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Workplace health assessments and long-term cardiovascular risk: a propensity score-matched emulated target trial among 2 091 415 Swedish workers

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Objectives This study aimed to estimate the effect of participating in a workplace health assessment on cardiovascular disease incidence among Swedish workers. Further, to examine if the association differed by sex, age, and education, and whether the association changed with longer follow-up.

Methods Registry data was used to emulate a target trial of participation in a workplace health assessment versus no assessment, with 1:4 propensity score matching and a 10-year follow-up. A total of 418 283 individuals (45% women, median age 43 years) who participated in an occupational health profile assessment (HPA) from 1995 until 2021 were included. Controls (N=1 673 132) from the general working population were matched on sex, birth year and educational level within the calendar year of the HPA.

Results There were 6670 cardiovascular disease (CVD) events among HPA participants and 31 779 among controls during follow-up. Hazard ratios (HR) for CVD events were significantly lower for HPA participants compared to controls (adjusted for birth cohort to account for secular trends in CVD risk across generations, sex, age, comorbidity, prior CVD and income): 0.90 (95% confidence interval 0.87–0.92). In unrestricted follow-up (29 years), the association was stable through about 10 years and weakened thereafter. Additionally, the reduction in CVD risk relative to matched controls was greater among women than men.

Conclusions Participation in workplace health assessments was associated with a lower 10-year risk of CVD, with a larger reduction relative to matched controls among women. Residual confounding and selection cannot be ruled out, so the findings are consistent with, but do not establish, a preventive effect.

Key terms health profile assessment; health risk appraisal; Sweden.

Cardiovascular disease (CVD) is the leading cause of death globally (1). A substantial part of CVD is preventable. In individual-level data from about 1.5 million people in 34 countries, five modifiable risk factors (body mass index, systolic blood pressure, non-HDL cholesterol, smoking and diabetes) accounted for more than half of 10-year CVD incidence (2). These risk factors are largely shaped by health behaviors such as physical activity, diet, and tobacco and alcohol use (3, 4). Physical inactivity alone is projected to cause about 500 million new cases of non-communicable disease by 2030, including coronary heart disease, stroke and hypertension (5). Preventive measures that reach large groups of adults are therefore needed to reduce the burden of CVD.

Workplaces present unique opportunities to implement health-promoting activities that improve health behaviors and reduce cardiovascular risk across diverse

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sociodemographic groups, potentially enhancing preventive reach and equity. One common approach is workplace health assessments, also called health risk appraisals, which typically involve evaluating health status through self-report and physical examinations, followed by individual and/or group-level feedback and sometimes additional interventions (6). In Sweden, the first large-scale health assessment program (the Health Profile Assessment, HPA) with a standardized method and procedure to evaluate lifestyle behaviors and health parameters was developed during the 1970s and 1980s (7, 8). The HPA measures several of these risk factors directly, including blood pressure, body mass, smoking and cardiorespiratory fitness. To date, approximately 800 000 assessments have been recorded in a centralized database since the 1990s. In a recent paper, we investigated the reach of the HPA over the period 1995 to 2021 (9), showing that while participants were not fully representative of the Swedish workforce, the program had reached various sociodemographic groups and work organizations, including those with low education or income and those born outside Europe – groups often considered hard to reach in non-workplace interventions (10).

The short-term effectiveness of workplace health promotion interventions on health behaviors and risk factors shows mixed findings (11). While some reviews of multicomponent interventions report improvements in physical activity, diet, and cardiometabolic risk (12–14), others have found no or inconsistent effects (15–17). We recently investigated how participation in repeated HPA over five years related to changes in weight, perceived health, and exercise frequency (18). Findings revealed that more frequent assessments over shorter periods were associated with positive effects on all three outcomes, while single or infrequent assessments had a limited impact. However, heterogeneity in intervention types, study populations, and methodology quality – with most studies showing low or very low quality (11) – makes short-term effectiveness difficult to conclude.

To our knowledge, no studies have explored workplace health assessment effects on long-term chronic disease outcomes such as CVD. A few studies reported mortality and cardiovascular event reductions among workers participating in health seminars or lifestyle programs in primary healthcare, compared with non-participants or matched controls (19–21). However, evidence is needed to understand whether workplace health assessments contribute to long-term disease prevention and workforce health sustainability.

The primary aim was to estimate the effect of participating in a workplace health assessment on cardiovascular disease incidence among Swedish workers. The secondary aim was to examine if the association differed by sex, age, and education, and whether the association changed with longer follow-up.

Methods

This observational study emulated a target trial to estimate the effect of participation in a workplace health assessment on cardiovascular disease incidence (22), using propensity score matching for confounding control and a ten-year restricted follow-up for the primary aim. The secondary aims used the same framework to examine effect modification at ten years and change in the association over unrestricted follow-up. Data was obtained from the HPA database for HPA participants and from national registers for matched controls. The process leading to a HPA has been described previously (9). In brief, the process is initiated by the employer, who chooses to offer a HPA to employees. Subsequently, a contract is established with a health assessment provider, most commonly an occupational health service. If the selected provider includes HPA in its service offerings – which approximately 90% of occupational health services in Sweden do – the employer and provider may agree to conduct the assessment. Employees are then informed of the opportunity and offered the option to participate. Participation in the HPA is voluntary and provided at no cost to the employee. The present study was approved by the Stockholm Ethics Review Board (Dnr 2015/1864-31/2; 2016/9-32; 2019-05711; 2023-07086-02) and conducted following the principles of the Declaration of Helsinki. All participants provided informed consent before HPA data collection. The research was classified as register-based, and no further consent was deemed necessary by the ethics board.

