

Journal of Clinical and Basic Cardiology

An Independent International Scientific Journal



Journal of Clinical and Basic Cardiology 1999; 2 (1), 64-68

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Work noise as a risk factor in myocardial infarction

H. Ising, W. Babisch, T. Günther¹

Noise has the potential to cause stress reactions. Acute increases of catecholamines or cortisol were observed under noise exposure with maximal levels ≥ 90 dB (A) or ≥ 120 dB (A) respectively. However, if the noise disturbed activities such as conversation, concentration, recreation, sleep, acute increases of catecholamines and/or cortisol were observed even at environmental noise levels ≥ 50 dB (A). In sleeping persons, traffic noise with such levels caused significant increases of catecholamines and/or cortisol, which became chronic if the noise exposure was persistently repeated. An interaction process between endocrine reactions and intracellular Ca/Mg shifts was detected, which leads to pathological alterations in the myocardium and the vascular walls. These findings led to the hypothesis that chronic noise-induced stress increases the risk of myocardial infarction (MI).

The hypothesis was tested in a case-control study with 395 MI patients (31–65 years) and 2148 controls. The relative risk of MI – adjusted for control variables (smoking, age, social status etc.) – was found to increase significantly and steadily with the loudness of work noise. Subjectively scaled work noise appeared to be the second greatest external risk factor in MI after smoking.

Noise-induced chronic stress seems to be an important risk factor in myocardial infarction. Since in the reported study there was possibly bias due to overreporting of subjective noise exposure, additional studies on the relationship between cardiovascular diseases and work-related stressors including subjective and objective noise assessment are needed to quantify the risk of MI due to work noise. *J Clin Basic Cardiol 1999; 2: 64–8.*

Key words: Noise stress, catecholamines, cortisol, calcium, magnesium, myocardial infarction

Noise has the potential to cause stress reactions ie, increase of catecholamines and/or cortisol. An interaction process between endocrine reactions and intracellular Ca/Mg shifts was detected which leads to pathological alterations in the myocardium and the vascular walls. The hypothesis that chronic noise-induced stress increases the risk of myocardial infarction was tested in a case-control study. The relative risk of MI – adjusted for control variables (smoking, age, social status etc.) – was found to increase significantly and steadily with the loudness of work noise. Work noise appeared to be the second greatest external risk factor in MI after smoking.

Introduction

Noise can act as a non-specific stressor inducing stress reactions which are in line with the general stress model [1–3]. Acute exposure to maximal sound pressure levels above 90 dB (A) has the potential to cause inner ear hearing loss and to stimulate the sympathetic nervous system into increasing the release of adrenaline and noradrenaline. Noise levels above 120 dB (A) increase cortisol in humans [4]. The activation of the sympathetic and endocrine systems is followed by changes in physiological function and in metabolism of the organism, including total peripheral resistance, cardiac output, blood lipids and rheological factors.

For moderate noise with maximal levels above 50 dB (A), noise has the potential to disturb activities such as communication, relaxation and sleep. Such noise disturbances are usually accompanied by endocrine reactions as mentioned above. Hence, in real life even moderate environmental noise exposure can increase the acute release of stress hormones (Fig. 1) [5–7].

Carefully controlled animal model studies were used to develop the hypothesis of noise as a cardiovascular risk factor. In order to be able to apply animal results to humans, acute noise-induced increases of stress hormones ie, catecholamines

