

Cardiovascular diseases and the work environment

A critical review of the epidemiologic literature on nonchemical factors

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KRISTENSEN TS. Cardiovascular diseases and the work environment: a critical review of the epidemiologic literature on nonchemical factors. *Scand J Work Environ Health* 1989;15:165–179. This is the first of two articles reviewing the epidemiologic research on cardiovascular diseases (CVD) and the work environment. It deals with a number of nonchemical factors, ie, physical inactivity at work, stressors at work, shift work, noise, cold, heat, and electromagnetic fields and waves. First the methodological quality of each of the empirical studies is assessed on the basis of epidemiologic criteria. Then the research literature on each of the aforementioned factors of the work environment is evaluated. It is concluded that the hypothesis of a causal relationship between physical inactivity at work and risk of CVD is substantially supported by the literature. As regards work stressors and shift work, several good studies have been published during the last 10 years strongly suggesting a causal relationship. Other studies have shown a relationship between noise and elevated blood pressure, but the quality of this literature is low. Heat and cold appear to have an acute effect on the incidence of CVD, but the possible chronic effect has seldom been investigated. Concerning electromagnetic fields and waves, it is concluded that more research is needed. The study of CVD and work ought to play a bigger role in research in the fields of occupational medicine and cardiovascular epidemiology in the future.

Key terms: cold, electromagnetic fields, electromagnetic waves, heat, hypertension, ischemic heart disease, noise, occupation, physical inactivity, shift work, stressors at work.

Over the past 10 years, many reviews of the literature about cardiovascular diseases (CVD) and the work environment have been published (1–12). These reviews have all been of the general narrative type, as explicit criteria for evaluating the literature have not been employed. The number of studies reported varies considerably, and no account is given of the method of literature search used or the criteria for including the individual studies.

The objectives of this and an ensuing article are (i) to record and integrate the epidemiologic literature on CVD and the work environment, (ii) to evaluate the research with the objective of elucidating possible causalities between work environment factors and CVD, (iii) if possible to point out areas where enough is known to start employing the research results for the purpose of prevention, (iv) to point out defects and deficiencies in existing research with the objective of strengthening and improving future research efforts.

The topic is delimited such that all relevant factors of the work environment have been included, while individual habits or characteristics have not, even though they are conceivably influenced by work conditions. In practice, therefore, the two reviews include passive smoking, but not active smoking; shift work, but not

sleeping disorders or coffee drinking; and monotonous high-paced work, but not type A behavior. To the extent that studies dealing with subjects beyond the realm of occupational medicine have been included, it has been done to elucidate the significance of occupational environmental factors.

The purpose of focusing on the influence of the occupational environment on CVD is not to question the research concerning the importance of the individual risk factors. This very extensive research has obtained clear results concerning conditions like smoking, cholesterol, blood pressure, hereditary characteristics, diet, obesity, and others, which are further elaborated in the research on the work environment and CVD. The objective is to expand and supplement the knowledge about the causes of CVD, which, in turn, increases the possibilities for preventing these diseases. This article deals with the nonchemical factors that might influence CVD, ie, physical inactivity during work, monotonous high-paced work and other psychosocial factors, shift work, noise, climate, and electromagnetic waves and fields. The chemical factors will be dealt with in a future article.

Materials and methods

This review of the literature has been limited to epidemiologic studies and reviews published in English, German, or one of the Scandinavian languages (or with summaries in one of these languages). In the areas of

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physical inactivity and noise the literature proved to be very extensive. For these subjects priority was given to the inclusion of the more recent studies, which have been supplemented with a number of classic studies and some very thorough reviews. In the other areas, the intention was to include all the relevant studies published.

The literature searches were carried out with the literature data bases Medlars, Toxline, Excerpta Medica, CIS abstracts, and Social Scisearch. Furthermore, a manual search was undertaken of the leading epidemiologic journals and of *Current Contents*, as well as via personal communication with experts in the relevant fields. A total of 2 000 references were checked, of which 700 were found relevant and, accordingly, read and evaluated.

The primary objective was to identify the risk factors in the work environment. By a risk factor I mean a factor which is causally related to CVD, ie, that the presence of this factor increases the risk for one or more types of CVD. (This phenomenon might happen via one of the (well-)established risk factors, such as elevated blood pressure, serum cholesterol, or "ischemic" electrocardiographic changes).

Each individual empirical investigation has been carefully evaluated as to the following five central methodological points: (i) the *time dimension*: in this respect attention was given to determining whether the design of the study made it possible to evaluate the temporal chronology between exposure and disease; (ii) *confounding*: individual confounders such as age, sex, smoking, diet, obesity, and social background were particularly recorded; (iii) *selection*: specific thought was given to one of the great methodological problems in occupational medicine, ie, the healthy worker effect; (iv) *measuring of exposures and disease*: retrospective studies especially often involve problems concerning the valid registration of earlier exposures; and (v) *adequate design and statistical analysis*: on this point, an evaluation was made of whether the design and analysis were optimal for testing the research hypotheses.

Each single study was given a score between "x" and "xxxxx" for methodological quality according to the quality of the study with regard to the five points mentioned. I used this procedure because soundly executed studies ought to carry more weight in the total evaluation of a given research field.

