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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1. Supplementary Material
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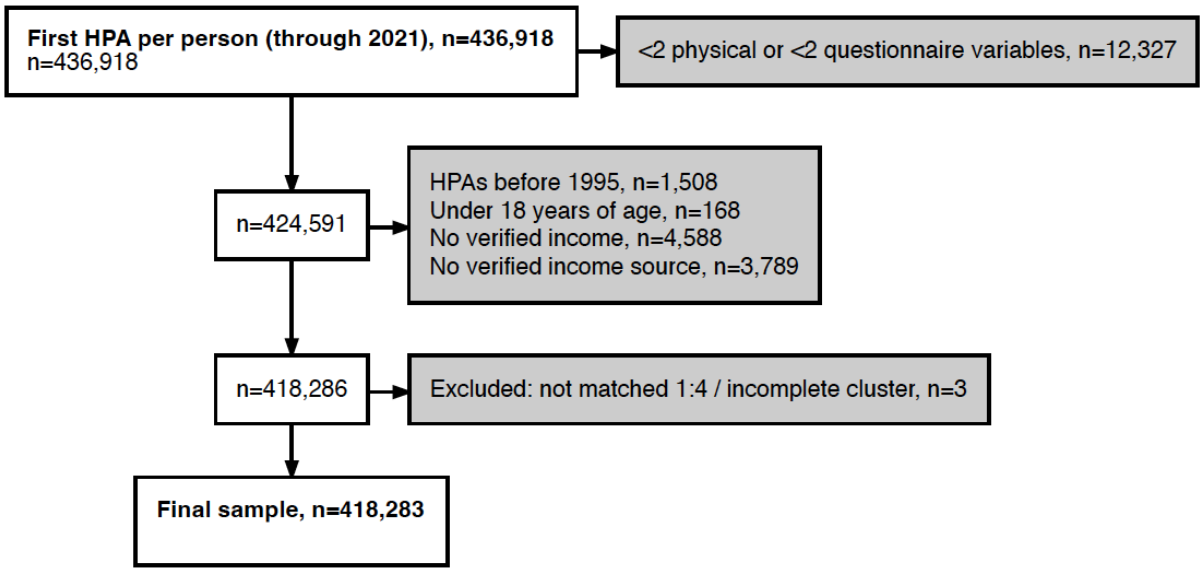

Figure S1. Flow chart of HPA participants.