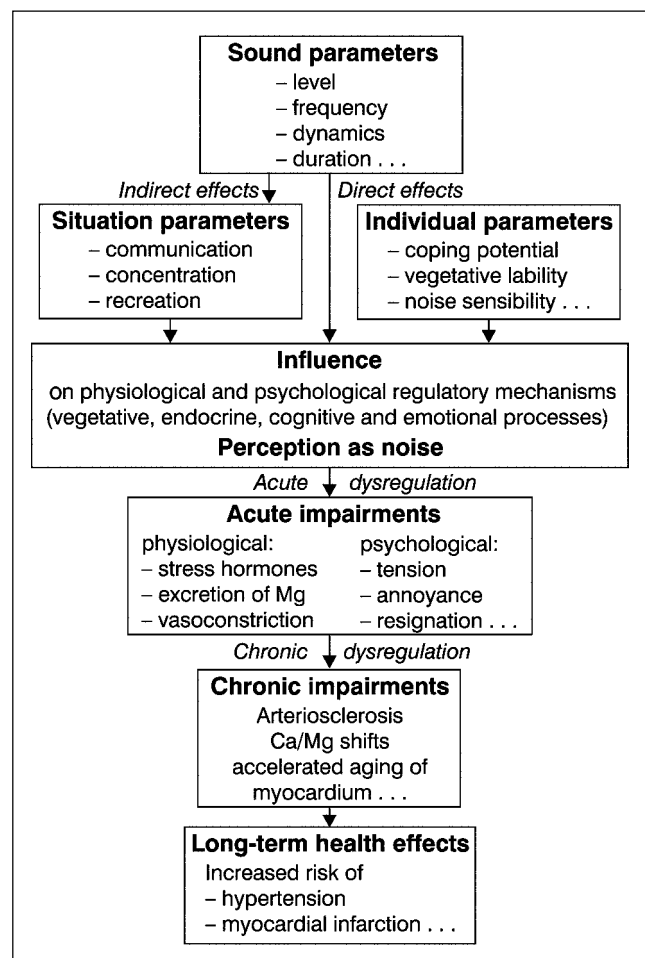


Figure 1. Model of noise perception and psychophysiological effects of noise, risk factors and cardiovascular diseases

Received February 27th, 1998; accepted May 18th, 1998.

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Table 1. Effects of noise exposure and Mg-deficiency in rats*

Treatment		Effect						Death rate
4 months Mg in diet	3 months Noise Leq/Lmax	Urine Noradrenaline [ng/g Cre]	Body weight [g]	Myocardium Hydroxyprol. [mg/g dry wt.]	Ca [mg/g d.w.]	Mg [mg/g d.w.]	Ca/Mg	
control	ambient	18 ± 4	402 ± 8	3.0 ± 0.1	3.0 ± 0.2	37.5 ± 0.8	0.08	0
suboptimal	ambient	23 ± 4	335 ± 23	3.0 ± 0.1	3.5 ± 0.5	38.0 ± 1.7	0.09	0
suboptimal	69/86 dB	37 ± 11	336 ± 10	3.0 ± 0.1	4.3 ± 0.2	37.9 ± 1.3	0.11	0
deficient	ambient	98 ± 17	206 ± 3	3.9 ± 0.1	6.2 ± 0.7	31.2 ± 1.4	0.20	38 %
deficient	69/86 dB	129 ± 19	156 ± 7	4.6 ± 0.1	6.7 ± 0.6	29.8 ± 1.8	0.23	62 %
deficient	75/86 dB	172 ± 26	145 ± 6	5.6 ± 0.9	8.0 ± 0.9	26.8 ± 0.8	0.30	75 %

* Noradrenaline excretion was measured during the 4th week of noise exposure; Death rate is related to the 4 months of treatment; all other parameters were determined at the end of the experiment (mean values ± S.E.).

and cortisol in humans and animals were compared and shown to be qualitatively similar [8].

In an animal study with persistently repeated noise exposure a chronic increase of noradrenaline was found [9]. In this experiment, 6 groups of rats were fed diets with normal, suboptimal, or deficient content of magnesium respectively. Magnesium deficiency was used as a model for a stressor which acts synergistically with noise. Three levels of noise exposure were used for 3 months: control condition and mean levels of 69 dB and 75 dB. Mg-deficient diet alone as compared with controls resulted in a marked chronic increase of noradrenaline excretion (444 %) (Tab. 1), which increased further with noise (69 dB: 32 %; 75 dB: 76 %). This shows that Mg-deficiency and noise are stressors which act synergistically. Using noradrenaline as an indicator of the total stress, the death rate, the collagen and Ca content of the myocard and the quotient of Ca/Mg increased with increasing stress. Similar Ca/Mg shifts were found in the vascular walls of chronically noise-stressed rats [10].

Further analysis of the experimental results led to an interaction model between chronic stress and intracellular electrolyte shifts [11–12] (Fig. 2). Chronic stress caused a loss of extracellular Ca [9]. A decrease of Mg was correlated to an increased physiological noise sensitivity, ie, to more severe noradrenaline releases in animals and humans under noise exposure [9, 11–14]. We found a positive feedback mechanism between stress – caused by noise and/or other stressors – and intracellular Ca/Mg shifts which may lead to a circulus vitiosus and therefore increase the cardiovascular risk.

