Employee Cardiometabolic Risk Following a Cluster-Randomized Workplace Intervention From the Work, Family and Health Network, 2009–2013

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Objectives. To examine whether workplace interventions to increase workplace flexibility and supervisor support and decrease work–family conflict can reduce cardiometabolic risk.

Methods. We randomly assigned employees from information technology (n = 555) and long-term care (n = 973) industries in the United States to the Work, Family and Health Network intervention or usual practice (we collected the data 2009–2013). We calculated a validated cardiometabolic risk score (CRS) based on resting blood pressure, HbA_{1c} (glycated hemoglobin), HDL (high-density lipoprotein) and total cholesterol, height and weight (body mass index), and tobacco consumption. We compared changes in baseline CRS to 12-month follow-up.

Results. There was no significant main effect on CRS associated with the intervention in either industry. However, significant interaction effects revealed that the intervention improved CRS at the 12-month follow-up among intervention participants in both industries with a higher baseline CRS. Age also moderated intervention effects: older employees had significantly larger reductions in CRS at 12 months than did younger employees.

Conclusions. The intervention benefited employee health by reducing CRS equivalent to 5 to 10 years of age-related changes for those with a higher baseline CRS and for older employees.

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Work is a key social determinant of health and well-being and provides many opportunities and resources as well as exposures to health risks. It is central in shaping inequalities in health. One of the key organizational conditions shaping workplace risk or protection is the ability to go to work without creating undue hardship and strain on family responsibilities and obligations, thus determining work–family conflict.[1] Work–family conflict is of growing concern in the United States, as the majority of women with younger children work outside the home and more workers need to care for older adults, including parents, partners, close friends, and family.[2,3] Dual wage–earning families are common, potentially increasing work demands for the entire family and reducing opportunities for home care. As a result, men and women increasingly experience major work and family responsibilities.

About 70% of US workers report some interference between work and nonwork.[4] Such interference is reported to degrade employee health, but few experimental approaches have been used and even fewer had strong disease biomarkers. Furthermore, recent workplace wellness program studies using strong randomized designs reported limited positive results,[5–7] including some improvements in health behaviors and intentions to change

behaviors but no differences in clinical or self-reported health outcomes[5–7]or medical or pharmaceutical spending or utilization.[7] In this light, it is important to consider workplace redesign practices that directly influence stressful workplace conditions.[8] This perspective is aligned with a social determinants of health approach in which the work itself—how it is organized and structured—shapes risks for health and well-being.

Rather than viewing the workplace as a venue for delivering health care or wellness benefits implicitly or explicitly asking workers to manage their stress as individuals, we see work and the structure of the workplace as a potential determinant of health.[9] Our framework focuses on reducing toxic environments, not on asking workers to adapt to risky working conditions. By using strong randomized experimental designs, such as what we used in this study, we can better assess causal impacts of changing workplace conditions on health, in this case cardiometabolic disease risk.

Previous systematic reviews[10–13] of observational studies have linked work stress to mortality and cardiovascular morbidity, and several studies from the Work, Family and Health Network (WFHN) have linked work–family conflict to health and cardiovascular risks.[14,15] A pilot supervisor support intervention was associated with decreases in blood pressure.[15] The WFHN intervention positively affected objectively measured sleep, recently recognized by the American Heart Association as important for heart health.[16] In systematic reviews and meta-analyses,[11,17] risks were found to be about 1.2 and somewhat higher (1.3) when issues of reverse causality were addressed. Selection into more stressful jobs related to preexisting poor health, low educational attainment, or other sources of disadvantage may still affect results.[15,18] Both confounding by other conditions and selection into stressful jobs reduces confidence in observational studies; experimental designs are needed for strong causal inference.

Biological mechanisms underlying the link between work stress and cardiovascular disease include coagulation, inflammation, and cardiovascular reactivity[13] as well as atherosclerosis and general metabolic syndromes,[19] but the strongest links between job strain and cardiovascular risk rest largely on prospective epidemiologic studies. However, as noted by Kivimäki and Kawachi, "The strongest evidence for causation derives from experimental manipulation of the exposure (work stress) to see whether it can affect outcomes of interest. Experimental evidence of this sort remains extremely sparse in the area of work stress."[19(p2)]

We provide experimental evidence that a workplace intervention designed to increase employee control over work time, train supervisors to support personal and family life, and reduce low-value work that leads to workers feeling overloaded can improve employees' cardiovascular health.

