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Work stress and health risk behavior

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Work stress and health risk behavior

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This contribution discusses current knowledge of associations between psychosocial stress at work and health risk behavior, in particular cigarette smoking, alcohol consumption and overweight, by reviewing findings from major studies in the field published between 1989 and 2006. Psychosocial stress at work is measured by the demand–control model and the effort–reward imbalance model. Health risk behavior was analyzed in the broader context of a health-related Western lifestyle with socially and economically patterned practices of consumption. Overall, the review, based on 46 studies, only modestly supports the hypothesis of a consistent association between work stress and health risk behavior. The relatively strongest relationships have been found with regard to heavy alcohol consumption among men, overweight, and the co-manifestation of several risks. Suggestions for further research are given, and the need to reduce stressful experience in the framework of worksite health promotion programs is emphasized.

Key terms alcohol consumption; cigarette smoking; demand-control model; effort–reward imbalance model; health-related lifestyle; overweight; review.

"Stress" is a latent construct that indicates a state of elevated activation of the autonomic nervous system with coordinated manifestations at the affective, cognitive, and behavioral levels. In many, but not all instances, stress is provoked by the presence of a stressor (ie, an acute or chronic extrinsic demand that taxes or threatens an individual's ability to cope). Threat to control and loss of control and associated rewards go along with intense, long-lasting negative emotions and autonomic arousal, particularly so in cases in which an important task has to be accomplished and people cannot escape from the situation (1). The duration and intensity of stress reactions vary according to personal and interpersonal coping resources and capabilities (2). Yet, to a large extent, they are determined by features of extrinsic stressors. It is evident that work and employment can be a source of multiple stressors, apart from their beneficial aspects for personal well-being and welfare (3).

While occupational health research has long been concerned with material (ie, physical and chemical) stressors at work, the nature of work and employment underwent significant changes in recent decades, with a shift in the prevalence of health-adverse work environments from material to mental and emotional stressors. Today, fewer jobs are defined by physical demands, and more are delineated by mental and emotional demands. Computer-based information processing is becoming part of a growing number of job profiles, and employment in the service sector continues to increase. In a macroeconomic context, with the advent of globalization, work pressure has increased considerably along with growing job insecurity and job loss.

This situation provides two main challenges to occupational stress research. First, how can we define and measure the major stressors of modern worklife at a level of generalization that allows for their use in a wide range of different occupations? Second, how can we achieve progress in understanding the mechanisms leading from exposure to work stressors to the development of stress-associated physical and mental diseases?

The following sections are mainly devoted to the second question, but a short answer to the first question is needed for the interpretation of the hypotheses and research findings related to the question of mechanisms.

Theoretical models of work stress

Several theoretical concepts were developed, and concepts continue to be developed, to explain the health effects of stressful work. [For a recent review, see the paper by Antoniou & Cooper (4)]. Two such models

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have received special attention recently, the demand–control model and the effort–reward imbalance model.

The former model identifies stressful work by job-task profiles that are characterized by high quantitative demands (eg, work pressure) in combination with low control (5). Importantly, a low level of control or decision latitude manifests itself in two ways, first, as lack of decision authority over one's tasks and, second, as a low level of skill utilization, as evidenced by monotonous, repetitive work. As an additional analytical dimension, social support at work was added to this model. Accordingly, the highest job strain is expected to occur in jobs that are described by high demand, low control, and low support at work or social isolation.

The effort–reward imbalance model builds on the notion of contractual reciprocity (6) that lies at the core of the work contract, with respect to which accomplished tasks are reciprocated by adequate rewards (money, esteem and career opportunities, including job security). The model claims that lack of reciprocity occurs frequently under the following three conditions and that failed reciprocity in terms of "high cost" and "low gain" elicits strong negative emotions and sustained stress reactions: (i) "dependency" (due to a lack of alternative choice in the labor market); (ii) "strategic choice" (anticipatory investments in order to increase future promotion prospects); (iii) "overcommitment" (a motivational pattern of excessive work-related performance and achievement that may be part of a person's psychological profile or result from a competitive work environment).

