responds to noise must not be per se "adverse". For example, thresholds of acute changes in EEG, finger pulse amplitude, stress hormones - the whole startle reaction - may be interpreted in terms of no/lowest observed effect levels, using the terminology used in toxicological science (NOEL and LOEL) (Dieter 1995). However, they may not have pathological significance. Furthermore, due to improvements of measuring techniques, thresholds tend to decline to levels without clinical relevance. Therefore, NOEL and LOEL may not be suitable for decision making in general.

It is sometimes suggested to refer to the excess of "normal values" (exceedances) of physiological factors as a criterion of effect. However, even such exceedances are not necessarily associated with an increased risk. Physiological normal values are usually defined by statistical grounds of distributions. However, once there is quantitative evidence that subjects with a chronically high biological value above normal run a higher risk for subsequent disorders, then we call this factor a risk factor per definition, and it is of clinical relevance. In such cases, we are looking at no/lowest observed adverse effect levels (NOAEL and LOAEL), which indeed may have implication for noise policy according to the WHO recommendations. NOAELs are commonly used in public health policy for preventive action. In order to obtain reasonably safe standards, it is usual to start from the NOAEL and to apply additional safety or uncertainty factors (Arnold et al. 1997; Neus and Boikat 2000; WHO 1994).

Decisions on whether or not any effect is adverse, require expert judgement. The severity of a health outcome is an important determinant of the adversity of an effect and implies variable action levels for public health policy (Babisch 2002; Babisch 2004a; Englert 2004; Griefahn et al. 2002; Health Council of The Netherlands 2003). This is outlined in *Figure 12*, which was taken from the "Handbuch der Umweltmedizin" (Wichmann and Ihme 1999), and adapted for the issue of noise effects (Babisch 2002). Since the diagram was originally designed for chemical exposures, the lowest effect grade ("internal exposure") was substituted by "annoyance". Unlike chemical noxious substances like lead or cadmium, for example, noise as such does not accumulate in the organism. One cannot measure "noise" in the organism, only its effects. Next in the pyramid comes "physiological changes of unknown significance" followed by "pathological changes", "morbidity" and "mortality" or "life span". Effects regarding the top three outcomes of the triangle may be attributed "adverse" according to the WHO criteria.

It should be mentioned in this context that pragmatic criteria are commonly used to define annoyance as an adverse outcome. However, these definitions do not refer to the severity of the outcome for the individual as such, but to the number of "annoyed" or "highly annoyed

subjects" in the exposed population. For example, 25% highly annoyed were considered as an adverse effect ("considerable annoyance") (Guski 2001; Maschke and Harder 1998).

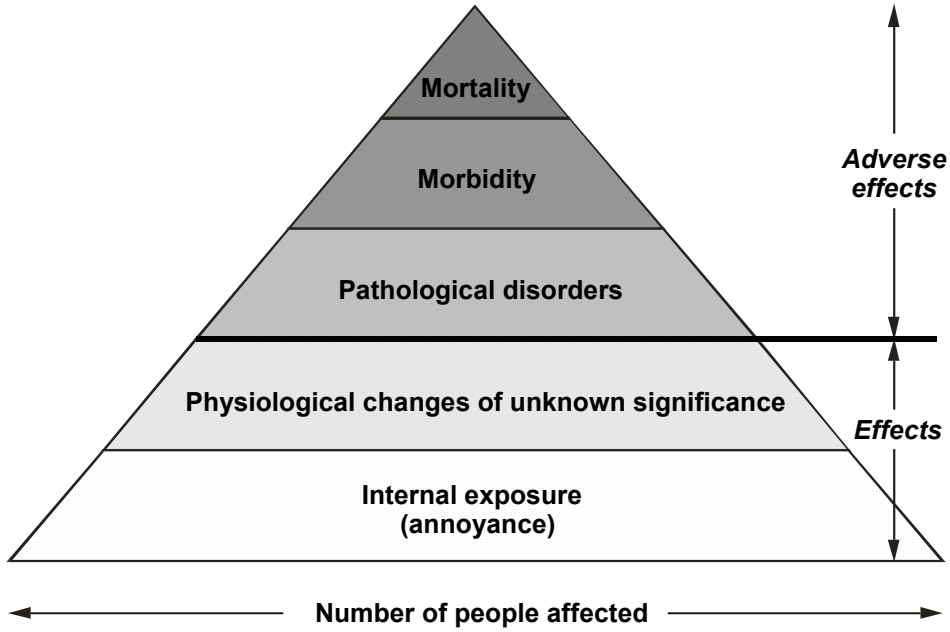


Figure 12. Severity of (noise) effects

In public discussions of environment and health hazards, the application of the statistical term "risk" often proves to be problematic. In colloquial language it is understood as a synonym for "danger", thus making competent reasoning rather difficult (Fülgraff 1992). While the term "danger" is used in defining a qualitative relationship between exposure factor and health, the term "risk" is used in the same relationship for a quantitative assessment (Zeger 1991). The statistical risk provides the probability for a certain damage occurring at a given point in time, either on the grounds of chronic exposure, or resulting from an acute event. Taking risks is a matter-of-course in everyday life. In this respect, decision making either for an individual or a community involves risk taking (Moghissi 1993). This may be seen in reference to the above mentioned system of values and standards prevailing in communities. The WHO-guidelines for drinking water, for instance, tolerate a cancer attributable lifespan and death outcome risk of $1{:}10^5$ (WHO 1993), reflecting a socially accepted background risk (Dieter and Grohmann 1995; Scheuplein 1993).

Since considerable parts of the population are exposed to high noise levels (EEA 2004), noise policy can have a significant impact on public health (Neus et al. 1995; Neus and Boikat 2000).

Due to the increasing number of people affected with the decreasing severity of the effect, even small individual risks and less severe health outcomes can be relevant for public health and decision-making. It has been shown, that moderate noise exposures implying a small individual risk, may cause more noise-induced cases of health impaired subjects than higher noise exposures. The number of people suffering from poor health due to aircraft noise is dominated by the larger number of people that is exposed to relatively moderate to low noise levels and not by those exposed to high noise levels (Franssen et al. 2004). This means that emphasis should be put on the reduction of noise in moderately exposed areas. However, public health policy cannot only consider population attributable risks (risk percentages), but must also consider individual risks (lifetime risk). In practice, noise policy should reduce noise, beginning with the highest exposures.

Decision-making will have to find common standards of acceptable risks, which may vary according to the cost-benefit considerations within and between communities and countries. Such practical standards may though vary due to the economic development and abilities, the cost-benefit considerations and the priority settings of a community or country. Health quality targets derived from scientific research are usually intended to minimize risks. Decision making in the political process is only partly scientifically based, but also due to economic limitations and concurring interests (Nijland et al. 2003). Different health-outcomes or indicators of well-being and quality of life imply different action levels. Environment and health policy must determine acceptable noise standards that consider the whole spectrum, from subjective well-being to somatic health (e.g. annoyance, physiological arousal, health risk).

7.2 Attributable risk percentage

Table 2 shows the occurrence of cardiovascular diseases in Germany for the years 1994 to 1999, grouped according to the international coding system of diseases (ICD code 9). For example, in the year 1999, 849,557 cases of ischaemic heart diseases (ICD 9, No. 410-414) including 133,115 cases of acute myocardial infarction (ICD 9, No. 410) were detected (Statistisches Bundesamt and Robert Koch-Institut 2005). The data on disease occurrence includes survivors and lethal cases that were treated in hospitals of the 'old' and 'new' German Federal States. Lethal cases of ischaemic heart diseases (IHD) in the year 1995 were 183,736 of 773,538 (24%); lethal cases of acute myocardial infarction (MI) were 87,739 of 133,311 (66%) (Statistisches Bundesamt 1998). As a general tendency, the standardized (standard population 'Germany 1987') disease-specific death rates due to cardiovascular disorders tended to decline over the years. This is is shown in *Table 3* (Statistisches Bundesamt and Robert

Koch-Institut 2005). Due to the change from ICD code 9 to ICD code 10, the figures before 1998 and from 1998 onwards cannot directly be compared.

Table 2. Disease occurrence in Germany (1994-1999)

ICD 9	1994	1995	1996	1997	1998	**1999**
Cardiovascular diseases (No. 390-459)	2,288,764	2,413,429	2,511,855	2,580,989	2,728,033	2,764,146
Acute rheumatic fever (No. 390-392)	2,038	1,887	1,515	1,421	1,391	1,292
Chronic rheumatic diseases (No. 393-398)	34,295	30,222	26,678	24,608	23,744	22,718
Hypertension and high blood pressure (No. 401-405)	148,692	154,640	159,122	166,656	185,083	186,822
Ischaemic heart diseases (No. 410-414)	703,996	773,538	794,615	813,294	855,563	**849,557**
Acute myocardial infarction (No. 410)	132,921	133,311	131,094	127,724	132,501	**133,115**
Diseases of the pulmonary circulatory system (No. 415-417)	34,898	34,817	34,497	34,785	37,758	38,481
Other heart diseases (No. 420-429)	493,463	522,327	561,507	582,354	625,543	638,996
Cerebral-vascular diseases (No. 430-438)	385,059	397,573	420,697	439,138	462,885	476,441
Diseases of arteries, arteriols and capillaries (No. 440-448)	184,437	189,142	193,638	198,684	207,743	215,100
Venous and other vascular diseases. (No. 451-459)	301,886	309,283	319,586	320,049	328,323	334,739

Table 3. Standardized disease-specific death rates per 100,000 subjects (1980-1997 and 1998-2003)

ICD 9	1980	1985	1990	1995	1996	1997
Cardiovascular diseases (No. 390-459)	719.0	650.1	561.2	478.4	466.4	450.3
Ischaemic heart diseases (No. 410-414)	221.9	226.4	211.8	208.9	203.0	197.7
Acute myocardial infarction (No. 410)	124.3	117.9	106.6	102.9	98.5	94.9
ICD 10	1998	1999	2000	2001	2002	2003
Cardiovascular diseases (No. I00-I99)	440.5	425.8	401.8	389.2	385.5	384.4
Ischaemic heart diseases (No. I20-I25)	100.3	100.1	93.3	90.6	90.8	88.5
Acute myocardial infarction (No. I21)	85.8	78.0	73.1	69.3	67.1	66.1

Standard: German population 1987

Table 4 shows the distribution of the general German population that is exposed to different levels of road traffic noise for the years 1992 and 1999 (Umweltbundesamt 1997; Umweltbundesamt 2001). The distribution of noise exposure during the day and the night was estimated on the basis of the German noise pollution model ('Lärmbelastungsmodell') (Babisch 2003a; Nolle and Pollehn 1989). No major changes can be seen throughout the years. Although the exposure data refer only to the 'old' Federal States of Germany; they can be approximately applied to the whole of Germany (Wende and Malow 1996). This has been supported by calculations and comparisons that were made with respect to road kilometres in former East and West Germany (Umweltbundesamt 1994). The approximation is further validated by the

number of annoyed people, which tend to be very similar in the 'old' and 'new' Federal States (Ortscheid and Wende 2002).

