

REVIEW ARTICLE

Occupational stress as a modifiable risk factor for atherosclerotic coronary artery disease: Evidence, mechanisms, and interventions

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Abstract

Atherosclerotic coronary artery disease continues to represent a major contributor to worldwide morbidity and mortality. Although conventional cardiovascular risk factors such as hypertension, dyslipidemia, diabetes mellitus, obesity, smoking, and sedentary lifestyle remain central to cardiovascular prevention, increasing attention has been directed toward psychosocial determinants, particularly occupational stress. Work-related stress, commonly conceptualized through the job strain and effort–reward imbalance models, has been associated with a higher incidence of coronary events, although the magnitude and mechanisms of this relationship remain incompletely understood. This review synthesizes current evidence on the relationship between occupational stress and coronary artery disease, drawing on major epidemiological studies and meta-analyses. Persistent occupational stress contributes to cardiovascular dysfunction through multiple interacting mechanisms. These include activation of the hypothalamic–pituitary–adrenal axis and sympathetic nervous system, promotion of systemic inflammatory activity, oxidative stress, autonomic imbalance, and unfavorable behavioral adaptations (e.g., smoking, unhealthy dietary habits, physical inactivity, and excessive alcohol intake). Collectively, these alterations may accelerate endothelial dysfunction, thrombogenicity, and atherosclerotic progression. Key studies, including INTERHEART and meta-analyses by Kivimäki et al., along with prospective data from Chandola et al. on metabolic syndrome, support both independent and cumulative effects of occupational stress on cardiovascular risk. Although its attributable risk is lower than that of traditional risk factors, occupational stress is modifiable, offering significant preventive potential. Intervention strategies include individual approaches (e.g., stress management, lifestyle changes, and psychological support), organizational measures (e.g., improving job control, reducing demands, and enhancing workplace support), and public health policies (e.g., occupational health programs and stress screening). Recognizing occupational stress as a modifiable and clinically relevant contributor to cardiovascular risk has important implications for prevention strategies and public health policies. Future research should prioritize longitudinal studies, biomarker identification, and the evaluation of cost-effective interventions.

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Citation: Popescu D-C, Diaconu M, Nechita A-C. Occupational stress as a modifiable risk factor for atherosclerotic coronary artery disease: Evidence, mechanisms, and interventions. *Eurasian J Med Oncol*.
doi: 10.36922/EJMO026130147

Received: March 27, 2026

Revised: June 1, 2026

Accepted: June 11, 2026

Published online: July 6, 2026

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Keywords: Occupational stress; Psychosocial factors; Coronary artery disease; Atherosclerosis; Cardiovascular risk; Job strain; Effort–reward imbalance; Inflammation

1. Introduction

Atherosclerotic coronary artery disease (CAD) remains one of the principal causes of morbidity and mortality worldwide. Traditionally, cardiovascular prevention strategies have focused on established risk factors such as hypertension, dyslipidemia, diabetes mellitus, smoking, obesity, and sedentary lifestyle. Nevertheless, growing scientific evidence suggests that psychosocial determinants, including occupational stress, may substantially contribute to cardiovascular risk and disease progression.¹

Socioprofessional stress—defined as stress experienced in the workplace and within associated social interactions—can be conceptualized in several forms: (i) job strain, described as the combination of high job demands and low decision latitude; (ii) effort–reward imbalance, referring to situations in which the effort invested at work is not matched by adequate rewards; and (iii) low social support, characterized by insufficient emotional, informational, or instrumental support from colleagues or supervisors.^{2–4}

Numerous epidemiological studies and meta-analyses have demonstrated a significant association between exposure to these forms of stress and an increased risk of myocardial infarction, angina pectoris, and other clinical manifestations of CAD.^{5–7} The mechanisms involved encompass both neuroendocrine responses—particularly activation of the hypothalamic–pituitary–adrenal (HPA) axis—and unhealthy behaviors that may arise as a consequence of chronic stress, such as smoking, excessive alcohol consumption, physical inactivity, and overeating.

Emerging evidence also suggests that the cardiovascular effects of occupational stress may differ significantly according to sex and gender. Women appear to exhibit greater HPA axis reactivity and heightened susceptibility to stress-induced coronary dysfunction, including microvascular ischemia and demand–supply mismatch ischemia, whereas gender-related occupational and societal disparities may further modulate stress exposure and cardiovascular vulnerability.