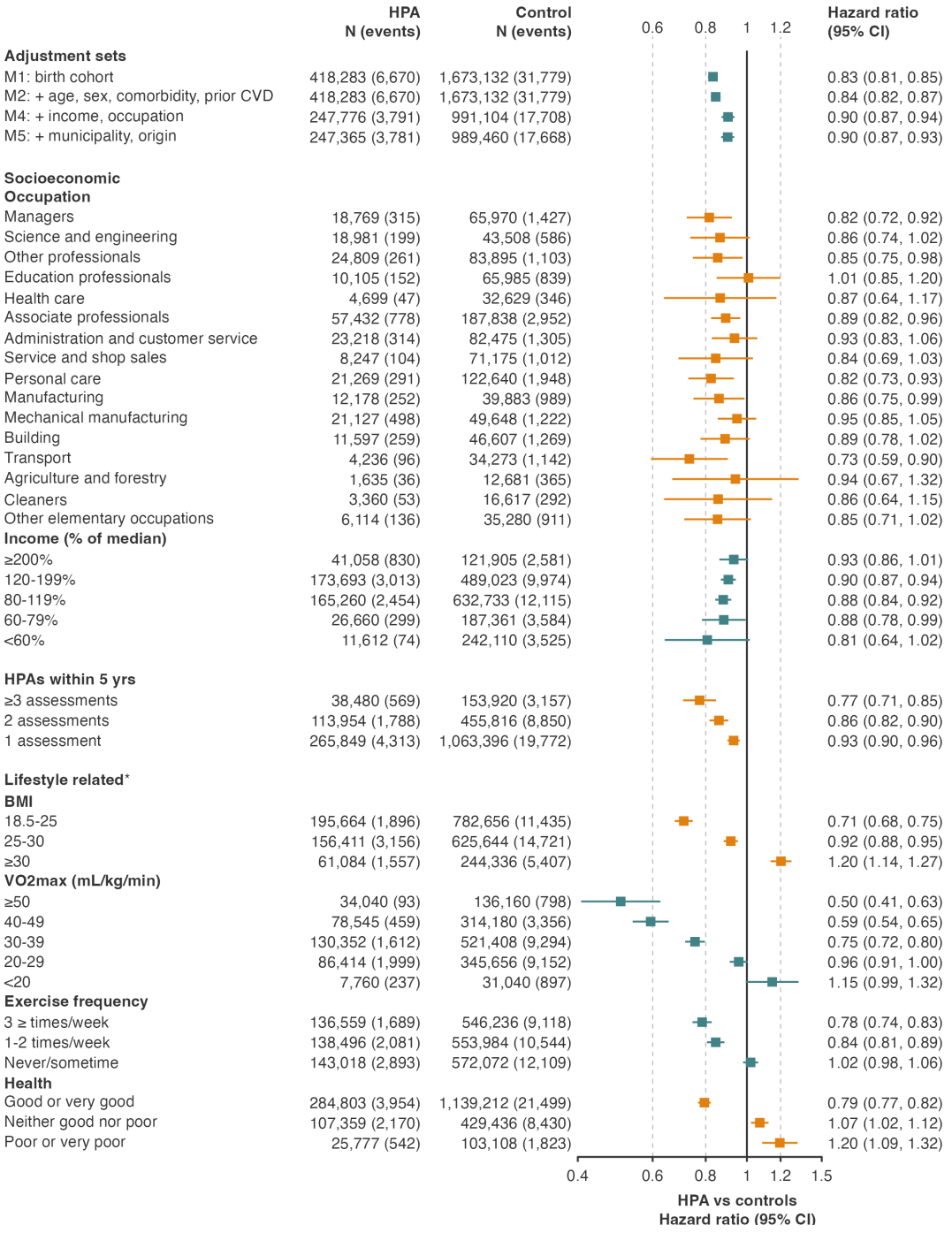

Figure S2. Hazard ratios (95% confidence intervals) for cardiovascular disease incidence among HPA participants compared with matched controls over the 10-year follow-up. The upper panel shows models with increasing adjustment (M1 to M5); M3 is the primary model, shown in Figure 1. All other estimates are based on M3. The middle panel shows subgroups by income and occupation, which were measured in both groups but not used for matching, so participants and controls within a subgroup are not necessarily from the same matched set. The lower panel shows variables measured only at the HPA (number of HPAs within five

years, BMI, VO_2max, self-rated exercise and self-rated health). Here each control was given the value of their matched participant, so each hazard ratio compares participants in one group with their own matched controls. Participants without a value, and their controls, were excluded from that analysis. These analyses do not test effect modification. The lifestyle of the controls is unknown and is likely similar across groups, since they were matched only on sex, birth year and education. Differences between groups therefore combine any difference in the effect of the HPA with the association between the lifestyle factor itself and CVD, and the two cannot be separated. The analyses show how strongly lifestyle is related to CVD among participants, compared with the size of the main result. As the difference across lifestyle groups is larger than the overall 10% difference, a modest difference in lifestyle between participants and controls could explain the main result. Participants with data: VO_2max 337,111 (81%), BMI 417,048, self-rated exercise 418,073 and self-rated health 417,939, of 418,283. M; Model, HPA; Health Profile Assessment, BMI; Body Mass Index.

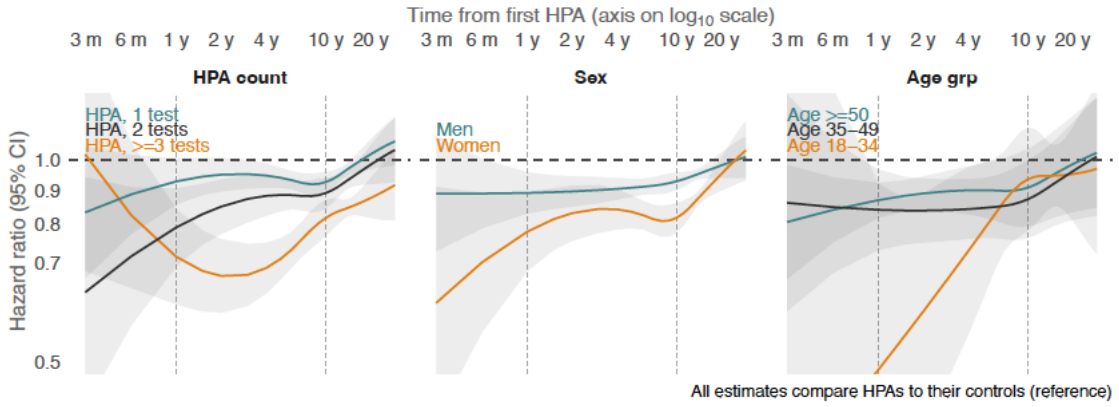

Figure S3. Time-varying smoothed hazard ratios (95% confidence intervals) for cardiovascular disease incidence among HPA participants compared with their matched controls. Flexible parametric survival models with unrestricted follow-up time are used, adjusting for birth cohort, age, sex, comorbidity, prior CVD, and income.

