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ORIGINAL RESEARCH

OUTCOMES AND QUALITY

Coronary Heart Disease Attributable to Psychosocial Stressors at Work

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ABSTRACT

BACKGROUND Psychosocial stressors at work, including job strain and effort-reward imbalance (ERI), have been associated with an increased risk of coronary heart disease (CHD). However, the proportion of CHD events attributable to these exposures has not been quantified in a prospective cohort study.

OBJECTIVES The purpose of this study was to estimate the fraction of CHD events attributable to psychosocial stressors at work in a 20-year prospective cohort study.

METHODS This prospective cohort study included employees from public and semipublic organizations in Quebec City, Canada, followed from 2004 and 2018. A total of 6,295 participants without cardiovascular disease at baseline were included. Job strain and ERI were assessed using validated instruments. Incident CHD events were identified through universally covered health care databases. Attributable fractions were estimated using the Kaplan-Meier method. Multiple imputation and inverse probability weighting were applied to address selection and confounding. The first 5 years of follow-up were excluded to minimize reverse causation.

RESULTS During 15-year follow-up, 669 CHD events occurred over 112,297 person-years, yielding a CHD incidence rate of 5.96 per 1,000 person-years. The attributable fraction for job strain was 18.2% (95% CI: 1.8%-34.7%), and for ERI, it was 3.3% (95% CI: −1.6% to 8.2%). Combined exposure to both stressors resulted in an attributable fraction of 19.5% (95% CI: 0.7%-38.4%).

CONCLUSIONS In this cohort, combined exposure to job strain and ERI accounted for approximately one-fifth of CHD events. Findings suggest that psychosocial stressors at work could be relevant targets for reducing the burden of CHD through prevention strategies. (JACC Adv. 2025;4:102160) © 2025 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

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ABBREVIATIONS AND ACRONYMS

AFs = attributable fractions
CHD = coronary heart disease
CVD = cardiovascular disease
ERI = effort-reward imbalance
KM = Kaplan-Meier

Coronary heart disease (CHD) is the worldwide leading cause of death and contributed to 185 million disability-adjusted life years in 2021.[1] The recent rise in CHD mortality rates among working-age North Americans is concerning.[2-4] Prevention efforts targeting modifiable CHD risk factors are crucial to alleviate this burden.

Attributable fractions (AFs) are essential measures to orient prevention efforts. They quantify the population-level reduction of disease achievable when preventing exposure to a specific risk factor. AFs estimate the proportion of disease cases that could be avoided if a given causal risk factor was eliminated. AFs account for both the risk increase and prevalence of a given risk factor, providing a straightforward assessment of its public health importance. AFs are therefore highly needed for informed decision-making and for prioritizing targets in prevention efforts.

Large international studies have estimated AFs to identify key behavioral and metabolic risk factors for CHD prevention.[5-7] However, these studies primarily focused on individual-level risk factors, with limited attention to upstream risk factors, such as those from the work environment. Psychosocial stressors at work from the job strain and effort-reward imbalance (ERI) models are well-established, modifiable risk factors from the work environment. Prospective epidemiological studies have consistently shown that workers exposed to job strain or ERI are at increased CHD risk.[8-12] High job strain occurs when high psychological demands are coupled with low decision latitude,[13] while ERI refers to the imbalance experienced when the effort required at work is disproportionately high compared to the rewards received.[14] The prevalence of workers exposed to either job strain or ERI is high, ranging from 20% to 25%.[15]

No prospective cohort study has estimated the fraction of CHD attributable to both job strain, ERI, and their combination. Estimating AFs from multiple stressors at work is challenging, with previous studies relying on single exposures[8,16,17] and indirect AF estimation method.[18,19] The current prospective study aimed to directly estimate the fraction of CHD events attributable to job strain, ERI, and their combination, among individuals without prior cardiovascular disease (CVD) and followed over 15 years. This study used rare longitudinal data on multiple work stressors and CHD incidence in the same cohort to assess their public health impact.

