The Association between Noise Exposure and Blood Pressure and Ischemic Heart Disease: A Meta-analysis

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It has been suggested that noise exposure is associated with blood pressure changes and ischemic heart disease risk, but epidemiologic evidence is still limited. Furthermore, most reviews investigating these relations were not carried out in a systematic way, which makes them more prone to bias. We conducted a meta-analysis of 43 epidemiologic studies published between 1970 and 1999 that investigate the relation between noise exposure (both occupational and community) and blood pressure and/or ischemic heart disease (*International Classification of Diseases*, Ninth Revision, codes 410–414). We studied a wide range of effects, from blood pressure changes to a myocardial infarction. With respect to the association between noise exposure and blood pressure, small blood pressure differences were evident. Our meta-analysis showed a significant association for both occupational noise exposure and air traffic noise exposure and hypertension: We estimated relative risks per 5 dB(A) noise increase of 1.14 (1.01–1.29) and 1.26 (1.14–1.39), respectively. Air traffic noise exposure was positively associated with the consultation of a general practitioner or specialist, the use of cardiovascular medicines, and angina pectoris. In cross-sectional studies, road traffic noise exposure increases the risk of myocardial infarction and total ischemic heart disease. Although we can conclude that noise exposure can contribute to the prevalence of cardiovascular disease, the evidence for a relation between noise exposure and ischemic heart disease is still inconclusive because of the limitations in exposure characterization, adjustment for important confounders, and the occurrence of publication bias. *Key words*: blood pressure, hypertension, ischemic heart diseases, meta-analysis, noise exposure. *Environ Health Perspect* 110:307–317 (2002). [Online 14 February 2002] *http://ehpnet1.niehs.nih.gov/docs/2002/110p307-317vankempen/abstract.html*

Noise is a persistent environmental problem. In Europe, about 450 million persons are exposed daily to equivalent noise levels of at least 55 dB(A); 113 million persons are exposed to equivalent noise levels of at least 65 dB(A); and 9.7 million persons are exposed to equivalent noise levels of 75 dB(A) or more (Appendix 1) (*1*).

Noise exposure is associated with a number of health effects. We can distinguish psychosocial responses such as annoyance, sleep disturbance, disturbance of daily activities and performance, and physical responses, such as hearing loss, hypertension and ischemic heart disease (*2*). Currently, there is much discussion about how noise can affect human health and well-being. Stress is supposed to play an important role and can be seen as an effect of the appraisal of noise or as a coping reaction of the body (fight–flight)—the so-called physiologic reflexes (*2*).

One of the models on noise and health that are being used at the moment is presented in Figure 1. It is an adapted version of the schematic presented by the Dutch Health Council (*2*) and assumes that health effects and/or status are determined by a combination of endogenous and exogenous factors such as physical and social environment and lifestyle. Noise exposure is only one of these exogenous factors. This process may be modified by personal characteristics such as attitude and coping style. According to this model, noise exposure can induce biochemical, physiologic, or psychosocial changes such as disturbance of sleep and daily activities, stress, and annoyance. These changes fall more or less within the normal range of biologic variation. Whether these changes are of any significance to health depends above all on the degree to which the function of organ systems or psychosocial functioning is affected, the reversibility and duration of the changes and the possibilities for recovery or compensation, and the possible loss of resilience (*2*). Noise-induced sleeping problems and their influence on mood and performance the next day are part of every normal life. However, at some point sleeping problems or sleep disturbance may become clinically significant as normal physical, mental, and social functioning are hampered. An effect such as the elevation of blood pressure caused by noise exposure might fall largely within normal homeostasis. However, given a certain population distribution of, for instance, systolic blood pressure, even a small shift due to environmental exposure may yield a substantial increase in the prevalence and mortality of cardiovascular disease (*2*).

Using this model, in the present study we focus on the physical responses to noise exposure: blood pressure changes and cardiovascular disease risk. Although many observational studies and reviews on noise exposure and cardiovascular effects have been carried out, epidemiologic evidence is still limited (*3–22*). With the preponderant influence of lifestyle and genetic predisposition, it is difficult to gain insight into the contribution of noise to cardiovascular disease (*2*). Therefore, the results presented in these observational studies are not consistent. Most of the previous reviews were not carried out systematically, so they are prone to bias (*23*). Only two of the reviews have quantified the association between noise and cardiovascular disease (*3,24*). In the review of Duncan et al. (*24*) the results of different noise exposure situations were combined. However, the situation in which people are exposed may influence their response. The second study reviewed only occupational studies (*3*).

To gain more insight into the relation between noise exposure and its potential health impact, we performed a meta-analysis on observational studies investigating the relation between noise and blood pressure and/or ischemic heart disease. A meta-analysis or quantitative overview is a systematic review that employs statistical methods to combine and summarize data from several studies (*23*). By means of a meta-analysis we can also gain more insight into the sources of heterogeneity among study results: The findings of observational studies are often distorted by different sources of bias (*25*), causing a fair amount of

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heterogeneous variation on study level (*26*). This variation among research results may be explained by differences in individual study characteristics with respect to the study population or design (*26*).

Materials and Methods

Data collection. Observational studies involving the association between noise exposure and blood pressure and/or ischemic heart disease, published between 1970 and 1999 in English, German, or Dutch, were identified in MEDLINE, EMBASE, BIOSIS, and SCISEARCH (see also Appendix 2 for search strategy). To ensure that most of the studies could be identified, we manually scanned journals, reports, and proceedings in epidemiology, noise, cardiovascular disease, and (public) health. From relevant literature in the field of noise and health, we checked references for additional studies. Overall, we identified more than 500 publications.

Studies meeting the following criteria were included for data extraction:

- Title and/or abstract of the given survey had to involve occupational noise exposure or community noise exposure in relation to blood pressure or ischemic heart disease (or both). In the given studies, the relation between noise exposure and blood pressure and/or ischemic heart disease had to be studied in a study population of adults who were not defined as a population with a certain illness or disorder.
- The survey had to quantify and/or describe the relation between objective noise exposure [in dB(A) and blood pressure and/or the relation between objective noise exposure (in dB(A)] and ischemic heart disease [*International Classification of Diseases*, Ninth Revision, codes 410–414 (*27*)].

We limited the studies to adults because the findings in children are difficult to interpret with regard to possible health risks in their later life (*8*). We chose the equivalent sound level [L_{Aeq}, in dB(A)] as a measure of exposure because it is the measure that is most commonly used (see Appendix 1).

We excluded studies published before 1970 for several reasons: They contained little of the quantitative information necessary for a meta-analysis; they were often (quasi)-experimental; and the epidemiologic and methodologic quality is relatively poor with respect to the current scientific standards (*18*). We also excluded studies using hearing loss or defective hearing as a proxy for (previous) noise exposure because it is impossible to differentiate between hearing loss caused by noise exposure and hearing loss caused by other factors. Also, it is difficult to detect differences in exposure (level) when it is possible that the effects derive from defective hearing rather than other measures of exposure. Furthermore, it is possible that atherosclerosis and/or hypertension increase the risk for hearing loss (*11*). In addition, we exluded surveys assessing noise exposure on the basis of subjective ratings, as given by the study subjects in a questionnaire. Subjective indicators are susceptible to observation bias (caused by overreporting) and recall bias (*13*).

Data extraction. From studies that met these criteria (*28–75*), we extracted the following data via a structured data-extraction form: data about study characteristics (authors, year of publication, period and location of the study, design), population characteristics (number of respondents, sex, age, inclusion and exclusion criteria), exposure assessment, and effect measurement. For each study, the data extraction was done by at least two persons working in noise research and/or statistics, and was discussed afterwards. Furthermore, a noise expert looked at the noise measurements presented in the studies. He checked whether the presented sound levels in the article were realistic given the presented methods of noise assessment.

The main effects under investigation were blood pressure, hypertension, the use of antihypertensive and/or cardiovascular medication, angina pectoris, myocardial infarction, and ischemic heart disease. To compare the studies, we calculated the following outcome variables.

Using the extracted average blood pressure values presented in the studies and noise levels [dB(A)], we calculated blood pressure change (mmHg) per noise level increase of 5 dB(A) ($\beta_{i,bloodpressure}$) and its variance for both systolic and diastolic blood pressure.