This review aims to provide an in-depth analysis of the existing literature investigating the role of socioprofessional stress in the pathogenesis, progression, and prognosis of atherosclerotic CAD. By presenting and discussing the principal studies and meta-analyses in this field, this review highlights the clinical and public health implications of occupational stress, as well as potential directions for intervention and prevention.

2. Methodology

This study was designed as a narrative review aimed at synthesizing current evidence regarding the relationship

between socioprofessional stress and atherosclerotic CAD. For the purpose of this review, a manual literature search was conducted between January 2024 and March 2025 across major scientific databases, including PubMed, ScienceDirect, and Google Scholar. The search strategy employed combinations of the following keywords: occupational stress, socioprofessional stress, job strain, effort–reward imbalance, coronary artery disease, atherosclerosis, psychosocial factors, and cardiovascular risk.

Both original research articles (cohort and case–control studies) and secondary sources, including meta-analyses and narrative or systematic reviews, were considered, provided they specifically assessed the association between work-related stress and the risk of atherosclerotic CAD.

The inclusion criteria comprised publication in internationally recognized peer-reviewed journals (e.g., *The Lancet*, *BMJ*, *Circulation*, *European Heart Journal*, and *Psychosomatic Medicine*); the use of representative study populations and/or rigorous methodologies for assessing occupational stress and cardiovascular status; and a clear definition of a significant association between socioprofessional stress and CAD.

Studies lacking quantitative data or focusing exclusively on other forms of stress (e.g., posttraumatic or family-related stress) without incorporating an occupational dimension were excluded. Priority was given to studies considered clinically relevant, methodologically robust, or highly influential in the field of psychosocial cardiovascular risk research.

Given the narrative nature of the review, the Preferred Reporting Items for Systematic Reviews and Meta-Analyses methodology was not applied. Instead, the final body of evidence was subjected to a thematic qualitative synthesis, with particular emphasis on biological and behavioral mechanisms, epidemiological evidence, biomarkers of chronic stress, sex-related differences in stress response, and implications for clinical practice and public health policies.

Generative artificial intelligence tools (ChatGPT, version GPT-5.1, OpenAI, United States) were used exclusively for minor linguistic adjustments intended to improve text clarity and readability. No artificial intelligence tools were used for data extraction, data interpretation, or reference generation.

3. Traditional cardiovascular risk factors: Setting the context for occupational stress

To place the role of socioprofessional stress into proper context, it is useful to briefly review the classical risk factors

for atherosclerotic CAD, which have been consistently confirmed by numerous clinical trials and epidemiological studies. These include:

- (i) Arterial hypertension, where increased arterial pressure accelerates atherosclerosis and raises myocardial workload.
- (ii) Dyslipidemia, characterized by elevated plasma levels of low-density lipoprotein cholesterol and triglycerides, which promote atherogenesis.
- (iii) Smoking, whose toxic constituents cause endothelial injury and increase the risk of atherothrombosis.
- (iv) Obesity, associated with insulin resistance, dyslipidemia, and hypertension.
- (v) Diabetes mellitus, where chronic hyperglycemia leads to endothelial dysfunction and impaired lipid metabolism.
- (vi) Physical inactivity, which compromises cardiovascular function and facilitates weight gain.

In addition to these well-established factors, psychosocial determinants have gained increasing recognition, among which socioprofessional stress plays a significant contributory role in cardiovascular risk modulation.¹

4. Individual factors and susceptibility to stress

Although socioprofessional stress is a well-documented risk factor for atherosclerotic CAD, not all individuals respond uniformly to chronic stress exposure. This interindividual variability can be explained by a complex interplay of genetic, psychological, and socioeconomic factors.

4.1. Genetic factors

Several studies have identified genetic polymorphisms that may influence neuroendocrine stress responses. For instance, polymorphisms in the glucocorticoid receptor gene (*NR3C1*) can modulate sensitivity to cortisol, thereby affecting HPA axis reactivity. Similarly, functional variants of the serotonin transporter gene (*5HTTLPR*) have been associated with heightened stress reactivity and an increased risk of depression and anxiety—conditions that may amplify the adverse cardiovascular effects of chronic stress exposure.⁸