METHODS

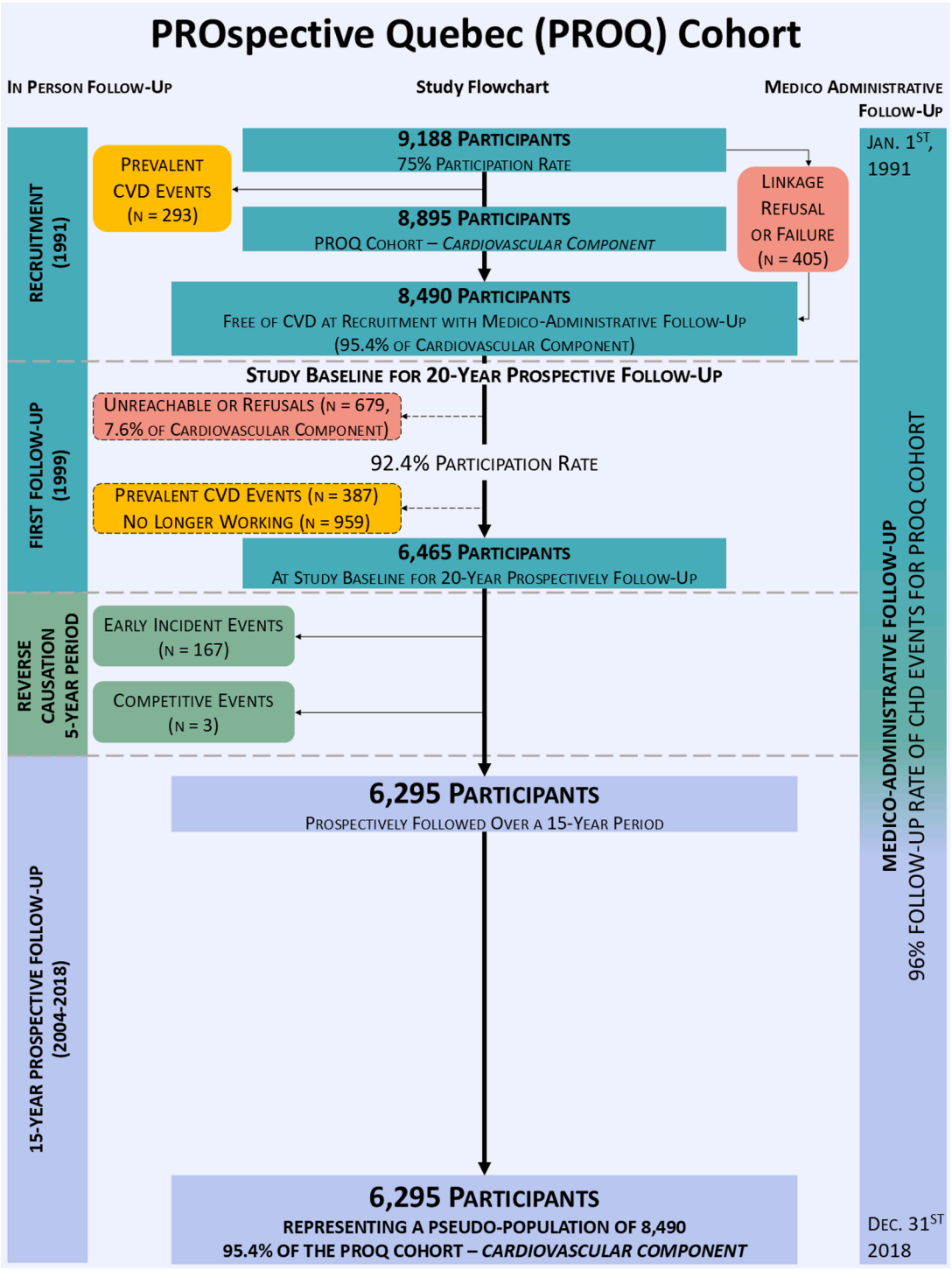
STUDY POPULATION. The PROQ (PROspective Quebec Study on Work and Health) recruited 9,188 white collar workers from 19 organizations in 1991 to 1993.[20] CHD events were identified for 96% of the PROQ participants, up to December 31, 2018. Informed consent was obtained from all participants, and the study was approved by Institutional Review Boards.

The current study baseline started in 1999 to 2001, during which both job strain and ERI were measured. Participation at this follow-up was 92.4%. At this time, 6,465 participants were still employed, free of CVD, reachable, and consented to participate. To control for potential reverse causation, the first 5 years of follow-up were excluded, resulting in a final study sample of 6,295 workers without an early incident CHD event (**Figure 1**, Supplemental Methods, Supplemental Table 1). The data that support the findings of this study are available from Dr Trudel upon reasonable request (xavier.trudel@crchudequebec.ulaval.ca).

PSYCHOSOCIAL STRESSORS AT WORK. All psychosocial stressor at work were measured at the 1999 to 2001 follow-up. Components of the job strain model were measured using the 18-item scale from the validated French translation[21] of the Job Content Questionnaire.[22] Psychological demands measured excessive workload, task interruption, and conflicting responsibilities. Decision latitude measured skill discretion and decision autonomy. Scores for psychological demands (range: 9-36) and decision latitude (range: 24-96) were calculated. Workers were categorized as having high psychological demands (≥24) or low decision latitude (≤72) based on median cutoffs from the Quebec working population, as recommended.[23] The recommended quadrant method was used to classify workers into high strain, passive and active jobs vs low strain jobs (the reference group) (**Central Illustration**).[13]

