




ORIGINAL RESEARCH

# Respective Mediating Effects of Social Position and Work Environment on the Incidence of Common Cardiovascular Risk Factors

Nicolas Hoertel, MD, MPH, PhD; Marina Sanchez Rico, MPH; Frédéric Limosin , MD, PhD; Joël Ménard, MD, PhD; Céline Ribet, PhD; Sébastien Bonenfant, MSc; Marcel Goldberg , MD, PhD; Marie Zins, MD, PhD; Pierre Meneton , PhD

**BACKGROUND:** Social position and work environment are highly interrelated and their respective contribution to cardiovascular risk is still debated.

**METHODS AND RESULTS:** In a cohort of 20 625 French workers followed for 25 years, discrete-time survival analysis with reciprocal mediating effects, adjusted for sex, age, and parental history of early coronary heart disease, was performed using Bayesian structural equation modeling to simultaneously investigate the extent to which social position mediates the effect of work environment and, inversely, the extent to which work environment mediates the effect of social position on the incidence of common cardiovascular risk factors. Depending on the factor, social position mediates 2% to 53% of the effect of work environment and work environment mediates 9% to 87% of the effect of social position. The mediation by work environment is larger than that by social position for the incidence of obesity, hypertension, dyslipidemia, diabetes, sleep complaints, and depression (mediation ratios 1.32–41.5, 6.67 when modeling the 6 factors together). In contrast, the mediation by social position is larger than that by work environment for the incidence of nonmoderate alcohol consumption, smoking, and leisure-time physical inactivity (mediation ratios 0.16–0.69, 0.26 when modeling the 3 factors together).

**CONCLUSIONS:** The incidence of behavioral risk factors seems strongly dependent on social position whereas that of clinical risk factors seems closely related to work environment, suggesting that preventive strategies should be based on education and general practice for the former and on work organization and occupational medicine for the latter.

**Key Words:** Bayesian structural equation modeling ■ cardiovascular risk factors ■ French cohort ■ social position ■ survival analysis with reciprocal mediating effects ■ work environment

The social conditions in which people live determine for a large part how healthy they are.<sup>1</sup> In particular, individuals of low social status, as measured by educational level, occupational class, or income, are more exposed to common cardiovascular risk factors<sup>2</sup> and have a higher risk of coronary heart disease.<sup>3</sup> Thus, smoking and heavy alcohol consumption as well as leisure-time physical inactivity and obesity are more

prevalent in individuals of low social status,<sup>4–8</sup> thereby increasing their risk of diabetes, hypertension, and dyslipidemia.<sup>6,8</sup> Socially disadvantaged individuals are also more exposed to depression<sup>9</sup> and sleep disorders,<sup>10</sup> which are significant risk factors for cardiovascular diseases.<sup>11,12</sup> The reasons individuals of low social status are more exposed to cardiovascular risk factors are multiple, including educational and cultural attainment

Correspondence to: Pierre Meneton, PhD, INSERM, UMR\_1142, Campus des Cordeliers, 15 rue de l'Ecole de Médecine, 75006 Paris, France. E-mail: pierre.meneton@spim.jussieu.fr

Supplementary Material for this article is available at <https://www.ahajournals.org/doi/suppl/10.1161/JAHA.121.021373>

For Sources of Funding and Disclosures, see page 12.

© 2022 The Authors. Published on behalf of the American Heart Association, Inc., by Wiley. This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

JAHA is available at: [www.ahajournals.org/journal/jaha](http://www.ahajournals.org/journal/jaha)

## CLINICAL PERSPECTIVE

### What Is New?

- The study uses Bayesian structural equation modeling to simultaneously investigate the respective contribution of social position and work environment to the incidence of common cardiovascular risk factors.
- The association of work environment with the incidence of behavioral risk factors (nonmoderate alcohol consumption, smoking, leisure-time physical inactivity) is largely mediated by social position; inversely, the association of social position with the incidence of clinical risk factors (obesity, hypertension, dyslipidemia, diabetes, sleep complaints, depression) is largely mediated by work environment.

### What Are the Clinical Implications?

- Preventive strategies should focus on education and general practice for behavioral risk factors and on work organization and occupational medicine for clinical risk factors.

that can influence risk-prevention behaviors, the ability to cope with illness, and the importance given to the care of one's own health, as well as material deprivations that can determine how well individuals adopt healthy lifestyles and access health care.<sup>13</sup>

The conditions in which people work for several hours every day during several decades also have a major effect on their health.<sup>14</sup> Like individuals of low social status, those with bad working conditions, mostly evaluated by high work stress, are more exposed to common cardiovascular risk factors and have an increased risk of coronary heart disease.<sup>15</sup> Thus, the prevalence of nonmoderate alcohol consumption,<sup>16</sup> smoking,<sup>17</sup> and leisure-time physical inactivity<sup>18</sup> as well as that of obesity,<sup>19</sup> hypertension,<sup>20</sup> and diabetes<sup>21</sup> is higher in individuals with bad working conditions. An adverse work environment also frequently induces chronic psychological stress that can increase the risk of sleep disorders<sup>22</sup> and depression.<sup>23</sup>

Disentangling the respective contributions of social position and work environment to cardiovascular risk is difficult because they are highly interrelated; the better one's social position, the better one's work environment tends to be.<sup>24</sup> However, working conditions are not completely determined by social position, including occupational grade, as these conditions can vary substantially for the same job.<sup>25</sup> This complexity is illustrated by the literature that reports reciprocal mediations and/or moderations between social position and work environment in the determination of cardiovascular risk. Thus, several studies suggest that

working conditions mediate to some extent the association between social status and cardiovascular risk.<sup>26</sup> For example, skill discretion (ie, the opportunity to develop new skills at work independently of previous education) partially mediates the effect of occupation on the incidence of myocardial infarction in 3 prospective population studies conducted in Copenhagen<sup>27</sup> and job control contributes significantly to the association between occupational grade and the incidence of coronary heart disease in the Whitehall II study.<sup>28</sup> We recently reported that work environment may mediate a large part of social inequalities in the incidence of common cardiovascular risk factors in a cohort of French workers.<sup>29</sup> This mediating effect varies substantially from one risk factor to another, explaining 30% to 40% of social gradients in the risk of leisure-time physical inactivity, obesity, diabetes, and dyslipidemia and 60% to 90% of gradients in the risk of hypertension, sleep complaints, and depression.

Inversely, some studies suggest that the association between working conditions and cardiovascular risk is influenced by social status. For example, the increased risk of myocardial infarction associated with low job control and adverse physical working conditions in the Netherlands longitudinal GLOBE (Gezondheid en Levens Omstandigheden Bevolking Eindhoven) study<sup>30</sup> is substantially attenuated after adjusting for education and occupation. The increase in cardiovascular mortality associated with long working hours in the Northern Ireland mortality study<sup>31</sup> as well as the association between job strain and the risk of coronary heart disease in North Italian employed men<sup>32</sup> are also strongly dependent on occupational class. Likewise, the increased cardiovascular mortality associated with job strain and effort-reward imbalance at work in the Valmet study is significantly smaller after controlling for education or occupation.<sup>33</sup> Although work environment cannot be seen as determining social position, both simultaneously influence at any moment the incidence of cardiovascular risk factors and the association of this incidence with work environment may also be partly explained by social position. This hypothesis was tested by performing discrete-time survival analyses with reciprocal mediating effects between work environment and social position using Bayesian structural equation modeling in the same cohort of French workers previously used.<sup>29</sup>

## METHODS

The data underlying the findings of this study are not publicly available for legal reasons related to data privacy protection. The GAZEL (Gaz and Electricité) cohort has a data sharing policy but a legal authorization must first be obtained from the French National Committee

for the Protection of Privacy and Civil Liberties. Email address to contact the staff is gaze@inserm.fr.

## Study Population

The analyses were performed in a cohort of 20 625 middle-aged individuals working at the French National Gas and Electricity Company and followed since 1989 (GAZEL cohort).<sup>34</sup> These workers, aged 35 to 50 at inception, lived throughout French metropolitan territory in various settings from rural areas to urban centers and were very diverse in terms of socioeconomic status, health, and health-related behaviors.<sup>35</sup> They were very motivated to participate in the cohort as indicated by the high acceptance rate at the time of recruitment (45%) and the very low attrition rate during follow-up (<1%). The response rate to annual self-administered questionnaires also remained high throughout follow-up (average of 75%) with only <5% of the individuals included in the cohort who never sent back any questionnaire. All workers sent written informed consent to participate in the study, which received approval from both the Ethics Evaluation Committee of the French National Institute of Health and Medical Research and the National Committee for the Protection of Privacy and Civil Liberties.

Compared with individuals in the same age range randomly selected from the French population,<sup>36</sup> cohort participants were less exposed to cardiovascular risk factors such as smoking, physical inactivity, and obesity (Table S1). Because of the industrial nature of the company, the sex ratio was unbalanced in favor of men and the social gradient was reduced with an overrepresentation of secondary educational level, intermediate occupational grade, middle income class, and an underrepresentation of primary educational level, blue collar/clerk occupational grade, low income class (Table S1).

## Assessment of Social Position

Four self-reported socioeconomic indicators whose distribution is shown in Table S2 were considered at baseline. Educational attainment was classified into 3 levels: university, secondary school, or primary school. Wealth included financial and housing assets minus liabilities of all household members and was divided into 3 classes: the rich, the middle class, or the poor who respectively declared over i304 898, between i76 225 and i304 898, or <i76 225. Income comprised monthly earnings of all household members and was ranked as high (above i3811), middle (between i2592 and i3811) or low (below i2592). These thresholds for wealth and income were dictated by the inquiry that originally included 10 categories for each indicator and the need to balance the number of workers between the groups. Occupation was reduced from a 10-level classification in the original inquiry to 3 grades: management, intermediate, or blue collar/clerk.

Given that these indicators represent interdependent and complementary aspects of social position and that their effects accumulate to some extent, a global measure was calculated by giving for each indicator a score of 1 to the less favored group, 3 to the most favored group, and 2 to the intermediary group, by summing the scores and by dividing the sum by the number of available indicators for each worker. For the analyses, this global measure, whose distribution is shown in Figure S1, was divided into 3 groups (high, middle, low) reproducing as much as possible the average distribution of socioeconomic indicators. As described in Figure S2, it is highly correlated to these indicators.

## Assessment of Work Environment

A total of 25 self-reported occupational exposures were used to characterize working conditions at baseline (Table S3), as previously described.<sup>37</sup> These include a series of physical, biomechanical, and organizational factors such as commuting time, working with the public, outdoor work, night shift work, regular work hours, on-call work, standing work posture, hard work posture, handling heavy loads, exposure to vibrations, working with a screen, working in the cold, working in the heat, exposure to noise, work involving specific risks (electrocution, gas intoxication, falls, machine injuries, burns, or road traffic accidents), and work administratively classified as unhealthy. Subjective factors as the extent to which work was considered to be physically demanding, nerve racking, or satisfactory were also retained, as well as psychosocial factors (decision latitude, psychological demand, social support at work, extrinsic effort, reward, overcommitment) that were assessed using the job content questionnaire<sup>38</sup> and the effort-reward imbalance score.<sup>39</sup>

These occupational exposures were not considered separately but combined into a global measure of work environment that was calculated by giving for each exposure a score of 1 to the nonexposed group, 2 to the exposed group, and 1.5 to the intermediary group whenever the exposure encompasses 3 levels, by summing the scores and by dividing the sum by the number of available exposures for each worker. This global measure, whose distribution is shown in Figure S3, was divided into tertiles (good, average, bad) for the analyses. As shown by Table S4, it is highly correlated to the global measure of social position ( $P<0.0001$ ): the worse the work environment, the lower the social position.