For each possible risk factor, the aggregate research was evaluated on the basis of the following three criteria: (i) the methodological quality of each individual study, (ii) the number of studies, and (iii) the consistency of the study results. The basis for this procedure was that a hypothesis about causality is not "proved" once and for all, but is confirmed or invalidated through steadily cumulating research activity. Ultimately, it will always be a matter of judgment as to whether sufficient evidence exists for causality to be considered established beyond all reasonable doubt.

As should now be evident, this literature review places itself somewhere between the general narrative literature review on one hand and meta-analysis on the other. Regarding the methodological problems involved with literature reviews and especially meta-analysis, see the two thorough works by Greenland (13) and Thacker (14). The decision not to carry out an actual meta-analysis was made because the studies in this field are too heterogeneous with regard to concepts, methods, and quality.

There are grounds to specify that the weight in this and the future review has been put on the epidemiologic and methodological qualities of the recorded studies. No attempt has been made to explain or discuss the physiological processes which might be conceived to explain the relationships found.

Discussion is sometimes heard — and with good reason — as to whether conclusions in literature reviews are too highly colored by the personal attitudes of the author. This possibility can best be avoided if one seeks to make the criteria employed as explicit as possible — which has been done in the present review. Furthermore, it is useful to realize that the readers of literature reviews have the possibility of monitoring all information and conclusions because all the basic material is publicly accessible. This possibility of monitoring the data material is normally not present when it comes to normal empirical studies.

The literature which has been collected in connection with the project has been entered in a personal computer data base by the use of the PELIKAN system (Personligt Litteratur Kartotekssystem), developed by MT Damsgaard for this project. The system is a personal, dynamic system which makes it possible to search under all combinations of topics, authors, years, etc.

Results

Physical inactivity at work

Physical inactivity at work has become increasingly widespread due to the technological and structural developments over the past hundred years. In the epidemiologic literature, the effect of physical inactivity on the incidence of CVD has mainly been investigated in three ways, namely, by relating CVD to (i) physical activity at work, (ii) physical activity during leisure time, (iii) physical fitness. The question of physical inactivity at work can best be elucidated by a review of the research in these three fields collectively.

The two great pioneers within this field of research are Morris and Paffenbarger. During the 1950s and 1960s, Morris and his colleagues published a number of studies of bus drivers, conductors, train personnel, etc (15—21). All of these studies evidenced a clear trend for a higher incidence of ischemic heart disease (IHD) among persons with physically inactive jobs. In the 1970s, Morris and his co-workers began investigating

physical inactivity during leisure time and IHD among civil servants (22–26). A clear relation was also found between physical inactivity and IHD. Paffenbarger et al studied the same complex of problems in a cohort of longshoremen (27–32) and in two studies of former students (33–39).

All of these studies are characterized by a high methodological quality (from “xxx” to “xxxxx”), and all of them show a clear trend towards a higher incidence of IHD and other CVD among the physically inactive. Moreover, IHD in the physically active was shown to take on less severe forms. Both research teams exhaustively treated the problems of selection, as well as confounding, and both concluded that the relation between physical inactivity and IHD is causal. Moreover, it was found that physical activity has to have a certain vigorousness in order to reduce the risk for IHD.

Morris’ and Paffenbarger’s now classic studies have been followed by a large number of similar studies which would require too much space to report in this communication. Instead, the reader is referred to the numerous reviews which have been published since 1982 (40–50). The authors of these reviews do not agree on the requisites which must be fulfilled to allow an assertion of conclusive evidence, but a high degree of consensus exists: (i) about two-thirds of the published studies show a positive correlation between physical inactivity and risk for CVD (especially IHD); (ii) no studies show a negative correlation; (iii) the lack of a relationship in some of the studies is probably connected with methodological problems related to the process of selection or to the measurement of physical activity; (iv) the relation between physical activity and CVD seems partly to come about via the traditional risk factors, eg, serum cholesterol, blood pressure, and obesity.

Of the aforementioned reviews, there is special reason to highlight the very systematic one by Powell et al (47) of 43 studies. Powell et al chose as a basis Hill’s criteria for causality (51) and classified the 43 studies according to their methodological qualities in approximately the same way as in the present study. On this basis, it could be demonstrated that the relation between physical inactivity and CVD was clearer with better methodological quality. While the median relative risk for IHD was 1.9 for the inactive when all studies were taken into consideration, it was 2.0 in the better studies, and 2.4 in the best. This pattern clearly supports the hypothesis of a causal relationship.

This positive conclusion is supported by well-executed studies which have been published subsequent to the review by Powell et al (52–54). Moreover, also worth mentioning is that well-known prospective population studies such as the Framingham Study, the Multiple Risk Factor Intervention Trial (MRFIT), and the Western Collaborative Group Study support the hypothesis of a causal relation between physical inactivity and IHD (55–59).

In summary, it must be concluded that an explanation of the epidemiologic research in this field is very difficult without the hypothesis of a causal relation between physical inactivity and IHD (and probably other forms of CVD) being accepted. The number of studies is very large, and the methodological quality very high, most studies having been given a rating of “xxx” to “xxxxx” for methodological quality. Furthermore, as also found by Powell et al (47), there is a relation between epidemiologic quality and corroboration of the hypothesis about physical inactivity, as the hypothesis is the most unequivocally confirmed by the best studies. On the basis of the review by Powell et al, the relative risk for CVD in the physically inactive must be estimated to be at least 2.0 when this group is compared to the physically active.