The WFHN designed a group-randomized experiment to intervene in work conditions to improve cardiovascular health, mental health, sleep, and workplace productivity.[20] We report on the primary aim related to cardiovascular risk in 2 different industries: the long-term care industry and an information technology (IT) industry. These 2 industries with their different wages and occupations provide an opportunity to test the intervention in distinct contexts.

The workplace intervention was designed to increase family-supportive supervisor behaviors and employee control over work time to decrease work–family conflict and improve health.[21,22] We hypothesized that the intervention would decrease employees' risk of developing cardiovascular disease, which was assessed by a validated cardiometabolic risk score (CRS).[23]

We tested a secondary hypothesis: that the effect of the intervention on cardiometabolic risk would be moderated by baseline cardiometabolic risk and age.[24] We reasoned that intervention effects would be more apparent for vulnerable employees with higher baseline cardiometabolic risk or who were older. Younger employees with lower baseline cardiometabolic risk would not benefit as much in terms of risk reduction although they may have benefited in terms of other outcomes, such as psychological distress.[25]

METHODS

The group-randomized experimental study design has been described previously[14,22,26] and registered, with our primary cardiovascular outcome the change in the modified Framingham risk score from baseline to 12 months follow-up (we collected data 2009–2013). We recruited participants from 2 companies with multiple work sites (long-term care facilities) or multiple work units (IT) in the United States. Employees were eligible to participate if they were at least 18 years old. Each participant provided written informed consent and completed a baseline preintervention survey with biomarker collection. Follow-up assessments included in-person interviews and biomarker collection. Our analytic sample for cardiovascular risk consisted of individuals with both baseline and 12-month postintervention follow-up data, of whom there were 973 in long-term care and 555 in IT, as specified in our registration with

ClinicalTrials.gov. Figure 1 is the CONSORT (consolidated standards of reporting trials) diagram, which shows each study step with its associated sample size. The unit of randomization was the worksite or work unit, not the individual.

We evaluated measurements at 2 time points: baseline (preintervention) and our major endpoint (the 12-month postintervention follow-up). Recruitment spanned September 2009 to July 2011. One company consisted largely of female, low-wage direct care workers and the other company consisted of male and female high- and moderate-salaried technical workers. We randomly assigned work units to either intervention or usual practice using an adaptive randomization technique[27] modified for each industry.[26] For long-term care, facilities or nursing homes (n = 30) were the units of randomization. For the IT industry, work units analogous to departments were the units of randomization (n = 56). All regular employees in IT groups were eligible to participate. In long-term care, direct care workers (e.g., registered nurses, certified nursing assistants) who worked for 22.5 hours or more per week on at least some day shifts were eligible to participate. We calculated power for estimated intervention effects on the modified Framingham score based on pilot work,[28] conservatively concluding that we had sufficient power to detect effects with 15 groups per condition and a minimum sample size of 20 employees per group.

Recruitment materials emphasized describing connections between employees' working conditions and health. Trained study site managers introduced the study to employees and coordinated project implementation. To minimize bias, using separate and blinded field interviewers, we obtained informed consent and collected data from employees in intervention and usual practice groups at baseline and follow-up. We collected baseline data approximately 1 month before intervention. We collected self-reported measures with a 60-minute computer-assisted personal interview at each time point. We describe biomarkers, including blood pressure measures, dried blood spots for HbA_{1c} and cholesterol, and weight and height measures for body mass index in the "Study Outcomes" section. Employees received up to $60 for completing data collection.

Study Oversight

A data safety and monitoring board reviewed the trial. Work, Family and Health steering committee members