Table 4. Exposure to road traffic noise in Germany (1992 and 1999)

	Percentage of the population [%]			
Average Sound Pressure Level [dB(A)]	Road traffic day 1992	Road traffic night 1992	Road traffic day 1999	Road traffic night 1999
>45 - 50	16.5	17.7	16.4	17.6
>50 - 55	15.8	14.7	15.8	14.3
>55 - 60	17.9	9.8	18.0	9.3
>60 - 65	15.6	4.3	15.3	4.2
>65 - 70	9.1	2.9	9.0	2.9
>70 - 75	5.2	0.2	5.1	0.2
>75	1.5	0.0	1.5	0.0

Both, the exposure data and the disease data of the year 1999 are considered for a quantitative risk assessment regarding the association between road traffic noise and cardiovascular risk. The exposure-effect risk curve derived from the meta analysis (see chapter 6) is applied. The following formulas are used to calculate the attributable fractions (AR%), the population attributable risk percentages (PAR%) and the absolute numbers of affected subjects (PAR) for each noise category (Hennekens and Buring 1987):

AR% = (RR-1) / RR * 100

PAR% = P_e/100 * (RR-1) / (P_e/100 * (RR-1) + 1) * 100

PAR = PAR% * N_d

where

RR = Relative risk (odds ratios are estimates of the relative risk)

P_e = Percentage of the population exposed

N_d = Number of subjects with disease (disease occurrence)

The results are shown in *Table 5*. For each noise category the attributable proportions and the number of subjects with expected incidence of myocardial infarction (MI) due to road traffic noise are given. All higher noise categories above the reference category of ≤60 dB(A) contribute nearly equally to the total number of subjects at risk. This is due to the larger number of exposed subjects in lower noise categories, where the relative risk is lower. In total, 3.2% of

myocardial infarctions in Germany are due to the road traffic noise (if the noise hypothesis is true). This accounts for approx. 4,289 MI cases per year, of which approx. 66% are lethal. The respective number of IHD cases per year would be 27,376 (approx. 24% lethal) for Germany, if the same dose-effect curve for the relative risk is considered (see conclusions).

Table 5. Risk estimation (risk of myocardial infarction due to road traffic noise)

Road traffic noise 1999	**Risk of myocardial infarction due to road traffic noise**				
Average Sound Pressure Level during the day (6-22 h) [dB(A)]	Percentage exposed [%]	Relative risk OR	Attributable fraction AR%	Population attributable risk percent PAR%	Number of subjects per year
<= 60	69.1	1.00	0.00	0.00	0
>60 - 65	15.3	1.05	4.76	0.76	1,011
>65 - 70	9.0	1.09	8.26	0.80	1,070
>70 - 75	5.1	1.19	15.97	0.96	1,278
>75	1.5	1.47	31.97	0.70	932
Sum				**3.22**	**4,289**

8. Conclusions

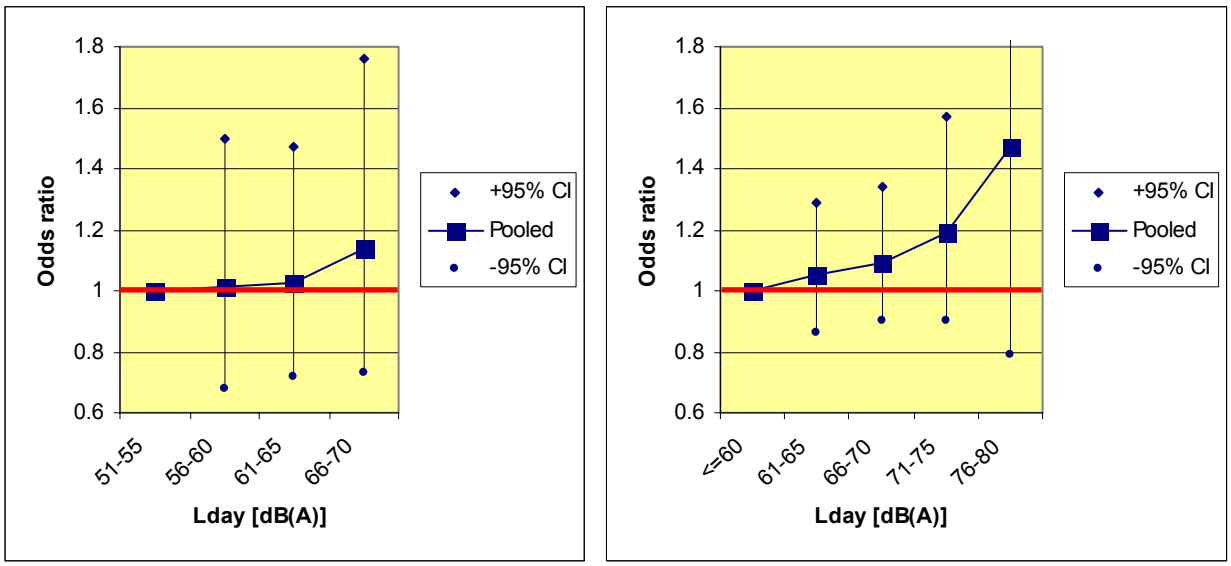
The evaluation process of studies (review), used in this report considered the "necessary" criteria: peer-reviewed publication in an international journal, reasonable quantitative control of possible confounding (not only descriptive comparisons), objective assessment of exposure and outcome. "Additional" criteria for the ranking were: type of study (analytic vs. descriptive) and dose-response assessment (not only dichotomous "high" vs. "low"). The approach differs from that of an earlier meta-analysis (Kempen et al. 2002) in that their regression coefficients were calculated for the entire dose response curve within a single study (e.g., the increase in risk per 5 dB(A)), which then were pooled between studies. Since higher exposure categories usually consist of smaller numbers of subjects than the lower categories, regression coefficients across noise levels tend to be influenced by the lower categories. This may lead to an underestimation of the risk in higher noise categories. The approach presented here pooled the effect estimates of single studies within each noise category, thus giving more weight to the higher noise categories and accounting for possible non-linear associations.

Using all the four "necessary" criteria, five analytic studies emerged that refer to the association between road traffic noise and myocardial infarction (MI). The studies were carried out in Caerphilly (UK) and Speedwell (UK) (Babisch et al. 1999), and Berlin (Germany) (Babisch et al. 2005; Babisch et al. 1994). These studies were also considered in the meta-analysis from Kempen et al. (Kempen et al. 2002), with the exception, that the newest study was not known at that time. If cross-sectional studies are allowed, another two descriptive studies from Caerphilly (UK) and Speedwell (UK) can be taken into account (Babisch et al. 1993a). They were also considered in the earlier meta-analysis. Study subjects were men; mainly for reasons of sampling efficiency and statistical power. In middle-aged groups of subjects, males are more prone to cardiovascular diseases (Yusuf et al. 2001a; Yusuf et al. 2001b). Furthermore, the confounding impact of hormonal status of females was excluded by this restriction. However, it seems to be reasonable to assume that, in relative terms (relative risk), females may be just as affected by noise stress as males, if the relevant confounding factors are taken into consideration.

Figures 13 and 14 show the two risk curves for descriptive and analytic studies (Hennekens and Buring 1987). The graphs show the pooled effect estimates (odds ratios) and the 95% confidence intervals for each noise category. Whereas the cross-sectional studies (*Figure 13*) cover the sound level range of L_{day} from >50 to 70 dB(A), the cohort and case-control studies (*Figure 14*) cover the range from ≤60 to 80 dB(A). Both curves together can serve as a basis for a quantitative risk assessment. From *Figure 13* it can be seen that below 60 dB(A) for L_{day}, no

notifiable increase in MI risk is to be detected. Therefore for the time-being, L_{day} = 60 dB(A) can be seen as NOAEL ("no observed adverse effect level") for the relationship between road traffic noise and myocardial infarction (Babisch 2002). For noise levels greater than 60 dB(A), the MI risk increases continuously, and is equal or greater than 1.2 for noise levels of 70 dB(A) and higher. This can be seen in *Figure 14* and *Table 1*, with relative risks ranging from 1.1 to 1.5 across the noise level range from 61 to 80 dB(A) for reference L_{day} <60 dB(A).

It should be mentioned that the risk estimates, in general, were found to be higher in subjects that had lived in the exposed areas for a longer time (Babisch et al. 2005; Babisch et al. 1999; Babisch et al. 1994). This is in accordance with the noise hypothesis and the effects of chronic noise stress (Lercher and Kofler 1996; Thompson 1997). Induction or latency periods of more than 10 years have to be considered (McCarron and Smith 2005; Rose 2005). However, for the calculation of population attributable risk percentages the figures for the whole population are relevant with the natural distributions of residence time.



Figures 13 and 14. Pooled effect estimates (meta analysis) for descriptive and analytic noise studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction (odds ratio ± 95% confidence interval (CI))

For a quantitative assessment of the association between community noise and hypertension, only the Okinawa study (Japan), formally fulfils the inclusion criteria set here (Matsui et al. 2004). It considers aircraft noise from a military airfield and provides dose-response data. Two studies that were carried out around Amsterdam's airport (The Netherlands) appear on the list

of studies, if the dose-response criterion is neglected. Cardiovascular diseases in general (Knipschild 1977b) and specific outcomes, including hypertension and ischaemic heart diseases (angina pectoris, ECG abnormalities, heart trouble) were clinically assessed (Knipschild 1977a). One of those studies was considered in the earlier meta-analysis, i.e. the only one on aircraft noise and hypertension. If dose-response studies of self-reported prevalence of diseases are included, two more peer-reviewed studies can be listed. These studies were carried out in Berlin (Germany) with respect to road traffic noise and myocardial infarction (Babisch et al. 1994) and Stockholm (Sweden) with respect to aircraft noise and hypertension (Rosenlund et al. 2001). More research is needed regarding the association between community noise and hypertension.