Health Profile Assessment participants

Between 1 January 1990 and 31 December 2021, the HPA database comprised 436 918 unique individuals who performed a HPA. To ensure completion of a HPA, inclusion criteria required data for at least two of six core questionnaire variables (exercise, smoking, diet, perceived health, stress, and back/neck pain) and two of five physical examination variables (estimated maximal oxygen intake [VO_2max], height, weight, systolic and diastolic blood pressure). Additionally, records before 1995 with low assessment rates and data inconsistencies, individuals under 18 years of age, those lacking verified salary or salary source data, and those who had no match were excluded. Eligibility spanned ages 18 to 85 years. Eighteen is the minimum age for regular employment in Sweden, and the upper bound of 85 retains workers active beyond the usual retirement window, since Sweden has no fixed retirement age and pension withdrawal is flexible, most commonly 65–67 years. The final study population comprised 418 283 individuals, of whom 1758 were >65 years (supple-

mentary figure S1). Information on those who declined a HPA is not available.

Matched controls from the Swedish working population

Controls were drawn from Statistics Sweden. For every calendar year from 1995 to 2021, the base population was all individuals aged ≥18 employed in Sweden, giving 243 440 352 person-years, of which 127 403 387 had a verified salary and confirmed employment and formed the eligible control pool. Controls were randomly selected from this pool and matched to participants as described below, giving 1 673 132 matched controls.

The Health Profile Assessment

The HPA method consists of three core components: a standardized questionnaire on physical activity, lifestyle, perceived health and symptoms; a physical examination of body mass, height, waist circumference, blood pressure and cardiorespiratory fitness; and a concluding behavior-change dialogue of about 20–40 minutes delivered with motivational interviewing. Assessments are stored in a central database maintained by the HPI Health Profile Institute, which developed and standardized the method. Standardization includes a written protocol and a 3–4 day training and certification for coaches, who are usually nurses or physiotherapists in occupational health services.

Covariates and matching variables

Sociodemographic and work-related characteristics were obtained from Statistics Sweden through linkage using participants' personal identification numbers. The place of origin was categorized as Sweden, Europe, and outside of Europe. Civil status was categorized into Partner and Single. Educational level was obtained using the Swedish standard classification of education, a seven-level classification system that categorizes educational attainment by duration and level, ranging from pre-gymnasium education <9 years to research education. Occupation (available from 2001) was classified according to the Swedish Standard Classification of Occupations, which aligns with the International Standard Classification of Occupations (23). Occupation was defined at both the major and sub-major group levels, with a similar classification approach as applied in previous studies (9, 24, 25). Annual income from employment was categorized into five categories based on percentages of the median in the population (26). Income was included as a confounder in the outcome model rather than as a matching variable. Matching on income would have required a new control population, for which outcome data were not available. Municipality of residence was metropolitan, dense or rural (27). Comorbidity used the register-adapted Charlson index in four categories (0, 1, 2, ≥3), with the outcome ICD codes removed and modeled separately (28). Baseline characteristics are shown in table 1.

Lifestyle variables were available only for participants, collected at the HPA. Body mass index (BMI) was computed from measured height and weight. Cardiorespiratory fitness was estimated as VO_2max from the Åstrand submaximal cycle ergometer test (29). Exercise frequency was self-reported using the statement "I exercise for the purpose of maintaining/improving my physical fitness, health and well-being…" with the alternatives never, sometimes, 1–2 times/week, 3–5 times/week, or ≥6 times/week. General health was self-rated health using the statement "I perceive my physical and mental health as..." with the alternatives very poor, poor, neither good nor bad, good, or very good. Number of HPA within a five-year period after their first HPA was categorized into 1, 2, or ≥3 HPA. These variables are presented in supplementary figure S2.

Outcome

The Swedish National Patient Register and the National Cause of Death Register were used to gather information on CVD diagnoses. All participants were followed from their first HPA, or for controls, from the date of HPA of their matched HPA participant (defined as time zero), to the first CVD event after time zero, death, or until 2024-06-05. Incident CVD events were defined as ICD-10 codes I21, I25, I46, I63, I64, I65, or I66.

Statistical analysis

The target trial protocol and its emulation are summarized in table 2, following the TARGET statement (30). HPA participants were matched to four controls (1:4 nearest-neighbor, no replacement) via propensity-score matching using logistic regression on sex, birth year (continuous), and educational level (7 levels), stratified by calendar year. Before matching, 77 of 405 covariate-year combinations (19%) had absolute standardized mean differences (SMD) exceeding 0.1, and 36 (9%) exceeded 0.25 (median SMD=0.03, range <0.01–0.34). After matching, all covariate-year combinations had an absolute SMD <0.1 (median <0.001, maximum=0.07).