Stressors related to the organization of work

In the general reviews that have been mentioned in the introduction to this article, stressors in work (often called psychosocial factors) play a very little role. Most authors are skeptical towards the research results in this area; others ignore them completely. Some mention that it is very difficult to define and measure these factors in a sufficiently valid manner. An exception is found in the two reviews by Theorell et al (11, 12), which specifically deal with stressors at work.

However, there are a number of very thorough reviews that deal with the research on work, stress, and CVD. One of the first was Rosenman & Friedman (60), who, among other things, mentioned their own classic study of the relation between stress at work and elevated serum cholesterol (61). The relation between stress and serum cholesterol has not been the object of any particular interest among epidemiologists since then, but is now perhaps about to be taken up again (62). The most important of the earlier reviews are House, (63) and Cooper & Marshall (64). These reviews include a broad and imprecise account of psychosocial factors such as work load, role conflicts, low job satisfaction, alienation, interpersonnel conflicts, high job demands, life events, etc. Generally, the epidemiologic level of the studies was rather low, and there was a lack of a comprehensive conceptual instrument. The research dealing with life events, which previously played a rather large part, has moreover been thoroughly referred to and commented on by Theorell in his more recent reviews of this field (12, 65, 66).

In 1980, Cooper (67) published a small article about work stress in white- and blue-collar jobs in which he demonstrated that stress and stress-related disorders are primarily a low-status problem, as blue-collar jobs are characterized by monotony, lack of influence, employment insecurity, etc. Almost simultaneously, Karasek (68) published the first article about the job strain model which was to influence the research of the 1980s in this field. The studies from 1982 on demonstrate this influence clearly, as Karasek’s model played a large part in all of them (11, 69–73). In the following

review, the newer research results in this area are stressed. For the older research, the reader is referred to the aforementioned articles.

The field is characterized by a lack of consensus concerning concepts and definitions. In the following text, the word "stress" is used for a condition of the individual which is characterized by a combination of arousal and aversion. Stress has a psychological, a physiological, and a behavioral aspect and can, accordingly, be assessed in a number of essentially different ways. In connection with CVD, one is especially interested in chronic stress. "Stressors" refers to conditions in the environment which can cause stress. Common characteristics for stressors are one or more of the following: (i) lack of control, (ii) lack of meaning, (iii) lack of predictability, (iv) over- or understimulation, and (v) conflict. Man is not passive to stressors, but employs a number of coping strategies which can be mental and/or behavioral. Stress can be conceived to increase the risk for CVD in two ways, ie, directly, via psychophysiological processes, and indirectly, via stress-related behavior (smoking, diet, type A behavior, etc). In this article, I am interested in elucidating the direct causal relation. Finally it should be mentioned that the word "psychosocial" is used as a somewhat imprecise designation for the whole area of topics.

After this brief clarification of concepts, it is time to turn to the empirical studies in the field. First comes those which have tested Karasek's model. According to the model, it is a combination of high job demands and a lack of influence on one's own work, "strain," which increases the probability of chronic stress and, thereby, CVD. High job demands are not in themselves detrimental to health if they are combined with influence on one's own work (thereby providing the possibility of employing adequate coping strategies). Thus, these possibilities for coping separate the manager from the assembly-line worker.

Eight independent studies were found in which Karasek's model was tested, and all eight support the hypothesis (74—85). Four of the studies employed a completely or partially prospective design, while three are case-referent studies, and one is cross-sectional. The number of subjects varied greatly from a cohort of about one million persons (80, 81) to 22 cases and 66 reference persons (74, 75). In addition, concerning the measurement of job strain and CVD, varying methods were employed. The studies have been carried out in the United States (US), Sweden, and Finland. The epidemiologic quality is generally high, as five studies have "xxx," two have "xxxx," and one has "xxxxx" as their ratings.

The high methodological quality combined with very different study designs and methods should a priori give good possibilities for falsification of the hypothesis. The relative risk for CVD among persons in the strain group varies in the different studies from 1.3 to about 4, and a slight tendency exists for the highest

risk ratios to be found in the best studies. The overall conclusion therefore must be that Karasek's hypothesis has been strongly confirmed by the studies mentioned.

Of the investigations which do not employ Karasek's job strain model, four should be mentioned that were published during 1977—1983, and all of which have been rated "xxx" or "xxxx" for methodological quality. They are the study by Zorn et al of sea pilots (86), the study by Theorell et al of construction workers (87, 88), the study by Hartvig & Midttun of bus and truck drivers (89), and the one by Kittel et al of bank employees (90, 91). The work-related stressors described in these investigations vary. The study by Zorn et al deals with great responsibility, as well as with long and irregular work hours. Theorell et al (88) employed the concept work load and described the job of construction workers as a "strenuous repetitive physical effort with a rushed tempo [p 153]." Hartvig & Midttun (89) wrote that the job of drivers is characterized by "tempo/time-limit/urgency in job [p 356]." Kittel et al noted that the job of bank personnel was characterized by insecurity, frustration, and competition, while those employed in a semi-public savings bank (reference group) had quiet, secure jobs.