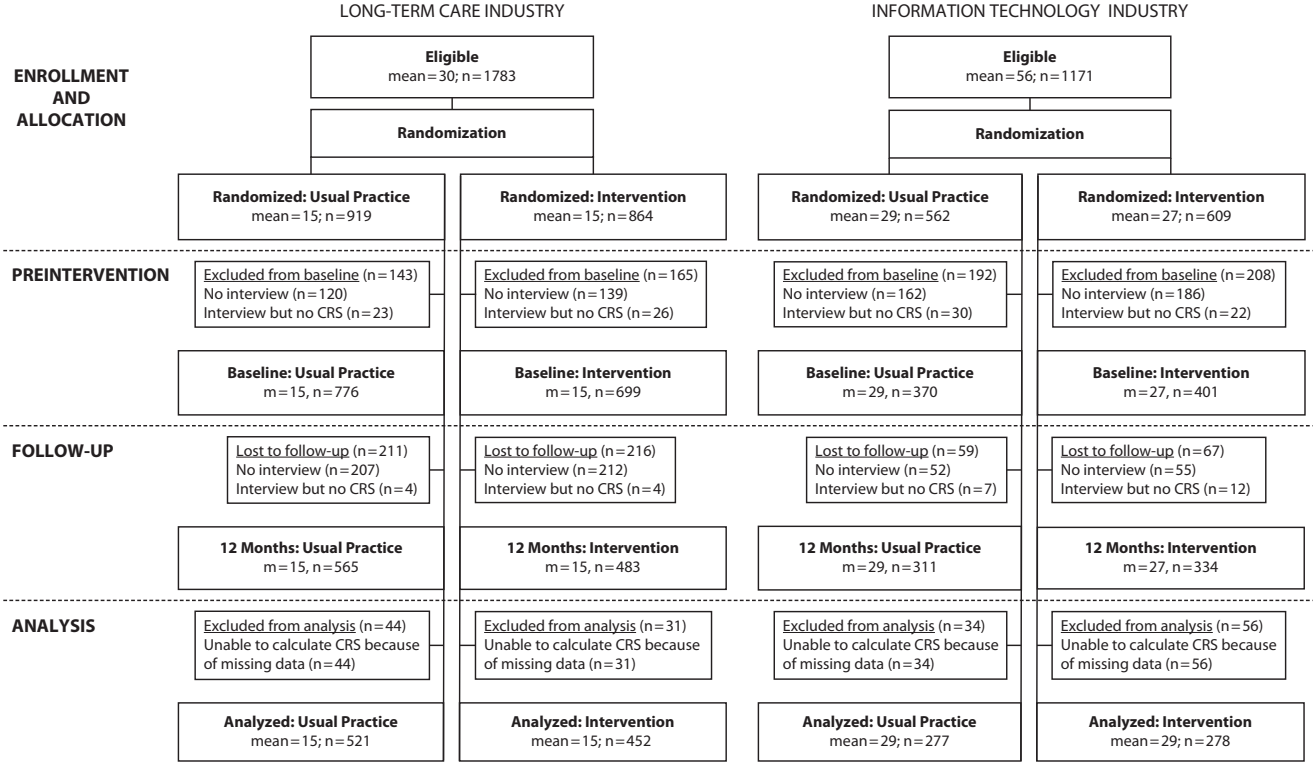

FIGURE 1— CONSORT Diagram: United States, 2009–2013

Note. CRS = cardiometabolic risk score; mean = mean cluster size.

who oversaw the trial can attest that the study was performed in accordance with protocols and the statistical analysis plan and vouch for accuracy and completeness of reported analyses.

Study Intervention

The intervention consisted of supervisor training focused on increasing supportive work–family behaviors and work redesign activities aimed to modify practices and interactions in workplaces between employees and their direct supervisors. The intervention was a structural and social change process designed specifically to increase employees' control over work time and supervisors' awareness and support of work–family balance; the intervention integrated pilot interventions that independently improved employee health.[29,30] Frontline managers participated in online and in-person training on strategies to demonstrate support for employees' personal and family lives while supporting employees' job performance. Employees and managers attended participatory training sessions where teams identified new work practices to increase employees' control over work time and to help reduce low-value tasks. Intervention activities unfolded over 12 weeks. We invited nonsupervisory employees to between 5 hours (in long-term care) and 7.5 hours (in IT) of training, and we invited managers to 4 additional hours of training.[21,22] Detailed training and support materials are available online (https://workfamilyhealthnetwork.org).

Study Outcomes

The primary outcome was CRS, a sex-stratified and validated score in the Framingham Offspring cohort to predict subsequent 10-year cardiovascular event risk.[23] We created the score using sex-specific algorithms based on 6 biomarkers:

1. systolic blood pressure (mm Hg),
2. body mass index (kg/m^2),
3. glycated hemoglobin (HbA_{1c}, %),
4. tobacco consumption (smoker vs nonsmoker),
5. HDL (high-density lipoprotein) cholesterol (mg/dl), and
6. total cholesterol (mg/dl).