Using all the extracted prevalences (incidences) and/or relative risks (RR) and noise levels [dB(A)], we calculated the natural logarithm of the relative risk (lnRR) and its variance per 5 dB(A). For studies comparing two exposure groups, we used the following equations:

$$\beta_i = ln(RR) \times \left(\frac{5}{\Delta db(A)} \right), \quad [1]$$

$$\sigma_i = \left(\frac{(ln\,RR_{hi}) - (ln\,RR_{lo})}{3.92} \right) \times \left(\frac{5}{\Delta dB(A)} \right), \quad [2]$$

where β_i is estimated $ln(RR)$ per 5 $dB(A)$; RR is relative risk extracted from study or calculated with Epi-info 604 (Centers for Disease Control and Prevention, Atlanta, GA, USA; World Health Organization, Geneva, Switzerland); $\Delta dB(A)$ is the difference in noise levels between the index and reference group; σ_i is the estimated standard error of β_i; RR_{hi} is the upper level of RR of the 95% confidence interval; RR_{lo} is the lower level of RR of the 95% confidence interval (see also Appendix 3).

In studies where two or more exposure groups were compared, betas were estimated with the SAS procedure PROC REG (SAS Institute, Inc., Cary, NC, USA). In this case, each group was compared to the lowest exposure group. From each participating study, we extracted one or more estimates of the above-mentioned outcome variables and their variance. Because not all the required data were directly available, recalculations were done. Equations and methods used are presented in Appendix 4.

Data aggregation. The extracted estimates had to be unconfounded by age and sex. Also, they had to refer to a homogeneous study population: White-collar workers are not a good control group for blue-collar workers (*13*) because the difference in noise at work might be associated with other factors of the workplace, which are also related

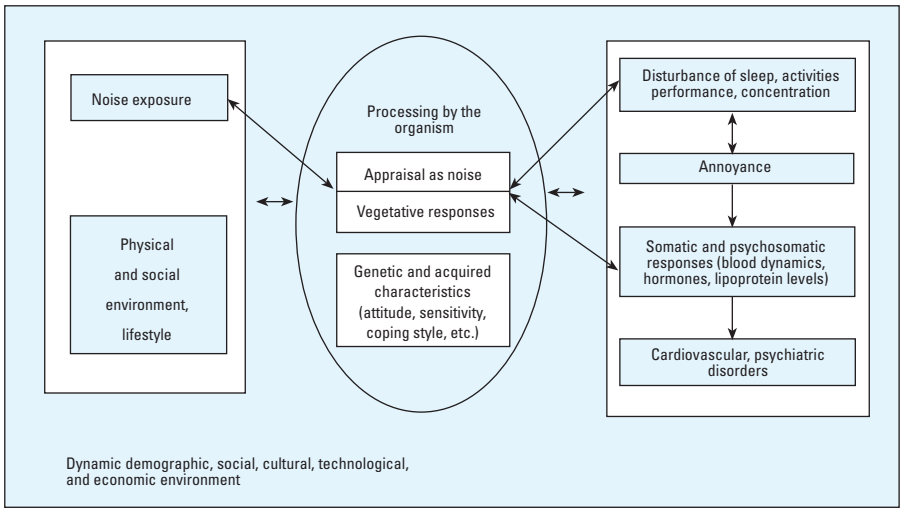

Figure 1. Conceptual model of the interaction of noise with humans and the occurrence of effects on health and quality of life (*2*).

to the health outcome. Furthermore, there may be differences with regard to lifestyle, social status, and psychosocial factors. Therefore, for the occupational studies researched in this article, we include only estimates from studies investigating the association between noise and ischemic heart disease and/or blood pressure that are well matched with regard to control (referent) groups. Because the populations in the community noise studies were considered relatively homogeneous, no extra criteria were applied. These adjusted estimates were aggregated, taking into account the variance. The true value was assumed to be normally distributed [mean (μ_{true})] and to have a standard error (σ_{true}). Through meta-analysis, we estimated μ_{true} and σ_{true}, given a number of outcome measures y_i (i = 1,…,*n*) with standard error σ_i. To estimate these parameters, we used a Random Effects Model (REM). REM acknowledges the occurrence of variation of true effects between studies but regards them as unknown effects to be estimated by assuming that the effects observed in the sample of studies analyzed are drawn from a population of studies (*26*).

To summarize the data, we calculated summary estimates of the extracted estimates unconfounded by age and sex and containing homogenic populations. Because the effects of noise sources might differ, we present the summary estimates for occupational noise exposure, road traffic noise exposure, and air traffic noise exposure separately. Afterward, we transformed the estimated betas for hypertension, use of antihypertensive and/or cardiovascular medication, angina pectoris, myocardial infarction, and ischemic heart diseases into a $RR_{5\ dB(A)}$ and 95% confidence interval.

Subgroup analysis. To investigate how these summary estimates might be affected by heterogeneity, we performed subgroup analyses (Table 1). For the association between occupational noise exposure and blood pressure as well as hypertension, we also calculated a summary estimate for those selected estimates that adjusted for body mass index (BMI).

Sensitivity analysis and publication bias. We examined the sensitivity of the results to any single estimate for occupational noise exposure and blood pressure as well as hypertension. This was done by removing the estimates one by one from the analysis and recalculating the summary estimate.

One of the most important problems in meta-analysis is that some studies do not get published. If the reasons that studies remain unpublished are associated with their outcome (publication bias), the validity of meta-analysis can be seriously threatened. To indicate the extent of publication bias in the present study, we made funnel plots. A funnel plot is a scatter plot of the studies' effect estimates against the inverse of the standard error. It is based on the fact that the precision in estimating the underlying effect will increase as the sample size of studies increases. In the absence of bias, the plot should resemble a symmetrical funnel (*76*).

Table 1. Subgroup analyses.

Factor under study	Subgroup of studies
Measurement of exposure	Sound level meters (SLM)
	Both a personal dosimeter (PDM) and a sound level meter (SLM)
	Job titles
	Exposure measurement was not reported
Blood pressure measurement	1 time
	> 1 time
Definition of hypertension used	Systolic blood pressure ≥ 95 mmHg and/or diastolic blood pressure ≥ 160 mmHg and/or use of antihypertensives
	Systolic blood pressure ≥ 95 mmHg and/or diastolic blood pressure ≥ 160 mmHg
Inclusion of treated hypertensives	Including treated hypertensives
	Excluding treated hypertensives
Sex of study population	Males
	Females
	Both sexes
Age of study population	18–35 years
	35–65 years
	18–65 years
Study location	Asia
	North America
	Europe (including Israel)
	South Africa
Publication period	1990s
	1980s
	1970s[a]
Study design[b]	Longitudinal studies, presenting 10-year incidences
	Cross-sectional studies, presenting prevalences

[a]This subgroup was included for hypertension. [b]This subgroup analysis was performed only for the association between myocardial infarction and IHD.

Results

Descriptives. Tables 2 and 3 show some characteristics of the studies involved in the data extraction. The occupational studies were all cross-sectional; from the cohort studies (*31,34,51,53*) only baseline results were available. The occupational studies were performed among a great variety of industries throughout the world within a broad exposure range: The $L_{Aeq,8hr}$ varied from 48 to 116 dB(A) (see Appendix 1). The community studies encompassed two case–control studies (*65,66*) and two cohort studies (*67–72*). They were carried out among equivalent sound levels (6–22 hrs and 7–19 hrs) of 38–80 dB(A) in Europe. In community noise studies, noise exposure is usually calculated, whereas occupational studies mainly tried to measure the noise exposure. The sample sizes of the studies varied from 46 (*30*) to 35,150 persons (*74*).

Exposure–response estimates. We studied the influence of noise exposure on blood pressure both for occupational and for road and air traffic noise exposure (see Figures 2 and 3). A statistically significant increase in blood pressure level was evident only in studies for occupational noise exposure: For systolic blood pressure we estimated an increase of 0.51 (0.01–1.00) mmHg/5 dB(A) (Figure 2). In the case of air traffic noise exposure and blood pressure increase, it refers only to military air traffic noise, not to civilian air traffic noise (*64*). Figures 2 and 3 show that the effect of occupational noise exposure on blood pressure differs among the studies.

The association between occupational noise exposure and hypertension is statistically significant: We found an $RR_{5\ dB(A)}$ of 1.14 (1.01–1.29) (Figure 4, Table 4).

Compared with the occupational studies, the community studies contained relatively few estimates per effect (Figures 5 and 6). Road traffic noise exposure is positively associated (nonsignificant) with myocardial infarction and ischemic heart diseases (Figure 5). Effects positively associated with air traffic noise exposure were hypertension, angina pectoris, the use of cardiovascular medicines, and consultation of a specialist and/or general practitioner (GP) (Figure 6). Only the association with air traffic noise exposure and hypertension was statistically significant: $RR_{5\ dB(A)}$ 1.26 (1.14–1.39) (Figure 5, Table 4). However, these results were based on only one study (*73*).

