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Long working hours at midlife and arterial stiffness at older age in a 24-year prospective cohort

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Abstract

Backgrounds This study aims to examine whether long working hours, repeatedly assessed at midlife, is associated with higher arterial stiffness at older age in a 24-year prospective study of white-collar workers in Quebec City, Canada.

Methods This study relied on a prospective cohort, initiated in 1991–1993 (T1) with two follow-ups after 8 years (T2, 1999–2001) and 24 years (T3, 2015–2018). Participants ($N = 1,629$, 51.3% women, mean age 37 ± 6.4 at T1) were randomly selected for arterial stiffness measurement at T3 using carotid-femoral pulse wave velocity (PWV). Long working hours (> 40 h/week) were assessed at T1 and T2. Mean differences in PWV were estimated using generalized linear models, accounting for sociodemographic factors, lifestyle-related risk factors, clinical factors and psychosocial stressors at work.

Results Among participants who remained actively employed over the study period (age range: 21–59 at T1), long working hours at T1 were associated with a + 0.54 m/s (95% CI: 0.05; 1.02) increase in PWV, while repeated exposure at T1 and T2 was associated with a + 1.50 m/s (95% CI: 0.78; 2.21) increase. No association was observed among participants who retired between T2 and T3.

Conclusion The present study suggests that exposure to long working hours during midlife is associated with higher arterial stiffness, among aging workers. Workplace preventive strategies reducing long working hours may be effective to mitigate long-term arterial stiffening.

Keywords Occupational stress, Work environment, Arterial stiffness, Cardiovascular disease

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Background

The cardiovascular continuum is defined as a sequence of events, initiated by exposure to upstream risk factors, progressing to asymptomatic vascular damage and leading to cardiovascular diseases (CVD) [1]. Arterial stiffness is an asymptomatic CVD marker, resulting from reduced ability of large proximal arteries to dilate and retract [2]. Arterial stiffness is associated with increased CVD risk, independently of blood pressure (BP) [3]. Evidence suggest that tackling upstream and modifiable risk factors may alter the pathogenic process leading to CVD, by reducing arterial stiffness progression [4].

Long working hours is a frequent and modifiable upstream risk factor from the work environment. According to the World Health Organization and International Labor organization (WHO / ILO), the number of individuals exposed to long working hours is increasing worldwide [5]. In 2023, about 20% of workers in the US [6] and 15% in Canada [7] worked more than 40 h, which exceeds the standard threshold for weekly working hours in many industrialized countries, including the US and Canada [8]. The association between long working hours and an increased risk of CVD, including coronary heart disease and stroke, has been documented in systematic reviews and meta-analyses [9, 10]. According to the WHO/ILO, long working hours were responsible for 745,000 deaths from ischemic heart diseases and stroke worldwide, in 2016 [5].

There is sparse evidence about the effect of long working hours on arterial stiffness. In a previous cross-sectional study conducted in Japan, older men (≥ 50 years old) working overtime had higher arterial stiffness [11]. In a recent prospective cohort study, workers exposed to long working hours at baseline had higher arterial stiffness progression over 5 years [12]. Both studies have used different methods to assess arterial stiffness and neither have used carotid-femoral pulse wave velocity (PWV), recognized as the reference assessment method [13, 14]. Moreover, none has assessed repeated exposure over time. Workers repeatedly exposed to long working hours over time may have higher PWV, when compared to those exposed for a shorter period.

The first objective of the present study was to examine the association between baseline exposure to long working hours at midlife and arterial stiffness, assessed at older age using carotid-femoral PWV, compared to those working ≤ 40 h/week. The second objective was to examine the association between repeated exposure to long working hours and arterial stiffness, compared to those never exposed.

Methods

Study design and population

This study relied on the PROspective Québec (PROQ) Study on Work and Health, described elsewhere [15]. The cohort was composed of white-collar workers from 19 public and semi-public organisations in Quebec City, Canada. Their professional activities encompassed the entire range of white-collar positions, including senior management, professional, technical and office workers. All workers were invited to participate. A total of 9,188 white-collar workers (participation rate: 75%) were recruited in 1991–1993 (T1). Follow-up participation included 8,120 workers (90% of those eligible) in 1999–2001 (T2) and 6,744 (81% of those eligible) in 2015–2018 (T3).