We calculated scores for respondents who had both baseline and 12-month assessments. Higher scores indicate higher estimated risk (%) of developing cardiovascular disease 10 years later. We conducted study-specific equivalency analyses on dried blood spot biomarkers and included control values in shipments to the biomarker laboratory for this specific purpose. We used these serum equivalencies for dried blood spot measures.[31]

Statistical Analysis

Summaries of continuous variables are presented as means ±SDs for normally distributed data and as frequencies (percentages) for categorical variables. We compared baseline covariates between study groups by using the χ^2 test for categorical variables and the Student *t* test for continuous variables.[26]

We performed analyses on complete cases separately for each of the 2 industries, resulting in 4 models (an overall intervention effect model for each industry and a moderation analysis by baseline CRS for each industry). For all 4 models, we performed analyses at the participant level, using linear mixed effects models to estimate the mean between-group difference in 10-year cardiovascular risk from baseline to 12 months. All models included random effects for participants to account for temporal correlation and random effects for workgroups to account for clustering of participants in workgroups.

To assess the overall impact of the intervention on 10-year cardiovascular risk, we followed a difference-in-difference approach that included an indicator for time (with baseline set as the reference), an indicator for intervention group (with usual practice serving as the reference), and the interaction between time and intervention. The effect of the intervention was represented by a 2-way interaction model parameter that represented the difference in relative average difference in CRS between intervention individuals relative to usual practice individuals across time. In addition to these terms, for long-term care, we included a control for the number of employees in a cluster. For the IT cohort, we also included the number of employees in a cluster and categorical indicators of unit function (core or support), whether that unit's baseline data were collected before or after a major organizational change (a merger) was announced, and a categorical indicator of whether the unit was reorganized during the study period.

We intended these control variables to address potential structural differences between treatment and control groups that randomization might not have fully addressed. Our analysis suggests that other potential differences, such as race and gender, between treatment and control groups were slight, and replications of analyses with controls adjusting for any differences in those characteristics were consistent with the main analysis.

To test the a priori hypothesis that the effect of the intervention on 10-year cardiovascular risk would be moderated by baseline CRS risk, we adapted linear mixed models to include

a 3-way interaction for the moderating variable of interest (baseline CRS). The 3-way interaction parameter represented the moderating effect of baseline CRS risk on the difference in relative average difference in outcomes between intervention individuals relative to usual practice individuals across time after controlling for potential confounders. We assessed evidence of any differential intervention effects on the primary outcome with the use of a likelihood ratio test for the 3-way interaction term.

As a sensitivity analysis, we also conducted intent-to-treat with last observation carried forward for those with missing data at the 12-month follow-up. All statistical tests were 2-sided, and statistical significance was defined at $P < .05$. We performed statistical analyses in SAS version 9.3 (SAS Institute, Cary, NC).

RESULTS

Table 1 shows the characteristics of participants by industry and intervention conditions. Overall, treatment and control groups were well balanced in terms of sociodemographic and health characteristics for both industries. In long-term care, control group respondents were slightly older, and a larger proportion were Hispanic and foreign born compared with respondents in the intervention group. In the IT cohort, a larger proportion of control group respondents were Asian/Pacific Islander (difference = 19) and foreign-born; the number of participants in these categories was small and did not influence results. Attrition rate did not differ by treatment group in either industry (Figure 1). Participants lost to follow-up were similar in treatment and control groups in the long-term care sample. In IT, participants lost to follow-up were slightly younger (no difference by treatment group) and slightly less likely to be non-Hispanic White and native-born (data available on request).

We estimated the amount of the intervention received by employee intervention session attendance. In IT, intervention sessions were attended by 75% of the analytic sample; 9% of employees (n = 25) attended fewer than half of the sessions, and 4% (n = 10) attended no sessions. In long-term care, intervention sessions were attended by 67% of the analytic sample; 29% of employees (n = 122) attended fewer than half of the sessions, and 9% (n = 38) attended no sessions.

Intervention Effects on Cardiometabolic Risk Score

Table 2 presents results from the evaluation of the overall intervention effects on CRS by industry. We present CRS by intervention and usual practice for each industry at baseline and 12 months with 95% confidence intervals (CIs). Although CRS in both usual practice groups at 12 months were slightly higher than at baseline, and CRS in intervention groups were slightly lower, the CIs overlap. We observed no statistically significant intervention main effect in either industry (reduction in CRS in the long-term care industry cohort: B = −0.27; 95% CI = –0.63, 0.08; in the IT cohort: B = −0.21; 95% CI = –0.62, 0.20).