Subgroup analyses. The results of the subgroup analyses for the occupational studies are presented in Figures 7 and 8. These figures show that for the influence of occupational noise exposure on blood pressure

Table 2. Study characteristics of the occupational studies included for data extraction.

Study (reference)	Country	Design	Population, age	No.	Industry	Exposure levels[a] [dB(A)]	Exposure measurement	Effects[b]	Adjustments[c]
Parvizpoor, 1976 (*28*)	Iran	Cross	M, 19–59	1,233	Textile mill	≤ 96	Not reported	B	1,2,3
Malchaire, 1979 (*29*)	Belgium	Cross	M, > 20	2,111	Car assembly and wire mill	93–100; 93–97	Dosimetry	B	1,2
Ising, 1980 (*30*)	Germany	Cross	M, 25–51	46	Brewery	95 ± 0,7; 82 ± 1,2	SLM & PDM	A	2,4
Lees, 1980 (*31*)	Canada	Cohort	M & F	140	Production and handling areas	≤ 85 , > 90	Company records	B, C	—
Kornhuber, 1981 (*32*)	Germany	Cross	M & F	97	Motor works	97–111	SLM	A	—
Singh, 1982 (*33*)	India	Cross	M & F?, 30–35	111	Army	88–107	Job history & SLM	A	2
Aro, 1984 (*34*)	Finland	Cohort	M & F, 21–61	388	Metal industry	64.8 ± 15.8	SLM	A	2,3,22
Belli, 1984 (*35*)	Italy	Cross	M ?, 35–56	940	Textile mill	78–105	SLM	B	2
Van Dijk, 1984 (*54*)	Netherlands	Cross	M, ≤ 65	238	Various[d]	78–98	SLM & noise exposure anamneses	A, B	2,5
Van Dijk, 1984 (*55*)	Netherlands	Cross	M, 18–63	257	Shipyard	82–91; 91-111	SLM & PDM	A, D	2,4,5
Van Dijk, 1987 (*56*)	Netherlands	Cross	M, 17–61	421	Various[e]	≤ 80; 81–85; 86-90; 91–95; > 95	SLM & PDM	A, D	2,4,6,7,8
Korotkov, 1985 (*36*)	Russia	Cross	M, 33–36	207	Seamen	93; 65	Acoustic data	A, B	2,5
Talbott, 1985 (*37*)	United States	Cross	M, 40–63	350	Various[f]	89; 81	SLM &PDM	A, B	2,9,10,11,12,13
Wu, 1987 (*47*)	Taiwan	Cross/Case[g]	M, 30–59	2,730	Shipyard company	> 85; < 80	SLM	A, B	1,2,4,5
Idzior-Walus, 1987 (*39*)	Poland	Cross	M, 20-55	784	Riveters and farmers	105-116	Not reported	A, B, C	1,2,12,14,15
Tarter, 1990 (*45*)	United States.	Cross	M, 35–65	269	Automobile plant		Dosimetry	B, D	2,16
Hirai, 1991 (*41*)	Japan	Cross	M, 20–59	1,756	Quiet office	85–115; < 85;	Not reported	A, B	2,3
Green, 1991 (*40*)	Israel	Cross	M, 25–65	162		74–102	PDM	A	2,4,13,17
Zhao, 1991 (*52*)	China	Cross	F, 18–50	1,101	Textile mill	75–104	SLM & noise survey	B	2,3,5
Tomei, 1991 (*38*)	Italy	Cross	M, 25–55	300		80–92; 70	Not reported	A, B	2,5,18
Lang, 1992 (*42*)	France	Cross	M, 18–60	1,986	Various[h]	85–100; ≤ 80	SLM & interview	A, B	2,4
Hessel, 1994 (*51*)	South Africa	Cohort	M, 27–40	973	Mine	80–111	Job titles	A	2,4,11,12,16
Fogari 1,2, 1994/95 (*43,44*)	Italy	Cross	M & F, 18–60	8,811	Metal company	≤ 55; 56–80; >80	SLM	A, B	2,3,4,5,19,22
Kristal-Boneh, 1995 (*53*)	Israel	Cohort	M & F, 20–65	3,106	Various[i]	≤ 65 - > 90	SLM	A	2,3,11,12,20,22
Wu, 1996 (*47*)	Taiwan	Cross	M & F, 81–71	222	Lead battery manufacturing factory	67 and 96	PDM	A	2,4,22–25
Saha, 1996 (*46*)	India	Cross	M, 20–55	156	Thermal power station	48–66; 90–113	SLM	A, B	2,4
Zhao, 1998 (*48*)	China	Cross	M & F, 18–58	1,593	Chemical fertilizer factory	53.0–96.7	SLM	B	2,3,22
Talbott, 1999 (*50*)	United States	Cross	M, 40–63	643	Stamping and assembly plant	57.0–100.1	SLM	A, B, D	4,11,12,21

Abbreviations: F, female; M, male; PDM, personal dosimeter; SLM, sound level meter.
[a]$L_{Aeq,8hr}$, the measurement range of the study. [b]A = blood pressure; B = hypertension; C = cardiovascular disease; D = use of medication for heart diseases. [c]1 = socioeconomic status; 2 = age; 3 = type of job; 4 = body mass index (or quetelet index); 5=duration of exposure/no. of working years; 6 = shift work; 7 = stress symptoms; 8 = annoyance index; 9 = education level; 10 = marital status; 11 = alcohol consumption; 12 = smoking (behavior); 13 = hearing loss; 14 = residence; 15 = physical activity at work; 16 = ethnicity; 17 = heart rate; 18 = suffering from hypertension; 19 = cholesterol level; 20 = coffee consumption; 21= medical history of hypertension; 22 = sex; 23 = blood lead level; 24 = ambient lead concentration; 25 = working history. [d]Production divisions of a livestock company, chocolate factory, engineering shop, printing office, division of mechanical woodworking and metalworking company. [e]Metal company, livestock company, synthetic processing company, metal processing company, and chemical industry. [f]Fabricage and production of metal parts and heating elements. [g]Cross and case-referent study design. [h]Mechanical or chemical industry, offices, garages, and restaurants. [i]Metal work, textile sector, light industry, electronics, foodstuffs, and plywood.

Table 3. Study characteristics of the community studies included for data extraction.

Study	Country	Design	Population, age	No.	Exposure			Effects investigated[b]	Adjustments[c]
					Source	Level [dB(A)][a]	Measurement		
Knipschild, 1976 (*57,58*)	Netherlands	Cross	F, 40–49	1,741	Road	55–60, 65–70	?	B,C,E,G	1,2,3,4,5,6,7,8,9
Von Eiff, 1980 (*59*)	Germany	Cross	M & F, 20–59	931	Road	>50, 66–73	Calc	B,C	1,10,11,12,13,14,15
Neus,1983 (*60*)	Germany	Cross	M & F,	117	Road	< 57, > 66	?	A,	1,11,16
Schulze, 1983 (*61*)	Germany	Cross	M & F, 20–75	700	Road	64-67, 72–75	SLM ?	B,H	17,18,19,20
Wölke, 1983 (*62*)	Germany	Before–after	M & F, >18	350	Road	76, 60	?	B	1, 11, 16
Knipschild, 1984 (*63*)	Netherlands	Cross	M & F, 41–43	2,878	Road	<55–80	SLM	A, B	11
Van Brederode, 1989-a (*64*)	Netherlands	Cross	M & F, 18–55	396	Road	40–75	Calc	A, H	1,2,10,11,21,22
Berlin a, 1994 (*65,66*)	Germany	Case–control	M, 41–70	243	Road	60–80	Calc	F	1,2,17,23,24,25
Berlin b, 1994 (*65,66*)	Germany	Case–control	M, 31–70	4,035	Road	60–80	Calc	F	1,2,23,24,25
Berlin c, 1994 (*66*)	Germany	Cross	M, 31–70	2,169	Road	60–80	Calc	F	1
Caer, 1993-1999 (*67–69,71,72*)	U.K.	Cohort	M, 45–59	2,512	Road	51–70	SLM & PDM	A,B,E,F,H	1,2,3,4,10,16,26
Speed, 1993-1999 (*67,68,70–72*)	U.K.	Cohort	M, 45–63	2,348	Road	51–70	SLM	A,E,F,H	1,2,3,4,10,16,26
Knipschild 1976 (*73*)	Netherlands	Cross	M & F, 35–64	5,828	Air	66–77, 55–66	Calc	B,D,E,G	1,2,3,11
Knipschild, 1976 (*74*)	Netherlands	Cross	M & F, < 99	35,150	Air	55–72	Calc	B,C,D,G	1,11
Van Brederode,1989 b (*64*)	Netherlands	Cross	M & F, 18–55	432	Air[d]	< 63 – > 75	Calc	A, H	1,2,10,11,21,22
RIVM/TNO, 1998 (*75*)	Netherlands	Cross	M & F, > 18	11,812	Air	38–75	Calc	D	1,11