The primary analysis estimated the effect of HPA participation on CVD over ten years, modeled with Cox proportional hazards. The primary model (M3) adjusted for birth cohort (birth year in ten-year groups), age at time zero, sex, comorbidity, prior CVD and income. Sex and birth year (as birth cohort), which were also matching variables, were kept in the model, giving doubly

Table 1. Characteristics of the study population at baseline. The main comparison examines controls versus Health Profile Assessment (HPA) participants. The HPA population is further stratified by the total number of HPA received within five years of their first HPA, with the characteristics at the first HPA presented in the table. Percentages for categories are among individuals with known values; for 'unknown', the percentage is of the column total. Sex, civil status, income, Charlson comorbidity index and prior CVD had no missing values. [CVD=cardiovascular disease.]

Characteristic	Main comparison		Baseline characteristics (first HPA) for participants stratified on number of HPA within 5-years		
	Control (N=1 673 132)	HPA participants (N=418 283)	1 HPA (N=265 849)	2 HPA (N=113 954)	≥3 HPA (N=38 480)
	N (%)	N (%)	N (%)	N (%)	N (%)
Sex					
Men	915 852 (55)	228 951 (55)	144 883 (54)	61 780 (54)	22 288 (58)
Women	757 280 (45)	189 332 (45)	120 966 (46)	52 174 (46)	16 192 (42)
Place of origin					
Outside Europe	119 356 (7.1)	15 429 (3.7)	10 384 (3.9)	3900 (3.4)	1145 (3.0)
Europe	140 275 (8.4)	26 455 (6.3)	17 297 (6.5)	6974 (6.1)	2184 (5.7)
Sweden	1 413 368 (84)	376 383 (90)	238 157 (90)	103 075 (90)	35 151 (91)
Unknown	133 (<0.1)	16 (<0.1)	11 (<0.1)	5 (<0.1)	0 (0)
Civil status					
Partner	770 652 (46)	201 363 (48)	125 379 (47)	56 355 (49)	19 629 (51)
Single	902 480 (54)	216 920 (52)	140 470 (53)	57 599 (51)	18 851 (49)
Education level					
Primary	158 955 (9.5)	38 762 (9.3)	24 044 (9.1)	11 071 (9.7)	3647 (9.5)
Secondary	788 168 (47)	196 718 (47)	121 321 (46)	55 855 (49)	19 542 (51)
Tertiary	717 780 (43)	181 607 (44)	119 670 (45)	46 746 (41)	15 191 (40)
Unknown	8229 (0.5)	1196 (0.3)	814 (0.3)	282 (0.2)	100 (0.3)
Occupation (since 2001)					
Managers	92 616 (6.6)	27 775 (7.3)	17 448 (7.3)	7541 (7.3)	2786 (8.1)
Science and engineering	60 371 (4.3)	28 864 (7.6)	18 969 (7.9)	7595 (7.3)	2300 (6.7)
Health care	44 095 (3.1)	6597 (1.7)	4050 (1.7)	1805 (1.7)	742 (2.2)
Education	89 473 (6.4)	14 235 (3.8)	10 278 (4.3)	3140 (3.0)	817 (2.4)
Other professionals	115 713 (8.3)	36 622 (9.7)	25 085 (10)	8957 (8.6)	2580 (7.5)
Associate professionals	257 277 (18)	86 154 (23)	54 634 (23)	23 310 (22)	8210 (24)
Administration and customer service	116 571 (8.3)	35 794 (9.5)	22 373 (9.3)	9794 (9.5)	3627 (11)
Manufacturing	61 328 (4.4)	20 838 (5.5)	13 438 (5.6)	5572 (5.4)	1828 (5.3)
Mechanical manufacturing	72 450 (5.2)	33 576 (8.9)	19 814 (8.2)	10 589 (10)	3173 (9.2)
Building	68 878 (4.9)	19 197 (5.1)	11 587 (4.8)	5349 (5.2)	2261 (6.6)
Agriculture and forestry	18 260 (1.3)	2742 (0.7)	1739 (0.7)	712 (0.7)	291 (0.8)
Service and shop sales	105 214 (7.5)	13 388 (3.5)	9231 (3.8)	3268 (3.2)	889 (2.6)
Transport	51 209 (3.7)	7024 (1.9)	4484 (1.9)	1948 (1.9)	592 (1.7)
Cleaners	24 125 (1.7)	5275 (1.4)	3198 (1.3)	1578 (1.5)	499 (1.5)
Personal care	169 653 (12)	30 803 (8.1)	18 151 (7.5)	9677 (9.3)	2975 (8.7)
Other elementary occupations	52 951 (3.8)	9673 (2.6)	6123 (2.5)	2794 (2.7)	756 (2.2)
Unknown	272 948 (16)	39 726 (9.5)	25 247 (9.5)	10 325 (9.1)	4154 (11)
Income (percentage of median)					
<60	242 110 (14)	11 612 (2.8)	8246 (3.1)	2533 (2.2)	833 (2.2)
60–79	187 361 (11)	26 660 (6.4)	17 343 (6.5)	7074 (6.2)	2243 (5.8)
80–119	632 733 (38)	165 260 (40)	104 603 (39)	45 951 (40)	14 706 (38)
120–199	489 023 (29)	173 693 (42)	109 959 (41)	47 510 (42)	16 224 (42)
≥200	121 905 (7.3)	41 058 (9.8)	25 698 (9.7)	10 886 (9.6)	4474 (12)
Municipality of residence					
Metropolitan municipalities	547 126 (33)	115 662 (28)	74 589 (28)	29 732 (26)	11 341 (29)
Dense municipalities	742 431 (44)	204 714 (49)	131 304 (49)	55 528 (49)	17 882 (47)
Rural municipalities	381 967 (23)	97 671 (23)	59 860 (23)	28 588 (25)	9223 (24)
Unknown	1608 (<0.1)	236 (<0.1)	96 (<0.1)	106 (<0.1)	34 (<0.1)
Charlson comorbidity index score					
0	1 618 305 (97)	406 296 (97)	258 078 (97)	110 767 (97)	37 451 (97)
1	25 883 (1.5)	5563 (1.3)	3600 (1.4)	1477 (1.3)	486 (1.3)
2	27 036 (1.6)	6106 (1.5)	3961 (1.5)	1627 (1.4)	518 (1.3)
≥3	1908 (0.1)	318 (<0.1)	210 (<0.1)	83 (<0.1)	25 (<0.1)
CVD incidence (before HPA)	16 307 (1.0)	3162 (0.8)	2138 (0.8)	785 (0.7)	239 (0.6)
CVD incidence (within ten years)	31 779 (1.9)	6670 (1.6)	4313 (1.6)	1788 (1.6)	569 (1.5)
CVD incidence (unrestricted)	60 471 (3.6)	13 228 (3.2)	8154 (3.1)	3807 (3.3)	1267 (3.3)