Studies that are not given a high ranking according to the above mentioned criteria may serve as additional sources of information when assessing the evidence of such an association. This was done in previous reviews and by expert committees (see section 3). Support for the noise hypothesis comes also from sensitivity analyses, in which effect modifying factors, in particular, exposure/noise-dose modifying factors were considered. Noise effects were slightly larger, when longer residence-time, room orientation (facing the street) and window opening habits were considered.

No particular risk groups could be identified on the basis of epidemiological research on cardiovascular effects of community noise. The assessment of dose-effect relationships sometimes suggested a cut-off level, above which the risk tends to increase. From a biological point of view, one would expect a continuous increase in risk with increasing noise level. However, adaptation, habituation and coping may be reasons for an empirical threshold of effect. Decisions with respect to guidelines values usually refer to a quantitative risk assessment of populations (e.g. population attributable risk percent). However, prevention strategies – for ethical reasons - should not ignore the individual risks of highly exposed subjects, even if their number may be small.

With respect to night noise exposure, nearly no information is available from epidemiological studies on the cardiovascular effects of long-term noise exposure of the bedroom during the night. Only one study distinguished between the exposures of the bedroom and the living room in the statistical analyses (Maschke et al. 2003b). The results suggested slightly higher effect estimates for the prevalence of hypertension with respect to the noise exposure of the bedroom (during the night) compared with the exposure of the living room (during the day). However, the difference was small (odds ration 1.9 vs. 1.5), which means that it still remains an open question whether the night exposure or the overall exposure throughout the whole day is the driving force. The study has some methodological limitations that were addressed in the summary of

the major technical report and in a recent advisory report of the Dutch Health Council (Health Council of the Netherlands 2004). They are mainly concerned with the fact that the study population consisted of a selected, predominantly older and health conscious group of persons that might have already suffered from other health problems (risk group). A few studies that looked at the association between subjective responses to community noise and cardiovascular outcomes suggest a closer relationship with sleep-related annoyance /disturbance reaction rather than with non-sleep related annoyance/disturbance (Babisch et al. 2005; Babisch et al. 1999; Bellach et al. 1995; Maschke et al. 2003a; Niemann and Maschke 2004). Closing the bedroom window or, vice versa, sleeping with the bedroom window open, was associated with a lower or higher risk, respectively (Lercher 1996). The same was found with respect to changing the bedroom with the living room because of noise. These findings may indicate that night-time noise may be more a determinant of noise-induced cardiovascular effects than the daytime exposure. However, daytime activity patterns and expectations of the individuals are much more inhomogeneous than during the night, which tends to dilute the statistical association of true effects with the day-noise exposure.

Given the situation that only few data are available from epidemiological studies with respect to effects on sleep (exposure of the bedroom during the night), there does not seem to be any other way of reasoning, than inferring night noise recommendations or guidelines from the results of studies that refer to the noise exposure during the daytime period (L_{day}) or the whole day (L_{dn}, L_{24h}). L_{den} (Directive 2002/49/EC 2002), in this context, appears to be a useful noise indicator for decision-making and regulatory purposes. Penalties of 5 dB(A) and 10 dB(A) are usually given to the evening period and the night period, respectively. It can be used for noise mapping and refers normally to the most exposed facade, which incorporates a certain degree of exposure misclassification regarding cause-effect relationships. This weighted indicator was introduced to assess the relationship between sound level and noise annoyance (Directive 2002/49/EC 2002). However, it may not be adequate for (somatic) health-related noise effects' research. Non-weighted separate exposure indicators, such as L_{day}, $L_{evening}$ or L_{night}, may be more appropriate when assessing physiological responses to the noise.

In urban settings, night-time average noise levels (22-6 h) for road traffic tend to be approx. 7-10 dB(A) lower than daytime average noise levels - relatively independent (no freeways) of the traffic volume of the street (Evans et al. 2001; Ullrich 1998; Utley 1985). In such cases, L_{den} is approx. 2 to 3 dB(A) higher than L_{day} (Bite and Bite 2004). 24h noise levels of road traffic were found to be 1 to 3 dB(A) lower than daytime noise levels (Rylander et al. 1986). The differences between L_{den} and L_{dn} are small (Miedema and Oudshoorn 2001). Therefore, in epidemiological studies in which the relative effect of road traffic noise is studied, the sound emission during the daytime can as well be viewed as an approximate relative measure of the overall sound

emission including the night. This seems to be further justified because existing noise regulations usually consider a 10 dB(A) difference between the day and the night. The NOAEL of 60 dB(A) for L_{day} corresponds, in this respect, with 50 dB(A) for L_{night}. This approximation can only be made with respect to road traffic noise. For train or aircraft noise no such approximation can be made.

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 (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.

The dose-effect curve presented in this report refers to the incidence of acute myocardial infarction (MI) as a health outcome. It is suggested that similar calculations could be made with respect to all ischaemic heart diseases (IHD) - using the same curve for the relative risk. The international code of diseases ICD 9, No. 410-414 comprises code No. 410 (acute myocardial infarction) and codes No. 411-414 (other acute and sub-acute forms of ischaemic heart disease, old myocardial infarction, angina pectoris, atherosclerosis and other forms of chronic ischaemic heart disease). These health endpoints are similarly considered in the noise hypothesis (see reaction schema in *Figure 1*) (Babisch 2002; Passchier-Vermeer and Passchier 2000). It may be a reasonable assumption that the relative risks for these endpoints show a similar relationship across noise categories. In cross-sectional and prospective cohort studies, the relative risks found for these health endpoints did look similar, within the statistical boundaries of uncertainty (confidence intervals) (Babisch et al. 1993a) (Babisch et al. 1999). However, the severe and manifest event of a myocardial infarction can be most reliably assessed in studies (Bormann et al. 1990).

The present report is clearly focussed on ill-health as an outcome of the adverse effect of noise. A common dose-effect curve for the relationship between road traffic noise (outdoors) and the risk of myocardial infarction was developed. This curve can be used for a quantitative risk

assessment and the calculation of attributable cases in a community. However, decisions regarding limit values have to be made for the whole spectrum from discomfort (annoyance) to ill-health (disease) (Babisch 2002; Jansen et al. 1996; Lindström 1992). The effect threshold for an increase in risk of ischaemic heart disease, including myocardial infarction due to road traffic noise, was found to be around 60-65 dB(A) for $L_{day} \approx L_{den}$. The effect threshold, if any, for serious annoyance tends to be lower, e.g. 55 dB(A) according to WHO recommendations (WHO 2000).

Whereas quality targets at the lower end of the effects scale may be more flexible, quality targets at the upper end may be more obligatory. For example, for ethical reasons (equality principle) it does not seem to be justified if (ill-)health-based limit values are varied according to the type of living area as expressed in land development plans (e.g. residential, mixed or commercial). On the other hand, regarding nuisance, different limit values could be accepted.

10. References

Note: References in the text with preceding '#' (e.g. #10) refer to the studies listed in Table A1.

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Appendix

Table A1. Studies on cardiovascular effects of community noise

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 01 CS	Halle - Germany *(Karsdorf and Klappach 1968)*	Schoolchildren 7-10th. grade >269, mf	(O) Road traffic noise level in school	(O) Blood pressure	(2) Sex, grade in school
# 02 CS	Vicinity of airports - Soviet Union *(Karagodina et al. 1969)*	Population 145000 mf	(O) Distance from airport	(O) Cardiovascular diseases	(0)
# 03 CS	Vicinity of airports - Soviet Union *(Karagodina et al. 1969)*	Schoolchildren 9-13 yr ?, mf	(O) Distance from airport	(O) Blood pressure abnormalities, autonomic vascular changes	(0)
# 04 CS	Munich, - Germany *(Eiff et al. 1974; Rohrmann 1974)*	Adults 21-60 yr 392, mf	(O) Aircraft noise level	(O) Blood pressure	(1) Age, sex, education, years in residence, socio-demographic factors, alcohol consumption, smoking, use of contraceptives, prevalence of multiple diseases
# 05 CS	Amsterdam - The Netherlands *(Knipschild 1977a)*	Adults 35-64 yr 5828, mf	(O) Aircraft noise level	(O) Clinical blood pressure, hypertension treatment, angina pectoris, heart trouble, pathological ECG, pathological heart shape, use of cardiovascular drugs	(2) Age, sex, relative body weight, smoking, size of village
# 06 SU	Amsterdam - The Netherlands *(Knipschild 1977b)*	Population 15-64 yr 18025, mf	(O) Aircraft noise level	(O) Contact rate (during one week) with general practitioner for cardiovascular diseases	(2) Age, sex, other diseases
# 07 t-EC	Amsterdam - The Netherlands *(Knipschild 1977c)*	Pharmacies 8 yrs trend	(O) Change of aircraft noise level	(O) Purchase of antihypertensive and cardiovascular drugs by pharmacies	(0) Change in population size
# 08 CS	Doetinchem - The Netherlands *(Knipschild and Sallé 1979)*	Housewives 40-49 yr 1741, f	(O) Road traffic noise level	(O) Blood pressure, hypertension, angina pectoris, ECG ischaemia, pathological heart shape	(1) Age, physical activity, civil status, relative body weight, smoking, financial situation

Table A1 - continued.