4.2. Psychological factors

Personality traits and coping mechanisms play a pivotal role in determining how stress impacts cardiovascular health. Type A personality characteristics—such as excessive competitiveness, time urgency, and hostility—have been associated with an increased risk of CAD. In

contrast, individuals exhibiting psychological resilience demonstrate a greater capacity to adapt to stress through effective coping strategies, optimism, and strong social support networks, resulting in a lower cardiovascular risk. Affective disorders, particularly depression and anxiety, which frequently coexist with occupational stress, have been independently linked to autonomic dysfunction and chronic low-grade inflammation, both of which contribute to cardiovascular disease development.⁹

4.3. Socioeconomic factors

Individuals with lower socioeconomic status and educational attainment are disproportionately exposed to cardiovascular risk through multiple pathways. These include employment in occupations characterized by high demands and low control, limited access to healthcare services and cardiovascular screening, and increased exposure to additional stressors such as financial insecurity and reduced social support. Socioeconomic disadvantage has been consistently associated with poorer cardiovascular outcomes and heightened vulnerability to stress-related disease.¹⁰

4.4. Sex- and gender-related differences in stress susceptibility

Growing evidence indicates that biological sex and gender-related social determinants substantially influence both exposure to occupational stress and cardiovascular responses to chronic stress. Women are disproportionately represented in occupations characterized by high emotional demands, reduced decision latitude, and effort–reward imbalance, particularly in healthcare, education, and caregiving professions. In addition, gender-related societal expectations, unpaid domestic responsibilities, and workplace discrimination may amplify chronic psychosocial stress exposure in women.

Experimental and clinical studies have demonstrated sex-specific neuroendocrine responses to stress. Women appear to exhibit greater HPA axis activation and enhanced cortisol and catecholamine responses following psychological stressors, whereas men more frequently demonstrate hemodynamic responses characterized predominantly by blood pressure elevation. These differences may contribute to distinct cardiovascular phenotypes associated with stress exposure.

Importantly, stress-related cardiovascular events in women are more commonly associated with coronary microvascular dysfunction, endothelial dysfunction, vasospasm, and myocardial oxygen supply–demand mismatch rather than plaque rupture alone. Acute emotional or physical stress may therefore precipitate

ischemia in the absence of obstructive coronary disease, contributing to conditions such as microvascular angina, stress-induced cardiomyopathy, and type 2 myocardial infarction.

Recent evidence further suggests that sex-related biological mechanisms and gender-associated psychosocial factors interact synergistically, influencing both stress perception and cardiovascular vulnerability. These observations underscore the importance of incorporating sex-stratified analyses into future occupational stress research and cardiovascular prevention strategies.¹¹

5. Definition and conceptualization of socioprofessional stress

5.1. The demand–control model

The demand–control model introduced by Karasek¹² conceptualizes occupational stress as the result of an imbalance between workplace demands and the degree of autonomy available to employees. According to this framework, psychological strain becomes particularly significant when high professional demands coexist with limited decision-making authority. The model evaluates two principal dimensions: (i) psychological job demands, including workload intensity and time pressure, and (ii) decision latitude, referring to employees' capacity to influence how and when work-related tasks are performed. The interaction between these dimensions determines different occupational stress profiles and their potential health consequences.

According to the interaction between job demands and decision latitude, four major occupational profiles can be identified:

- (i) High-strain occupations, characterized by elevated demands combined with limited control, are associated with increased psychological burden and higher cardiovascular risk.
- (ii) Active occupations involve high demands together with substantial autonomy and may stimulate professional development and engagement.
- (iii) Passive occupations combine low demands with reduced control and are frequently associated with decreased motivation and reduced professional satisfaction.
- (iv) Low-strain occupations, characterized by lower demands and greater autonomy, are generally considered the most favorable from a health perspective.

The original framework was subsequently extended

through the inclusion of workplace social support, leading to the development of the demand–control–support model. This expanded perspective emphasizes that supportive professional relationships and effective supervisory support may attenuate the adverse effects associated with excessive demands and limited autonomy. Consequently, occupational stress is now understood not only as a function of workload and control, but also of the quality of interpersonal interactions within the work environment.¹²

5.2. The effort–reward imbalance model

The effort–reward imbalance model proposed by Siegrist¹³ describes occupational stress as a consequence of disproportionate reciprocity between professional effort and the rewards received in return. Such rewards may include financial compensation, professional recognition, career advancement opportunities, and long-term job security.

Central components of the effort–reward imbalance model framework include the imbalance between intense professional effort and insufficient occupational reward, together with the concept of overcommitment, which reflects excessive personal involvement in work-related activities despite inadequate recognition or compensation. Persistent exposure to these conditions may amplify physiological stress responses and contribute to adverse cardiovascular outcomes.