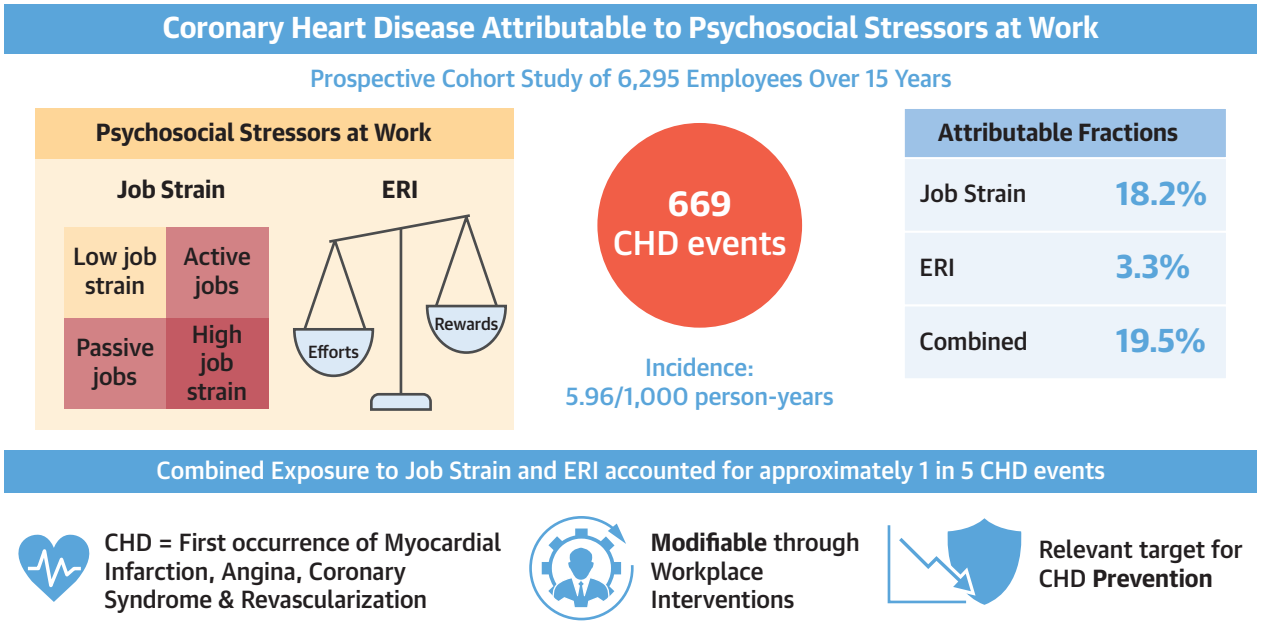
Reward at work was measured using 9 questions from the validated French translation of the ERI instrument.[14] The reward scale (range: 9-27) was dichotomized at the median (<25). Psychological demands, assessed using 9 items from the Job Content Questionnaire French validated translation (range: 9-36, median ≥24), served as a proxy for effort.[21] The psychometric qualities of this ERI version have been demonstrated.[24] As recommended, ERI was defined by a ratio >1.[14] This binary definition is consistent with the original theoretical

FIGURE 1 Study Flowchart of the PROQ Cohort



This figure outlines the selection of participants from the PROQ cohort for the CHD analysis. Of 9,188 initially recruited participants, 8,895 were retained in the cardiovascular component. After excluding those with prevalent CVD or failed data linkage, 8,490 participants were eligible for medico-administrative follow-up. The analytic sample included 6,295 participants free of CVD and actively working at study baseline, with exclusions for refusals, nonworking status, and early incident or competing events. A pseudo-population was generated using inverse probability weighting to address selection processes and minimize reverse causation. Visual Annotations: Yellow boxes indicate exclusions based on data validity (eg, prevalent CVD). Red boxes denote losses due to refusal, unreachable participants, or linkage failure. Dotted lines represent statistical adjustments using inverse probability weighting (IPW) to construct a pseudo-population. Abbreviations: CHD = coronary heart disease; CVD = cardiovascular disease; PROQ = PROspective Quebec.

CENTRAL ILLUSTRATION Coronary Heart Disease Attributable to Psychosocial Stressors at Work



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This Central Illustration summarizes key findings from a 15-year prospective cohort study of 6,295 employees, investigating the contribution of psychosocial stressors at work to CHD. Exposure to job strain and ERI were assessed using validated instruments. A total of 669 CHD events were identified through administrative health databases. Attributable fractions were 18.2% for job strain, 3.3% for ERI, and 19.5% for combined exposure. These exposures are modifiable through workplace interventions and represent relevant targets for CHD prevention. Abbreviations as in **Figures 1 and 2**.

framework[14] and has been widely used in prior epidemiological studies, including the only study to date that estimated AFs for ERI,[19] thus favoring comparability across studies.

Combined exposure to job strain and ERI was categorized into 4 levels as described previously.[11] Double exposure was defined as having both high job strain and ERI. Participants with either high job strain or ERI, but not both, were categorized as having intermediary exposure. Low exposure included participants in passive or active jobs and/or those with low rewards, without an ERI. The unexposed group included only those with low job strain and without low rewards, reflecting the theoretical minimum risk level (Supplemental Figure 1).[25]

CORONARY HEART DISEASE EVENT DEFINITION. CHD events were identified over a 15-year follow-up period (2004-2018) using medico-administrative databases, which provide near-complete coverage of all hospitalizations, emergency visits, outpatient consultations for Quebec residents.[26]

A validated algorithm was applied to ascertain CHD events. This algorithm was developed and validated against chart-abstracted diagnoses from electronic medical records, considered the gold standard (sensitivity: 77.0%; specificity: 97.5%; positive predictive value: 75.3%) (Supplemental Figure 2).[27]

Incident CHD events included the first occurrence of myocardial infarction, angina, acute and chronic coronary syndrome (International Classification of Diseases-9: 410-414; International Classification of Diseases-10: I20-I25), coronary revascularization, either by coronary angioplasty or bypass surgery (Canadian Classification of Diagnostic, Therapeutic, and Surgical Procedures [48.02, 48.03, 48.09, 481]; Canadian Classification of Health Interventions [1. IJ.50, 1.IJ.57.GQ, 1.IJ.76]). CHD deaths were identified in the provincial death registry.

Participants were identified as having had an incident CHD event if they had: 1) 1 hospitalization with specified diagnosis or procedure codes; 2) 2 or more outpatient consultations within 1 year with such diagnosis; or 3) CHD as the primary cause of

death. Prevalent CVD events occurring before the study baseline were identified using similar algorithms applied to medico-administrative databases[27,28] as well as self-reported questionnaires and were subsequently excluded.