Effects by Baseline Cardiometabolic Risk Score

In prespecified subgroup analyses, we compared intervention impacts on workers who had higher baseline CRS. Baseline CRS moderated intervention effects (Table 2). For both industries, intervention employees who had higher CRS at baseline exhibited significant decreases in CRS at 12-month follow-up (Figure 2). Specifically, the intervention decreased CRS for long-term care workers (B = −0.08; 95% CI = −0.13, −0.04) and for IT workers (B = −0.07; 95% CI = −0.12, −0.01) whose baseline CRS was higher. To put the effect magnitude into perspective, we express these differences relative to age-related differences in risk. In supplementary analyses, magnitudes of effects for the average worker (−0.31 for long-term care and −0.18 for IT) were comparable with age-related increases in CRS equivalent to 5.5 years in the IT cohort and 10.3 years in the long-term care cohort.

Intervention Effects by Age

In supplementary analyses, we found that these effects were more apparent in older employees. Figure A (available as a supplement to the online version of this article at http://www.ajph.org) shows that older employees (≥ 45 years) who had higher CRS at baseline were more likely to decrease CRS at 12 months than were younger employees (< 45 years) who also had higher CRS at baseline. Note that older employees had higher CRS at baseline on average than younger employees.

In the intention-to-treat analyses shown in Table A (available as a supplement to the online version of this article at http://www.ajph.org), results were qualitatively similar to those of the complete case analyses. In particular, main effect terms remained nonsignificant, and the CRS moderating effects were similar in magnitude and statistical significance (IT: $P = .01$; long-term care: $P < .001$).

TABLE 1— Selected Characteristics of Study Participants by Industry: United States, 2009–2013

	Long-Term Care			IT		
	Intervention (n = 452), Mean ±SD or No. (%)	Control (n = 521), Mean ±SD or No. (%)	Δ	Intervention (n = 278), Mean ±SD or No. (%)	Control (n = 277), Mean ±SD or No. (%)	Δ
Baseline sociodemographics						
Age, y	37.7 ± 12.4	39.6 ± 12.1	*	46.6 ± 8.9	46.1 ± 8.6	
Male sex	34 (7.5)	47 (9.0)		160 (57.6)	173 (62.5)	
Married/partnered	277 (61.3)	341 (65.5)		224 (80.6)	222 (80.1)	
Caregiver	140 (31.0)	149 (28.6)		66 (23.7)	65 (23.5)	
Race/ethnicity						
White	291 (67.2)	315 (61.6)		195 (70.4)	185 (66.8)	
Black	70 (16.2)	62 (12.1)		11 (4.0)	5 (1.8)	
Hispanic	51 (11.8)	91 (17.8)	*	23 (8.3)	18 (6.5)	
Asian/Pacific Islander	. . .	. . .	. . .	32 (11.6)	51 (18.4)	*
Asian, other	. . .	. . .	. . .	14 (5.1)	15 (5.4)	
Other	21 (4.9)	43 (8.4)	*	2 (0.7)	3 (1.1)	
Foreign-born	121 (26.8)	158 (30.3)		59 (21.2)	82 (29.6)	*
Postsecondary education, No. (%)	259 (57.3)	317 (70.0)		266 (95.7)	270 (97.5)	
Children ≤ 18 y in household	211 (46.7)	246 (47.2)		130 (46.8)	139 (50.2)	
Baseline health characteristics						
10-y CRS, %	7.4 ± 8.0	8.1 ± 7.9		10.3 ± 7.9	9.6 ± 7.0	
Systolic blood pressure, mm Hg	114.8 ± 12.3	114.8 ± 13.5		120.2 ± 13.2	117.8 ± 13.5	*
BMI, kg/m^2	29.5 ± 7.0	29.5 ± 6.9		28.7 ± 5.4	28.0 ± 5.7	
HbA_{1c}, %	5.5 ± 0.6	5.5 ± 0.6		5.7 ± 0.6	5.6 ± 0.5	
Smoker	127 (28.1)	154 (29.6)		18 (6.5)	17 (6.1)	
HDL cholesterol, mg/dl	64.1 ± 5.8	63.1 ± 5.3	*	65.3 ± 5.4	64.4 ± 4.7	*
Total cholesterol, mg/dl	191.2 ± 28.9	190.8 ± 28.4		194.3 ± 27.2	190.5 ± 23.0	
12-mo health characteristics						
10-y CRS, %	7.3 ± 7.8	8.3 ± 8.4		10.2 ± 7.8	9.7 ± 7.2	
Systolic blood pressure, mm Hg	111.5 ± 12.0	112.3 ± 13.9		116.8 ± 12.5	115.9 ± 14.0	
BMI, kg/m^2	29.5 ± 6.8	29.8 ± 6.8		28.7 ± 5.6	27.9 ± 5.7	
HbA_{1c}, %	5.4 ± 0.8	5.5 ± 0.7		5.6 ± 0.6	5.6 ± 0.5	
Smoker	109 (24.1)	146 (28.0)		18 (6.5)	15 (5.4)	
HDL cholesterol, mg/dl	62.4 ± 6.1	61.9 ± 5.8		61.3 ± 5.2	61.4 ± 5.5	
Total cholesterol, mg/dl	197.4 ± 28.8	199.5 ± 28.0		201.2 ± 27.7	202.4 ± 23.9	

