Feature

Effects of Noise on Health

Chapter 3 of a report on *Noise and Health* prepared by a committee of the Health Council of The Netherlands

In 1994, the Health Council of The Netherlands was asked to prepare an advisory report on the effects of noise on human health. In the report to the Minister of Health, Welfare, and Sports, and to three other Ministers in The Netherlands, Professor L. Ginjaar, President of the Health Council of the Netherlands, said that the committee responsible for the report asked him to draw the Minister's attention to the following:

A Health Council Committee concluded in 1971 that noise exposure is an important public health problem. Since then, the knowledge about the effects of noise on health has increased considerably. However, this has not lead to new insights. Also, today noise exposure presents a considerable public health problem, as the data in the committee's report illustrate. This implies that the measures that have been taken in the last decades have had only a limited effect, which is partly due to the increase in exposure. In my opinion, abatement of noise annoyance, of noise-induced hearing loss, and of other effects of noise on health should be an important part of a public health policy.

The report consists of an Executive Summary and four chapters: 1) Noise as a Public Health Problem, 2) Health and Noise Exposure, 3) Effects of Noise on Health, and 4) Noise Exposures in The Netherlands and Their Effects. This feature article is a slightly edited version of Chapter 3, Effects of Noise on Health, which is reprinted with the permission of Dr. W. Passchier-Vermeer, scientific secretary of the Health Council of the Netherlands. — Ed.

Noise-Induced Hearing Loss

Occupational Noise Exposure

Relations Between Noise Exposure and Noise-Induced Hearing Loss

The second edition of ISO 1999 "Acoustics -Determination of occupational noise exposure and estimation of noise-induced hearing impairment" (ISO90) gives a calculation method for the determination of hearing threshold levels of populations exposed to all types of noise (steady-state, intermittent and impulse) during working hours. The noise exposure is characterized by the noise exposure level, L_{EX}. In this report L_{EX} is denoted by $L_{EX,occ}$, indicating the exposure concerns *occupational* noise. Relations are given between $L_{EX,occ}$ and noise-induced permanent threshold shift (NIPTS) for frequencies in the range from 500 to 6000 Hz, and for exposure times up to 40 years. These relations are expressed in statistical terms (median values of NIPTS as well as values from the 0.05 to the 0.95 fractile). The relations show that NIPTS is a phenomenon which occurs predominantly in the higher frequency range from 3000 to 6000 Hz; the effect is largest at a frequency of 4000 Hz. With increasing equivalent sound level and increasing exposure time, hearing loss also occurs at the lower frequencies, more specifically at 2000 Hz. For prolonged occupational noise exposure, ISO 1999 shows that permanent threshold shift is not induced by noise with $L_{EX,occ}$ values at and below A-weighted sound levels of 75 dB(A). (*In this report, the unit of level is the decibel (dB). However, the unit symbol is followed by (A) to denote the A-weighted sound level in decibels [dB(A)]. — Ed.*).

The value of 75 dB(A) below which there occurs no noise-induced permanent threshold shift from occupational noise exposure had already been given by the World Health Organization in 1980 (WHO80). Also, the draft Physical Agents Directive of the European Union specifies this level at 75 dB(A).

Exposure to Impulse Noise

There is evidence that temporary effects on hearing from exposure to impulse or impact noise are different from those of exposure to more or less steady-state noise. However, epidemiological studies could not show any systematic difference between permanent threshold shifts from occupational exposure to impulse or impact noise and from steady-state noise (Pas89a) with the same equivalent sound level. Regarding shooting noise this seems to hold only for equivalent sound levels up to 85 dB(A) over a period of 8 hours; for higher equivalent sound levels shooting noise may be more damaging than should have been expected from its equivalent sound level (Smo82).

At very high levels mechanical damage of the hearing organ may occur. To avoid this, adults should not be exposed to *peak* levels exceeding 140 dB. Possibly, for children a lower value is appropriate. This value is, as yet, unknown.

Identifying Sensitive Persons

ISO 1999 shows that variation in human sensitivity to noise-induced permanent threshold shift increases with noise exposure level; variation is considerable at high equivalent sound levels. However, there are as yet no tests for identifying individuals that may be susceptible to noise-induced hearing loss before the hearing damage occurs. ISO 1999 presumes females and males to be equally susceptible.

Non-Occupational Noise Exposure

Non-occupational noise exposure can be divided into four categories:

- exposure to environmental noise in the living environment such as: traffic, industrial and residential noise
- exposure to noise from home-based activities
- exposure to noise from traffic during travel between home and work/school
- exposure to noise during leisure (*noise includes music*).

The committee is of the opinion that extrapolation of the calculation scheme in ISO 1999 to (a combination of) the daily non-occupational noise exposures as specified above is justified. This implies that, for adult populations, a permanent threshold shift is not induced by noise with $L_{Aeq,24h}$ values at and below 70 dB(A), whether noise exposure is prolonged or not.

The committee regrets the lack of information on the patterns of exposure of populations to non-occupational noise. Due to this lack of information, only global estimates can be made and general conclusions be drawn concerning noise-induced hearing loss from non-occupational noise exposure.

Susceptible Groups

A pregnant woman's exposure to noise may affect the hearing of the unborn child. The two epidemiological studies that examined the hearing acuity of young children with mothers who had been exposed to occupational noise during pregnancy, both showed an increase in percentages of children with high-frequency hearing loss. On the basis of these results, the committee concludes that equivalent sound levels of 85 dB(A) or higher during an 8-hour working day appear to be detrimental to the hearing of the unborn child. It recommends that further research should be undertaken to verify whether, at equivalent sound levels lower than 85 dB(A), increased hearing loss in young children occurs, especially when it concerns exposure to low frequency noise and vibrations.

Data from animal experiments indicate that young children may be more susceptible to noise-induced permanent threshold shift than adults. Such an increased susceptibility has not been confirmed by epidemiological studies in human populations. Spreng (Spr90) considers a difference of 5 dB(A) applicable for certain types of exposures. (*This concerns exposures with rapid increases of the sound level, such as in the case of low-flying fighter-jets. The middle ear of children may then react differently from that of adults.*)

Males exposed to occupational noise, who have high plasma cholesterol levels in blood, have an increased risk of noise-induced hearing loss in comparison to occupational noise exposed male populations with normal cholesterol levels (Axe85a).

Social Consequences of Hearing Loss

The main social consequence of hearing damage concerns the inability to understand speech under day to day living conditions. Since speech is the most common means of communication between people, a decreased understanding of speech should be considered a severe social handicap.

In the case of the combination of age-related hearing loss (presbycusis) and occupational noise-induced hearing loss, decrease in speech intelligibility is a process which may proceed over years. Understanding speech first starts to become difficult in noisy surroundings (cafeterias, parties, noisy meetings). Next, difficulties occur during church services, theatrical performances and public meetings, even when people with hearing damage place themselves close to the speaker. Once the hearing impaired start compensating for their handicap, others will recognize the decreased hearing capacity. In

the next stage, telephone calls start to present problems and conversations in fairly quiet surroundings become difficult, the more so when they involve strangers. Eventually, understanding the speech of close friends and family starts to become critical. A decreased hearing capacity can be partially compensated by lip reading, even without the hearing-handicapped listener being aware of it.

Even small values of hearing damage may have an effect on understanding speech in normal live. In investigations of groups of people with noise-induced hearing loss, a decrease in speech understanding has been observed at hearing threshold levels from 10 dB, averaged over 2000 and 4000 Hz and averaged over both ears (Smo86, Pas85). When the hearing threshold level exceeds 30 dB, again averaged over 2000 and 4000 Hz and over both ears, the hearing damage becomes a noticeable social handicap (Smo86, Pas87a,b).

Classification of Health Effects

The committee is of the opinion that there is sufficient evidence for a causal relationship between noise and hearing loss. Exposure-effect functions are specified in ISO 1999. For occupational noise exposure $L_{EX,occ}$ is taken as noise measure and for non-occupational noise exposure $L_{Aeq,24h}$ is the measure to be used. Observation thresholds correspond to a value of $L_{EX,occ}$ of 75 dB(A) and a value of $L_{Aeq,24h}$ of 70 dB(A).

Although there is sufficient evidence for a causal relationship between occupational noise exposure during pregnancy and hearing loss in babies, the available data do not allow it to be specified whether and to what extent hearing loss occurs below a value of $L_{EX,occ}$ of 85 dB(A).

Noise-induced Stress-related Health Effects

Stress

The reactions to a stressor can be of a psychological, behavioral and somatic nature. Psychological effects concern feelings of fear, depression, frustration, irritation, anger, helplessness, sorrow and disappointment. Examples of behavioral reactions to a stressor are social isolation, aggression and resort to excessive use of alcohol, tobacco, drugs or food. Psychological and behavioral stress may have a direct or indirect effect on physiological processes within the body. (*In this respect, it is not always obvious in the analysis of the results of an epidemiological study whether observed differences in behavior of a noise-exposed group and of a group of people not exposed to noise should be considered as a direct or indirect result of exposure to noise or as a confounding factor. Take as an example effects of traffic noise on the prevalence of ischaemic heart disease and on smoking, presuming that smoking is a risk factor for this heart disease. It could be argued that smoking is associated with stress and that due to stress from daily exposure to high levels of road traffic noise, the relative number of people smoking and the cigarettes smoked increase. Smoking should then not be considered as a confounding factor and corrections should not be applied on the test results with respect to this factor. On the other hand, should smoking be considered as a confounder, corrections should be applied, when exposure to noise is associated with ischaemic heart disease in the analysis of the test results.*) A great number of laboratory experiments have demonstrated changes in various somatic, physiological and biochemical factors in humans due to acute noise exposure. These experimental studies show that noise should be considered as an unspecific stressor that stimulates central nervous system and hormonal activity (Isi93, Mar88, Mar90).

Research into long-term noise-induced stress-related health effects has been limited mainly to cardiovascular disorders. To a far lesser extent, epidemiological research has been carried out regarding changes in biochemical parameters and parameters of the immune system. There is a complicated interaction between the hormonal and immune system. Hormones produced by the pituitary gland interact with immune factors, whereas both hormones and immune factors have an impact on the brain. The connections with parts of the limbic system, the system which largely determines emotional activity, are also of importance.

Research into the chronic effects of long-term exposure to noise involves inherent difficulties:

- Cardiovascular and biochemical changes are non-specific; a number of other factors may cause these changes, some of the factors possibly not yet being identified. A major problem in epidemiological research is to control these factors.
- In epidemiological research, it is time-consuming and difficult to obtain good quantitative data about the noise exposure, especially about past exposure. For example, noise maps of cities may be used in road traffic noise studies. Using these maps could give a non-systematic misclassification of the noise exposure of some inhabitants. Such a misclassification will obscure a noise-induced effect.
- People intervene to a certain extent in their own living and working situation, e.g. by moving to more quiet surroundings or by changing their job.

This may result in a selection in which people who are *noise-proof* will remain in noisy situations and those who are not will leave the situation.
- There are great differences in individual susceptibility.

Cardiovascular Effects in the Working Environment

Epidemiological research into the long-term stress-related health effects has been focused on changes in the blood pressure of workers exposed to occupational noise and on the prevalence of hypertension among these workers (Dij84, Isi80a, Isi93; for other references see Pas93a,b). Hypertension has been defined, according to the World Health Organization, as a systolic blood pressure of at least 160 mmHg (*1 mmHg corresponds to approximately 0.13 kPa*) and/or a diastolic blood pressure of at least 95 mmHg.

The committee concludes that prolonged exposure to occupational noise may contribute to increased blood pressure and hypertension. These effects have been shown to occur at equivalent sound levels during the working day of at least 85 dB(A). Effects of chronic exposure at lower noise levels such as in offices have hardly been studied.

Other noise-induced effects on the cardiovascular system have been observed in workers exposed to high or extremely high equivalent sound levels during the working day, such as an increase in abnormalities in the electrocardiogram, more heart beat irregularities, faster pulse rate, faster increase in heart rate during a physical test and slower recovery of vascular constriction during a noise exposure test. Apart from abnormalities in the electrocardiogram, the other noise-induced effects seem not be detrimental to health, taken into account the extent of the effects in so far they were due to noise exposure.

Cardiovascular Effects in the Living Environment

Long-term effects of exposure to noise in the living environment have only been investigated in relation to road and air traffic noise to which people are exposed in their own homes (Alt87, Alt89, Bab88, Bab90, Bab92, Bab93a,b, Bie89a,b, Isi80b, Isi93, Jon92b, Kni76; for other references see Pas93a,b). These exposures are usually much lower than those to occupational noise, but the exposed population is much greater. A complicating factor in the determination of noise exposure in homes is that people are not only exposed to traffic noise, but also to various, often even louder, noises from other sources. Furthermore, housing features (e.g. single or double glazing) and personal habits (e.g. closing windows, moving to quieter sides of the house, staying indoors during the summer) affect the actual noise exposure.

Several studies on the effects of traffic noise have had the occurrence of changes in blood pressure and hypertension, and the risk of ischaemic heart disease as their subject. Epidemiological studies show that, in general, there are no obvious effects from exposure to traffic noise on the mean systolic and diastolic blood pressure, except in children. However, the committee considers the observed increase of, at most, 10 to 15 mmHg (Coh80, Kar68) in the average systolic and diastolic blood pressure in children to be of a temporary nature and not relevant for permanent health damage.

The committee draws the following conclusions from the results of epidemiological research:

- there is little evidence for an increased risk of hypertension and of ischaemic heart disease in people living in areas with traffic noise at outdoor equivalent sound levels (from 0600 to 2200 hours) below 70 dB(A).(*There are some indications that this value might have to be lowered to 65 dB(A) once the results of additional studies become available.*)
- the relative risk of ischaemic heart disease and of hypertension starts to increase for persons living in areas with road or air traffic noise at equivalent sound levels above 70 dB(A) (from 0600 to 2200 hours).

Biochemical Effects

Epidemiological studies on the effects of high to very high environmental and occupational noise exposures on the biochemical (*this concerns specific hormones and metal-ions (Mg^{2+})*) composition of the blood of exposed people mostly show noise-induced changes which should be expected if noise acts as stressor. Several studies also show changes which indicate an increased risk of ischaemic heart disease (Bab88, Bab90, Bab92, Bab93, Isi80b,c). However, there are only limited data available. Therefore the committee is unable to establish to what extent changes in blood composition occur under which particular environmental and occupational circumstances. However, laboratory studies with volunteers show that such effects may occur.

Effects on the Immune System

No epidemiological investigations except for the Caerphilly and Speedwell Collaborative Heart Disease Studies (Bab92, Bab93) have been carried out into the effects of noise on the immune system. This study has revealed an increased concentration of

leucocytes in blood in the case of exposure to high levels of road traffic noise.

Effects on the immune system might ultimately lead to an increased prevalence of infectious diseases, such as influenza and inflammations, and possibly cancer. No epidemiological studies concerning such effects of noise exposure have been reported.

Effects on the Unborn Child

In view of the available research data, it cannot be excluded that noise exposure of pregnant women to air traffic noise in the *living* environment may affect the birthweight of the baby. Should a reduced weight at birth occur, this is only at noise exposures with L_{dn} values greater than 62 dB(A) (more than 40 Ke). The available data virtually exclude an aircraft noise-induced risk of the occurrence of congenital defects.

The studies on the health of babies whose mothers were exposed to *occupational* noise during pregnancy suggest that there does not seem to be a higher risk of lower birthweight and of premature birth; the results with regard to congenital defects are contradictory, whereas those related to increased risk of spontaneous or imminent abortion and death at birth are questionable.

Susceptible Groups

People highly annoyed by low levels of road traffic noise have an increased risk of hypertension. Men exposed to high levels of road traffic noise in the living environment and also exposed to occupational noise have an increased risk of ischaemic heart disease compared to men exposed to road traffic noise only (Bab90). Pregnant women exposed to occupational noise show an increased risk of hypertension during pregnancy, compared to pregnant women not exposed to occupational noise. People with noise-induced sleep disturbance have an increased risk of hypertension and ischaemic heart disease compared with people in the same living environment without sleep disturbance (Isi93). Exposure of hospitalized patients to relatively high levels of noise from sources inside or outside the hospital delays recovery and wound healing.

Classification of Health Effects

The committee is of the opinion that the following classifications are applicable:

- biochemical effects: limited evidence
- hypertension: sufficient evidence
- ischaemic heart disease: sufficient evidence
- effects on immune system: limited evidence
- birthweight: limited evidence
- congenital defects: evidence suggesting lack of a causal relationship.

For occupational industrial noise-induced hypertension, the observation threshold probably has a value of $L_{EX,occ}$ below 85 dB(A). For groups exposed to values of $L_{EX,occ}$ of 90 dB(A) and above the relative risk is 1.7.

For environmental road- and air traffic noise-induced hypertension the observation threshold has a value of $L_{Aeq,06\text{-}22h}$ of 70 dB(A) (measured outdoors). For ischaemic heart disease the same value is applicable. Groups exposed to higher values (70 to 80 dB(A)) will have a relative risk of hypertension and of ischaemic heart disease of about 1.5.

Psycho-social Effects

Subjects studied in epidemiological research with respect to psycho-social effects from noise in the living environment include noise annoyance, effects on psycho-social well-being and the question of whether noise-induced feelings of irritation have such an impact that they increase the number of admissions to psychiatric hospitals. Effects studied in the working environment concern annoyance and increased absenteeism from the worksite.

Noise annoyance is a feeling of resentment, displeasure, discomfort, dissatisfaction or offence which occurs when noise interferes with someone's thoughts, feelings or actual activities. The capacity of a given sound to annoy depends on its physical characteristics including sound level, spectral characteristics and variations with time. These variables are characterized by onset times, durations and repetition rates. However, annoyance also depends on non-acoustical, cognitive factors, such as fear with regard to the noise source, the conviction that the noise exposure could be reduced by third parties, individual noise sensitivity, the degree to which an individual feels able to control the noise, whether the noise stems from a new situation or technology, and, to a lesser extent, the recognition that the noise source gives rise to problems other than mere noise exposure or that it results from an important economic activity. Demographic variables - age, sex, socio-economic status - are almost unrelated to annoyance from a given noise source.

Noise annoyance and psycho-social well-being can both be evaluated using questionnaires. Psycho-social well-being concerns depression, relaxation, activity, passivity, aggression, general well-being and social aspects, such as group interaction and willingness to help.

Annoyance in the Living Environment

Noise from Road Traffic, Trains and Airplanes
Recently defined exposure-effect functions relate annoyance to exposure to various types of traffic noise in the living environment (Mie92). Severe annoyance by noise from several types of traffic (aircraft, highway traffic, other road traffic, railroad traffic) starts to occur at L_{dn} values of 42 dB(A), annoyance starts at L_{dn} values of 37 dB(A) and some annoyance at 32 dB(A). (*Miedema defines severe annoyance as annoyance of at least 72 (on a scale with a lower boundary of 0, corresponding to being not at all annoyed, and an upper boundary of 100, corresponding to being extremely annoyed) (Mie92).*) These L_{dn} values were measured outdoors, in front of the dwellings. Annoyance increases most with L_{dn} for aircraft noise, followed by highway traffic noise, other road traffic noise and railroad noise. For the traffic noise exposures that were considered, there is a close relationship between $L_{Aeq,24h}$, L_{etm} and L_{dn}, due to a high correlation between the equivalent sound levels during the day and those during the night.

Noise from High-speed Trains
Noise from high-speed trains is of special importance these days, in view of the plans for a high-speed train network in Europe, including The Netherlands. Based on measurements made abroad, on the acoustical characteristics of this type of train noise and on the projected future use of high-speed trains, De Jong (Jon93) concludes that annoyance from noise produced by high-speed trains in the Netherlands will not exceed annoyance caused by conventional trains with equal L_{etm} values.

Noise from Helicopters and Small Aircraft

Noise from helicopters differs from that of conventional airplanes due both to the characteristic sound of the rotating blades (blade slap) and to the helicopter's lower speed, which make helicopters audible during a longer period. Furthermore, helicopters do not only pass an area, but often also circle above it for some time. This last comment is also applicable to some types of small aircraft.

Annoyance from passing helicopters and small aircraft has been found to be comparable to annoyance from conventional aircraft only when the duration of the noise is taken into account. Expressing exposure to helicopter noise in Ke is therefore not advisable, as this measure does not take the duration of noise events into account.

Noise of Low-flying Fighter Jets

Noise of low-flying fighter jets, with flight paths with a minimum height of 75 meter, contrasts with that of civil aviation in several aspects:

- under the low-flying corridor, the maximum sound level of an overflight is relatively high
- this very high level is not restricted to the surroundings of the airport, since low-flying corridors can be situated elsewhere
- the onset time of the noise from a low-flying fighter jet is relatively short.

It is estimated that noise from low-flying fighter jets is as annoying as noise from conventional aircraft with a 10 dB(A) higher equivalent sound level (Pas93a,b). Other effects in addition to annoyance, are to be expected. These include psychological effects such as fear and panic in adults and children.

Other Noise Sources in the Living Environment
Noise from stationary sources, such as industry, railroad shunting yards and artillery-ranges is more annoying than traffic noise, especially when the noise contains impulse or impact components (Vos85a, Vos85b). Annoyance from shunting-yards is comparable to that from passing trains for L_{dn} values up to about 60 dB(A), but is much more annoying at higher levels (Mie92).

There exists a relation between annoyance from indoor noises from neighboring dwellings and the sound insulation between dwellings: low values of sound insulation resulting in high percentages of people annoyed. Due to the large variability in the levels of outdoor noises from neighbors and noises in the neighborhood of private homes, e.g., people shouting, slamming car doors, the sound of car horns and lawn-mowers, and the variety of non-acoustical factors that also determine annoyance, it is difficult if not impossible to determine exposure-effect relations for these types of noises.

Accumulation of Noise Exposures
When people are exposed to more than one environmental noise source, annoyance is cumulated. Weighted summation of the annoyance effects provides a fair description of the accumulation (Mie93, Vos92). The resulting annoyance from two noise sources is sometimes much larger than the annoyance expected from the most disturbing source alone (Mie93).

Psycho-social Well-being

The limited research carried out with respect to effects from exposure to road traffic noise on psycho-social well-being does not permit a definite conclusion. Two investigations showed psycho-social well-being to be decreased in people living in

very noisy areas (equivalent sound levels during the daytime over 70 dB(A)) compared to that of people living in quiet surroundings. This concerned social orientation, activity and depression. The third investigation showed psycho-social well-being of people not to be related to the noise level as determined in front of their dwelling, but to their noise sensitivity and to the extent to which noise penetrates into their bedroom and disturbs sleep (Ohr89, Ohr91).

Effects on Admission to Psychiatric Hospitals

For some people, psychological stress may lead to admission to psychiatric hospitals. A number of factors other than noise exposure in the living environment are involved in such admissions. The effect of aircraft noise in this respect has been studied in the vicinity of Heathrow Airport in the past twenty years. Taking into account several intervening factors, the most recent analysis showed a statistically significant increase in the percentage of admissions to psychiatric hospitals with exposure to aircraft noise. In areas with L_{dn} levels of more than 70 dB(A) (B more than 55 Ke) due to air traffic noise, admission to psychiatric hospitals was higher than in areas with L_{dn} values of less than 65 dB(A) (B less than 45 Ke); the prevalence ratio found was 1.1 (Kry90). However, since a causal relationship was shown in only one investigation and in only one analysis, the committee is of the opinion that care should be taken to generalize this relation to other situations and other populations.

Annoyance in the Work Environment

No relations have been established between noise annoyance experienced during working hours and noise level (Mie85). Only a very small part of the variance in annoyance in the workplace is attributable to variations in noise exposure. The following non-acoustic variables have a much greater effect than noise level on annoyance during working hours:

- meaningfulness and information content of the noise (discussions by colleagues in the surroundings of the workplace score high in this respect)
- predictability, avoidability and controllability of the noise
- attitude of the workers towards the noise source
- task demand
- individual susceptibility.

Annoyance in offices is already considerable at equivalent sound levels above 55 dB(A) during working hours. The few available results of epidemiological investigations show that 35% to 40% of the office workers exposed to an equivalent sound level of 55 to 60 dB(A) are severely annoyed. In industrial situations similar percentages of annoyed workers occur at equivalent sound levels over 85 dB(A). These results do not allow the determination of observation thresholds for annoyance in office and industrial workers.