COVARIATES. Blood pressure was measured according to guidelines with a sphygmomanometer,[29] and participants were classified as hypertensive if the average of 3 readings was elevated (≥140/90 mm Hg) or if they reported antihypertensive drug use. Diagnoses of diabetes and dyslipidemia, as well as related medication use, were self-reported. Waist and hip circumferences were measured by a trained research nurse,[20] and the waist-to-hip ratio was calculated with at-risk thresholds of >0.95 for men and >0.8 for women.[30] Current smoking status and alcohol intake were measured by standardized questionnaires. Self-reported weekly alcohol intake was classified as abstinent, low risk (≤15 drinks for men, ≤10 drinks for women), or high risk.[31] Leisure physical activity was measured by frequency and duration of sessions to classify participants as low (<1 session/week), moderately active (1-2 sessions/week), or active (≥3 sessions/week).[32]

CAUSALITY INFERENCE FRAMEWORK. Estimates of AFs rely on the assumption of a causal relationship between the exposure and the outcome.[33] The associations between job strain and ERI with CHD have been studied over 3 decades, with consistent evidence from meta-analyses and prospective studies supporting a causal effect.[8,10,12,34] Quality assessments demonstrated that previous evidence most likely underestimates the true causal effect.[34-36] The current study has been built on this extensive body of evidence. It has been shaped in all parts to avoid previous limitations and use the most updated causal inference methods.

STATISTICAL ANALYSIS. Missing data were handled through sequential multiple imputation, creating as many data sets as the percentage of missing data.[37] Descriptive analyses were performed on these imputed data sets and then averaged across imputations. Marginal Cox models were used to calculate HRs as described in detail elsewhere.[11] The proportional hazards assumption of the Cox model was visually verified based on Schoenfeld residuals and deemed satisfied.

The Kaplan-Meier (KM) approach estimated AFs by accounting for changes in participants at risk over time.[38] Person-years were calculated until a CHD event, a competing risk, or the end of follow-up (December 31, 2018). Noncardiovascular deaths were censored with the Fine and Gray estimator.[39]

The initial 5 years of follow-up were subtracted from the survival curves, which covered a 20-year period:

$$AF_{15} = 1 - \left[\frac{P_{20}\,(Y_0 = 1) - P_5\,(Y_0 = 1)}{P_{20}\,(Y = 1) - P_5(Y = 1)}\right]$$

$P_x(Y_0 = 1)$ represents the cumulative CHD incidence at time x, assuming no exposure. This was estimated using a weighted KM estimator among *unexposed participants*. Confounding weights balanced the study population in terms of age, sex, education level, marital status, lifestyle factors (smoking, alcohol consumption, physical activity), anthropometric measures (at-risk waist-hip ratio), and metabolic factors (diabetes, hypertension, dyslipidemia) across exposure groups. Covariates for confounding adjustment were measured at study baseline (1999-2001), while covariates for selection were assessed at recruitment (1991-1993). Confounding weights were combined with selection inverse probability weights and applied to the imputed data sets, creating a pseudo-population controlling for these biases.[38,40] Selection and confounding inverse probability weights were multiplied and truncated at the 99th percentile to avoid extreme weight.[41]

The denominator $P_x(Y = 1)$ represents the observed CHD incidence and was estimated using a KM estimator weighted solely for selection, among the *entire* population. This reflects the incidence in the population, accounting for selection.

The KM approach, based on counterfactual reasoning (*ie,* estimating what would have happened in an unexposed population), estimates CHD incidence while controlling for selection and confounding. This method estimates an AF by accounting for the prevalence and risk associated with each exposure level. The KM approach estimates AFs for both binary exposures (*eg,* ERI), and categorical exposures (*eg,* job strain quadrant and combined exposure to job strain and ERI). These AFs reflect the potential reduction in CHD events if all exposures were lowered to the minimum risk level.[25] A nonparametric bootstrap (200 replicates) estimated AF standard errors within each imputed data set,[42] with 200 replicates as recommended for standard error estimation,[43] providing 95% CIs. The difference in AFs between men and women was tested using the Wald test.[44] Differences were not statistically significant. Therefore, the analyses are presented for the total population.

SUPPLEMENTARY ANALYSES. Supplementary analyses included a parsimonious model to assess overadjustment by potential mediating factors[45] and

alternative exposure definitions and AF calculations,[33,46] for comparison with prior studies, including a tertile-based categorization for ERI to better align with the theoretical minimum risk levels.[25]

RESULTS

A total of 6,295 participants (52% women) were included (**Table 1**). At the study baseline, the average age was 45 years; 17% of participants were smokers, 4% were heavy drinkers, and 23% were physically active less than once per week. The prevalence of diabetes, hypertension, dyslipidemia, and high waist-hip ratio was 3%, 22%, 26%, and 31%, respectively. Regarding psychosocial stressors at work, 37% held passive jobs, 28% held active jobs, and 18% were exposed to high job strain, and 25% were exposed to ERI. Double exposure to high job strain and ERI was present in 10%; intermediary exposure (either high job strain or ERI) in 24%, and low exposure (passive or active jobs and/or with low rewards, without an imbalance) in 55% of participants.

A total of 669 incident CHD events occurred over the 15-year follow-up, with a crude incidence rate of 5.96 per 1,000 person-years.