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 09 CS	Bonn - Germany *(Eiff and Neus 1980; Eiff et al. 1981b; Neus et al. 1983)*	Adults 20-59 yr 931, mf 20-49 yr 165, mf	(O) Road traffic noise level	(S,O) Hypertension, myocardial infarction Blood pressure	(2) Age, sex, nationality, income, coffee/tea consumption, smoking, employment status, physical activity, social class, hearing,
# 10 CS	Los Angeles - United States *(Cohen et al. 1980; Cohen et al. 1981)*	Schoolchildren 3-4th. grade 262, mf	(O) Aircraft noise level at school	(O) Blood pressure	(3,4) Grade in school, ethnic group, social class, family size, obesity, height, hearing, noise at home, years in residence, months in school
# 11 p-CO	Los Angeles - United States *(Cohen et al. 1981)*	Schoolchildren 3-4th. grade 163, mf (1 yr follow-up)	(O) Aircraft noise level at school	(O) Blood pressure	(3,4) Grade in school, ethnic group, social class, family size, obesity, height, hearing, noise at home, migration, months in school, noise abatement
# 12 r-CO (PM)	Erfurt - Germany *(Schulze et al. 1983)*	Adults 20-75 yr 700, mf	(O) Road traffic noise level	(O) Incidence data: Ischaemic heart disease, hypertension	(1) Age, sex, socio-demographic factors
# 13 SU	Erfurt - Germany *(Schulze et al. 1983)*	Adults 20-75 yr 700, mf	(O) Road traffic noise level	(O) Purchase of antihypertensive and cardiovascular drugs from pharmacies	(1) Age, sex, socio-demographic factors
# 14 CS	Amsterdam - The Netherlands *(Knipschild et al. 1984)*	Adults 41-43 yr 2878, mf	(O,S) Road traffic noise level, noise annoyance	(O) Blood pressure, hypertension	(2) Years in residence, sex, socio-economic factors
# 15 CS	Luebeck - Germany *(Hense et al. 1989; Herbold et al. 1989)*	Adults 30-69 yr 2359, mf	(O,S) Road traffic noise level, subjective rating of type of road	(O) Blood pressure, hypertension	(3) Age, sex, body mass index, alcohol consumption, education, employment status, years in residence, room orientation
# 16 p-CO	Bonn - Germany *(Eiff et al. 1987; Otten et al. 1990)*	Adults 20-35 yr 192, mf (3 yrs follow-up)	(O) Road traffic noise level	(O) Blood pressure	(2) Sex, migration, weight, years in residence
# 17 CS	Groningen, Twenthe, Leeuwarden, Amsterdam, - The Netherlands *(Altena and et al. 1989; Pulles et al. 1990)*	Adults 22-55 yr 829, mf	(O) Road traffic noise level, military aircraft noise level	(O) Blood pressure, ischaemic heart disease	(3) Age, sex, smoking, relative body weight, family history of hypertension, employment status, alcohol consumption, shift work, use of contraceptives, treatment of hypertension, blood cholesterol

Table A1 - continued.

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 18 CS	Village near Erfurt, - Germany *(Wölke et al. 1990)*	Adults All ages 352, mf	(O) Road traffic noise level	(O) Contact rates due to cardiovascular diseases and hypertension	(1) Age, sex, socio-economic status, social activities
# 19 r-CO	Village near Erfurt, - Germany *(Wölke et al. 1990)*	Adults All ages 139, mf 5 yrs follow-up	(O) Road traffic noise level	(O) Contact rates due to cardiovascular diseases and hypertension	(1) Age, sex, socio-economic status, social activities
# 20 CS	Münsterland - Germany *(Ising et al. 1990)*	Children 9-13 yr 94, mf	(O) Military aircraft noise Low altitude flight zones	(O) Blood pressure, heart rate	(2) Age, sex
# 21 CS	Franken - Germany *(Ising et al. 1990; Schulte and Otten 1991)*	Children 9-13 yr 433, mf	(O) Military aircraft noise Low altitude flight zones	(O) Blood pressure, heart rate	(2) Age, sex
# 22 CS	Flight zones - Germany *(Ising et al. 1991a; Ising et al. 1991b)*	Children 12-17 yr 467, mf	(O) Military aircraft noise Low altitude flight zones	(O) Blood pressure, heart rate	(3) Age, sex, body mass index
# 23 SU	Flight zones - Germany *(Schulte and Otten 1993b)*	Adults 20-60 yr 7189, mf	(O) Military aircraft noise Low altitude flight zones	(S) Self-reported treatment for hypertension	(1) Sex
# 24 CS	Muensterland - Germany *(Schulte and Otten 1993b)*	Adults 20-60 yr 413, mf	(O) Military aircraft noise Low altitude flight zones	(O) Blood pressure, hypertension	(2) Sex, smoking
# 25 CS	Franken - Germany *(Schulte and Otten 1993b)*	Adults 40-60 yr 424, mf	(O) Military aircraft noise Low altitude flight zones	(O) Blood pressure, hypertension	(2) Sex, smoking
# 26 CS	Westphalia - Germany *(Schmeck 1991; Schmeck and Poustka 1993)*	Children 4-17 yr 376, mf	(O) Military aircraft noise Low altitude flight zones	(O) Blood pressure, heart rate, skin conductivity	(1,2) Sex, psycho-social factors, socio-economical status
# 27 CS	Caerphilly - United Kingdom *(Babisch and Gallacher 1990; Babisch et al. 1993a; Babisch et al. 1988)*	Adults 45-59 yr 2512, m	(O) Road traffic noise level	(O) Blood pressure, ECG ischaemia, angina pectoris, myocardial infarction, ischaemic heart disease	(3) Age, social class, body mass index, employment status, marital status, smoking, family history of IHD, physical activity during leisure, pre-existing diseases

Table A1 - continued.

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 28 CS	Speedwell - United Kingdom *(Babisch and Gallacher 1990; Babisch et al. 1993a; Babisch et al. 1993b)*	Adults 45-63 yr 2348, m	(O) Road traffic noise level	(O) Blood pressure, ECG ischaemia, angina pectoris, myocardial infarction, ischaemic heart disease	(3) Age, social class, body mass index, smoking, family history of IHD, physical activity at leisure, pre-existing diseases
# 29 CS	Tyrol - Austria *(Lercher 1992b)*	Children 8-12 yr 796, mf	(O) Distance from highway	(O) Blood pressure	(1) Age, sex
# 30 CS	Tyrol - Austria *(Lercher 1992a; Lercher 1996; Lercher and Kofler 1993; Lercher and Kofler 1995)*	Adults 25-65 yr 1989, mf	(O,S) Road traffic noise level, noise annoyance	(O) Blood pressure, hypertension, angina pectoris, hypotension, myocardial infarction	(1,3) Age, sex, education, relative body weight, life-style, working conditions, socio-demographic factors, window opening
# 31 CS	Southern Taiwan - Taiwan *(Wu et al. 1993)*	Children 7-12 yr 1050, mf	() Mute-deaf vs. normal hearing	(O) Blood pressure	(3) Age, sex, body mass index,
# 32 p-CC	Berlin - Germany *(Babisch et al. 1992; Babisch et al. 1994)*	Adults 41-70 yr 243, m	(O) Road traffic noise level	(O) Myocardial infarction	(3) Age, body mass index, smoking, employment status, education, social class, work noise, room orientation, years in residence
# 33 p-CC	Berlin - Germany *(Babisch et al. 1992; Babisch et al. 1994)*	Adults 31-70 yr 4035, m	(O,S) Road traffic noise level, noise annoyance	(O) Myocardial infarction	(3) Age, body mass index, smoking, employment status, education, social class, work noise, shift work, marital status, area, room orientation, years in residence
# 34 CS	Berlin - Germany *(Babisch et al. 1992; Babisch et al. 1994; Wiens 1995)*	Adults 31-70 yr 2193, m	(O,S) Road traffic noise level, noise annoyance	(S) Self-reported treatment of myocardial infarction and hypertension	(3) Age, body mass index, smoking, education, social class, room orientation, years in residence
# 35 GP (CS)	General population - Germany *(Bellach et al. 1995; Müller et al. 1994)*	Adults 40-65 yr 1002, mf	(S) Noise at home, noise annoyance	(S) Hypertension, angina pectoris, myocardial infarction	(3) Age, sex, overweight, social class, smoking, employment status, alcohol consumption, physical activity, neuroticism, education, marital status
# 36 CS	Munich - Germany *(Evans et al. 1995)*	Schoolchildren 3-4th. grade 135, mf	(O) Aircraft noise level	(O) Blood pressure	(4) Age, social class, ponderosity (body fat)

Table A1 - continued.

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 37 CS	Bratislava - Slovak Republic *(Regecová and Kellerová 1995)*	Preschool children 3-7 yr 1542, mf	(O) Road traffic noise level	(O) Blood pressure, heart rate	(2) Age, height, weight, Quetelet-index, child's position in the family (first-born), familial social care
# 38 CS	Tokyo - Japan *(Yoshida et al. 1997)*	Adults 20-60 366, f	(O) Road traffic noise level	(S) Symptoms including heart disease and hypertension	(1) Age, type of housing, duration of residence
# 39 CO	Munich - Germany *(Evans et al. 1998)*	Schoolchildren 3-4th. grade 217, mf 2 yrs follow-up	(O) Aircraft noise level	(O) Blood pressure	(4) Age, socioeconomic status, hearing test, ponderosity
# 40 CS	Sydney - Australia *(Morrell et al. 1998; Morrell et al. 2000)*	Primary school children ≥ 3th. grade 1230, mf	(O) Aircraft noise level	(O) Blood pressure	(3) Age, height, weight, gender, adiposity, child activity, child and family history of high blood pressure, other noise sources, eating behaviour, language background, cluster sampling, type of housing, pulse rate
# 41 p-CO	Sydney - Australia *(Morrell et al. 2000)*	Primary school children ≥ 3th. grade 628, mf	(O) Change in aircraft noise level	(O) Blood pressure	(3) Height, weight, skin-fold thickness, physical activity, family history of high blood pressure, baseline blood pressure, ambient temperature at time of blood pressure measurement
# 42 p-CO	Caerphilly - United Kingdom *(Babisch et al. 1999)*	Adults 45-59 yr 2512, m 10 yrs follow-up	(O) Road traffic noise level	(O) Ischaemic heart disease (IHD)	(3) Age, social class, body mass index, employment status, smoking, physical activity during leisure, family history of IHD, prevalence of IHD, pre-existing diseases, migration
# 43 p-CO	Speedwell - United Kingdom *(Babisch et al. 1999)*	Adults 45-63 yr 2348, m 10 yrs follow-up	(O) Road traffic noise level	(O) Ischaemic heart disease (IHD)	(3) Age, social class, body mass index, smoking, physical activity during leisure, family history of IHD, prevalence of IHD, pre-existing diseases, migration
# 44 p-CO	Caerphilly, Speedwell (pooled) - United Kingdom *(Babisch et al. 2003b; Babisch et al. 1999)*	Adults 45-63 yr 3950, m 6 yrs follow-up	(O,S) Road traffic noise level, noise annoyance, noise disturbance	(O) Ischaemic heart disease (IHD)	(3) Age, social class, body mass index, smoking, physical activity during leisure, family history of IHD, prevalence of IHD, pre-existing diseases, migration, room orientation, window opening, years in residence

Table A1 - continued.