## Determination of Cardiovascular Risk Factors

Twelve self-reported risk factors that have previously been shown to be independent predictors of cardiovascular events in the cohort<sup>40</sup> were retained for the analyses. Three nonmodifiable risk factors were used

as model covariates for adjustment purpose at baseline: sex, age divided into tertiles, and parental history of early coronary heart disease coded as a binary variable that referred to the occurrence of the disease before the age of 60 on father's or mother's side. Nine modifiable risk factors were used as binary outcome variables in mediation models: smoking, nonmoderate alcohol consumption (<14 or >27 drinks/week in men, <7 or >20 drinks/week in women), leisure-time physical inactivity, obesity (body mass index  $\geq 30$  kg/m<sup>2</sup>), hypertension, dyslipidemia, diabetes, sleep complaints, and depression. The inquiry into the occurrence of hypertension, dyslipidemia, diabetes, and sleep complaints asked to report the condition if it appeared during the past year. Body mass index was calculated from reported weight and height values. Depression was assessed with the Centre of Epidemiologic Studies Depression scale and defined as a score  $\geq 17$  in men and  $\geq 23$  in women.<sup>41</sup> The inquiry into alcohol consumption and smoking referred to habits during the week before filling in the questionnaires. Leisure-time physical inactivity was defined by the lack of sport practice whatever its frequency (occasionally, regularly, or competition).

## Statistical Analysis

Bayesian structural equation modeling was used to perform discrete-time survival analyses with reciprocal mediating effects<sup>42–44</sup> in order to simultaneously estimate the extent to which the association between work environment and the incidence of modifiable cardiovascular risk factors may be explained by social position and vice versa, assuming that the effect of social position is the same across work environments and that the effect of work environment is the same across social positions. These analyses combine prior distributions for parameters with the data likelihood to form posterior distributions for the parameter estimates. In these analyses, in the absence of precise data about the relationships examined, diffuse (ie, noninformative) priors were used as the default.<sup>45</sup> The choice to use Bayesian statistics was driven by several reasons: (1) more can be learned about parameter estimates that do not have a normal distribution, (2) complex types of models comprising a substantial number of parameters or disparate types of data like those presented in this report can be analyzed, and (3) analyses can be made less computationally demanding despite the fact that models include numerous categorical outcomes and latent variables resulting in many dimensions of numerical integration that are computationally cumbersome or sometimes impossible using maximum likelihood estimation.<sup>45</sup>

The occurrence of each risk factor was self-reported every year from 1990 to the year of the first detection of the factor or to the year of the last completed questionnaire, whichever occurred first in

workers who were not exposed to the factor at baseline, up to 2014 (25 years of follow-up at most with an average duration of  $20.5 \pm 7.8$  [SD] years). Workers lost ( $n=619$ ) or who died ( $n=707$ ) during follow-up were not excluded, nor were those who have had nonfatal cardiovascular events ( $n=1694$ ) because the number of events occurring before the first detection of risk factors, that is, the situation in which these events can have potential confounding effects, was negligible compared with the number of incident risk factors. We chose to extend the follow-up after retirement given that social position can exert its effect before and after retirement and that risk factors whose incidence is influenced by work environment early during the working period can affect the incidence of other risk factors later after retirement. All models used high social position and good work environment as reference groups and were adjusted for nonmodifiable risk factors (sex, age, parental history of early coronary heart disease) but not for modifiable risk factors in order to avoid adjustment for potential descendants, that is, consequences rather than causal antecedents of measured outcomes.

Latent variables in the models represent the propensity to have each risk factor during follow-up and were measured by 25 discrete-time survival indicators, that is, by the occurrence of each risk factor at each year of the 25-year follow-up. These latent variables (labeled with the name of each risk factor) merely simplify the presentation as the direct effect of each explanatory variable (social position, work environment and for adjustment purpose, sex, age, parental history of early coronary heart disease) on survival indicators can be identified with a single path. This specification is equivalent to one in which each explanatory variable has an effect on each of the 25 discrete-time survival indicators with these 25 effects being constrained to be equal. As the entire model is linear (because the discrete-time survival part of the model may be interpreted in terms of a linear regression using a latent response formulation), indirect effects can be estimated using the product-of-coefficients approach and represent “natural indirect effects.”<sup>43,46</sup> Direct effects in the models are equivalent to “natural direct effects” whereas the total effect is given by the sum of direct and indirect effects.<sup>47</sup> More specifically, because our models simultaneously estimate the bidirectional association between social position and work environment, in the models testing risk factors separately, indirect effects represent both the mediating effect of work environment in the association between social position and the incidence of each risk factor and the mediating effect of social position in the association between work environment and the incidence of each risk factor; direct effects represent the effect of social position on the incidence of each risk factor that is not mediated by work environment as well

as the effect of work environment on the incidence of each risk factor that is not mediated by the social position. In the models testing simultaneously behavioral, clinical, or all risk factors, the indirect effects represent respectively the total mediating effects of work environment in the associations between social position and the incidence of risk factors (ie, the total indirect effect obtained by summing indirect effects for each risk factor) as well as the total mediating effects of social position in the associations between work environment and the incidence of risk factors; the direct effects represent the sum of the effects of work environment on the incidence of risk factors that are not mediated by social position and of the effects of social position on the incidence of risk factors that are not mediated by work environment.

Note that the associations were assessed using a probit link. The probit regression coefficients give the change in the Z score or probit index for 1 unit in the predictor and cannot be interpreted as conventional effect sizes.<sup>48</sup> Significance of estimates were evaluated using Bayesian 95% credibility interval of the posterior parameter distributions, which allows for a strongly nonnormal distribution.<sup>49</sup> The size of the mediating effects was calculated both in absolute terms and as mediation proportion 95% credibility intervals. The latent variables underlying survival indicators have a mean of 0 and an SD of 1 and thus the raw coefficients may be interpreted as capturing the effect, measured in SDs, of a unit change in explanatory variables.<sup>50</sup> The mediation models allow for the possibility of compensatory effects, that is, that some indirect effects are positive and others are negative.<sup>51</sup> For example, they would explain the association between work environment and the incidence of each risk factor if the total indirect effect (ie, the effect of work environment that is mediated by social position) would be positive with no additional (ie, direct) effect of work environment on the incidence of each risk factor. Because all path coefficients were simultaneously examined, no paths in any of the models were set to 0. Therefore, goodness-of-fit measures are not relevant in evaluating these models because they do not inform on the “correctness” of the models but rather only provide a summary of how well the observed correlations match the models when several paths are set to 0.<sup>46</sup> It is important to carefully consider convergence in Bayesian analyses. The convergence criterion used is that a proportional scale reduction factor is close enough to 1 for each parameter. Bayesian analyses use Markov chain Monte Carlo algorithms to iteratively obtain an approximation to the posterior distributions of the parameters. The proportional scale reduction approach to determining convergence compares the parameter variation within each chain to that across chains to make sure that the different chains do not converge to different values. The proportional

scale reduction criterion essentially requires the between-chain variation to be small relative to the total of between- and within-chain variation.<sup>52</sup> To gain further evidence of convergence, each model was run with longer chains (using Mplus option FBITERATIONS in the ANALYSIS command to request a fixed number of Bayes iterations up to 10 000) while checking that the parameter values did not significantly change and that the proportional scale reduction remained close to 1. In addition, the results of each model were tested for their sensitivity to prior distributions, hypothesized normal, by specifying different combinations of estimates and variances. All structural equation models were implemented by using the software Mplus 7.1.<sup>45</sup>

## RESULTS

### Prevalence of Cardiovascular Risk Factors at Baseline and Their Incidence During Follow-Up According to Social Position and Work Environment

The prevalence of most risk factors at baseline is inversely associated with social position and work environment as reported in Table 1. The exceptions are the prevalence of sex, age, nonmoderate alcohol consumption, and dyslipidemia that are directly associated with social position, the prevalence of age being also directly associated with work environment. Likewise, the incidence of modifiable risk factors during follow-up is inversely associated with social position and work environment except for the incidence of nonmoderate alcohol consumption, which is directly associated with social position (Table 2).

### Reciprocal Mediating Effects of Work Environment and Social Position on the Incidence of Each Cardiovascular Risk Factor

Low social position and bad work environment at baseline are associated with an increased incidence of each cardiovascular risk factor during follow-up as shown by estimates of total effects (all  $P < 0.001$ ) (Figure 1 and Table 3). Work environment has a significant mediating effect on the association between social position and the incidence of each risk factor with a mediation proportion ranging from 9% to 87% depending on the factor (all  $P < 0.001$ ) (Table 3). Social position also has a significant mediating effect on the association between work environment and the incidence of each risk factor with a mediation proportion ranging from 2% to 53% (all  $P < 0.001$  except for depression [ $P < 0.01$ ] and diabetes [ $P < 0.05$ ]) (Table 3). It is noteworthy that the mediating effect of work environment is larger than that of social position

**Table 1. Prevalence of Cardiovascular Risk Factors at Baseline According to Social Position and Work Environment**

	Social position			Work environment		
	High (n=4666)	Middle (n=11 217)	Low (n=4740)	Good (n=6677)	Average (n=6994)	Bad (n=6947)
Sex						
Men	82.7	72.2	64.4	62.5	71.8	83.6
Women	17.3	27.8	35.6	37.5	28.2	16.4
Age, y						
35–41	29.5	32.8	36.8	33.5	33.8	31.8
42–45	33.9	33.8	30.4	29.4	32.6	36.9
46–50	36.6	33.4	32.8	37.1	33.6	31.3
Parental history of early coronary heart disease						
No	88.8	87.8	86.3	88.6	88.2	86.5
Yes	11.2	12.2	13.7	11.4	11.8	13.5
Nonmoderate alcohol consumption						
No	36.0	36.8	41.9	39.0	36.9	37.5
Yes	64.0	63.2	58.1	61.0	63.1	62.5
Smoking						
No	72.6	71.8	69.6	73.6	72.2	68.8
Yes	27.4	28.2	30.4	26.4	27.8	31.2
Leisure-time physical inactivity						
No	75.2	66.4	57.7	67.1	66.7	66.2
Yes	24.8	33.6	42.3	32.9	33.3	33.8
Obesity						
No	96.5	95.1	93.2	96.1	95.6	93.5
Yes	3.5	4.9	6.8	3.9	4.4	6.5
Hypertension						
No	92.6	91.4	90.8	92.3	92.1	90.2
Yes	7.4	8.6	9.2	7.7	7.9	9.8
Dyslipidemia						
No	87.0	88.0	88.3	89.8	88.2	85.7
Yes	13.0	12.0	11.7	10.2	11.8	14.3
Diabetes						
No	98.8	98.6	97.9	98.9	98.7	98.0
Yes	1.2	1.4	2.1	1.1	1.3	2.0
Sleep complaints						
No	83.5	81.2	77.3	84.5	81.0	77.0
Yes	16.5	18.8	22.7	15.5	19.0	23.0
Depression						
No	81.2	76.2	67.4	83.0	76.0	68.8
Yes	18.8	23.8	32.6	17.0	24.0	31.2

The percentages refer to the number of workers in each social position or work environment.

on the incidence of obesity, hypertension, dyslipidemia, diabetes, sleep complaints, and depression (the mediation ratio, estimating the ratio of the mediating effect by work environment to the one by social position, ranges from 1.32 to 41.5) whereas the opposite is true on the incidence of nonmoderate alcohol consumption, smoking, and leisure-time physical inactivity (mediation ratio ranging from 0.16 to 0.69) (Table 3). Note that concerning the incidence of depression, using different cutoff values on the Centre of Epidemiologic Studies Depression scale (ie, 17 in men and 23 in women or 19 in both sexes) does not

change the observation that the mediating effect of work environment is much larger than that of social position (mediation ratio 22.4, Table S5).