Abbreviations: Caer, Caerphilly; Calc, exposure assessment by calculations; F, female; M, male; PDM, personal dosimeter; SLM, sound level meter; Speed, Speedwell.
[a]For road traffic noise expressed as $L_{Aeq,6–22\ hr}$, and for air traffic noise expressed as $L_{Aeq,7–19\ hr}$; this is the measurement range of the study. [b]A = blood pressure change; B = hypertension; C = use of antihypertensives; D = use of cardiovascular medication (including antihypertensives); E = angina pectoris; F = myocardial infarction; G = consultation GP/specialist; H = coronary heart diseases. [c]1 = age; 2 = anthropometric data (BMI, etc); 3 = smoking; 4 = physical activity at work; 5 = shortness of breath at exertion; 6 = chronic cough; 7 = lung pathology; 8 = cholesterol; 9 = diabetes;10 = alcohol consumption; 11 = sex; 12 = professional status; 13 = income; 14 = coffee consumption; 15 = tea consumption; 16 = socioeconomic status; 17 = education level; 18 = professional activity; 19 = working conditions; 20 = living conditions; 21 = hypertension in parents and siblings; 22 = primary appraisal; 23 = duration of residence; 24 = working; 25 = noise in workplace; 26 = family history. [d]Military air traffic noise.

change, a statistically significant increase in systolic blood pressure could be distinguished for five subgroups: *a*) studies adjusting for BMI: 0.82 (0.00–1.65) mmHg/5 dB(A); *b*) studies investigating both males and females: 0.65 (0.34–0.95) mmHg/5 dB(A); *c*) studies including treated hypertensives: 0.67 (0.12–1.22) mmHg/5 dB(A); *d*) studies carried out during the 1990s: 0.56 (0.04–1.08) mmHg/5 dB(A); and *e*) studies using sound level meters (SLM) for exposure assessment: 0.87 (0.05–1.69) mmHg/5 dB(A). For diastolic blood pressure change, no subgroups that indicated a statistically significant change in blood pressure could be distinguished.

Figure 8 shows that a statistically significant association between occupational noise exposure and hypertension could be identified for six subgroups: *a*) studies adjusting for BMI: $RR_{5\ dB(A)}$ 1.60 (1.10–2.32); *b*) studies investigating populations 18–65 years of age: $RR_{5\ dB(A)}$ 1.18 (1.12–1.25). This differs from the $RR_{5\ dB(A)}$ estimated for studies investigating populations 18–35 years of age [$RR_{5\ dB(A)}$ 0.93 (0.79–1.10)]; *c*) studies investigating both males and females: $RR_{5\ dB(A)}$ 1.25 (1.13–1.39); *d*) studies carried out

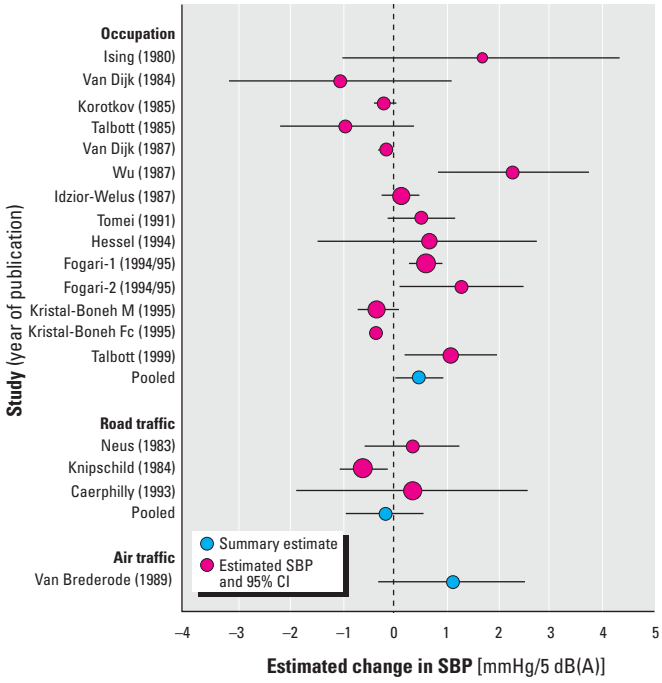

Figure 2. The association between noise exposure and systolic blood pressure change, adjusted for sex, age, and work type. Abbreviations: CI, confidence interval; SBP, systolic blood pressure change. The dotted vertical line corresponds to no effect of occupational noise exposure on systolic blood pressure. The measurement ranges of the studies included were occupational noise exposure $L_{Aeq,8hr}$ 50–116 dB(A); road traffic noise exposure $L_{Aeq,6–22hr}$ 51–80 dB(A); and air traffic noise exposure $L_{Aeq,7–19hr}$ 63 to > 75 dB(A). This estimate has a large variance.

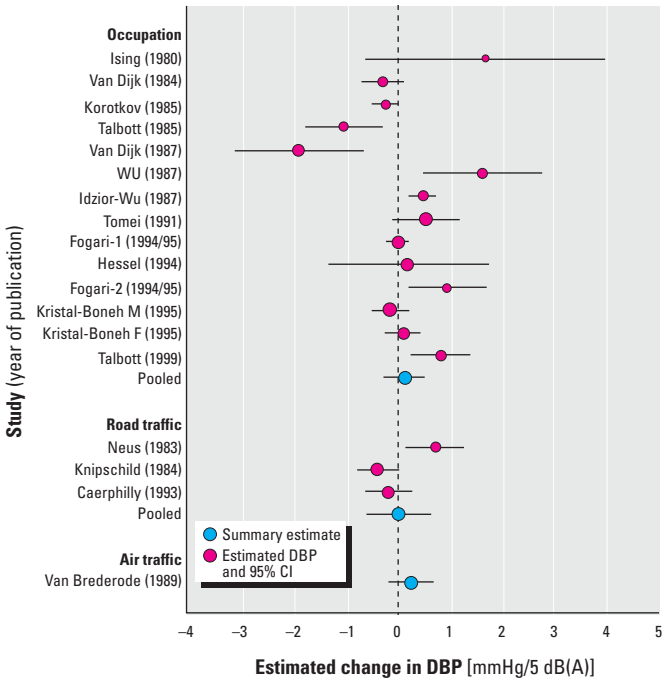

Figure 3. The association between noise exposure and diastolic blood pressure change, adjusted for sex, age, and work type. Abbreviations: CI, confidence interval; DBP, diastolic blood pressure change; F, female; M, male. The dotted vertical line corresponds to no effect of occupational noise exposure on diastolic blood pressure. The measurement ranges of the studies included were occupational noise exposure $L_{Aeq,8hr}$ 50–116 dB(A); road traffic noise exposure $L_{Aeq,6–22hr}$ 51–80 dB(A); and air traffic noise exposure $L_{Aeq,7–19hr}$ 63 to > 75 dB(A).

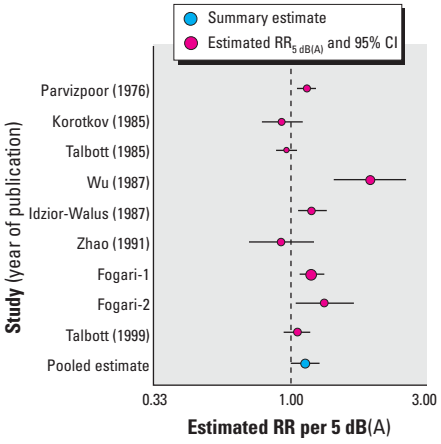

Figure 4. The association between occupational noise exposure and hypertension, adjusted for age, sex, and work type. CI, confidence interval. The dotted vertical line corresponds to no effect of occupational noise exposure. Measurement range of the studies, $L_{Aeq,8hr}$, 55–116 dB(A).

Table 4. Summary estimates, expressed as $RR_{5\ dB(A)}$, for the association between noise exposure, hypertension, and ischemic heart diseases, adjusted for sex and age.