The importance of Ca/Mg shifts was confirmed by post mortem studies of hearts from victims of ischaemic heart diseases (IHD, ICD 410–414¹). The tissue samples were taken from areas of the myocardium not affected by the infarction and the results were stable after controlling for several confounders [15]. The results in Table 2 show that in humans the ratio of Ca/Mg increases with age. A differentiation between ischaemic heart disease (IHD) death and non-IHD death revealed that the myocardium of IHD death looked older with respect to Ca/Mg increases. Since chronic noise stress increased the ratio of Ca/Mg and the collagen content of the rat myocardium (which can be interpreted as accelerated aging [16]), and increased Ca/Mg-ratios were found in the myocardium of IHD deaths, we conclude that chronic noise stress accelerates the aging of the heart also in humans.

Table 2. Age dependency of myocardial Ca and Mg in ischaemic heart disease [IHD] deaths and non IHD deaths (mean value ± SD, number in brackets)

Group		Age		
		< 45 years	45–64 years	> 64 years
Ca [μg/g]	Non IHD	43 ± 15 (175)	50 ± 14 (281)	57 ± 22 (155)
	IHD	48 ± 10 (48)	53 ± 17 (389)	58 ± 21 (188)
Mg [μg/g]	Non IHD	183 ± 28	173 ± 34	178 ± 30
	IHD	170 ± 29	157 ± 30	156 ± 27
Ca/Mg	Non IHD	0.24	0.29	0.32
	IHD	0.28	0.34	0.37

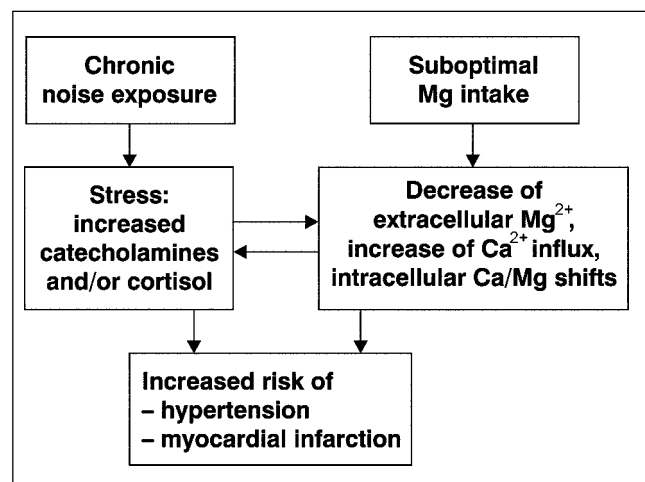


Figure 2. Interaction model of noise-induced endocrine reactions and electrolyte shifts resulting in increased cardiovascular risk

In the following studies it is shown that persistent traffic noise exposure leads to chronic increases of stress hormones. A cross-sectional study with about 200 females, who lived for several years in streets with low or high traffic noise, showed a significant increase of the noradrenaline excretion in subjects whose bedroom windows were facing a noisy street. Additionally, noise-disturbed persons had significantly higher noradrenaline levels than undisturbed persons [17].

In another study, 43 males and females living in either noisy or quiet (control group) streets for several years, were monitored for 2–3 nights (a total of 102 testperson nights). Persons who lived in noisy streets had significantly higher excretion

¹ICD 410: acute myocardial infarction, ICD 411–414: other acute and chronic ischaemic heart diseases

of noradrenaline and cortisol, but adrenaline was unchanged. In one third of all the nights, the cortisol excretion of the noise group exceeded the normal range compared to only 4 % in the control group [18].

Chronically increased noradrenaline is known to have detrimental effects upon the heart. One of the side effects of chronically increased cortisol is arteriosclerosis. Therefore, we formulated the hypothesis that chronic noise exposure increases the risk of myocardial infarction and tested it in an epidemiological study.

Methods

In order to study the relationship between work noise as a stressor and myocardial infarction, a population based case-control study was carried out. Men aged 31 to 65 years (normal working upper age limit) who had been treated for acute MI (ICD 410) in the major Berlin (West) hospitals were considered as "cases". They were interviewed by a physician and questioned about potential control variables (age, social class, education, employment status, shift work, smoking habits, body mass index, family status and residential area) as well as about work noise (see below) and their home address. From the 583 MI cases participating, 188 (32 %) were unemployed and therefore excluded from the analysis. This yielded a total of 395 employed men in the MI group. Less than 5 % of the cases refused to co-operate. However, because some hospitals were not included, 80–85 % of the source population ultimately co-operated.