Effect on Absenteeism in the Work Environment

Epidemiological studies suggest that the absentee rate increases if workers are exposed to higher equivalent sound levels during work. This was demonstrated for various industrial situations at equivalent sound levels higher than 75 dB(A) in one study (Mel92: CORDIS-study: Cardiovascular Occupational Risk Factor Detection in Israel (*In this study the prevalence ratio was 1.2 for equivalent sound levels from 75 to 85 dB(A) and 1.7 for higher sound levels.*) and in another study in the coal and steel industry (Sch91) at equivalent sound levels higher than 90 dB(A) (*in this study the prevalence ratio was 1.1*). On a small scale, a statistically significantly higher absentee rate was shown in office workers who were (very) frequently exposed to clearly audible noise events, compared to those seldom exposed to such events (Sch82) (*in this study the prevalence ratio was 1.3*). However, some of these studies have insufficiently taken into account confounding variables and others are flawed in other aspects. Therefore, the committee concludes that no causal relationships between absentee rate and industrial occupational noise exposure or exposure to noise in offices has yet been demonstrated conclusively.

Sensitive Groups

People annoyed by noise in the workplace show an increased post-work irritability which might affect their general well-being. Noise-sensitive people, people with fear of certain noise sources and people feeling they have no control over a noise situation (i.e., feel an abuse of power) have an increased risk of severe annoyance.

Classification of Health Effects

The committee is of the opinion that the following classifications are applicable:

- annoyance in the living and work environment: sufficient evidence
- psycho-social well-being: limited evidence
- admission to psychiatric hospitals: limited evidence
- absenteeism from work: limited evidence.

Exposure-effect functions have been specified (Mie93) for annoyance from environmental expo-

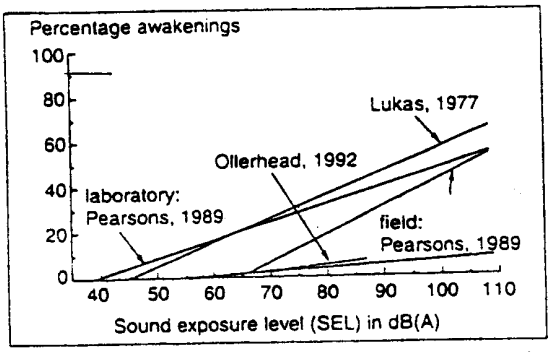

Fig. 1. Relations between the percentage of people with awakenings due to a night-time noise event and the indoor sound exposure level of such an event.

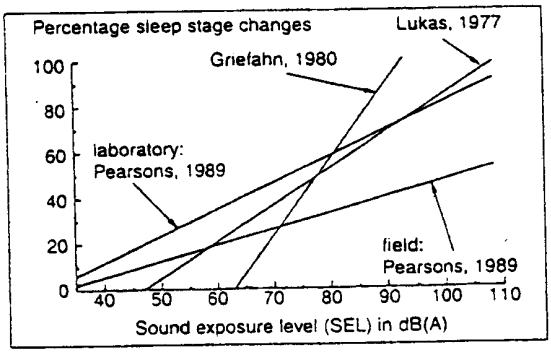

Fig. 2. Relations between the percentage of people with sleep stage changes due to a night-time noise.

sure to traffic and industrial noise. The observation threshold for severe annoyance corresponds to a value of L_{dn} of 42 dB(A).

Exposure-effect functions for annoyance from occupational noise exposure in offices as well as in industrial situations are lacking. Observation thresholds for office noise and for industrial noise exposure are well below $L_{EX,oce}$ values of 55 respectively 85 dB(A). At these values, the fraction of workers severely annoyed amounts to 35% to 40%.

Sleep Disturbance

Effect of Noise on Sleep

Night-time noises can disturb sleep (Gri76, Gri90a,b, Hof91, Hof92b, Jur83, Luk75, Mie93, Ohr83, Ohr88, Oll92, Pea89, WNN93). According to the advisory report of the Health Council on airplane noise and sleep (GR91), external factors such as noise may affect sleep in different ways, resulting in:

- degradation of sleep quality
- disturbance of functioning or performance the next day
- disturbance of mood the next day

Since many of the underlying experimental and epidemiological studies concern exposure to a wide range of types of noise sources, the conclusions about the influence of aircraft noise on sleep in the former Health Council report are also largely applicable to exposure to other types of intermittent traffic noises.

Effects on Sleep Quality

Sleep quality may be affected in various ways:

- by changes in sleep pattern
- by changes in sleep stages from deeper to less deep sleep
- by awakening during the sleeping period
- by changes in subjective assessment of sleep quality
- by changes in cardiovascular and hormonal parameters
- by changes in the immune system

Sleep Pattern

Night-time noise of sufficient intensity changes the sleep pattern in such a way that it increases the time awake during the sleep period and increases sleep latency (the time between lights out and falling asleep). According to the committee the results of experimental and epidemiological research do not permit the assessment of a level above which the sleep pattern starts to worsen. It is recognized, however, that at high levels of traffic noise a significantly greater percentage of the exposed population reports difficulties falling asleep than at lower levels.

Changes in Sleep Stages and Awakening

The sleep stages can be determined from electro-encephalograms (EEGs), measured while the subject is falling asleep and during sleep. The EEG is a continuous recording of the electrical activity of the cerebral cortex. The EEG, together with the electro-oculogram (EOG), indicate the sleep stages: W (waking), 1, 2, 3, 4, REM (Rapid Eye Movements).

For intermittent noise exposures such as produced by aircraft, trains and road traffic, various exposure-effect relations between the characteristics of night-time noise exposure and awakening and sleep stage changes have been derived. Figures 1 and 2 show these exposure-effect relations. The curves proposed by Griefahn (Gri76) and by Lukas (Luk75) are mainly derived from laboratory experiments. The curves of Pearsons (Pea89) distinguish between laboratory and epidemiological studies. The curve derived from the research by Ollerhead (Oll92) concerns epidemiological research. Comparison of the exposure-effect relations from field and laboratory studies supports the hypothesis that habituation results in fewer awakening reactions. This, however, seems less correct for

changes between different stages of sleep, a statement which is supported by the results of the joint European field investigation (Jur83) into sleep disturbance. In the two field studies (Pea89, Oll92), the onset of noise-induced awakenings is found to be at a SEL of about 60 dB(A), measured indoors. The onset of noise-induced changes between sleep stages is found at a SEL value of about 35 dB(A). Based on the preliminary exposure-effect relation derived from the two field studies, the total number of awakenings and sleep stage changes during all nights of the year have been estimated as a function of the equivalent sound level indoors during the night (2300-0700h) due to aircraft noise, where this equivalent sound level during the night has been taken on a yearly basis (Pas94). This equivalent sound level has been taken as the noise exposure measure in the preparation of legal requirements concerning night-time flights around main airports in the Netherlands (WNN93).

Subjective Sleep Quality

The subjectively experienced quality of sleep of people exposed to high levels of night-time noise is lower than that for non-exposed people, even for persons who have lived for years in noisy surroundings (Jur83, Ohr89, Ohr90, Ohr91, Sch90, Mie93). In one investigation (Mie93) data on self-reported sleep disturbances due to traffic noise were gathered from questionnaires on noise annoyance. Analysis of these data indicated that at outdoor equivalent sound levels during the night (2300-0700h) from 40 dB(A) subjective sleep quality started to decrease. The committee is of the opinion that there is yet insufficient information to permit determination of the exact exposure-effect relation between subjective sleep quality and night-time noise, especially not at the lower noise exposure values, but that it is justified to take an equivalent sound level of 40 dB(A) during the night as observation threshold.

Cardiovascular and Hormonal Parameters During Sleep

Night-time noise exposure may increase heart rate during the night; habituation to this effect does not seem to occur. The observation threshold is equal to a SEL value of 40 dB(A), measured indoors.

The effect of night-time noise on the endocrine system has so far not been investigated in epidemiological studies, but only in a laboratory study (Gru92, Mas92). The latter study concerned changes in epinephrine and norepinephrine excretion in urine as a function of aircraft noise exposure (*The hormones epinephrine and norepinephrine are also denoted by adrenaline and noradrenaline. They are hormones related to stress.*). Statistically significant effects could be observed at indoor equivalent sound levels of 35 dB(A) (64 overflights). The study has been reported (Isi93) to show a high correlation between epinephrine levels and sleep stage changes. The committee is of the opinion that further research is necessary before conclusions can be drawn for hormonal effects.

The Immune System During Sleep

Only in a Japanese laboratory study by Osada (Osa68, Osa69, Osa72, Osa74) in the period from 1968 to 1974 were changes in the percentages of leucocytes and granulocytes in blood measured. The committee does not consider the results of the Osada research as proof of an effect of noise exposure during sleep on immune system function. Although noise exposure at night may affect the immune system, as daytime noise exposure may do, experimental confirmation of such an effect is still lacking.

After-Effects

The performance during the day, in relation to noise exposure during the previous night, is usually measured by testing reaction time. Epidemiological research showed that the reaction time of residents exposed to night-time noise for years was longer when they had been exposed to more noise during the previous night (Jur83). The committee is, however, of the opinion that the available data do not allow levels to be specified at which these noise-induced effects on performance the next day start to occur. Most studies into the effect of night-time noise on mood the succeeding day showed a decrease in mood of persons exposed to high levels of night-time noise. An outdoors equivalent sound level of 60 dB(A) during the night is the observation threshold.

Sensitive Groups

Ill people, older people and people with sleeping difficulties show more noise-induced sleep disturbance, especially with respect to inability to fall asleep (after being awakened), than do other adults. Older people also have an increased risk of being awakened by night-time noise (WNN93).

Classification of Health Effects

According to the committee the following classifications apply:

- changes in sleep pattern: sufficient evidence
- changes in sleep stages and awakening: sufficient evidence
- subjective sleep quality: sufficient evidence
- heart rate frequency: sufficient evidence
- hormonal system: limited evidence

- immune system: inadequate evidence
- mood next day: sufficient evidence
  performance next day: limited evidence

Although the committee has concluded that there is sufficient evidence for a causal relationship between night-time noise exposure and various effects on sleep, exposure-effect functions are lacking for some of these effects. Exposure-effect functions have been specified for awakening and for sleep stage changes with exposure specified as SEL-values (Pea89). Observation thresholds for the various noise-induced effects are with exposure specified as SEL-values:

- awakening: a SEL value (measured indoors) of 60 dB(A)
- sleep stage changes: a SEL value (measured indoors) of 35 dB(A)
- changes in heart rate: a SEL value (measured indoors) of 40 dB(A)

The number of awakenings and sleep stage changes have been related to the equivalent sound level during the night due to aircraft noise (near main airports) with the noise exposure taken on a yearly basis (Pas94). (*For this specific situation the observation threshold might be an equivalent sound level of 16 dB(A) taken over 7 hours during the night and measured indoors.*)

The observation threshold for subjectively experienced deterioration of sleep quality is found at a value of $L_{Aeq,night}$ of 40 dB(A), measured outdoors. After-effects, the day following night-time noise exposure, on mood and, presumably performance, have observation thresholds at night-time equivalent sound levels of 60 dB(A) measured outdoors.

Effects on Performance

Laboratory studies with test subjects have shown that noise exposures may have a significant effect on performance. While a task is being performed, noise may in test subjects increase arousal, alter the choice of task strategy, and decrease attention to the task. Noise may also affect social performance, mask speech and impair communication and it may distract attention from relevant social cues. When a task involves auditory cues, and these auditory signals are masked by noise, this will have an effect on task performance.

Even relatively low noise levels may have acute adverse effects. It is obvious from laboratory experiments that to a large extent habituation occurs. Performance of a task involving motor and monotonous activities is not always disturbed by noise; noise (music) can also enhance performance in these situations.

Due to the complex character of noise-induced effects on task performance and the many non-acoustical factors involved, no exposure-effect relations were drawn up.

People whose performance strategies are already limited for other reasons and people who are faced with multiple tasks, putting requirements on short-term memory, may be more vulnerable to the distracting effects of noise.

Epidemiological research into effects on performance of schoolchildren has shown that these children, when exposed to very high levels of aircraft or road traffic noise (equivalent sound levels during schooltime over 70 dB(A), measured outside the school) do show an impaired performance in cognitive tasks. They are distracted more easily and make more mistakes when they are exposed daily to high noise levels, while at school (Coh80, Kar68).

The committee is of the opinion that there is limited evidence for a causal relationship between noise exposure as experienced under normal living conditions and decreased performance in adults. There is sufficient evidence in the case of schoolchildren.

Combinations of Noise Exposures

People may be exposed to different noise sources in the same situation, e.g., to a combination of road traffic and train noise in the living environment. People may also be exposed to different noise sources, acting on them in different situations at different times, such as a combination of occupational noise during working hours and road traffic noise while at home.

Accumulated Effects from Different Sources in the Same Situation

Miedema and Vos studied annoyance from two or more environmental noise sources; their work resulted in models for these accumulated noise effects (Vos92, Mie93). Further research may show whether these models are also appropriate for stress-related environmental noise-induced health effects and for sleep disturbance. The combined effect of different noise sources on hearing levels is related to the equivalent sound level of the combined exposure.

Accumulated Effects from Different Sources at Different Locations

Concerning noise-induced hearing loss, the committee considers it appropriate to estimate the accumulated effect of combined exposures based on the equivalent sound level over the total relevant exposure period.

The only epidemiological research into the combination of noise exposure in the living and in the working environment on stress-related effects (cardiovascular and biochemical parameters) showed that effects of road traffic noise in the living environment are more pronounced in men who were also working in high noise levels (equivalent sound levels over 90 dB(A)) than in men without occupational noise exposure (Bab90). In this respect, occupational noise exposure may be considered a risk factor for ischaemic heart disease for people exposed to high levels of environmental noise.

Concerning annoyance, the preliminary conclusion from the scarce epidemiological research is that, irrespective of the extent of the noise exposure at work, only those persons annoyed by noise during working hours show an increase in post-work irritability from noise sources at home (Mel92).

Whether noise exposure during the daytime affects sleep quality the night after the exposure was only tested in laboratory research ((Fru88a,b, Fru90). The results were contradictory. One investigation showed noise exposure during the daytime to stimulate recovery processes of neural- and endocrine functions during sleep and another investigation showed no such effect.

Interaction of Noise with Other Agents

Effects on Hearing

Noise may interact with drugs and industrial agents to produce additive or even synergistic effects on hearing. The ototoxic properties of certain drugs, such as aminoglycoside antibiotics (the mycine drugs) are heightened by exposure to noise. Although high doses of salicylates (aspirin) accompanied by noise exposure can produce temporary hearing loss, increased permanent hearing loss does not seem to occur.

Several case reports have been published on acute and chronic effects of carbon monoxide on hearing. The hearing loss resulting from carbon monoxide exposure appears to be reversible in most cases and is associated with toxic effects in the central nervous system. In one epidemiological study, noise-induced hearing loss in welders and plant assembly workers appeared to be influenced by exposure to carbon monoxide.

Epidemiological studies on workers suggest that carbon disulfide, carbon tetrachloride, trichlorethylene and n-butanol induce sensorineural hearing loss. However, the number of studies and the size of the populations studied seem too small to allow a decision about a possible interaction between noise and solvents on hearing.

Heavy metals have also been mentioned as possible industrial ototoxic agents, but very few studies have tested this suggestion.

Noise and vibrations may have a combined effect on hearing. Several epidemiological studies showed that groups of workers exposed to noise and hand-arm vibrations had a noise-induced hearing loss that was more frequent and greater than that in groups of workers exposed only to noise or only to hand-arm-vibrations. The effects were more pronounced in workers suffering from vibration-induced white finger syndrome. All epidemiological studies concerned exposures to very high noise levels and very intense hand-arm-vibrations. For whole-body vibrations (rather than hand-arm) a smaller effect on hearing levels was observed in groups of workers exposed to a combination of noise and vibrations than in groups of workers exposed to noise only.

Other Health Effects

Epidemiological research into the effect of combined exposure to noise and other environmental agents on health other than on hearing is scarce. Investigations are usually carried out in the laboratory with test persons or with animals. Forestry workers using vibrating and noisy tools, with several years of daily exposure to noise, vibration and cold, showed bradycardia. In laboratory experiments it could be shown that other stressors, such as heat and whole-body vibration do exert, when combined with noise, a greater effect on pulse rate, blood pressure and catecholamines than does noise exposure alone. Notwithstanding the data from laboratory research, the committee does not deem it possible to draw any quantitative conclusions applicable to real-life

Summary of Noise-induced Effects

Table 1 summarizes the present data on the effects on health of exposure to noise. The observation thresholds are given in the measures used in the pertinent literature. The use of such a measure in the table does not necessarily imply it to be recommended for use in practical situations or regulations.

With respect to the use of noise exposure measures for the estimation of noise-induced health effects, table 1 shows that for all of these effects, with the exception of some aspects of sleep disturbance, observation thresholds are expressed in the equivalent sound level determined over a selected representative period during the 24 hour day. Usually, the existing exposure-effect relations characterize noise exposure by an equivalent sound level over a representative period. However, dependent upon the noise effect under consideration, these representative periods are different. Therefore, the committee concludes that there is, as yet, no single noise

Table 1.

(Possible) long-term effects of exposure to noise, classification of the evidence for a causal relationship, and data on the observation threshold.

Effect	Classification[a] of evidence	Situation[b]	Observation threshold		
			Measure	Value in dB(A)	In/out[c]
hearing loss	sufficient	occ	$L_{EX,occ}$	75	in
		env recr	$L_{Aeq,24h}$	70	in
		occ unb	$L_{EX,occ}$	<85	in
hypertension	sufficient	occ ind	$L_{EX,occ}$	<85	in
		env road	$L_{Aeq,06-22h}$	70	out
		env air	$L_{Aeq,06-22h}$	70	out
ischaemic heart disease	sufficient	env road	$L_{Aeq,06-22h}$	70	out
		env air	$L_{Aeq,06-22h}$	70	out
biochemical effects	limited	occ			
		env			
immune effects	limited	occ			
		env			
birthweight	limited	occ			
		env air			
congenital effects	lack	occ			
		env			
psychiatric disorders	limited	env air			
annoyance	sufficient	occ off	$L_{EX,occ}$	<55	in
		occ ind	$L_{EX,occ}$	<85	in
		env[d]	L_{dn}	42	out
absentee rate	limited	occ ind			
		occ off			
psycho-social well-being	limited	env			
sleep disturbance, changes in:					
sleep pattern	sufficient	sleep			
awakening	sufficient	sleep	SEL	60	in
sleep stages	sufficient	sleep	SEL	35	in
subjective sleep quality	sufficient	sleep	$L_{Aeq,night}$	40	out
heart rate	sufficient	sleep	SEL	40	in
hormones	limited	sleep			
immune system	inadequate	sleep			
mood next day	sufficient	sleep	$L_{Aeq,night}$	<60	out
performance next day	limited	sleep			
performance	limited	occ env			
	sufficient	school	$L_{Aeq,school}$	70	out

Notes to Table 1.

a classification of evidence of causal relationship between noise and health.

b occ = occupational situation, ind = industrial, off = office, env = living environment, recr = recreational environment, road = road traffic, air = air traffic, sleep = sleeping time, unb = unborn: exposure of pregnant mother, school = exposure of children at school.

c value relates to indoor or outdoor measurement. in the netherlands, the difference between the level measured outdoors and that indoors is 15 to 25 db(a) for dwellings with single glazing.

d observation thresholds for traffic and industrial noise; the observation threshold is lower for environmental impulse noise.

measure, such as $L_{Aeq,24h}$, from which all noise-induced health effects can be estimated, without a specification of the type of noise source, the situation and the period of the day, during which the exposure occurs. This seems especially appropriate for the estimation of sleep disturbance in real live situations, since a reliable relation between measures of night-time exposure and measures related to the 24-hour period does not exist.

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Bab 88

Traffic Noise and Cardiovascular Risk. The Caerphilly Study, First Phase. Outdoor Noise Levels and Risk Factors

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ABSTRACT. As part of the Caerphilly study, traffic noise exposure and risk for ischemic heart disease (IHD) were studied in a sample of 2,512 men aged 45–59 yr. The traffic noise emission level ranged between L_{eq} = 51–70 dB(A) (6–22 hr, 10 min). No association was found between traffic noise and prevalence of IHD. Associations were found between noise and potential IHD risk factors including systolic blood pressure, oestradiol, total cholesterol, plasma viscosity, antithrombin III (increases), cortisol, and platelet count (decreases). Not all results supported the hypothesis that traffic noise increases the risk for IHD. The possibility of confounding cannot be excluded, although an extensive range of potential confounding variables were included in the analysis.

EPIDEMIOLOGIC EVIDENCE suggests that noise exposure may be associated with extra-aural effects such as blood pressure.[1,2] Most evidence that exists supporting an effect on blood pressure comes from occupational noise studies. As to environmental noise, i.e., road traffic noise, aircraft noise, few epidemiological investigations have been made.[3] Research has concentrated on effects on blood pressure, and the results are contradictory. With regard to IHD risk, all known and potentially new risk factors should be considered, including blood pressure, blood lipids, thrombosis factors, hormones, immunological factors, and electrolytes.

Noise (unwanted sound) acts as an environmental or occupational stressor on the human organism. Different types of noise interfere with certain activities of the individual. Even low levels of traffic noise may disturb recreation [e.g., 40 dB(A)] at home, whereas higher noise levels at work or when riding in a car [e.g., 70 dB(A)] may not interfere with other activities. Individuals show different levels of acceptance toward different types of noise and intensity. Extra-aural noise effects are moderated by psychological effects. For this reason, the averaged noise intensity as a one-figure criteria for the whole day noise exposure is an inappropriate measure with regard to the psychological reaction. However, in multifactorial designs the independent effects of different noise sources and their interaction effects can be studied. In principle, noise research can also focus on one specific noise source if methodological assumptions are made, such as a random selection design.

In this paper, cross-sectional data on traffic noise and IHD risk factors are presented from the Caerphilly study. The Caerphilly study is a prospective epidemiological study, in which prevalence and incidence of IHD is related to variables thought to be determinants of cardiac risk. The present results concern outdoor noise levels and data collected during the initial medical examinations of subjects (first phase), and will be used for comparison with other epidemiologic noise research. In further analyses, occupational noise exposure as estimated by questionnaire (follow-up phase) will be considered for all subjects. In a subsample,

combined effects of traffic and work noise (personal noise dosemeters) will be studied. Also, with the aid of a questionnaire, information on indoor traffic noise exposure at home will be estimated. Full details of the Caerphilly study and the present state of noise analysis are given elsewhere.[3-5]

Method

Caerphilly is a town with approximately 45,000 inhabitants. All men who were at entry to the study between the ages of 45–59 yr inclusive and who were residents in the town and a few surrounding villages were invited to attend a heart disease screening clinic. Of 2,818 men identified as being eligible, 2,512 (89%) were examined.

Subjects completed demographic, medical, and personality questionnaires. For IHD categorization, the London School of Hygiene chest pain questionnaire[6] was administered and a 12-lead electrocardiogram (ECG) recorded, which was Minnesota coded by two experienced coders. The prevalence of ischemic heart disease (IHD) was assessed using the questionnaire (angina pectoris, myocardial infarction) and the ECG recordings (ECG ischemia) following the classifications (probable, possible, none) used in the Whitehall study.[7] Blood pressure was measured after several minutes rest in the sitting position. A random zero sphygmomanometer (R. Z.) was applied, but due to failure of the apparatus a regular mercury sphygmomanometer (Reg.) was used also. A fasting blood sample was taken later. In blood serum, enzyme activities of aspartate aminotransferase and alkaline phosphatase, total protein and the fraction albumin, total bilirubin and uric acid concentration were determined; in whole blood the glucose level was measured. In plasmal oestradiol, testosterone and cortisol, total triglycerides, total cholesterol and the subfractions LDL, VLDL, and HDL were determined. Among the hemostatic and hematological factors were viscosity, fibrinogen concentration and clotting time measurements clotting fibrinogen, thrombin time, heparin thrombin clotting time, factor VII and antithrombin III, all of which were determined in plasma; and hemoglobin concentration, platelet and white cell count were determined in whole blood. The concentration of magnesium in erythrocytes was determined also.