### Reciprocal Mediating Effects of Work Environment and Social Position on the Incidence of Behavioral, Clinical, or All Cardiovascular Risk Factors

When modeling simultaneously the incidence of nonmoderate alcohol consumption, smoking, and leisure-time physical inactivity, the mediating effects of work

**Table 2. Incidence of Modifiable Cardiovascular Risk Factors During the 25-Year Follow-Up According to Social Position and Work Environment**

	Social position			Work environment		
	High (n=4666)	Middle (n=11 217)	Low (n=4740)	Good (n=6677)	Average (n=6994)	Bad (n=6947)
Nonmoderate alcohol consumption	28.6	27.5	22.7	25.0	27.2	27.6
Smoking	4.7	4.7	5.6	4.5	5.0	5.1
Leisure-time physical inactivity	15.1	15.9	16.9	14.5	15.8	17.3
Obesity	5.4	6.7	7.6	5.3	6.3	8.1
Hypertension	13.8	14.2	15.2	12.2	14.1	15.4
Dyslipidemia	16.2	16.9	17.5	15.2	16.4	18.1
Diabetes	3.7	4.1	4.6	3.0	4.0	5.1
Sleep complaints	20.2	21.7	22.4	18.6	22.0	23.9
Depression	9.7	10.2	10.1	7.1	10.2	12.9

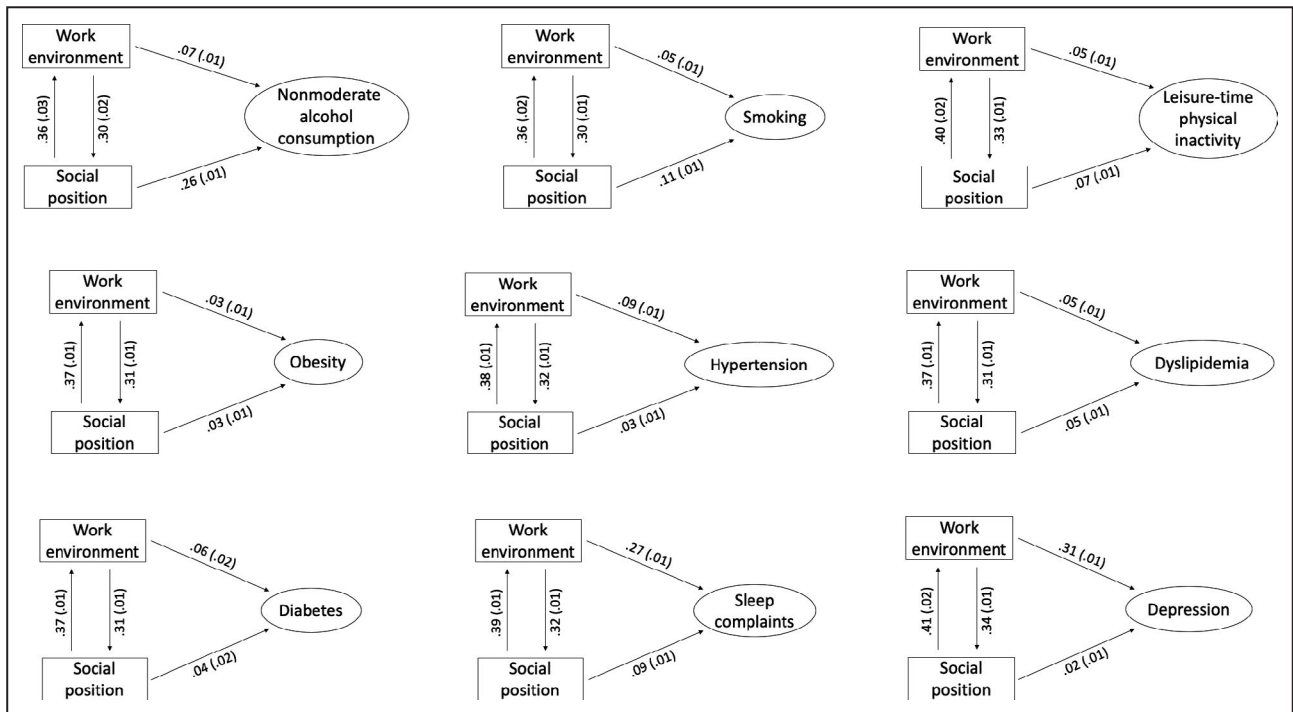
The incidence is expressed as the number of cases/1000 person-years in each social position or work environment.

environment and social position represent respectively 12% and 46% of the global effects of social position and work environment on the incidence of these risk factors (all  $P < 0.001$ ) with a mediation ratio of 0.26 (Figure 2 and Table 4). The mediating effects of work environment and social position are respectively 58% and 9% (all  $P < 0.001$ , mediation ratio 6.67) when modeling simultaneously the incidence of obesity, hypertension, dyslipidemia, diabetes, sleep complaints, and depression, and 41% and 14% (all  $P < 0.001$ , mediation

ratio 3.03) when modeling the incidence of all risk factors together (Figure 2 and Table 4).

### Reciprocal Mediating Effects of Work Environment and Social Position on the Incidence of Behavioral and Clinical Cardiovascular Risk Factors by Sex

To explore potential sex differences that could be masked in aggregate analyses, we assessed the



**Figure 1. Discrete-time survival analysis with reciprocal mediating effects by social position and work environment on the incidence of each cardiovascular risk factor using Bayesian structural equation modeling.**

Explanatory variables are represented by rectangles, latent variables by ellipses, and direct effects by straight arrows pointing from cause to effect with estimates and SDs in parenthesis. Note that explanatory variables used for adjustment purposes (sex, age, parental history of early coronary heart disease) are included in each model but not represented in the figure.

**Table 3. Discrete-Time Survival Analysis With Reciprocal Mediating Effects by Social Position and Work Environment on the Incidence of Each Cardiovascular Risk Factor Using Bayesian Structural Equation Modeling**

	Assuming that work environment mediates the effect of social position on the incidence			Assuming that social position mediates the effect of work environment on the incidence			Mediation ratio (mediation by work environment/mediation by social position)
	Estimate	SD	95% CI	Estimate	SD	95% CI	
Nonmoderate alcohol consumption							
Total effect	0.281*	0.005	0.272–0.290	0.142*	0.009	0.127–0.159	
Indirect effect	0.024*	0.003	0.018–0.030	0.077*	0.005	0.066–0.086	
Mediation, %	0.085*	0.011	0.064–0.106	0.533*	0.038	0.470–0.620	0.16
Smoking							
Total effect	0.132*	0.009	0.114–0.149	0.085*	0.009	0.066–0.100	
Indirect effect	0.018*	0.003	0.013–0.023	0.034*	0.003	0.029–0.039	
Mediation, %	0.137*	0.015	0.107–0.165	0.405*	0.031	0.356–0.473	0.34
Leisure-time physical inactivity							
Total effect	0.089*	0.007	0.077–0.102	0.072*	0.007	0.058–0.086	
Indirect effect	0.020*	0.002	0.015–0.024	0.023*	0.002	0.019–0.027	
Mediation, %	0.219*	0.017	0.184–0.249	0.316*	0.023	0.278–0.366	0.69
Obesity							
Total effect	0.042*	0.013	0.019–0.062	0.042*	0.009	0.028–0.060	
Indirect effect	0.012*	0.002	0.009–0.017	0.009*	0.003	0.003–0.014	
Mediation, %	0.290*	0.075	0.237–0.512	0.220*	0.046	0.097–0.268	1.32
Hypertension							
Total effect	0.066*	0.007	0.053–0.079	0.100*	0.007	0.086–0.112	
Indirect effect	0.034*	0.003	0.029–0.039	0.010*	0.002	0.007–0.014	
Mediation, %	0.510*	0.034	0.456–0.583	0.103*	0.012	0.080–0.127	4.92
Dyslipidemia							
Total effect	0.066*	0.009	0.050–0.085	0.067*	0.008	0.052–0.083	
Indirect effect	0.020*	0.002	0.015–0.024	0.014*	0.002	0.010–0.019	
Mediation, %	0.293*	0.025	0.253–0.353	0.216*	0.020	0.174–0.255	1.36
Diabetes							
Total effect	0.066*	0.029	0.010–0.107	0.071*	0.025	0.026–0.109	
Indirect effect	0.021*	0.007	0.009–0.033	0.014†	0.007	0.001–0.023	
Mediation, %	0.330*	0.186	0.286–0.948	0.187†	0.054	0.005–0.220	1.72
Sleep complaints							
Total effect	0.202*	0.012	0.180–0.221	0.303*	0.010	0.285–0.322	
Indirect effect	0.106*	0.005	0.096–0.114	0.030*	0.004	0.025–0.038	
Mediation, %	0.529*	0.022	0.482–0.562	0.099*	0.009	0.087–0.119	5.32
Depression							
Total effect	0.145*	0.011	0.125–0.167	0.316*	0.009	0.298–0.333	
Indirect effect	0.126*	0.006	0.114–0.139	0.007‡	0.002	0.002–0.012	
Mediation, %	0.866*	0.040	0.791–0.951	0.021‡	0.007	0.007–0.035	41.5

Each model included sex, age, and parental history of early coronary heart disease for adjustment purposes. Estimates are reported with SDs and 95% credibility intervals (95% CI).

\*P<0.001.