Regardless of these and other differences, the four studies reached the same conclusion, ie, a connection between stressors at work and an increased incidence of CVD. The study by Kittel et al of bank and semi-public savings bank employees is a particularly convincing study. It has not been possible in the epidemiologic literature to find investigations of an acceptable quality (at least "xxx") which did not show a relation between stressors at work and CVD.

Occasionally, studies are published which seemingly invalidate the hypothesis about a causal relationship between stressors at work and CVD. For example, among the car assembly workers in the study by Baxter et al (92), a very low incidence of IHD was found. A closer inspection shows that the study dealt with an open cohort with a very considerable turnover of personnel and without control of primary or secondary selection, that persons who left the cohort were not followed, that there was no control for any confounders other than age and sex, and that the reference population (the total population) had been an unfortunate choice. In connection with this review, it has been decided not to include the rather large number of studies of this category, as they must be regarded as uninformative.

In a number of studies, CVD is not used, but blood pressure or the prevalence of hypertension are employed as end points. As the risk for CVD is generally assumed to increase steadily with increasing blood pressure, studies on blood pressure are included. Eight studies on stressors at work and blood pressure/hypertension have been found which have a sufficiently high epidemiologic level to be informative (74, 89—91, 93—98). Of these, only Kornhuber & Lissner (98) did

not find a relation between stressors at work (piece work and high responsibility) and blood pressure.

In the study by Aro et al (93, 94), the relation between work stressors and blood pressure was not particularly clear. In this investigation self-reported stressors and stress symptoms were employed. In a recent study of bus drivers, Winkelby et al (99) found a negative correlation between self-reported stressors and the drivers' blood pressure. Winkelby et al discussed whether this finding can be attributed to the fact that hypertensive persons have a tendency to underestimate or deny stressors at work and found support for this hypothesis in other studies. On a number of occasions, Theorell has also described this tendency for denial of emotions and/or problems in hypertensives and/or persons with increased risk of CVD (70, 73, 100). This hypothesis is important to take into consideration in future research.

In summary, it can be concluded that, especially during the 1980s, many high-quality studies have been conducted which show a relation between stressors at work and the risk of CVD. Particularly Karasek's work strain model has been confirmed in a convincing way, and it is interesting to note that this dimension has now been included in a couple of the classic cardiovascular population studies: the Whitehall and Framingham studies (79, 85, 101).

The hypothesis about a relation between work stressors, chronic stress, and CVD not only has empirical support, but it has also been confirmed by animal experiments and is physiologically plausible. (For a discussion of this topic, the reader is referred to references 69, 71–73, 102, 103.)

Shift work

In the following discussion, the term shift work is used as a collective designation for all inconvenient work hours which break with the biological and/or social diurnal rhythm (night shift, rotating meeting times, and actual shift work with three to five shifts).

Shift work is not mentioned at all in the general literature about the work environment and CVD. In the literature about possible consequences for health due to shift work, CVD is mentioned, but it is concluded that shift work does not increase the risk for CVD (104–106).

In contrast to this conclusion, in 1984, Åkerstedt et al (107) published a review about shift work and CVD which concluded that shift work probably increases the risk for CVD. This result was based partly on a re-evaluation of earlier studies and partly on more recent Swedish research. This article was followed in 1985 by yet another review by Orth-Gomer (108) with a similar conclusion.

In the following text, the empirical studies in this area are reviewed, whereafter lack of consensus in the field is discussed.

Several studies from the period 1976–1984 investigated the influence of shift work on serum lipids,

blood pressure, and other cardiovascular risk factors (97, 98, 109–114). Half of these studies were cross-sectional, while the remainder were small-scale intervention studies in which, for example, clockwise and counter-clockwise rotation systems were compared. The background for this comparison is that the biological day is a little longer than the chronological one, and therefore clockwise rotation should give fewer undesirable health effects than counter-clockwise rotation. All the studies have been given "xx" or "xxx" ratings for their epidemiologic quality. These studies show no relation between shift work and blood pressure (97, 98, 109, 110), while there is a tendency towards a rise in serum lipids in connection with shift work or counter-clockwise rotation (109, 110, 112–114). The evidence from these studies, though, is not very strong due to the designs employed.

The next group of studies comprises those that have actual CVD as an end point (77, 80, 81, 115–126). There are 10 investigations, of which some are described in several references.

The classic studies in this field were carried out by the two Norwegian pioneers Thiis-Evensen and Aanonson, who published their results during the period 1949–1964 (115, 116, 125, 126). They investigated angina pectoris, hypertension, IHD, etc, by the use of a study design which was essentially cross-sectional. The methodological quality must today be rated as low ("xx"). These researchers found no relation between shift work and CVD or even a tendency towards lower morbidity among shift workers.

In 1972, Taylor & Pocock (124) published their study of mortality and shift work. For death due to CVD, they found a standardized mortality ratio of 92 for day workers, 102 for shift workers, and 132 for ex-shift workers who had transferred to day work (the so-called dropout group). Taylor & Pocock concluded that there was no relation between shift work and mortality. In this connection, Taylor & Pocock committed, in my opinion, two mistakes. For one thing, they made comparisons with the national mortality instead of using the day workers as a reference group. For another, they overlooked the trend in the material as they compared the number of deaths in each group with the "expected."

All these early studies point concurrently towards a methodological problem which is particularly large in shift work research: the selection processes. Everything indicates that both primary and secondary selection is particularly extensive in connection with shift work. Therefore, in reality, each study which does not explicitly take this fact into account must be regarded as inconclusive.