Noise exposure,[a] outcome	$RR_{5\ dB(A)}$	95% CI	No. of estimates	Measurement range [dB(A)]
Occupation				
Hypertension[b]	1.14	1.01–1.29*	9	55–116
Road traffic				
Hypertension	0.95	0.84–1.08	2	< 55–80
Use of antihypertensives	0.96	0.76–1.22	2	> 50–73
Consultation of GP/specialist	0.91	0.73–1.12	1	55–70
Angina pectoris	0.99	0.84–1.16	2	51–70
Myocardial infarction[c]	1.03	0.99–1.09	3	51–80
IHD-total[c]	1.09	1.05–1.13*	2	51–70
Air traffic				
Hypertension	1.26	1.14–1.39*	1	55–72
Use of antihypertensives	0.99	0.87–1.14	1	55–72
Consultation of GP/specialist	1.10	0.95–1.27	2	55–77
Use of cardiovascular medicines	1.05	0.99–1.11	2	38–77
Angina pectoris	1.03	0.90–1.18	1	55–72

CI, confidence interval.
[a]The noise exposure measures differed between the noise exposure sources: occupational noise exposure expressed in $L_{Aeq,8hr}$, in dB(A), road traffic noise exposure expressed in $L_{Aeq,\ 6–22hr}$, in dB(A), and air traffic noise exposure expressed in $L_{Aeq,7–19hr}$, in dB(A). [b]Adjusted for age, sex, and work type. [c]Only prevalence estimates. *$p < 0.05$.

in Europe: $RR_{5\ dB(A)}$ 1.60 (1.58–1.62); *e*) studies using SLMs for exposure assessment: $RR_{5\ dB(A)}$ 1.32 (1.05–1.67); *ff*) studies carried out during the 1990s [$RR_{5\ dB(A)}$ 1.14 (1.00–1.31)] and 1970s [$RR_{5\ dB(A)}$ 1.15 (1.06–1.24)].

For the association between road traffic noise exposure and ischemic heart diseases (IHD) (Figure 5), data aggregation produced statistically significant summary estimates for the cross-sectional studies (*71*) [$RR_{5\ dB(A)}$ 1.09 (1.05–1.13)]. After the results of the reported 10-years' incidence were combined, the effect of road traffic exposure on IHD was eliminated (*72*) [$RR_{5\ dB(A)}$ 0.97 (0.90–1.04)]. With respect to the association between road traffic noise and myocardial infarction, no significant differences between prevalence and incidence could be noticed.

Sensitivity analysis and publication bias. Sensitivity analysis through one-by-one exclusion of studies revealed that the results of the meta-analysis for occupational noise exposure and blood pressure as well as for occupational noise exposure and hypertension were not significantly affected by separate studies.

Because only a few estimates were available for most of the studied effects, it was possible to make funnel plots only for blood pressure changes and hypertension associated with occupational noise exposure. Figure 9 presents the results for hypertension. The figure shows that studies finding a relatively small effect have been published less often.

Discussion

Main results. For this meta-analysis, we studied 43 occupational and community studies with a wide range of effects, varying from blood pressure changes to a myocardial infarction. With respect to the association between noise exposure and blood pressure, we noticed small blood pressure differences. A significant increase in systolic blood pressure was evident for occupational noise exposure. The results of the occupational studies tally with the results of an earlier review conducted by Passchier-Vermeer (*3*) evaluating 21 occupational studies; this review presented increases of the mean systolic and diastolic blood pressure of 3.9 and 1.7 mmHg for persons in exposed groups compared to persons in reference groups, respectively (*3*). Our results concerning community noise studies correspond to the results of a research review by Babisch (*8*), who concluded that no consistent findings can be seen and that there was little epidemiologic evidence of an increase in blood pressure in subjects exposed to traffic noise. Furthermore, we can conclude that the results of the occupational studies investigating blood pressure are contradictory (Figures 2 and 3). From the results with respect to the subgroup analyses for blood pressure, no sources of heterogeneity could be identified, however. The finding that road traffic noise exposure is not associated with the risk of hypertension agrees with that of Babisch (*8*), who concluded that there was little epidemiologic evidence of an increased risk of hypertension in subjects exposed to traffic noise. In the present study, we found a statistically significant association for occupational noise exposure with hypertension: $RR_{5\ dB(A),\ occup}$ = 1.14 (1.01–1.29). Passchier-Vermeer (*3*) also found a significant increase in risk of hypertension; an RR of 1.7 for noise levels exceeding 85 dB(A) was recorded. Duncan et al. (*24*) found an increase in the odds of developing hypertension as a function of increasing noise levels above 20 Kosten units [equivalent to $L_{Aeq,7–19hr}$ 55 dB(A)]. However, comparing is difficult because the results of the different exposure situations were combined.

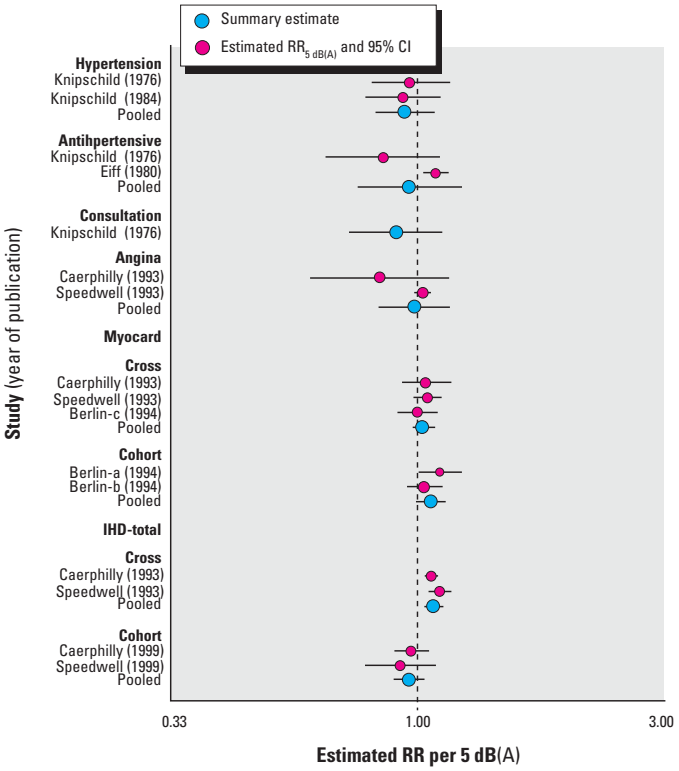

Figure 5. The association between road traffic noise exposure [$L_{Aeq,6–22hr}$, in dB(A)] and hypertension or ischemic heart disease, adjusted for age and sex. CI, confidence interval. The dotted vertical line corresponds to no effect of road traffic noise exposure. The measurement ranges of the studies included were hypertension < 55–80 dB(A); use of antihypertensives > 50–73 dB(A); consultation of a GP/specialist 55–70 dB(A); angina pectoris 51–70 dB(A); myocardial infarction 51–80 dB(A); and ischemic heart disease 51–70 dB(A).

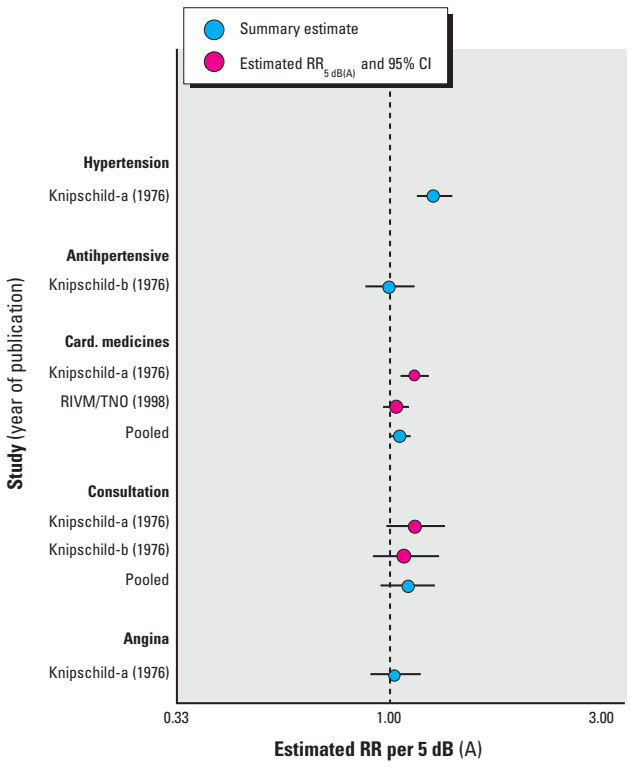

Figure 6. The association between air traffic noise exposure [$L_{Aeq,7–19hr}$, in dB(A)] and hypertension or ischemic heart disease, adjusted for age and sex. CI, confidence interval. The dotted vertical line corresponds to no effect of air traffic noise exposure. The measurement ranges for the studies included were hypertension 55–72 dB(A); use of antihypertensives 55–72 dB(A); use of cardiovascular medicines 38–77 dB(A); consultation of a GP/specialist 55–77 dB(A); and angina pectoris 55–72 dB(A).