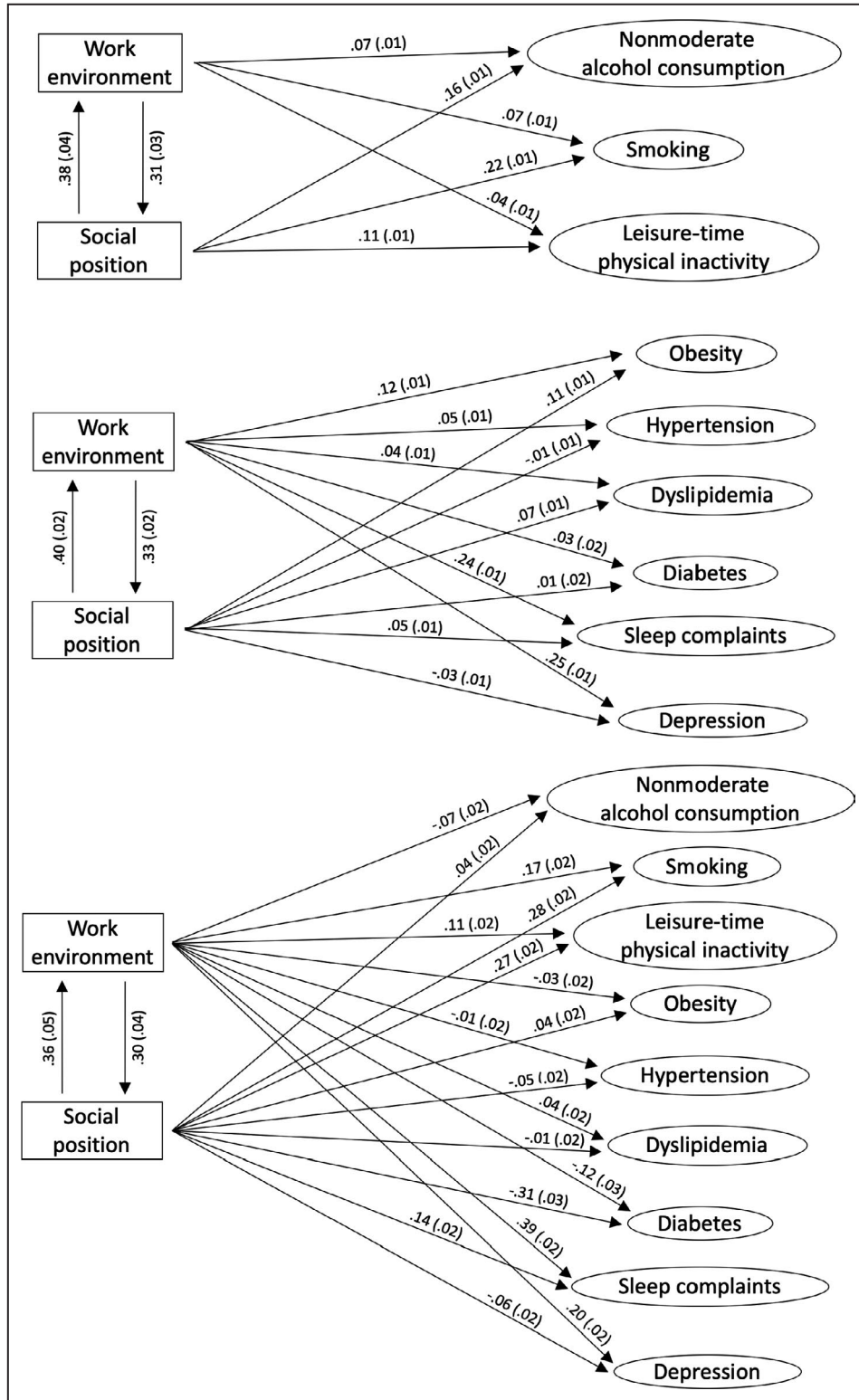
†P<0.05.

‡P<0.01.

reciprocal mediating effects of work environment and social position on the incidence of behavioral or clinical risk factors, separately in men and women. In both sexes, the mediation ratios are well below 1 when modeling simultaneously the incidence of nonmoderate

alcohol consumption, smoking, and leisure-time physical inactivity (0.15 in men and 0.07 in women) whereas they are well above 1 when modeling simultaneously the incidence of obesity, hypertension, dyslipidemia, diabetes, sleep complaints, and depression (6.56 in





**Figure 2.** Discrete-time survival analysis with reciprocal mediating effects by social position and work environment on the incidence of behavioral, clinical, or all cardiovascular risk factors using Bayesian structural equation modeling.

Explanatory variables are represented by rectangles, latent variables by ellipses, and direct effects by straight arrows pointing from cause to effect with estimates and SDs in parenthesis. Note that explanatory variables used for adjustment purpose (sex, age, parental history of early coronary heart disease) are included in the models but not represented in the figure.

**Table 4. Discrete-Time Survival Analysis With Reciprocal Mediating Effects by Social Position and Work Environment on the Incidence of Behavioral, Clinical, or All Cardiovascular Risk Factors Using Bayesian Structural Equation Modeling**

	Assuming that work environment mediates the effect of social position on the incidence			Assuming that social position mediates the effect of work environment on the incidence			Mediation ratio (mediation by work environment/mediation by social position)
	Estimate	SD	95% CI	Estimate	SD	95% CI	
Behavioral risk factors							
Total effect	0.564*	0.026	0.516–0.618	0.335*	0.029	0.281–0.393	
Indirect effect	0.067*	0.011	0.049–0.091	0.154*	0.016	0.123–0.188	
Mediation, %	0.119*	0.015	0.092–0.152	0.461*	0.035	0.392–0.530	0.26
Clinical risk factors							
Total effect	0.491*	0.031	0.435–0.551	0.789*	0.025	0.745–0.837	
Indirect effect	0.285*	0.016	0.255–0.317	0.069*	0.008	0.053–0.084	
Mediation, %	0.580*	0.024	0.537–0.634	0.087*	0.008	0.070–0.102	6.67
All risk factors							
Total effect	0.594*	0.072	0.462–0.747	0.781*	0.069	0.648–0.919	
Indirect effect	0.243*	0.038	0.173–0.322	0.105*	0.021	0.069–0.153	
Mediation, %	0.409*	0.046	0.323–0.503	0.135*	0.022	0.095–0.180	3.03

The models included sex, age, and parental history of early coronary heart disease for adjustment purpose and tested either nonmoderate alcohol consumption, smoking and leisure-time physical inactivity together (behavioral risk factors), obesity, hypertension, dyslipidemia, diabetes, sleep complaints and depression together (clinical risk factors), or all risk factors together. Estimates are reported with SDs and 95% credibility intervals (95% CI).

\*P<0.001.

men and 7.21 in women) (Table S6). These results suggest that the very different mediating effects of social position and work environment on the incidence of behavioral and clinical risk factors are present in both sexes.

### Reciprocal Mediating Effects of Work Environment and Social Position on the Incidence of Behavioral and Clinical Cardiovascular Risk Factors When Social Position Is Assessed by Specific Socioeconomic Indicators

As the global measure of social position combining the 4 socioeconomic indicators may weaken the observed associations if some indicators are more weakly linked to the incidence of risk factors than others, we assessed the reciprocal mediating effects of work environment and each socioeconomic indicator on the incidence of behavioral or clinical risk factors. For each indicator, the mediation ratios (mediation by work environment/mediation by socioeconomic indicator) are well below 1 when modeling simultaneously the incidence of no-moderate alcohol consumption, smoking, and leisure-time physical inactivity whereas they are well above 1 when modeling simultaneously the incidence of obesity, hypertension, dyslipidemia, diabetes, sleep complaints, and depression (Table S7). The figures are respectively 0.43 and 8.94 for education, 0.22 and 5.53 for wealth, 0.09 and 13.3 for income, and 0.38

and 8.14 for occupational grade. These results suggest that education, wealth, income, and occupation are similarly involved in the determination of behavioral and clinical risk factors.

### Reciprocal Mediating Effects of Work Environment and Social Position on the Incidence of Behavioral and Clinical Cardiovascular Risk Factors According to Follow-Up Duration

Given that social position and work environment were assessed only at baseline, we cannot exclude that the 2 variables improved over time at different rates as workers moved up the job ladder, thus potentially modifying the magnitude of their association. To test this possibility, we have assessed the reciprocal mediating effects of work environment and social position on the incidence of behavioral and clinical risk factors after 12 years of follow-up. The mediation ratio is well below 1 (0.25) when modeling simultaneously the incidence of nonmoderate alcohol consumption, smoking, and leisure-time physical inactivity whereas it is well above 1 (5.03) when modeling simultaneously the incidence of obesity, hypertension, dyslipidemia, diabetes, sleep complaints, and depression (Table S8). These results, which are very similar to those observed after 25 years of follow-up, suggest that the magnitude of the association between social position and work environment remains relatively constant over time.

## Sensitivity to Prior Distributions and Convergence of Proportional Scale Reduction in the Model Evaluating Reciprocal Mediating Effects by Social Position and Work Environment on the Incidence of Depression

The sensitivity of mediation ratios (mediation by work environment/mediation by social position) to prior distributions was very small in all models that adequately converged with proportional scale reduction values close to 1 after 1000 iterations. As an example, the sensitivity and convergence are reported in Tables S9 and S10 for the model evaluating reciprocal mediating effects by social position and work environment on the incidence of depression.

## DISCUSSION

In the present study, we found that low social position is associated with a higher incidence of cardiovascular risk factors as expected from the literature that reports inverse associations between several indicators of socioeconomic status and the prevalence and/or incidence of sleep disorders,<sup>10</sup> depression,<sup>9</sup> diabetes,<sup>6</sup> obesity,<sup>7</sup> smoking,<sup>6,8</sup> leisure-time physical inactivity,<sup>4</sup> heavy alcohol consumption,<sup>5</sup> dyslipidemia,<sup>6,8</sup> and hypertension<sup>6,8</sup> in populations from high-income countries. We also find that bad work environment is associated with an increased incidence of cardiovascular risk factors in agreement with studies reporting such inverse associations with specific occupational exposures. For example, job strain has been associated with increased risk of nonmoderate alcohol consumption,<sup>16</sup> smoking,<sup>17</sup> leisure-time physical inactivity,<sup>18</sup> obesity,<sup>19</sup> hypertension,<sup>20</sup> diabetes,<sup>21</sup> sleep disorders,<sup>22</sup> and depression<sup>23</sup> in several populations.