The two models identify complementary aspects of a stressful psychosocial work environment with focus on task control and participation in the former model and focus on reward and contractual fairness in employment in the latter model. Both models are measured by standardized questionnaires with validated psychometric properties (7, 8). They have been extensively tested using a variety of study designs, for example, prospective observational studies, case–control and cross-sectional investigations, laboratory experiments, "naturalistic" studies with ambulatory monitoring techniques, and intervention studies. [For a review, see the papers by Belkic et al (9) and Tsutsumi & Kawakami (10)]. Before reviewing recent evidence on their role in explaining health risk behavior, we need to understand the pathways linking work stress with physical and mental disorder in more detail.

The challenge of mechanisms: health risk behavior

A common way of explaining the pathophysiological mechanisms underlying the association between work stress and stress-related diseases, such as cardiovascular diseases, metabolic disorders or depression, assumes two separate mechanisms, one acting "directly" via the organism's main stress axes (11) and a second "indirect" pathway operating via behavioral decisions or habits of people exposed to work stressors (12). These choices or habits generally concern health risk behavior, such as a diet rich in saturated fat and calories, cigarette smoking, high alcohol consumption, and lack of physical exercise.

Despite its plausibility, this approach faces the conceptual problem of nondetermination as no additional information is provided to predict under what conditions "direct" or "indirect" pathways are expected to occur. Accordingly, in many epidemiologic studies on work stress and health using multivariate regression models, odds ratios of respective disease risks are adjusted for health risk behavior, in an attempt to estimate "direct" effects of work stress on health (13). In other investigations, the role of health risk behavior as a mediator of the association between work stress and disease is analyzed, exploring the relevance of "indirect" effects (14).

While this difficulty is far from being resolved, the first task consists of examining the available empirical evidence of associations between work stress and health risk behavior more closely in terms of the two models. What is the common denominator of a set of behaviors that are commonly labeled "health risk behavior", and how consistent are the associations across the single health risk behaviors and across the populations under study? Once these questions are further clarified and once the available evidence has been assessed, suggestions for future research strategies can be developed.

To our knowledge, the term "health risk behavior" has never been properly defined. As a wide range of behavior, unconscious as well as conscious, has a potential impact on health, a scientifically useful definition turns out to be hard to reach. As a first step towards clarification, one may ask what external constraints limit the range of behavioral choices. In this regard, the concept of health-related lifestyle is of interest. A health-related lifestyle is defined as a "collective pattern of health-related behavior based on choices from options available to people according to their life chances [p 901]" (15, 16). Health-related activities typically consist of practices related to food, exercise, personal hygiene, smoking, alcohol, drug use, risk of accident, relaxation, and preventive check-ups. These types of behavior are not randomly distributed across societies, but are clustered according to a society's opportunity structure.

The way behavioral choices, options, and the life chances embedded in a societal opportunity structure are interrelated has been elucidated by the famous sociologist Max Weber. According to Weber (17), the concept of lifestyle is based on two notions, "life conduct" and

"life chances". Life conduct reflects people's preferences or choices that are influenced by social norms and values, whereas life chances refer to the material constraints that limit these preferences, such as financial resources, availability of goods, or mechanisms of social control. In this regard, social status and a broader economic and cultural context are powerful determinants of health-related lifestyles, and specifically of the patterns of consumption that are preferred.

Most importantly, a Western lifestyle evolved from the process of industrialization with a huge impact on population health (15). After severe poverty in the early stage of industrialization, economic progress and the development of a welfare state were experienced by a growing proportion of industrial populations. This progress included the availability of food rich in calories and fat, as well as rich in vitamins and proteins, better housing, energy consumption, transport, education and general hygiene. In terms of population health, Western industrialization was associated with a marked increase in life expectancy and a change in the pattern of prevailing diseases, the epidemiologic transition from infectious to chronic diseases. At the same time, highly prevalent "diseases of affluence", such as coronary heart disease, metabolic disorders, certain cancers (especially lung cancer), and accidents, were largely triggered by a health-adverse lifestyle that was characterized by reduced physical activity, by frequent consumption of fat and meat, and by the consumption of drugs such as alcohol and cigarettes. Consequently, during the first stage of the epidemiologic transition, these patterns of consumption associated with wealth were more prevalent among socially and economically privileged groups. At the later stage of the process of industrialization, however, this social pattern changed. With the spread of Western lifestyles across social boundaries "diseases of affluence" increasingly became the "diseases of the poor" (18).