Evidence summarized within the effort–reward imbalance framework indicates that sustained imbalance between high occupational effort and insufficient reward may contribute to prolonged psychophysiological stress responses, adverse behavioral adaptations, and poorer cardiovascular health. This model is therefore considered complementary to Karasek's demand–control framework, because it emphasizes reciprocity, fairness, and perceived imbalance between occupational effort and reward rather than job autonomy alone.¹³

5.3. Social support

Social support, both in the workplace (e.g., from colleagues and supervisors) and in personal life (e.g., from family and friends), represents an important buffering factor against stress. Adequate social support can attenuate perceived stress and enhance coping capacity, whereas its absence may intensify stress perception and reduce resilience. Low social support has been associated with increased vulnerability to cardiovascular disease and poorer health outcomes, underscoring its relevance as a key psychosocial determinant in occupational health.⁴

6. Pathophysiological mechanisms linking stress to atherogenesis

6.1. Neuroendocrine activation and inflammatory response

Sustained occupational stress induces prolonged activation of both the HPA axis and the sympathetic nervous system. Persistent elevation of cortisol and catecholamine levels contributes to hemodynamic overload, autonomic dysregulation, endothelial injury, and amplification of inflammatory signaling pathways. Increased production of proinflammatory mediators such as interleukins and tumor necrosis factor- α may further accelerate atherosclerotic processes and vascular dysfunction.¹⁴

6.2. Interaction with behavioral risk factors

Chronic psychosocial stress is frequently associated with maladaptive behavioral responses that may further aggravate cardiovascular risk. Individuals experiencing persistent occupational strain often demonstrate higher rates of tobacco use, alcohol consumption, unhealthy dietary behaviors, a sedentary lifestyle, and reduced adherence to preventive measures. These behavioral patterns may act synergistically with biological stress responses in promoting cardiovascular disease progression.²

6.3. Endothelial dysfunction and hypercoagulability

Chronic stress may directly impair endothelial function through increased oxidative stress and reduced bioavailability of nitric oxide, a critical mediator of vascular homeostasis. In parallel, elevated levels of catecholamines and cortisol promote prothrombotic states, characterized by increased fibrinogen levels and enhanced platelet aggregation. These alterations contribute to endothelial injury, plaque instability, and a heightened risk of acute coronary events, thereby linking stress-related neuroendocrine dysregulation to clinically relevant cardiovascular outcomes.²

7. Markers of chronic stress and cardiovascular risk

Beyond classical cardiovascular biomarkers such as NT-proBNP and C-reactive protein, emerging markers of chronic stress provide deeper insight into the underlying pathophysiological mechanisms linking stress exposure to cardiovascular disease.

7.1. Inflammatory biomarkers

Long-term exposure to psychosocial stress has been associated with persistent low-grade inflammatory activation. Elevated circulating concentrations of

inflammatory mediators such as interleukin-6 and tumor necrosis factor- α have been observed in individuals exposed to chronic stress conditions. In addition, C-reactive protein remains an established biomarker used for cardiovascular risk stratification and reflects the inflammatory component of stress-related cardiovascular dysfunction.¹⁵

7.2. Markers of neuroendocrine activation

Markers of neuroendocrine stress responses offer valuable information regarding HPA axis and sympathetic nervous system activation. Salivary and plasma cortisol levels are commonly used indicators of HPA axis activity, with persistently elevated values reflecting chronic stress exposure. Catecholamines (e.g., adrenaline and noradrenaline) are closely linked to sympathetic overactivation and hemodynamic stress. In addition, neuropeptide Y has emerged as a biomarker associated with maladaptive stress responses, vasoconstriction, and the development of arterial hypertension. Collectively, these markers capture the neuroendocrine dimension of stress-related cardiovascular risk.¹⁴

7.3. Epigenetic biomarkers

Stress-induced epigenetic modifications may exert long-term effects on cardiovascular health. DNA methylation of the *NR3C1* gene, which encodes the glucocorticoid receptor, can alter cortisol sensitivity and disrupt feedback regulation of the HPA axis. Furthermore, altered expression of specific microRNAs—such as miR-146a and miR-21—has been implicated in vascular inflammation, immune dysregulation, and the progression of atherosclerosis. These epigenetic changes provide a potential mechanistic link between chronic stress exposure and sustained cardiovascular vulnerability.⁸

7.4. Heart rate variability reactivity to stress

A substantial body of evidence has demonstrated significant alterations in heart rate variability (HRV) parameters in response to experimentally induced and real-life stress. Most studies report a reduction in high-frequency components of HRV, indicating decreased parasympathetic activity, alongside an increase in low-frequency components, reflecting sympathetic activation. Stress induction protocols have included cognitively demanding tasks, exposure to noise, and socially stressful situations, with consistent HRV responses observed across different methodologies.