Our analyses indicate that work environment has a mediating effect on the associations between social position and the incidence of risk factors in line with studies reporting that psychosocial exposures at work contribute to the link between socioeconomic status and the incidence of coronary heart disease.<sup>26–28</sup> The mediating effect of work environment varies largely from one risk factor to another, explaining 9% to 87% of the associations between social position and the incidence of these factors. Inversely, we observe that social position has a mediating effect on the associations between work environment and the incidence of risk factors, which also varies largely depending on the factor, explaining 2% to 53% of the associations. This observation is in agreement with data suggesting that the association between working conditions and cardiovascular risk is influenced by socioeconomic status, although mediation was not assessed per se in these studies.<sup>30–33</sup>

The important finding is that the relative mediating effects of work environment and social position on the incidence of risk factors are very different depending on the nature of these factors. Indeed, both in men and women, the mediating effect of social position is 4-fold higher than that of work environment on the incidence of nonmoderate alcohol consumption, smoking, and leisure-time physical inactivity whereas it is more than 6-fold smaller than that of work environment on the incidence of obesity, hypertension, dyslipidemia, diabetes, sleep complaints, and depression. In other words, although social position and work environment are highly interrelated,<sup>24</sup> a pattern emerges suggesting that incentives for behavioral risk factors mainly depend on social position and only distantly on workplace. Conversely, work environment appears to be a closer determinant of the incidence of clinical risk factors than social position that would have a more distant role. To interpret the respective roles of social position and work environment in determining the incidence of behavioral and clinical risk factors, it is necessary to recall that these factors form an extensive network of reciprocal relationships where each factor predicts, and/or is predicted by, several other factors.<sup>40</sup> Four categories of factors can be distinguished: nonmodifiable factors (gender, age, parental history of early coronary heart disease) that only predict and are not predicted by other factors; behavioral factors (nonmoderate alcohol consumption, smoking, leisure-time physical inactivity) that form very few associations with each other, predict several clinical factors, and are predicted by a small number of nonmodifiable or clinical factors; upstream clinical factors (obesity, sleep complaints, depression) that form a few associations with each other, predict many downstream clinical factors, and are predicted by many nonmodifiable or behavioral factors; and downstream clinical factors (hypertension, dyslipidemia, diabetes) that form many associations with each other, predict very few factors but are predicted by a large number of nonmodifiable behavioral factors or upstream clinical factors.<sup>40</sup> The present results suggest that the influence of work environment on the incidence of clinical factors, especially upstream clinical factors as previously reported,<sup>37</sup> largely exceeds the influence that social position exerts on the incidence of these factors through its effect on the incidence of behavioral factors. In other words, the influence of social position on the incidence of upstream clinical factors would be mainly mediated by work environment and not by behavioral factors.<sup>29</sup> The same interpretation can be applied to the incidence of downstream clinical factors that would be more influenced by work environment, directly and through its effect on the incidence of upstream clinical factors, than by social position and its effect on the incidence of behavioral factors. Inversely, work environment is expected to

have a weak influence on the incidence of behavioral factors as it does not determine per se social position and because upstream and downstream clinical factors have a limited influence on the incidence of behavioral factors.

This study has some strengths and several limitations. One strength is the use of global measures of work environment and social position whose rationale has been discussed elsewhere.<sup>29,37</sup> The main reason is that specific socioeconomic indicator or occupational exposure captures only partial aspects of social position or work environment whereas their combinations allow the assessment of this position or environment as a whole, the reality that people face. A second strength is the use of Bayesian structural equation modeling to perform discrete-time survival analysis with reciprocal mediating effects,<sup>42–44</sup> which is an appropriate method among a few others.<sup>53</sup> It nevertheless requires important assumptions in order to make causal claims, for example, the absence of confounding for each association that forms part of the mediation structure.<sup>43</sup> Among the other limitations of the study, one is the external validity of the findings that were obtained in a cohort of workers who were not representative of the French working population as discussed in the Methods. A second is that socioeconomic indicators, occupational exposures, and cardiovascular risk factors were self-reported and may therefore be relatively imprecise. A third is the lack of information concerning the potential evolution of social position and work environment during follow-up; this likely weakens the associations with the incidence of cardiovascular risk factors given that both social position and work environment probably improve with time as workers move up the job ladder, thus reducing the probability of occurrence of risk factors.

## CONCLUSIONS

In conclusion, our results show a reciprocal mediation between social position and work environment on the incidence of common cardiovascular risk factors. The proportions of the mediating effects, which are very variable depending on the factor, suggest that the incidence of behavioral risk factors is strongly dependent on social position whereas that of clinical risk factors is closely related to work environment both in men and women. In addition to providing insights into the mechanisms that underlie the associations of social position and work environment with the incidence of cardiovascular risk factors, these findings suggest different ways of improving preventive strategies, based on education and general practice for behavioral factors and involving work organization and occupational medicine for clinical factors.

## ARTICLE INFORMATION

Received February 21, 2021; accepted November 3, 2021.

### Affiliations

Département de Psychiatrie, Institut de Psychiatrie et Neurosciences de Paris, Faculté de Médecine, Hôpital Cochin-Celton, INSERM, UMR\_S1266, AP-HP, Université de Paris, Issy-les-Moulineaux, France (N.H., M.S.R., F.L.); UMS\_011, INSERM, Université Paris-Saclay, Villejuif, France (C.R., S.B., M.G., M.Z.); and UMR\_1142, INSERM, Sorbonne Université, Université Paris 13, Paris, France (J.M., P.M.).

### Acknowledgments

The authors express their thanks to EDF-GDF, especially to the Service Général de Médecine de Contrôle and to the Caisse centrale d'action sociale du personnel des industries électrique et gazière. We also wish to acknowledge the Population-based Cohort Unit, UMS 011 INSERM Versailles St-Quentin University, which is responsible for the GAZEL database management.

### Sources of Funding

The GAZEL Cohort Study was funded by EDF-GDF and INSERM and received grants from the Cohortes Santé TGIR Program, Agence Nationale de la Recherche (ANR-08-BLAN-0028), Agence Française de Sécurité Sanitaire de l'Environnement et du Travail (AFSSET-EST-2008/1/35), and the Caisse d'assurance Maladie des Industries Électrique et Gazière (CAMIEG).

### Disclosures

None.

### Supplementary Material

Tables S1–S10

Figures S1–S3

## REFERENCES

- Marmot M. Achieving health equity: from root causes to fair outcomes. *Lancet*. 2007;370:1153–1163. doi: 10.1016/S0140-6736(07)61385-3
- Lynch J, Davey Smith G, Harper S, Bainbridge K. Explaining the social gradient in coronary heart disease: comparing relative and absolute risk approaches. *J Epidemiol Community Health*. 2006;60:436–441. doi: 10.1136/jech.2005.041350
- Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88:1973–1998. doi: 10.1161/01.CIR.88.4.1973
- Beenackers MA, Kamphuis CB, Giskes K, Brug J, Kunst AE, Burdorf A, van Lenthe FJ. Socioeconomic inequalities in occupational, leisure-time, and transport related physical activity among European adults: a systematic review. *Int J Behav Nutr Phys Act*. 2012;9:116. doi: 10.1186/1479-5868-9-116
- Bloomfield K, Grittner U, Kramer S, Gmel G. Social inequalities in alcohol consumption and alcohol-related problems in the study countries of the EU concerted action 'Gender, Culture and Alcohol Problems: a Multi-national Study'. *Alcohol Alcohol Suppl*. 2006;41:26–36. doi: 10.1093/alcalc/agl073
- Kanjilal S, Gregg EW, Cheng YJ, Zhang P, Nelson DE, Mensah G, Beckles GL. Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971–2002. *Arch Intern Med*. 2006;166:2348–2355. doi: 10.1001/archinte.166.21.2348
- McLaren L. Socioeconomic status and obesity. *Epidemiol Rev*. 2007;29:29–48. doi: 10.1093/epirev/mxm001
- Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health*. 1992;82:816–820. doi: 10.2105/AJPH.82.6.816
- Chang-Quan H, Zheng-Rong W, Yong-Hong L, Yi-Zhou X, Qing-Xiu L. Education and risk for late life depression: a meta-analysis of published literature. *Int J Psychiatry Med*. 2010;40:109–124. doi: 10.2190/PM.40.1.i
- Jehan S, Myers AK, Zizi F, Pandi-Perumal SR, Jean-Louis G, Singh N, Ray J, McFarlane SI. Sleep health disparity: the putative role of race, ethnicity and socioeconomic status. *Sleep Med Disord*. 2018;2:127–133.