To establish a noise map for the area under study, continuous measurements (hourly readings) of A-weighted average sound pressure level (L_{eq}) were carried out on all streets with local bus service on 3 consecutive weekdays. These long-term measurements included all busy roads (continuous traffic flow during daytime) and many side streets (single-event traffic) of the area under study. Similar measurements were taken at some dead end streets (off main roads) to obtain information about the lowest traffic noise conditions exploratively. From these measures, L_{eq} levels for different periods of the day (day: 6–18 hr; evening: 18–22 hr; night: 22–6 hr) were obtained. In addition, short-term measurements of L_{eq} (30 min) were carried out during representative periods of day (10–18 hr) at all other relevant streets. The addresses of all study participants were inspected. Average distances of the houses from the road were documented. From these values traffic noise emission (L_{eq} 6–22 hr, 10 m distance from the center of the road = LEQD10) and immission (at the facades = LEQD0) levels were calculated for every subject's home. These noise levels are the basic figures to describe outdoor traffic noise in the study.

Statistical analysis of results was by analysis of variance (*F* test), in which traffic noise was considered as a four-level factor. Univariate analyses were supplemented by multivariate analyses of variance, in which the potential confounders of age, social class, marital status, employment status, shift work, family history of myocardial infarction, type A behavior, body mass index, physical activity, smoking habit, and alcohol consumption were treated as covariates when necessary. Analyses of variance (*F* test) or contingency (chi square test) were carried out to detect associations between confounders and noise. Analyses of covariance were carried out to detect confounders that had statistical influence on risk factors. Discontinuous variables were dichotomized for this purpose. Relevant covariates (regression coefficient significant) were kept in each analysis to reduce variance. Individual contrasts were checked by *t* test. To avoid possible influence of disease or its treatment, subsamples were formed by excluding subjects with relevant positive medical histories on the basis of anamnestic inquiries. With respect to IHD prevalence and medical history, chi square tests of contingency were carried out. All statistical test interpretations are based on the 5% criteria ($p \leq .05$, two-tailed).

Results

Long-term measurements at 24 different sites revealed a mean difference of 1 dB(A) of L_{eq} between the time periods "day" and "evening," almost independent of traffic volume [range: 1–3 dB(A), correlation: $r = 0.96$]. Similar comparison for the periods "day" and "night" showed a mean difference of 8 dB(A), also almost independent of traffic noise volume [range: 6–10 dB(A), correlation: $r = 0.94$]. In low noise areas the difference tended to be slightly smaller. L_{eq} was relatively stable during 10–18 hr, allowing an estimation of $L_{eq\,6\text{–}22hr}$ by any hourly L_{eq} measure within this period with an accuracy of 3 dB (2 dB in noisy areas). On this basis, traffic noise emission (6–22 hr) was also estimated from short-term measurements. The emission level in the whole sample varied between 51–70 dB(A), and classification of the streets was done in 5-dB categories with regard to LEQD10 and LEQD0. Homes with very low traffic volume (< 5 vehicles/10 min) were grouped into the lowest category. The distribution of subjects over the four noise categories is given in Table 1. Due to random composition of the sample, only one-fifth of the subjects were living in streets with LEQD10 > 60 dB(A), which is in agreement with large community surveys.[8]

Of the potential confounders, only social class and employment status showed a statistical dependency on

Table 1.—Number of Men in Traffic Noise Categories of a Total Sample of 2,512 Men Aged 45–59 yr in Caerphilly

	Noise category [dB(A)]			
	51–55	56–60	61–65	66–70
Traffic noise emission (LEQD10)	1850	211	318	133
Traffic noise immission (LEQD0)	1845	237	264	166

traffic noise. The relative frequency of unemployed men was high in the low noise areas [LEQD10 ≤ 60 dB(A)], and the rate of men with higher social status was lower in the 56–60 dB(A) category than in the others.

The relationships between traffic noise emission and prevalent IHD and its different manifestations as well as medical history of heart attack, stroke, and high blood pressure are given in Table 2. No statistically significant effects were detected. Prevalent IHD ranged from 17.5% at the lowest noise category through 16.6% and 17% with increasing noise to 18.8%.

Table 3 gives mean blood pressure and heart rate readings for men in each traffic noise emission category. Results are given for each sphygmomanometer separately. The data refer to the total sample. Highest mean systolic pressures of 146 mmHg (R. Z.) and 144 mmHg (Reg.) were recorded in men exposed to the most noise. Lowest systolic pressures of 137 mmHg (R. Z.) and 138 mmHg (Reg.) resp. were found in men in the middle noise exposure categories. For men exposed to the least traffic noise, systolic pressures of 140 and 142 mmHg resp. were found. The *F* test of variance showed significance for traffic noise emission and immission ($p \leq .01$ and .05 resp.). However, contrast analyses revealed that this was due to the decrease in the moderately exposed groups. The *t* test between the extreme groups of traffic noise exposure became significant ($p \leq .05$) in a subsample where subjects with positive history of heart attack, stroke, high blood pressure, or thyroid troubles became excluded, which yields to lower readings in the lowest noise category of about 2 mmHg. The corresponding data are given elsewhere.[9] Diastolic pressures of 84–85 mmHg and 89–92 mmHg were recorded between noise categories in the total sample; no association with noise exposure was detected. Heart rate, ranging from 71–72 beats/min, showed no relationship with traffic noise. Accounting for covariates did not influence the results substantially.

Table 4 gives mean hormone levels for each noise emission category. Highest oestradiol levels of 264 pmol/L were found in men exposed the most to traffic noise, and lowest levels of 242 pmol/L were found in men in the 61–65 dB(A) noise category. For men exposed to the least traffic noise, the mean value for oestradiol was 249 pmol/L. The *F* test of variance showed statistical significance for traffic noise emission and immission ($p \leq .05$ and .01 resp.), which was due to the decrease in the 61–65 dB(A) category and the increase in the 65–70 dB(A) category (*t* test: $p \leq .05$). Testosterone levels varied little between the extreme noise exposure levels, indicating a fall from 22.6 to 21.1 nmol/L, but were highest, with a mean value of 25.0 nmol/L, in men exposed to 56–60 dB(A). The *F* test of variance was significant ($p \leq .001$) for traffic noise emission and immission, but the *t* test for extreme group differences only showed significance for traffic noise emission ($p \leq .05$). Cortisol levels only showed a significant association with traffic noise with respect to the immission level (*F* test: $p \leq .05$) due to a decrease between the extreme groups of noise exposure from 432–409 nmol/L in the highest noise category (*t* test: $p \leq .01$). The highest values were found in men in the 61–65 dB(A) category (446 nmol/L). For traffic noise emission, no such characteristic was found. Through all categories the values varied between 432 and 435 nmol/L. Covariates had only little influence on cell means and did not affect the results of statistical tests substantially.

Table 5 gives mean values for serum lipids according to noise emission level. Highest total cholesterol values of 5.93 mmol/l were found in the highest noise exposure group, while in the other categories the values ranged from 5.66–5.70 mmol/l. The *F* test of variance showed no significance; however, the *t* test contrast between the extreme groups of noise exposure was significant ($p \leq .05$) for traffic noise emission only, independent of covariates. HDL cholesterol showed a sim-

Table 2.—Traffic Noise Emission and Medical History and Prevalent IHD

	LEQD10 [dB(A)] Relative frequency (%)				Total frequency (%) (N = 2,512)
	51–55	56–60	61–65	66–70	
Heart attack	5.8	6.7	4.7	5.3	5.7
Stroke	1.2	1.4	1.3	3.0	1.4
High blood pressure	19.1	21.3	17.3	12.8	18.7
Angina pectoris*	7.9	6.6	8.2	4.5	7.6
Myocardial infarction*	10.3	10.9	8.2	10.5	10.1
ECG-ischemia	3.9	1.9	4.1	6.0	3.9
IHD (a combination of the above)	17.5	16.6	17.0	18.8	17.4

*These criteria are not mutually exclusive.

Table 3.—Traffic Noise Emission and Blood Pressure and Heart Rate

	LEQD10 [dB(A)] [mean (standard deviation)]			
	51–55	56–60	61–65	66–70
Systolic blood pressure (mmHg)				
Random zero device*	140.4(19.8)	137.3(19.6)	136.9(23.4)	146.3(22.3)
Regular device*	141.9(19.3)	138.0(15.5)	139.3(15.9)	143.5(21.5)
Diastolic blood pressure (mmHg)				
Random zero device*	84.9(12.6)	84.3(12.5)	84.4(16.1)	84.5(12.3)
Regular device*	91.8(12.1)	89.4 (9.2)	90.9(10.5)	91.3(12.2)
Heart rate (beats/min)	71.5(12.5)	70.7(11.5)	71.7(11.9)	71.4(12.4)

*Random zero device, N = 42%; regular device, N = 58%.

Table 4.—Traffic Noise Emission and Hormone Levels

	LEQD10 [dB(A)] [mean (standard deviation)]			
	51–55	56–60	61–65	66–70
Oestradiol (pmol/L)	249 (61)	247 (64)	242 (59)	264 (69)
Testosterone (nmol/L)	22.6 (7.3)	25.0 (8.5)	22.2 (7.8)	21.1 (6.8)
Cortisol (nmol/L)	432 (141)	433 (124)	435 (142)	432 (140)

Table 5.—Traffic Noise Emission and Lipid Levels

	LEQD10 [dB(A)] [mean (standard deviation)]			
	51–55	56–60	61–65	66–70
Total cholesterol (mmol/L)	5.70(1.13)	5.66(1.18)	5.68(1.13)	5.93(1.27)
HDL cholesterol (mmol/L)	1.11(0.33)	1.07(0.30)	1.13(0.33)	1.16(0.36)
LDL cholesterol (mmol/L)	3.77(1.04)	3.78(1.10)	3.74(1.08)	3.88(1.21)
VLDL cholesterol (mmol/L)	0.81(0.58)	0.80(0.63)	0.81(0.53)	0.89(0.57)
Total triglycerides (mmol/L)	2.03(1.33)	1.91(1.53)	1.96(1.14)	1.99(1.22)

ilar trend, with highest values of 1.16 mmol/l in the highest noise exposure group. Lowest values of 1.07 mmol were found in men in the 56–60 dB(A) category. For men exposed the least to traffic noise, the mean value was 1.11 mmol/l. The F test of variance was significant only for traffic noise immission when covariates were considered ($p \leq .05$), which was due to an increase between the extreme groups of noise exposure (t test: $p \leq .01$). Here the maximum level was 1.18 mmol/l. VLDL cholesterol and LDL cholesterol showed no association with traffic noise. The values ranged from 3.74–3.88 for VLDL and 0.81–0.89 for LDL. Total triglyceride, ranging from 1.91–2.03 mmol/l, showed no significant relationship with traffic noise level.

Table 6 provides mean values for thrombosis-related factors according to noise emission level. White cell count, fibrin clotting time, thrombin time, and heparin-thrombin clotting time were not associated with noise emission. Plasma viscosity showed no significant association in variance analysis, but there was a homogeneous increase in men with increasing traffic noise emission and immission level from 1.71–1.73 cp. Although a marginal difference, this trend was statistically significant when regression analyses were performed (r = +0.05, $p \leq .05$). Fibrinogen values ranged from 3.77–3.95 g/l. Association to traffic noise level was curvilinear with highest values in men exposed to most traffic noise and lowest values in the 61–65 dB(A) group. The F test of variance was significant only for traffic noise immission ($p \leq .05$) and when covariates were considered, which influenced the results. The t test of extreme group differences was significant ($p \leq .05$). Platelet count tended to decrease with greater noise exposure for emission and immission level (F test: $p \leq .01$)—from 282–259 × 10^9/L—although the highest values of 294 × 10^9/L were found in the 56–60 dB(A) category. The t test for extreme group differences was significant also ($p \leq .05$). These effects were independent of covariates used in the analyses. Antithrombin III was highest at 119% of standard in the 61–65 dB(A) category with respect to traffic noise emission and at 120% of standard in the 66–70 dB(A) category with re-

spect to traffic noise immission. Lowest values (112 and 113% resp. of standard) were found in the 56–60 dB(A) noise category. In men exposed to the least traffic noise, the values were 115% of standard. The *F* test of variance was significant for traffic noise emission and immission ($p \leq .01$), independent of covariates. The *t* test of extreme group differences was significant only for traffic noise immission.

Table 7 gives mean values for other biochemical factors that have been investigated according to noise emission level. No associations were found between traffic noise and aspartate aminotransferase, alkaline phosphatase, total protein, glucose, uric acid, hemoglobin, and ethythrocyte magnesium. Albumin values were lowest in men exposed most to noise (43.5 g/L) and varied in the other categories between 44.3 and 44.6 g/L. The *F* test of variance was significant ($p \leq .001$) for traffic noise emission and immission, independent of covariates. The same was true with extreme group differences (*t* test: $p \leq .001$). Bilirubin showed highest values at 10.8 mmol/L in men of the highest category of noise emission, but lowest values in the 55–60 dB(A) category (9.1 mmol/L), whereas those men least exposed to traffic noise had a mean value of 9.8 mmol/L. The values were slightly different for noise immission level: *F* test of variance and *t* test of extreme group differences ($p \leq .05$) were significant only for traffic noise emission, independent of covariates.

Table 8 summarizes the results concerned with risk factors. For emission and immission level of traffic noise on the basis (*t* test) of extreme group differences (category 51–55 dB(A) vs. 66–70 dB(A)). Tables 2–8 refer to cell frequencies given in Table 1. Because of the missing tests, the number of cases varied slightly between the measurements and assays.

Discussion

Traffic noise indices (L_{eq}) for different time periods of day ("day," "evening," and "night") normally show very high correlation,[10–12] as shown in this study. Consequently, they were equally good predictors for day and night disturbances.[13,14] In the Caerphilly study, $L_{eq.6-22hr}$ was a fairly good descriptor for noise load of streets to allow distinction between high- and low-noise areas with respect to other time periods of the day. Traffic noise level generally tends to be relatively stable during daytime (11–18 hr) independent of traffic noise volume,[10] which was also found in Caerphilly. This also enabled assessment of $L_{eq.6-22hr}$ on the basis of short-term measurements. $L_{eq.6-22hr}$ is a common measure for characterization of traffic noise conditions, and was an appropriate measure in this study, where a large number of addresses, distributed randomly over the town, had to be considered.

In this study of a large and representative sample of men, no association between traffic noise and preva-

Table 6.—Traffic Noise Emission and Thrombosis Factors

	LEQD10 [dB(A)] [mean (standard deviation)]			
	51–55	56–60	61–65	66–70
Platelet count (10⁹/L)	282 (75)	294 (77)	270 (68)	259 (75)
White cell count (10⁹/L)	7.11 (2.04)	7.20 (2.26)	6.70 (1.91)	6.94 (1.87)
Plasma viscosity (cp)	1.71 (0.09)	1.71 (0.09)	1.72 (0.10)	1.73 (0.12)
Fibrinogen (g/L)	3.77 (0.82)	3.86 (0.99)	3.79 (0.80)	3.95 (0.99)
Fibrin clotting time (sec)	13.1 (2.4)	13.1 (2.5)	12.9 (2.6)	13.3 (2.6)
Thrombin time (sec)	17.6 (1.6)	17.8 (1.5)	17.8 (3.1)	18.0 (1.7)
Heparin thrombin clotting time (sec)	31.1 (11.8)	31.4 (12.8)	29.5 (12.3)	30.6 (9.4)
Antithrombin III (% of standard)	115 (21)	112 (22)	119 (19)	117 (17)

Table 7.—Traffic Noise Emission and Other Biochemical Variables

	LEQD10 [dB(A)] [mean (standard deviation)]			
	51–55	56–60	61–65	66–70
Aspartate aminotransferase (IU/L)	25 (11)	24 (10)	27 (13)	26 (12)
Alkaline phosphatase (IU/L)	88 (34)	93 (32)	87 (30)	85 (30)
Total protein (g/L)	72.3 (4.2)	71.9 (3.9)	72.8 (4.3)	73.3 (5.0)
Albumin (g/L)	44.6 (2.9)	44.3 (3.0)	44.6 (2.9)	43.5 (3.0)
Bilirubin (mmol/L)	9.8 (5.0)	9.1 (4.8)	9.7 (4.4)	10.8 (5.1)
Glucose (mmol/L)	5.0 (1.2)	5.2 (2.0)	5.0 (1.1)	5.2 (1.9)
Uric acid (μmol/L)	360 (77)	359 (77)	365 (74)	360 (78)
Hemoglobin (g/L)	154 (11)	154 (12)	154 (12)	155 (11)
Ery-magnesium (mmol/kg)*	5.58 (0.57)	5.55 (0.63)	5.60 (0.62)	5.53 (0.76)

*Number of cases: 1,269 due to incomplete sampling.

Table 8.—Summary of the Results of *t* Test Statistic (two-tailed) Based on Extreme Group Differences of Noise Exposure [51–55 dB(A) vs. 65–70 dB(A)]

Effect variable	Significance		Tendency of association
	Traffic noise emission	Traffic noise immission	
Systolic blood pressure	$p \leq .05$	NS	+
Diastolic blood pressure	NS	NS	
Heart rate	NS	NS	
Cortisol	NS	$p \leq .01$	−
Oestradiol	$p \leq .05$	$p \leq .05$	+
Testosterone	$p \leq .05$	NS	−
Total cholesterol	$p \leq .05$	NS	+
HDL cholesterol	NS	$p \leq .05$	+
LDL cholesterol	NS	NS	
VLDL cholesterol	NS	NS	
Total triglycerides	NS	NS	
Platelet count	$p \leq .01$	$p \leq .01$	−
White cell count	NS	NS	
Plasma viscosity	$p \leq .10$	NS	+
Fibrinogen	NS	$p \leq .05$	+
Fibrin clotting time	NS	NS	
Thrombin time	NS	NS	+
Heparin thrombin clotting time	NS	NS	
Antithrombin III	NS	$p \leq .05$	+
Asparate aminotransferase	NS	NS	
Alkaline phosphatase	NS	NS	
Total protein	NS	NS	
Albumin	$p \leq .001$	$p \leq .001$	−
Bilirubin	$p \leq .05$	NS	+
Glucose	NS	NS	
Uric acid	NS	NS	
Ery-magnesium	NS	NS	
Hemoglobin	NS	NS	

lence of IHD was detected. This may be because there is no association, but there are other possible explanations. Subjects with evidence of heart disease are survivors and seriously underrepresent the full picture of IHD. Secondly, current traffic noise exposure can only give, at best, a crude estimate of past exposure. From a causal point of view, noise must persist over an extended period before a pathogenic outcome can be expected.

In the study of possible causal factors of heart disease, incidence data constitute a far more certain basis for conclusions than cross-sectional analyses of prevalent data. In the prospective approach, measurements are made of dietary, biochemical, or environmental factors, and levels are related to the subsequent development of heart disease. We are currently collecting such evidence in Caerphilly.

The main focus of the present paper, however, is the associations between noise exposure and known and possible risk for IHD. In this, the limitations outlined above still apply, but with much less force. Alterations in risk factors will occur before the actual IHD event. Looking at risk factors instead of IHD cases avoids the problem of low numbers of cases. Even small changes of mean values for risk factors may substantially increase the risk for disease in a population.

We have found no evidence of significant associations between traffic noise exposure and diastolic blood pressure, heart rate, VLDL and LDL cholesterol, triglycerides, fibrin clotting time, thrombin time, heparin-thrombin clotting time, white cell count, uric acid, total protein, glucose, hemoglobin, erythrocyte magnesium, alkaline phosphatase, and aspartate aminotransferase. Associations were found between traffic noise and systolic blood pressure, oestradiol, testosterone, cortisol, total cholesterol, HDL cholesterol, platelet count, fibrinogen, antithrombin III, plasma viscosity, albumin, and bilirubin. Of these variables, lower values of HDL cholesterol, antithrombin III, and testosterone are associated with higher IHD risk.[15–19] Higher values of systolic blood pressure, oestradiol, cortisol, total cholesterol, fibrinogen, plasma viscosity, and platelet count are associated with higher IHD risk.[16,20–26] If comparisons are made of risk factor values between men in extreme noise categories, the rise in systolic blood pressure, oestradiol, total cholesterol, fibrinogen and plasma viscosity (positive correlation with noise), and the fall in testosterone with greater noise exposure is consistent with the hypothesis that noise exposure increases IHD risk. In contrast, however, the fall in cortisol and platelet count and the rise in HDL cholesterol and antithrombin III do not support this hypothesis.

Interpretation of relationships between noise and IHD risk factors is further complicated in some cases where curvilinear associations occurred (albumin, bilirubin, testosterone, platelet count, fibrinogen, antithrombin III). However, other variables not showing a linear trend seem to indicate a threshold for physiolog-

ical noise effects (oestradiol, cortisol, total cholesterol, HDL cholesterol, viscosity, systolic blood pressure). This is what can be expected from community surveys on noise annoyance that show an increase in percentage of highly annoyed inhabitants at $L_{dn} > 60$ dB(A).[27] Because of the suburban character of the area, no higher levels of traffic noise other than $L_{eq} = 70$ dB(A) (6–22 hr) were obtained for this study. All results so far must be treated carefully. Adequate statistical control for the effects of confounding is always difficult. In this study, an extensive range of potential confounding variables were measured including age, social class, marital status, employment status, shift work, family history of myocardial infarction, type A behavior, body mass index, physical activity, smoking, alcohol consumption, and prevalence of diseases. The variables were included in each analysis whenever they covaried with noise or the IHD risk factor. In spite of these procedures, the possibility of confounding by an unknown factor cannot be excluded, and would be an attractive explanation in the absence of a theoretical basis for curvilinear effects of traffic noise.

Many risk factors were considered. Here the problem of mass significance testing arises. Some of the findings could be chance effects. From longitudinal data (new IHD incidence rates in the follow-up phase), a risk estimation for the Caerphilly cohort will be drawn. Only a few independent (endogenous) factors with considerable predictive power will remain. Accounting for the statistical impact of traffic noise (exogenous factor) on the relevant risk factors will allow a quantitative risk estimation (relative risk of disease occurrence) for traffic noise exposed groups.

At present, no final conclusions should be drawn from the Caerphilly survey. Follow-up investigations are in process, and the outcome of longitudinal data may or may not confirm the findings. Noise annoyance data, residence time, and indoor exposure (room orientation) will also be considered. A noise questionnaire has been introduced into the survey. Additional noise measurements will incorporate other noise indices, e.g., maximum noise level. A twin study in another community by the same group of investigators with identical methods applied—The Speedwell Study[4]—is in progress, which will give further evidence.

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SLEEP DISTURBANCE, PSYCHO-SOCIAL AND MEDICAL SYMPTOMS—A PILOT SURVEY AMONG PERSONS EXPOSED TO HIGH LEVELS OF ROAD TRAFFIC NOISE

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A pilot survey was undertaken to elucidate sleep quality, as well as psycho-social and medical symptoms and mood, among people who had lived for many years in an area with high levels of road traffic noise during night hours and inhabitants of a quiet control area: 106 personal interviews were performed and specific questionnaires on sleep and mood answered by 63 persons during three consecutive days. It was found that both sleep quality and mood (social orientation, activity, wellbeing and extroversion) were depressed in the noisy area as compared with a control area. Symptoms of tiredness, headache and nervous stomach disorders were more frequent. A significant relationship between sensitivity to noise and sleep quality was also found. From this pilot study hypotheses may be formulated about a relationship between environmental noise and different psycho-social and medical symptoms. It is suggested that similar studies on a larger scale are performed to elucidate long-term effects of noise.

1. INTRODUCTION

There is considerable literature on the effects of road traffic noise on sleep (for a general review, see reference [1]). Most studies have been concerned with the physiological effects measured by EEG, EMG and ECG during sleep, either in laboratory experiments or in restricted field studies where the noise exposure has been manipulated in different ways. Recent studies [2, 3] have elucidated after-effects measured the day after noise-disturbed sleep. Results from the joint European study [3] based on 70 subjects and 1000 nights showed an increased performance and an increase in subjective sleep quality after a noise reduction of 6–14 dB(A) in the bedrooms. These effects were considered to be indicators of health effects.