Both time-domain measures (e.g., Standard Deviation of Normal-to-Normal intervals and Root Mean Square of Successive Differences) and frequency-domain indices (e.g., high- and low-frequency power) have shown

sensitivity to stress-related autonomic changes, offering a comprehensive assessment of autonomic nervous system dynamics. These stress-induced HRV alterations underscore a shift toward sympathetic dominance and parasympathetic withdrawal, supporting the utility of HRV as a noninvasive biomarker for stress assessment and monitoring in both clinical and research settings.¹⁶

8. Epidemiological and clinical evidence

8.1. Meta-analysis of the 2012 study by Kivimäki *et al.*

In the 2012 meta-analysis published in *The Lancet* by Kivimäki *et al.*⁷, the association between occupational stress and the risk of coronary heart disease was investigated using individual participant data. The study pooled data from 13 European cohort studies, comprising a total of 197,473 participants free of coronary heart disease at baseline. Among these, approximately 15% reported exposure to job strain.

Over a mean follow-up period of 7.5 years, a total of 2,358 major coronary events were documented. After adjustment for age and sex, individuals exposed to occupational stress exhibited a 23% higher risk of developing coronary heart disease compared with unexposed individuals (hazard ratio = 1.23; 95% confidence interval = 1.10–1.37). Importantly, the association remained statistically significant after excluding coronary events occurring within the first three or five years of follow-up, suggesting that the findings were not driven by reverse causality.

The relationship between job strain and coronary heart disease was consistent across multiple subgroups, including sex, age categories, and socioeconomic strata. The population-attributable fraction of coronary heart disease related to occupational stress was estimated at approximately 3.4%, indicating a modest but measurable impact at the population level. However, Kivimäki *et al.*⁷ reported that these results may not be directly generalizable beyond European populations. They concluded that although reducing occupational stress could contribute to lowering the incidence of coronary heart disease, the expected impact would likely be smaller than that achieved by targeting traditional risk factors such as smoking or hypertension.

8.2. Recent developments and post-pandemic occupational stress

More recent evidence has further strengthened the association between occupational stress and adverse cardiovascular outcomes, particularly in the context of evolving workplace dynamics following the COVID-19

pandemic. The rapid expansion of remote and hybrid work models has introduced new psychosocial stressors, including social isolation, blurred work–life boundaries, increased digital workload, and reduced organizational support.

Recent studies suggest that prolonged occupational stress in post-pandemic work environments may contribute to sustained autonomic imbalance, sleep disturbances, depressive symptoms, and unhealthy lifestyle behaviors, all of which may adversely affect cardiovascular health. These findings have reinforced the growing recognition of psychosocial stress within contemporary cardiovascular prevention frameworks, including the 2021 European Society of Cardiology Guidelines on cardiovascular disease prevention.¹⁷

8.3. Chronic work-related stress and metabolic syndrome: Evidence from Chandola *et al.*

In a prospective study published in *The British Medical Journal* in 2006, Chandola *et al.*² examined the relationship between chronic work-related stress and the development of metabolic syndrome. The study included 10,308 British civil servants aged 35–55 years who were followed for a mean duration of 14 years as part of the Whitehall II cohort.

Participants who were frequently exposed to occupational stress had more than a two-fold higher risk of developing metabolic syndrome compared with those without job strain (age- and employment-grade-adjusted odds ratio = 2.25; 95% confidence interval = 1.31–3.85). This association remained significant after adjustment for unhealthy behaviors, including smoking, poor diet, and physical inactivity.

A clear social gradient in the prevalence of metabolic syndrome was observed, with employees in lower occupational grades exhibiting a higher risk. Occupational stress partially explained this gradient, suggesting a mediating role between socioeconomic position and metabolic risk. These findings underscore chronic work-related stress as an independent and clinically relevant risk factor for metabolic syndrome, with important implications for cardiovascular prevention strategies.