For the control group, a random sample of the male German speaking population of Berlin (West) with a similar age distribution of the cases was drawn up by the local registration office and a questionnaire with identical questions as answered by the cases, was sent to them yielding a participation rate of 64 % (ie, 68 % of all men who received the questionnaire because 4 % were incorrectly addressed). 3228 men aged 31–65 years completed the questionnaire. 1080 (34 %) of them were unemployed so that for the analysis the control group consisted of 2148 employed men. The age distributions of cases and controls are shown in Table 3A and are found to be quite similar. Obvious hints at the aim of the study (in particular noise) were avoided, both in the questionnaires and in interviews with the patients.

Subjective work noise was quantified by the questionnaire. The instruction for the subjects was: "Of the following noise sources please select which best describes how loud it is at your workplace: 1) refrigerator, 2) typewriter, 3) electric lawnmower, 4) electric drill, 5) pneumatic drill."

Multiple logistic regression analyses were performed using the PC-Windows 6.0 version of the SPSS statistical soft-

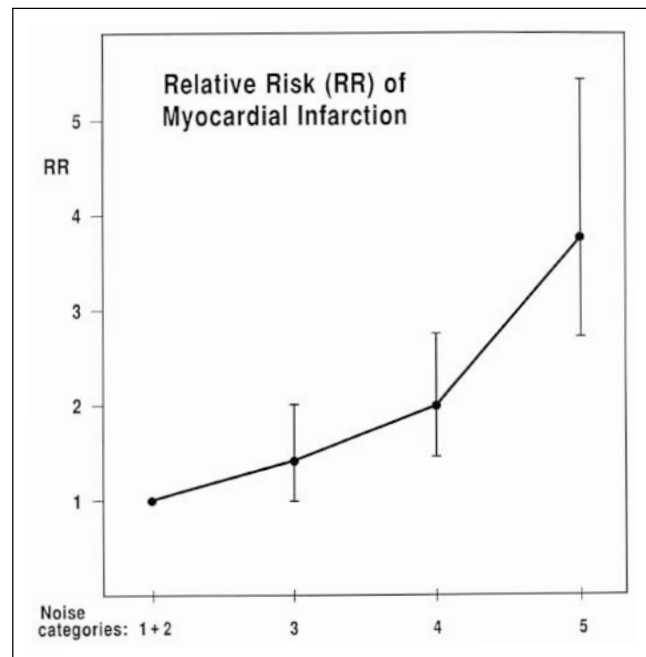


Figure 3. Relative risk (RR) of MI in men (and 95 % confidence intervals) by work noise category. Work noise was assessed by subjective loudness comparison with typical noise sources (1+2: refrigerator + typewriter, 3: electric lawn-mower, 4: electric drill, 5: road drill). Relative risk of MI (ICD 410) was adjusted for covariates (smoking, body mass index, age, social class, education, marital status, shift work, housing area) using logistic analysis.

ware package, and test-based confidence limits of relative risks were calculated.

It was possible to calculate population attributable risk percentage (PAR) since the control group was almost a representative sample of the male German working population of Berlin (West). PAR was calculated according to the formula $PAR \% = [(RR - 1) / RR] P_{case}$ with P_{case} as percentage of exposed cases. The work noise-related PAR was calculated as the sum of three terms for the noise categories 3, 4 and 5, each similar to this formula.

Because the response rate was only 64 %, we compared the social class distribution of our control group with official census data. The biggest difference was between the percentage of workers in our control group (29 %) and in the source population (37 %). This difference was approximately corrected in the PAR-calculation by multiplication with 29/37.

Results

The distributions of cases and controls in respect to subjective work noise categories are shown in Table 3B. Additionally the relative risks (RR) and the 95 % confidence intervals in relation to the sum of categories 1 + 2 are given. In contrast to Figure 3 these relative risks are not model adjusted for control variables.