Knowledge of long-term health effects such as psycho-social and medical symptoms due to sleep distrurbance induced by road traffic noise (see reference [4]) is sparse. Some studies have suggested that persons in areas marked by noise exposure have a higher frequency of psychiatric disorders, manifested as admission rates to psychiatric hospitals [5]. In view of the difficulties in controlling for such confounding factors as socio-economic differences, these results must be interpreted with care [6]. A higher use of tranquilizers, a more frequent consultation of psychiatrists and a higher rate of admission to a psychiatric clinic were found in an area affected by high levels of road traffic noise [7]. Results from a laboratory sleep study [8] showed that exposure to road traffic noise during nine nights for a period of two weeks caused decreased sleep quality, performance and mood, and increased tiredness. No habituation was seen for these effects. If people who have lived in areas with high levels of road traffic for many years are affected in a similar way, they may develop long-term effects on their psycho-social wellbeing and medical symptoms.

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2. AIM OF STUDY

The aim of the present study was to describe sleep quality, mood and possible long-term effects of disrupted sleep, such as different medical and psycho-social symptoms, in areas with heavy road traffic noise. Annoyance to traffic noise and other disturbances in the environment as well as noise sensitivity and different background variables (for example, age, length of residence, sex and employment status) were to be considered.

3. MATERIALS AND METHODS

3.1. SITES

The Highway and Street Office in Gothenburg, Sweden, intended to reduce the noise levels in a thoroughfare in Gothenburg by prohibiting heavy vehicles during the night and reducing speed limits from 70 to 50 km/h. They turned to us to evaluate the effect of these traffic regulations. A summary of the study before and after traffic regulations has been presented in a paper at the Fifth Congress of Noise as a Public Health Problem in Stockholm (1988). In this paper the study before the traffic regulations is described.

Two sites were chosen for the study, at different distances from the highly trafficked main thoroughfare. Site N (noisy) close to the thoroughfare and site C (control), about 300 m from the thoroughfare, were residential areas with small privately owned two-storey houses or blocks of three-storey flats.

3.2. NOISE DESCRIPTION AND MEASUREMENT

Noise levels were measured outdoors at a representative site in each area. Measurements were made for five weekdays in each area and were averaged. The traffic flow was measured hour for hour using a counting device which could discriminate between long and short vehicles, in order to define heavy vehicles. The exposure was expressed as the total number of vehicles, number of heavy vehicles, outside L_{eq} levels and number of maximum noise events above 75, 80 and 85 dB(A) (outside). The noise exposure for the two sites is shown in table 1.

There was only sparse road traffic at site C on local streets during the daytime. The noise reduction from the façade in the houses was about 25 dB(A), which means that 445 vehicles with a noise level of more then 50 dB(A) max. could be heard inside the houses.

Noise exposure for each single respondent was not measured but, when analyzing the different effect variables, separate analyses were made to control for window insulation

TABLE 1

Noise exposure

	Noisy site N	Control site C
24 hour		
L_{eq}(dB(A))	71·8	56
Numbers of vehicles	24 886	
Number of heavy vehicles	4 603	
Night, 22:00-06:00		
Number of vehicles	1 606	
Number of heavy vehicles	228	
>75 dB(A)	445	
>80 dB(A)	92	
>85 dB(A)	5	

(noise insulation windows *vs.* no insulation) and the location of the bedrooms (toward the street or in the rear of the house).

3.3. INTERVIEWS

At each site, one person above 18 years of age in each household, who had resided there for at least one year, was selected for a personal interview. After the interview, the respondents received a special questionnaire on sleep quality and mood to be answered on three consecutive days. This was done to try to mask the intention of the survey by presenting the investigation as a general study of environment and health.

The interview questionnaire contained 34 questions and was divided into four sections. The first section contained general questions about length of residence, satisfaction with the dwelling and the surroundings. The respondents were also asked about disturbances in the environment, such as noise from road traffic, neighbors, aircraft, industries and exhaust from motor vehicles, dust or soot in the air and odors from industries. Individuals who expressed annoyance from these disturbances were asked about the extent ("not very", "rather" and "very annoyed") and whether they were concerned about damage to the house from the road traffic. A question was also asked about which activities were disturbed by road traffic noise, such as ability to have the windows open, interference with speech, interference with telephone calls, listening to radio/TV, rest and relaxation, and difficulty in falling asleep and awakening.

The second section contained questions about sleep. The questions were graded as follows: on sleep quality, from 1 to 4 ("bad", "rather bad", "not very good" and "very good"); on difficulty in falling asleep, from 1 to 4 ("not at all", "not very", "rather" and "very difficult"); on time for falling asleep, less than 15 minutes, 15-30 minutes, 30-60 minutes and more than 60 minutes. The number of awakenings and difficulty in going back to sleep after having woken up (yes/no) were also assessed. Questions about feelings in the morning were graded from 1 to 4 ("very tried", "rather tired", "rather alert" and "very alert"). Questions were also asked in this section about use of sleeping pills and ear plugs.

The third section contained questions about medical and psycho-social symptoms. Some of these questions were chosen from an early investigation by Edwards [9], who studied symptoms after acute loss of sleep. The respondents answered how often (every day, every week, once a month and more seldom or never) they felt very tired, irritable and cross, depressed, anxious and nervous, felt they wanted to be left alone, or had headaches or a nervous stomach. If any of these symptoms were experienced daily, they were asked to give a reason for it. They were also asked whether they experienced other kinds of symptoms.

The fourth section of the questionnaire contained questions about employment, and about subjective noise sensitivity, graded from 1 to 4 ("not sensitive", "not very sensitive", "rather sensitive" and "very sensitive").

At the end of the interview all respondents were requested to answer a special sleep questionnaire [2] and a mood questionnaire (MACL) [10] on three consecutive days between Monday and Friday. Questions about tiredness during the evening and the day (graded 1-10), stress and worries during the day (graded 1-10) and bedtime were answered in the evening. In the morning, questions about the time needed to fall asleep (in minutes), difficulty in falling asleep (yes/no) and time to wake up were answered, together with questions about the number of awakenings and difficulty in gong back to sleep (yes/no). Body movements, sleep quality, tiredness and irritability in the morning were evaluated on a scale graded from 1 to 10 with endpoint markings. The respondents were also asked to report whether anything in particular had affected their sleep during the night.

The mood questionnaire (MACL), which contains 60 different adjectives and measures degree of wellbeing, extroversion, activity, relaxation and social orientation, was answered immediately after the evening and morning questionnaires on sleep.

3.4. ANALYSIS OF THE RESULTS

The results on sleep, medical and psycho-social symptoms and mood are described in three sections, after each of which is presented an analysis of population characteristics and annoyance from noise in the environment and sleep disturbance from noise. Differences in mean values between sites were tested with Student's t-test (two-sided) and χ^2 test. If the variables were not normally distributed, the Mann–Whitney U-test was used. The relationships between different variables were evaluated using Pearson's correlation test or, when appropriate, Spearman's correlation test. The overall results are discussed in the final section.

4. RESULTS

4.1. DESCRIPTIONS OF THE POPULATION SAMPLES

The total number of interviews at sites N and C together was 106 (69 and 37). The average drop-out was 20% (19 and 21). The number of respondents who answered the special sleep and mood questionnaire for three days was 63 (40 and 23 at sites N and C). Some characteristics of the different population samples are shown in table 2. As can be seen in the table, the time of residence was shorter at site N. The average age and the proportion working did not differ significantly between the sites. There was no difference in the distribution between sexes and the average noise sensitivity between the populations of the two sites.

4.2. EXPERIENCE OF THE ENVIRONMENT

At site N, an average of 46% did not feel satisfied with the environment, as opposed to 5.4% at site C. The main source of disturbance was road traffic noise (an average of 94.5% were annoyed and 67.5% were very annoyed), as opposed to 19% annoyed and 3% very annoyed at the control site.

TABLE 2

Population characteristics

	Noisy site N	Control C	p-values (two-tailed)
Number of interviews	69	37	
Number of questionnaires	40	23	
Time of residence:			
Mean	11·9	18·8	0·04[1]
Variance	1-54	1-52	
Age:			
Mean	44·8	50·8	0·12[1]
Variance	19-84	20-87	
Females (%)	57·9	48·7	—
Males (%)	42·1	51·3	—
Percentage working (%)	54	71	—
Noise sensitivity (mean)	2·2	2·1	—

[1] Mann–Whitney U-test.

Sleep disturbances dominated the disturbance pattern at site N. Those who were annoyed by road traffic noise reported that the noise disturbed their rest and recreation (49%), made it difficult to fall asleep (37%) and woke them (50%): 87% could not keep their windows open because of the noise; while 43% of those who owned their houses and 8% of rentees reported fear of damage to the house from traffic vibrations.

4.3. SLEEP QUALITY

The mean values for sleep quality as measured by the interview questionnaire are reported in table 3. It can be seen that sleep quality was worse in the noisy area, with greater difficulty in falling asleep, a tendency to greater difficulty in going back to sleep, poorer sleep quality and greater tiredness in the morning. Only a small number of the respondents used sleeping pills or ear plugs (five at site N, as opposed to one at site C).

TABLE 3

Mean values for sleep variables (interviews)

	Noisy site N	Control C	p-values (two-tailed)
N	69	37	
Difficulty in falling asleep (mean)	2.1	1.3	0·001 [2]
>30 minutes to fall asleep (%)	34	19	0·36 [1]
Awakenings	1·2	0·8	0·15 [3]
Difficulty in going back to sleep (%)	36	10	0·08 [1]
Sleep quality (mean)	2·9	3·5	0·0008 [2]
Tiredness (mean)	2·2	2·7	0·01 [3]

[1] χ^2-test.
[2] Student's t-test.
[3] Mann–Whitney U-test.

A similar but more detailed and precise response pattern was derived from the specific sleep questionnaire. As there was a smaller number of respondents to this questionnaire, a t-test was performed to ensure that these respondents did not differ from the total sample. The test showed no difference in sleep quality. The results of the specific sleep questionnaire are shown in Table 4. The table shows that it took an average of 19 minutes longer to fall asleep at site N. However, due to the variation in time to fall asleep, no significant differences were found. The number of awakenings was significantly higher at site N and a higher percentage of awakenings was caused by traffic noise at this site. Concerning body movements and sleep quality, a significant difference was found between site N and site C. At site N, the respondents were more tired and more irritatable in the mornings, and also more tired during the day.

Sleep quality was not correlated with length of residence, age, sex, employment status type of window or location of the bedroom. The lack of a relationship between sleep quality and the type of window and the location of the bedroom might be due to selection: that is, those who are more concerned or sensitive to noise may have changed their windows and the location of the bedroom.

Subjective sensitivity to noise was correlated to several sleep measures. The more sensitive the subject the longer it took to fall asleep ($r = 0{\cdot}21$, $p > 0{\cdot}05$), the more awakenings were reported ($r = 0{\cdot}27$, $p > 0{\cdot}01$) and the more tired he/she was in the morning ($r = 0{\cdot}25$, $p > 0{\cdot}01$). The correlation between sleep quality and noise sensitivity increased when only area N was included in the analysis (correlation coefficients between

TABLE 4

Mean values for sleep variables (specific sleep questionnaire)

	Noisy site N	Control C	*p*-values (two-tailed)
N	40	23	
Difficulty in falling asleep (%)	37	8	0·05 [a]
Time to fall asleep (in minutes)	44	25	0·10 [b]
Awakenings	2·2	1·2	0·04 [b]
Awakenings from traffic noise (%)	57	4	
Body movements (1–10)	5·0	3·6	0·02 [b]
Sleep quality (1–10)	6·2	8·2	0·002 [b]
Morning			
Tired-alert (1–10)	5·0	7·0	0·004 [b]
Irritable-friendly (1–10)	6·5	7·8	0·02 [b]
Day			
Tired-alert (1–10)	6·5	7·8	0·01 [b]

[a] χ^2-test.
[b] Student's *t*-test.
[c] Mann-Whitney *U*-test.

0·28 and 0·43, as opposed to between 0·21 and 0·27 when both sites were included). This means that individuals who are sensitive to noise and live in a highly noise-exposed area will have poorer sleep quality than those who are not sensitive to noise.

4.3.1. *Sleep quality and experience of disturbances in the environment*

Sleep quality was also related to annoyance from road traffic noise in the environment ($r = 0·43$) and to feelings of satisfaction with the neighborhood ($r = 0·39$). All correlations were significant at the 0·1% level. Other sleep measures (difficulty in falling asleep, awakening and tiredness in the morning) were also correlated to these environmental factors, but these correlations were lower.

Concerning the relation between specific activity disturbances from road traffic noise and sleep quality, those who reported disturbance in rest and relaxation tended to be more tired in the morning ($p = 0·06$). Those who reported difficulties in falling asleep because of road traffic noise had greater difficulties in going back to sleep ($p = 0·003$), a tendency to decreased sleep quality ($p = 0·07$) and a tendency to increased tiredness in the morning ($p = 0·08$). Finally, those who reported that they were awakened by road traffic noise had poorer sleep quality ($p = 0·002$) and needed longer to fall asleep ($p = 0·04$).

Again, there was a close relationship between noise sensitivity and disturbance of rest and relaxation from road traffic noise ($p = 0·009$) and awakenings ($p = 0·001$).

4.4. MEDICAL AND PSYCHO-SOCIAL SYMPTOMS

The extent of reported symptoms at the different sites is shown in Figure 1: the left *p*-value refers to symptoms every day or week and the right *p*-value to symptoms every day, week and month. A significantly higher proportion of individuals reported that they were "very tired" in the noisy site. At site N, there were also increased reports of "headaches" and "nervous stomach" and a tendency to increased feelings of wanting to be left alone.

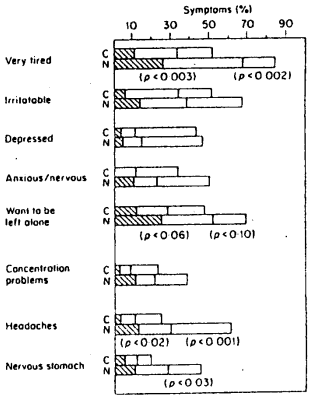

Figure 1. Medical and psycho-social symptoms. ▩, Every day; ▨, several times a week; □, several times a month.

The respondents at site C who had reported different psycho-social symptoms on a daily basis gave the reasons for these to be pregnancy, work, personality, poor sleep quality and high age, respectively. At site N, the majority of the respondents mentioned sleep deprivation, road traffic noise and work as reasons for the symptoms they experienced every day.

Some of the population characteristics were related to the different symptoms Older respondents were more often anxious or nervous ($r = 0·20$, $p = 0·05$). Those who were worried about damage to their homes from traffic vibration more often also reported anxiousness and nervousness ($p = 0·04$). The correlation between age and anxiousness or nervousness decreased to $r = 0·08$ when only those who rented apartments were taken into account. Thus the increased reports of anxiousness or nervousness were probably due to worries about damage to homes. Time of residence was found to be correlated with "wanting to be left alone" ($r = 0·23$, $p = 0·05$) and headaches ($r = 0·25$, $p = 0·01$) The longer the time of residence, the lower the frequency of these symptoms. (This could be due to selectivity: i.e., those who experienced such symptoms to a high extent may have moved away from the area.) Employment and sex were not correlated with any symptoms. Subjective noise sensitivity was correlated with "depressed" ($r = 0·22$, $p = 0·05$), "anxious and nervous" ($r = 0·31$, $p = 0·01$) and "concentration problems" ($r = 0·24$, $p = 0·04$). The more sensitive to noise the individual was, the more frequent were these symptoms.

4.4.1. *Medical symptoms and experience of disturbances in the environment*

It was found that the degree of annoyance from road traffic noise was significantly related to the frequency of "headaches" ($r = 0·32$, $p < 0·01$), "nervous stomach" ($r = 0·27$, $p < 0·01$), feeling "very tired" ($r = 0·25$, $p < 0·01$) and "anxious and nervous" ($r = 0·21$, $p < 0·05$).

The occurrence of medical symptoms was also analyzed in relation to activity disturbances from road traffic noise. Only those who reported noise disturbance of rest and sleep had reported different symptoms. It was found that those who were disturbed during rest and relaxation were more often very tired (24% *vs* 3%, $p = 0{\cdot}04$). Those who had difficulties in going to sleep because of noise more often reported "concentration problems" (36% *vs.* 8%, $p = 0{\cdot}02$). Those who reported that they were awakened by road traffic noise were more often "anxious and nervous" (43% *vs.* 5%, $p = 0{\cdot}005$), "depressed" (27% *vs.* 2%, $p = 0{\cdot}01$) and had "concentration problems" (31% *vs.* 7%, $p = 0{\cdot}04$).

4.5. MOOD

The results of the mood questionnaire are shown in Table 5. The respondents at site N had lower mood values in the mornings on four parameters (wellbeing, activity, social orientation and extroversion). A tendency towards a lesser degree of relaxation was also seen. Mood as measured in the evenings did not differ significantly between the sites. The only correlation found between the different population characteristics and mood was for age. Older respondents were more socially oriented.

TABLE 5

Mean values from the mood questionnaire

	Noisy site N	Control C	p-values (two-tailed)
Number	40	23	
Morning			
Wellbeing	2·74	3·10	0·02 †
Activity	2·58	3·05	0·004 †
Social orientation	2·90	3·21	0·04 †
Relaxation	2·88	3·16	0·08 †
Extroversion	2·37	2·64	0·05 †

† Student's t-test.

There was generally a close relationship between the different symptoms and mood, all correlations being between 0·33 ($p < 0{\cdot}01$) and 0·71 ($p < 0{\cdot}001$). The results from repeated questionnaires over several days (the mood questionnaire) are thus in good agreement with the respondent's judgement of the different psycho-social and medical symptoms over a longer time period (the personal interview).

4.6. SLEEP QUALITY, MEDICAL AND PSYCHO-SOCIAL SYMPTOMS AND MOOD

The correlation between sleep quality and medical and psycho-social symptoms is shown in Table 6. It can be seen that sleep quality was related to all symptoms except "wanting to be left alone", and tiredness in the morning to all symptoms except "anxious and nervous". Other sleep parameters were related to only some of the symptoms. Difficulty in falling asleep was related to "depressed" ($r = 0{\cdot}24$), "anxious and nervous" ($r = 0{\cdot}26$) and "headaches" ($r = 0{\cdot}29$).

Sleep quality was highly correlated to the different mood parameters ($r = 0{\cdot}55$ to $r = 0{\cdot}64$, $p < 0{\cdot}001$). A strong relationship was also seen between tiredness in the morning and during the day and the mood parameters ($r = 0{\cdot}52$ to $r = 0{\cdot}82$, $p < 0{\cdot}001$).

TABLE 6

Correlations (r_s) between medical and psycho-social symptoms and sleep quality ($n = 106$)

	Sleep quality	Tiredness in the morning
Very tired	0·30	0·43
Irritatable/cross	0·32	0·22
Depressed	0·38	0·22
Anxious/nervous	0·31	0·19
Want to be left alone	0·15	0·33
Headaches	0·34	0·32
Nervous stomach	0·30	0·32
Concentration problems	0·24	0·23

$r = 0{\cdot}21$ ($p < 0{\cdot}05$); $r = 0{\cdot}25$ ($p < 0{\cdot}01$); $r = 0{\cdot}32$ ($p < 0{\cdot}001$).

5. DISCUSSION

A summary and schematic description of the relation between noise, noise sensitivity and the variables investigated is given in Figure 2(a), and the variables with significant interrelationships (significance level > 0·05) are shown in Figure 2(b). The results indicate that people living for many years in an area marked by a great deal of noise report that they still experience disturbances during rest, have difficulties in falling asleep and are frequently awakened by road traffic noise. This lack of habituation to noise is in agreement with results from other studies [4, 7, 11].

When sleep was measured by using the repeatedly given sleep questionnaire, the results showed that the number of awakenings was doubled and that the individuals experienced their sleep quality as worse than at the control site. During the morning as well as the day, the average tiredness was more severe. The difference in sleep quality between noisy and control sites was 24%, which is about the same as in laboratory experiments, where sleep quality decreased by 17% when subjects were exposed to road traffic noise for nine nights during a two-week period [8]. Increased tiredness in the morning and day were more pronounced than in previous laboratory studies [2, 8]. In these laboratory studies students with a mean age of 24 years (18–30 in 1982) with respect to 24·5 (22–34 in 1988) were registered. Since the younger persons in this study were more tired in the morning than the older ones, the difference in age between the field and the laboratory studies could not be due to differences in age. One possible reason for the more pronounced effects in this field study is the long time of noise exposure.

Other studies [12–14] have shown that older people are more likely to be disturbed by noise during sleep than are younger people. This conclusion was based on EEG findings and behavioural measures. This was not our finding in this study, which indicates that older people do not necessarily subjectively experience their sleep as worse than do younger people, even if they have less stage 4 sleep than younger people. In fact, Saletu [15] questioned the connection between the sleep stage nomenclature and 'sleep depth". He found a positive relationship between stage 4 and subjectively experienced "light sleep", while a great amount of stage 2 was indicative of a shortened sleep latency, a restful and uninterrupted sleep process and a refreshed feeling in the morning. As regards sex and noise-disturbed sleep, no firm evidence has previously been found of differences between men and women [16], nor could such differences in sleep disturbances be found in this study.

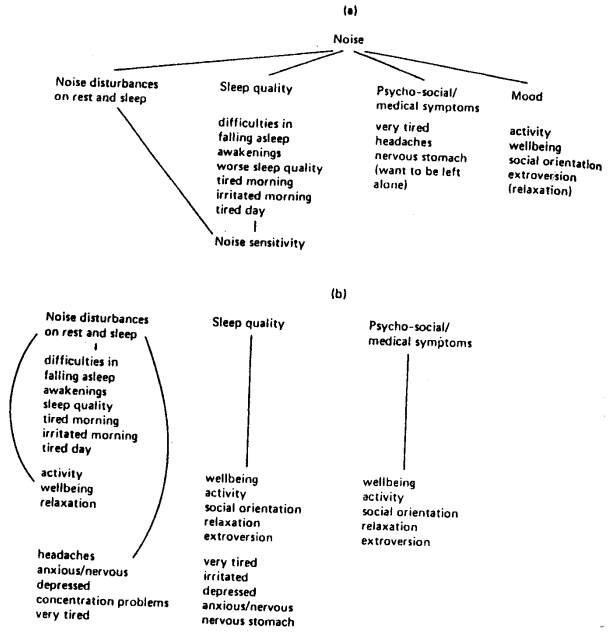

Figure 2. A schematic description of the relation between noise and the variables investigated. (a) Relation between noise, noise sensitivity and effect variables; (b) relation between variables.

Psycho-social and medical symptoms such as tiredness, headaches and nervous stomach were more frequent in the noisy area. Also, a tendency towards a desire to be alone was seen in the noisy area, which is in accordance with decreased social orientation as measured with the mood questionnaire. As these symptoms could not be linked to differences in background factors, they are probably caused by noise exposure. This interpretation is supported by the strong relation between noise disturbances of rest/relaxation, difficulties in falling asleep and awakenings and the symptoms of more frequent headaches, nervous stomach and being very tired. An increased tiredness has also been found after exposure to road traffic noise in a previous laboratory experiment [8].

The symptoms were closely interrelated to the mood variables and are probably measuring the same effect. The symptoms are derived from direct questions and mood through a more sophisticated scale. Psycho-social symptoms have not previously been studied in relation to noise-disturbed sleep. Effects on mood after exposure to noise during the night have previously been studied in laboratory experiments by Ehrenstein *et al.* [17] and Öhrström [2, 8]. The effects of mood were found to be more pronounced in the present study, probably due to the fact that noise is always present in the home environment, while the temporary exposure in a laboratory has a less pronounced effect on the subjects' mood because they know that the noise exposure will soon cease.

A significant relationship was found between subjective noise sensitivity and reported disturbances from noise during sleep and certain sleep quality parameters. This finding is in accordance with results from earlier field studies [4] and with the findings of laboratory experiments [8] whose noise-sensitive and non-noise-sensitive subjects were exposed to road traffic noise.

Psycho-social symptoms were also associated with noise sensitivity, but not with mood. Individuals who were noise-sensitive more often reported anxiety and nervousness, feelings of depression and concentration problems. A relationship between noise sensitivity and neuroticism (EPI) was also found in a laboratory study [18]. Similar results have been presented by Poenaru *et al.* [19]. They found that individuals who complained about noise had more personal disorders: nervousness, anguish, anxiety and phobia. A test of attenuation also showed that individuals who complained about noise were significantly less rapid in the execution of tasks and that they made more mistakes.