8.4. The INTERHEART study: Evidence from Rosengren *et al.*

The INTERHEART study, published in *The Lancet* in 2004 by Rosengren *et al.*³, evaluated the association between psychosocial risk factors and the risk of acute myocardial infarction. This large international case–control study included 11,119 patients with a first acute myocardial infarction and 13,648 control subjects from 52 countries,

representing diverse geographic regions and ethnic backgrounds.

Participants were assessed for work-related stress, home stress, financial stress, and major life events during the preceding year. Depression and perceived control over life circumstances were also evaluated. Frequent work-related stress was reported in 23% cases compared with 17.9% of controls, while permanent work stress was reported in 10% of cases versus 5% of controls. After adjustment for age, sex, geographic region, and smoking, work-related stress was associated with a significantly increased risk of myocardial infarction.

Similarly, frequent home-related stress (11.6% vs. 8.6%) and permanent domestic stress (3.5% vs. 1.9%) were more prevalent among cases than controls. Overall stress—whether occupational, domestic, or both—was associated with a higher risk of myocardial infarction, particularly among individuals reporting persistent stress exposure. Severe financial stress and major life events were also more common among cases and independently associated with increased risk.

Depression was reported by 24% of patients compared with 17.6% of controls, further supporting its role as a psychosocial risk factor for myocardial infarction. The authors concluded that psychosocial stressors are consistently associated with acute myocardial infarction across populations worldwide.

8.5. The role of social support: Evidence from Orth-Gomér *et al.*

In a study published in *Psychosomatic Medicine* in 1993, Orth-Gomér *et al.*⁴ investigated the relationship between social support and the incidence of coronary heart disease in middle-aged Swedish men. The study included a random sample of 736 men aged 50 years, all born in 1933 and residing in Gothenburg, Sweden. Participants were followed for six years to assess the incidence of myocardial infarction and coronary heart disease-related mortality.

Social support was evaluated across two dimensions: emotional attachment, reflecting support from close personal relationships, and social integration, representing support from a broader social network. Lower levels of emotional attachment were observed among men who developed coronary heart disease, with a near-significant association ($p = 0.07$). In contrast, reduced social integration was significantly associated with incident coronary heart disease ($p = 0.04$). Multivariable analyses demonstrated that both dimensions of social support remained independent predictors of new coronary events, alongside smoking. These findings highlight the importance of social relationships as psychosocial

determinants of cardiovascular health.

8.6. Occupational restructuring and depressive symptoms: Evidence from Netterstrøm *et al.*

In a study published in the *Scandinavian Journal of Work, Environment & Health*, Netterstrøm *et al.*¹⁸ examined the development of depressive symptoms and the incidence of depression among Danish civil servants exposed to major organizational restructuring. No significant differences in depressive symptoms or incident depression were observed between employees affected by mergers or job changes and control subjects. These findings suggest that not all forms of occupational stress uniformly translate into adverse mental health outcomes, highlighting the heterogeneity of stress-related health effects.

8.7. Job strain and body mass index: Evidence from Nyberg *et al.*

In a pooled analysis published in *PLoS ONE*, Nyberg *et al.*⁶ examined the association between job strain and body mass index in a large European population. Job strain was associated with both underweight and obesity, demonstrating a U-shaped relationship between occupational stress and body mass index. Longitudinal analyses further showed that both weight gain and weight loss were linked to the onset of job strain over time. However, these associations were modest in magnitude, suggesting a limited impact at the population level. Overall, these findings indicate that occupational stress may be related to alterations in body weight, but its contribution to cardiovascular risk through obesity appears relatively small and complex.

9. Practical implications and multi-level intervention strategies

9.1. Assessment of psychosocial risk in clinical practice

In light of the available evidence, the integration of occupational stress assessment into routine clinical practice is recommended, particularly in patients with established cardiovascular risk factors. The use of standardized questionnaires, such as the Job Content Questionnaire and the Effort–Reward Imbalance Questionnaire, may facilitate the identification of individuals at increased psychosocial risk. Incorporating psychosocial risk evaluation into cardiovascular risk stratification may improve the detection of vulnerable patients and support a more comprehensive, patient-centered preventive approach.^{3,13}

Structured occupational stress screening may be facilitated through integrated multidisciplinary cardiovascular prevention programs involving

cardiologists, psychologists, occupational medicine specialists, rehabilitation specialists, and primary care physicians. Repeated psychosocial assessment during follow-up may help identify persistent stress exposure and improve long-term adherence to preventive strategies.