A third of the population at baseline, in 1991–1993, was randomly selected from the entire PROQ cohort [16]. A second random sampling was performed, to include 50% of those who were actively employed in 2015–2018 (T3). In total, 3,411 participants were randomly selected for arterial stiffness assessment (Fig. 1).

Participants with prevalent CVD ($N = 170$), defined as cardiovascular events occurring before the start of the study, and those working less than 21 h per week ($N = 259$) at baseline or at the first follow-up were excluded. After applying these selection criteria, 2,982 participants were identified as eligible for the present study. A total of 309 participants were excluded at T2 (1999–2001) and 629 at T3 (2015–2018) because of losses to follow-up or deaths ($n = 938$). Additionally, 368 participants were excluded because they refused arterial stiffness assessment or had invalid measures. Finally, participants with missing values on long working hours ($N = 3$) and covariates ($N = 44$) were also excluded. The final study sample therefore consisted of 1,629 participants. Excluded participants were older, had higher BMI, and presented higher systolic and diastolic BP compared to included participants. They were also more likely to have lower education levels, be current smokers, and have hypertension and diabetes, while being less likely to engage in leisure physical activity or moderate alcohol consumption (Table S1).

Data collection

At all three data collection, workers completed a self-administered questionnaire on sociodemographic, lifestyle-related risk factors, clinical risk factors and psychosocial stressors at work.

Long working hours

The primary independent variable was self-reported weekly worked hours in the respondents' paid job. Long working hours were defined as hours of work more than usual full-time working hours, namely 40 h per week. At each time, working hours were grouped into the

