Cardiovascular diseases and psychosocial factors at work

Maladies cardiovasculaires et facteurs psychosociaux au travail

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Summary

Besides the ‘classic’ cardiovascular risk factors (high blood pressure, dyslipidaemia, metabolic syndrome and diabetes), the work environment is playing an increasingly significant role in cardiovascular morbidity and mortality. Several elements contribute to the effect of the work environment: physical factors, chemical factors, shift work and psychosocial factors. The effects of psychosocial factors on the aetiology and progression of cardiovascular disease have been confirmed by several studies. Identification of these work-related psychosocial factors must be taken into account when evaluating cardiovascular risk factors, in order to ensure better prevention.

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MOTS CLÉS
Maladies cardiovasculaires ; Travailleur tendu ; Déséquilibre effort récompense ; Facteurs psychosociaux au travail

Résumé

À côté des facteurs de risque cardiovasculaires «classiques» (hypertension artérielle, dyslipidémie, syndrome métabolique, diabète), l’environnement professionnel joue un rôle de plus en plus important dans la morbidité et la mortalité cardiovasculaire. Plusieurs éléments contribuent à l’action de l’environnement professionnel : les facteurs physiques, les facteurs chimiques, le travail posté et les facteurs psychosociaux. Les effets des facteurs psychosociaux sur l’étiologie et la progression des maladies cardiovasculaires sont étayés par de nombreux travaux. L’identification de ces facteurs psychosociaux au travail doit être prise en compte lors de l’évaluation des facteurs de risque cardiovasculaires afin d’assurer une meilleure prévention.

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Abbreviations: AHT, arterial hypertension; BP, blood pressure; CVD, cardiovascular disease; OR, odds ratio; PSF, psychosocial factor; RR, relative risk.

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Introduction

There has been a significant increase in the incidence of CVDs in industrialized countries over the last 40 years. CVD was the leading cause of mortality until the turn of the 21st century, when deaths caused by tumours surpassed deaths caused by CVD in France. The ranking of causes differs markedly according to sex, as CVD is the second cause of mortality in men after tumours, whereas this order is reversed for women [1]. Recent developments in the monitoring of ischaemic heart disease have revealed two trends: a drop in cardiovascular mortality [2,3], particularly a decrease in deaths caused by acute coronary syndrome, which reflects progress made in medical case management; and a slowing down or even stabilizing of the decline in the incidence of coronary disease, which indicates that efforts are required in terms of primary prevention [4,5].

Three major groups of determinants are usually identified for cardiovascular morbidity and mortality: personal determinants (age, sex); biological determinants; and behavioural determinants. In France, primary prevention mainly focuses on the ‘classic’ risk factors, i.e. bioclinical and behavioural determinants [4,6]. These factors are, primarily, high BP, dyslipidaemia, smoking, diabetes and metabolic syndrome. This approach to prevention needs to be supplemented for several reasons.

First of all, according to various studies [4,7], these ‘classic’ factors can explain 50 to 80% of cases of CVDs. The INTERHEART study, which measured the association of nine modifiable risk factors with myocardial infarction in 52 countries, revealed that PSFs (stress at home or at work, financial stress, life events) accounted for 32.5% of the population attributable risk for myocardial infarction, putting them in third place behind risks associated with lipids and cigarette smoking [8]. This means that proper management of the PSFs in primary prevention could, in theory, reduce the number of infarctions by 32%.

Secondly, prevention that focuses on the ‘classic’ factors facilitates the individual approach, centred on the use of risk prediction charts. These charts are used to calculate the cardiovascular risk of an individual, by identifying the level or presence of a number of ‘classic’ risk factors. This method is an appealing concept; the approach focuses on bioclinical or behavioural factors, without taking into account other risk factors and psychosocial determinants, in particular. Modelling becomes approximate as soon as the person does not belong to the reference group used to develop the risk prediction equation. These charts have trouble predicting the overall cardiovascular risk [6,9].