robust estimation and protection against residual confounding if the propensity score model is misspecified. Proportional hazards were assessed with Schoenfeld residuals. The assumption held for HPA participation (P=0.39). Some covariates deviated, most clearly prior CVD, but the estimate was unchanged when individuals with prior CVD were excluded (supplementary table S2). Because controls were matched to participants, the estimate is the average treatment effect in the treated (ATT) and does not represent assessing the whole workforce (figure 1).

All participants and controls had a verified income, and sex, birth year, civil status, comorbidity and prior CVD had no missing values. Missing values for other

Table 2. Target trial emulation protocol. Following the TARGET statement (30). [CVD=cardiovascular disease; HPA=Health Profile Assessment.]

Protocol component	Target trial specification	Emulation using observational data
Eligibility criteria	Inclusion: employed in Sweden, aged 18–85 years, with verified salary confirming employment.	HPA participants from the HPA database linked to Statistics Sweden registers; controls from the employed population with verified salary.
	Exclusion: not employed.	
	Prior CVD does not affect eligibility; it is recorded at baseline and handled in the analysis.	Prior CVD retained and adjusted for in the primary analysis, excluded in a sensitivity analysis.
		Age range justified in Methods (Eligibility). Study flow and final sample in supplementary figure S1.
Treatment strategies	Intervention: complete a HPA (standardized questionnaire, physical examination, behaviour-change dialogue).	Intervention: first recorded HPA (1995–2021), requiring ≥2 of 6 questionnaire items and two of five examination measures.
	Comparator: no assessment.	Comparator: no HPA record.
		HPA components described in Methods.
Assignment procedures	Random assignment to HPA or no HPA, open-label.	1:4 nearest-neighbor propensity score matching (logistic regression on sex, birth year, education), stratified by calendar year.
		Balance assessed by standardized mean differences (see Methods, Statistical analysis).
		Assignment assumed independent of potential outcomes conditional on measured covariates; unmeasured organizational and behavioral factors may violate this.
Time zero	Follow-up begins at assignment, aligning eligibility, treatment, and start of follow-up.	Date of the first HPA for participants and the same calendar date for their matched controls (see Methods, Outcome).
Follow-up period	From time zero to the first CVD event, death (competing event), 10 years (primary), or administrative end (secondary).	Through linked registers until 2024-06-05.
		Censoring at CVD event, death, 10 years (primary), or 29 years (secondary).
	Non-informative censoring assumed.	
Outcome	First incident CVD event (myocardial infarction, chronic ischemic heart disease, cardiac arrest, stroke, cerebrovascular disease), from medical records or cause of death.	Swedish National Patient Register and Cause of Death Register.
		ICD-10 I21, I25, I46, I63, I64, I65, I66. Prior CVD defined by the same codes before time zero (see Methods, Outcome).
Causal contrast (estimand)	Intention-to-treat effect of assignment to HPA versus no HPA at time zero, regardless of later participation or behavior.	Groups compared as classified at time zero.
	Relative measure: cause-specific hazard ratio, death censored.	Because controls were matched to participants, the estimand is the average treatment effect in the treated (ATT); it does not represent assessing the whole workforce.
	Absolute measure: risk difference at 10 years.	
	Fine-Gray subdistribution hazard specified as a sensitivity analysis.	Effect estimates reported in Results.
Identification assumptions	Exchangeability, positivity, consistency, no interference, non-informative censoring.	Exchangeability conditional on the matched variables and primary-model covariates.
		Positivity supported by the large control pool and post-matching balance.
		Consistency given the standardised HPA. Administrative censoring assumed non-informative; death censored in the primary analysis and competing in Fine-Gray.
		Main threat: unmeasured confounding by health consciousness, employer factors, and lifestyle (measured only in participants), quantified by an E-value reported in Results.
Statistical analysis plan	Primary: Cox model, 10-year follow-up, pre-specified confounders.	Primary model (M3): birth cohort, age, sex, comorbidity, prior CVD, income. Proportional hazards assessed (see Methods).
	Secondary: effect modification by sex, age, education; change in the association over unrestricted follow-up.	Effect modification by interaction terms.
	Sensitivity: alternative confounder sets (M1 to M5), one-year landmark, excluding prior CVD, ages 18–65, Fine-Gray.	Exploratory: number of HPA (baseline-classified and immortal-time-corrected) and lifestyle variables available only in participants.
		Full specification in Methods; code at 10.5281/zenodo.21103683