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 45 SU	Paris - France (Vallet et al. 1999)	Adults 628, mf in-patients of 7 doctors for 1 week	(O) Aircraft noise contour (high vs. low)	(O) High blood pressure	(1,3) Age, gender, income, education, type of housing, family status, weight, tobacco consumption, alcohol and drug intake, doctor
# 46 CS	Sollentuna - Sweden *(Bluhm et al. 1998)*	Adults 19-80 yr 759, mf	(O) Road traffic noise level, railway noise level	(S) Self reported medical diagnosis of hypertension during the last 5 years	(1,3) Age, sex, ethnic background, education level, employment status, type of housing, smoking, outdoor exercise, fruit and vegetable consumption
# 47 CS	Sollentuna -Sweden *(Bluhm et al. 2001)*	Adults 19-80 yr 631, mf	(O) Road traffic noise level	(S) Self reported medical diagnosis of hypertension during the last 5 years	(1,3) Age, sex, ethnic background, education level, employment status, type of housing, smoking, outdoor exercise, fruit and vegetable consumption
# 48 EC	62 municipalities around Amsterdam - Netherlands *(Franssen et al. 1999; Franssen et al. 2002)*	Adults general population	(O) Distance from airport (postcode)	(O) Hospital admission rates due to cardiovascular diseases	(2) Age, sex
# 49 CS	Okinawa - Japan *(Matsui et al. 2001; Matsui et al. 2004)*	Adults 20-79 yr 28781, mf	(O) Aircraft noise level	(O) Blood pressure, clinical hypertension	(3) Age, sex, body mass index
# 50 CS	Stockholm - Sweden *(Rosenlund et al. 2001)*	Adults 19-80 yr 2959, mf	(O) Aircraft noise level	(S) Self-reported medical diagnosis of hypertension	(3) Age, sex, smoking, education, physical activity, fruit consumption, type of housing
# 51 TS	Madrid - Spain (Tobias et al. 2001)	In-patients at a major hospital 3 yrs (1096 days)	(O) Variations in noise level at 5 stations in the city (ecological epproach)	(O) Emergency admissions for all causes and specific causes including circulatory	(3) Air pollutants (SO_2, TSP, NO_x, NO_2, O_3), lag models, temperature, humidity, day of the week, influenza epidemics, seasonality, autoregressive terms
# 52 CS	Inn Valley, Tyrol - Austria *(Lercher et al. 2000)*	Adults 20-75 yr 572, mf	(O) Road traffic and railway noise	(O) Blood pressure, clinical hypertension	(3) Age, sex, education, satisfaction with the environment, type of housing, coping with the noise, body mass index, weather sensitivity, family history of hypertension, shift work

Table A1 - continued.

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 53 CS	Inn Valley, Tyrol - Austria *(Evans et al. 2001; Lercher et al. 2002)*	Children 4th grade 115, mf	(O) Road traffic and railway noise	(O) Blood pressure	(1) Age, sex, mother's education, family size, people/room, type of housing, body mass index
# 54 CS	Pancevo - Serbia *(Belojevic and Saric-Tanaskovic 2002)*	Adults population >20 yr 2874, mf	(S) Road traffic noise annoyance	(S) Self-reported treatment of hypertension and medically diagnosed myocardial infarction	(3) Age, sex, body mass index, smoking
# 55 CO	204 municipalities - Netherlands *(Hoek et al. 2002)*	Adults 55-69 yr 4492, mf 8 yrs follow-up	(O) Distance from major road	(O) All-cause mortality, specific mortality including cardiopulmonary mortality	(3) Age, sex, smoking, school education, blue collar job, neighbourhood socioeconomic score, body mass index, alcohol consumption, food consumption (fat, vegetables, fruit), air pollutants (black smoke, nitrogen dioxide)
# 56 CS	Fukuoka - Japan *(Goto and Kaneko 2002)*	Adults 407, f	(O) Aircraft noise zones	(O) Blood pressure	(2) Age, smoking, drinking, salt intake, anti-hypertensive medication
# 57 r-CO	Fukuoka - Japan *(Goto and Kaneko 2002)*	Adults 183, f 8 yr follow-up	(O) Aircraft noise zones	(O) Blood pressure	(2) Age, smoking, drinking, salt intake, anti-hypertensive medication
# 58 CS	Berlin Spandau - Germany *(Maschke 2003; Maschke et al. 2003a)*	Adults 16-90 yr 1718, mf	(O,S) Road traffic noise level, aircraft noise contour, noise annoyance	(O) Period prevalence (2 yr) of medical examinations because of hypertension, angina pectoris, myocardial infarction, assessed in a clinical interview	(3) Age, sex, smoking, alcohol consumption, physical activity at work and at leisure, hearing loss, body mass index, socio-economic index, noise sensitivity, season of the examination
# 59 CS	62 municipalities Amsterdam - Netherlands *(Franssen et al. 2004)*	Adults population >18 yr 11812, mf	(O) Aircraft noise level, aircraft noise annoyance	(S) Medication for cardiovascular diseases or high blood pressure, sleep medication	(3) Age, sex, educational level, smoking, urbanisation, ethnicity
# 60 CS	Stockholm (preliminary results) - Sweden *(Bluhm et al. 2004)*	Adults 45-65 417, m	(O) Aircraft noise level, aircraft noise annoyance	(S) Angina pectoris treatment, diagnosed myocardial infarction, diagnosed hypertension, use of anti-hypertension drugs	(2) Age, smoking, noise sensitivity, hearing deficits

Table A1 - continued.

Number of study (as given in the text) **Type** of study [1]	**Location** Town, Country **References**	**Subjects** Age Number Sex [2]	**Exposure** O=objective (noise level outdoors) S=subjective (annoyance)	**Outcome** O=objective (measurement or clinical interview) S=subjective (self-reported in a postal questionnaire survey)	**Covariates** 0=no control, 1=group comparison, 2=stratification or standardisation, 3=model adjusted (regression), 4=matching
# 61 p-CC	Berlin - Germany (Babisch 2004b; Babisch et al. 2005; Babisch et al. 2003a)	Adults 20-69 yr 4115, mf	(O,S) Road traffic noise level, noise annoyance	(O) Myocardial infarction	(3) Age, smoking, body mass index, employment status, marital status, shift work, educational level, noise sensitivity, diabetes mellitus, high blood pressure, family history of myocardial infarction, high cholesterol
# 62 CS	8 cities - Europe LARES Group (Niemann and Maschke 2004)	Adults adult population 5442, mf	(S) Noise annoyance, sleep disturbance	(S) Self-reported multiple morbidity including hypertension and myocardial infarction	(3) Age, sex, socio-economic status, smoking, body mass index, alcohol consumption, sports, size of city, marital status, housing problems, established EU citizen

1) SU = survey, EC = ecological study, PM = proportional morbidity study, CS = cross-sectional study, GP = general population follow-up study, CC = case-control study, CO = cohort study; TS = time series study; p- = prospective, r- = retrospective, t- temporal panel
2) m = males, f = females, mf = males and females

Table A2. Studies on effects of community noise on mean blood pressure readings

Study		Mean blood pressure difference [1]		Sound level (outdoors) [2]	Significance
Number	Location	Systolic [mmHg]	Diastolic [mmHg]	dB(A)	Sytolic / Diastolic
Children					
# 01	Halle	+9 to +16 (age)	+12 to +16 (age)	$L_{phon,mean}$ indoor: quiet vs. 70	unknown
# 03	Russian airports	higher rate of blood pressure abnormalities		Close to vs. far from airport	
# 10	Los Angeles	+3 to +7 (years of enrolment)	+3 to +4 (years of enrolment)	$L_{max,mean}$ indoor: 56 vs. 74	$p < 0.05$ / $p < 0.10$
# 11	Los Angeles	-2 to +7 (years of enrolment)	+1 to +7 (years of enrolment)	$L_{max,mean}$ indoor: 57 vs. 79	n.s. / n.s.
# 20	Münsterland [3]	-1	-1	150 m area vs. 75 m area	n.s / n.s
# 21	Franken [3]	0 to +9 (sex)	0 to +3 (sex)	150 m area vs. 75 m area	$p < 0.001$ / $p < 0.01$
# 22	Flight zones in Germany [3]	0 to +2 (sex, area)	0 to +2 (sex, area)	Control area vs. 75 m area	n.s. / n.s.
# 26	Westphalia [3]	+1 to +2 (sex)	-1 to 2 (sex)	Low vs. High	n.s. / n.s.
# 29	Tyrol	-2	-2	L_{24h}: <50 vs. >= 64	n.s./ n.s.
# 31	Taiwan	-3 to 10 (age)	-1 to 12 (age)	Mute-deaf vs. normal hearing	$p < 0.001$./ $p < 0.001$
# 36	Munich	2	0	L_{24}h: 59 vs. 68	$p < 0.10$ / n.s.
# 37	Bratislava	+1 to +5 (Kindergarten/Residence)	+2 to +5 (Kindergarten/Residence)	L_{dn}: <60 vs. >70	$p < 0.001$ / $p < 0.001$
# 39	Munich	D: +4	D: +2	L_{24h}: 53 vs.62	$p < 0.01$ / $p < 0.10$
# 40	Sydney	-1	-1	ANEI: 15 to 45	n.s. / n.s.
# 41	Sydney	0	0	Change in ANEI: -5 to +5	n.s. / n.s.
# 53	Inn Valley	2	0	L_{dn}: <50 vs. > 60	$p < 0.10$ / n.s.

Table A2 – continued.