Because of the small sample, no definite conclusions can be drawn from the present study. However, it is hypothesized that exposure to high levels of road traffic noise during the night, which causes acute sleep disturbances and affects subjective sleep quality, also has more long-term effectss on psycho-social wellbeing and mood. Noise-sensitive individuals (rather + very sensitive to noise), in this case 30% of the sample, appear to suffer more from these effects. Further studies on a larger scale will, however, be necessary to confirm these findings. The study does not make it possible to draw any conclusions about dose-response relationships. Studies in areas with different noise exposure levels are therefore needed.

ACKNOWLEDGMENT

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NATURAL FREQUENCY INFORMATION FOR CIRCULAR AND ANNULAR PLATES

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This paper presents the results of an extensive literature search and review of available sources of numerical natural frequency information for stationary circular and annular elastic plates. In addition to the source, information is given regarding the specific plate theory, boundary conditions, geometric properties and material properties used to determine the natural frequency information. Sources of information are tabulated according to the above parameters for easy reference and selection of desired information. A description of the various plate theories, including the basic governing partial differential equation of motion, is also given. This information may be particularly useful to experimentalists and designers when natural frequency data are required without the need for detailed analysis.

1. INTRODUCTION

This paper presents the results of an extensive literature search and review of available sources of numerical natural frequency information for stationary, homogeneous, isotropic circular and annular elastic plates of uniform thickness. The references cited all have some numerical information regarding the natural frequencies, dimensionless frequency parameters, or frequency ratios referenced to some frequency of interest for circular and annular plates subject to various boundary conditions.

In addition to the source, information is given regarding the specific plate theory, boundary conditions, geometric properties and material properties used to determine the natural frequency information. A wide range of these parameters is covered by the sources, and most common cases should be found in the tables often associated with more than one source.

A description of the various plate theories is also given. This includes the basic governing partial differential equation of motion and a brief discussion of the salient features and assumptions of each theory.

2. DISCUSSION

The information in the tables is arranged in ascending order of date of publication. For completeness, even early investigations are included. Table 1 lists all sources of numerical natural frequency information for stationary, homogeneous, isotropic circular plates of uniform thickness. Table 2 lists all sources of numerical natural frequency information for stationary, homogeneous, isotropic annular plates of uniform thickness. Extensive surveys of additional topics in plate mechanics may be found in the papers by Leissa [1–7].

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TRAFFIC NOISE, WORK NOISE AND CARDIOVASCULAR RISK FACTORS : The Caerphilly and Speedwell Collaborative Heart Disease Studies

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As part of two large heart surveys, associations between traffic noise exposure and cardiovascular risk factors were studied. The Caerphilly sample (small town, total sample) consisted of 2512 men aged 45 to 59 years and the Speedwell sample (suburb of a major city, random sample) of 2030 men of same age group. Both studies have a prospective design; cross-sectional results are presented here. Acoustic measurements were carried out in both areas. Among the possible risk factors for ischaemic heart disease studied were blood pressure, blood coagulation, blood lipids and other biochemical factors. Statistically significant noise effects were detected for systolic blood pressure, total cholesterol, HDL cholesterol, total triglycerides, blood viscosity, platelet count and glucose level, although not all of these were consistent with noise being a risk factor for heart disease. In a subsample, the additional influence of work noise as determined by noise dosimetry was studied in 255 men, taking the use of ear protection into account. The associations between traffic noise and risk factors were more pronounced in men who also were exposed to high work noise levels.

INTRODUCTION

The Caerphilly study and the Speedwell study are two closely linked epidemiological heart surveys carried out in the United Kingdom (The Caerphilly And Speedwell Collaborative Group 1984). The objectives are to identify already known and possible new risk factors with independent predictive power for ischaemic heart disease (IHD). A prospective study design has been used, in which a cohort of subjects is recruited and examined, and then followed forward in time. New cases of disease which develop are identified and can be related to the initial measurements. With respect to noise, changes in physiological factors with time will be observed and related to the traffic noise exposure which was determined by short- and long-term measurements in the streets.

Also of interest in this paper are the combined effects of traffic noise and occupational noise on other risk factors. This was studied in a subsample of the Caerphilly cohort. Work noise was determined by use of personal noise dosimeters at work in a quasi-random sample. Therefore a wide variety of work places were assessed.

METHODS

Caerphilly is a small town in South Wales, while Speedwell refers to a district of the major English city of Bristol. Both representative samples are based on populations of 45 to 59 year old men. From Caerphilly a 100% sample of men was selected using the electoral roll and general practitioner records as the basic sample frame. Speedwell men were drawn from a 100% sample of the age-sex registers of the 16 general practitioners working out of two health centers in the area. In both samples, a response rate of about 90% was obtained. Altogether about 4500 men (2512 and 2030, respectively) were seen (Babisch and Gallacher 1990).

The "Dosimeter"-subsample was drawn from a random sample of 500 Caerphilly men to which 84 men who lived on noisy streets were added. The latter were added to ensure that there were adequate subjects with high exposure to noise in the sample. Due to certain social factors (unemployment, disablement, retirement), the eligible sample was reduced to 380 subjects. Due to refusals of men and employers (no dosimeters were allowed for miners) and missing values, a final sample of 255 men was obtained.

The clinical examinations included ECG recordings, blood pressure readings and fasting blood samples as well as medical and socio-demographic questionnaires (The Caerphilly Collaborative Group 1985). Both studies have a common core protocol. Survey methods have been made as similar as possible.

Long-term and short-term noise measurements of the A-weighted sound pressure level were carried out in the streets, to establish traffic noise maps of the areas (Babisch and Ising 1986). The subjects' homes were randomly distributed due to the sampling procedure. In accordance with the noise measurements, the subjects were grouped into 5 dB categories of the traffic noise emission level, in terms of L_{eq} referring to the period from 6 a.m. to 10 p.m, and a distance of 10 m from the streets. Daytime outdoor noise level was used as a general descriptor of traffic noise load in the streets. Work noise measurements were carried out by dosimetry. On two to three consecutive days, the men were fitted with personal noise dosimeters (impulse characteristic) for the entire shift to determine the A-weighted average sound pressure level L_{eq}. To avoid overload, the instruments were set to the 80-130 dB(AI) range.

RESULTS

Traffic noise in the total samples

At present, cross-sectional data analyses referring to the initial phases are available. Associations between outdoor traffic noise level and IHD risk factors are given here, as have been presented at the 5th international congress on "Noise as a Public Health Problem" (Babisch and Gallacher 1990). Further results are given elsewhere (Babisch et al. 1988). The statistical analyses followed a two-factorial variance design. Noise and area were treated as independent factors. Table 1 shows the distribution of men over the noise categories. A set of variables considered as potentially confounding were generally treated as covariates in the analyses. These included age, social class, relative body weight, smoking, alcohol consumption, physical activity and family history. The following results refer to adjusted group means. Group differences were standardized for the standard deviations of the variables in the entire samples. Figure 1-6 show the results for total plasma cholesterol, total plasma triglycerides, whole blood glucose, plasma viscosity, platelet count, and systolic blood pressure.

Table 1. Distribution of men in traffic noise categories (Caerphilly and Speedwell total samples).

Noise Category : $L_{eq,6\text{-}22h,10m}$ [dB(A)]	51-55	56-60	61-65	66-70	Total
Caerphilly sample	1850	211	318	133	2512
Speedwell sample	1410	234	183	203	2030

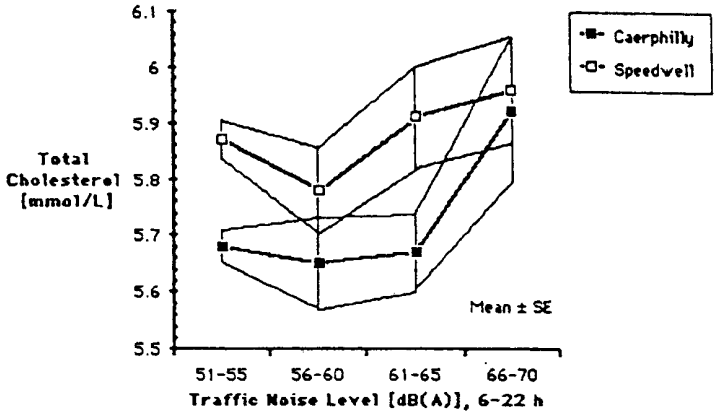

Fig. 1. Group means of risk factors related to traffic noise level.
—Plasma cholesterol—

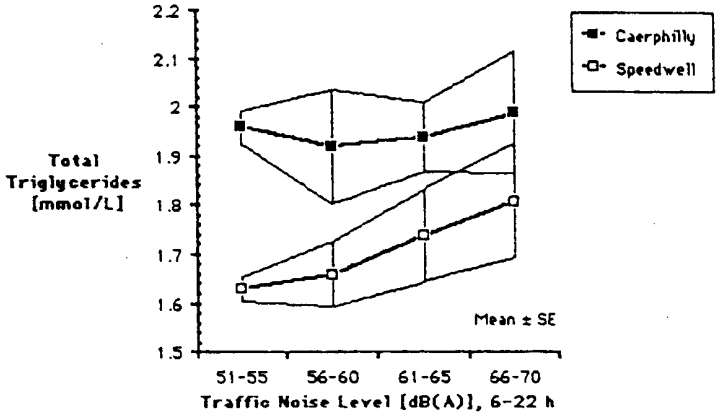

Fig. 2. Group means of risk factors related to traffic noise level.
—Plasma triglycerides—

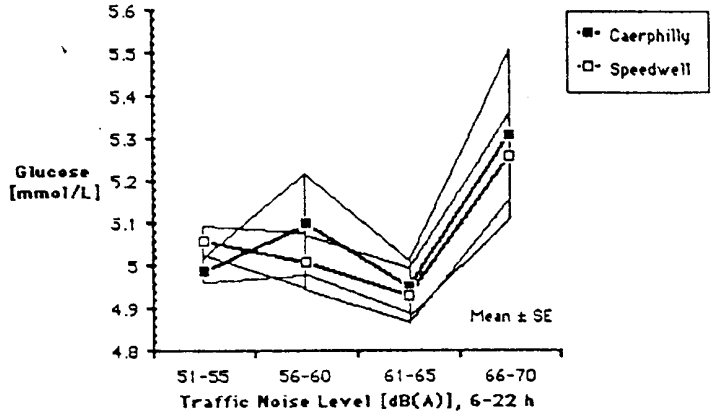

Fig. 3. Group means of risk factors related to traffic noise level.
—Whole blood glucose—

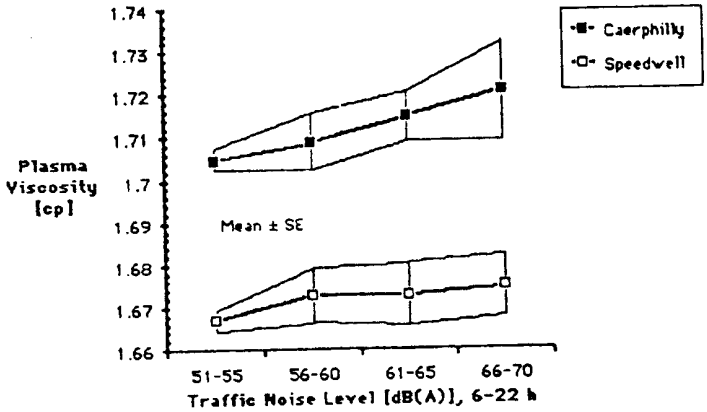

Fig. 4. Group means of risk factors related to traffic noise level.
—Plasma vicosity—

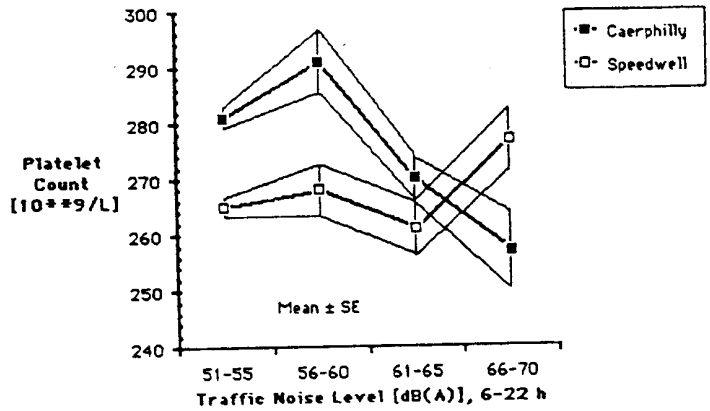

Fig. 5. Group means of risk factors related to traffic noise level.
—Platelet count—

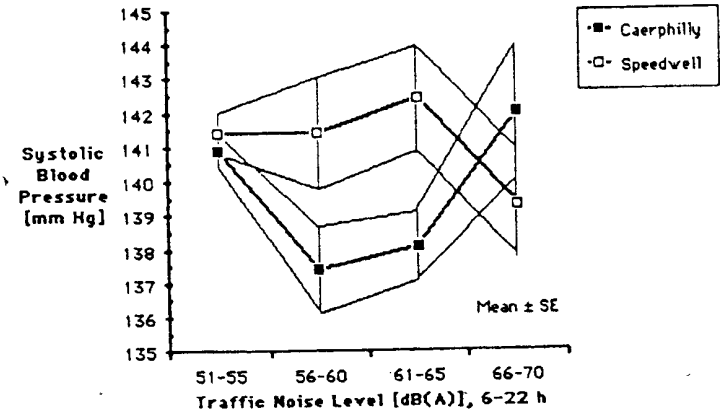

Fig. 6. Group means of risk factors related to traffic noise level.
—Systolic blood pressure—

The most consistent noise effects in both samples were found for blood lipids. Men in the noisiest category had the highest mean total cholesterol levels. From the graphs it can be seen that the effect showed evidence of a threshold at about 60 to 65 dB(A). As compared to the control group, the increase was about 21% (0.24 mmol/L) and 7% (0.09 mmol/L) of the standard deviations, and reached statistical significance in the Caerphilly sample. LDL cholesterol and HDL cholesterol showed similar associations.

Total plasma triglycerides were independent of the traffic noise level in the Caerphilly sample. However, there was a marked significant increase with noise in the Speedwell sample. In terms of the standard deviation, men in the noisiest category had an 18% (0.18 mmol/L) higher group mean than the controls.

Whole blood glucose also showed evidence of a threshold at about 65 dB(A) for higher levels in the noisiest category. This was consistent in both samples. The extreme group differences were about 24% (0.32 mmol/L) and 16% (0.20 mmol/L) and reached statistical significance. However, in Caerphilly men, higher levels were also observed in a moderately noise-exposed group.

A marginal, but in the pooled sample statistically significant, increase of plasma viscosity of 16% (0.016 cp) and 9% (0.008 cp) with noise level was found, higher plasma viscosity levels indicating a higher thrombotic risk.

The findings for platelet count were contradictory. In Caerphilly a decrease of 32% (24×10^9/L) and in Speedwell an increase of 18% (12×10^9/L) were found, both being statistically significant.

Also the findings for blood pressure were contradictory. Since no effect at all was detected for diastolic blood pressure, a slight tendency towards higher systolic blood pressure readings of 6% (1.1 mm Hg) in the highest noise category was found in Caerphilly men, while lower readings were found in Speedwell men. Furthermore these findings were difficult to interpret because of U-shaped curve characteristics (F-Test: $p < 0.01$). The increase in systolic blood pressure in the Caerphilly cohort was more pronounced (21% of SD, 4 mm Hg), if men with a history of hypertension and heart troubles (possible treatment) became excluded (Babisch 1985). Similar data for Speedwell men (hypertension) were missing.

Table 2 sums up the results. For the two samples, the standardized group differences of risk factors [% SD] between men in the noisiest and the quietest traffic noise category are given for each sample. The

Table 2. Mean differences in IHD-risk factors between extreme groups of traffic noise exposure (Caerphilly and Speedwell total samples).

Extreme group difference L_{eq}, 6-22 h, 10 m: (51-55 vs. 65-70 dB(A))	Caerphilly Δ [% SD]		Speedwell Δ [% SD]		Pooled sample (2-factorial)
Plasma cholesterol	+ 21	$p < 0.05$	+ 7	n. s.	$p < 0.05$
Plasma HDL-cholesterol	+ 15	n. s.	+ 11	$p < 0.05$	$p < 0.05$
Plasma LDL-cholesterol	+ 6	n. s.	+ 11	n. s.	n. s.
Plasma VLDL-cholesterol	+ 19	n. s.	- 3	n. s.	n. s.
Plasma triglycerides	+ 2	n. s.	+ 18	$p < 0.01$	n. s.
Plasma viscosity	+ 16	$p < 0.10$	+ 9	n. s.	$p < 0.05$
Plasma fibrinogen	+ 11	n. s.	- 3	n. s.	n. s.
Heparin thrombin clotting time	- 3	n. s.	- 2	n. s.	n. s.
Whole blood glucose	+ 24	$p < 0.05$	+ 16	$p < 0.05$	$p < 0.01$
Whole blood platelet count	- 32	$p < 0.01$	+ 12	$p < 0.05$	n. s.
Whole blood leucocyte count	- 10	n. s.	- 1	n. s.	n. s.
Systolic blood pressure	+ 6	n. s.	- 9	n. s.	n. s.
Diastolic blood pressure	- 6	n. s.	- 10	n. s.	n. s.

Significance based on T-test and F-test statistics for extreme group differences
Δ = Difference ("high" minus "low" traffic noise group) SD = Standard deviation

sign indicates increases or decreases with noise. Test statistics (T-Test, F-Test) are given for these extreme group differences. The table includes results for further risk factors measured. Results of inferential statistics are also given for the pooled sample, using area as a factor.

Diastolic blood pressure, VLDL cholesterol, plasma fibrinogen, white cell count and clotting tests failed to show an association with traffic noise. Platelet count and systolic blood pressure showed contradictory relations with noise. Higher levels of these variables are associated with a higher cardiovascular risk (O'Brien et al. 1975; Daniel et al. 1982; Baker et al. 1982; Tuomilehto et al. 1984). Total cholesterol, LDL and HDL cholesterol, total triglycerides, blood glucose and plasma viscosity were raised in traffic noise-exposed men, indicating a higher cardiovascular risk (Carlson and Böttiger 1981; Shaper et al. 1985; Yarnell et al. 1985; The Caerphilly and Speedwell Collaborative Group 1988), except for HDL cholesterol which showed an inverse relationship with IHD in some heart studies (Gordon et al. 1977).

Traffic noise and work noise in the subsample

A question that arises in work noise studies is how to treat ear protection in the analyses. The effect on sound reduction can hardly be estimated (type of ear protector, noise spectrum, inappropriate use). Surprisingly, many studies on non-auditory effects of noise seemed to have ignored this fact. In the present study the use of ear protection was questioned on a five-point scale. From a statistical point of view, ear protection acts as a confounder. However, if ear protection is available, its actual use by the subjects might be comparable to closing the windows in homes located on noisy streets. Individuals with greater acceptance of the noise will use ear protection less often, if they are not forced to. This reflects the aspect of coping. One might suspect that as long as the subjects can control the stimulus (effectiveness of supplied ear protection), they show lower stress reactions. In non-auditory noise research, the role of ear protection will be different as compared to hearing loss research, where the physical noise energy is the primary factor. In this study, men who acknowledged wearing ear protection, in general, were grouped into the "low" work noise category regardless of frequency of its use. Two-thirds of the men with "high" work noise answered that they never used ear protection. This might partly be due to the wide variety of work places being sampled including many which, although not in an industrial setting and so not being recognized as being noisy, nevertheless, produced high noise levels.

Table 3. Case numbers in traffic and work noise categories (Caerphilly subsample).

Traffic noise	Work noise	Number
low	low	114
low	high	53
high	low	57
high	high	31

The statistical analyses followed a two-factorial design. Traffic noise and work noise were treated as independent factors besides the interaction term. In order to achieve reasonable case numbers in each cell, each factor was reduced to two levels (Babisch and Ising 1988). The grouping criteria were set to >90 vs. ≤90 dB(AI) for occupational noise ("high/low"), which is about to reflect the hearing protection criteria, and >60 vs. ≤60 dB(A) for traffic noise ("high/low"). Table 3 shows the case numbers in each factor cell. The results refer to adjusted group means (analyses of covariance). Group differences were standardized for the standard deviation of each risk factor variable in the subsample.

Figures 7-12 show the results for total plasma cholesterol, total plasma triglycerides, whole blood glucose, plasma viscosity, platelet count, and systolic blood pressure. All "group means" refer to adjusted values considering the possible confounders mentioned above. For comparison the graphs also show the results for the subsample if work noise is not considered as parameter. There were marked differences in the effects of traffic noise on risk factors between men who additionally were exposed to much work noise and those who were not.

For example, for men in the two work noise categories ("high" versus "low") the differences between the high and low traffic noise category were +74% (0.92 mmol/L) versus +2% (0.02 mmol/L) of the standard deviation for total cholesterol ($p < 0.01$), +57% (0.67 mmol/L) versus -3 % (-0.03 mmol/L) for LDL cholesterol ($p < 0.10$), +46% (0.043 cp) versus +24% (0.022 cp) for plasma viscosity ($p < 0.05$), +58% ($35 \leq 10^9$/L) versus +30% (18×10^9/L) for platelet count ($p < 0.01$) and +22% (4.1 mm Hg) versus -20% (-3 mm Hg) for systolic blood pressure (n. s.).