Lastly, analysis of premature mortality (prior to age 65 years) caused by CVD reveals disparities in the level of reduction between the different social groups. There is still a higher rate of cardiovascular mortality among groups with a lower socioeconomic status or among individuals with a lower level of education [10,11]. This disparity clearly raises the question of the role of socioprofessional determinants in health inequalities.

For all of these reasons, the long-debated role of PSFs in the onset of CVD is now clearly established. Several studies in literature have considered both the emotional impact and the quality of life impact on cardiovascular health. The stress factors associated with cardiovascular risk include the following: type A behaviour (stress hyperactive individuals with a strong sense of competitiveness) [12]; type D behaviour (negative affectivity and inhibited relationships) [13]; anxiety disorders (panic disorders, anxiety) [14,15]; and mood disorders [16]. On the other hand, some other factors, such as social support, are regarded as more protective. More specifically, our article will deal with the role of PSFs at work. Numerous factors linked with occupational exposure have been identified. A great number of published studies have evaluated the role and mode of action of work-related PSFs on cardiovascular morbidity and mortality. There is such a large number and variety of such studies [17], in fact, that it is impossible to present a complete bibliography of them. The principal aim of this article is to review the main achievements in this area. First, we will discuss the theoretical approaches and the development of the main tools used to evaluate PSFs in the workplace, and then we will endeavour to reveal the major trends in the cardiovascular field, illustrating these with a selection of results.

Methods

We searched through several Medline databases, using keywords such as ‘psychosocial factors’, ‘ischaemic heart disease’, ‘stress at work’, ‘job strain’ and ‘arterial hypertension (AHT)’. We examined the articles published between 1979 (the year Karasek’s first article was published) and 2008. The articles selected refer explicitly to Karasek’s model and Siegrist’s model, and discuss cardiovascular morbidity and mortality linked to AHT and ischaemic heart disease. We restricted ourselves to epidemiological studies published in peer-reviewed journals. We selected the standard reference works or those that have been cited very often.

Psychosocial factors at work

Over the last few years, jobs have been characterized by an overall reduction in strenuous physical activity—although this reduction is only slight among the exposed groups—and increasing job demands [18]. These PSFs at work are linked to the individual, collective and organizational aspects of the occupational activity. They are likely to have an effect on health and, in particular, they include psychological job demands (excessive workloads, time pressure, ambiguous roles and workplace insecurity) and poor labour-management relations. The quantification of exposure to job strain has been evaluated by several theoretical models. The two most commonly used general models are the Karasek model [19,20] and the Siegrist model [21,22]. Other more recent concepts have been developed, relating to organizational justice or violence in the workplace. We will restrict ourselves to the Karasek and Siegrist models, which are the subject of the highest number of studies.

The PSF evaluation model, put forward by Karasek, is based on a self-report questionnaire that evaluates an individual’s perception of work through several dimensions: the first dimension is psychological demand and focuses on the
psychological stress associated with task complexity and execution, time pressure, work interruptions, unplanned tasks and contradictory demands. The second dimension is decision latitude, which measures the amount of control an individual has over their own work (freedom to choose how to carry out the work) and skill use (the possibility of using one's qualifications and developing new ones). Individuals are positioned based on these two dimensions, using an algorithm to categorize them into one of four groups (Fig. 1): the high demand, low decision latitude combination is labelled a high-strain job; the high demand, high decision latitude combination is labelled an active job; the low demand, low decision latitude combination is labelled a passive job; and the low demand, high decision latitude (ideal situation) is labelled a low-strain job. To these two dimensions, a third has been added to evaluate relationships between coworkers and superiors (social support in the workplace) [23]. The underlying hypothesis is that the high psychological demand and low decision latitude combination, categorized as job strain, increases the risk of CVD, especially where there is little social support.