During the years 1978–1983, Koller et al published two studies (121, 122) and Angersbach et al one (118) which indicate a higher incidence of CVD among shift workers. In their first study, Koller et al found a higher prevalence of cardiovascular complaints in the drop-

out group (121). In their second, an increased prevalence of medically diagnosed CVD was found for both shift workers and dropouts, as well as a clearly increasing prevalence of CVD with age among the shift workers in contrast to the day workers (122). In their historical prospective study (118), Angersbach et al found that shift workers had a slightly higher incidence of CVD than day workers, while the dropout group had an incidence of more than twice that of the shift workers. These three studies do not have a high methodological level ("xx," "xxx," and "xxx"), but the tendencies found can be regarded as suggestive.

During the same period, Michel-Briand et al (123) published a study of shift workers and health with the use of a population of retired workers. The authors concluded that shift work increases the risk for CVD, but the methodological quality of the study is exceptionally low ("x"), and the investigation must be regarded as inconclusive.

Finally, during the period 1982–1986, Swedish researchers published three studies of higher methodological quality ("xxx," "xxx," and "xxxx"), which today form the best basis for an evaluation of a possible relation between shift work and CVD. In a case-referent study by Alfredsson et al (77, 78, 117), the shift workers had a relative risk of 1.3 for myocardial infarction. When shift work was combined with monotony or noise, the relative risk was 1.4. The reference persons were matched for sex, age, and residence.

The following study by Alfredsson et al employed a prospective design (80, 81). About one million persons were followed with regard to hospitalizations for, among other things, IHD. For men with shift work, the relative risk was 1.2, while for women it was 1.5. In this study, the risk among shift workers was probably underestimated, as there was no control with the dropout group.

A study of paper-mill shift workers by Knutsson et al (119, 120) is, to date, the best in the field. It is a historical prospective study of 504 workers over a 14-year period. Employing a two-year latency period, they found the following relative risks for IHD (fatal as well as nonfatal): 2–5 years of shift work, 1.5; 6–10 years, 2.0; 11–15 years, 2.2; 16–20 years, 2.8; ≥ 21 years, 0.4. The combined relative risk was 1.4. There was control for age, sex, and smoking. The very low risk for workers with the longest exposure was explained by the healthy worker effect.

As a supplement to these Swedish studies, a Danish study of mortality and occupation can be mentioned in which the total adult Danish population was followed for ten years via registers with the purpose of registering all deaths. It was found that, for men, eight of the ten occupations with the highest CVD mortality could more or less be characterized as shift work. They were cooks, factory workers, independent bakers, servants, drivers in salvage corps, self-employed persons in the hotel and restaurant field, self-employed taxi drivers, and salaried taxi drivers (127).

As has appeared from this review, there is a positive correlation between study result and quality so that the better studies in the field consistently find a modestly higher incidence of CVD among shift workers (and dropouts). The relative risk of 1.4 found by Knutsson et al is consistent with the results of other studies in the field and must, for the present, be regarded as the most reasonable estimate.

Future research on this topic should give attention to the following three methodological problems (107, 108, 128): (i) the previously mentioned problems in connection with primary and secondary selection; (ii) the existence of a possible threshold value, so that shift work for a number of years is necessary in order to increase the risk for CVD; (iii) the other differences that possibly exist between day work and shift work, such as noise, chemical agents, monotony, etc.

Noise

In most of the comprehensive reviews on the relation between the work environment and CVD, noise is not mentioned at all, and in the remainder the subject occupies a modest position. The literature about noise and CVD has been exhaustively covered by Rosenman (9, 10), who has five references about this subject. In those instances in which noise is mentioned, it is concluded that more research is needed.

On the other hand, there are several very extensive and exhaustive reviews about noise and nonauditive health effects, of which many deal with CVD. Since 1970, more than 25 such reviews have been published, and, of these, the vast majority are from the period 1977–1981. The most extensive and exhaustive are by Cohen & Weinstein (129) (with 143 references), Dejoy (130) (with 46 references), Hattis & Richardson (102) (with 397 references), Welch (131) (with 99 references), Westman & Walters (132) (with 123 references), and Thompson (133) (with several hundred references).

Of these reviews the one by Thompson (133) deserves particular emphasis, as it is one of the thoroughest reviews in all the epidemiologic literature. In connection with the project, 47 foreign (primarily East European) studies were ordered and translated. All the studies were evaluated methodologically by a team of experts in a number of well-defined areas. Thompson and her colleagues concluded, among other things, that the vast majority of studies have a low methodological level.

Most of the reviews on noise and nonauditive effects emphasize that noise is a stressor and that the nonauditive effects spring from this fact. In this connection, it is important to differentiate between sound and noise, as noise is defined as unwanted or uncomfortable sound. The following factors are responsible for turning sound into noise: (i) lack of control, (ii) lack of predictability, (iii) lack of meaningfulness, (iv) the volume of the sound, (v) other characteristics of the sound (intermittent sound, disharmonious sound,

etc). It is apparent that these points correspond to the previously mentioned common features of stressors.