In Table 4, the distribution of cases and controls in relation to control variables and the sum of the work noise categories 3+4+5 are compared (for significance of distribution differences between cases and controls see Tab. 5). The sum of the work noise categories 1+2 was used as a reference and the relative risks of MI adjusted with respect to the control variables were determined, using multiple logistic analysis. In Figure 3, the model adjusted relative risks of MI together with the 95 % confidence intervals are plotted against the work noise

Table 3. Distributions of MI cases and controls

3A Age distribution			3B Work noise distribution			
Age [years]	MI	Controls	Noise categ.	MI	Controls	RR (CI)
31–40	42 (10.6 %)	161 (7.5 %)	1+2	149 (37.7 %)	1221 (56.8 %)	1
41–50	122 (30.0 %)	619 (29.4 %)	3	71 (18.0 %)	397 (18.5 %)	1.47 (1.07–2.01)
51–55	115 (29.1 %)	752 (35.0 %)	4	88 (22.3 %)	358 (16.7 %)	2.01 (1.49–2.72)
56–65	116 (29.4 %)	616 (28.5 %)	5	87 (22.0 %)	172 (8.0 %)	4.14 (3.01–5.71)
Total	395	2148	CI: 95 % confidence interval			

Table 4. Distribution of cases and controls in control variables and work noises

	MI	Controls
Social class [low]	35.2 %	27.3 %
Education [college degree]	14.7 %	28.0 %
Marital status [divorced + widowed]	16.2 %	8.4 %
Residential area [outskirts]	38.7 %	44.9 %
Shift work [yes]	14.2 %	14.4 %
Body mass index [$> 27.7 \text{ kg/m}^2$]	30.7 %	23.3 %
Current smoking [yes]	68.1 %	38.2 %
Work noise [categories 3+4+5]	62.3 %	43.2 %

Table 5. Results of full model analysis

Variable	RR
Social class [worker/others]	0.71*
Education [college degree/others]	0.60
Marital status [divorced + widowed/others]	1.86
Residential area [outskirts/inner city]	0.85
Shift work [yes/no]	0.72
Body mass index [kg/m^2]	1.05
Current smoking [yes/no]	3.10
Work noise [categories (3+4+5)/(1+2)]	2.47

* Most of the other MI risk factors (smoking, high BMI etc.) are more prevalent in workers

categories. The relative risk of MI increased significantly and steadily with work noise categories.

The results of the full model analysis are listed in Table 5. Stratification of the results into three age groups shows that in the youngest age group the relative risk of MI increased more conspicuously with subjective work noise than in older age groups (Tab. 6).

The population attributable risk percentage (PAR) was $\text{PAR} = 0.325$. When the above mentioned correction for social class bias was applied, the result was $\text{PAR} = 0.255$, suggesting that 26 % of all MI in the source population may be attributable to subjective work noise. Consequently, subjective work noise appeared to be the second greatest risk factor for MI after smoking ($\text{PAR} = 0.46$).

Discussion

It was most surprising to find that subjective work noise appeared to be the second important risk factor of MI after smoking when external factors were considered. Internal risk factors ie, blood lipids, blood viscosity and blood pressure are not taken into account in this comparison. However, since the subjective noise rating was assessed after the MI or "retrospectively", it may have been influenced by the experience of MI, thereby causing a systematic over-estimation of noise by the MI patients. Such an over-estimation of subjective noise could be due to the well-known fact that noise in combination with other stressful conditions is rated higher than noise alone. Probably this subjective noise aggravation is more pronounced in the MI group than in controls. However, it is unlikely that this effect is large because in a case-control study comparing noise level measurements with subjective noise ratings it was shown that only 8 % of the controls and 16 % of the MI patients over-estimated the subjective noise load [19].

The 32 % of non-responders in the controls entails the possibility of selection bias. The most probable effect seems to be a distortion in relation to social class. Subsequently we checked this and found that selection bias resulted in 29 %

Table 6. Relative risks of MI (95 % Confidence intervals) in dependency of age and of work noise categories (categories 1 + 2 = reference)

Noise category:	3		4		5	
Age group	RR	CI	RR	CI	RR	CI
31–45 years	2.1	(1.07/4.30)	4.0	(1.90/8.52)	5.6	(2.37/13.07)
46–55 years	1.4	(0.85/2.15)	1.7	(1.08/2.67)	3.9	(1.86/5.13)
56–65 years	1.0	(0.56/1.99)	1.9	(1.03/3.33)	4.2	(2.25/7.95)

workers in the control group whereas the source population contained 37 % workers. Therefore selection bias may have influenced the results but only to a limited extent.

The duration of work noise exposure was not assessed. However, exposure misclassification due to missing information on the length of exposure could dilute the true noise effect if this occurred at random. Bias was more likely to have a conservative impact since the migration rate would probably be higher due to noise at the workplace.