KAPLAN-MEIER CURVES. **Figures 2A to 2C** presents the cumulative incidence of CHD by psychosocial stressors at work. Participants exposed to high job strain (**Figure 2A**) and ERI (**Figure 2B**) had a higher cumulative CHD incidence compared to those with low strain and those unexposed to ERI. **Figure 2C** presents cumulative CHD incidence for combined exposure. Double exposure had the highest cumulative CHD incidence, followed by that of intermediate exposure. Cumulative CHD incidence among participants with low exposure increased slightly faster when compared to that among unexposed participants, a differentiation that was observed approximately 7 years after study baseline.

ATTRIBUTABLE FRACTIONS. The estimated AF of CHD events, adjusted for age, sex, marital status, education, hypertension, cholesterol, diabetes, smoking, alcohol consumption, physical activity, and waist-hip ratio was 18.2% (95% CI: 1.8%-34.7%) for job strain (**Table 2**). For ERI exposure, the AF was 3.3% (95% CI: −1.6% to 8.2%), and for combined job strain and ERI exposure, the AF was 19.5% (95% CI: 0.7%-38.4%).

RESULTS OF SUPPLEMENTARY ANALYSES

Results were comparable when using a parsimonious model adjusted for age, sex, and education only (Supplemental Table 2). Using the binary definition of high job strain (regrouping passive and active groups with the low job strain group) resulted in lower AFs (Supplemental Table 3). Tertile analyses of ERI yielded a higher AF of 7.6% (95% CI: −2.6 to 17.6). AFs calculated using Miettinen's formula,[33,46] yielded similar estimates (Supplemental Table 4).

DISCUSSION

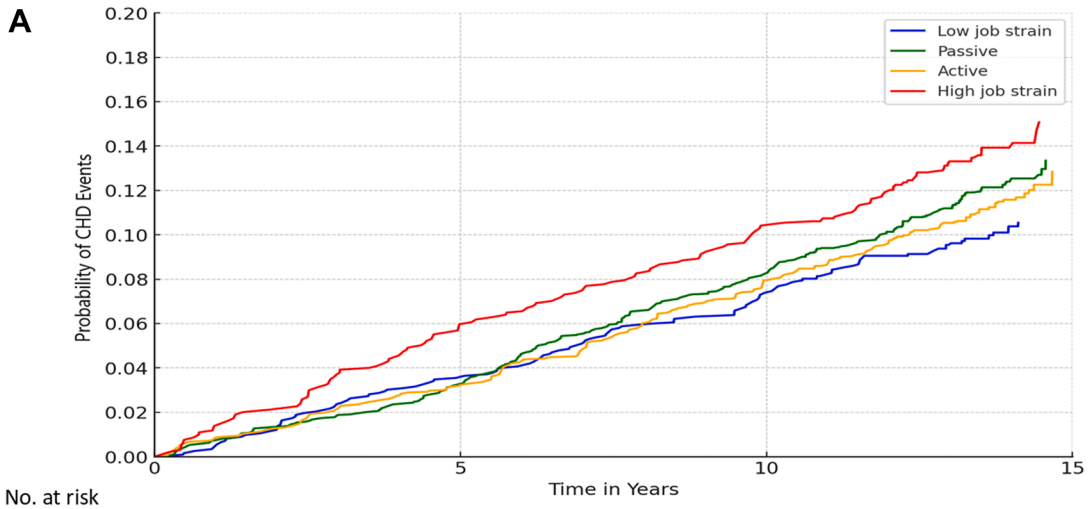
MAIN FINDINGS. The present study shows that psychosocial stressors at work are important contributors to incident CHD events. Combined exposure to job strain and ERI was estimated to contribute to one-fifth of CHD events. Job strain alone explained most of this burden, contributing to 18% of CHD events. These estimates represent the proportion of CHD events that could be prevented by reducing exposure to job strain and ERI for all workers, to levels observed in unexposed individuals.

COMPARISON WITH PREVIOUS STUDIES. The present study is the first to provide direct prospective cohort estimates of the fraction of CHD events attributable to multiple psychosocial work stressors, including job strain, ERI, and their combination. Previous AFs estimation for CHD due to these stressors is scarce, having mostly relied on indirect methods that combine prevalence data with relative risks derived from different populations.[17-19]

Our approach to estimating AFs aligns with the theoretical minimum risk level used in large-scale CHD studies.[7,25] This approach requires well-differentiated exposure levels including a true minimum risk level. The theory-based quadrant method used here made it possible to define a true minimum risk level, by treating passive and active jobs as distinct categories. This contrast with prior studies in which these categories were grouped into the unexposed group in a binary categorization.[8,16,18,19] Our approach is coherent with previous prospective studies showing that not only high job strain but also passive and active jobs are associated with adverse cardiovascular outcomes.[10,47-49] The AF for job strain observed here therefore represents the benefit of reducing exposure across all exposed categories to the true minimum risk level. In comparison, AFs for job strain have previously been estimated in only one longitudinal analysis using individual participant data from a European cohort consortium, where a binary exposure definition was applied and estimates ranged from 3.4% to 4.3%.[8,16]