Today, more than 200 studies on noise and CVD have been published, of which about half are epidemiologic and half experimental (both animal and human experiments). It would exceed the framework of this article to include and refer to all the epidemiologic studies. Instead, an attempt has been made to include all the studies that fulfill at least one of the following criteria: (i) published in 1980 or later, (ii) published in English, (iii) reasonable epidemiologic quality ("xxx" or more), (iv) often cited in other reviews. Those interested in the remainder of the literature are referred to the reviews mentioned.

Table 1 shows a comprehensive survey of the quality and results of 47 studies. About half of them were published in 1981 or later, and these studies are, thus, not mentioned in the thorough reviews from the period 1979–1981 (102, 131, 133). Five of the studies concern noise from airplanes, five deal with traffic noise, and the remainder concentrate on noise at the workplace. During the later years, there has been a development in this field toward increasing methodological quality, as well as an increase in the number of studies from the US and Western Europe.

The table shows that about half of the studies revealed a positive correlation between noise and CVD, while a fifth showed weak or inconsistent correlation, and one-third gave no correlation. However, the trend in the table is very relevant. A clear correlation is shown between study quality and the results, so that the share of positive studies increases with increasing quality. Among studies with low epidemiologic quality ("x" or "xx"), there is 41 % positive, against 60 % positive among the best ("xxx" or "xxxx"). This trend in the table supports the hypothesis of a causal relation between noise and CVD.

However, it is apparent that the epidemiology in this area is still of relatively low quality. The most important deficiencies are the following: (i) about two-thirds of the studies are cross-sectional, and there is a lack of studies which control for the time factor and the possible selection processes; (ii) in most studies, blood pressure and/or hypertension is used as an end point, and studies of other cardiovascular end points are desirable; (iii) few of the studies differentiate between noise and sound, and, in future studies, noise ought explicitly to be regarded as a stressor (eg, a sound level of 80 dB can be a stressor for persons who perform cognitive work, while a level of 95 dB from a plane engine need not be a stressor for the pilot); (iv) many studies have insufficient or inappropriate control for confounders; (v) several studies use defective hearing as a measure for previous noise exposure (139, 142, 147, 151, 159, 161, 163, 170, 173). As this method has been commonly used since Jonsson & Hansson published their article in *The Lancet* in 1977 (151) and as it is specific for noise research, a short account is given of why the method is deficient: (i) defective hearing

Table 1. Methodological quality and results of 47 epidemiologic studies of cardiovascular diseases (CVD) and noise. The criteria for methodological quality are explained in the text. The table is based on the following references: 78, 83, 93, 96–98, 134–174.

Result ^a	Methodological quality									
	x		xx		xxx		xxxx		x—xxxx	
	N	%	N	%	N	%	N	%	N	%
0	6	66.7	5	27.8	4	22.2	—	—	15	31.9
(+)	1	11.1	4	22.2	4	22.2	—	—	9	19.1
+	2	22.2	9	50.0	10	55.6	2	100.0	23	48.9
Total	9	100	18	100	18	100	2	100	47	100

^a 0 = no relationship between noise and CVD, (+) = slight or inconsistent relationship between noise and CVD, and + = positive relationship between noise and CVD.

due to noise exposure has a relatively low sensitivity as a measure for previous noise exposure; (ii) it can be very difficult to differentiate between loss of hearing due to noise exposure and loss of hearing due to other reasons, which gives a low specificity; (iii) loss of hearing due to noise is, strictly speaking, not due to noise but due to sound (see the preceding discussion); (iv) it is possible that atherosclerosis and/or hypertension increases the risk for loss of hearing (146, 175–177); (v) loss of hearing protects against noise; (vi) the persons to whom a certain noise is most stressing have a greater tendency to use hearing protectors and, thus, have a lower risk for loss of hearing. It is remarkable that seven out of the 15 negative studies in table 1 employ this highly defective method. If these studies were removed from the table, the percentage of negative studies would fall from 32 to 20 %.

Despite these methodological deficiencies in the epidemiologic research, the conclusion must be that this research yields reasonable support for the noise-CVD hypothesis, which actually can be regarded as a partial hypothesis in relation to the question of a causal relationship between stressors and CVD. The hypothesis is furthermore supported by experimental research and is biologically plausible (102, 129, 130, 132).

Cold

In the reviews on environmental factors and CVD, cold is mentioned by Rosenman (9, 10) and by Hopkins & Williams (5), but none of these reviews deal with the subject thoroughly as no more than two empirical studies are quoted. Likewise, CVD occupies a modest position in works on cold and health (178). However, there exist two Finnish reviews which specifically treat the subject of cold and CVD — Harjula's from 1982 (179) and Vuori's from 1987 (180). Vuori's review is particularly exhaustive (94 references).

In the following discussion, a part of the epidemiologic literature on this topic is examined with a view to elucidating the most important results and methodological problems. In 1966, Rose began his article "Cold Weather and Ischaemic Heart Disease" in this way:

"It is well known that the incidence of ischaemic heart disease tends to be higher in winter than in summer [p 97]" (181). His own investigation clearly confirms this well-known fact, and the same must be said of the wave of studies which were carried out on the subject during the 1970s (182—189). In these studies, virtually everyone used the same method, as CVD mortality (and sometimes other causes of death) during the year was related to the outdoor air temperature on the day the deaths occurred. Some of the studies also recorded rain, snow, air humidity, barometric pressure, wind, air pollution, etc.