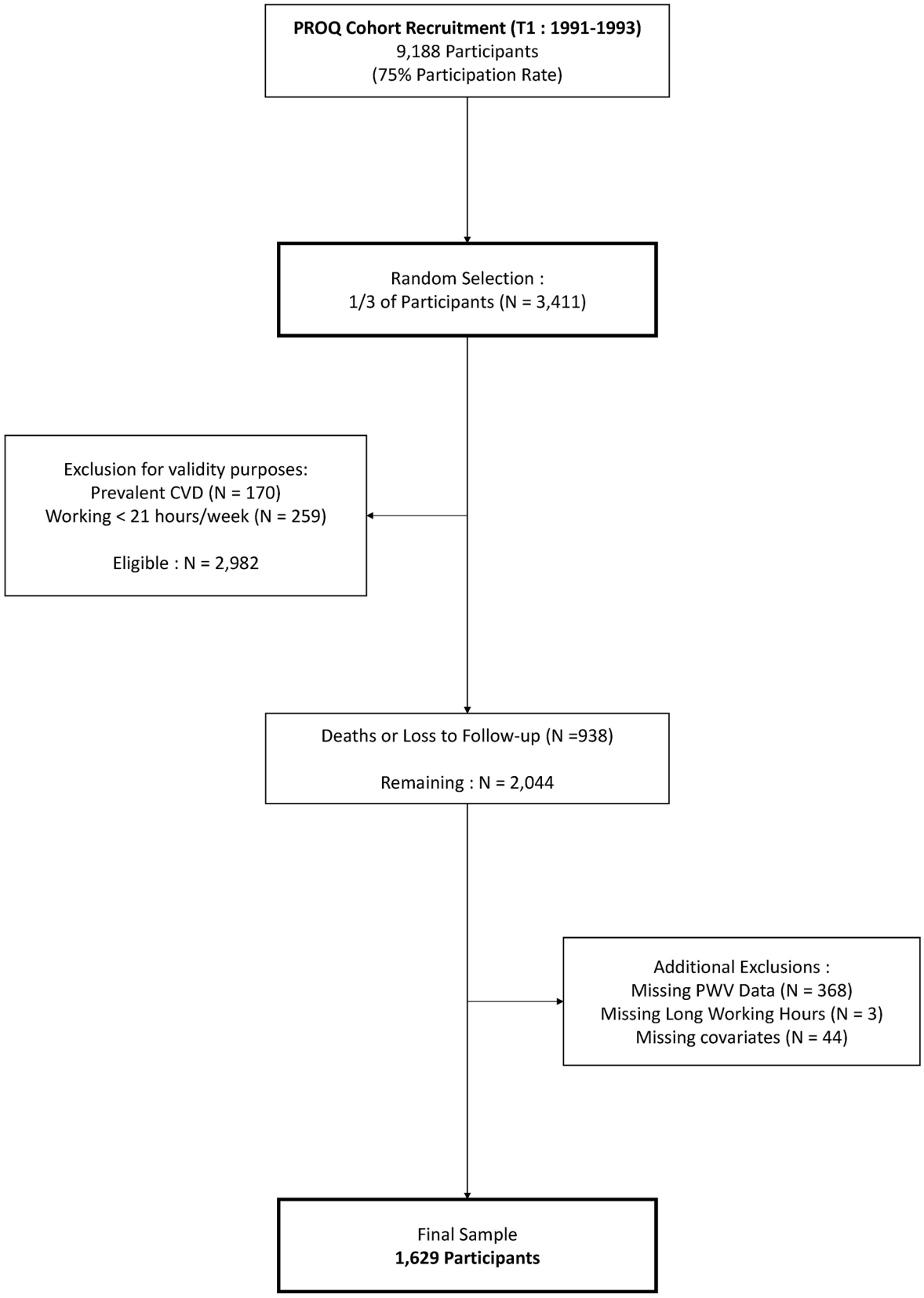

Fig. 1 PROspective Quebec Study Flowchart. footnote: Abbreviation used: Cardiovascular Disease (CVD), Pulse Wave Velocity (PWV)

following categories: ≤ 40 h (reference category) and >40 h per week. Repeated exposure was defined as working more than 40 h per week at both T1 (1991–1993) and T2 (1999–2001). Participants were categorized into three exposure groups: [1] never exposed at T1 nor T2 – reference category [2], exposed once (at either T1 or T2), and [3] exposed at both T1 and T2.

Arterial stiffness

Arterial stiffness was assessed at the last follow-up (T3) using carotid femoral PWV, the gold standard, in accordance with international recommendations [17]. Measurements were performed using the *Complior Analyse Device* (Alam Medical, France) [18], which simultaneously records the carotid and femoral pulse waveforms. PWV was calculated as the ratio of the measured carotid-femoral distance to the pulse transit time between these two sites.

For each participant, PWV was measured twice. If the difference between the two measurements exceeded 0.5 m/s, a third measurement was conducted. The average of all valid measurements was used for analysis. Inter- and intra-observer reproducibility was assessed in previous studies and found to be excellent [19, 20].

Covariates

Sociodemographic variables included age (continuous), sex (men and women), and education (less than college, college completed, and university completed).

Lifestyle-related risk factors included alcohol consumption, smoking status and leisure physical activity. Alcohol consumption was categorized into three categories, based on the weekly frequency of intake: low consumption (fewer than 1 drink per week), moderate consumption (1–10 drinks per week for women and 1–15 for men), and high consumption (more than 10 drinks per week for women and more than 15 for men) [21]. Smoking status was defined as the daily consumption of at least one cigarette per day (yes/no). Leisure physical activity was assessed using a validated question on the duration and frequency of their physical activity: inactive (<1 session per week), insufficiently active (1–2 sessions per week), and active (≥3 sessions per week) [22].

Clinical risk factors included body mass index (BMI), BP and diabetes. BMI was assessed using body weight and height measured by a trained nurse and calculated as the ratio between weight in kilograms and the square of height in meters. BP was measured following the American Heart Association protocol [23] by trained staff using a mercury sphygmomanometer. In brief, BP was measured at rest after the participant had been seated for 5 min. The averages of two BP measurements taken at T1 and three measurements taken at T2, 1 to 2 min apart. Participants were also asked if they had ever been diagnosed with diabetes by a healthcare professional or if they were currently taking medication to manage high BP (antihypertensive medication), with responses categorized as 'yes' or 'no'.

Psychosocial stressors at work from the demand-control model were also assessed, using the validated French version of the Job Content Questionnaire [24, 25]. This model includes psychological demands (9 items), which refer to the quantity of work, time constraints, interruptions, conflicting demands and the intensity of intellectual effort required. It also measures decision latitude (9 items), referring to opportunities for learning, autonomy and participation in the decision-making process. Psychological demands and decision latitude were dichotomized at the median observed in a random sample of the general Quebec working population, with high psychological demands classified as ≥24 and low job control as ≤72 [26]. Job strain was defined as a combination of high psychological demands and low decision latitude.