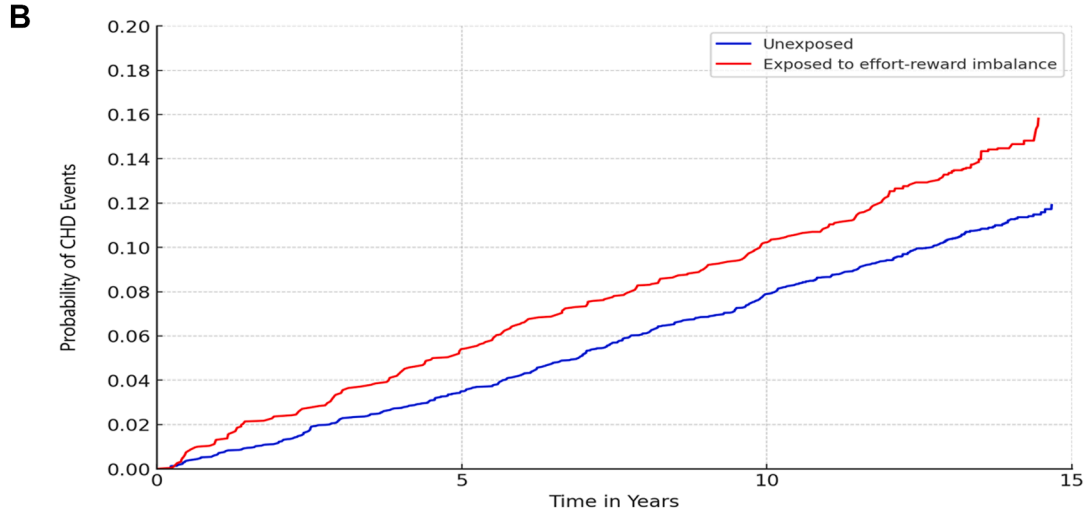
Our main analysis suggests a modest AF of 3% (95% CI: −1.6 to 8.2) for ERI exposure considered

FIGURE 2 **Coronary Heart Disease by Psychosocial Stressors at Work**



No. at risk

Low Job Strain	1,111	1,073	1,030	0
Passive Jobs	2,305	2,237	2,142	0
Active Jobs	1,750	1,698	1,612	0
High Job Strain	1,129	1,081	1,042	0



No. at risk

Balance State	4,745	4,603	4,406	0
Imbalance State	1,550	1,486	1,420	0

Kaplan-Meier curves display the cumulative incidence of CHD events over 15 years according to exposure to psychosocial stressors at work. A shows CHD risk by job strain categories (low strain [reference], passive, active, and high strain). High job strain and passive jobs were associated with a higher cumulative incidence of CHD. B compares participants exposed vs unexposed to ERI, with higher cumulative incidence observed in the exposed group. C presents cumulative incidence across combined exposure groups: neither exposure, job strain only, ERI only, and both exposures. The highest risk was observed among participants jointly exposed to both job strain and ERI. ERI = effort–reward imbalance; other abbreviation as in Figure 1.

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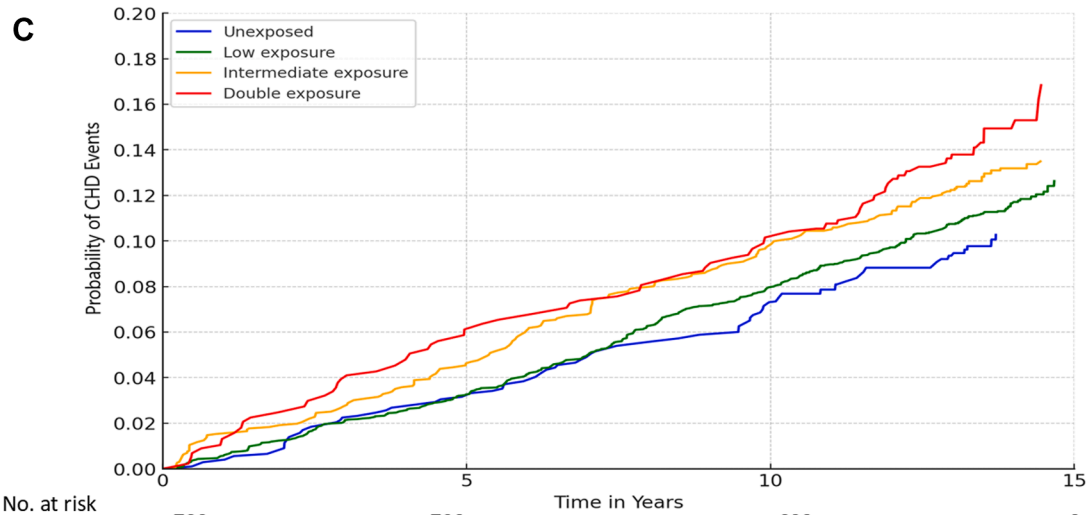
FIGURE 2 Continued



No. at risk				
Unexposed	739	720	690	0
Low Exposure	3,479	3,373	3,227	0
Intermediary Exposure	1,476	1,425	1,356	0
Double Exposure	601	571	553	0

separately. A previous study has reported a modest AF for ERI (1.7%) but used an indirect estimation method, making comparisons challenging.[19] Our supplementary analysis using tertiles instead of the usual binary categorization suggests a potentially higher AF for ERI (7.6%) when a finer categorization is used (Supplemental Table 4).

The combined effect of job strain and ERI has rarely been quantified despite evidence that these psychosocial stressors at work may exert independent effects on a number of health outcomes, including CHD.[11,12,50] In our study, estimating AFs for their combined exposure provides a broader estimate of the potential burden attributable to work-related stressors. This combination reflects real-world workplace conditions, where multiple adverse psychosocial stressors often co-occur, potentially amplifying the overall CHD burden.