The reverse relation between temperature and CVD mortality was found in several countries in Northern Europe and in Canada, the US, Israel, and Australia. The relation seems to be the strongest in the United Kingdom, a fact which some authors connect with the quality of housing (184, 190, 191). Occasionally, one finds factors other than cold to be significant as well, such as humidity, snow, wind, and pollution, but in all the studies temperature is clearly the most important factor. In those in which age-specific mortality rates are calculated, one finds the same trend in all the age groups, but a somewhat stronger trend with the older persons. Several studies discuss how large a role respiratory diseases play with respect to seasonal variation since these diseases could be perceived as the eliciting death cause in many cases registered as CVD deaths. The general conclusion is that the association between temperature and CVD mortality is real. In connection with these studies, there is no particular reason to relate the results with methodological quality. Most of the studies are on a medium methodological level ("xxx"), and the vast majority reach the same conclusion.

The crucial question is how these results should be interpreted. Two different interpretations are possible: (i) exposure to cold entails a permanent increase in the risk for CVD and (ii) exposure to cold increases the risk for acute manifestations of CVD (as, for example, myocardial infarction, stroke, angina attacks) in connection with the actual exposure. These hypotheses are both causal, but have, of course, very different implications. The latter hypothesis is primarily of importance for persons with existing CVD, while the former means that cold is a risk factor in the development of CVD.

Both Harjula and Vuori seem to conclude that the empirical research confirms both hypotheses, which of course is possible in principle (179, 180). However, there only seems to be empirical support for the latter hypothesis, which deals with the acute effect. As all studies in the field deal with seasonal variations in CVD in connection with outdoor temperature, in reality, it is impossible to know if the exposure to cold entails a lasting increased risk.

From the point of view of occupational medicine, it would be relevant to know if exposure to cold increases the risk for developing CVD, but as far as is

known there have been no studies of, for example, slaughterhouse workers, fishery workers, or cold storage workers that elucidate this hypothesis. Such studies would be highly desirable.

Heat

Among the reviews cited concerning the environment and CVD, only Millar (8) considered it proved that heat is a risk factor for CVD. He wrote: "... heat (is) known to increase the risk of cardiovascular disease [p 239]." Furthermore, heat was mentioned as a possible risk factor by Rosenman (9, 10), Goldhaber (2), and Harlan et al (4).

No reviews especially concerned with heat and CVD have been found, but in the review by Dukes-Dobos, "Hazards of Heat Exposure" from 1981 (192), tachycardia is mentioned as an effect of several months in a hot job, while hypertension and myocardial damage are mentioned as results of many years in a hot job.

The effect of heat on the incidence of CVD has been investigated in a manner similar to that used for cold. In Rogot & Padgett's study on mortality in American cities (188), a U-shaped relation is found so that the incidence of CVD is highest in connection with cold and heat, but lowest with temperatures in the range of 16—27°C. A trend towards an increasing incidence of CVD in very hot months was also found by Heyer et al in Dallas, Texas (193), by Avierinos in Egypt (194), and by Al-Yusuf et al in their study of patients from Kuwait (195).

As was the case with cold, the relation can be explained in two ways, as an acute effect and as a lasting effect. The study by Al-Yusuf et al supports the hypothesis of an acute effect, as the total increase in CVD hospitalizations during the hot period can be explained by rehospitalizations of patients with previous infarctions.

While there is an apparent lack of studies on CVD in workers exposed to cold, three investigations exist on heat exposure on the job and CVD. Two of them are cross-sectional studies on the blood pressure of heat-exposed workers (196, 197). They focus on workers in a metallurgical plant in Brazil and in a glass factory in Canada. Both studies found a clear positive correlation between blood pressure and exposure to heat. Both have been given an "xx" rating for methodological quality, as little or no account was taken of selection, the time factor, confounding, etc.

The third study is a historical prospective mortality study of steelworkers in the US (198). It was found that CVD mortality was 10 % lower for the workers exposed to heat than for a reference group which was not exposed to heat. Further analysis showed, however, that the CVD mortality was relatively high for those workers who had been employed for less than six months in jobs with heat exposure (199). This finding could indicate a healthy worker effect, and there-

fore the CVD mortality among persons exposed to heat was probably underestimated in this study.

All things considered, it must be concluded that the hypothesis about an acute increased risk of CVD attacks in persons with existing disease is confirmed, while the hypothesis about a lasting effect of heat exposure is neither confirmed nor invalidated. On this subject, too, more extensive and better research is desirable.

Electromagnetic waves and fields, high- and low-frequency sound, etc

Several case reports mention IHD and atherosclerosis as possible consequences of irradiation in connection with treatment for Hodgkin's disease and other forms of cancer. A review of these case reports is beyond the limits of this article. A good review of this subject was made by Dunsmore et al (200) and Rosenman (9). Boivin & Hutchison's follow-up study (201) of 957 patients who had received radiotherapy for Hodgkin's disease showed a higher IHD mortality among the irradiated patients than among the referents. The difference was on the border of statistical significance. As far as is known, no studies have been carried out on CVD in employees with occupational exposure to radiation.