Analyses

Generalized linear equations were used to examine the association between long working hours and PWV. PWV mean differences and 95% confidence intervals (CI) were computed. Models were adjusted for sociodemographic factors (Model 1), then additionally adjusted for lifestyle-related risk factors, clinical risk factors and psychosocial stressors at work (Model 2). Analyses were conducted in the total sample and separately for actively employed and inactive participants at the time of PWV assessment (T3). Inactive participants were defined as being retired from the workforce or working less than 21 h per week. The potential effect modification by sex and job strain exposure was examined using a multiplicative interaction term with long working hours. This interaction term was not statistically significant in any model (p-value range: 0.44–0.46). Two post-hoc sensitivity analyses were conducted, replacing baseline systolic BP with 1- baseline mean arterial pressure and 2-systolic BP changes over the study period. The present study was approved by the ethics review board of CHU de Quebec- Université Laval. All patients provided informed consent. All analyses were performed with *SAS v.9.4 software*. This study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Results

Table 1 presents the baseline distribution of the population. The study population was composed of 835 women (51.3%) and 794 men (48.7%), resulting in a total of 1,629 participants. The average age of participants was 37 years at baseline (standard deviation [SD]: 6.4) and 46.5% had a university degree. Regarding lifestyle-related risk factors, most participants were non-smokers (84.8%), and

Table 1 Description of the study population at baseline (1991–1993) ($n = 1,629$)

	Total Workers Actively Employed in 1991–1993	Actively Employed Workers in 2015-18	Inactive Participants in 2015-18
	N = 1,629	*N* = 533 (32.7%)	*N* = 1,096 (67.3%)
Sex			
Women	835 (51.3)	286 (53.7)	549 (50.1)
Men	794 (48.7)	247 (46.3)	547 (49.9)
Mean age (year, SD)	37.0 (6.4)	31.2 (5.0)	39.7 (5.1)
Education			
Less than college	366 (22.5)	73 (13.7)	293 (26.7)
College	505 (31.0)	194 (36.4)	311 (28.4)
University	758 (46.5)	266 (49.9)	492 (44.9)
Alcohol intake			
Low	650 (39.9)	238 (44.7)	412 (37.6)
Moderate	929 (57.0)	290 (54.4)	639 (58.3)
High	50 (3.1)	5 (0.9)	45 (4.1)
Smoking	248 (15.2)	79 (14.8)	169 (15.4)
Physical activity			
Inactive	336 (20.6)	196 (36.8)	421 (38.4)
Insufficently active	676 (41.5)	230 (43.2)	446 (40.7)
Active	617 (37.9)	107 (20.0)	229 (20.9)
Mean BMI, (kg/m^2, SD)	24.1 (3.6)	23.7 (3.5)	24.2 (3.6)
Hypertension medication (yes)	23 (1.4)	4 (0.8)	19 (1.7)
Mean Systolic BP, (mmHg, SD)	115.3 (13.2)	114.1 (12.3)	115.9 (13.6)
Mean Diastolic BP, (mmHg, SD)	72.8 (9.8)	70.3 (9.2)	74 (9.8)
Diabetes (yes)	18 (1.1)	5 (1.0)	13 (1.2)
Job strain exposure	351 (21.6)	124 (23.3)	227 (20.7)
Long working hours (> 40 h/week)			
Baseline exposure [1]	121 (7.4)	29 (5.4)	92 (8.4)
Repeated exposure[2]			
Never exposed at T1 nor T2	1444 (88.6)	476 (89.3)	968 (88.3)
Exposed once, at T1 or T2	139 (8.5)	44 (8.3)	95 (8.7)
Exposed at both T1 and T2	46 (2.8)	13 (2.4)	33 (3.0)

Note: SD = Standard Deviation; BP = Blood Pressure; BMI = Body Mass Index

[1] Baseline exposure at T1 (1991–1993)

[2] Repeated exposure at T1 (1991–1993) and T2 (1999–2001)

a majority were moderate drinkers (57.0%). According to physical activity, 41.5% were identified as insufficiently active, 37.9% were active, and 20.6% were inactive. Mean BMI was 24.1 kg/m^2 (SD: 3.6), while mean systolic and diastolic BP were 115.3 mmHg (SD: 13.2) and 72.8 mmHg (SD: 9.8), respectively. A small proportion of participants were taking antihypertensive medication (1.4%) or had diabetes (1.1%). According to occupational risk factors, 21.6% were exposed to job strain and 7.4% were exposed to long working hours at baseline. Participants who remained actively employed over the study period were younger at baseline (31.2 years vs. 39.7 years). This subgroup also had a lower average systolic BP, less alcohol consumption and a lower prevalence of diabetes.