In contemporary advanced societies, a clearcut social gradient of morbidity and mortality is observed, leaving those with lower status within the social hierarchy in poorer health (19). Part of this social gradient is explained by a higher prevalence of health-adverse behavior, in particular unhealthy diet, overweight, lack of physical exercise, and cigarette smoking, among lower socioeconomic status groups. These groups may also pay less attention to their body in terms of personal hygiene, risk-reducing behavior, and preventive measures, including medical check-ups (20, 21).

In the next section we ask to what extent this socially patterned health-adverse lifestyle is determined by stressful exposures in general, and by chronic work stress in particular (15).

We focus on exposure to a stressful psychosocial work environment in terms of the two models, demand–control and effort–reward imbalance, by evaluating empirical evidence on its association with a health-adverse lifestyle. This review is restricted to the main health risk behaviors of cigarette smoking (table 1), alcohol consumption (table 2), and body weight (table 3), as these types of behavior were the most often assessed in studies dealing with work stress in terms of the two models. Moreover, overweight or weight gain can be considered a crude proxy measure of exposure to a sedentary lifestyle and unhealthy diet as these conditions often cluster among individuals (22).

This review is based on a literature search of investigations published in established peer reviewed journals between 1989 and 2006. Relevant articles were identified using PubMed. The search term included items related to either work-stress model and the selected lifestyle variables (eg, overweight, weight gain, BMI, obes*). In addition, the bibliographies of the retrieved articles were hand-searched for additional studies. No study was excluded on the grounds of methodological quality. Altogether, 46 articles were identified.

Work stress and health risk behavior

Cigarette smoking

Altogether 24 cross-sectional or prospective studies published between 1989 and 2006 are included in a review with summarized findings in table 1 (23–46). Most of the investigations are cross-sectional and are focused on the demand–control model. More studies analyze smoking status than smoking intensity. The results with the demand–control model can be summarized as follows: The full model has been confirmed in a minority of studies only (23–26). Single components have only partially been found to be significantly related to smoking status (27–33) and, if so, more often among women than among men. Eight studies did not report any association between model components and smoking status (34–41). Concerning smoking intensity, the evidence is similar. Of the 10 studies exploring smoking intensity, two found full support (34, 42), and three found partial support (41, 43, 44), while findings were negative in five studies (23, 26, 32, 37, 45). The only prospective study on smoking intensity resulted in a negative finding (45). Concerning the effort–reward imbalance model, only two studies have been published, one restricted to a male sample with positive findings on smoking intensity (46) and one exploring smoking status and intensity among men and women and in which the full model was supported only among the women (23).

In conclusion, cigarette smoking is not consistently related to work stress, as measured by the demand–control model. Evidence concerning the effort–reward

Table 1. Work stress [effort–reward imbalance (ERI) model or demand–control (DC) model] and cigarette smoking. In most studies, the full models were tested first (DC, ERI) and the single components were tested thereafter. Results on components are cited only if they provide information in case the full model was not confirmed. [D = high job demands, C = low job control, E = high effort, R = low reward, + = hypothesis confirmed, – = hypothesis not confirmed (refuted or no significant association)]