The effort-reward imbalance model was introduced later by Siegrist. This model distinguishes two sources of effort and three types of reward (Table 1). It defines and analyses the socioemotional risks in the work environment in terms of the mismatch between the efforts invested and the rewards—whether symbolic (esteem, recognition) or pecuniary—because the efforts are part of a process in which the rewards are expected. It is based on the hypothesis that significant and continued efforts and low rewards can have a damaging effect on health. In the Karasek model, the decision latitude dimension is pivotal, whereas in the Siegrist model, it is the concept of social reciprocity (the right to legitimately expected rewards) that is the central tenet. These two models have been extensively validated by studies on the incidence of CVD [24–26].

### Mechanisms

The different studies published on this subject report two underlying mechanisms. These two complementary mechanisms seem to have a direct effect on pathophysiological processes, as well as an indirect effect through the adoption of high-risk habits [7].

The direct mechanism posits a chronically hyperactive sympathetic nervous system with a rise in BP and left ventricular hypertrophy, hypercatecholamineaemia, an alteration in immune and inflammatory responses and early-onset atherosclerosis [27–29]. The entire circulatory system is affected, although a greater number of studies have focused on the coronary system. Changes in other target organs have been observed, particularly in the carotid and cerebral artery territories [30].

The indirect mechanism involves behavioural factors such as obesity, lack of physical activity, alcohol and/or tobacco consumption, lack of access to health care or the metabolic syndrome [31–33]. The premise is that PSFs in the workplace contribute to weakening self-control, which in turn encourages an unhealthy lifestyle, with increased consumption of alcohol and/or tobacco and a tendency to become overweight or obese.

### Results

The two models for evaluating PSFs at work have revealed a link between these demands and cardiovascular morbidity (hypertension in particular) and mortality, and their sometimes predictive role. The data sources are varied (mortality or morbidity databases, monitoring system) and most of the studies were conducted in industrialized countries [7]. The main indicators used in the literature are AHT, ischaemic heart disease and death from cardiac causes [17].

### Table 1 Dimensions of the Siegrist model.

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<th>Extrinsic efforts</th>
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<th>Rewards</th>
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Arterial hypertension

High BP is a recognized cardiovascular risk factor, with no threshold effect, associated with cardiovascular morbidity and mortality \cite{34,35}. Although it is most often labelled as ‘essential’, AHT is associated with several risk factors such as age, lack of physical activity, obesity and alcohol consumption.

The Karasek model contributes greatly to explaining CVD because of the many studies that have revealed the role of PSFs in the appearance of AHT \cite{17}. A thesis devoted to this subject summarized these studies \cite{36}. The results observed depend on the type of study (longitudinal, cross-sectional, case-control) and the type of BP measurement. There are a far greater number of studies involving occasional BP measurement. Although most of these reveal a connection between PSFs and increased BP, the correlation is less constant due to the limited number of measurements and the BP variability. On the other hand, studies using ambulatory BP monitoring have the advantage of avoiding BP variability.

One of the first longitudinal studies to use ambulatory BP monitoring was by Schnall et al., who included 195 men monitored over 3 years. They showed that the average BP measured at two different periods was higher in stressed workers than in others \cite{37}. Furthermore, individuals who were exposed to job strain during the initial period, but not during the second period 3 years later, showed a decrease of approximately 5/3 mmHg in the work BP reading taken during the second period. A study by Landsbergis et al. \cite{38} focused on 213 men and showed that, at work, the systolic BP of employees exposed to job strain with over 25 years of service was 4.8 mmHg higher than that of unexposed employees. This difference reached 7.8 mmHg when systolic BP was measured at home after work. The studies conducted by Schnall et al. and Landsbergis et al. represent a major step forward in understanding the mechanisms that can explain cardiovascular morbidity. On one hand, they suggest that reducing exposure to job strain or removing the individual from the environment leads to a reduction in BP \cite{37}; on the other hand, they reveal that exposure has a cumulative effect \cite{38}.