11. Grandner MA, Alfonso-Miller P, Fernandez-Mendoza J, Shetty S, Shenoy S, Combs D. Sleep: important considerations for the prevention of cardiovascular disease. *Curr Opin Cardiol*. 2016;31:551–565. doi: 10.1097/HCO.0000000000000324
12. Van der Kooy K, van Hout H, Marwijk H, Marten H, Stehouwer C, Beekman A. Depression and the risk for cardiovascular diseases: systematic review and meta analysis. *Int J Geriatr Psychiatry*. 2007;22:613–626. doi: 10.1002/gps.1723
13. Havranek EP, Mujahid MS, Barr DA, Blair IV, Cohen MS, Cruz-Flores S, Davey-Smith G, Dennison-Himmelfarb CR, Lauer MS, Lockwood DW, et al.; American Heart Association Council on Quality of Care and Outcomes Research COEAP, Council on Cardiovascular and Stroke Nursing, Council on Lifestyle and Cardiometabolic Health, and Stroke Council. Social determinants of risk and outcomes for cardiovascular disease: a scientific statement from the American Heart Association. *Circulation*. 2015;132:873–898. doi: 10.1161/CIR.0000000000000228
14. Burgard SA, Lin KY. Bad jobs, bad health? How work and working conditions contribute to health disparities. *Am Behav Sci*. 2013;57:1105–1127. doi: 10.1177/0002764213487347
15. Sara JD, Prasad M, Eleid MF, Zhang M, Widmer RJ, Lerman A. Association between work-related stress and coronary heart disease: a review of prospective studies through the job strain, effort-reward balance, and organizational justice models. *J Am Heart Assoc*. 2018;7:e008073. doi: 10.1161/JAHA.117.008073
16. Heikkilä K, Nyberg ST, Fransson EI, Alfredsson L, De Bacquer D, Bjorner JB, Bonenfant S, Borritz M, Burr H, Clays E, et al.; IPD-Work C. Job strain and alcohol intake: a collaborative meta-analysis of individual-participant data from 140,000 men and women. *PLoS One*. 2012;7:e40101. doi: 10.1371/journal.pone.0040101
17. Heikkilä K, Nyberg ST, Fransson EI, Alfredsson L, De Bacquer D, Bjorner JB, Bonenfant S, Borritz M, Burr H, Clays E, et al.; IPD-Work C. Job strain and tobacco smoking: an individual-participant data meta-analysis of 166,130 adults in 15 European studies. *PLoS One*. 2012;7:e35463. doi: 10.1371/journal.pone.0035463
18. Fransson EI, Heikkilä K, Nyberg ST, Zins M, Westerlund H, Westerholm P, Väänänen A, Virtanen M, Vahtera J, Theorell T, et al. Job strain as a risk factor for leisure-time physical inactivity: an individual-participant meta-analysis of up to 170,000 men and women: the IPD-Work Consortium. *Am J Epidemiol*. 2012;176:1078–1089. doi: 10.1093/aje/kws336
19. Nyberg ST, Heikkilä K, Fransson EI, Alfredsson L, De Bacquer D, Bjorner JB, Bonenfant S, Borritz M, Burr H, Casini A, et al.; IPD-Work C. Job strain in relation to body mass index: pooled analysis of 160,000 adults from 13 cohort studies. *J Intern Med*. 2012;272:65–73. doi: 10.1111/j.1365-2796.2011.02482.x
20. Landsbergis PA, Dobson M, Koutsouras G, Schnall P. Job strain and ambulatory blood pressure: a meta-analysis and systematic review. *Am J Public Health*. 2013;103:61–71. doi: 10.2105/AJPH.2012.301153
21. Nyberg ST, Fransson EI, Heikkilä K, Ahola K, Alfredsson L, Bjorner JB, Borritz M, Burr H, Dragano N, Goldberg M, et al.; IPD-Work C. Job strain as a risk factor for type 2 diabetes: a pooled analysis of 124,808 men and women. *Diabetes Care*. 2014;37:2268–2275. doi: 10.2337/dc13-2936
22. Linton SJ, Kecklund G, Franklin KA, Leissner LC, Sivertsen B, Lindberg E, Svensson AC, Hansson SO, Sundin Ö, Hetta J, et al. The effect of the work environment on future sleep disturbances: a systematic review. *Sleep Med Rev*. 2015;23:10–19. doi: 10.1016/j.smrv.2014.10.010
23. Theorell T, Hammarström A, Aronsson G, Träskman Bendz L, Grape T, Hogstedt C, Marteinsdottir I, Skoog I, Hall C. A systematic review including meta-analysis of work environment and depressive symptoms. *BMC Public Health*. 2015;15:738. doi: 10.1186/s12889-015-1954-4
24. Benach J, Solar O, Santana V, Castedo A, Chung H, Muntaner C, EMCONET N. A micro-level model of employment relations and health inequalities. *Int J Health Serv*. 2010;40:223–227. doi: 10.2190/HS.40.2.d
25. Moncada S. Working conditions and social inequalities in health. *J Epidemiol Community Health*. 1999;53:390–391. doi: 10.1136/jech.53.7.390
26. Marmot M, Theorell T. Social class and cardiovascular disease: the contribution of work. *Int J Health Serv*. 1988;18:659–674. doi: 10.2190/KTC1-N5LK-J1PM-9GRQ
27. Andersen I, Burr H, Kristensen TS, Gamborg M, Osler M, Prescott E, Diderichsen F. Do factors in the psychosocial work environment mediate the effect of socioeconomic position on the risk of myocardial infarction? Study from the Copenhagen Centre for Prospective Population Studies. *Occup Environ Med*. 2004;61:886–892. doi: 10.1136/oem.2004.013417
28. Marmot M, Bosma H, Hemingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet*. 1997;350:235–239. doi: 10.1016/S0140-6736(97)04244-X
29. Meneton P, Hoertel N, Wiernik E, Lemogne C, Ribet C, Bonenfant S, Ménard J, Goldberg M, Zins M. Work environment mediates a large part of social inequalities in the incidence of several common cardiovascular risk factors: findings from the Gazel cohort. *Soc Sci Med*. 2018;216:59–66. doi: 10.1016/j.socscimed.2018.09.042
30. Huisman M, Van Lenthe F, Avendano M, Mackenbach J. The contribution of job characteristics to socioeconomic inequalities in incidence of myocardial infarction. *Soc Sci Med*. 2008;66:2240–2252. doi: 10.1016/j.socscimed.2008.01.049
31. O'Reilly D, Rosato M. Worked to death? A census-based longitudinal study of the relationship between the numbers of hours spent working and mortality risk. *Int J Epidemiol*. 2013;42:1820–1830. doi: 10.1093/ije/dyt121
32. Ferrario MM, Veronesi G, Bertù L, Grassi G, Cesana G. Job strain and the incidence of coronary heart diseases: does the association differ among occupational classes? A contribution from a pooled analysis of Northern Italian cohorts. *BMJ Open*. 2017;7:e014119. doi: 10.1136/bmjopen-2016-014119
33. Brunner EJ, Kivimäki M, Siegrist J, Theorell T, Luukkonen R, Riihimäki H, Vahtera J, Kirjonen J, Leino-Arjas P. Is the effect of work stress on cardiovascular mortality confounded by socioeconomic factors in the Valmet study. *J Epidemiol Community Health*. 2004;58:1019–1020. doi: 10.1136/jech.2003.016881
34. Goldberg M, Leclerc A, Bonenfant S, Chastang JF, Schmaus A, Kaniewski N, Zins M. Cohort profile: the GAZEL cohort study. *Int J Epidemiol*. 2007;36:32–39. doi: 10.1093/ije/dyl247
35. Zins M, Leclerc A, Goldberg M. The French GAZEL cohort study: 20 years of epidemiological research. *Adv Life Course Res*. 2009;14:135–146.
36. Lanoe JL, Makdessi-Raynaud Y. L'état de santé en France en 2003: santé perçue, morbidité déclarée et recours aux soins à travers l'enquête décennale santé. *Etudes et Résultats*. 2005;436:1–12.
37. Meneton P, Lemogne C, Herquelot E, Bonenfant S, Czernichow S, Ménard J, Goldberg M, Zins M. Primary cardiovascular disease risk factors predicted by poor working conditions in the GAZEL cohort. *Am J Epidemiol*. 2017;186:815–823. doi: 10.1093/aje/kwx152
38. Karasek R, Baker D, Marxer F, Ahlborn A, Theorell T. Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. *Am J Public Health*. 1981;71:694–705. doi: 10.2105/AJPH.71.7.694
39. Siegrist J, Starke D, Chandola T, Godin I, Marmot M, Niedhammer I, Peter R. The measurement of effort-reward imbalance at work: European comparisons. *Soc Sci Med*. 2004;58:1483–1499. doi: 10.1016/S0277-9536(03)00351-4
40. Meneton P, Lemogne C, Herquelot E, Bonenfant S, Larson MG, Vasani RS, Ménard J, Goldberg M, Zins M. A global view of the relationships between the main behavioral and clinical cardiovascular risk factors in the GAZEL prospective cohort. *PLoS One*. 2016;11:e0162386. doi: 10.1371/journal.pone.0162386
41. Le Port A, Gueguen A, Kesse-Guyot E, Melchior M, Lemogne C, Nabi H, Goldberg M, Zins M, Czernichow S. Association between dietary patterns and depressive symptoms over time: a 10-year follow-up study of the GAZEL cohort. *PLoS One*. 2012;7:e51593. doi: 10.1371/journal.pone.0051593
42. Gelman A, Carlin JB, Stern HS, Rubin DB. *Bayesian data analysis*. Chapman and Hall/CRC; 2004.
43. Pratschke J, Haase T, Comber H, Sharp L, de Camargo CM, Johnson H. Mechanisms and mediation in survival analysis: towards an integrated analytical framework. *BMC Med Res Methodol*. 2016;16:27. doi: 10.1186/s12874-016-0130-6
44. Raykov T, Zajacova A, Gorelick PB, Marcoulides GA. Using latent variable modeling for discrete time survival analysis: examining the links of depression to mortality. *Struct Equ Modeling*. 2018;25:287–293. doi: 10.1080/10705511.2017.1364969
45. Muthén BO Bayesian analysis in Mplus: a brief introduction. 2010. Available at: <https://www.statmodel.com/download/IntroBayesVersi%203.pdf>. Accessed November 16, 2021.
46. Blanco C, Hoertel N, Wall MM, Franco S, Peyre H, Neria Y, Helpman L, Limosin F. Toward understanding sex differences in the prevalence of posttraumatic stress disorder: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *J Clin Psychiatry*. 2018;79:16m11364. doi: 10.4088/JCP.16m11364

- 
47. Pearl J. Interpretation and identification of causal mediation. *Psychol Methods*. 2014;19:459–481. doi: 10.1037/a0036434
  48. Lee SY, Song XY, Cai JH. A Bayesian approach for nonlinear structural equation models with dichotomous variables using logit and probit links. *Struct Equ Modeling*. 2010;17:280–302. doi: 10.1080/10705511003659425
  49. MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: distribution of the product and resampling methods. *Multivariate Behav Res*. 2004;39:99. doi: 10.1207/s15327906mbr3901\_4
  50. Hoertel N, Blanco C, Olfson M, Oquendo MA, Wall MM, Franco S, Leleu H, Lemogne C, Falissard B, Limosin F. A comprehensive model of predictors of suicide attempt in depressed individuals and effect of treatment-seeking behavior: results from a national 3-year prospective study. *J Clin Psychiatry*. 2018;79:17m11704. doi: 10.4088/JCP.17m11704
  51. MacKinnon DP, Krull JL, Lockwood CM. Equivalence of the mediation, confounding and suppression effect. *Prev Sci*. 2000;1:173–181.
  52. Gelman A, Rubin DB. Inference from iterative simulation using multiple sequences. *Stat Sci*. 1992;7:457–511. doi: 10.1214/ss/1177011136
  53. VanderWeele TJ. A unification of mediation and interaction: a 4-way decomposition. *Epidemiology*. 2014;25:749–761. doi: 10.1097/EDE.0000000000000121

**Table S1:** Behavioral and socioeconomic characteristics of cohort participants at baseline compared to randomly selected individuals from the French population in the same age range.

	Men			Women		
	GAZEL cohort	Randomly selected	P	GAZEL cohort	Randomly selected	P
<b>N</b>	15011	2683	-	5614	4333	-
<b>Age, y (mean <math>\pm</math> SD)</b>	44.5 $\pm$ 2.9	44.9 $\pm$ 3.1	-	41.7 $\pm$ 4.2	42.4 $\pm$ 4.6	-
<b>Smoking, n (%)</b>	4632 (31.0)	848 (35.7)	<0.0001	1200 (21.7)	1218 (31.4)	<0.0001
<b>Physical inactivity, n (%)</b>	3895 (30.8)	1432 (53.4)	<0.0001	1811 (40.5)	2582 (59.6)	<0.0001
<b>Obesity, n (%)</b>	707 (5.5)	306 (11.4)	<0.0001	162 (3.5)	426 (9.8)	<0.0001
<b>Education, n (%)</b>						
Primary school	967 (6.6)	548 (20.4)	<0.0001	409 (7.5)	913 (21.1)	<0.0001
Secondary school	10644 (72.4)	1509 (56.3)		4343 (79.6)	2267 (52.3)	
University	3094 (21.0)	626 (23.3)		702 (12.9)	1153 (26.6)	
<b>Occupation, n (%)</b>						
Blue collar/clerk	2151 (14.3)	1243 (52.2)	<0.0001	1529 (27.3)	2502 (62.7)	<0.0001
Intermediate	8384 (55.9)	656 (27.5)		3635 (64.8)	1019 (25.5)	
Management	4459 (29.8)	484 (20.3)		442 (7.9)	472 (11.8)	
<b>Income, n (%)</b>						
Low	4234 (41.1)	1370 (51.0)	<0.0001	1155 (34.5)	2420 (55.9)	<0.0001
Middle	3777 (36.7)	678 (25.3)		1404 (41.9)	1007 (23.2)	
High	2288 (22.2)	635 (23.7)		788 (23.6)	906 (20.9)	

The percentages in parenthesis refer to the number of men or women participating in the cohort or randomly selected from the French population. Univariate comparisons were performed with chi-square test or Fisher's exact test when necessary.