Study	Dependent variable	Design	Work-stress measure	
			ERI	DC
Peter et al, 1991 (46)	Smoking intensity	Cross-sectional	ERI +	
Kouvonen et al, 2005 (23)	Smoking status	Cross-sectional	ERI +[a]	DC +[a]
	Smoking intensity		ERI +[a]	DC –
Reed et al, 1989 (45)	Smoking intensity	Prospective	..	DC –
Pieper et al, 1989 (27)	Smoking status	Cross-sectional	..	C +[b]
Green et al, 1990 (34)	Smoking status	Cross-sectional	..	DC –
	Smoking intensity			DC +
Netterstrom et al, 1991 (35)	Smoking status	Cross-sectional	..	DC –
Johansson et al, 1991 (36)	Smoking status	Cross-sectional	..	DC –
Alterman et al, 1994 (28)	Smoking status	Cross-sectional	..	C +
Greenlund et al, 1995 (37)	Smoking status	Cross-sectional	..	DC –
	Smoking intensity			DC –
Hellerstedt et al, 1997 (24)	Smoking status	Cross-sectional	..	DC +
Niedhammer et al, 1998 (29)	Smoking status	Cross-sectional	..	D +, C +
Landsbergis et al, 1998 (30)	Smoking status	Prospective	..	C +
Kawakami et al, 1998 (38)	Smoking status	Cross-sectional	..	DC –
Amick et al, 1998 (39)	Smoking status	Cross-sectional	..	DC –
Otten et al, 1999 (43)	Smoking intensity	Cross-sectional	..	C +[b]
Jönsson et al, 1999 (31)	Smoking status	Cross-sectional	..	D +[a], C +[a]
Brisson et al, 2000 (32)	Smoking status	Cross-sectional	..	D +[a]
	Smoking intensity			DC –
Van Loon et al, 2000 (40)	Smoking status	Cross-sectional	..	DC –
Kuper et al, 2003 (44)	Smoking intensity	Cross-sectional	..	D +, C +
Tsutsumi et al, 2003 (41)	Smoking status	Cross-sectional	..	DC –
	Smoking intensity			D +
Lallukka et al, 2004 (25)	Smoking status	Cross-sectional	..	DC +[a]
Lindstrom 2004 (26)	Smoking status	Cross-sectional	..	DC +
	Smoking intensity			DC –
Gun Kang et al, 2005 (33)	Smoking status	Cross-sectional	..	D +
John et al, 2006 (42)	Smoking intensity	Cross-sectional	..	DC +

[a] Females.
[b] Males.

imbalance model is still scarce. Available results are not only limited due to the fact that few prospective investigations have been conducted so far, but also because the dynamics of "smoking careers", including smoking cessation and relapse, have not been explored.

Alcohol consumption

Findings from 18 cross-sectional or prospective studies published between 1989 and 2006 are summarized in table 2. As the measure of alcohol intake varies considerably between studies, their comparability is limited. Again, most of the studies are cross-sectional (25, 28, 29, 37, 39, 40, 44, 47–51) and concern the demand–control model. Six studies are based on longitudinal data (30, 45, 52–55). It is of interest to note that four of the studies found at least partial support for a role of work stress in heavy alcohol consumption or alcohol dependency (52–55), particularly among men. This conclusion holds true for both work-stress models. In contrast, most of the cross-sectional studies failed to find the hypothesized association (29, 37, 39, 40, 48, 49).

Body weight

Altogether 22 reports published between 1989 and 2006 on associations between work stress and body weight are available (table 3). Most of the studies are cross-sectional and concern the demand–control model. The studies vary considerably with regard to the confounders included in the analyses, as well as with regard to measures of weight. Data on weight change were restricted to longitudinal designs, whereas the cross-sectional studies were bound to a comparison of the mean body mass index, the waist-to-hip ratio, or the distribution of overweight or obesity between groups.