The use of antihypertensives (an indirect indicator for hypertension) was not associated with community noise exposure. Air traffic noise exposure was positively associated with the consultation of a GP or specialist, the use of cardiovascular medicines, and angina pectoris. In cross-sectional studies, road traffic noise exposure increases the risk of myocardial infarction and ischemic heart diseases (IHD-total). However, the results for IHD-total contradicted the results of the follow-up studies, in which this effect was not evident (*71,72*).

The hypothesis that the association of noise exposure with IHD might differ among the different noise sources is not confirmed by our results: Comparing the random effect estimates per effect between air traffic noise and road traffic noise (Table 4) shows that air traffic noise exposure is more strongly associated with blood pressure and/or IHD than is road traffic noise exposure. However, these differences are not statistically significant. A possible explanation of the observed differences may be found in the study of Miedema and Oudshoorn (*77*). Recently, they published the results of a pooled analysis on noise exposure and annoyance. These results indicated that air traffic noise is more annoying than road traffic noise (*77*).

Studies included. We can thus conclude that epidemiologic evidence on noise exposure, blood pressure, and IHDs is still limited: With respect to blood pressure and hypertension, results were contradictory, and for IHDs only a few studies are available. One can raise some criticism of the individual studies: First, the studies are mainly cross-sectional. This confounds both the determination of the direction of the causation and the accurate

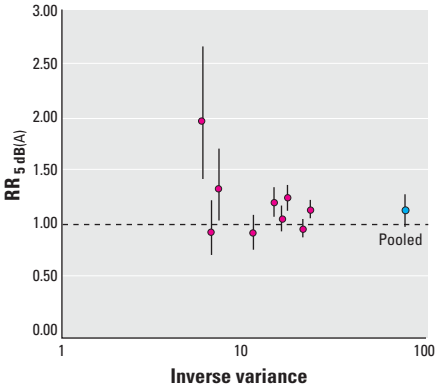

Figure 9. Funnel plot of the results of occupational studies investigating the relation between occupational noise exposure [$L_{Aeq,8hr,}$ in dB(A)] and the risk of hypertension, adjusted for age, sex, and work type.

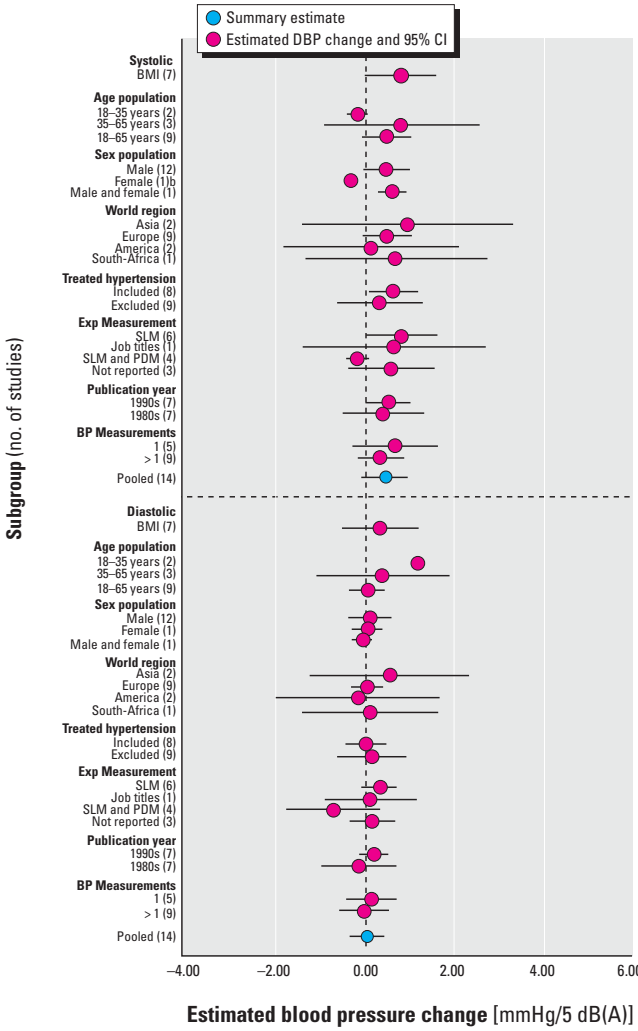

Figure 7. Subgroup analysis for the association between occupational noise exposure [$L_{Aeq,8hr,}$ in dB(A)] and systolic blood pressure (SBP) and diastolic blood pressure (DBP), adjusted for age, sex, and work type. CI, confidence interval. The dotted vertical line corresponds to no effect of occupational noise exposure on blood pressure. This estimate has a standard error of 60 mmHg/5 dB(A).

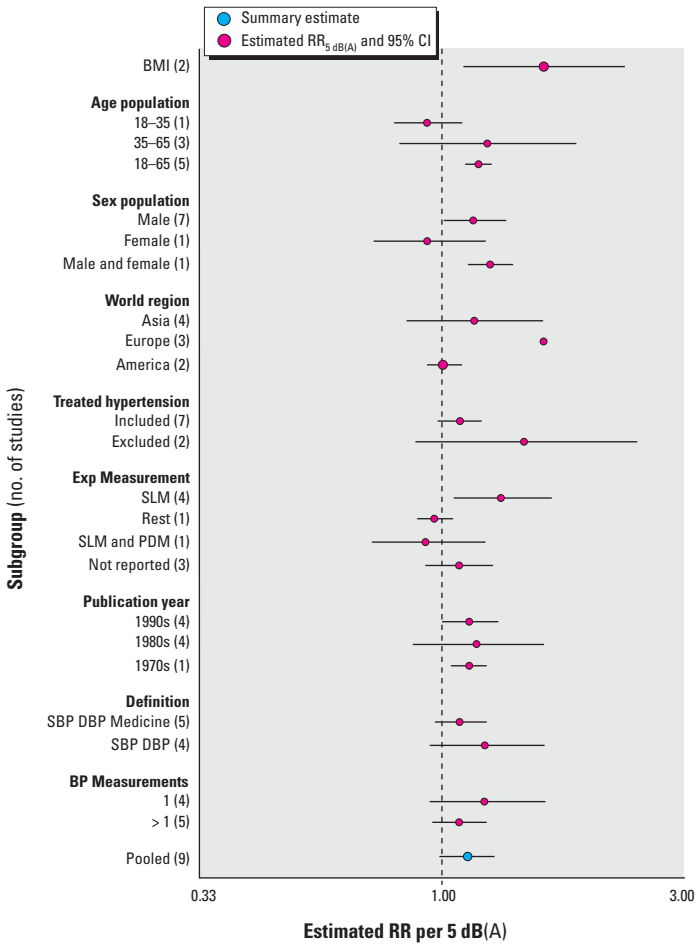

Figure 8. Subgroup analysis for the association between occupational noise exposure [$L_{Aeq,8hr,}$ in dB(A)] and hypertension, adjusted for age, sex, and work type. Abbreviations: BMI, adjusted for sex, age, blue-collar workers, and body mass index; CI, confidence interval; PDM, personal dosimeter; SBP DBP, studies defining hypertension as systolic blood pressure ≥ 96 mmHg and/or diastolic blood pressure ≥ 160 mmHg; SBP DBP Medic, studies defining hypertension as systolic blood pressure ≥ 95 mmHg and/or diastolic blood pressure ≥ 160 mmHg and/or use of antihypertensives; SLM, sound level meter. The dotted vertical line corresponds to no effect of road traffic noise exposure.

estimation of noise exposure (*22*). To produce persistent effects such as coronary heart disease, noise may have to be of certain intensity and to be present for a certain length of time. Another problem when investigating chronic diseases in cross-sectional studies is the problem of self-selection in community studies and the healthy-worker effect in occupational studies. In community studies, somewhat sensitive subjects may move out of the polluted areas, diluting the effect of interest (*8*). In occupational studies, subjects may leave the job because of cardiovascular diseases due to noise or because of the noise itself. These effects tend to diminish the magnitudes of the effect estimates (*13*).

Furthermore, noise exposure was often poorly characterized. In the occupational studies, noise exposure was assessed mainly by fixed measurements with sound level meters. Also, data on the use of ear protection were largely missing. In community studies, exposure was often calculated. But from the literature it was not possible to derive whether these models were validated. Although noise exposure was assessed at people's homes, the fact that people work outside the home during the day was not taken into account. The characterization of personal exposure is a general problem in environmental epidemiologic studies, especially concerning long-term effects. In general, the reporting of noise-related factors, such as fluctuation of noise levels, duration of exposure, frequency (Hz), and peak or continuous noise, was incomplete. Other reviewers came to the same conclusion (*3,8,24*). Adjustments for the position of the living and/or sleeping room(s) were often not made. Also the blood pressure was not always measured in a standard way, and often only a single blood pressure measurement was done. The definition of hypertension was often based on this single measurement. In addition, studies did not always adjust for important modifying factors, such as BMI, smoking, and alcohol consumption. The aspects mentioned in the above section might have led to misclassification on both exposure and effect, which will bias the effect in the direction of no effect.