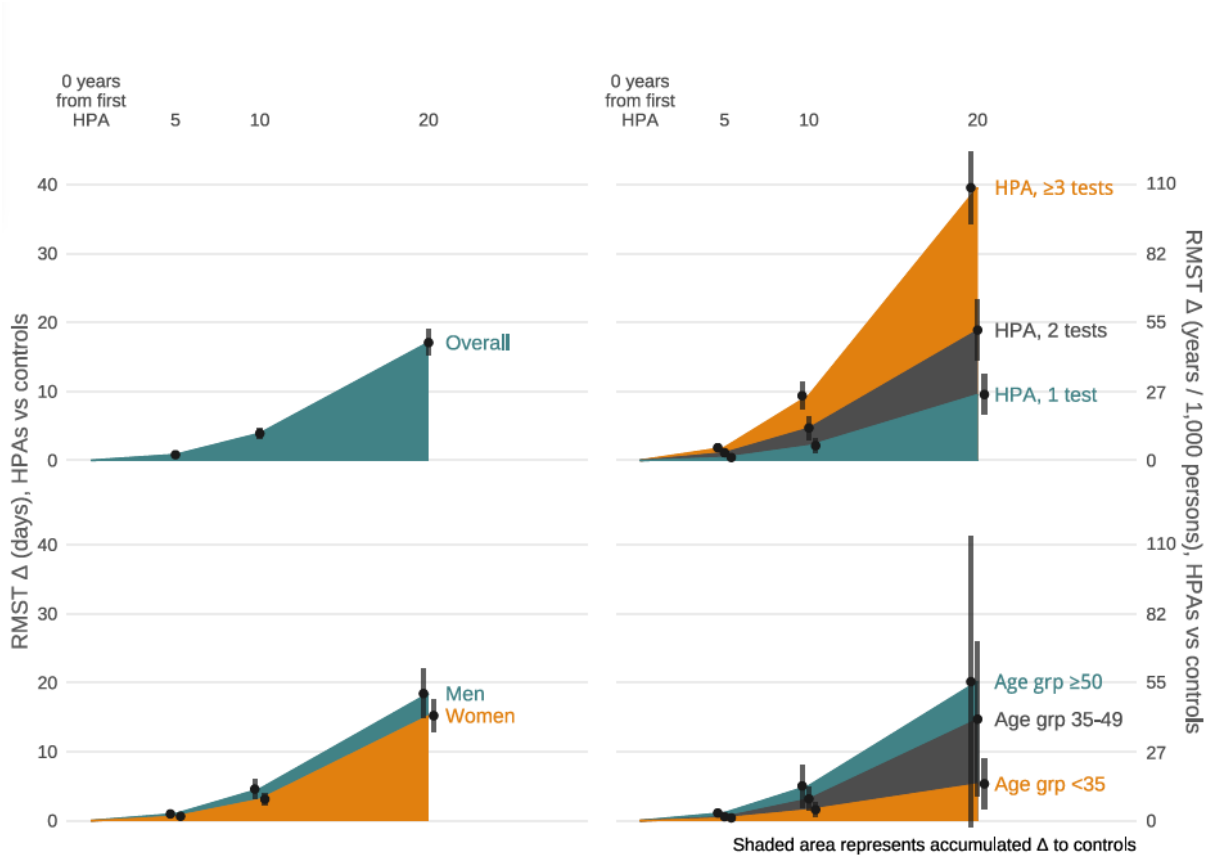

Figure S4. Restricted mean survival time differences between HPA participants and their matched controls at 5-, 10-, and 20-years follow-up. Area plots showing cumulative survival time differences (95% confidence intervals) for the overall population and stratified by number of HPAs within 5 years, sex, and age group. The color areas represent additional cardiovascular disease-free survival time after an HPA gained by HPA participants compared to controls, with the height of the area plot indicating mean differences in days per individual (on the left axis) and in years per 1,000 person (on the right axis) at each time point.