Table 2 presents mean PWV (m/s) in 2015–2018 according to long working hours at baseline (1991–1993) among the total sample ($n = 1,629$), as well as stratified for actively employed workers (subsample $n = 533$) and inactive participants (subsample $n = 1,096$) in 2015–2018 (T3). In the crude models, arterial stiffness was slightly higher among those exposed to long working hours, in the total sample (+0.68 m/s [95% CI (0.37; 0.98]), as well as among actively employed workers (+0.90 m/s [95% CI (0.40; 1.41]) and inactive participants (+0.52 m/s [95% CI (0.15; 0.90]) at T3 (2015–2018). In the fully adjusted models, long working hours at baseline remained associated with arterial stiffness among actively employed workers (+0.54 m/s [95% CI (0.05; 1.02)]). No association was observed in the total sample (+0.12 m/s [95% CI (-0.16; 0.40)]) or among inactive participants (-0.04 m/s [95% CI (-0.38; 0.31)]).

Table 3 presents mean PWV (m/s) in 2015–2018 according to repeated exposure to long working hours at baseline (T1: 1991–1993) and at first follow-up (T2: 1999–2001). In the crude model, mean PWV was higher among participants who were repeatedly exposed to

Table 2 Pulse wave velocity (m/s) in 2015–2018 according to long working hours at baseline (T1: 1991–1993)

Baseline exposure to long working hours (> 40 h/week)		*N*	Pulse Wave Velocity, m/s (95% CI)		
			Crude Model	Adjusted Model 1	Adjusted Model 2
Total (*n* = 1,629)	Unexposed at T1 (ref)	1,508	8.06	8.10	8.10
	Exposed at T1	121	+ 0.68 (+ 0.37, + 0.98)	+ 0.14 (-0.15, + 0.42)	+ 0.12 (-0.16, + 0.40)
Actively Employed Workers in 2015-18 (*n* = 533)	Unexposed at T1 (ref)	504	7.63	7.65	7.65
	Exposed at T1	29	+ 0.90 (+ 0.40, + 1.41)	+ 0.54 (+ 0.05, + 1.03)	+ 0.54 (+ 0.05, + 1.02)
Inactive Participants in 2015-18 (*n* = 1,096)	Unexposed at T1 (ref)	1,004	8.27	8.32	8.32
	Exposed at T1	92	+ 0.52 (+ 0.15, + 0.90)	-0.01 (-0.36, + 0.34)	-0.04 (-0.38, + 0.31)

Ref: Reference category, m/s: meter per second, CI: confidence intervals

Adjusted model 1: Adjusted for age, sex, education

Adjusted model 2: Model 1 plus alcohol intake, smoking, physical inactivity, body mass index, systolic blood pressure, diabetes diagnosis, hypertension medication, and job strain

Table 3 Pulse wave velocity (m/s) in 2015–2018 according to repeated exposure to long working hours at baseline (T1: 1991–1993) and at first follow-up (T2: 1999–2001)

Repeated exposure to long working hours (> 40 h/week)		*N*	Pulse Wave Velocity, m/s (95% CI)		
			Crude Model	Adjusted Model 1	Adjusted Model 2
Total (*n* = 1,629)	Never exposed at T1 nor T2 (ref)	1444	8.06 m/s	8.11 m/s	8.11 m/s
	Exposed once, at T1 or T2	139	+ 0.21 (-0.08; +0.50)	-0.13 (-0.39; +0.14)	-0.16 (-0.42; +0.11)
	Exposed at both T1 and T2	46	+ 0.95 (+ 0.46, + 1.43)	+ 0.25 (-0.20, + 0.70)	+ 0.22 (-0.22, + 0.67)
Actively employed workers at T3 (2015-18) (*n* = 533)	Never exposed at T1 nor T2 (ref)	476	7.64 m/s	7.67 m/s	7.67 m/s
	Exposed once, at T1 or T2	44	-0.14 (-0.55; +0.27)	-0.37 (-0.76; +0.03)	-0.38 (-0.78; +0.01)
	Exposed at both T1 and T2	13	+ 2.07 (+ 1.35, 2.80)	+ 1.46 (+ 0.75; +2.17)	+ 1.50 (+ 0.78, + 2.21)
Inactive participants at T3 (2015-18) (*n* = 1,096)	Never exposed at T1 nor T2 (ref)	968	8.27 m/s	8.33 m/s	8.33 m/s
	Exposed once, at T1 or T2	95	+ 0.36 (-0.01; +0.73)	-0.02 (-0.36; +0.32)	-0.06 (-0.39; +0.28)
	Exposed at both T1 and T2	33	+ 0.46 (-0.14, + 1.06)	-0.23 (-0.79, 0.34)	-0.28 (-0.84, + 0.28)