Bias meta-analysis. Compared to earlier reviews on noise exposure and cardiovascular effects, our study was performed systematically: We defined inclusion and exclusion criteria and used one consistent measure of association for comparing study results. Furthermore, this study provided estimates based on more recent studies, stratified analyses by various study characteristics (subgroup analyses), and analyses to assess publication bias.

However, some aspects must be kept in mind when interpreting our results. Several studies contained exposure groups that had no clear-cut noise range [e.g., people exposed to < 80 dB(A)]. To calculate an effect estimate, a noise expert made a "best guess" of the L_{Aeq}. This "best guess" was based on the information available in the literature (*28–75*). These choices might have influenced the strength of the calculated associations.

For the meta-analysis, we presented the results of an exponential model, which meant that a constant RR per noise unit is assumed, and this suggests an exponential relation between noise exposure and the effect concerned. It was not possible to indicate a threshold value (Appendix 3). This is not consistent with studies that state that there is a threshold value of 70 dB(A) (*1,79*).

A serious threat to the validity of a meta-analysis is publication bias. With respect to occupational noise exposure and hypertension, the funnel plot (Figure 9) shows that studies with negative results are sometimes missing because they were not available. For this association, we concluded that there is an indication for publication bias. Another possible explanation is that there are some poor studies (e.g., with misclassification of exposure) reporting a false positive association. For the other effects under study, it was not possible to make funnel plots because few studies were available.

The results of the occupational studies were not consistent. The subgroup analyses suggested that for the association of occupational noise exposure with blood pressure and hypertension, no sources of heterogeneity could be identified despite the fact that the occupational studies were performed among a great variety of industries. Our results show that with respect to the association between traffic noise exposure and ischemic heart diseases (IHD-total), study design might be a possible source of heterogeneity.

Biologic mechanisms. The literature suggests that noise-induced cardiovascular effects must be seen as the consequence of stress. Stress can arise in several ways in relation to noise. We can distinguish physiologic and psychologic pathways. In experimental studies that studied the effects of short-term noise exposure, acute biochemical, physiologic, and cardiovascular changes have been found (*3,22*). These mark a common physiologic stress reaction of short duration that occurs as a consequence of the activation of the autonomous nervous and hormone system. It appeared that the acute effects referred to were the same as the effects caused by an ordinary stress reaction.

Some authors assume that the effect of noise on the auditory system is transmitted to the Reticular Arousal System and the hypothalamus, where both neuronal and hormonal (hypothalamus–pituitary–adrenal axis) activity may be activated (*3,22*). Stress can also be the consequence of the appraisal of noise (*2*).

A stress situation can lead to the following effects, which are primary risk factors for coronary heart disease. First, directly as a result of stress, the body secretes adrenal medullary hormones (catecholamines) such

Appendix 1. Noise measures.

To judge noise levels and their possible impacts on health, several noise measures are available. These measures start from a physical quantity to which corrections are applied that account for the human noise sensitivity. These corrections depend on the frequency, noise characteristics (impulse, intermittent, continuous), and the noise source (*1*). Noise measures relevant for this article are explained here.

Noise frequency. Noise is a physical phenomenon consisting of alternating compression and expansion of air that propagates in all directions from a source. These alternating compressions and expansions can be described as small changes in pressure around atmospheric pressure. The frequency of the alternations [expressed in Hertz (Hz)] determines the pitch of a sound (*1*): A high-pitched tone (e.g., 4,000 Hz) has a squeaking sound; a low-pitched tone (e.g., 200 Hz) a humming sound.

Sound pressure level. Sound pressure level (L) is a measure of the air vibrations that make up sound. Because the human ear can detect a wide range of sound pressure levels [10–102 Pascal (Pa)], they are measured on a logarithmic scale with units of decibels (dB). Decibels are used to indicate the loudness of sound (*1*).

Sound level. The human ear is not equally sensitive to sounds of different frequencies. Therefore, a spectral sensitivity factor is used that rates sound pressure levels at different frequencies in a way comparable to that of the human hearing organ; this is called A-weighting. The biophysical quantity A-weighted sound pressure level (L) is expressed as dB(A) and is referred to as sound level (*1*).

Equivalent sound level. Sound levels fluctuate within time. For these fluctuating sound levels, the equivalent sound level ($L_{Aeq,T}$) over a period of time T is determined. Common exposure periods T are from 7 to 23 hr ($L_{Aeq,7–23hr}$) (often used in community noise studies), and 8 hr (workday) (often used in occupational noise studies) (*1*).

as noradrenaline. The effects of these hormones will be the rise of peripheral resistance and the increase of blood pressure and heart rate (*22*). Second, indirectly stress may affect human behavior and thus can contribute to cardiovascular disease, for example, by increased smoking, alcohol consumption, and use of medicines (*3*).

According to Morrell et al. (*4*), heart diseases caused by noise exposure may occur more often in susceptible subgroups within populations through physiologically mediated aggravation of existing physical or mental conditions or through precipitation of complications—for example, triggering of dysrhythmias in persons with heart disease.

We can conclude that the biologic mechanism of the relation between noise exposure and cardiovascular effects seems plausible but is very complex.

Conclusions. The results of this meta-analysis are consistent with a slight increase of cardiovascular disease risk in populations exposed to air traffic and/or road traffic noise.