Part of the literature about radio-frequency radiation has been reviewed by Resnekov (202), who wrote that most studies are East European and difficult to evaluate due to the terminology and methodology employed. After the deliberate beaming of microwaves into the building of the US embassy in Moscow, the employees were examined, but no adverse health effects were traced (203). Neither could negative health effects of microwave radiation be shown in an extensive study of 40 000 men in the American Navy (204). In this study, mortality, as well as hospitalizations and disablement, were end points. Gonzales (3) and Harlan et al (4) concluded in the early 1980s that further research in this field is greatly needed, and this conclusion is still valid.

A Swedish (205) and an English (206) study examined workers with long-term exposure to electric fields. The Swedish investigation (205) dealt with employees of high-voltage substations and the English one (206) with electrical power transmission and distribution workers. In neither of these thorough cross-sectional studies was it possible to trace any relation between exposure to electric fields and cardiovascular morbidity.

In contrast to these negative studies concerning electric fields, Perry & Pearl, in a recent investigation (207), found a relation between power-frequency magnetic fields and CVD. Hospitalizations among residents in multistory apartment buildings were examined. Persons who lived close to the perpendicular electrical supply cables were classified as exposed, while the remainder were considered unexposed. The study resembled a randomized trial, as neither the residents nor

the housing agency knew where the cables were placed or had any suspicion of any adverse effect on health. Sixty-three percent more IHD hospitalizations were found among those exposed than among the unexposed ($P=0.056$). This finding was unexpected as magnetic fields had previously mainly been related to leukemia and psychiatric disorders. In a review article from 1982, Easterley (208) referred to the experimental literature concerning cardiovascular risk from exposure to static magnetic fields and developed a model for assessing cardiovascular risk. In Miller's article "Exposure Guidelines for Magnetic Fields" (209), the risk for CVD was only dealt with sporadically.

Very few authors have discussed the possible relationship between low-frequency noise (0–100 Hz) and CVD. Broner's 1978 review (210) on the effects of low-frequency noise on people treats the effects on the cardiovascular system only very peripherally, and the same is true of Landström's review from 1979 (211). In 1985, Danielsson & Landström published the results of an experimental investigation which showed increased blood pressure under infrasonic exposure (212). With respect to this finding, the authors remarked that it was the first study in the field. Apparently, no investigation of a possible relation between high-frequency noise (over 20 000 Hz) and CVD has been published (213).

All things considered, it must be concluded that there is little agreement between the extension of the mentioned exposures and the research activity. This lack is possibly related to the fact that those people who are exposed to electromagnetic waves and fields generally do not have knowledge of the exposure. From an ethical point of view, one could say that especially this fact ought to incite increased research activity.

Concluding remarks

In his book *Occupational Epidemiology* Monson wrote: "There are only few studies in which a clear-cut association has been found between work and cardiovascular disease. This probably is a reflection of both biology and methodology. Most cardiovascular disease probably occurs for reasons unrelated to a person's occupation. The rate ratio of work-related disease is therefore low [p 205]" (7). Other authors of reviews have been less cautious, but Monson's remarks are probably a manifestation of the general attitude among both cardiovascular and occupational medicine epidemiologists.

This review has attempted to examine a large number of epidemiologic studies. A particular objective has been to include all studies of medium or high quality. In the evaluation of causality, the investigations examined have been scored from "x" to "xxxxx" for methodological quality.

The cognitive basis has been that a hypothesis about causality cannot be "proved," but is confirmed or invalidated through well-performed studies.

Concerning physical inactivity, the research is very extensive and of high quality. The results are, moreover, consistent and biologically plausible. There are still unsolved problems in this area of research, but it appears beyond any reasonable doubt that a causal relation exists between physical inactivity and CVD. It would appear that the labor market will, in an even increasing degree, continue to be characterized by sedentary work, and employees will have to compensate for this risk factor in their spare time.

Research dealing with monotonous, high-paced work and other stressors at work and with shift work is far less extensive and occasionally of a slightly lower quality. With regard to quality and quantity, however, great progress has been made, especially in the 1980s, with Sweden as the center. The hypotheses about causal relation in these two areas must be considered well-founded, but further research with the inclusion of the conventional risk factors is clearly necessary.

Concerning noise, the quality of the research is clearly lower than in the other areas already mentioned. There is a very large number of investigations, however, and a tendency for the better studies to support a causal hypothesis to a greater extent than the studies of poorer quality do. Noise seems primarily to increase the risk for CVD via increased blood pressure, but the effect is so modest for the individual exposed that it would be difficult to demonstrate in epidemiologic studies with actual CVD morbidity or mortality as the end points.

Most of the research concerning cold and heat has focused on seasonal variations in the incidence of CVD. It has been clearly shown that the highest incidence is found in particularly cold or hot months and the lowest at medium temperatures. Both cold and heat can be the eliciting factor in persons with existing CVD. However, whether long-term occupational exposure to cold or heat permanently increases the risk for CVD has not been sufficiently clarified. Research on this topic is very desirable.

Concerning electromagnetic waves and fields, as well as infrasound and ultrasound, the research has been modest. Some studies suggest cardiovascular effects, and again it is concluded that further research is desirable.

Generally, this review has found considerably more evidence for a relation between the work environment and CVD than earlier reviews. This difference is partly due to the fact that more empirical studies have been included, partly to the employment of a method-critical approach, and partly to the fact that there has been a tremendous expansion in this research in recent years. On this basis, it is recommended that occupational factors in the future receive a more prominent place in cardiovascular research and that researchers in occupational medicine give more attention to cardiovascular diseases.

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