Moreover, the difference between subjective and objective noise rating seems to be of major importance. This difference is explained schematically in Figure 1. In general, sound parameters, which can be measured objectively, determine subjective noise perception to within 30 % to 40 %. Situative and personal influences together determine subjective noise perception to the same degree [20]. Therefore, the correlation of noise effects with subjective noise parameters is expected to be closer than with objective noise parameters. For this reason, it can also be concluded that in the presented paper the main effects of noise seem to have acted indirectly by disturbing activities and thus lead to psychophysiological stress effects. A direct effect of noise as an external physical risk factor is possible at levels above 90 dB (A) but seems to be of lesser practical importance.

However, if our results do reflect a true noise effect, then there should also be a correlation between objective work noise parameters, ie, noise levels, and cardiovascular risk. This is possible – although such a correlation was not demonstrated until now – since nearly all studies on this relationship have shortcomings arising from two problems. The first problem is the suitability of the control group. Since the ideal "no noise" does not exist, objective noise studies must compare groups with clearly increasing noise levels using the group with the lowest exposure as a reference. Most studies, however, compare two groups with noise levels above and below 85 dB (A). The second problem occurs at levels above 85 dB (A). In western industrialized countries ear protectors must be provided above 85 dB (A). If the partial use of ear protectors is not taken into account, noise effects will be underestimated. This is because people who suffer from noise stress are more likely to use ear protectors than those who are less sensitive to noise. People who are working in a noise level below 85 dB (A) and are therefore not provided with ear protectors, will have a higher noise exposure than workers with 90–100 dB (A) external noise, who use ear protectors because their noise level is reduced by 20–30 dB by the ear protectors. This seems to be the reason why one otherwise well designed case-control study [21] failed to show any cardiovascular risk of noise.

Additional arguments for the hypothesis of noise as an external risk factor of MI stems from studies on noise-induced acute [22] and chronic [10, 23–25] increases of some of the accepted internal risk factors of MI, ie, increased blood pressure and total cholesterol. However, the problem of ear protectors has to be considered in these studies as well.

A dose-dependent increase of the hypertension rate was found among about 1000 female textile workers in China [26] who worked for several years without ear protection under a noise exposure of up to 104 dB (A). Since no ear protection was used in this cross-sectional study, the noise exposure was unadulterated, whereas in most of the other studies on blood pressure and work noise, an undefined use of ear protection caused an error in noise exposure of 20–30 dB. Since subjective noise scaling avoids the problem of ear protectors the existing studies of this type will be mentioned shortly.

In a prospective cohort study [27] with more than 2000 test persons (duration: 17 years) the relation of subjective noise load and hypertension was investigated. Persons who stated that they had worked in noisy workplaces for several years had a significantly higher risk of hypertension. The relative risk was 1.74 and the 95 % confidence interval 1.002–3.015.

In another prospective cohort study [27] with 1002 persons (about half males and half females, duration: 11 years) the relation of work related stressors (ie, shift work, time pressure, heat, cold, air pollution, noise etc.) and disease was investigated. After adequate adjustment for confounders including smoking and body mass index, a noise related relative MI risk of 2.78 (95 %-confidence interval: 1.01–7.63) was found and a population attributable risk percentage of PAR = 0.15. This value is lower than in our study, which may indicate an effect of overestimation. Additionally, it can be explained by the low percentage of females working under loud work noise exposure and the fact that we studied only male subjects. However, in this cohort study the work-noise-related relative risk of MI was approximately double in women as compared with men.

In these two prospective studies, the noise-related relative risks of hypertension and MI are rather small. However, since a high percentage of the population is exposed to work-noise, the social medical relevance of these results is high. It has to be stressed that work-noise exposure was not defined as exposure to mean noise levels above 85 dB (A) but as an answer to the question: "Are you exposed to noise at work?" The result was that more than 10 % of the population is subjectively exposed to noise at work.

Conclusion

In agreement with our hypothesis, which is based on detailed empirical evidence, subjective work noise appears to be a major risk factor in MI. However, the presented study and the few studies from literature which are not seriously impaired for methodological reasons, are not sufficient to quantify the noise-related risk of MI. Therefore an interdisciplinary study on the relationship between cardiovascular diseases and work-related stressors, including subjective and objective noise assessment, was started to check this result and to quantify the noise related risk of MI [28].

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