Table S1. Number of HPAs and cardiovascular disease: baseline-classified versus time-varying (immortal-time-corrected) estimates. Hazard ratios for incident CVD by number of HPAs completed within five years, among HPA participants (reference = 1 assessment), from Cox proportional-hazards models adjusted for the primary-model covariates (birth cohort, age, sex, comorbidity, prior CVD, and income). In the baseline-classified ("naïve") analysis each participant is assigned their final assessment count, fixed from baseline. In the time-varying analysis assessment count is modelled as a time-dependent exposure in a counting-process model: each participant contributes follow-up at the count actually held at each point in time, entering the two- or three-assessment state only on the date of the corresponding assessment, so that person-time before each assessment is attributed to the lower count. This removes immortal-time bias, the guaranteed ("immortal") survival a participant must accrue to reach a higher count. Correcting for immortal time does not remove confounding (e.g., by health-consciousness), the residual association is not interpreted causally.

Number of HPAs	Naïve HR (95% CI)	Time-varying HR (95% CI)
1 assessment	1.00 (reference)	1.00 (reference)
2 assessments	0.96 (0.90–1.01)	0.99 (0.93–1.05)
≥3 assessments	0.86 (0.79–0.94)*	0.92 (0.84–1.01)

* p = 0.001; other comparisons p ≥ 0.09.

Table S2. One-year landmark analysis (delayed entry), excluding instead of adjusting for prior CVD, only including ages 18-65 at first HPA. All analyses based on the primary model (M3).

Sensitivity analysis	Population (n)	Events	Hazard ratio (95% CI)
M3, primary (cause-specific; death censored)	2,091,415	38,449	0.895 (0.871–0.920)
M3, competing risks (Fine–Gray; death competing)	2,091,415	38,449	0.900 (0.879–0.922)
M3, one-year landmark (delayed entry)	2,076,720	36,252	0.896 (0.871–0.921)
M3, excluding prior CVD	2,000,200	31,946	0.902 (0.876–0.928)
M3, ages 18–65 at first HPA	2,082,625	37,759	0.897 (0.873–0.922)

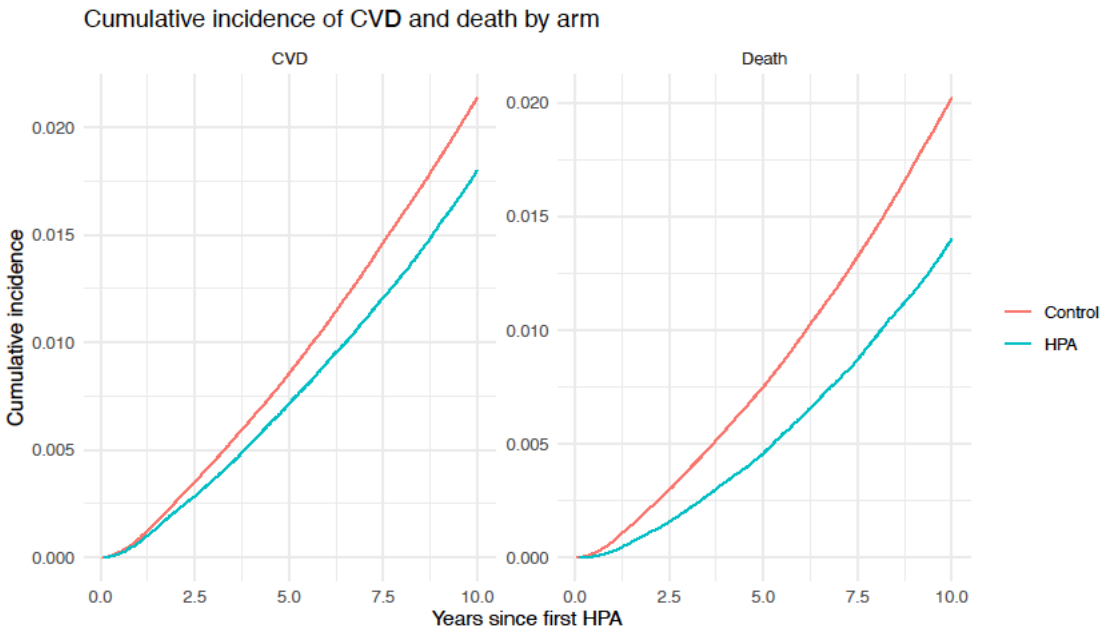

Figure S5. Cumulative incidence of cardiovascular disease and of death, by study arm. Cumulative incidence functions (CIFs) over 10 years for incident CVD and for death as a competing event, in the 1:4 propensity-score-matched cohort. CIFs were estimated with the Aalen–Johansen estimator, treating death as a competing risk for CVD. The CVD curves were similar between arms, consistent with a modest relative reduction. Death was less frequent among HPA participants; because participants self-selected into assessment and matching did not balance health behaviors, this difference likely reflects selection of a healthier population in addition to any true effect.