With regard to the effort–reward imbalance model, one prospective and one cross-sectional study found full support (56, 57), and one cross-sectional investigation found partial support (58). The demand–control model

Table 2. Work stress [effort–reward imbalance (ERI) model or demand–control (DC) model] and alcohol consumption. Results on components are cited only if they provide information in case the full model was not confirmed. [D = high job demands, C = low job control, E = high effort, R = low reward, + = hypothesis confirmed, – = hypothesis not confirmed (refuted or no significant association)]

Study	Dependent variable	Design	Work stress measure	
			ERI	DC
Puls et al, 1998 (47)	Alcohol consumption (intensity)	Cross-sectional	ERI +	C +
Head et al, 2004 (52)	Incident alcohol dependence (CAGE)	Prospective	ERI + [a]	DC –
Kouvonen et al, 2005 (48)	Alcohol consumption (heavy drinking)	Cross-sectional	ERI –	DC –
Reed et al, 1989 (45)	Alcohol consumption (intensity)	Prospective	..	DC –
Romelsjö et al, 1992 (53)	Severe alcohol problems (hospitalization or mortality)	Longitudinal	..	D +, C +
Alterman et al, 1994 (28)	Alcohol consumption	Cross-sectional	..	D + [a]
Ragland et al, 1995 (49)	Alcohol consumption (high versus low)	Cross-sectional	..	DC –
Greenlund et al, 1995 (37)	Alcohol consumption (intensity)	Cross-sectional	..	DC –
Crum et al, 1995 (54)	Alcohol dependence (DSM III)	Prospective	..	DC + [a]
Hemmingsson et al, 1998 (55)	Alcohol dependence (hospitalization)	Prospective	..	C + [a]
Amick et al, 1998 (39)	Alcohol consumption (intensity)	Cross-sectional	..	DC –
Niedhammer et al, 1998 (29)	Alcohol consumption (high versus low)	Cross-sectional	..	DC –
Landsbergis et al, 1998 (30)	Alcohol consumption (frequency)	Prospective	..	DC –
Roxburgh, 1998 (50)	Alcohol consumption (high versus low)	Cross-sectional	..	D + [b]
San Jose et al, 2000 (51)	Alcohol consumption (heavy drinking)	Cross-sectional	..	D +
van Loon et al, 2000 (40)	Alcohol consumption	Cross-sectional	..	DC –
Lallukka et al, 2004 (25)	Alcohol consumption (Yes versus no)	Cross-sectional	..	D +
	Alcohol consumption (heavy drinking)			DC –
Kuper et al, 2003 (44)	Alcohol consumption (intensity)	Cross-sectional	..	D +

[a] Males.
[b] Females.

Table 3. Work stress (effort–reward imbalance [ERI] model or demand–control [DC] model) and body weight. Results on components are cited only if they provide information in case the full model was not confirmed. [BMI = body mass index, D = high job demands, C = low job control, E = high effort, R = low reward, + = hypothesis confirmed, – = hypothesis not confirmed (refuted or no significant association)]

Study	Dependent variable	Design	Work stress measure	
			ERI	DC
Kivimäki et al, 2002 (56)	BMI	Prospective	ERI +	C +
Ostry et al, 2004 (58)	BMI	Cross-sectional	E + [a]	D + [a]
			R + [b]	
Kouvonen et al, 2005 (57)	BMI	Cross-sectional	ERI + [b]	DC +
Reed et al, 1989 (45)	BMI	Prospective	..	DC –
Netterstrom et al, 1991 (35)	BMI	Cross-sectional	..	DC –
Georges et al, 1992 (64)	Central body fat distribution	Cross-sectional	..	DC –
Theorell et al, 1993 (65)	BMI	Cross-sectional	..	DC –
Hellerstedt et al, 1997 (24)	BMI	Cross-sectional	..	DC + [b]
Wamala et al, 1997 (59)	Obesity (BMI>28.6 [b])	Cross-sectional	..	DC +
Landsbergis et al, 1998 (30)	Overweight (BMI>27.8 [a])	Cross-sectional prospective	..	DC –
	Weight gain			DC –
Amick et al, 1998 (39)	BMI	Cross-sectional	..	DC –
Emdad et al, 1998 (66)	BMI	Cross-sectional	..	DC –
Martikainen et al, 1999 (61)	Weight gain	Prospective	..	C +
Steptoe et al, 1999 (62)	BMI	Cross-sectional	..	C + [b]
Niedhammer et al, 1998 (29)	Overweight (BMI>26.9 [a] / >27.2 [a])	Cross-sectional	..	D + [b]
Jönsson et al, 1999 (31)	BMI	Cross-sectional	..	D + [a]
Brisson et al, 2000 (32)	BMI	Cross-sectional	..	DC –
Kuper et al, 2003 (44)	BMI	Cross-sectional	..	C + [b]
Hannerz et al, 2004 (63)	Weight gain	Prospective	..	D +
	Weight loss			D +
Ishizaki et al, 2004 (67)	BMI	Cross-sectional	..	DC –
	Waist to hip ratio			DC –
Lalluka et al, 2005 (68)	Weight gain	Prospective	..	DC –
Kivimäki et al, 2006 (60)	Weight gain	Prospective	..	DC + [a]
	Weight loss			DC + [a]