Ref: Reference category, m/s: meter per second, CI: confidence intervals

Adjusted model 1: Adjusted for age, sex, education

Adjusted model 2: Model 1 plus alcohol intake, smoking, physical inactivity, body mass index, systolic blood pressure, diabetes diagnosis, hypertension medication, and job strain

long working hours when compared to those exposed once, and those never exposed (+ 0.95 m/s [95% CI (0.46; 1.43)]). The association between repeated exposure to long working hours and PWV remained statistically significant among actively employed workers (+ 1.50 m/s [95% CI (0.78; 2.21)]) in the fully adjusted model. In the total sample, the adjusted estimate for repeated exposure was + 0.22 m/s [95% CI (-0.22; 0.67)] and for inactive participants, it was – 0.28 m/s [95% CI (-0.84; 0.28)], neither of which reached statistical significance. Repeated exposure to long working hours remained associated with PWV and estimates were of comparable magnitude in post-hoc analyses, replacing baseline systolic BP with mean arterial pressure or systolic BP changes over the study period (*not shown*).

Discussion

The present study relied on a prospective cohort composed of men and women followed over 24 years. Results suggest that long working hours at midlife are associated with higher arterial stiffness, among participants who remained actively employed over the whole study period. This association was robust to adjustment for socio-demographics, lifestyle-related risk factors, clinical risk factors and psychosocial stressors at work. Workers repeatedly exposed to long working hours had a 1.5 m/s higher PWV, which is of notable clinical importance. For instance, a meta-analysis has shown that a 1 m/s increase in PWV is associated with age-, sex-, and risk factor–adjusted increases in risk of 14% for total CVD events, and 15% for both CVD and all-cause mortality [2].

To our knowledge, the current study is the first to examine the association between repeated exposure to long working hours and arterial stiffness. It is also the first to use carotid-femoral PWV, which is recognized as the reference standard measurement for arterial stiffness [17]. One previous prospective study has reported an association between baseline exposure to long working hours and arterial stiffness progression over 5 years [12], which is consistent with our results. In the current study, ageing workers repeatedly exposed to long working hours at midlife had higher PWV. This result underlines the importance of assessing exposure duration. Indeed, using a single time point exposure to long working hours may lead to misclassification and underestimation of the true effect [27]. Arterial stiffness integrates both the effect and duration of exposure to risk factors. Thus, PWV is a summary measure, capturing arterial damage as product of age and exposure to risk factors, including their progression and cessation over time [28]. Findings from the present study suggest that the harmful effects of long working hours on arterial stiffness may accumulate over the working life leading to higher PWV among older workers with prolonged exposure.

Pathophysiological mechanisms could explain the adverse effect of long working hours on arterial stiffness. Repeated exposure to long working hours may lead to increased activity of the sympathetic nervous system (catecholamines) and the hypothalamic-pituitary-adrenal axis (glucocorticoids). Activation of the sympathetic nervous system is a natural physiological response to life stressors. However, overactivity of the sympathetic nervous system is an important mediator of hypertension and related damage to the cardiovascular system. Measures of arterial stiffness, including PWV, increase with increasing sympathetic activity in healthy subjects and in patients with hypertension or existing CVD, independently of BP [29, 30]. Prolonged exposure to long working hours, through activation of the sympathetic nervous system, may promote progressive damage to arterial walls. Moreover, the sympathetic nervous system is one of the pathways activating the renin-angiotensin system. Therefore, in conjunction with other risk factors, exposure to long working hours can trigger vasoconstriction, endothelial dysfunction, cellular proliferation, and inflammation that promote arterial stiffness [31–33].