**Table S2:** Distribution of socioeconomic indicators at baseline.

		<b>N</b>	<b>%</b>
<b>Education</b>	<b>University</b>	3,796	18.8
	<b>Secondary school</b>	14,987	74.4
	<b>Primary school</b>	1,376	6.8
<b>Wealth</b>	<b>Rich</b>	2,669	21.0
	<b>Middle class</b>	7,924	62.4
	<b>Poor</b>	2,110	16.6
<b>Income</b>	<b>High</b>	3,076	22.5
	<b>Middle</b>	5,181	38.0
	<b>Low</b>	5,389	39.5
<b>Occupational grade</b>	<b>Management</b>	4,901	23.8
	<b>Intermediate</b>	12,019	58.3
	<b>Blue collar/clerk</b>	3,680	17.9



**Table S3:** Occupational exposures characterizing work environment at baseline.

		N	%
<b>Commuting time</b>	<b>Short</b>	5,930	29.1
	<b>Average</b>	7,559	37.2
	<b>Long</b>	6,858	33.7
<b>Working with the public</b>	<b>No</b>	10,960	60.3
	<b>Yes</b>	7,216	39.7
<b>Outdoor work</b>	<b>No</b>	9,520	49.6
	<b>Yes</b>	9,675	50.4
<b>Night shift work</b>	<b>No</b>	13,022	68.5
	<b>Yes</b>	5,995	31.5
<b>Regular work hours</b>	<b>No</b>	3,649	20.1
	<b>Yes</b>	14,478	79.9
<b>On-call work</b>	<b>No</b>	14,968	75.5
	<b>Yes</b>	4,869	24.5
<b>Standing work posture</b>	<b>No</b>	5,088	45.1
	<b>Yes</b>	6,184	54.9
<b>Hard work posture</b>	<b>No</b>	5,385	57.4
	<b>Yes</b>	3,999	42.6
<b>Handling heavy loads</b>	<b>No</b>	5,580	65.4
	<b>Yes</b>	2,957	34.6
<b>Exposure to vibrations</b>	<b>No</b>	5,635	72.1
	<b>Yes</b>	2,184	27.9
<b>Working with a screen</b>	<b>No</b>	2,527	14.0
	<b>Yes</b>	15,505	86.0
<b>Working in the cold</b>	<b>No</b>	5,594	73.3
	<b>Yes</b>	2,036	26.7
<b>Working in the heat</b>	<b>No</b>	5,609	75.1
	<b>Yes</b>	1,856	24.9
<b>Exposure to noise</b>	<b>No</b>	5,498	68.8
	<b>Yes</b>	2,492	31.2
<b>Work involving specific risks</b>	<b>No</b>	6,607	35.8
	<b>Yes</b>	11,833	64.2
<b>Unhealthy work</b>	<b>No</b>	18,932	95.2
	<b>Yes</b>	950	4.8
<b>Physically demanding work</b>	<b>No</b>	7,336	36.7
	<b>Average</b>	7,423	37.2
	<b>Yes</b>	5,204	26.1
<b>Nerve-racking work</b>	<b>No</b>	7,053	34.7
	<b>Average</b>	8,442	41.5
	<b>Yes</b>	4,832	23.8
<b>Satisfactory work</b>	<b>No</b>	3,469	20.7
	<b>Average</b>	7,127	42.5
	<b>Yes</b>	6,185	36.8
<b>Decision latitude</b>	<b>Low</b>	4,006	28.6
	<b>Average</b>	5,240	37.5
	<b>High</b>	4,743	33.9
<b>Psychological demand</b>	<b>Low</b>	4,192	29.9
	<b>Average</b>	5,266	37.6
	<b>High</b>	4,542	32.5
<b>Social support at work</b>	<b>Low</b>	4,266	30.7
	<b>Average</b>	5,347	38.5
	<b>Strong</b>	4,291	30.8
<b>Extrinsic effort</b>	<b>Low</b>	2,633	24.7
	<b>Average</b>	4,473	41.9
	<b>High</b>	3,567	33.4
<b>Reward</b>	<b>Low</b>	3,329	31.2
	<b>Average</b>	3,738	35.0
	<b>High</b>	3,606	33.8
<b>Over-commitment</b>	<b>No</b>	7,003	65.6
	<b>Yes</b>	3,670	34.4

Some exposures were binary while others were graded on a 3-level scale or divided into tertiles.

**Table S4:** Interdependence of social position and work environment at baseline.

		Work environment			p
		Good	Average	Bad	
Social position	High	2,127	1,530	1,009	<0.0001
		31.9	21.9	14.5	
	Middle	3,393	3,902	3,919	
		50.8	55.8	56.4	
	Low	1,157	1,562	2,018	
		17.3	22.3	29.1	

The association between work environment and social position was assessed by Chi-square test. The percentages given with counts refer to the number of workers in each type of work environment.

**Table S5:** Discrete-time survival analysis with reciprocal mediating effects by social position and work environment on the incidence of depression using Bayesian structural equation modeling and different cut-off values on the CES-D scale.

		Assuming that work environment mediates the effect of social position on the incidence			Assuming that social position mediates the effect of work environment on the incidence			Mediation ratio (mediation by work environment/mediation by social position)
		Estimate	SD	95% CI	Estimate	SD	95% CI	
<b>Depression (cut-offs 17 and 23)</b>	<b>Total effect</b>	0.145***	0.011	0.125-0.167	0.316***	0.009	0.298-0.333	41.5
	<b>Indirect effect</b>	0.126***	0.006	0.114-0.139	0.007**	0.002	0.002-0.012	
	<b>Mediation (%)</b>	0.866***	0.040	0.791-0.951	0.021**	0.007	0.007-0.035	
<b>Depression (cut-off 19)</b>	<b>Total effect</b>	0.148***	0.014	0.112-0.170	0.299***	0.011	0.273-0.313	22.4
	<b>Indirect effect</b>	0.116***	0.007	0.101-0.130	0.010**	0.003	0.002-0.016	
	<b>Mediation (%)</b>	0.782***	0.059	0.708-0.948	0.035**	0.011	0.007-0.051	

Depression was defined by using either different cut-offs in men and women (respectively 17 and 23 on the CES-D scale) or the same cut-off in both sexes (19 on the CES-D scale). Each model included sex, age and parental history of early coronary heart disease for adjustment purpose. Estimates are reported with standard deviations (SD) and 95% credibility intervals (95% CI). \*\*\* <0.001, \*\* <0.01.

**Table S6:** Discrete-time survival analysis with reciprocal mediating effects by social position and work environment on the incidence of behavioral or clinical cardiovascular risk factors using Bayesian structural equation modeling stratified by sex.

			Assuming that work environment mediates the effect of social position on the incidence			Assuming that social position mediates the effect of work environment on the incidence			Mediation ratio (mediation by work environment/mediation by social position)	
			Estimate	SD	95% CI	Estimate	SD	95% CI		
Men	Behavioral risk factors	Total effect	0.392***	0.019	0.358-0.431	0.214***	0.020	0.174-0.253	0.15	
		Indirect effect	0.036***	0.007	0.022-0.052	0.133***	0.014	0.107-0.162		
		Mediation (%)	0.093***	0.018	0.057-0.132	0.625***	0.053	0.522-0.740		
	Clinical risk factors	Total effect	0.501***	0.019	0.464-0.541	0.740***	0.011	0.714-0.760		6.56
		Indirect effect	0.318***	0.016	0.288-0.351	0.072***	0.006	0.060-0.084		
		Mediation (%)	0.636***	0.022	0.593-0.677	0.097***	0.008	0.082-0.113		
Women	Behavioral risk factors	Total effect	0.477***	0.031	0.433-0.549	0.138***	0.038	0.061-0.211	0.07	
		Indirect effect	0.010*	0.008	0.004-0.028	0.043*	0.031	0.017-0.104		
		Mediation (%)	0.021*	0.017	0.008-0.058	0.312*	0.030	0.204-0.623		
	Clinical risk factors	Total effect	0.430***	0.038	0.360-0.509	0.896***	0.023	0.852-0.940		7.21
		Indirect effect	0.136***	0.034	0.073-0.205	0.040***	0.010	0.021-0.060		
		Mediation (%)	0.317***	0.058	0.195-0.422	0.044***	0.011	0.023-0.066		

The models included age and parental history of early coronary heart disease for adjustment purpose and tested either non-moderate alcohol consumption, smoking and leisure-time physical inactivity together (behavioral risk factors), or obesity, hypertension, dyslipidemia, diabetes, sleep complaints and depression together (clinical risk factors). Estimates are reported with standard deviations (SD) and 95% credibility intervals (95% CI). \*\*\* <0.001, \* <0.05.

**Table S7:** Discrete-time survival analysis with reciprocal mediating effects by socioeconomic indicator and work environment on the incidence of behavioral or clinical cardiovascular risk factors using Bayesian structural equation modeling.

			Assuming that work environment mediates the effect of socioeconomic indicator on the incidence			Assuming that socioeconomic indicator mediates the effect of work environment on the incidence			Mediation ratio (mediation by work environment/mediation by socioeconomic indicator)	
			Estimate	SD	95% CI	Estimate	SD	95% CI		
Education	Behavioral risk factors	Total effect	0.347***	0.038	0.270-0.417	0.213***	0.032	0.150-0.273	0.43	
		Indirect effect	0.056***	0.013	0.032-0.081	0.079***	0.013	0.056-0.107		
		Mediation (%)	0.160***	0.028	0.104-0.214	0.376***	0.050	0.287-0.483		
	Clinical risk factors	Total effect	0.429***	0.031	0.369-0.492	0.704***	0.025	0.656-0.750		8.94
		Indirect effect	0.250***	0.019	0.213-0.288	0.046***	0.006	0.034-0.059		
		Mediation (%)	0.581***	0.031	0.525-0.644	0.065***	0.007	0.051-0.080		
Wealth	Behavioral risk factors	Total effect	0.284***	0.032	0.224-0.344	0.134***	0.028	0.084-0.194	0.22	
		Indirect effect	0.018***	0.006	0.007-0.032	0.038***	0.010	0.020-0.060		
		Mediation (%)	0.063***	0.020	0.028-0.104	0.285***	0.072	0.159-0.448		
	Clinical risk factors	Total effect	0.536***	0.032	0.471-0.598	0.915***	0.022	0.871-0.957		5.53
		Indirect effect	0.201***	0.021	0.161-0.243	0.062***	0.007	0.048-0.077		
		Mediation (%)	0.376***	0.027	0.321-0.427	0.068***	0.007	0.054-0.082		
Income	Behavioral risk factors	Total effect	0.231***	0.028	0.192-0.304	0.108***	0.032	0.058-0.184	0.09	
		Indirect effect	0.013*	0.009	0.002-0.033	0.070***	0.010	0.052-0.093		
		Mediation (%)	0.056*	0.033	0.009-0.121	0.638***	0.166	0.425-1.091		
	Clinical risk factors	Total effect	0.339***	0.025	0.287-0.387	0.768***	0.021	0.720-0.803		13.3
		Indirect effect	0.212***	0.014	0.185-0.240	0.036***	0.005	0.025-0.047		
		Mediation (%)	0.623***	0.033	0.565-0.696	0.047***	0.006	0.033-0.059		
Occupational grade	Behavioral risk factors	Total effect	0.505***	0.025	0.454-0.550	0.334***	0.028	0.278-0.385	0.38	
		Indirect effect	0.083***	0.012	0.059-0.107	0.143***	0.015	0.115-0.171		
		Mediation (%)	0.164***	0.019	0.127-0.202	0.428***	0.035	0.364-0.502		
	Clinical risk factors	Total effect	0.614***	0.031	0.547-0.670	0.925***	0.023	0.874-0.960		8.14
		Indirect effect	0.414***	0.020	0.375-0.456	0.077***	0.008	0.060-0.093		
		Mediation (%)	0.676***	0.022	0.638-0.721	0.083***	0.008	0.068-0.098		

The models included sex, age and parental history of early coronary heart disease for adjustment purpose and tested either non-moderate alcohol consumption, smoking and leisure-time physical inactivity together (behavioral risk factors), or obesity, hypertension, dyslipidemia, diabetes, sleep complaints and depression together (clinical risk factors). Estimates are reported with standard deviations (SD) and 95% credibility intervals (95% CI). \*\*\* <0.001, \* <0.05.