Study		Mean blood pressure difference [1]		Sound level (outdoors) [2]	Significance
Number	Location	Systolic [mmHg]	Diastolic [mmHg]	dB(A)	Sytolic / Diastolic
Adults					
# 04	Munich	2	3	$L_{max,mean}$: <87 vs. >95	n.s. / n.s.
# 09	Bonn	1	1	L_{day}: <60 vs. >65	n.s. / n.s.
# 14	Amsterdam	-2	-1	L_{day}: <65 vs. >=65	$p < 0.05$ / $p < 0.10$
# 15	Luebeck	-1 to +1 (sex)	-1 to +2 (sex)	L_{day}: <=60 vs. >65	n.s. / $p < 0.10$
# 16	Bonn	D: -5 to +8 (sex)	D: -1 to +3 (sex)	L_{day}: <55 vs. >63	n.s. / n.s.
# 17	Groningen, Twenthe, ...	-1 to +5 (type of noise)	-1 to 2 (type of noise)	Approx. L_{den}: <=60 vs. >65	$p < 0.05$ / n.s.
# 24	Münsterland [3]	-2 to +1 (flight zones)	-1	Control area vs. Flight zones	n.s. / n.s.
# 25	Franken [3]	-4 to +2 (flight zones)	-2 to +1 (flight zones)	Control area vs. 75 m area	n.s. / n.s.
# 27	Caerphilly	1	-1	L_{day}: 51-55 vs. 66-70	n.s. / n.s.
# 28	Speedwell	-3	-1	L_{day}: 51-55 vs. 66-70	n.s. / $p < 0.05$
# 30	Tyrol	-5 to -3 (annoyance)	-3 to -1 (annoyance)	L_{24h}: <50 vs. >= 64	$p < 0.05$ / $p < 0.05$
# 52	Tyrol	n. s.	n. s.	L_{dn}: <50 vs. >60	$p < 0.05$ / $p < 0.05$
# 56	Fukuoka	4	1	Approx. L_{dn}: <60 vs. >= 70	n.s / n.s
# 57	Fukuoka	D: 0	D: -4	Approx. L_{dn}: <60 vs. >= 70	n.s / n.s

D = Difference in change of blood pressure

1) = High exposure minus low exposure (extreme group comparison)

2) = Outdoor noise level if not otherwise indicated

3) = Low-altitude military aircraft noise

Table A3. Studies on effects of community noise on the prevalence of hypertension

Study (Number, Location) **Relative Risk** [1) 2)] **(95% confidence interval)**		**Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})**						
		46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 05	N = 5828							
Amsterdam (aircraft)								
Clinical hypertension - mf			1.0		1.73 (1.38-2.16)		---	---
Clinical hypertension - m			1.0		1.81 (1.23-2.66)		---	---
Clinical hypertension - f			1.0		1.68 (1.28-2.22)		---	---
Hypertension - mf			1.0		1.47 (1.24-1.73)		---	---
Hypertension - m			1.0		1.49 (1.07-2.07)		---	---
Hypertension - f			1.0		1.46 (1.20-1.76)		---	---
# 08	N = 1741							
Doetinchem (road)								
Clinical hypertension - f		---	---	1.0	---	0.93 (0.65-1.34)	---	---
# 09	N = 926							
Bonn (road)								
Hypertension - mf		---	1.0		---	1.52 (1.15-1.02)		---
Hypertension - m		---	1.0		---	1.44 (0.98-2.10)		---
Hypertension - f		---	1.0		---	1.63 (1.07-2.48)		---
# 12	N = 700							
Erfurt (road)								
Clinical hypertension - mf		---	---	---	1.0	---	2.40 ($p < 0.05$)	---

Table A3 – continued.

Study (Number, Location) Relative Risk [1) 2)] (95% confidence interval)		Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})						
		46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 14	N = 2878							
Amsterdam (road)								
Clinical hypertension - mf		---	1.0	0.74 (0.53-1.06)	0.83 (0.58-1.19)	0.59 (0.37-0.95)	1.03 (0.68-1.58)	0.11 (0.00-1.19)
				1.0			0.87 (0.65-1.16)	
Clinical hypertension - m		---		1.0			0.93 (0.61-1.45)	
Clinical hypertension - f		---		1.0			0.83 (0.56-1.24)	
# 15	N = 2359							
Luebeck (road)								
Clinical hypertension - m				1.0	1.25 (0.84-1.86)	1.05 (0.74-1.49)		---
Clinical hypertension - f				1.0	0.84 (0.57-1.25)	0.52 (0.35-0.76)		---
# 18	N = 253							
Erfurt (road)								
Clinical hypertension - mf		---	---	1.0	---	---	2.35 (1.37-4.05)	---
# 30	N = 1985							
Tyrol (road)								
Hypertension - mf		---	1.0			0.83 (0.64-1.10)		---
Hypertension - mf		---		1.0		0.81 (0.61-1.09)		---
# 34	N = 2193							
Berlin (road)								
Hypertension - m		---		1.0	0.92 (0.58-1.45)	1.10 (0.77-1.58)	0.86 (0.57-1.28)	1.55 (0.82-2.93)
		---		1.0		1.00 (0.77-1.57)		1.00 (0.71-1.42)

Table A3 – continued.

Study (Number, Location) **Relative Risk** [1) 2)] **(95% confidence interval)**	**Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})**						
	46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 38 N = 366							
Tokyo (road)							
Hypertension - f	1.0		0.60 (030-1.21)	0.63 (0.28-1.42)	1.09 (0.51-2.33)	0.53 (0.16-1.52)	---
Hypertension - f	1.0		0.61 (0.33-1.14)		0.83 (0.41-1-65)		---
# 46 N = 481							
Sollentuna (rail)							
Hypertension - mf	1.0	---	0.8 (0.3-1.8)		---	---	---
# 46 N = 658							
Sollentuna (road)							
Hypertension - mf	1.0 [3)]	1.8 (1.0-3.2)			---	---	---
Hypertension - m	1.0 [3)]	1.0 (0.4-2.3)			---	---	---
Hypertension - f	1.0 [3)]	3.3 (1.4-7.3)			---	---	---
# 47 N = 631							
Sollentuna (road) (n = 281)							
Hypertension - mf	2.0 (0.7-5.7) [4)]	2.0 (0.8-5.1)	3.0 (1.1-8.4)		---	---	---
Hypertension - mf	1.0 [3)]	1.47 (0.83-2.61)			---	---	---
- (>10 y in residence)	1.0 [3)]	2.4 (1.09-5.39)			---	---	---
Hypertension - m	1.0 [3)]	1.4 (0.6-3.2)			---	---	---
Hypertension - f	1.0 [3)]	1.8 (0.8-4.1)			---	---	---
# 49 N = 28781							
Okinawa (aircraft)							
Clinical hypertension - mf	---	1.0		1.1 (1.0-1.2)	1.1 (0.9-1.2)	1.37 (1.19-1.57)	---
Clinical hypertension (borderline) - mf	---	1.0		1.0 (1.0-1.1)	1.1 (1.0-1.2)	1.17 (1.05-1.30)	---

Table A3 – continued.

Study (Number, Location) Relative Risk [1) 2)] (95% confidence interval)	Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})						
	46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 50 N = 2959							
Stockholm (aircraft)							
Hypertension - mf	1.0	1.1 (0.7-1.9)	1.5 (0.9-2.5)	2.1 (0.8-5.3)	---	---	---
Hypertension - mf	1.0		1.6 (1.0-2.5)		---	---	---
Hypertension - m	1.0		1.7 (0.9-3.3)		---	---	---
Hypertension - f	1.0		1.4 (0.8-2.8)		---	---	---
# 58 N = 1351							
Berlin Spandau (road) (n = 279)							
Hypertension - mf (day, living room)	1.0		1.29 (0.75-2.24)	1.12 (0.60-2.09)	1.51 (0.78-2.93)	---	---
Hypertension - mf (night, bedroom)	1.0	1.66 (1.07-2.56)	1.88 (1.10-3.22)		---	---	---
- (windows open)	1.0	4.53 (1.02-20.2)	6.13 (1.28-29.2)		---	---	---
# 58 N = 1351							
Berlin Spandau (aircraft) [5)]							
Hypertension - mf	1.0			1.09 (0.80-1.48)	1.51 (0.55-4.16)	---	---
# 60 N = 417							
Stockholm (aircraft)							
Hypertension - m	1.0		1.64 (1.21-2.21)		---	---	---

m = males, f = females, mf = males and females

1) Calculated as risk ratio, odds ratio or proportional morbidity ratio

2) Clinical hypertension = blood pressure measurement, Hypertension = clinical interview or self-administered questionnaire

3) Reference category includes 40-45 dB(A)

4) Reference category is 40-45 dB(A)

5) Approximation (contours according to German Aircraft Noise Act using equivalence parameter q=4 based on older prognostic data)

Table A4. Studies on effects of subjective responses to noise on the prevalence of hypertension

Study (Number, Location) **Relative Risk** [1) 2)] **(95% confidence interval)**		**Noise Annoyance/Disturbance/Rating [Categories]** [3)]			
		1	**2**	**3**	**4+5**
# 14	N = 2878				
Amsterdam (road)					
Clinical hypertension - mf		1.0		0.83 (0.57-1.17)	
# 15	N = 2359				
Luebeck (road)					
Clinical hypertension. - m		1.0		1.32 (0.98-1.79)	
Clinical hypertension - f		1.0		0.75 (0.56-1.02)	
# 30	N = 1986				
Tyrol (road)					
Hypertension - mf		1.0		0.92 (0.72-1.20)	
# 34	N = 2193				
Berlin (road)					
Hypertension - m (road)		1.0		1.22 (0.98-1.53)	1.29 (1.05-1.60)
Hypertension - m (aicraft)		1.0		1.21 (0.94-1.56)	1.06 (0.84-1.34)
# 35	N = 1002				
German general population sample (road)					
Hypertension - mf		1.0		0.92 (0.60-1.42)	
Hypertension - m		1.0		1.18 (0.64-2.19)	
Hypertension - f		1.0		0.90 (0.51-1.61)	
Hypertension - mf (sleep disturbance)		1.0		2.32 (1.16-4.65)	
# 54	N = 2874				
Pancevo (road)					
Hypertension - m		1.0		1.2 (0.8-1.8)	1.8 (1.0-2.4)
Hypertension - f		1.0		0.9 (0.6-1.3)	1.1 (0.8-1.7)

Table A4 – continued.