variables are shown in table 1; occupation is available only from 2001. Individuals with missing values were excluded from M4 and M5. No imputation was used.

Two secondary analyses used the same model. Effect modification by sex, age and education was tested with interaction terms over the ten-year follow-up (figure 1). Change in the association with time since the HPA was examined over unrestricted follow-up (≤29 years) with flexible parametric survival models (restricted cubic splines, two internal knots, log cumulative hazard scale), estimating time-varying hazard ratios (HR) at 90, 183, 365, 547 and 730 days and annually thereafter. Precision falls at longer follow-up, where estimates rest on the earliest-enrolled participants (figure 2; subgroups in supplementary figure S3).

Sensitivity analyses tested robustness (supplementary table S2). Five adjustment sets were compared: M1 (birth cohort), M2 (+ age, sex, comorbidity, prior

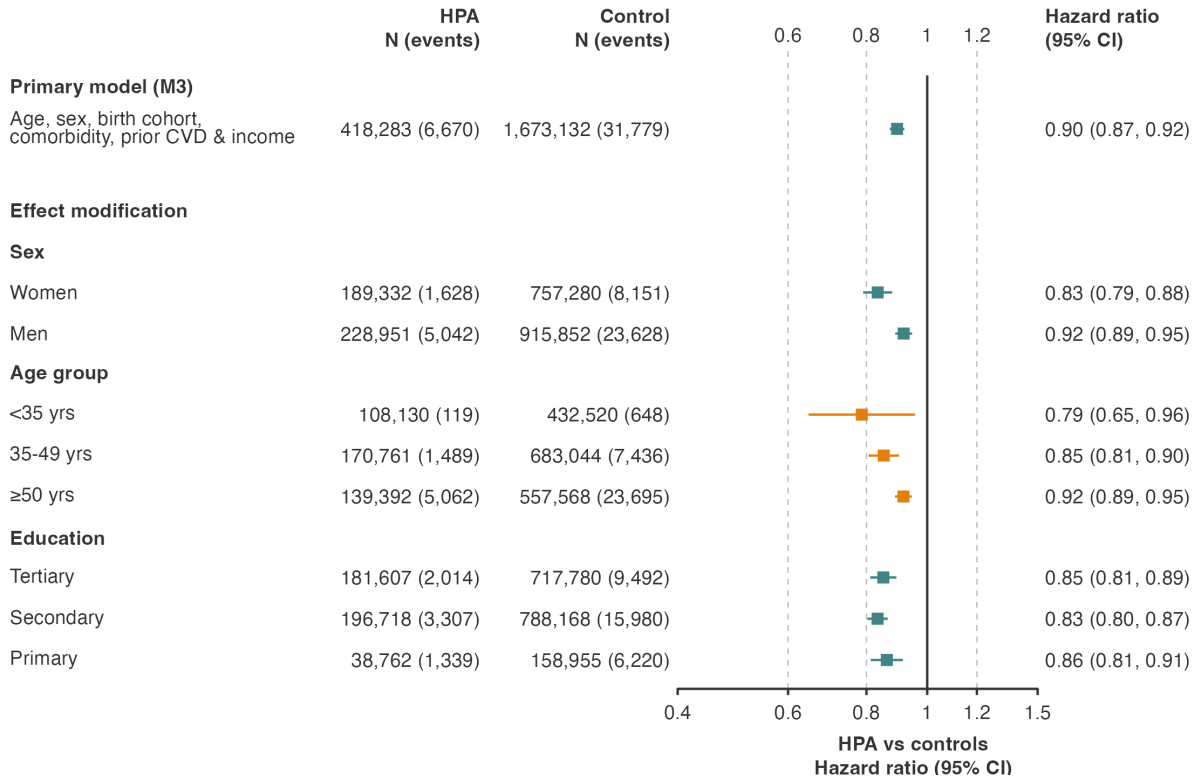

Figure 1. Hazard ratios for cardiovascular disease incidence among Health Profile Assessment (HPA) participants compared with matched controls over the 10-year follow-up. Results are shown overall and by sex, age group and education, all based on the primary model (M3). As these were matching variables, each subgroup compares participants with their own matched controls. [CI=confidence interval.]