Emerging digital health technologies may further enhance stress monitoring and cardiovascular prevention. Mobile health applications, wearable devices, smartwatches, and telemedicine platforms may allow continuous assessment of HRV, physical activity, sleep quality, blood pressure, and behavioral adherence, thereby supporting earlier identification of high-risk individuals and improving continuity of care.¹⁹⁻²¹

9.2. Individual-level interventions

Interventions targeting stress reduction at the individual level may include behavioral therapy, coping-skills training, mindfulness-based approaches, relaxation techniques, and structured psychological counseling. Lifestyle optimization—including regular physical activity, smoking cessation, healthy dietary habits, and moderation of alcohol intake—remains a central component of cardiovascular prevention in individuals exposed to chronic occupational stress.

Social support also plays a critical role by encouraging the development of supportive networks involving family, friends, and colleagues, as well as participation in activities that enhance social cohesion and emotional well-being. These interventions have been shown to mitigate stress-related autonomic and inflammatory responses, thereby contributing to cardiovascular risk reduction.^{4,9}

Digital-health-supported interventions may additionally improve patient engagement and lifestyle modification. Remote counseling programs, smartphone applications promoting physical activity and stress reduction, wearable-based rehabilitation monitoring, and telemedicine-assisted follow-up have shown promising effects on adherence, cardiovascular rehabilitation participation, and behavioral risk factor control in high cardiovascular-risk populations.¹⁹⁻²¹

9.3. Organizational-level interventions

At the organizational level, balanced work design is fundamental and may be achieved through measures such as reducing excessive workload, establishing clear and realistic objectives, providing constructive feedback, and ensuring rewards that are proportional to the effort invested. Increasing employees' control over daily decision-making processes and actively involving them in project planning and management can further attenuate job strain.

Fostering a culture of social support within the workplace is equally important. This can be accomplished by strengthening collegial relationships through team-building activities, as well as by offering opportunities for mentorship and access to psychological counseling services. Organizational interventions addressing both structural and relational aspects of work have the potential to substantially reduce occupational stress and its adverse health consequences.^{7,9}

9.4. Public health implications

At the population level, recognizing socioprofessional stress as a modifiable risk factor for atherosclerotic CAD justifies targeted investments in occupational health policies. These may include regulations governing working hours and mandatory rest periods, legislative measures aimed at preventing workplace harassment and ensuring safe working conditions, and the development of national programs dedicated to stress screening and counseling in high-demand occupational sectors.

Such public health initiatives align with contemporary cardiovascular prevention frameworks and emphasize the need for multisectoral collaboration to address psychosocial determinants of cardiovascular disease.^{1,3}

10. Comparative perspectives and emerging approaches

10.1. Individual versus organizational interventions

While the previous section focused primarily on practical intervention strategies, the following section provides a comparative and critical perspective regarding the relative effectiveness, limitations, and emerging directions of occupational stress interventions in cardiovascular prevention.

A key aspect of reducing occupational stress is in the comparative evaluation of intervention strategies. Individual-level interventions—such as relaxation techniques, mindfulness-based approaches, and psychological counseling—may improve the psychophysiological stress responses and enhance individual coping capacity. However, their effectiveness tends to be limited when the underlying work environment remains persistently stressful.

In contrast, organizational-level interventions—including workload reduction, increasing job autonomy, and promoting a supportive work environment—have been shown to exert a more substantial and sustained impact on long-term health outcomes. These strategies address the structural determinants of occupational stress and are therefore more likely to produce durable reductions

in cardiovascular risk.^{1,5}

10.2. The role of pharmacological interventions

In selected patients with severe chronic stress, depression, anxiety, or high cardiovascular risk, pharmacological therapies may represent adjunctive strategies within a broader multidisciplinary approach. Beta-blockers may attenuate sympathetic nervous system hyperactivation and reduce the hemodynamic effects of stress exposure, particularly in individuals with concomitant hypertension, tachyarrhythmias, or established CAD.