Study (Number, Location) Relative Risk [1) 2)] (95% confidence interval)		Noise Annoyance/Disturbance/Rating [Categories] [3)]			
		1	**2**	**3**	**4+5**
# 58	N = 1351				
Berlin Spandau (road)					
Hypertension - mf (day)		1.0		0.97 (0.62-1.52)	
Hypertension - mf (night)		1.0		1.17 (0.71-1.92)	
# 58	N = 1351				
Berlin Spandau (aircraft)					
Hypertension - mf (day)		1.0		1.18 (0.83-1.67)	
Hypertension - mf (night)		1.0		1.30 (0.81-2.09)	
# 62	N = 5442				
8 European cities (general traffic noise)	n = 936				
Hypertension - mf		1.0	1.1 (n.s.)	1.6 ($p < 0.05$)	
- elderly		1.0		1.15 (n.s.)	
# 62	N = 5442				
8 European cities (general neighbourhood noise)	n = 936				
Hypertension - mf		1.0	1.3 ($p < 0.05$)	1.7 ($p < 0.05$)	
- elderly		1.0		0.9 (n.s.)	
Hypertension – mf (noise-induced sleep disturbance)		1.0		1.5 ($p < 0.05$)	
- elderly		1.0		0.9 (n.s.)	

m = males, f = females, mf = males and females

1) Calculated as risk ratio, odds ratio or proportional morbidity ratio

2) Clinical hypertension = blood pressure measurement, Hypertension = interview or self-administered questionnaire

3) Categories: 1 = never, not at all, dead end street, not affected; 2 = seldom, a little, side street, not affected; 3 = sometimes, moderate, busy road, affected; 4+5 = often+always, much+very much, major trunk road, affected

Table A5. Studies on effects of community noise on the prevalence of ischaemic heart disease

Study (Number, Location) **Relative Risk** [1] **(95% confidence interval)**	**Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})**						
	46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 05 N = 5828							
Amsterdam (aircraft)							
Angina pectoris - mf	---	1.0		1.11 (0.82-1.50)		---	---
Angina pectoris - m	---	1.0		1.49 (0.89-2.51)		---	---
Angina pectoris - f	---	1.0		0.95 (0.65-1.38)		---	---
Heart trouble mf	---	1.0		1.40 (0.98-2.01)		---	---
Heart trouble - m	---	1.0		1.16 (0.73-1.82)		---	---
Heart trouble - f	---	1.0		1.94 (1.07-3.49)		---	---
ECG ischaemia - mf	---	1.0		1.16 (0.92-1.47)		---	---
ECG ischaemia - m	---	1.0		1.05 (0.71-1.56)		---	---
ECG ischaemia - f	---	1.0		1.23 (0.92-1.65)		---	---
# 06 N = 18025							
Amsterdam (aircraft)							
Cardiovascular diseases [2] - mf	1.0	approx. 0.5	approx. 1.3	approx. 2.0	---	---	---
	1.0		1.80 (1.25-2.59)		---	---	---
Cardiovascular diseases [2] - m	1.0		1.91 (1.08-3.40)		---	---	---
Cardiovascular diseases [2] - f	1.0		1.72 (1.07-2.70)		---	---	---
# 08 N = 1741							
Doetinchem (road)							
Angina pectoris - f	---	---	1.0	---	0.65 (0.27-1.55)	---	---
ECG ischaemia - f	---	---	1.0	---	1.06 (0.70-1.59)	---	---
# 09 N = 931							
Bonn (road)							
Myocardial infarction - mf	---	1.0		---	1.30 (0.44-3.56)		---

Table A5 – continued.

Study (Number, Location) Relative Risk [1] (95% confidence interval)	Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})						
	46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 17 N = 829							
Groningen, Twenthe, Leeuwarden, Amsterdam (aircraft)							
Ischaemic heart diseases - mf	1.0	0.77 (0.36-1.64)	1.12 (0.55-2.28)	1.48 (0.74-2.94)	1.13 (0.51-2.48)	0.92 (0.23-3.71)	---
	1.0		1.45 (0.87-2.41)		1.23 (0.63-2.39)		---
# 17 N = 829							
Groningen, Twenthe, Leeuwarden, Amsterdam (road)							
Ischaemic heart diseases - mf	1.0	1.03 (0.45-2.33)	0.98 (0.40-2.41)	0.51 (0.19-1.40)	0.52 (0.20-1.35)	0.54 (0.16-1.81)	---
	1.0		0.75 (0.39-1.45)		0.52 (0.25-1.08)		---
# 27 N = 2512							
Caerphilly (road)							
Angina pectoris - m	---	1.0	0.94 (0.51-1.73)	1.17 (0.73-1.87)	0.52 (0.19-1.44)	---	---
Myocardial infarction - m	---	1.0	1.00 (0.58-1.70)	0.90 (0.56-1.44)	1.22 (0.63-2.33)	---	---
ECG ischaemia - m	---	1.0	0.51 (0.16-1.65)	1.10 (0.56-2.20)	1.24 (0.44-3.50)	---	---
Ischaemic heart disease - m	---	1.0	0.95 (0.61-1.46)	1.10 (0.77-1.56)	1.15 (0.67-1.96)	---	---
# 28 N = 2348							
Speedwell (road)							
Angina pectoris - m	---	1.0	1.10 (0.67-1.80)	1.09 (0.64-1.85)	1.10 (0.65-1.87)	---	---
Myocardial infarction - m	---	1.0	1.02 (0.57-1.84)	1.22 (0.70-2.11)	1.07 (0.59-1.93)	---	---
ECG ischaemia - m	---	1.0	0.88 (0.39-1.97)	0.95 (0.43-2.12)	1.44 (0.71-2.92)	---	---
Ischaemic heart disease - m	---	1.0	0.96 (0.63-1.45)	1.23 (0.81-1.86)	1.25 (0.82-1.89)	---	---

Table A5 – continued.

Study (Number, Location)		Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})						
Relative Risk [1] **(95% confidence interval)**		**46-50**	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 30	N = 1985							
Tyrol (road)								
Angina Pectoris - mf		---	1.0	2.01 (1.18-3.44)				---
Myocardial Infarction - mf		---	1.0	0.96 (0.50-1.85)				---
Angina Pectoris - mf		---	1.0		2.13 (1.23-3.69)			---
Myocardial Infarction - mf		---	1.0		0.77 (0.37-1.62)			---
# 34	N = 2193							
Berlin (road)								
Myocardial infarction - m			1.0		0.75 (0.32-1.74)	0.87 (0.47-1.64)	1.08 (0.57-2.06)	1.41 (0.54-3.67)
			1.0		0.83 (0.50-1.38)		1.16 (0.67-2.01)	
# 38	N = 366							
Tokyo (road)								
Heart disease - f		1.0		2.04 (0.46-9.11)		4.89 (1.12-21.4)		---
# 58	N = 1351							
Berlin Spandau (road)	(n = 279)							
Angina pectoris - mf (day, living room)		1.0		2.57 (0.33-19.7)	3.99 (0.46-34.4)	2.01 (0.20-20.7)	---	---
Angina pectoris - mf (night, bedroom)		1.0	3.07 (0.71-13.2)	3.24 (0.62-16.8)		---	---	---
Myocardial infarction - mf (day, living room)		1.0		0.83 (0.09-7.32)	1.41 (0.13-15.5)	small sample	---	---
Myocardial infarction - mf (night, bedroom)		1.0	2.28 (0.27-19.2)	1.17 (0.07-20.2)		---	---	---
# 58	N = 1351							
Berlin Spandau (aircraft) [3]								
Angina pectoris - mf			1.0		1.65 (0.83-3.31)	---	---	---
Myocardial infarction - mf			1.0		0.40 (0.05-3.27)	---	---	---

Table A5 – continued.

Study (Number, Location) **Relative Risk** [1)] **(95% confidence interval)**	**Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})**						
	46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 60 N = 417							
Stockholm (aircraft)							
Angina pectoris - m	1.0		0.86 (0.28-2.63)		---	---	---
Myocardial infarction - m	1.0		2.59 (0.93-7.24)		---	---	---

m = males, f = females, mf = males and females

1) Calculated as risk ratio, odds ratio or proportional morbidity ratio

2) Cardiovascular diseases include high blood pressure

3) Approximation (contours according to German Aircraft Noise Act using equivalence parameter q=4 based on older prognostic data)

Table A6. Studies on effects of community noise on the incidence of ischaemic heart disease

Study (Number, Location) **Relative Risk** [1] **(95% confidence interval)**	**Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})**						
	46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 12 N = 700							
Erfurt (road)							
Myocardial infarction - mf	---	---	---	1.0	---	4.40 ($p < 0.05$)	---
# 32 N = 243							
Berlin (road) (n = 155)							
Myocardial infarction - m		1.0		1.48 (0.57-3.85)	1.19 (0.49-2.86)	1.25 (0.41-3.81)	1.76 (0.11-28.8)
		1.0		1.31 (0.66-2.60)		1.31 (0.45-3.77)	
- (≥15 y in residence)		1.0		2.34 (0.48-11.4)	0.98 (0.31-3.00)	0.98 (0.24-3.12)	2.10 (0.12-35.0)
		1.0		1.30 (0.51-3.32)		1.15 (0.32-4.10)	
# 33 N = 4035							
Berlin (road) (n = 2582)							
Myocardial infarction - m		1.0		1.16 (0.82-1.65)	0.94 (0.62-1.41)	1.07 (0.67-1.65)	1.46 (0.77-2.79)
		1.0		1.06 (0.80-1.38)		1.17 (0.81-1.67)	
- (≥15 y in residence)		1.0		1.10 (0.62-1.93)	0.97 (0.61-1.55)	1.17 (0.73-1.88)	1.71 (0.88-3.34)
		1.0		1.02 (0.70-1.47)		1.32 (0.89-1.96)	
# 42 N = 2512							
Caerphilly (road)							
IHD - m (preliminary 5 y follow-up) [2]	---	1.0	1.24 (0.66-2.32)	1.30 (0.77-2.20)	0.52 (0.16-1.69)	---	---
IHD - m (10 y follow-up) [2]	---	1.0	1.07 (0.68-1.68)	0.87 (0.58-1.30)	1.07 (0.60-1.91)	---	---
# 43 N = 2348							
Speedwell (road)							
IHD - m (preliminary 3 y follow-up) [2]	---	1.0	0.60 (0.26-1.49)	1.26 (0.63-2.50)	0.72 (0.31-1.75)	---	---
IHD - m (10 y follow-up) [2]	---	1.0	0.67 (0.42-1.07)	0.76 (0.48-1.22)	0.92 (0.61-1.41)	---	---

Table A6 – continued.