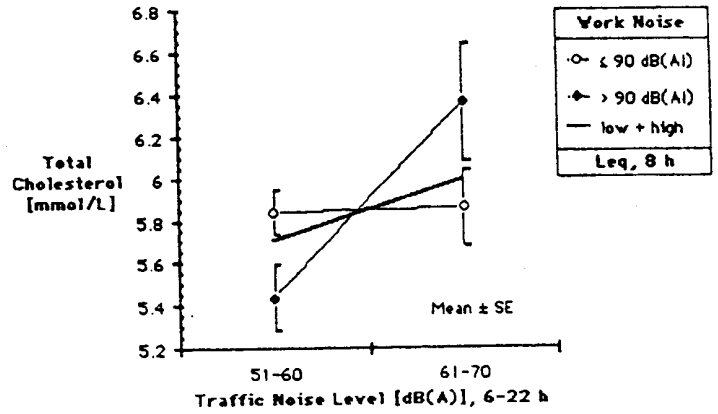

Fig. 7. Group means of risk factors related to traffic noise level and work noise level.
—Plasma cholesterol—

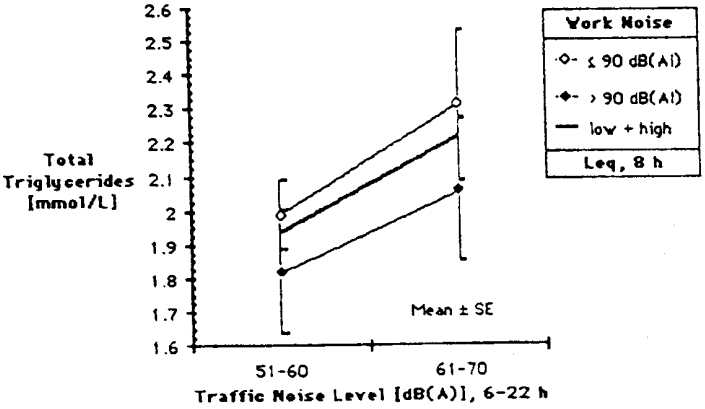

Fig. 8. Group means of risk factors related to traffic noise level and work noise level.
—Plasma triglycerides—

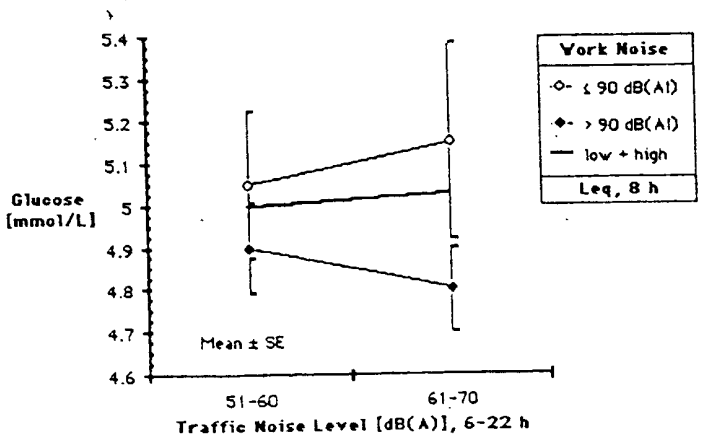

Fig. 9. Group means of risk factors related to traffic noise level and work noise level.
—Whole blood glucose—

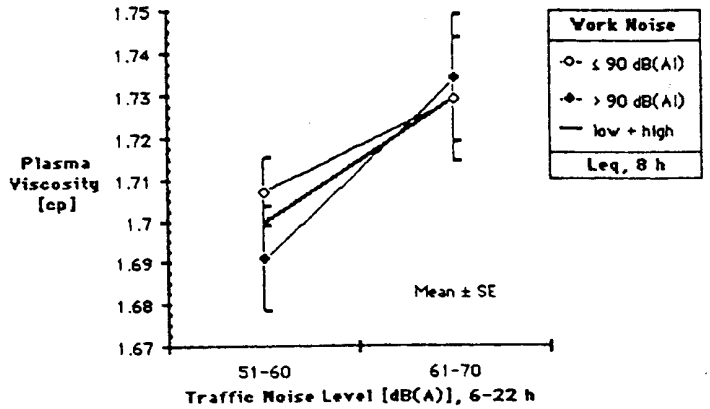

Fig. 10. Group means of risk factors related to traffic noise level and work noise level.
—Plasma viscosity—

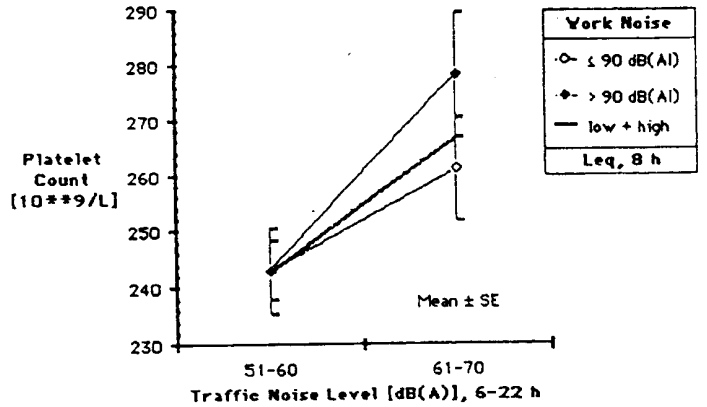

Fig. 11. Group means of risk factors related to traffic noise level and work noise level.
—Platelet count—

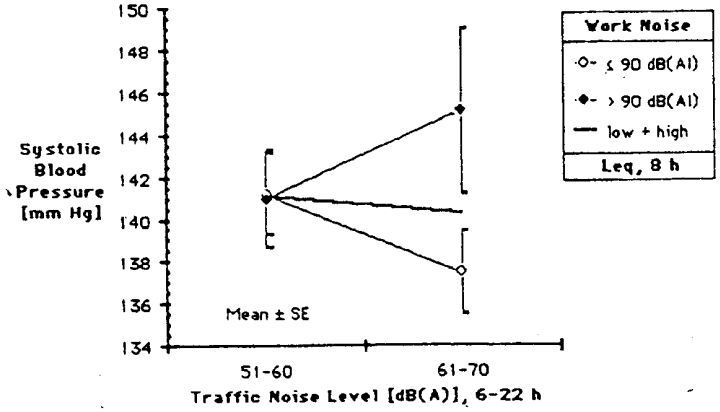

Fig. 12. Group means of risk factors related to traffic noise level and work noise level.
—Systolic blood pressure—

Table 4. Mean differences in IHD-risk factors between groups of high and low traffic noise exposure as a function of work noise exposure (Caerphilly subsample).

Extreme group difference [%]	Traffic noise (L_{eq}, 6-22h, 10m)		Work noise (L_{eq}, 8h)	Interaction
Work noise:	low Δ [% SD]	high Δ [% SD]	Significance level	
Plasma cholesterol	+ 2	+ 74	p < 0.01	n. s.	p < 0.01
Plasma HDL-cholesterol	- 5	- 3	n. s.	n. s.	n. s.
Plasma LDL-cholesterol	- 3	+ 57	p < 0.10	n. s.	p < 0.05
Plasma VLDL-cholesterol	+ 13	+ 55	p < 0.05	n. s.	n. s.
Plasma triglycerides	+ 27	+ 19	n. s.	n. s	n. s.
Plasma viscosity	+ 24	+ 46	p < 0.05	n. s.	n. s.
Plasma fibrinogen	+ 15	+ 29		n. s.	n. s.
Heparin thrombin clotting time	- 33	- 14	p < 0.10	n. s.	n. s.
Whole blood glucose	+ 7	- 7	n. s.	n. s.	n. s.
Whole blood platelet count	+ 30	+ 58	p < 0.01	n. s.	n. s.
Whole blood leucocyte count	- 5	+ 18	n. s.	p < 0.10	n. s.
Systolic blood pressure	- 20	+ 22	n. s.	n. s.	n. s.
Diastolic blood pressure	- 5	+ 3	n. s.	n. s.	n. s.

Significance based on F-test statistics
Δ = Difference ("high" minus "low" traffic noise group) SD = Standard deviation

Table 4 summarizes the results. Standardized differences between the two traffic noise groups are given for each work noise category. Statistically significant interactions between noise and area were observed only for blood lipids.

If the traffic noise factor was split into 3 levels (≤=60, 61-65, ≤=66 dB(A)), the effect of work noise appeared to be greater. Since cell frequencies became very small in the higher noise categories (N = 15), the results for total cholesterol and systolic blood pressure only, are presented for illustrative purposes (Figures 13-14). The differences between the extreme traffic noise categories then increased to +89% (1.11 mmol/L) versus +5% (0.28 mmol/L) for total cholesterol and +44% (8.4 mm Hg) versus -1% (-1.2 mm Hg) for systolic blood pressure in dependency on the work noise level.

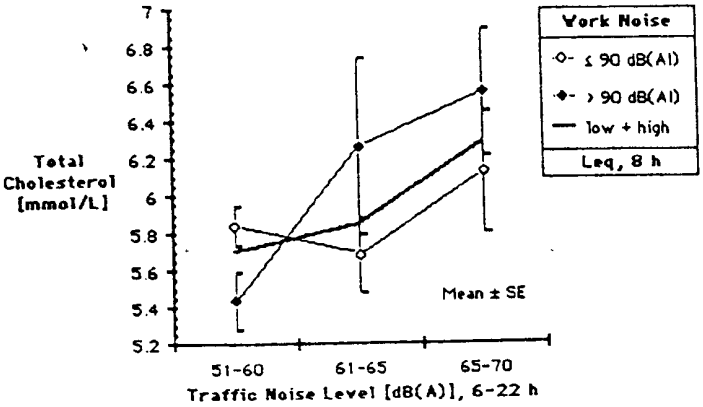

Fig. 13. Group means of plasma cholesterol and systolic blood pressure related to traffic noise level and work noise level.
—Plasma cholesterol—

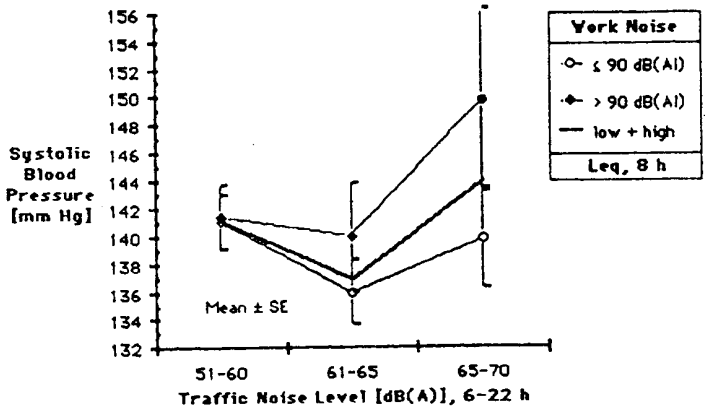

Fig. 14. Group means of plasma cholesterol and systolic blood pressure related to traffic noise level and work noise level. —Systolic blood pressure—

CONCLUSIONS

The effects of traffic noise on risk factors for ischaemic heart disease were studied in two samples of 45 to 59 year old men cross-sectionally. Blood lipids, plasma viscosity and glucose levels turned out to be associated with traffic noise level. Men in the highest noise category consistently showed the highest readings. The results for blood pressure were contradictory between the two samples. At the present stage, the findings must be interpreted cautiously. It cannot be concluded that the relationships reported here were necessarily free of confounding influences although a number of standard confounding factors were considered. The reported effects were marginal and often of borderline significance. Follow-up investigations will provide further evidence about the relationship between traffic noise load and cardiovascular risk in both cohorts under study. Information about room orientation, residence time, noise annoyance and occupational noise exposure will then be taken into consideration, which might explain inconsistent findings.

It was shown in a subsample of the Caerphilly cohort that taking into account work noise exposure reduces unexplained variance, to reveal larger effects of traffic noise. Effects on blood pressure and blood lipids, in particular, turned out to be more pronounced in subjects who also were exposed to high levels of work noise. Since the relatively crude two-level criteria of work noise was successful, it provides encouragement to use questionnaire data to distinguish between high and low noise exposed subjects with respect to their occupational environment. This is being done in the follow-up phases of the Caerphilly and Speedwell heart disease studies for the entire cohorts.

In conclusion, this study suggests that even small differences among group means might be relevant for public health because of the large number of people affected by traffic noise in our communities. More epidemiological noise research is needed in the environmental field and relative risk estimations are required. The Caerphilly and Speedwell collaborative heart disease studies will provide such data on the basis of incidence data and longitudinal changes of risk factors.

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Traffic Noise and Cardiovascular Risk: The Caerphilly and Speedwell Studies, Second Phase. Risk Estimation, Prevalence, and Incidence of Ischemic Heart Disease

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ABSTRACT. As part of the Caerphilly and the Speedwell collaborative heart disease studies, associations between outdoor traffic noise level, risk factors for ischemic heart disease, and prevalence and incidence of ischemic heart disease were studied in two samples of 2 512 and 2 348 men, respectively, who were 45–63 y of age. Compared with the lowest noise category [$L_{eq,6-22\,h}$ = 51–55 dB(A)], the subjects in the highest noise category [$L_{eq,6-22\,h}$ = 66–70 dB(A)] showed a slightly worse risk factor profile with respect to 9 identified endogenous risk factors. Logistic regression analysis suggested a marginal increase in risk (relative risk = 1.1) for ischemic heart disease incidence for these men based on risk factors. The prevalence of ischemic heart disease was slightly higher (relative risk = 1.2) in this noise group. The observed incidence of major ischemic heart disease within an observation period of approximately 4 y was slightly lower (relative risk = 0.8) for men in the highest noise group.

EXPERIMENTAL STUDIES have shown that acute noise exposure results in various physiological and biochemical reactions.[1,2] Noise acts as an nonspecific stressor on the human organism.[3] Individual differences determine whether the sympathetic-adrenal-medullary system or the pituitary-adrenal-cortical system will be more affected.[4] However, such temporary laboratory-based effects do not reveal much about health risk resulting from prolonged noise exposure, such as persistent traffic noise at home or work noise. Because many of the acute effects are also postulated as cardiovascular risk factors, a hypothesis has been suggested that chronic noise exposure raises the risk of heart disease; in particular, that traffic noise causes heart disease.

This hypothesis is being tested cross-sectionally and prospectively in two cohorts in the Caerphilly and Speedwell collaborative heart disease studies. In these separate but closely linked surveys, the relationships among road traffic noise level and cardiovascular risk factors, prevalence, and incidence of ischemic heart

disease (IHD) have been investigated. In the cross-sectional phases of the Caerphilly study[5] and the Speedwell study,[6] a number of known and possibly new endogenous (biological) risk factors (e.g., blood pressure) and exogenous risk factors (determinants, e.g., noise) for IHD were examined. The idea behind this model is that an exogenous factor affects health via endogenous factors within the organism (exogenous risk factor → endogenous risk factors → disease).

Methods

Caerphilly men were chosen from the electoral roll and general practitioner records, and Speedwell men were selected from the age-sex registers of 16 general practitioners working out of two health centers. First-phase data were collected from 1979–1983 and from 1979–1982, respectively, whereas second-phase examinations were conducted during 1984–1988 and 1982–1985, respectively. Response rates in the source populations were 89% (Caerphilly) and 92% (Speedwell), resulting in samples of 2 512 men, aged 45–59 y, for Caerphilly and 2 348 men, aged 45–63 y, for Speedwell. Both study populations were representative samples of the general population. The average follow-up intervals were 61 (± 5) and 38 (± 3) mo (mean ± standard deviation). Full details on prevalence data[5-7] and incidence data[8,9] are described elsewhere.

Traffic noise exposure was operationalized by measurement of the traffic noise emission level at 10 m distance from the center of the streets ($L_{eq,6-22\ h,10\ m\ distance}$). Measurements for a given street were then linked to all individual subjects living on that street. These measurements were classified according to 5 dB(A) categories ranging from 51–55 to 66–70 dB(A). Because the difference between the day and night average A-weighted sound pressure levels was independent of 24-h traffic volume, the emission level was viewed to be a valid indicator for noise load. Type of housing was very similar in both cohorts, and the majority of subjects' homes were situated within a distance of 12 m from the streets, which produced nearly identical results for emission and immission level.

With respect to IHD categorization, the London School of Hygiene chest pain questionnaire was administered and a 12-lead electrocardiogram (ECG) recorded, which was Minnesota coded by two experienced coders. The prevalence of IHD was assessed using the questionnaire (angina pectoris, myocardial infarction) and the ECG recordings (ECG ischemia). The incidence of major IHD was coded if one of the following criteria was fulfilled: death due to IHD (ICD 410–414), clinical myocardial infarction (notified admission to hospital due to acute myocardial infarction coded ICD 410), or ECG myocardial infarction (major ECG changes by Minnesota coding).

The following findings are taken from three major technical reports.[10-12] They refer to the total samples and subsamples of 2 158 (Caerphilly) and 2 118 (Speedwell) men for whom complete data were available on all the variables considered. These were the IHD prevalence and incidence effect variables, the 9 identified endogenous risk factors of systolic and diastolic blood pressure, total and high-density lipoprotein (HDL) cholesterol, total triglycerides, glucose, plasma viscosity, fibrinogen and white cell count. The control variables (dummy-coded if necessary) were age; social status ("professional + intermediate + skilled nonmanual," "skilled manual + partly skilled + unskilled"); family status ("married," "single," "others"); employment status ("employed," "unemployed," "retired," "retired for health reason); smoking ("never," "ex-smoker," "current smoker"), which showed distributional differences over noise categories in the Caerphilly cohort. Control variables, which showed distributional differences over noise categories in Speedwell, were age; social status ("professional + intermediate," "skilled nonmanual," "skilled manual," "partly skilled + unskilled"); family history of myocardial infarction ("positive first grade relative," "negative"); physical activity at work ("active," "not active + occasionally active + retired"); smoking ("never," "ex-smoker," "current smoker"); and body mass index. Furthermore, the results were controlled for possible confounding in a subsample of men with no history of heart attack, stroke, venous thrombosis, diabetes mellitus, gout, and liver and kidney disease in the first phase, which involved 1 452 (Caerphilly) and 1 744 (Speedwell) disease (and treatment) -free men, respectively.

Results

A total of 132 Caerphilly men (5.3%) and 106 Speedwell men (4.5%) died prior to follow up. Eight men in each area could not be contacted, but 12 of these 16 were known to be alive. Of the men available for re-examination, 96% completed a chest pain questionnaire and 93% had a repeat ECG. In total, 153 (Caerphilly) and 98 (Speedwell) major events occurred. In Table 1 are provided migration statistics for the men in each traffic noise category during follow-up intervals; 88% (Caerphilly) and 84% (Speedwell) did not move, whereas 96% (Caerphilly) and 94% (Speedwell) had no change in noise exposure.

Not all of the risk factors considered in the first cross-sectional phases showed predictive power for IHD incidence. Univariate analyses[8,12] revealed the 9 risk factors given in Table 2 to be predictive of IHD incidence. The table gives age-standardized relative odds for each quintile of the risk factor distributions, compared with the quintile with the lowest values in both cohorts. IHD incidence was predicted on the basis of risk factors by calculating multiple models, using logistic regression techniques. This was done independently for each cohort, once by including only endogenous risk factors as predictors and once by adding covariates (as described above) into the logistic model to control for exogenous factors, other than traffic noise. The IHD incidence in each noise category was calculated by summing the predicted logits within each noise group. This was done for the total and the subsample of disease-free (first phase) men to control for prevalent chronic diseases.

In Tables 3 and 4 are shown the number of men; the prevalent cases; the predicted and observed cumulative incidence cases in each noise category for each en-

Table 1.—Migration Statistics for Caerphilly and Speedwell Men During Follow-up Intervals

Traffic noise category [dB(A)]	No. men	No change in address*		No change in traffic noise category†	
Caerphilly					
51–55	1 850	1 617	87.4%	1 754	97.5%
56–50	211	193	91.5%	209	99.5%
61–65	318	280	88.1%	310	99.4%
66–70	133	115	86.5%	126	98.4%
Total	2 512	2 205	87.8%	2 399	98.0%
Speedwell					
51–55	1 633	1 332	81.6%	1 538	96.2%
56–60	262	216	82.4%	225	87.2%
61–65	214	186	86.9%	191	91.0%
66–70	239	186	77.8%	197	85.3%
Total	2 348	1 920	81.8%	2 151	93.5%

*Or death.
†Of men who did not move out of the area.

tire sample, for the total samples with complete data for risk factors and control variables, and for the subsamples of disease-free subjects in each cohort. In Tables 5 and 6 the corresponding prevalence ratios and incidence risk ratios (95% confidence intervals in brackets) are given. In Table 7 are shown similar data for the pooled sample, using the Mantel-Haenszel estimate for calculating standardized relative risks. Adjustments for control variables (covariates) were carried out by logistic regression analyses providing odds ratios as approximations of relative risks.

Prevalence ratios in the highest traffic noise group, compared with the lowest in the Caerphilly/Speedwell/pooled cohort were 0.5–0.6/1.1–1.3/0.9–1.0 for angina pectoris, 1.0–1.2/1.1–1.2/1.1–1.1 for myocardial infarction, 1.2–1.5/1.3–1.4/1.3–1.4 for ECG ischemia, and 1.1–1.2/1.2–1.3/1.2–1.2 for any IHD, depending on the stratum (sample—disease-free subsample) of analysis. The incidence risk ratios for major IHD predicted on the basis of risk factor prevalence by logit summing for the extreme group comparison were 1.1–1.1/1.0–1.1/1.1–1.0, and in contradiction to the observed incidence risk (odds) ratios were 0.5–0.9/0.7–0.8/0.6–0.8. None of the results was statistically significant.

Summary and discussion

In two representative samples (prospective cohort studies), cross-sectional and longitudinal associations between road traffic noise and nine cardiovascular risk factors and major ischemic heart disease were examined. Endogenous risk factors were understood as mediators of the noise-IHD relationship. Because noise acts nonspecifically on the organism and because of individual differences and disposition, the pathways of noise processing may be different, with greater emphasis on either the sympathicotonic or humoral axis. This means, for example, that one subject may react more with lipid changes, the other with changes in blood pressure. In terms of public health policy, the disease outcome (in this case IHD) is the overall factor of interest, regardless of mediating mechanisms reflected in individual risk factor profiles.

In men of the 66–70 dB(A) outdoor traffic noise category [$L_{eq,\ 6\text{–}22\ h,\ 10\ m\ distance}$ = 66–70 dB(A)], the relative risk for prevalent myocardial infarction was approximately 10% higher, for prevalent ECG ischemia approximately 30% higher, and for any ischemic heart disease approximately 20% higher than in those of the 51- to 55-dB(A) category. This corresponds to approximately 10% higher relative risk for the incidence of major IHD predicted from risk factor profiles by logit summing within the follow-up periods of approximately 4 y in men of the highest noise group. In this context, however, it is very difficult to resolve the fact that the observed relative risk of IHD incidence was lower in the highest noise category. None of the findings was significant.

The results have been controlled for a variety of potential confounding factors. However, the incidence data rely on very few cases in the higher noise categories. This reflects a limitation of our study. Inasmuch as it was not designed as a noise study per se, the subjects were distributed unequally among the traffic noise categories. However, a relative risk of 1.5 in the pooled sample would have been significant.

Rejection of the noise hypothesis would be one possible interpretation of the findings. Of the three sources of error—chance, bias, and confounding—chance would be the most attractive interpretation of any of the findings because of the small magnitude and the large variability, including the null value within the confidence intervals.

The results concerning the relative risk of IHD incidence tend not to be stable. A few more (observed) cases in the highest noise group would result in point estimates of the relative risks above 1.0, thus reflecting consistency with prevalence and predicted relative risks. Longer observation periods of the cohorts would provide more stable results on the basis of more cases, especially in this numerically weak group.

The prevalence data for these terms show less variability. Because the men in our study had, on average,

Table 2.—Relative Odds of Ischemic Heart Disease among "Fifth" of the Endogenous Risk Factors

Caerphilly (n = 2 512) Speedwell (n = 2 348)	Quintiles 1 (low)	2	3	4	5 (high)	x^2 trend p value
Systolic blood pressure						
Caerphilly	1.0*	0.7	1.2	1.2	1.3	.017
Speedwell	1.0	3.2	4.8	4.6	6.5	<.001
Diastolic blood pressure						
Caerphilly	1.0	1.8	1.5	1.2	2.5	.017
Speedwell	1.0	0.9	1.3	1.8	2.0	.005
Total cholesterol						
Caerphilly	1.0	0.9	1.1	1.6	1.7	.018
Speedwell	1.0	0.9	0.9	1.6	1.9	.016
HDL cholesterol						
Caerphilly	2.0	1.2	1.3	1.3	1.0	.056
Speedwell	2.3	3.1	2.7	0.9	1.0	.002
Total triglycerides						
Caerphilly	1.0	2.3	2.2	3.2	3.5	<.001
Speedwell	1.0	2.2	3.6	4.4	4.5	<.001
Glucose						
Caerphilly	1.0	1.6	0.9	0.9	2.2	.068
Speedwell	1.0	1.1	1.1	1.3	1.3	.423
Fibrinogen						
Caerphilly	1.0	1.6	1.6	1.9	3.3	<.001
Speedwell	1.0	1.1	2.6	2.8	6.3	<.001
Plasma viscosity						
Caerphilly	1.0	2.1	2.0	3.3	4.1	<.001
Speedwell	1.0	1.8	1.5	6.0	5.2	<.001
White cell count						
Caerphilly	1.0	1.0	1.8	2.4	3.7	<.001
Speedwell	1.0	1.5	1.5	1.6	2.6	.007

*Relative odds.

Table 3.—IHD Prevalence and Incidence in the Caerphilly Cohort

Caerphilly	Traffic noise level, LEQD10 [dB(A)] 51–55	56–60	61–65	66–70	Total
Total sample	1 850†	211	318	133	2 512
Prevalent angina pectoris*	146	14	26	6	192
Prevalent myocardial infarction*	190	23	26	14	253
Prevalent ECG ischemia*	73	4	13	8	98
Prevalent IHD*	324	35	54	25	438
Incident IHD	107	17	22	7	153
Subsample without prevalent IHD	1 526	176	264	108	2 074
Incident IHD	68	11	17	4	100
Sample of men with complete data for risk factors and covariates	1 592	179	277	110	2 158
Incident IHD	84	13	19	3	119
Predicted incident IHD	88.5	11.8	14.9	7.0	122.2
Predicted incident IHD, adjusted	80.5	10.6	13.7	6.2	111.0
Subsample of men with no disease history and complete data for risk factors and covariates	1 064	125	187	76	1 452
Incident IHD	42	7	9	1	59
Predicted incident IHD	57.0	7.2	10.1	4.5	78.8
Predicted incident IHD, adjusted	51.8	6.5	9.3	4.0	71.6

*These criteria are not mutually exclusive. Predicted values on the bases of risk factor prevalence.
†Number of men.