[a] Males.
[b] Females.

was fully confirmed in four studies (24, 57, 59, 60) and partially confirmed in eight studies (29, 31, 44, 56, 58, 61–63); in ten investigations there was no association between job strain or single-model components and body weight or weight change, respectively (30, 32, 35, 39, 45, 64–68).

However, these inconclusive findings on the relationship between work stress and body weight may reflect the failure to take into account the possibility that stress may cause both weight gain and weight loss. Weight change is usually analyzed in one direction (ie, weight gain as a function of work stress). This conclusion seems well justified in view of the obesity epidemic in advanced societies (69), but weight loss has also been shown to be a consequence of stressful experience (70). The hypothesis of a bi-directional effect of work stress on weight received recent support from two prospective studies. In both investigations work stress increased the likelihood of weight gain among obese employees, whereas the likelihood of weight loss was higher among employees with a low body mass index (60, 63).

Co-manifestation of health risk behaviors

Epidemiologic evidence indicates that the clustering or co-manifestation of health-risk behaviors increases cardiovascular risk above and beyond the one expected on the basis of the sum of the separate effects (22). Therefore, it is important to know whether and to what extent exposure to work stress is related to the clustering of health-adverse behaviors. A recent publication based on a cross-sectional study of some 36 127 public-sector employees in Finland, showed a dose–response relationship between work stress in terms of effort–reward imbalance and the number of health risk behaviors (figure 1) (71).

Suggestions for future research and conclusions

This review indicates that work-related stress is not consistently associated with the health-adverse lifestyle that continues to be highly prevalent in advanced societies, particularly so among population groups with a low socioeconomic status. The few robust findings point to an association with changing body weight (mostly weight gain, but possibly weight loss as well) and, among men, heavy alcohol consumption. Evidence on a relationship between work stress and cigarette smoking is inconclusive.

Overall, few studies have so far tested the effort–reward imbalance model, while most have been directed towards the demand–control model. Yet, in the few available studies, the former model seems to explain health risk behavior at least as well as the latter model, although only a few reports tested the two models simultaneously.

Limitations of available evidence concern a relative paucity of longitudinal research and a lack of intervention studies. In addition, health risk behavior was assessed in relatively crude ways, neglecting the dynamics over time (cessation; relapse) and, with few exceptions, their clustering and their bi-directional association with work stress.

As information on a mediating role of health risk behavior in the association between work stress and cardiovascular or other disease outcomes from prospective studies is scarce, the relative contribution of the two pathways mentioned earlier (ie, of direct versus indirect effects of work stress on health) cannot be further clarified.

The first suggestion for future research therefore concerns a rigorous test of these two pathways in available and newly designed epidemiologic investigations. Importantly, changes in health risk behavior should be monitored as should changes in exposure to work stress over time. As is well known from birth cohort studies (72, 73), childhood and adolescence are important stages in the formation of health-risk behavior. Therefore, the contribution of work stress in adult life towards maintaining, intensifying, or quitting these types of behavior deserves attention in a life course perspective.