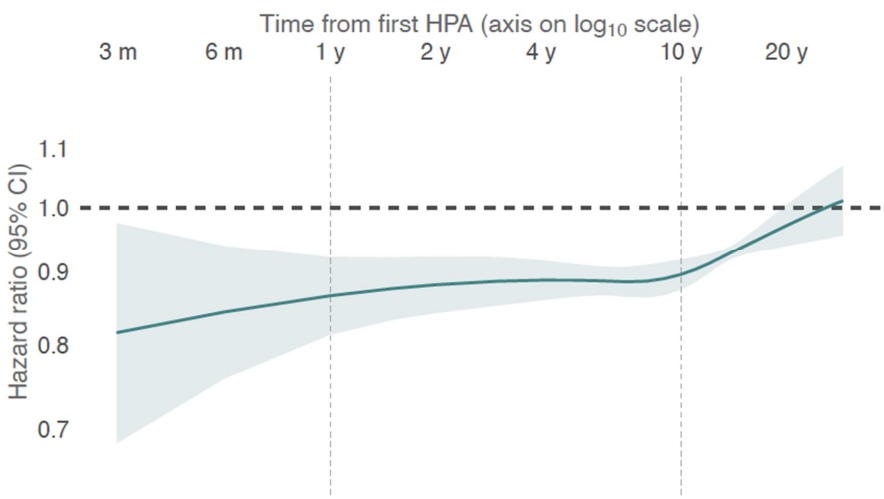

Figure 2. Time-varying smoothed hazard ratios for cardiovascular disease (CVD) incidence among Health Profile Assessment (HPA) participants compared with their matched controls. Flexible parametric survival models with unrestricted follow-up time are used, adjusting for birth cohort, age, sex, comorbidity, prior CVD, and income. [CI=confidence interval.]

CVD), M3 (+ income, the primary model), M4 (+ occupation) and M5 (+ municipality and place of origin) (supplementary figure S2). A one-year landmark analysis (follow-up from 365 days, delayed-entry risk sets) addressed reverse causation and early treatment effects. The primary model was also refitted excluding matched sets in which any member had prior CVD, and restricting to participants aged 18–65 years. Competing risk from death was assessed with Fine-Gray subdistribution hazards and cumulative incidence functions (supplementary figure S5). Immortal-time bias in the number of HPA was checked with a counting-process Cox model, with one HPA as reference (supplementary table S1). To gauge unmeasured confounding, E-values were computed for the primary estimate and for its confidence limit closer to the null, treating the low-incidence hazard ratio (<2% at ten years) as a risk ratio; the E-value is the minimum risk-ratio association an unmeasured confounder would need with both exposure and outcome, beyond the measured covariates, to explain the association fully.

Exploratory analyses examined assessment frequency (number of HPA within five years) and lifestyle-related characteristics available only for participants (BMI, estimated VO_2max, exercise frequency, self-rated health). For these, each control was given the value of their matched participant, and participants without a value were excluded with their controls. As the variables were not measured in controls, these analyses do not test effect modification (supplementary figure S2).

Population-level effect was summarized with marginal effects from the primary model: the absolute risk difference at ten years from marginal survival, its reciprocal as the number associated with one fewer CVD event, and restricted mean survival time differences at 5, 10 and 20 years (figure 3; subgroups in supplementary figure S4).

Analyses used R 4.4.2 with MatchIt, survival, flexsurv, EValue and marginaleffects packages (31–35). Code available at 10.5281/zenodo.21103683.

Results

A total of 418 283 HPA participants were matched to 1 673 132 controls, yielding 5 625 047 person-years of observation among HPA participants and 22 311 743 person-years among controls. Median follow-up time was 13.2 years [Interquartile range (IQR) 8.8–17.7 and range 0.05–29.4]. Within ten years, there were 6670 CVD events among participants and 31 779 among controls (1.78 and 2.13 per 1000 person-years); over the full follow-up there were 13 228 and 60 471 events (2.35 and 2.71 per 1000 person-years).

Baseline characteristics were similar after matching, with small differences in the non-matching variables civil status, municipality, and comorbidity (table 1). Income differed most and was the main non-matching confounder. Characteristics were similar across participants with one, two, or three or more HPA.