Selective serotonin reuptake inhibitors have demonstrated beneficial effects in patients with depression and cardiovascular disease, particularly through improvement of depressive symptoms, autonomic regulation, and treatment adherence. Landmark trials such as SADHART and ENRICH highlighted the importance of addressing depression in patients with CAD, although the impact on hard cardiovascular outcomes remains variable.^{22,23}

In addition, statins may exert pleiotropic anti-inflammatory and endothelial-protective effects beyond lipid lowering, potentially counteracting some of the vascular consequences of chronic psychosocial stress.²⁴ Nevertheless, pharmacological treatment should not be regarded as a substitute for addressing the psychosocial and organizational determinants of occupational stress, but rather as part of a comprehensive cardiovascular prevention strategy.

10.3. International models of successful intervention

Several countries have implemented occupational health policies that have demonstrated tangible benefits in stress reduction and employee well-being. For example, Sweden and the Netherlands have adopted progressive labor and occupational health policies, including reductions in working hours, increased professional autonomy, and the implementation of mandatory workplace wellness programs.

In addition, the formal recognition of burnout as an occupational or medical condition in certain healthcare systems has facilitated earlier diagnosis, improved access to support services, and greater institutional accountability. These international experiences highlight the potential effectiveness of policy-level interventions and underscore the importance of integrating occupational health into broader public health and cardiovascular prevention strategies.^{5,6}

11. Future research directions

In light of the convergent findings of the studies analyzed, it has become increasingly evident that socioprofessional stress supports the view that socioprofessional stress is a modifiable risk factor for atherosclerotic CAD. This relationship appears to be partially mediated by traditional cardiovascular risk factors—such as arterial hypertension, dyslipidemia, and obesity—while a growing body of research highlights direct effects of stress on vascular endothelial function, inflammatory pathways, and metabolic regulation.

Despite this progress, important knowledge gaps persist. Methodological heterogeneity across studies—particularly with regard to the subjective assessment of stress exposure—continues to limit the comparability of results and the strength of causal inference. Consequently, there is a pressing need for large-scale longitudinal studies designed to monitor the temporal evolution of stress-related inflammatory biomarkers and metabolic intermediates, as well as to evaluate the cost-effectiveness of occupational stress-reduction interventions implemented at the population level and their impact on cardiovascular morbidity and mortality.

Future research should prioritize large-scale longitudinal studies integrating sex-stratified analyses, particularly given the growing evidence of sex- and gender-related differences in stress perception, neuroendocrine activation, endothelial dysfunction, and susceptibility to stress-induced ischemia. Additional investigation is also warranted regarding the use of wearable technologies and HRV-based monitoring systems for real-time occupational stress assessment.

The integration of telemedicine, mobile health applications, and wearable monitoring systems into cardiovascular prevention programs may further improve stress-related risk stratification, long-term follow-up, and adherence to lifestyle interventions. These technologies may be particularly valuable for individuals exposed to chronic occupational stress by enabling continuous monitoring, personalized feedback, and remote multidisciplinary support.

In parallel, the transition toward remote and hybrid work environments following the COVID-19 pandemic has fundamentally altered occupational stress patterns. Although remote work may reduce commuting-related stress and improve flexibility in certain populations, it may also increase social isolation, digital fatigue, sedentary behavior, and difficulties in separating professional and

personal life. The long-term cardiovascular implications of these evolving occupational models remain insufficiently understood and represent an important emerging research domain. Future studies should additionally evaluate the cost-effectiveness of occupational stress reduction strategies and their long-term impact on cardiovascular morbidity and mortality.

12. Conclusion

This narrative review highlights the growing evidence supporting occupational stress as a clinically relevant contributor to atherosclerotic CAD. The relationship appears to involve complex interactions between neuroendocrine activation, inflammatory dysregulation, endothelial dysfunction, and adverse behavioral adaptations. Available epidemiological evidence suggests that chronic work-related stress may independently contribute to cardiovascular morbidity, although its attributable risk remains lower than that associated with major traditional cardiovascular risk factors.

Accordingly, the implementation of effective stress prevention and management strategies may play a crucial role in reducing the overall burden of cardiovascular disease. Furthermore, organizational-level interventions and public health policies aimed at ensuring appropriate working conditions, adequate social support, and a balanced relationship between occupational demands and individual resources have the potential to improve cardiovascular health at the population level.

Acknowledgments

None.

Funding

None.

Conflict of interest

The authors declare no conflict of interest.

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Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data

Not applicable.

Further disclosure

During the preparation of this manuscript, ChatGPT (OpenAI) was used to assist with language editing and minor improvements in clarity and readability. The authors have critically reviewed and edited the manuscript and take full responsibility for the content of this publication.

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