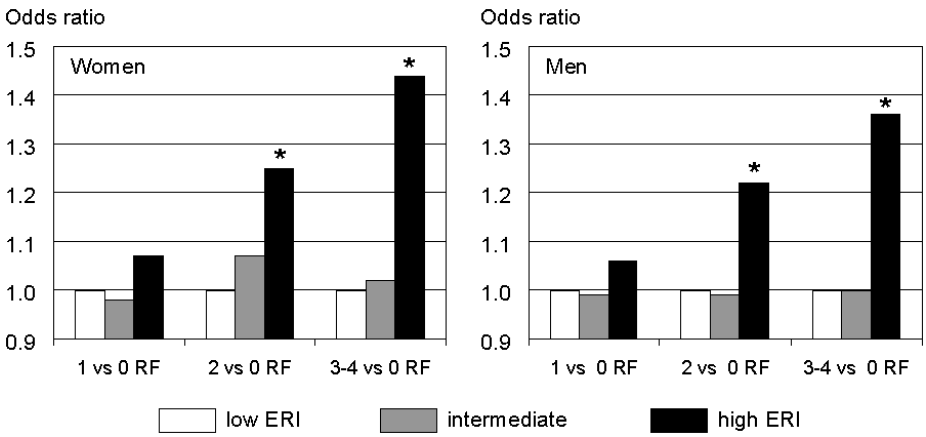

Figure 1. Work stress [effort–reward imbalance (ERI) model] and co-manifestation of health risk behavior (current smoker, body mass index ≥25 kg/m^2, physically inactive, and heavy drinker) among 36 127 public-sector employees in Finland. The results are based on the report of Kouvonen et al (71). (* P<0.05, RF = number of risk factors)

As a second suggestion, monitoring studies are required that assess the circumstances, attitudes, and emotions associated with health risk behavior more intensely. Respective designs should include the registration of the activity of the autonomic nervous system, of markers of hormonal activity, and of immune competence. Third, basic neuroscience research is promising in advancing our understanding of the role of the brain reward system in stress and addictive behavior. For instance, the "cascade theory of reward" suggests that prolonged stress provokes neuroregulatory dysfunction in dopamine release in mesolimbic cortical structures (74). This pathway, with inputs from the prefrontal cortex, is implicated in reinforcement and reward expectancy, as well as in addictive behavior (75).

In a recent experimental study using functional magnetic resonance imaging with reward-based paradigms, hyperactivations in the medial prefrontal, anterior cingulate, and dorsolateral prefrontal cortex were observed in a group of healthy adults with high susceptibility to reward frustration as compared with a group with low susceptibility. Susceptibility to reward frustration was measured by scores on the scales assessing effort–reward imbalance at work (76). The findings suggested that exposure to chronic frustration after effort compromises the adaptive responsiveness of parts of the cortical system to received or omitted rewards. The extent to which such compromised responsiveness contributes to an intensification of bodily stress reactions or to addictive tendencies is yet unknown.

A fourth suggestion concerns the design and realization of intervention studies in which theory-based measures of organizational and personnel development aim at reducing stressful experience at work. If the health risk behavior of the participants of such interventions would diminish as a function of respective measures, the findings would strengthen the ecological validity of experimental and epidemiologic evidence.

In conclusion, this review suggests that at least some part of the burden of disease attributable to a health-adverse psychosocial work environment is explained by an unhealthy lifestyle, in particular heavy alcohol consumption, overweight, and cigarette smoking. Moreover, work stress increases the probability of co-manifestation for several risk factors for individuals, predisposing them to chronic disease development. Findings support measures of a health-promoting lifestyle as part of worksite intervention programs. However, one should be cautious in regarding expected effects for at least two reasons. First, health risk behavior is acquired through socialization as part of a lifestyle at earlier stages in the life course (73). As they are embedded in, and reinforced by, people's everyday social environment, it may be difficult to induce and maintain behavioral change. Second, in addition to indirect effects, work stress affects health in the long run by direct effects via sustained activation of the autonomic nervous system and enhanced neuroendocrine responses (77). Intervention efforts therefore should be directed towards structural measures of task redesign, organizational justice, and promotion prospects, including job security, in order to reduce the sources of stressful experience. These measures are further enhanced by investments in improved leadership and improved skills of coping with demands, threats, and inadequate rewards at work.

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