Over the ten-year follow-up, participants had a lower rate of CVD than their matched controls [M3: HR 0.90, 95% confidence interval (CI) 0.87–0.92; figure 1]. Adjustment for income accounted for most of the difference from the birth-cohort-only model, while age, sex,

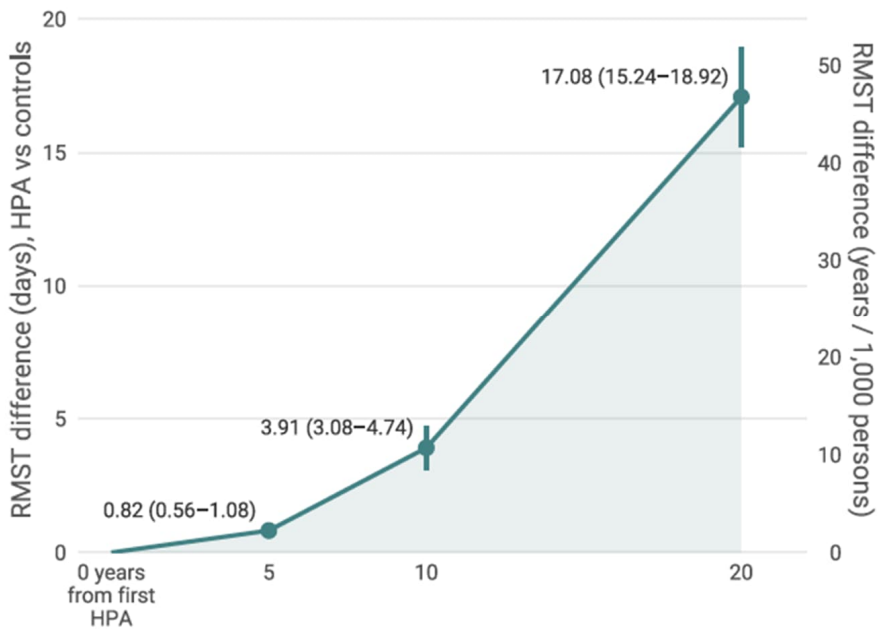

Figure 3. Restricted mean survival time (RMST) difference between Health Profile Assessment (HPA) participants and matched controls at 5, 10 and 20 years. Each point is the difference in cardiovascular disease (CVD)-free time (area between the survival curves) accumulated up to that horizon, with 95% confidence intervals (CI).

comorbidity and prior CVD changed the estimate only marginally (M1, M2, M4, M5, supplementary figure S2). The E-value was 1.46 for the point estimate and 1.39 for the confidence limit closer to the null, so an unmeasured confounder would need a risk-ratio association of ≥1.46 with both exposure and outcome, beyond the measured covariates, to explain the association. For the pre-specified subgroups, the reduction relative to matched controls was greater in women than men, with a trend toward greater reduction in younger participants (figure 1).

Exploratory analyses are reported in the supplement. The reduction relative to matched controls was larger among participants with higher income. Participants with repeated HPA or a more favorable lifestyle had a lower CVD risk than their matched controls, but these comparisons do not test effect modification as the controls do not have any performed HPA (supplementary-figure S2). In unrestricted follow-up, the HR was stable and below one for about ten years and then weakened, with CI including one after about 19 years (figure 2).

At the population level, the absolute risk difference at ten years was about 1.8 fewer CVD cases per 1000 participants, or one fewer event per 549 participants (95% CI 441–735). Restricted mean survival time differences were small at the individual level, with about 18 more CVD-free days at 20 years, equivalent to 49 CVD-free years per 1000 individuals (figure 3; subgroups in supplementary figure S4).

Discussion

In this study of over two million Swedish workers, participation in a workplace health assessment was associated with a 10% lower CVD risk over ten years compared with matched controls, and the reduction relative to controls was larger in women than men. The risk was stable and lower for about ten years and then weakened at longer follow-up. At the population level, participation in HPA was associated with 18 more CVD-free days at 20 years, or 49 CVD-free years per 1000 individuals. The E-value was modest, so a moderately strong unmeasured confounder, for example one linked to health consciousness or employer characteristics, could account for the association; the findings are consistent with, but do not establish, a preventive effect.

Our findings align with earlier long-term studies. One previous long-term follow-up study showed 17% lower overall mortality rates among participating employees compared to non-participants following attendance at a health seminar program lasting 2–3 weeks (19). The lower mortality observed is consistent with our findings, though direct comparison is limited by differences in intervention type and outcome measures. Further, a Swedish prevention program based in primary healthcare targeted individuals with at least one cardiovascular risk factor, promoting physical activity and healthy lifestyles (20). Over 22 years of follow-up, participants showed a 12% lower risk of first cardiovascular events, a 21% reduction in cardiovascular mortality, and a 17% decrease in all-cause mortality compared to a matched reference group from the general population. Long-term follow-up of a Swedish community-based health intervention comprising health examinations, lifestyle-directed dialogues, and group activities showed 29% reduced all-cause mortality compared to national controls after 24 years (36). Our estimates were of similar size, suggesting that occupational health services delivering health assessments can potentially serve as an effective platform for CVD prevention.