Study (Number, Location) Relative Risk [1] (95% confidence interval)		Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})						
		46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 44	N = 3950							
	(n = 2578)							
Caerphilly + Speedwell (road)								
IHD - m (6 y follow-up) [2]		---	1.0	0.71 (0.46-1.11)	0.68 (0.44-1.03)	1.07 (0.70-1.65)	---	---
- (≥15 y in residence)		---	1.0	0.70 (0.40-1.20)	0.60 (0.35-1.03)	1.20 (0.72-2.03)	---	---
- window orientation		---	1.0	0.82 (0.51-1.31)	0.64 (0.39-1.04)	1.16 (0.73-1.86)	---	---
		---	1.0		0.65 (0.40-1.06)	1.18 (0.74-1.89)	---	---
- - (≥15 y in residence)		---	1.0	0.82 (0.46-1.46)	0.49 (0.25-0.95)	1.30 (0.73-2.32)	---	---
- window orientation + opening		---	1.0	0.69 (0.42-1.12)	0.64 (0.44-1.03)	1.31 (0.78-2.21)	---	---
- - (≥15 y in residence)		---	1.0	0.67 (0.36-1.24)	0.45 (0.20-0.98)	1.59 (0.85-2.97)	---	---
# 61	N = 4115							
Berlin (road)	(n = 2857)							
Myocardial infarction - m			1.0		1.01 (0.77-1.31)	1.13 (0.86-1.49)	1.27 (0.88-1.84)	---
			1.0		1.01 (0.77-1.31)	1.18 (0.93-1.49)		---
- (≥10 y in residence)			1.0		1.17 (0.81-1.69)	1.31 (0.88-1.97)	1.81 (1.02-3.21)	---
			1.0		1.17 (0.81-1.69)	1.45 (1.03-2.05)		---
Myocardial infarction - f			1.0		1.14 (0.70-1.85)	0.93 (0.57-1.52)	0.66 (0.32-1.35)	---
			1.0		1.14 (0.70-1.85)	0.84 (0.55-1.27)		---

m = males, f = females, mf = males and females

1) Calculated as risk ratio, odds ratio or proportional morbidity ratio

2) Preliminary 3 yrs (Speedwell) and 5 yrs (Caerphilly) follow-up refer to phases 1-2, 10 yr follow-up refers to phase 1-3, 6 yr follow-up refers to phase 2-3 (information regarding room orientation and annoyance was only collected during phase 2)

Table A7. Studies on effects of subjective responses to noise on the prevalence or incidence of ischaemic heart disease

Study (Number, Location)		**Noise Annoyance/Disturbance/Rating [Categories]** [2)]			
Relative Risk [1)] **(95% confidence interval)**		**1**	**2**	**3**	**4**
# 30	N = 1986				
Tyrol (road)					
Angina pectoris - mf		1.0		1.32 (0.77-2.24)	
Myocardial infarction - mf		1.0		0.82 (0.44-1.51)	
# 35	N = 1002				
German general population sample (road)					
Angina pectoris - mf		1.0		1.09 (0.73-1.64)	
Angina pectoris - m		1.0		1.81 (1.03-3.16)	
Angina pectoris - f		1.0		0.86 (0.51-1.46)	
Angina pectoris - mf (sleep disturbance)		1.0		1.86 (0.94-3.70)	
Myocardial infarction - mf		1.0		1.04 (0.42-2.53)	
Myocardial infarction - mf (sleep disturbance)		1.0		1.32 (0.34-5.07)	
# 34	N = 2193				
Berlin (road)					
Myocardial infarction - m (road)		1.0		1.01 (0.69-1.46)	1.25 (0.88-1.77)
Myocardial infarction - m (aircraft)		1.0		1.45 (0.98-2.14)	1.08 (0.73-1.59)
# 44	N = 2914				
Caerphilly+Speedwell (road)					
IHD - m (relaxation")		1.0	0.86 (0.64-1.16)	0.89 (0.62-1.27)	1.39 (0.76-2.54)
IHD - m ("conversation")		1.0	0.96 (0.72-1.29)	0.91 (0.64-1.29)	1.23 (0.69-2.18)
IHD - m ("waking up")		1.0	1.10 (0.82-1.47)	1.01 (0.74-1.39)	1.38 (0.79-2.40)
IHD - m ("annoyance")		1.0	0.79 (0.58-1.08)	0.93 (0.68-1.27)	0.95 (0.52-1.75)

Table A7 – continued.

Study (Number, Location)		Noise Annoyance/Disturbance/Rating [Categories] [2]			
Relative Risk [1] **(95% confidence interval)**		**1**	**2**	**3**	**4**
# 54	N = 2874				
Pancevo (road)					
Myocardial infarction - m		1.0		0.6 (0.2-1.2)	1.7 (1.0-2.9)
Myocardial infarction - f		1.0		0.3 (0.2-0.9)	1.0 (0.4-2.0)
# 58	N = 1350				
Berlin Spandau (road)					
Angina pectoris - mf (day)		1.0		0.78 (0.21-2.84)	
Angina pectoris - mf (night)		1.0		1.20 (0.34-4.23)	
Myocardial infarction - mf (day)		1.0		1.27 (0.11-14.2)	
Myocardial infarction - mf (night)		1.0		small sample	
# 58	N = 1350				
Berlin Spandau (aircraft)					
Angina pectoris - mf (day)		1.0		1.12 (0.48-2.65)	
Angina pectoris - mf (night)		1.0		2.12 (0.80-5.61)	
Myocardial infarction - mf (day)		1.0		2.39 (0.47-12.0)	
Myocardial infarction - mf (night)		1.0		1.89 (0.20-18.1)	
# 61	N = 4115				
Berlin (road)					
Myocardial infarction - m (day)		1.0 [3]		1.08	1.12
Myocardial infarction - m (night)		1.0 [3]		1.21	1.33 ($p < 0.05$)
Myocardial infarction - f (day)		1.0 [3]		1.06	1.09
Myocardial infarction - f (night)		1.0 [3]		0.96	0.94

Table A7 – continued.

Study (Number, Location)		Noise Annoyance/Disturbance/Rating [Categories] [2]			
Relative Risk [1] **(95% confidence interval)**		**1**	**2**	**3**	**4**
# 61	N = 4115				
Berlin (aircraft)					
Myocardial infarction - m (day)		1.0 [3]		1.02	1.03
Myocardial infarction - m (night)		1.0 [3]		1.10	1.16
Myocardial infarction - f (day)		1.0 [3]		1.28	1.44
Myocardial infarction - f (night)		1.0 [3]		1.64	2.10 (p < 0.05)
# 62	N = 5442				
8 European cities (general traffic noise)	n = 936				
Heart attack - mf		1.0	1.1 (n.s.)	1.4 (n.s.)	
- elderly		1.0		0.77 (n.s.)	
# 62	N = 5442				
8 European cities (general neighbourhood noise)	n = 936				
Heart attack - mf		1.0	1.4 (n.s)	2.0 (n.s.)	
- elderly		1.0		1.2 (n.s.)	
Heart attack – mf (noise-induced sleep disturbance)		1.0		1.4 (n.s.)	
- elderly		1.0		1.2 (n.s.)	

m = males, f = females, mf = males and females, IHD = ischaemic heart disease

1) Calculated as risk ratio, odds ratio or proportional morbidity ratio

2) Categories: 1 = never, not at all, dead end street, not affected; 2 = seldom, a little, side street, not affected; 3 = sometimes, moderate, busy road, affected; 4 = often+always, much+very much, major trunk road, affected

3) Re-calculated on the basis of a continuous logistic regression coefficient (odds ratio per category)

Table A8. Studies on effects of community noise on medication / drug consumption

Study (Number, Location) Relative Risk [1] (95% confidence interval)		Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})						
		46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 05	N = 5828							
Amsterdam (aircraft)								
Cardiovascular drugs - mf		---	1.0		1.39 (1.12-1.72)		---	---
Cardiovascular drugs - m		---	1.0		1.11 (0.74-1.66)		---	---
Cardiovascular drugs - f		---	1.0		1.62 (1.25-2.08)		---	---
# 07	pharmacies				(5th year vs. 1st year)		---	---
Amsterdam (aircraft)							---	---
Hypnotics - mf		1.0			approx. 1.5 [2]		---	---
Sedatives - mf		1.0			approx. 1.6 [2]		---	---
Antacids - mf		1.0			approx. 1.7 [2]		---	---
Cardiovascular drugs - mf		1.0			approx. 2.4 [2]		---	---
Antihypertensive drugs - mf		1.0			approx. 5.5 [2]		---	---
# 09	N = 926							
Bonn (road)								
Cardiovascular drugs - mf			1.0		---	1.26 (0.79-2.00)		---
Hypnotics - mf			1.0		---	1.23 (0.77-1.98)		---
# 13	N = 700							
Erfurt (road)								
Sedatives - mf		---	---	---	1.0	---	2.5 ($p > 0.05$)	---
Hypnotics - mf		---	---	---	1.0	---	3.8 ($p < 0.05$)	---
Tranquillizer - mf		---	---	---	1.0	---	2.6 ($p > 0.05$)	---
Anthypertensive drugs - mf		---	---	---	1.0	---	5.0 ($p < 0.05$)	---
Cardiovascular drugs - mf		---	---	---	1.0	---	5.0 ($p < 0.05$)	---

Table A8 – continued.

Study (Number, Location) **Relative Risk** [1)] **(95% confidence interval)**	**Noise Level [dB(A)], outdoors (L_{day}, L_{dn}, L_{den})**						
	46-50	**51-55**	**56-60**	**61-65**	**66-70**	**71-75**	**76-80**
# 30 N = 1984							
Tyrol (road)							
Sedatives - mf	---	1.0	1.16 (0.61-2.18)	1.11 (0.56-2.19)	---	---	---
Sleeping pills - mf	---	1.0	2.33 (1.19-4.58)	1.88 (0.93-3.78)	---	---	---
Tranquillizer - mf	---	1.0	1.28 (0.89-1.84)	0.99 (0.66-1.48)	---	---	---
# 59 N = 11812							
Amsterdam (aircraft)							
Cardiovascular medication - mf	1.0	1.18 (1.01-1.38)	1.26 (0.98-1.61)	1.22 (0.67-2.21)	---	**---**	---
(including anti-hypertension drugs)					---	**---**	---
Sedatives or sleeping pills - mf					---	**---**	---
- prescribed	1.0	1.15 (0.93-1.42)	1.13 (0.78-1.64)	1.52 (0.67-3.42)	---	**---**	---
- non-prescribed	1.0	1.59 (1.20-2.11)	1.89 (1.21-2.95)	2.02 (0.77-5.30)	---	**---**	---
# 60 N = 417							
Stockholm (aircraft)							
Antihypertensive drugs - m	1.0		1.61 (1.15-2.25)			---	---

m = males, f = females, mf = males and females, IHD = ischaemic heart disease

1) Calculated as risk ratio, odds ratio or proportional morbidity ratio

2) Relative effect of change in purchase [RR (year 5)/ RR (year 1)], relative risks are approximation based on visual inspection of graphs in the reference