Table 4.—IHD Prevalence and Incidence in the Speedwell Cohort

Caerphilly	Traffic noise level, LEQD10 [dB(A)] 51–55	56–60	61–65	66–70	Total
Total sample	1 633†	262	214	239	2 348
Prevalent angina pectoris*	137	25	21	25	208
Prevalent myocardial infarction*	111	16	17	19	163
Prevalent ECG ischemia*	61	8	7	12	88
Prevalent IHD*	228	34	37	41	340
Incident IHD	72	8	10	8	98
Subsample without prevalent IHD	1 404	228	177	198	2 007
Incident IHD	38	5	4	4	51
Sample of men with complete data for risk factors and covariates	1 481	239	194	204	2 118
Incident IHD	60	6	10	6	82
Predicted incident IHD	56.3	10.3	7.5	7.8	81.9
Predicted incident IHD, adjusted	51.0	9.3	6.9	7.2	74.4
Subsample of men with no disease history and complete data for risk factors and covariates	1 218	202	153	171	1 744
Incident IHD	30	5	3	4	42
Predicted incident IHD	44.3	7.8	5.6	6.6	64.3
Predicted incident IHD, adjusted	40.1	7.1	5.1	6.2	58.5

*These criteria are not mutually exclusive. Predicted values on the bases of risk factor prevalence.
†Number of men.

Table 5.—IHD Prevalence Ratios and Incidence Risk Ratios for Differently Traffic-noise-exposed Groups (Caerphilly Cohort)

Caerphilly	Traffic noise level, LEQD10 [dB(A)] 51–55	56–60	61–65	66–70
Total sample (N = 2 512)				
Prevalent angina pectoris*	1.0	0.8 (0.5–1.4)†	1.0 (0.7–1.6)	0.6 (0.3–1.3)
Prevalent myocardial infarction*	1.0	1.1 (0.8–1.7)	0.8 (0.5–1.2)	1.0 (0.6–1.7)
Prevalent ECG ischemia*	1.0	0.5 (0.2–1.3)	1.0 (0.6–1.9)	1.5 (0.8–3.1)
Prevalent IHD*	1.0	1.0 (0.7–1.3)	1.0 (0.8–1.3)	1.1 (0.7–1.6)
Incident IHD	1.0	1.4 (0.9–2.3)	1.2 (0.8–1.9)	0.9 (0.4–2.0)
Subsample without prevalent IHD (n = 2 074)				
Incident IHD	1.0	1.4 (0.7–2.6)	1.4 (0.8–2.4)	0.8 (0.3–2.3)
Sample of men with complete data for risk factors and covariates (n = 2 158)				
Prevalent angina pectoris, adjusted*	1.0	0.9 (0.5–1.7)	1.2 (0.7–1.9)	0.5 (0.2–1.4)
Prevalent myocardial infarction, adjusted*	1.0	1.0 (0.6–1.7)	0.9 (0.6–1.4)	1.2 (0.6–2.3)
Prevalent ECG ischemia, adjusted*	1.0	0.5 (0.2–1.7)	1.1 (0.6–2.2)	1.2 (0.4–3.5)
Prevalent IHD, adjusted*	1.0	1.0 (0.6–1.5)	1.1 (0.8–1.6)	1.2 (0.7–2.0)
Incident IHD	1.0	1.4 (0.8–2.4)	1.3 (0.8–2.1)	0.5 (0.2–1.6)
-Incident IHD, adjusted	1.0	1.2 (0.7–2.3)	1.3 (0.8–2.2)	0.5 (0.2–1.7)
Predicted incident IHD	1.0	1.2 (0.7–2.2)	1.0 (0.6–1.7)	1.1 (0.5–2.4)
Predicted incident IHD, adjusted	1.0	1.2 (0.7–2.2)	1.0 (0.6–1.7)	1.1 (0.5–2.4)
Subsample of men with no disease history and complete data for risk factors and covariates (n = 1 452)				
Incident IHD	1.0	1.4 (0.7–3.3)	1.2 (0.6–2.5)	0.3 (0.1–2.4)
Predicted incident IHD	1.0	1.1 (0.5–2.4)	1.0 (0.5–1.9)	1.1 (0.5–3.0)
Predicted incident IHD, adjusted	1.0	1.1 (0.5–2.5)	1.0 (0.5–2.0)	1.1 (0.5–2.9)

Notes: Adjusted = adjusted for covariates. Relative risk is calculated as prevalence ratio, prevalence odds ratio, incidence risk ratio, or incidence odds ratio.
*These criteria are not mutually exclusive.
†Relative risk (95% confidence interval).

Table 6.—IHD Prevalence Ratios and Incidence Risk Ratios for Differently Traffic-noise-exposed Groups (Speedwell Cohort)

Speedwell	Traffic noise level, LEQD10 [dB(A)]			
	51–55	56–60	61–65	66–70
Total sample (N = 2 348)				
Prevalent angina pectoris*	1.0	1.1 (0.8–1.7)†	1.2 (0.8–1.8)	1.3 (0.8–1.9)
Prevalent myocardial infarction*	1.0	0.9 (0.5–1.5)	1.2 (0.7–1.9)	1.2 (0.7–1.9)
Prevalent ECG ischemia*	1.0	0.8 (0.4–1.7)	0.9 (0.4–1.9)	1.3 (0.7–2.5)
Prevalent IHD*	1.0	0.9 (0.7–1.3)	1.2 (0.9–1.7)	1.2 (0.9–1.7)
Incident IHD	1.0	0.7 (0.3–1.4)	1.1 (0.6–2.0)	0.8 (0.4–1.6)
Subsample without prevalent IHD (n = 2 007)				
Incident IHD	1.0	0.8 (0.3–2.0)	0.8 (0.3–2.3)	0.8 (0.3–2.1)
Sample of men with complete data for risk factors and covariates (n = 2 118)				
Prevalent angina pectoris, adjusted*	1.0	1.1 (0.7–1.8)	1.1 (0.6–1.9)	1.1 (0.7–1.9)
Prevalent myocardial infarction, adjusted*	1.0	1.0 (0.6–1.8)	1.2 (0.7–2.1)	1.1 (0.6–1.9)
Prevalent ECG ischemia, adjusted*	1.0	0.9 (0.4–2.0)	1.0 (0.4–2.1)	1.4 (0.7–2.9)
Prevalent IHD, adjusted*	1.0	1.0 (0.6–1.5)	1.2 (0.8–1.9)	1.3 (0.8–1.9)
Incident IHD	1.0	0.6 (0.3–1.3)	1.3 (0.7–2.5)	0.7 (0.3–1.7)
-Incident IHD, adjusted	1.0	0.6 (0.3–1.5)	1.3 (0.6–2.5)	0.7 (0.3–1.8)
Predicted incident IHD	1.0	1.1 (0.6–2.1)	1.0 (0.5–2.3)	1.0 (0.5–2.1)
Predicted incident IHD, adjusted	1.0	1.1 (0.6–2.2)	1.0 (0.5–2.3)	1.0 (0.5–2.2)
Subsample of men with no disease history and complete data for risk factors and covariates (n = 1 744)				
Incident IHD	1.0	1.0 (0.5–2.6)	0.8 (0.3–2.6)	1.0 (0.3–2.7)
Predicted incident IHD	1.0	1.1 (0.5–2.4)	1.0 (0.5–2.5)	1.1 (0.5–2.5)
Predicted incident IHD, adjusted	1.0	1.1 (0.5–2.3)	1.0 (0.4–2.5)	1.1 (0.5–2.5)

Notes: Adjusted = adjusted for covariates. Relative risk is calculated as prevalence ratio, prevalence odds ratio, incidence risk ratio, or incidence odds ratio.
*These criteria are not mutually exclusive.
†Relative risk (95% confidence interval).

lived in their homes for approximately 13 y prior to the beginning of the study,[12] the analytic power of the prevalence data corresponds in some respects to that of a population-based, retrospective case-control study. In addition, doubling or halving of traffic noise volume would yield a change in noise level of approximately 3 dB(A), causing little (nondifferential) misclassification. All other general arguments against prevalence studies (survivors, self-selection, or sensitive persons moving to low noise categories) would underestimate any true noise effect. We also suggest that the relative risks of IHD incidence predicted from risk factors by logit summing have a somewhat higher validity (although being unstable in terms of case numbers) than the observed relative risks. This is because here each individual contributes to the numerator with his risk factor constellation (continuous effect), rather than the numerator being restricted to only a very few diseased cases (discrete effect). This makes the outcome measure less susceptible to random variation. However, this argument depends on the validity of the model that endogenous factors are mediators of the noise-IHD relationship.

Selection bias is relatively unlikely to have occurred because of the high response rates in the total samples in the initial phase (about 90%) and follow-up phase (about 95%). The subjects were not aware of the noise focus, which also minimized the effects of recall bias. The same goes for information (interviewer) bias, due to the standardized, well-defined criteria for exposure and disease, including blind and double-blind techniques for objective measurements. Interviews were not conducted in the homes but in a clinic.

Confounding can never be identified fully. The results have been controlled for a number of potentially confounding factors, including standard factors like age, sex, social class, and the exogenous risk factors smoking, obesity, and hereditary disposition (controlling for endogenous factors would be inappropriate given our understanding of the noise hypothesis). Body mass index can be viewed, in part, as an indicator for the impact of nutrition on IHD but does not control, for example, for high or low cholesterol intake. On the other hand, there is no reason to believe that nutrition habits were distributed differentially over noise categories because the cohorts were relatively homogeneous with respect to the sociodemographic factors considered.

In general, nondifferential influences dilute the true effect, thus leading to a conservative interpretation of

Table 7.—IHD Prevalence Ratios and Incidence Risk Ratios for Differently Traffic-noise-exposed Groups (Caerphilly + Speedwell Cohort Pooled)

Caerphilly + Speedwell (Mantel Haenszel estimate)	Traffic noise level, LEQD10 [dB(A)]			
	51–55	56–60	61–65	66–70
Total sample (N = 4 860)				
Prevalent angina pectoris*	1.0	1.0 (0.7–1.4)†	1.1 (0.8–1.5)	1.0 (0.7–1.4)
Prevalent myocardial infarction*	1.0	1.0 (0.7–1.4)	0.9 (0.7–1.2)	1.1 (0.8–1.6)
Prevalent ECG ischemia*	1.0	0.7 (0.4–1.2)	1.0 (0.6–1.5)	1.4 (0.9–2.3)
Prevalent IHD*	1.0	0.9 (0.8–1.2)	1.1 (0.9–1.3)	1.2 (0.9–1.5)
Incident IHD	1.0	1.1 (0.7–1.6)	1.2 (0.8–1.7)	0.8 (0.5–1.4)
Subsample without prevalent IHD (n = 4 081)				
Incident IHD	1.0	1.1 (0.7–1.9)	1.2 (0.8–2.0)	0.8 (0.4–1.6)
Sample of men with complete data for risk factors and covariates (n = 4 276)				
Prevalent angina pectoris, adjusted*	1.0	1.0 (0.7–1.5)	1.1 (0.8–1.6)	0.9 (0.6–1.4)
Prevalent myocardial infarction, adjusted*	1.0	1.0 (0.7–1.5)	1.0 (0.7–1.5)	1.1 (0.7–1.7)
Prevalent ECG ischemia, adjusted*	1.0	0.7 (0.4–1.4)	1.1 (0.6–1.8)	1.3 (0.8–2.4)
Prevalent IHD, adjusted*	1.0	1.0 (0.7–1.3)	1.12(0.9–1.5)	1.2 (0.9–1.6)
Incident IHD	1.0	1.0 (0.6–1.6)	1.3 (0.9–1.9)	0.6 (0.3–1.2)
-Incident IHD, adjusted	1.0	0.9 (0.6–1.5)	1.3 (0.9–1.9)	0.6 (0.3–1.3)
Predicted incident IHD	1.0	1.2 (0.8–1.8)	1.0 (0.6–1.5)	1.1 (0.7–1.8)
Predicted incident IHD, adjusted	1.0	1.2 (0.7–1.8)	1.0 (0.7–1.6)	1.1 (0.6–1.8)
Subsample of men with no disease history and complete data for risk factors and covariates (n = 3 196)				
Incident IHD	1.0	1.2 (0.7–2.2)	1.1 (0.6–2.0)	0.7 (0.3–1.7)
Predicted incident IHD	1.0	1.1 (0.6–1.8)	1.0 (0.6–1.7)	1.1 (0.6–2.0)
Predicted incident IHD, adjusted	1.0	1.1 (0.6–1.8)	1.0 (0.6–1.7)	1.1 (0.6–2.0)

Notes: Adjusted = adjusted for covariates. Relative risk is calculated as prevalence ratio, prevalence odds ratio, incidence risk ratio, or incidence odds ratio.
*These criteria are not mutually exclusive.
†Relative risk (95% confidence interval).

findings. Misclassification may be present with re-ct to exposure, given that only outdoor noise levels re considered. The investigations in the second ase ("noise questionnaire") showed that there was difference between the noise groups in bedroom entation, and only in the Speedwell cohort in living m orientation, whereas windows were more often ot closed throughout the year in noisy areas.[12] The ections of all these effects again tend to play down l associations. We feel that the outdoor noise level is appropriate measure of exposure with respect to blic health because it considers all adverse effects of aling with the noise (keeping the windows closed y be a strain) and takes into account individual strat-ies for coping with the noise.

n environmental epidemiology we are normally con-rned about ambient (not acute toxic) concentrations agents and exposures that might have some patho-nic potential in the long term. Conclusions cannot be awn from one study alone. Even small relative risks ay have some relevance to public health when the mber of subjects affected is high.

In the Bonn traffic noise study[13] (males + females) and e Amsterdam aircraft noise study,[14] prevalence ratios for myocardial infarction of 1.3 and 1.2 (males) and 1.9 (females) for subjects of the extreme groups of noise exposure were found. The Doetinchem traffic noise study[15] also provided data on IHD, but no extreme groups were considered. The prevalence ratio for ECG ischemia was 1.1 (females) between the two halves of the sample, cut on a noise criterion. In most of these studies, the confidence intervals for the relative risk included the null value. All these findings are consistent with the findings in the Caerphilly and Speedwell studies, except for the actual IHD incidence relationship with traffic noise observed here.

Statistical proof of small effects has to be founded on large samples, but it is, in principle, feasible. On the other hand, the detection of relative risks below approximately 1.2 with observational epidemiological methods can hardly be achieved, even with very specific methods of measurements due to random variation, residual confounding, and insufficient control of similarly weak confounders.[16] The whole issue of weak associations was discussed recently.[17] Experimental epidemiologic methods (intervention studies) may be a useful tool in environmental noise research. Further traffic noise studies should comprise areas and subjects

exposed to levels higher than $L_{eq,day}$ = 70 dB(A) for larger magnitude of effect or look at predetermined sensitive subgroups.[18] Consistency, dose-response relationship, and biological plausibility are important issues in the interpretation of findings.[19]

The Caerphilly and Speedwell studies, although revealing no statistically significant results, could be one piece in the puzzle of quantifying the IHD risk for subjects exposed to traffic noise via meta-analysis techniques.[20] Ten years of follow-up data may provide a better picture of the IHD incidence risk for men in the cohort.

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Bab98

THE CAERPHILLY AND SPEEDWELL STUDIES, 10 YEAR FOLLOW-UP

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1. INTRODUCTION

This paper refers to a series of articles relating to different follow-up phases of two cohort studies in which the effects of road traffic noise on the cardiovascular system were investigated [1,2,3]. The hypothesis was tested that prolonged exposure to traffic noise at home increases the risk for ischemic heart disease (IHD). Full information about the 10 yr follow-up results is given elsewhere [4,5].

2. METHODS

Two cohorts of 2512 (Caerphilly, South Wales) and 2348 (Speedwell, England) middle aged men, aged 45-59 yr and 45-63 yr, respectively, were recruited in the United Kingdom to study the predictive power of already known and new risk factors for ischaemic heart disease (IHD) [1,2]. Both study designs followed identical protocols. First follow-up investigations were carried out after approximately 4 yr (second phase), second follow-up's after observation periods of 120 (standard deviation = 6) and 112 (standard deviation = 3) months, respectively, which approximates to 10 yr (third phase) [6]. Since a detailled noise questionnaire was only administered during the second phase, most of the follow-up analyses presented here refer to the observation period from phase 2 to phase 3. The reconstructed cohort for phase 2 of the Caerphilly sample consisted of 1951 men of the original cohort who were seen again at the clinic, plus 447 men of the same age range who had moved into the area since the original cohort was identified. This gave a total of 2398 men between 47 and 67 yrs of age. The reconstructed Speedwell cohort consisted of the 2055 men of the original cohort who were seen again at the phase 2 clinics, aged 48-66 y. The statistical noise analyses with respect to the reconstructed cohorts were carried out in pooled sample of 3997 men, aged 47-67 yr, who actually filled in the noise questionnaire during the second phase. The

average follow-up interval for these men was 61 (standard deviation = 6) and 75 (standard deviation = 6) months which approximates to 6 y.

Noise measurements were carried out in every street where the subjects lived. Outdoor noise levels were related to each individual's home. The subjects were grouped into 5 dB(A)-categories of the A-weighted average sound pressure level outdoors from 6–22 hr ($L_{eq,\ 6\text{-}22\ h}$). 24 hr continuous noise measurements were carried out in a number of different type of streets revealing day/night differences of 7 to 11 dB(A) of the average sound pressure level. This is a common finding in urban areas (no freeways). 24h noise levels are usually 1 to 3 dB(A) lower than daytime noise levels [7], which means that the daytime L_{eq} can be used as an indicator for the overall traffic noise emission from the streets. Subjective measures of annoyance and disturbance due to traffic noise at home were also assessed. The corresponding results are given in the original papers [4,5].

Incidence of ischemic heart disease was defined when a major IHD event occured between the follow-up phases. These events could be IHD death (coded ICD 410–414 on death certificate), definite clinical non-fatal myocardial infarction (MI) meeting WHO criteria regarding clinical history, ECG and enzyme changes, or ECG defined MI meeting WHO criteria [6]. Death certificates were available for all except three men who died before the final examination. In Caerphilly 94 % and in Speedwell 87 % of the 10 yr follow-up survivors were seen at the clinic again. A further 2 % and 3 %, respectively, had postal cardiovascular questionnaires after hospital admission for chest pain; then records from all local hospitals were searched for men from the cohorts who have been admitted to hospital with any diagnosis in the range ICD 410–414.

All statistical analyses on the relationship between traffic noise (dummy-coded) and IHD incidence were controlled (model adjusted) for the potentially confounding factors age, social class, maritial status, smoking, body mass index, family history of myocardial infarction, employment status, physical activity at leisure, prevalence of IHD and pre-existing-health conditions [4,5]. Furthermore, the analyses refering to the period between phase 2 and phase 3 were controlled for subjective noise sensitivity based on a single item and area (cohort) in the model. Room orientation (rooms facing the street or not) and window opening habits (windows closed or not) were considered in these analyses to improve individual exposure assessment. Where neither living rooms nor bedrooms were facing the street of the address (being checked for other noisy streets), subjects were grouped into a 15 dB(A) quieter noise category; which in fact was the quietest of 51-55 dB(A). Subjects who had answered that they never opened any windows facing the street when they spend time inside these rooms, were grouped into a 10 dB(A) quieter noise category. Simultaneous indoor/outdoor measurements carried out in 300 households in the areas revealed these average sound level differences in conditions of non-facing the street and open windows, and of facing the street and closed windows (only single framed windows). Years in residence before the subjects entered the study were considered in the analyses either by exclusion (subgroup ≥ 15 yr in residence) or interaction of residence period with noise level in the models. Unfortunately, this information was only available for the reconstructed cohorts (6 yr follow-up). Multiple logistic regression technique (using SPSS 6.0) was applied to calculate relative risk estimates (odds ratio) and 95%-confidence intervals (standard error).

3. RESULTS

10 yr follow-up (phase 1 to 3)

The 10 yr cumulative incidence of major IHD was 312 (of 2512) and 291 (of 2348) cases in Caerphilly and Speedwell, respectively. The mean age (recorded as age last birthday) of the men at recruitment was 52.1 yr (standard deviation = 4.4) and 54.2 yr (standard deviation = 4.4), respectively. The average annual incidence rates (unadjusted for decreasing size of cohorts) of 1.24 % and 1.32 % per year turned out to be very similar in both cohorts. Due to missing values in control variables the eligible sample size varies slightly between the variables. For 2369 and 2330 men, respectively, complete data were available in all the covariates considered in the multiple models. Table 1 gives odds ratios of the relationship between these variables and IHD incidence in the three samples. Smoking, IHD prevalence, family history of IHD, age, body mass index, unemployment, pre-existent disease and area were significantly associated with a higher IHD risk. In Table 2, crude and adjusted relative risks (odds ratios and 95%-confidence intervals) are shown in reference to the lowest traffic noise category of 51-55 dB(A). In Caerphilly, relative risks greater than 1 were found in the 56-60 dB(A)-category and the highest noise category of 66-70 dB(A), with marginal and non-significant odds ratios of ca. 1.1. In Speedwell, no relative risk greater than 1 was found for any higher noise categories. For moderate noise levels of 56-60 dB(A), a borderline significantly lower IHD risk was found compared with the reference group.

Table (1) Associations between control variables and IHD incidence

Control variable (C=Caerphilly 10 yr, S=Speedwell 10 yr, C+S=Pooled 6 yr)	Odds ratio C	S	C+S
Social class (manual vs. partly skilled or unskilled)	1.2	0.8	1.1
Social class (non-manual vs. partly skilled or unskilled)	1.4	1.2	1.2
Social class (professional or intermediat vs. partly skilled or unskilled)	1.0	0.9	1.1
Employment status (employed vs. unemployed)	—	—	0.7
Smoking (ex-smoker vs. non-smoker)	1.5	1.5	1.4
Smoking (current smoker vs. non-smoker)	2.4	2.3	2.1
Physical activity at leisure (active vs. inactive)	0.9	0.9	—
Family history of IHD	1.3	1.2	1.5
IHD prevalence	2.5	2.6	2.3
Prevalence of pre-existing diseases	1.5	2.0	1.5
Subjective noise sensitivity (not at all+a little+moderate vs. much+very much)	—	—	0.9
Age (per year)	1.06	1.06	1.05
Body mass index (per kg/m²)	1.06	1.03	1.06
Area (Speedwell vs. Caerphilly)	—	—	1.55

6 yr follow-up (phase 2 to 3)

The 5-6 yr cumulative incidence of major IHD was 161 (of 2398) and 191 (of 2055) subjects with events in the Caerphilly and Speedwell cohorts with mean age 57.4 yr (standard deviation = 4.5) and 57.3 (standard deviation = 4.3), respectively. Altogether 3997 men filled in the noise questionnaire. Due to missing values, adjusted analyses refer to the pooled sample of 3950 men of whom complete information on noise questionnaire and control variables was available. The men of the pooled sample had an average age of 57.3 yr (standard deviation = 4.5). The average annual incidence rate was 1.38 %. Table 2 gives relative risks for each traffic noise category. Relative risks greater than 1 were only seen in the highest noise category with odds ratios of ca. 1.1 in the crude

and adjusted analyses. These were not significant. When the noise level became corrected for window orientation the relative risk increased to 1.2 and further up to 1.3 when window opening habits was considered, still being not significant. Exclusion of subjects who had not been living at their last address when they were recruited for at least 15 years (which applies for a third of men), revealed odds ratios of 1.2 (address), 1.3 (corrected for window orientation), 1.6 (corrected for window orientation and window opening habits) for the remainder of men of the 66-70 dB(A)-noise-category compared to those in the quietest category of 51-55 dB(A). Again none of the results were significant due to the smaller numbers of men in the higher noise categories. At moderate noise levels odds ratios less than 1 were found and were sometimes significant. Another way to account for exposure period was to treat years in residence as an interaction term with noise level (noise * years of residence) in the model to replace the noise factor. The results are given in Table 2. Relative IHD risks greater than 1; 1.007 (address), 1.010 (corrected for window orientation) and 1.017 (corrected for window orientation and window opening habits) for a year's increase in residence at the given noise exposure (multiplicative model) were only found for men in the highest noise category - the latter was borderline significant ($p < 0.10$).