**Table S8:** Discrete-time survival analysis with reciprocal mediating effects by social position and work environment on the incidence of behavioral or clinical cardiovascular risk factors using Bayesian structural equation modeling and different follow-up durations.

			Assuming that work environment mediates the effect of social position on the incidence			Assuming that social position mediates the effect of work environment on the incidence			Mediation ratio (mediation by work environment/mediation by social position)	
			Estimate	SD	95% CI	Estimate	SD	95% CI		
25-year follow-up	Behavioral risk factors	Total effect	0.564***	0.026	0.516-0.618	0.335***	0.029	0.281-0.393	0.26	
		Indirect effect	0.067***	0.011	0.049-0.091	0.154***	0.016	0.123-0.188		
		Mediation (%)	0.119***	0.015	0.092-0.152	0.461***	0.035	0.392-0.530		
	Clinical risk factors	Total effect	0.491***	0.031	0.435-0.551	0.789***	0.025	0.745-0.837		6.67
		Indirect effect	0.285***	0.016	0.255-0.317	0.069***	0.008	0.053-0.084		
		Mediation (%)	0.580***	0.024	0.537-0.634	0.087***	0.008	0.070-0.102		
12-year follow-up	Behavioral risk factors	Total effect	0.568***	0.051	0.479-0.671	0.335***	0.048	0.246-0.432	0.25	
		Indirect effect	0.067***	0.015	0.039-0.097	0.156***	0.020	0.120-0.198		
		Mediation (%)	0.118***	0.019	0.078-0.153	0.465***	0.049	0.384-0.575		
	Clinical risk factors	Total effect	0.570***	0.036	0.493-0.633	0.846***	0.030	0.784-0.903		5.03
		Indirect effect	0.300***	0.018	0.267-0.336	0.089***	0.010	0.067-0.108		
		Mediation (%)	0.528***	0.027	0.479-0.589	0.105***	0.010	0.084-0.125		

Follow-up duration was 25 or 12 years. The models included sex, age and parental history of early coronary heart disease for adjustment purpose and tested either non-moderate alcohol consumption, smoking and leisure-time physical inactivity together (behavioral risk factors) or obesity, hypertension, dyslipidemia, diabetes, sleep complaints and depression together (clinical risk factors). Estimates are reported with standard deviations (SD) and 95% credibility intervals (95% CI). \*\*\* <0.001.

**Table S9:** Sensitivity to prior distributions in the model evaluating reciprocal mediating effects by social position and work environment on the incidence of depression using Bayesian structural equation modeling.

Prior distributions of the association between social position and the incidence of depression (estimate, variance)	Prior distributions of the association between work environment and the incidence of depression (estimate, variance)	Mediation ratio (mediation by work environment/mediation by social position)		
		Ratio	SD	95% CI
Unfixed	Unfixed	41.542	17.527	28.533-93.201
(0, 1)	(0, 1)	42.987	17.256	30.474-93.348
(0, 3)	(0, 3)	42.990	17.262	30.475-93.368
(0, 5)	(0, 5)	42.990	17.263	30.475-93.372
(0, 10)	(0, 10)	42.990	17.263	30.475-93.375
(3, 10)	(3, 10)	43.010	17.301	30.483-93.510
(1, 1)	(1, 1)	43.053	17.383	30.498-93.799
(1, 10)	(1, 10)	42.997	17.276	30.478-93.420
(3, 3)	(3, 3)	43.056	17.388	30.499-93.819

The model included sex, age and parental history of early coronary heart disease for adjustment purpose and ran after having standardized the covariates in the analysis such that any abnormal covariate scale was eliminated. Unfixed and fixed prior distributions were considered normal. Mediation ratios are reported with standard deviations (SD) and 95% credibility intervals (95% CI).

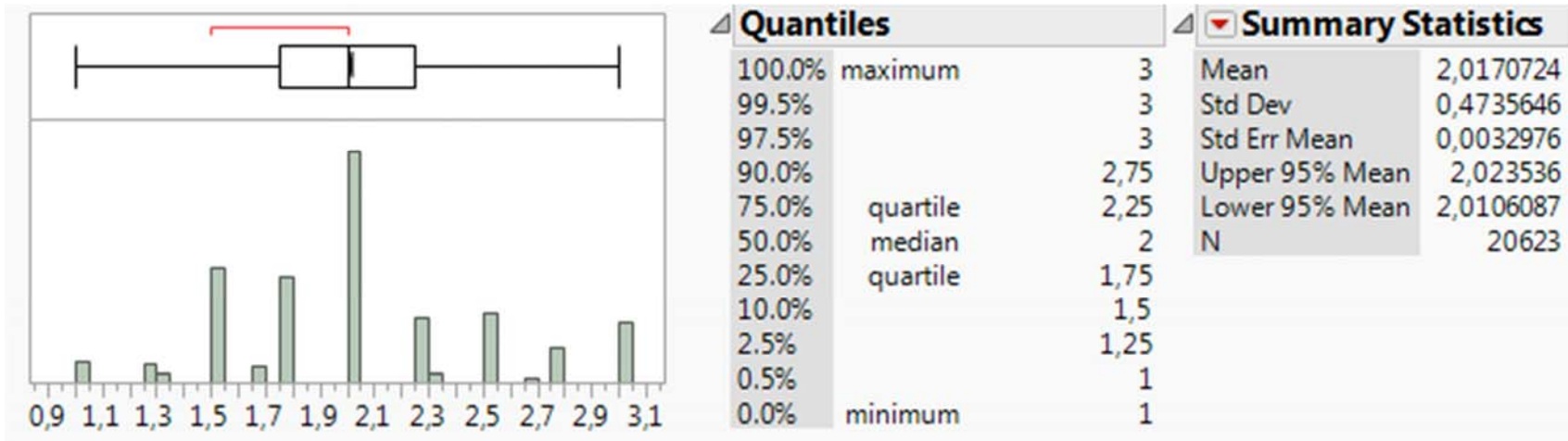
**Table S10:** Proportional scale reduction convergence in the model evaluating reciprocal mediating effects by social position and work environment on the incidence of depression using Bayesian structural equation modeling.

<b>Bayes iterations</b>	<b>Proportional scale reduction</b>
100	2.863
200	1.665
300	1.469
400	1.230
500	1.122
600	1.190
700	1.218
800	1.274
900	1.127
1000	1.102
1100	1.051

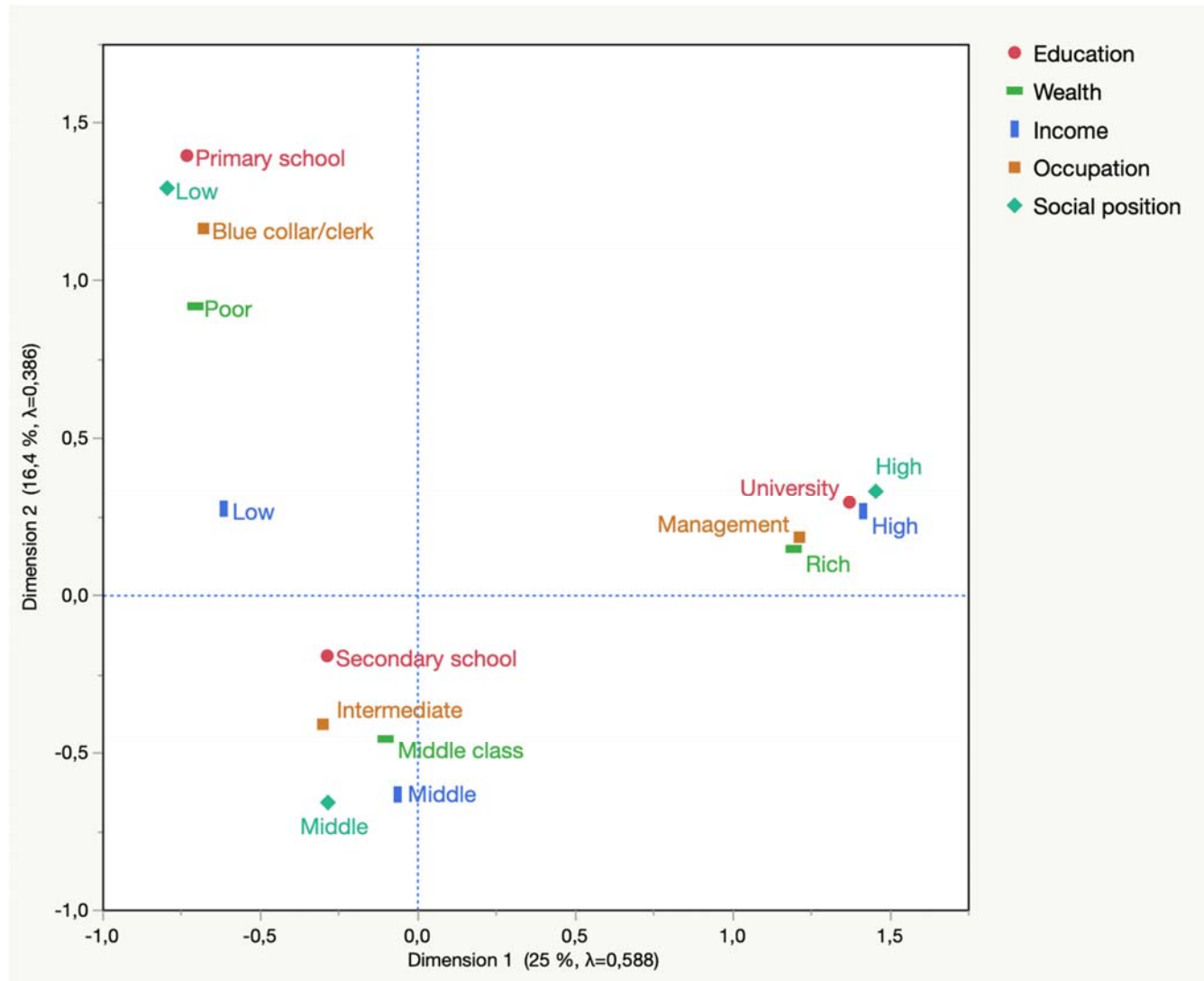
Proportional scale reduction values lower than 1.10 indicate adequate convergence.



**Figure S1:** Distribution and summary statistics of the global measure of social position. It is calculated by giving for each socioeconomic indicator a score of 1 to the less favored group, 3 to the most favored group and 2 to the intermediary group, by summing the scores and by dividing the sum by the number of available indicators for each worker.



**Figure S2:** Multiple correspondence analysis showing the association between the different socioeconomic indicators and the global measure of social position at baseline. The plot uses the two first dimensions which explain respectively 25.0 and 16.4% of the total inertia (57.4 and 13.2% with Greenacre adjustment).



**Figure S3:** Distribution and summary statistics of the global measure of work environment. It is calculated by giving for each occupational exposure a score of 1 to the non-exposed group, 2 to the exposed group and 1.5 to the intermediary group whenever the exposure encompasses three levels, by summing the scores and by dividing the sum by the number of available exposures for each worker.