PSYCHOSOCIAL WORK STRESSORS AND TRADITIONAL CHD RISK FACTORS. This study shows that the contribution of psychosocial stressors at work to CHD burden tends to be as important as that of some traditional CHD risk factors. For instance, our AF estimate that one-fifth of CHD events are attributable to job strain and ERI combined is in the same order magnitude as the 17% AF for myocardial infarction due to hypertension reported in the PURE (Prospective Urban Rural Epidemiology) study.[6]

PATHOPHYSIOLOGICAL MECHANISMS. The findings of this study align with the current understanding of pathophysiological mechanisms linking psychosocial stressors at work and CHD. Over the course of professional life, exposure to chronic stressors such as psychosocial stressors at work in addition to conventional cardiovascular risk factors can promote the occurrence and progression of coronary atherosclerosis with the activation of the sympathetic nervous system, the renin-angiotensin-aldosterone system, and the hypothalamo-pituitary-adrenal axis.[51] Furthermore, acute activation of the sympathetic nervous system can precipitate cardiovascular events by promoting atherosclerotic plaque disruption and platelet activation, driven by increased heart rate, elevated blood pressure, and coronary vasoconstriction.[51] These mechanisms provide solid biological plausibility for the effect of psychosocial stressors at work on CHD risk.

CAUSALITY. In the current study, causal interpretation of estimated AFs is supported by several converging considerations. First, the temporality of the association is ensured by the prospective design, where exposure to job strain and ERI precedes the

TABLE 1 Descriptive Statistics of the Study Sample at Baseline (N = 6,295)

Age (y)	45.2 ± 6.7
Sex	
Women	3,288 (52)
Men	3,007 (48)
Education level	
≤High school	1,628 (26)
College	1,748 (28)
University	2,919 (46)
Marital status	
In relationship	4,743 (75)
Smoking status	
Smoker	1,094 (17)
Alcohol consumption[a]	
Abstinent	393 (6)
Low-risk	5,626 (89)
High-risk	276 (4)
Physical activity (times per week)[b]	
<1/wk	1,463 (23)
1 or 2/wk	1,698 (27)
≥3/wk	3,134 (50)
Diabetes	
Yes	174 (3)
Hypertension	
Yes	1,370 (22)
Dyslipidemia	
Yes	1,640 (26)
Waist-to-hip ratio[c]	
At risk	1,922 (31)
Job strain	
Low job strain	1,111 (18)
Passive jobs	2,305 (37)
Active jobs	1,750 (28)
High job strain	1,129 (18)
Effort-reward imbalance	
Exposed	1,550 (25)
Combined exposure[d]	
Unexposed	739 (12)
Low exposure	3,479 (55)
Intermediary exposure	1,476 (24)
Double exposure	601 (10)

Values are mean ± SD or n (%). Descriptive statistics of participants at baseline, including sociodemographic characteristics, lifestyle factors, and exposure to psychosocial stressors at work. [a]Abstinent - reported no alcohol consumption. Low-risk weekly alcohol consumption defined as ≤15 for men and ≤10 for women. [b]Leisure physical activity levels defined as low (<1 20-30-minute session per week), moderate (one or two sessions per week), and active is 3 or more sessions per week. [c]The at-risk waist-to-hip ratio was calculated using a threshold of >0.95 for men and >0.8 for women. [d]Combined exposure was defined as follows: unexposed: low job strain and unexposed to low rewards; low exposure: passive or active job and/or unexposed to low rewards; intermediary exposure: job strain or effort-reward imbalance, but not both; double exposure: job strain and effort-reward imbalance.

onset of CHD events. Second, the consistency of findings across different methodological approaches and exposure categorizations reinforces the robustness of this relationship. The coherence of our results with prior prospective studies and meta-analyses further supports a causal interpretation.[8,10,12,34] Third, the observed AFs persist after full adjustment for a comprehensive set of potential confounders, including sociodemographic factors (age, marital status, education) and established cardiovascular risk factors (hypertension, cholesterol, diabetes, smoking, alcohol consumption, physical activity, and waist-to-hip ratio). This minimizes the likelihood that residual confounding explains the observed associations. Finally, the biological plausibility of this relationship is well established as described in the previous section.

GENERALIZABILITY OF FINDINGS. While the present study was conducted on a population of white-collar workers, important factors favor generalization: 1) the incidence of CHD was comparable in magnitude to the general adult population in the same region;[23,26] 2) white-collar work is a common occupational category in high-income countries;[52] 3) the prevalence of exposure to active, passive jobs, and high job strain observed in our population is similar to previous estimates from both U.S. and European studies, including occupational and cardiovascular cohorts (Supplemental Table 5). In addition, our observed risk estimates are consistent with a recent meta-analysis of prospective studies[10] which reported, among the 9 studies with at least 10 years of follow-up, HRs for CHD of 1.20 (95% CI: 1.05-1.36) for passive jobs, 1.13 (95% CI: 0.98-1.30) for active jobs, and 1.33 (95% CI: 1.16-1.53) for high job strain. However, it should be noted that blue-collar workers are also frequently exposed to psychosocial stressors at work, with studies also supporting stronger association with CHD.[52-54] These evidence suggest that the attributable burden may be comparable or higher in this group. Finally, it is important to note that the present study was conducted before the COVID-19 pandemic. Evidence suggests that there have been post-COVID increases in the prevalence of psychosocial stressors at work, including increased work intensity and unsocial working hours, particularly in the United States and Europe.[55,56] Teleworking has also been linked to increased demands.[57]