Other longitudinal studies conducted among both men and women confirm the correlation between BP and PSFs at work, with a stronger correlation among men. In a study by Ohlin et al. (involving 448 men and women), the results differed according to the sex of the individual \cite{39}. The correlation between job strain and increased BP was only seen among men (+7.7 mmHg for systolic BP and +5.6 mmHg for diastolic BP). A study by Guimont et al. \cite{40}, which monitored white-collar workers over a period of 7.5 years, showed a slight but significant increase in systolic BP among men exposed to job strain (+1.8 mmHg). This effect was less significant among women (+0.5 mmHg).

It is worth pointing out that certain longitudinal studies found no correlation between ambulatory BP and PSFs. Their authors hypothesized that this was due to the absence of a cumulative effect of exposure as a result of too short a monitoring period \cite{41} or because the individuals monitored were too young \cite{42}.

The number of cross-sectional studies far outweighs the number of longitudinal studies, but they are less reliable. We will mention a few that show an increase in ambulatory BP in the job strain group. This increase in BP is constant and sometimes persists outside of work. In a study that included a sample of 527 individuals aged between 25 and 64 years, Cesana et al. evaluated the correlation between ambulatory BP and PSFs in the workplace \cite{43}. The psychological demand and decision latitude combination made it possible to categorize each person into one of four exposure groups. Among the 385 normotensive individuals, systolic BP measurements at work and at rest were higher in the group exposed to a significant level of job strain (job strain +3.4 mmHg). The BP values then decreased in the passive group, followed by the active workers’ group, and lastly, the low-strain group. In other studies, ambulatory BP among individuals exposed to job strain was not only high while working and while at rest, but also remained high while the individuals were sleeping. In a study conducted in Belgium, Clays et al. \cite{44} showed that the average ambulatory BP measured within a 24-hour period was higher among the group of individuals perceiving the demands. This increase in BP persisted outside of work and during sleep. In another study that measured diastolic BP, the increase among exposed individuals was observed during work (+7.4 mmHg), leisure (+5.9 mmHg) and sleep (+7 mmHg) \cite{45}.

Generally speaking, case-control studies are much rarer. We can cite one such French study which, to our knowledge, was one of the first to be conducted among a working population in France. This case-control study, which evaluated the correlation between PSFs and AHT, revealed that this association was particularly strong among women \cite{46}. Compared with the low-strain group, the adjusted OR among women was 4.73 (1.36–16.42) for the passive group, 4.51 (1.24–16.43) for the active group and 3.20 (0.92–11.12) for the job strain group. Among men, the association was significant in the job strain group (2.60 [1.15–5.85]), the passive group (2.30 [1.01–5.26]) and the active group (2.39 [1.1–5.18]).

The conclusion that is largely shared in these different studies is that psychosocial stress is associated with the onset of the rise in BP, regardless of the type of study or population. The scope of this correlation is nevertheless a source of debate: certain authors believe that PSFs can increase BP at work but are not a predictive factor of long-term AHT \cite{47}. Other authors, on the other hand, emphasize the cumulative aspect of stress, which predicts the appearance of AHT: this is the case in a recent literature review (of 10 cohort studies and four case-control studies) published in 2009 by Sparrenberger et al., which highlights the role of chronic stress \cite{48}.

The Siegrist model has been backed up by several studies on AHT that is, or is not associated with other cardiovascular risk factors \cite{49,50}. In a cohort study conducted among blue-collar workers, Siegrist et al. reported that the presence of AHT was associated with a lack of career prospects (rewards) and an overinvestment in work \cite{51}. In a study conducted by Steptoe et al. among 197 workers, overinvestment was a predictive factor for systolic BP during the day among male workers exposed to psychological job demands \cite{52}.,
Ischaemic heart disease

Research work conducted on the role of PSFs in the onset of ischaemic heart disease has mainly focused on angina and myocardial infarction. It is worth noting that most of the research work was carried out over the last 20 years, which explains why the analyses were conducted using standardized diagnostic categories (stable angina, unstable angina, completed myocardial infarction), whereas current developments in coronary disease monitoring reveal that the notion of acute coronary syndrome takes precedence [53].