Table (2) Relative risk of IHD incidence for differently traffic-noise-exposed groups (odds ratio, 95%-confidence intervals)

Sample (C=Caerphilly, S=Speedwell, C+S=Pooled)	Traffic noise level [dB(A)] 51-55	56-60	61-65	66-70
C 10 yr - total, crude	1.00	1.04 (0.68-1.59)	0.96 (0.67-1.39)	1.11 (0.66-1.86)
C 10 yr - complete data, crude	1.00	1.06 (0.68-1.65)	0.91 (0.62-1.35)	1.06 (0.60-1.85)
C 10 yr - complete data, adjusted [1]	1.00	1.07 (0.68-1.68)	0.87 (0.58-1.30)	1.07 (0.60-1.91)
S 10 yr - total, crude	1.00	0.65 (0.41-1.01)	0.85 (0.54-1.33)	1.04 (0.70-1.54)
S 10 yr - complete data, crude	1.00	0.65 (0.41-1.02)	0.85 (0.54-1.33)	1.00 (0.66-1.49)
S 10 yr - complete data, adjusted [1]	1.00	0.67 (0.42-1.07)	0.76 (0.48-1.22)	0.92 (0.61-1.41)
C+S 6 yr - total, crude	1.00	0.78 (0.50-1.17)	0.72 (0.47-1.06)	1.13 (0.75-1.68)
C+S 6 yr - complete data, crude	1.00	0.76 (0.49-1.17)	0.71 (0.47-1.08)	1.08 (0.71-1.64)
C+S 6 yr - complete data, adjusted [1]	1.00	0.71 (0.46-1.11)	0.68 (0.44-1.03)	1.07 (0.70-1.65)
C+S 6 yr - as above, per year in residence	1.00	0.989 (0.971-1.007)	0.990 (0.974-1.006)	1.007 (0.992-1.023)
C+S 6 yr - subsample ≥15 yr in residence	1.00	0.70 (0.40-1.20)	0.60 (0.35-1.03)	1.20 (0.72-2.03)
Accounted for window orientation [2]				
C+S 6 yr - complete data, adjusted [1]	1.00	0.82 (0.51-1.31)	0.64 (0.39-1.04)	1.16 (0.73-1.86)
C+S 6 yr - as above, per year in residence	1.00	0.995 (0.976-1.014)	0.989 (0.971-1.007)	1.010 (0.993-1.028)
C+S 6 yr - subsample ≥15 yr in residence	1.00	0.82 (0.46-1.46)	0.49 (0.25-0.95)	1.30 (0.73-2.32)
Accounted for window orientation and window opening [2] [3]				
C+S 6 yr - complete data, adjusted [1]	1.00	0.69 (0.42-1.12)	0.64 (0.44-1.03)	1.31 (0.78-2.21)
C+S 6 yr - as above, per year in residence	1.00	0.988 (0.969-1.008)	0.983 (0.961-1.006)	1.017 (0.998-1.036)
C+S 6 yr - subsample ≥15 yr in residence	1.00	0.67 (0.36-1.24)	0.45 (0.20-0.98)	1.59 (0.85-2.97)

[1] Adjusted for covariates
[2] In case of no living or bedroom windows are facing the road: noise level = noise level minus 15 dB(A)
[3] In case of windows are kept close throughout the whole year: noise level = noise level minus 10 dB(A)

4. DISCUSSION

In the earlier cross-sectional analyses of the Caerphilly and Speedwell studies, adjusted non-significant relative risks for the prevalence of IHD between 1.2 and 1.3 were seen in

higher traffic noise level exposed men ($L_{eq,\ 6\text{-}22\ h}$ = 66-70 dB(A)) as compared to non-exposed men ($L_{eq,\ 6\text{-}22\ h}$ = 51-55 dB(A)) in both cohorts [3]. The prevalence of endogenous risk factors in these men suggested a relative risk of 1.1 for the incidence of IHD during the follow-up period. Intermediate 4-years follow-up analyses (phase 1 to 2) which were based on a very few incident cases (5-15 depending on the subgroups considered) revealed non-significant relative risks between 0.6 and 0.8 in men of the highest exposed noise category of the pooled cohort [3]. The data presented here refer to phase 3 of the prospective cohort studies carried out in the two locations. After 10 years of follow-up, the number of incident cases in the highest traffic noise group was considerably higher (18-50) than at phase 2, due to aging of the subjects and length of observation period. No statistically significant noise effects could be detected when the traffic noise level was considered as factor of exposure. The adjusted relative risks for men in the highest traffic noise category of 66-70 dB(A) were 1.1 and 0.9 in Caerphilly and Speedwell, respectively, after 10 yrs of follow-up, and 1.1 after 6 yrs of follow-up in the reconstructed pooled cohort which refers to the observation period from phase 2 to 3. To account for a longer induction period, a subsample of men was formed who had lived at least for 15 years at their present address when recruited. This led to a slightly higher adjusted relative risk for men of the 66-70 dB(A)-category of 1.2 in the pooled 6 yr follow-up cohort where the corresponding information was available. Given the range of the confidence intervals these findings can hardly be interpreted as noise effects. Nevertheless, the results are within the magnitude of effect that was to be expected, considering the findings of the cross-sectional phases and other new studies in mind [8].

From the methological point of view, the detection limit of observational epidemiological studies has to be considered as 1.2-1.3 for the risk or odds ratio because of the unknown impact of random variation and weak or residual confounding [9]. The precise measure of exposure (for magnitude of effect estimate), the precise measure of disease, the recruiting of large samples (for precision of effect estimate), and the comprehensive assessment of potential confounding factors (to reduce bias) is essential to handle the problem of small effects. In the present study, sample size (under aspects of noise research) was not a matter of influence because the noise team joined in with a running heart program. Also, the entire topic was new and suggestions from other studies about the magnitude of the effect hardly known. With regard to disease assessment, misclassification was minimized, and its impact on the results was estimated to be extremely low. This was due to the standardized protocol, the integrative use of all information obtained from the own clinical measurements, hospital and general practitioner records, and death certificates, and to the effort made to follow up each subject. A number of possible confounding factors were considered in the analyses. However, exposure misclassification may very well remain because the outdoor noise exposure differs from the perceived noise of the individual.

The 6 yr follow-up investigation of the pooled reconstructed cohorts gave an opportunity to further reduce exposure misclassification on the basis of the questionnaire information about room orientation and window opening habits. While adjusted relative risks of 1.1 and 1.2 were found for men in the 66-70 dB(A)-category in the total sample and in the 15 yr in residence subsample with respect to the outdoor noise level, the odds ratios rose to 1.2 and 1.3, respectively, when the orientation of the living and the bed-

room was included in the analyses. It increased further to 1.3 and 1.6, respectively, when the information about window opening habits was considered. Still, none of the results were significant. Living in streets with traffic noise levels of 66-70 dB(A) was associated with an increase in relative risk of 1.01 and 1.02 per year in residence, the latter which accounts for window orientation and window opening habits achieved borderline significance. The finding of an increase in effect estimate when improving exposure assessment is improved supports the noise hypothesis and may be interpreted to imply causality, to some extent.

From the Caerphilly and Speedwell studies on their own, it cannot be deduced that traffic noise (level) increases the risk for myocardial infarction or any other form of ischaemic heart disease. However, the results provide another source of information concerning the potential hazard of traffic noise. At present, opinion will have to be based on mostly non-significant but consistent findings amongst studies. The data presented here may feature future meta-analytic approaches, once further studies are available on this topic which is of great environmental concern [10].

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(2) Path analysis was performed with 12 variables obtained via the surveys. The path model explained about 50% of the variation in the annoyance responses.

(3) A strong effect of sleep disturbance on road traffic annoyance was confirmed by path analysis. It also showed important effects of hearing disturbance, satisfaction with the area, employment status and self-reported sensitivity to road traffic annoyance.

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PSYCHO-SOCIAL EFFECTS OF TRAFFIC NOISE EXPOSURE

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In this paper a study of psycho-social effects of exposure to high levels of road traffic noise is presented. A questionnaire was constructed to evaluate not only annoyance reactions and sleep disturbance effects of noise, but also more long-term effects on psycho-social well-being (PSW). PSW was evaluated by 26 questions concerning depression, relaxation, activity, passivity, general well-being and social orientation. The postal questionnaire was answered by 151 persons in a quiet city area and 97 persons in an area exposed to an L_{eq} level of 72 dB(A). The results showed that a higher proportion of those who lived in the noisy area in apartments with windows facing the street more often felt depressed. Those who had windows facing the courtyard, in the noisy area, however, were not more depressed than those who lived in the quiet area. Methodological difficulties in this type of study are also discussed in the paper.

1. INTRODUCTION

There is a great amount of literature on the effect of environmental noise on general annoyance and sleep disturbances. It is also well known that environmental noise may affect performance and conversation. As regards psychiatric symptoms, Abbey-Wickrama *et al.* in 1969 [1] found a significant relationship between psychiatric symptoms and exposure to aircraft noise around Heathrow Airport. But a renewed study by Tarnopolsky and Gattony in 1973 [2] could not confirm these earlier results. However, they did find a significant relationship between annoyance caused by noise and the prevalence of psychiatric symptoms. Relster [3] investigated two areas exposed to high and low levels of road traffic noise in Copenhagen in 1965. She found a greater use of tranquilizers (25% versus 17%) and a higher frequency of medical consultations for psychiatric problems (19% versus 12%) in the high-noise area.

In a survey in Gothenburg [4] among persons living at different distances from a road with high levels of road traffic noise, it was found that symptoms such as tiredness, headache and "nervous stomach" were felt more frequently by residents of the noisiest area. Mood was also negatively affected by noise.

Against this background, we wanted to elucidate possible health effects of noise such as psycho-social symptoms. A questionnaire was developed and tested in a survey in a noisy and a quiet area.

2. MATERIALS AND METHODS

2.1. QUESTIONNAIRE

The hypothesis and basis for the development of the questionnaire was that physical and psycho-social symptoms and reduced work capacity may occur as an effect of general

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annoyance and sleep disturbances caused by noise exposure. These symptoms, of course, may also be dependent upon other circumstances, for example chronic illness or difficulties in the family situation or work conditions. The individual capacity to handle stress might also be of importance for the development of different symptoms. The questionnaire concerned living and work environment, sleep and general questions. Psycho-social well-being (PSW) was evaluated by 26 questions. These questions were analysed by a multi-factorial analysis and divided into six factors: well-being, depression, relaxation, activity, passivity and social orientation. The score of each factor was made up by the sum of the score (1-4) on the questions within the factor. A total score was also calculated for psycho-social well-being (PSW) containing the sum of the scores of each question.

The questionnaire was distributed by mail together with an introductory letter. Two letters were sent after 10 and 20 days, respectively, as reminders to those who did not answer after the first or the second letter.

2.2. NOISE EXPOSURE

Two areas in the center of Gothenburg were chosen for the investigation, one quiet area with background noise levels below 50 dB(A) L_{eq} and another area exposed to high levels of road traffic noise. The noise exposure is shown in Table 1. The total number of vehicles was 34 000, of which about 10% were heavy vehicles. The L_{eq} level in dB(A) was 72 and the maximum noise levels during day and night were 91 and 84, respectively.

TABLE 1

Noise exposure

	Noisy area	Quiet area
Number of vehicles		
Total	34 000	—
Heavy	3 060	—
L_{eq}	72	< 50
Maximum dB(A)		
Day	91	—
Night	84	—

2.3. SUBJECTS

In each area, one person from each household was selected who was between 18 and 75 years and had resided in the area for at least one year. An equal number of men and women were chosen in each area, with as balanced an age distribution as possible. A total number of 450 persons was chosen. The response rate was 71% in the quiet area and 57% in the noisy area.

Only questionnaires from subjects who lived in apartments with windows facing the street were included. Some characteristics of the subjects are shown in Table 2. It can be seen from the table that, in the noisy area, the mean age and the working frequency was similar, but the time of residence was shorter.

TABLE 2

Subjects

	Noisy area	Quiet area
Number of respondents	97	151
Age (mean)	50·6	53·3
Time of residence (mean), L_{eq}	8·8	16·4
Working outside home (%)	58	53

3. RESULTS

3.1. SATISFACTION WITH THE ENVIRONMENT

The results from the experience of the environment of the noisy area are shown in Table 3. The table shows that road traffic noise and exhausts are experienced as rather or very annoying by about 60%. Vibrations from heavy traffic are reported to be annoying by 25%. Dust, soot and smell from industries are also reported as annoying.

TABLE 3

Environmental nuisance

Source	% Rather + very annoyed
Road traffic noise	65
Exhausts	58
Vibrations	25
Dusty/sooty industries	30
Smelly industries	12

In the noisy area, 14% were not satisfied with the environment and 23% wanted to move away from the area. In the quiet area only 1% was unsatisfied with the environment.

3.2. SLEEP QUALITY

Results as regards different sleep quality parameters are shown in Table 4. It can be seen from the table that a greater number of people had difficulty in falling asleep, sleep quality tended to be lower and persons felt less rested in the morning in the noisy area. The use of sleeping pills was greater as was the use of earplugs.

TABLE 4

Sleep quality

	Noisy area	Quiet area	*P*-value (one-sided test)
Difficulties in falling asleep (%)	30	17	0·05
Sleep quality 1-10 (mean)	7·2	7·7	0·06
Rested in the morning 1-10 (mean)	6·1	6·6	0·05
Use of sleeping pills (%)	24	14	0·04
Use of earplugs (%)	14	4	0·001

Several factors were related to sleep quality. Older people had significantly worse sleep quality, as well as those who were divorced or widowed or chronically ill. Those who had a lower score on PSW also had lower sleep quality. Sleep quality was also related to annoyance from noise and to working factors such as psychological and physical loads, feelings of tiredness after work and shift work. The work factors were related to sleep only in the noisy area, however, not in the quiet area.

3.3. PSYCHO-SOCIAL WELL-BEING (PSW)

The results regarding PSW are shown in Table 5. Only one aspect of PSW was different in the noisy area, this being the factor of depression. On the single questions having to do with this factor, people more often answered that they felt "resigned" and that "everything in life was a burden to them".

The relation between PSW and other factors was also investigated. PSW was lower among those who were chronically ill and those who more often were bothered by "nervous

TABLE 5

Psycho-social well-being (PSW)

	Noisy area	Quiet area	*P*-value (one-sided test)
Depression	17·2	17·9	0·01
(not worth living	3·63	3·76	0·08
resigned	3·28	3·53	0·002
insecure	3·45	3·57	—
nervous	3·60	3·90	—
feel life is a heavy burden	3·21	3·37	0·03)

stomach", tiredness and headaches. PSW was also related to sleep quality and to annoyance from noise and exhausts from road traffic. Finally, PSW was seen to be related to satisfaction at work, work load and tiredness after a workday.

4. COMMENTS

The primary aim of the study was to develop and test a questionnaire that could elucidate possible effects on psycho-social well-being of exposure to environmental noise. The questionnaire proved to fulfil the requirement, as differences in extent of depression were found between a quiet and a noise exposed area, and a significant relationship was found between annoyance caused by noise and PSW.

The response rate in this study was only 57–75%, and the results might have differed if all the persons selected had answered. Some important methodological questions may thereby be raised. Is it possible to increase the response rate? Is it possible to study long-term effects of environmental noise by using other methods? Do those who respond to the questionnaire differ from those who do not?

The known reasons for failing to respond in this study were that some of the questions, having especially to do with PSW, were experienced as being too personal. Some persons said that they never answered statistical investigations and some persons felt that they did not have the time. Thus it is important to motivate people to participate in the study.

As concerns the second question (whether it is possible to use other methods), it might be possible to follow the same persons living in a noisy area over a number of years instead of just using a control group. On the other hand, the duration of residence is much shorter in noisy areas and thus the drop-out rate would still be high. In regard to the third question, an analysis was made of the results on PSW of those who answered the questionnaire immediately, and of those who responded after the first and after the second reminder letters. The hypothesis was that those who answered last would show a greater similarity to those who did not answer. This analysis showed that those who answered early had a greater PSW in both the noisy and quiet areas and thus the results might reveal an overly positive PSW score. However, as this was the case for both areas, the differences in PSW between areas would probably be the same even if the response rate had been higher.

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Öhr 98

SLEEP DISTURBANCES BEFORE AND AFTER REDUCTION IN ROAD TRAFFIC NOISE

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1. INTRODUCTION

There is extensive evidence in the literature that environmental noise causes various adverse effects and the evidence are strongest for annoyance, sleep disturbance and performance by school children [1]. Concerning long term non-auditory health effects, e.g. effects of noise-induced sleep disturbances and psychosocial wellbeing, the evidence is weaker and more research is required. Longitudinal studies and intervention studies in connection with extensive noise abatement activities, 'natural experiments' are far more informative than cross-sectional studies.

2. BACKGROUND AND AIM

This paper deals with an investigation of sleep disturbances, annoyance and psychosocial symptoms among residents living at different distances from a highly trafficked road in Göteborg city, Sweden. Two studies were performed among people living in the same residential area in 1986 and 1987 before and after traffic regulations during night [2,3]. The results from these studies indicated that road traffic noise not only causes adverse effects on sleep quality and various daily activities, but may also cause more long term effects on psychosocial health and wellbeing. These previous studies also showed that prohibition of heavy vehicles during night was not sufficient to reduce adverse effects on sleep and general wellbeing. A tunnel for the road traffic is now being built to facilitate transport and to solve the noise problems in the residential area. This provided a good opportunity to perform a new study before and after the opening of the tunnel.

The aim of this investigation was to assess:

(1) the adverse effects on people of long term exposure to road traffic in terms of sleep quality, annoyance, activity disturbances and psycho-social wellbeing

(2) and how people living in the area are affected by the changed traffic situation.

3. METHOD AND MATERIALS

Method

A first study was performed in October – December 1997 before the opening of the new tunnel for road traffic. The tunnel was opened In January 27th 1998 and a first, minor, follow up study on sleep was performed 3 months later. During August – December 1998, the existing roads and green areas in the residential area will be renewed and the final follow up study is planned in 1999.

The area of investigation was divided into an exposure and a control area in which the houses were situated 25 – 67 m and 125 – 405 m respectively from the trafficked main road.

Evaluation of effects. The effects on the population were evaluated by a main questionnaire that was delivered by a project assistant at the door/mailbox together with an introductory letter to one, or two, persons in each household between 18 and 75 years of age. The questionnaire was similar to those previously used [2] contained questions about the dwelling, annoyance to different sources in the neighbourhood, sleep and sleep disturbances and health and psycho-social symptoms and wellbeing. A second questionnaire on sleep was given to those who had answered the main questionnaire to be answered during 3 consecutive days. A smaller sample also took part in a sleep study including questionnaires and registration of body movements by actigram during 3 nights. The actimeters (type AMI – Mini-Motionlogger Actigraf) have been used in studies of effects of aircraft noise [4].

Assessment of noise exposure. A noise level meter, Larson & Davis type LD 820, which was operated as a remote station via a wireless transmission system was used. Measurements were done 3-4 days continuously in 5 different positions in the garden. A mean for each position was calculated for: LAeq, L01, L90, LAmax, Noise events >70 dBA for 3 periods; 24 hours, daytime (06-22) and night time (22-06). Traffic statistics was obtained from the local Traffic Office.

Materials

The total number of respondents was 142 persons and the response rate was 62 % for the main questionnaire. Of these 116 persons (82%) answered the 3-day sleep questionnaire. 26 persons took part in the sleep-actimeter study in 1997 and 24 persons in the first follow up study (2 persons had moved from the area). These 26 persons were chosen to achieve a similar distribution according to age and sex in each area.

4. RESULTS

Noise exposure

The total number of vehicles in the 1997 study was 24 600 per 24 hours including 4600 heavy vehicles and 1 375 during night including 125 heavy vehicles (h.v.). After the opening of the tunnel in 1998 the number of vehicles decreased to 4 600 per day (740 h.v.) and to 550 per night (80 h.v.). The measured outdoor noise levels in LAeq 24h were reduced from 64 to 49 dB in the noise area. LAeq levels in the control area were not affected, LAeq 47 dB.

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Effects on the population

The population sample. Preliminary results of the main 1997-study [involving 50 persons in the noise area and 92 persons in the control area] revealed no significant differences between the noise and the control area in socio-demographic aspects, time of residence etc. However 55% of the respondents in the noise area had renovated the facade of their house versus 34 % in the control area. Perceived noise sensitivity was somewhat (sign.) higher in the noise area.

Annoyance and activity disturbances. Noise from road traffic caused annoyance reactions (rather + very annoyed) among 96 % of the respondents in the noise area as opposed to 13 % in the control area. Noise annoyance as measured by a 0-10 point scale was 8.9 versus 2.3. Traffic noise was reported to cause difficulties to fall asleep and awakenings by about 25 % in the noise area versus 2-3 % in the control area. The percentage of people who only "sometimes or seldom/never" kept their bedroom windows open when sleeping was 90 % in the noise area versus 59 % in the control area.

Psychosocial wellbeing *[based on the sum of 7 symptoms scored 1-4, according to their occurrence; daily, weekly, monthly o yearly]* was significantly lower in the noise area ($p=0.007$ X^2-test). The three symptoms "low social orientation", "nervous stomach", and "depressed" were the main reasons for this, whereas "headaches", "very tired" and "irritated or anxious/nervous" did not differ between the areas.

Sleep quality parameters (time to fall asleep, sleep quality [scale 1-10 and 1-5], alertness in the morning [scale 1-10 and 1-5] were significantly lower in the noise area. Number of reported awakenings did not differ between the areas.

The results based on the study on 116 persons [40 in noise and 76 in control area], who answered a more detailed sleep questionnaire during 3 consecutive nights, showed significantly lower alertness during day ($p=0.02$) and morning [$p=0.008$] in the noise area. The respondents in the noise area also needed a longer time to fall asleep (22 versus 15 minutes, $p=0.003$), they perceived they had moved more in the bed ($p=0.003$) and slept more badly ($p=0.02$). The number of reported awakenings per night was *not* more frequent in the noise area; 1.7 versus 1.6 per night. Of those who woke up, however, 19 % in the noise area versus 1 % in the control area reported awakenings due to noise.

Body motility. The preliminary analyses of the results from the study on body motility (actimeters) and perceived sleep quality among 24 persons before and after road traffic noise reduction are summarised below. The values represent average values for three nights per person before and after noise reduction.

The table shows few significant results. In spite of a reduction in number of vehicles from 1375 to 550 per night and in LAeq levels with 13 dB during night, no significant effects on the various sleep parameters were found in the noise area. (A tendency ($p=0.06$) was seen, however, for a better perceived sleep quality). There was a significant difference between noise and control areas before traffic reduction for mean activity per minute and wake minutes. The results are, however, unclear since these parameters showed higher values in the control area in the after-study.

Table 1. Mean values for sleep parameters based on actimetry and questionnaires.

	Noise site (n=11x3x2)		Control site (n=13x3x2)		Noise/ control*	Noise/ control**
Actimeter parameters:	*Before*	*After*	*Before*	*After*	*Before*	*After*
Sleep latency	8.9	3.9	4.7	5.2	0.09	>0.10
Mean activity per minute	3.3	3.0	2.7	3.4	0.04	>0.10
Wake minutes	55.3	45.8	38.3	57.2	0.03	>0.10
Sleep minutes	407	397	382	342	0.06	>0.10
% sleep	88.3	89.9	91	86	0.08	>0.10
Perceived sleep quality:						
Minutes for falling asleep	24	19	19	12	>0.10	>0.10
Awakenings	2.1	1.2	2.1	1.6	>0.10	>0.10
Sleep quality (1-10)	6.2	7.3	6.2	6.8	>0.10	>0.10
Moved in bed (1-10)	5.2	5.1	5.1	5.1	>0.10	>0.10
Alertness morning (1-10)	5.3	5.8	6.6	6.5	0.01	>0.10

** t-test, one-tailed, ** t-test, two-tailed*

5. COMMENTS AND CONCLUSIONS

No final conclusions can be drawn from the investigation before the final study is carried out in 1999, about 1 year after the reduction in traffic. The noise exposure situation will be analysed in detail and the relation between noise levels and effects will be analysed. E.g. indoor noise immission in the bedrooms are of specific importance since the windows most often was facing the garden and not the main road in the noise area.

The results obtained in this study on sleep quality and psychosocial symptoms are in accordance with the findings in the previous study 1986 and 1987 [2,3] in the same area. Among the studied sleep parameters, sleep quality and time for falling asleep and alertness during morning and day seems to be more indicative of noise-induced sleep disturbances from road traffic than reported awakenings.

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TRAFFIC NOISE

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