Appendix 2. Search profile used to identify studies on noise and blood pressure and/or ischemic heart disease.

```
BASE COMMAND ACCEPTED FOR ME66;MEDLINE;LAST-
UPDATE=3.11.1998
*** MEDLINE reloaded using MeSH 98 *** Copyright NLM. For
    details of copyright, liabilities and warranties see
    COPYRULES file: BASE ZC00
?
t hc
   1.00  852601 FIND CT D CARDIOVASCULAR DISEASES
   2.00    9236 FIND CT D NOISE
   3.00     330 FIND 1 AND 2
   4.00     298 FIND 3 AND PY>=1970
   5.00     200 FIND 4 AND LA=(EN;GE;DU)
   6.00  147130 FIND CT D BLOOD PRESSURE
   7.00     222 FIND 2 AND 6
   8.00     152 FIND 7 NOT 3
   9.00     138 FIND 8 AND PY>=1970
  10.00     106 FIND 9 AND LA=(EN;GE;DU)
  11.00     306 FIND 5 OR 10
  12.01       0 DUPLICATE CANDIDATES IN S= 11.00  (OUTPUT
                ONLY)
  12.02       0 DUPLICATES REMOVED FROM S= 11.00  (OUTPUT
                ONLY)
  13.00     306 UNIQUE               IN S= 11.00

BASE COMMAND ACCEPTED FOR EM74;Embase;LAST-
UPDATE=30.10.1998
Copyright Elsevier Science B.V. 1998. All Rights Reserved.
For Menu driven search enter : CALL MENU
t hc
   1.00  717226 FIND CT D CARDIOVASCULAR DISEASES
   2.00   13186 FIND CT D NOISE
   3.00     415 FIND 1 AND 2
   4.00     415 FIND 3 AND PY>=1970
   5.00     369 FIND 4 AND LA=(EN;GE;DU)
   6.00  106923 FIND CT D BLOOD PRESSURE
   7.00     196 FIND 2 AND 6
   8.00     130 FIND 7 NOT 3
   9.00     130 FIND 8 AND PY>=1970
  10.00     116 FIND 9 AND LA=(EN;GE;DU)
  11.00     485 FIND 5 OR 10
  12.01     120 DUPLICATE CANDIDATES IN S= 11.00  (OUTPUT
                ONLY)
  12.02     119 DUPLICATES REMOVED FROM S= 11.00  (OUTPUT
                ONLY)
  13.00     366 UNIQUE               IN S= 11.00
  14.00    9321 FIND CT=(NOISE;NOISE EXPOSURE;NOISE
INJURY;NOISE NUISANCE;NOISE
                  POLLUTION;NOISE
                  SOUND;NOISE,TRAFFIC;INDUSTRIAL NOISE)
  15.00    7355 FIND 14/IM=1
  16.00     299 FIND 5 AND 13
  17.00      80 FIND 16 AND 15
  18.00      67 FIND 10 AND 13
  19.00      36 FIND 18 AND 15
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POOLKEY            = BA93
DATABASE NAME      = Biosis Prev AB
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FIRST ENTRY        = 1.01.1993
LAST  UPDATE       = 26.10.1998 05:25
UPDATE PERIOD      = WEEKLY
?
t hc
   1.00    6796 FT=NOISE
   2.00  429631 FIND CARDIOVASCULAR OR ISCHAEM? OR ISCHEM?
OR ANGINA PECTORIS
                  OR MYOCARD? OR CORONARY OR VASCULAR
                  DISEASE? OR CARDIAC OR BLOOD PRESSURE
                  OR HYPERTENS?
   3.00  114159 FT=HEART
   4.00      92 FIND 1/(TI;UT;CT) AND (2 OR 3)/TI
   5.00     258 FIND 1/(TI;UT;CT) AND (2 OR 3)/(TI;UT;CT)
   6.00      40 FIND 5 AND (EXPOS? OR HAZARD? OR DISEASE?
                OR CARDIO?)/TI
   7.00     106 FIND 4 OR 6
   8.01      34 DUPLICATE CANDIDATES IN S=  7.00  (OUTPUT
                ONLY)
   8.02      34 DUPLICATES REMOVED FROM S=  7.00  (OUTPUT
                ONLY)
   9.00      72 UNIQUE               IN S=  7.00
****END OF TAB*
POOLKEY            = BA70


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NUMBER OF RECORDS  = 8.123.668
FIRST ENTRY        = 1.01.1970
LAST  UPDATE       = 31.12.1992 23:59
UPDATE PERIOD      = NONE
?
t hc
   1.00   15079 FT=NOISE
   2.00  449557 FIND CARDIOVASCULAR OR ISCHAEM? OR ISCHEM?
                OR ANGINA PECTORIS
                  OR MYOCARD? OR CORONARY OR VASCULAR
                  DISEASE? OR CARDIAC OR BLOOD PRESSURE
                  OR HYPERTENS?
   3.00  188779 FT=HEART
   4.00     241 FIND 1/(TI;UT;CT) AND (2 OR 3)/TI
   5.00     527 FIND 1/(TI;UT;CT) AND (2 OR 3)/(TI;UT;CT)
   6.00     139 FIND 5 AND (EXPOS? OR HAZARD? OR DISEASE?
                OR CARDIO?)/TI
   7.00     288 FIND 4 OR 6
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                ONLY)
   8.02      76 DUPLICATES REMOVED FROM S=  7.00  (OUTPUT
                ONLY)
   9.00     212 UNIQUE               IN S=  7.00
****END OF TAB*


POOLKEY            = IS74
DATABASE NAME      = SCISEARCH
NUMBER OF RECORDS  = 16.631.495
FIRST ENTRY        = 1.01.1974
LAST  UPDATE       = 6.11.1998 06:25
UPDATE PERIOD      = WEEKLY
?
t hc
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   2.00  500007 FIND CARDIOVASCULAR OR ISCHAEM? OR ISCHEM?
                  OR ANGINA PECTORIS OR MYOCARD? OR
                  CORONARY OR VASCULAR DISEASE? OR
                  CARDIAC OR BLOOD PRESSURE OR HYPERTENS?
   3.00  188999 FT=HEART
   4.00     261 FIND 1/(TI;UT;CT) AND (2 OR 3)/TI
   5.00     323 FIND 1/(TI;UT;CT) AND (2 OR 3)/(TI;UT;CT)
   6.00     107 FIND 5 AND (EXPOS? OR HAZARD? OR DISEASE?
                OR CARDIO?)/TI
   7.00     272 FIND 4 OR 6
   8.01     174 DUPLICATE CANDIDATES IN S=  7.00  (OUTPUT
                ONLY)
   8.02     174 DUPLICATES REMOVED FROM S=  7.00  (OUTPUT
                ONLY)
   9.00      98 UNIQUE               IN S=  7.00
****END OF TAB*

0 DUPLICATES REMOVED FROM S= 11.00  (OUTPUT ONLY)
  13.00

  15.00    7355 FIND 14/IM=1
  16.00     299 FIND 5 AND 13
  17.00
```

We feel it is especially significant that a range of observed end points is consistent with known cardiovascular disease progression. Small, transient, stress-related hemodynamic responses that are harmless on the individual level may result in slight but relevant shifts in blood pressure on the level of populations. In a smaller, susceptible proportion of the population, this shift may lead to an increase in diagnosed hypertension, medication use, visits to the GP, and eventually the prevalence of IHD, including angina pectoris and myocardial infarction (Figure 1). In this perspective, additional cases of myocardial infarction attributable to noise exposure can be regarded as merely the tip of the iceberg.

The evidence for a relation between noise and cardiovascular disease is still inconclusive, not only because of the complexity with regard to noise and health outlined here, but also because of limitations in exposure characterization, blood pressure measurement and/or definition of hypertension, adjustment for important confounders, and the occurrence of publication bias. Therefore, we recommend more large follow-up studies. Exposure characterization could be improved by repeated personal dose measurements in a representative sample of the study population and reporting of more noise-related factors such as exposure duration and intensity. Furthermore, we should study health end points such as angina pectoris and myocardial infarction as well as others.

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Appendix 3. Why the exponential model?

When we started this study, the shape of the relation between noise exposure and coronary heart disease was not clear: linear, exponential, with or without threshold value? To get an idea of the shape, we plotted the noise exposure levels (extracted from the studies) against the prevalence of the effect in question. These plots showed that it was not possible to indicate a threshold value. This result is not consistent with those of some other studies, that there is a threshold value of 70 dB(A) (*1,79*). Furthermore, the plots showed that the shapes of the dose–response relations were not specific. Therefore, we decided to use two models for the meta-analysis: an exponential model (as presented in the article) and an additive model, defined as

(a) $$\beta_{i,Additive} = \left(\frac{RR-1}{\Delta dB(A)}\right) \times 5,$$

(b) $$\sigma_{i,additive} = \left(\frac{RR_{hi} - RR_{lo}}{3.92}\right) \times \left(\frac{5}{\Delta dB(A)}\right),$$

where $\Delta dB(A)$ is the difference in noise levels between the index and reference group; *RR* is the relative risk extracted from study or calculated with Epi-info; RR_{lo} is the lower level of *RR*; RR_{hi} is the upper level of RR; $\beta_{i,additive}$ is the estimated change in risk per 5 dB(A); and $\sigma_{additive}$ is the estimated standard error of β_i.

The additive model assumes that the increase in prevalence per unit of noise [dB(A)] is constant. The exponential model assumes a constant RR per unit of noise, which suggests an exponential relation between noise exposure and the prevalence of the effect concerned. The results of the meta-analysis showed that the associations found per 5 dB(A) with the additive model were stronger than those found with the exponential model, but that both models led to the same conclusions. To find out whether the models were valid, we plotted the noise levels of the reference groups (extracted from the studies) against the beta per 5 dB(A) of the different exposure groups. These plots showed that neither model shows clear dependence on the background levels. Therefore, both models seem to fit the data. Because the exponential model is most commonly used, we present only the results of the exponential model.

Appendix 4. Equations used for recalculations.

(a) $$\beta_{i,bloodpressure} = \left(\frac{\Delta Bloodpressure}{\Delta dB(A)}\right) \times 5,$$

(b) $$SE_i = \frac{SD_i}{\sqrt{N}},$$

(c) $$\sigma_{i,bloodpressure} = \left[\left(\frac{\sqrt{\left(SE_i^2 + SE_{ii}^2\right)}}{\Delta \mathrm{dB(A)}}\right) \times 5\right]^2,$$

(i) $$L_{Aeq,\,7-19\,hr} = 0.555 \times B_{GL} + 44 \quad (74),$$

where $\Delta Bloodpressure$ is the difference in systolic or diastolic blood pressure; $\Delta dB(A)$ is the difference in sound levels; SE_i is the standard error of systolic or diastolic blood pressure in group i; SE_{ii} is the standard error of systolic or diastolic blood pressure in group ii; SD_i is the standard deviation of systolic or diastolic blood pressure; *N* is population β_i is the estimated change in blood pressure or risk per 5 *dB(A)*; σ_i is the estimated standard error of β_i. B_{GL} is traffic noise exposure in Kosten units. In The Netherlands, air traffic noise exposure (B_{GL}) is expressed in Kosten units (KE). Kosten developed this measure in 1963. Modifying factors are: maximum noise levels (LA,max) during the overflights, the total number of overflights and the overflight-times, averaged over one year with adjustment for the number of night overflights (*78*); and $L_{Aeq,\,7–19hr}$ is the equivalent noise exposure level during day-time in dB(A)

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