Several recent studies reveal a positive correlation between PSFs in the workplace and acute coronary events. In a longitudinal study conducted over a period of 12 years among over 10,000 white-collar workers in the United Kingdom with little exposure to physical demands, Chandola et al. [54] revealed a dose–response relationship between work stress evaluated according to the Karasek model and the onset of coronary artery disease (infarction or definite angina with treatment by nitrate compounds). By first adjusting for behavioural factors, then for metabolic syndrome, and lastly, for these two major factors, the authors showed that RR increased from 1.41 to 1.52 (for stress reported once) and from 1.56 to 1.61 (for stress reported twice).

A review of literature conducted by Belkic et al. [55] evaluated the results of 17 longitudinal studies investigating the correlation between job strain and cardiovascular mortality or coronary disease. Among these studies, eight provided evidence of a positive association, with RR ranging from 1.21 (1.08–1.35) to 4 (1.1–14.4) and three others showed a positive but non-significant association. Regarding the six studies that showed no association, the authors mentioned methodological biases linked to a faulty evaluation of exposure or job status.

A meta-analysis of studies published between 1979 (the year Karasek’s first article was published) and 2006 was conducted to estimate the RR of coronary disease associated with the occupational stress indicators evaluated by the Karasek and Siegrist models [7]. This meta-analysis was based on 14 longitudinal studies conducted in Europe, USA and Japan. The studies using the Karasek model included a total of 83,014 workers, while those using the Siegrist model included 11,528 workers. The RR of coronary disease or cardiovascular events adjusted for age and sex was 1.43 (1.15–1.84) for the studies using the Karasek model. After adjustments for cardiovascular risk factors, the RR remained positive but not significant (1.16 [0.94–1.43]). For the studies using the Siegrist model, the RR adjusted for age and sex was 1.58 (0.84–2.97). By adjusting for confounding factors, the RR reached 2.05 ([0.97–4.32]). Although the results are not all significant, by providing an objective summary of the 14 studies conducted between 1979 and 2006, this meta-analysis shows an excess risk of CVD among workers exposed to PSFs at work.

Finally, other cross-sectional studies or case-control studies have shown an association between coronary artery disease and PSFs. In a literature review conducted by Belkic et al., four cross-sectional studies out of eight and six case-control studies out of nine revealed a statistically significant relationship between PSFs at work and cardiovascular morbidity and mortality [55]. In a case-control study conducted in China, Xu et al. showed that the combination of a high effort-reward imbalance and overcommitment was associated with a risk of coronary disease, with a dose–response effect. This association was maintained when they adjusted for ‘classic’ risk factors (OR = 1, OR = 3.2 [1.8–6.2], OR = 5.5 [2.2–13.4]) [56].

Limitations

Several limitations can be identified. The first limitation relates to heterogeneity. The populations included in these different studies are variable (working population, general population). Exposure evaluations are sometimes heterogeneous, as some studies use job-exposure matrices, whilst others use instruments with items. The outcomes are not homogeneous either, as some relate to mortality and others to morbidity. This makes it more difficult to compare the different studies.

The second limitation is that these studies are not necessarily independent of one another. As a reminder, much of this research was conducted as part of the Whitehall cohort study.

Lastly, there is a publication bias. This is the tendency to over-represent in literature those studies that show significant results, to the detriment of studies that reveal an absence of significant results.

Conclusion

CVDs remain a major source of mortality and morbidity, and is costly for the health care system. These diseases are multifactorial in essence and the impact of PSFs at work has been evaluated in numerous studies. These studies show that individual risk factors do not fully explain the health of individuals and that PSFs at work play a role in the aetiology and progression of CVD. Although they are not yet unanimously recognized as a cardiovascular risk factor in their own right, PSFs at work must be taken into consideration in the same way as ‘classic’ risk factors. A combined approach that takes into account all of the risk factors is becoming increasingly important, given the increase in PSFs brought on by the changes that have taken place in the working environment over the last few years. This is a public health issue because collective prevention will be more effective once all of the factors, and in particular those related to the work environment, are identified and taken into account.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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