Indirect mechanisms could also explain the observed association. First, evidence suggests that long working hours could be associated with sleep deprivation, which could in turn increase cardiovascular risk [34, 35]. Second, individuals exposed to long working hours might be more likely to adopt or maintain unhealthy behaviors including physical inactivity due to lack of time for exercise, higher alcohol consumption and smoking [36]. Third, long working hours could also be associated with prolonged exposure to psychosocial stressors at work [37]. In a recent study conducted by our research team, job strain at midlife was associated with higher PWV among workers with high BP [38]. In the present study, association between long working hours and arterial stiffness was observed following adjustment for lifestyle-related risk factors and psychosocial stressors at work. Therefore, these risk factors unlikely explain the observed association.

Our study has limitations. First, exposure assessment at baseline and follow-up were spaced by approximately 8 years, which could have led to non-differential misclassification of the exposure. However, most workers (82%) remained in the same occupation between T1 and T2, in favor of exposure stability [39]. Second, 7.4% of the study population worked more than 40 h per week and only 2.8% were repeatedly exposed at both times. Therefore, study power was limited, hindering our ability to examine the adverse effect of long working hours using higher thresholds of exposure and to examine a potential dose-response relationship. Third, arterial stiffness was available at the last follow-up only. Therefore, longitudinal progression could not be assessed. Adjusting for BP changes over the study period led to similar estimates suggesting that the longitudinal progression of BP could not explain the observed association. Future studies using multiple assessment of PWV are needed to examine the adverse effect of long working hours on arterial stiffness longitudinal progression. Fourth, excluded participants had distinct cardiometabolic profiles compared to those included, likely due to the exclusion of individuals with prevalent CVD, which was done to ensure internal validity. Notably, no statistically significant association was found between exposure to long working hours and inclusion status ($p = 0.6$), suggesting that selection bias is unlikely. Finally, the generalizability of our findings might be limited to white-collar workers. The association between long working hours and arterial stiffness might differ for other occupational groups more frequently exposed to alternative working arrangements, such as shift work, night work, or irregular schedules [7, 40, 41]. Future research should explore how occupational group may modify the relationship between long working hours and cardiovascular outcomes, particularly in more diverse populations [9].

Our study also has important strengths. First, carotid-femoral PWV was used, which is considered as the gold standard measurement for arterial stiffness [17]. Second, the present study relied on a prospective cohort and long working hours were assessed at two different time points. The 24-year follow-up also allowed to examine the association between long working hours at midlife and arterial stiffness at older age. The association between age and arterial stiffness is characterized by non-linear

increases and faster progression over time [42]. Our long-term follow-up allowed to estimate the effect of long working hours at later stages of this pathogenic process. Moreover, the high participation proportion at each time, and the use of random sample for PWV assessment minimized the potential for selection bias. Finally, a large number of potential confounders were assessed and were adjusted for in the analyses.

Conclusion

The present study examined the association between long working hours and arterial stiffness. Results suggest that long working hours assessed at midlife are associated with higher arterial stiffness, among older workers. This association was of higher magnitude among participants repeatedly exposed to long working hours. Preventive workplace intervention aiming at reducing the prevalence of long working hours are needed to examine potential benefits on cardiovascular health, including the potential for such interventions to mitigate arterial stiffness progression. At the clinical level, the present study suggest that long working hours may be useful for the early identification of workers who may be at increased risk of developing asymptomatic vascular damage.

Abbreviations

CVD	Cardiovascular disease
BP	Blood pressure
WHO/ILO	World health organization and international labor organization
PWV	Pulse wave velocity
PROQ Study	PROspective québec study
BMI	Body mass index
CI	Confidence intervals
SD	Standard deviation

Supplementary Information

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Supplementary material 1

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Author contributions

CBS wrote the manuscript and led the analysis. MGO and MLR participated in revising and editing the manuscript. CB was the cohort principal investigator, supervised the data collection and study design. AM supervised arterial stiffness assessment and clinical interpretation. XT supervised the methodological aspects, reviewed and edited the manuscript, and is responsible for the overall content. All authors contributed significantly to the work and approved the final manuscript for publication.

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Data availability

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request (xavier.trudel@crchudequebec.ulaval.ca).

Declarations

Ethics approval and consent to participate

The study was approved by the ethics review boards of the Centre hospitalier universitaire de Québec - Laval University. Informed consent was obtained from all participants involved in the study.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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