Possible explanations for the observed lower CVD risk associated with HPA participation might include a direct intervention effect from HPA participation itself. Although a systematic review of meta-analyses found mixed effects of a wide range of single- and multicomponent workplace health promotion interventions on health behaviors and cardiometabolic risk factors (11), non-workplace interventions have shown more consistent benefits. For example, the Västerbotten Intervention Program is a systematic and county-wide CVD intervention program implemented in primary care in northern Sweden targeting inhabitants at the ages 40, 50, and 60 years. It includes similar core individual intervention components as the HPA – physical examination, comprehensive questionnaire, and health dialogue – and has shown significant beneficial effects on physical activity (37), obesity (38), and hypertension (39). Long-term follow-up has also demonstrated significant reductions in both all-cause and cardiovascular mortality rates (21). Similarly, lifestyle interventions in adults at risk for CVD show positive effects on both diet and physical activity, with subsequent lower risk for CVD events (40). However, the Look AHEAD trial is an example of an intensive lifestyle intervention in a randomized controlled trial setting, with no effects on cardiovascular outcomes despite significant beneficial effects on intermediate risk factors, including weight loss, higher fitness, and cardiometabolic risk factors (41). In a previous paper, we reported only small changes in weight, perceived health, and exercise frequency following participation in a HPA (18); however, multiple and more frequent assessments over a shorter period yielded small yet significant improvements in both perceived health and exercise frequency. This aligns with research showing that more than one encounter with motivational interviewing increases the likelihood of behavior change compared with a single encounter (42, 43). In the present study, participants with three or

more HPA had a lower CVD risk than those with one HPA (supplementary figure S2), but this association was weak and not statistically significant after correction for immortal time (supplementary table S1).

A selection effect may be another potential explanation for the results. Despite the target trial framework, matching, and adjustment, HPA participants may have had more favorable health behaviors and prerequisites for good health at baseline, as well as better health status during follow-up, compared to controls. Participants with a less favorable lifestyle had a CVD risk close to that of their matched controls, while participants with a more favorable lifestyle had a lower risk (supplementary figure S2). As lifestyle was not measured in controls, we could not test whether the groups differed, but this pattern is consistent with selection on lifestyle. This selection bias may operate at both individual and structural levels. At the individual level, those with initially healthier lifestyles may be both more likely to participate in HPA and more responsive to the behavioral interventions they provide. At the structural level, since HPA are conducted by occupational health services on behalf of employers as part of workplace health and work environment initiatives, selection may occur through organizational pathways. Individuals who are offered and choose to participate in HPA may be employed at companies with stronger health strategies and greater financial resources to invest in health initiatives, potentially creating a workplace environment that both attracts and supports healthier individuals. The identification of income as the primary non-matching confounder underscores this complexity, as it also operates simultaneously at multiple levels. At the individual level, higher income is linked to healthier baseline behaviors and health literacy (44, 45). At the structural level, companies with greater financial resources are more likely to contract occupational health services and implement workplace health strategies, thereby creating environments that both attract and sustain healthier employees (9).

The larger reduction among women may reflect differences in response to the HPA, differences in who chooses to participate, or both. Women may derive greater benefit from the motivational interviewing approach due to documented differences in health-seeking behavior (46, 47), while younger participants may have greater capacity for lifestyle modification and longer time horizons to accumulate cardiovascular benefits from behavior change, consistent with the life-course approach to CVD prevention (48, 49).

Major strengths of this study include the large sample size, long follow-up period, and methodological approach using an emulated target trial design. Limitations include the inability to determine which specific components of the HPA method or aspects of its delivery may have influenced intermediate factors or directly affected CVD risk among participants. Although the HPA follows a systematic and standardized process, it was not originally designed as a research study. Variability in the delivery of behavior-change dialogues, along with participant factors such as low self-efficacy, limited motivation, or low health literacy, may have affected outcomes. Additionally, workplace-related factors – including organizational prerequisites and culture – may be even more influential on intervention effectiveness, yet these are often overlooked in workplace health promotion research (50). Unmeasured confounding remains possible despite matching and adjustment. The study population was limited to Swedish workers with access to occupational health services, potentially limiting generalizability to other populations or healthcare systems. The analysis of multiple HPA is subject to immortal-time bias, since individuals must remain alive, event-free and employed to receive further assessments within five years. After correction for this, the association was weak and not statistically significant (supplementary table S1), and any remaining difference may reflect health consciousness and motivation rather than an effect of the HPA. Individual employment history was not reconstructed and may leave residual confounding; income and occupation capture part of it, and the E-value indicates the confounding strength that would be needed to explain the findings.

Concluding remark

Participation in workplace health assessments was associated with a 10% lower ten-year CVD risk, with a larger reduction relative to matched controls among women. Residual confounding and selection cannot be ruled out, so the findings are consistent with, but do not establish, a preventive effect. Randomized trials with long-term follow-up are needed to confirm them and to clarify which participants benefit most.

Ethics approval and consent to participate

The Stockholm Ethics Review Board approved the study (Dnr 2015/1864-31/2; 2016/9-32; 2019-05711; 2023-07086-02), which follows the principles of the Declaration of Helsinki. All participants provided informed consent before HPA data collection. The ethical board classified the research as register-based, with no requirement for additional consent from the participants.

Availability of data and materials

The data that support the findings of this study are available from the HPI Health Profile Institute (HPI) but restrictions apply to the availability of these data,

which were used under license for the current study, and so are not publicly available. Interested parties can request access to the data from the authors upon reasonable request and with the permission of HPI. For inquiries regarding data access, please contact HPI at peter.wallin@hpi.se.

Competing interests

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. Authors declare no conflict of interests.

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