TABLE 2 Attributable Fractions for Coronary Heart Disease Across Psychosocial Stressors at Work Over 15 Years (N = 6,295, No. of Events = 669)

Exposure Category	N (%)	CHD Events	CHD Incidence Rate (per 1,000 PY)	Adjusted HR[a] (95% CI)	Adjusted AF[a] (%) (95% CI)
Job strain[b]					
Low job strain (ref)	1,111 (18)	111	5.45	1.00	18.2 (1.8-34.7)
Passive jobs	2,305 (37)	240	5.77	1.26 (0.99-1.53)	
Active jobs	1,750 (28)	195	6.17	1.12 (0.84-1.41)	
High job strain	1,129 (18)	123	6.03	1.41 (1.12-1.70)	
Effort-reward imbalance[c]					
Balance state (ref)	4,745 (75)	478	5.54	1.00	3.3 (−1.6 to 8.2)
Imbalance state	1,550 (25)	191	6.88	1.17 (0.97-1.37)	
Combined exposure to job strain and effort-reward imbalance[d]					
Unexposed (ref)	739 (12)	65	4.84	1.00	19.5 (0.7-38.4)
Low exposure	3,479 (55)	365	5.77	1.18 (0.83-1.53)	
Intermediary exposure	1,476 (24)	163	6.19	1.27 (0.89-1.65)	
Double exposure	601 (10)	76	6.93	1.53 (1.06-1.99)	

Adjusted HRs, AFs, and incidence rates (per 1,000 person-years) for CHD over 15 years by categories of job strain, effort-reward imbalance, and combined exposure. HRs and AFs are adjusted for sociodemographic, lifestyle, and cardiovascular risk factors. [a]HRs were estimated using Cox proportional hazards models to assess the association between psychosocial stressors at work and the risk of CHD. AFs were calculated using the Kaplan-Meier method, indicating the proportion of CHD events that could be prevented if the exposure to specific work-related stressors was eliminated. Both HRs and AFs are adjusted for age, sex, marital status, education level, as well as for physical activity, alcohol consumption, tobacco smoking, hypertension, cholesterol, diabetes, and waist-to-hip ratio. [b]Job strain model categories are defined as follows: "low job strain" (reference) includes participants in jobs with low psychological demands and high decision latitude, "passive jobs" refer to jobs with low psychological demands and low decision latitude, "active jobs" include jobs with high psychological demands and high decision latitude, and "high job strain" reflects jobs characterized by high psychological demands and low decision latitude. [c]Effort-reward imbalance model categories are defined as follows: "balance state" (reference) includes participants with balanced effort and reward at work. "Imbalance state" refers to participants where the effort required at work is disproportionately high relative to the rewards received. [d]Combined exposure to psychosocial stressors was categorized as follows: "unexposed" (reference) includes participants with low job strain and balanced effort-reward conditions. "Low exposure" refers to participants with passive or active jobs and/or low rewards without imbalance. "Intermediary exposure" includes participants exposed to either high job strain or effort-reward imbalance, but not both. "Double exposure" refers to participants exposed to both high job strain and effort-reward imbalance.

AF = attributable fraction; CHD = coronary heart disease; PY = person-years.

These post-COVID increases could translate into a higher contribution of psychosocial stressors at work to the burden of CHD.

WORKPLACE INTERVENTIONS FOR CARDIOVASCULAR PREVENTION. Psychosocial stressors at work, including job strain and ERI, represent relevant targets for CHD prevention. Reducing the prevalence of these work stressors is achievable using appropriate workplace interventions.[15] Such interventions involve improving job control, reducing excessive work demands, and fostering supportive work environments. An evaluation of interventions of this type has shown reductions in both blood pressure means and hypertension prevalence using ambulatory monitoring.[58] Additionally, addressing these stressors can have broader health benefits, as prospective studies show that they increase the risk of other important debilitating conditions, such as diabetes[59] and depression.[60] Implementing strategies to reduce these stressors can therefore lead to substantial and lasting improvements in population health.

PUBLIC HEALTH AND POLICY IMPLICATIONS. The American Heart Association recognizes the workplace as a crucial setting for prevention and promotes comprehensive workplace wellness programs.[61] Our results suggest that incorporating interventions aimed at the reduction of psychosocial stressors at work to these programs may be effective in preventing a sizable number of CHD events. Furthermore, clinicians can play a role by raising awareness about psychosocial stressors at work and recognizing their impact on cardiovascular health. Some countries have implemented public health policies calling for surveillance and mitigation of psychosocial work stressors.[62] In the province of Quebec, employers recently have had the legal obligation to prevent psychosocial stressors at work.[63] Such societal measures are essential to support and guide organizations in reducing psychosocial stressors at work. These policies could, in turn, have a significant

impact on reducing the CHD burden, promoting long-term public health improvements.

CONCLUSIONS

In the present study, a large fraction of CHD events was attributable to job strain alone or in combination with ERI. These findings add new evidence on potential benefits for CHD prevention of reducing psychosocial stressors at work. Both job strain and ERI exposures should be acknowledged in prevention strategies to reduce the burden of CHD.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: Psychosocial stressors at work contribute to a large proportion of incident CHD events among women and men.

TRANSLATIONAL OUTLOOK: The effectiveness of preventive interventions on psychosocial stressors at work in reducing the CHD burden should be investigated in future studies.

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APPENDIX For supplemental tables, methods, and figures, please see the online version of this paper.