Note. BMI = body mass index (weight in kilograms divided by square of height in meters); CRS = cardiometabolic risk score; HDL = high-density lipoprotein; IT = information technology industry. Significance (Δ) is from the Mantel-Haenzel χ^2 test if dichotomous and from the *t* test using pooled or Satterthwaite variances, as appropriate. Race/ethnicity was self-reported. Actual sample sizes differ slightly for baseline sociodemographics: n = 944–973 for long-term care and 554–555 for IT.

*Statistically significant ($P < .05$) within industry differences.

DISCUSSION

The Work, Family and Health Study is among the first studies to use a fully randomized and intent-to-treat design to evaluate whether changing workplace conditions will affect employees' cardiovascular risk. Our findings in 2 different industries suggest common effects. Although there were no main effects of the workplace intervention in either industry for CRS, we did observe an intervention effect among those with higher baseline CRS (Figure 2). Intervention effects for those with elevated baseline CRS were on the order of the effects of lower age-related CRS of 5 to 10 years. Among those with a lower

TABLE 2— Multilevel Intervention Effects on Estimated 10-Year CRS by Industry: United States, 2009–2013

	CRS, Mean (95% CI) or B (95% CI)	
	Long-Term Care	IT
CRS: by group and time		
Usual practice, baseline	8.12 (7.25, 8.99)	9.99 (8.57, 11.41)
Usual practice, 12 mo	8.30 (7.43, 9.18)	10.09 (8.68, 11.51)
Intervention, baseline	7.36 (6.45, 8.28)	9.87 (8.29, 11.45)
Intervention, 12 mo	7.27 (6.36, 8.19)	9.77 (8.19, 11.35)
Overall: difference-in-difference models		
Main effect: time (ref = baseline)	0.18 (−0.06, 0.43)	0.10 (−0.18, 0.39)
Main effect: intervention (ref = usual practice)	−0.76 (−2.08, 0.56)	−0.12 (−2.44, 2.21)
2-way interaction: intervention × 12 mo	−0.27 (−0.63, 0.08)	−0.21 (−0.62, 0.20)
Moderation analysis: difference-in-difference models by baseline CRS		
Main effect: time (ref = baseline)	0.18 (−0.06, 0.42)	0.10 (−0.18, 0.39)
Main effect: intervention (ref = usual practice)	0.01 (−0.29, 0.31)	0.09 (−0.29, 0.48)
Main effect: baseline CRS	1.00 (0.98, 1.02)	1.00 (0.97, 1.03)
2-way interaction (12 mo × baseline CRS)	0.00 (−0.03, 0.03)	−0.01 (−0.05, 0.03)
2-way interaction (intervention × baseline CRS)	0.00 (−0.03, 0.03)	−0.00 (−0.04, 0.04)
2-way interaction (intervention × 12 mo)	−0.31 (−0.66, 0.05)	−0.18 (−0.58, 0.22)
3-way interaction (intervention × 12 mo × baseline CRS)	−0.08 (−0.13, −0.04)	−0.07 (−0.12, −0.01)

Note. CI = confidence interval; CRS = cardiometabolic risk score; ICC = intracluster correlation coefficients; IT = information technology industry. Intervention effects on estimated 10-y CRS by industry were calculated using multilevel models, controlling for number of employees in cluster for long-term care and controlling for whether cluster was assigned rather than randomized, number of employees in cluster, the function (core or support) used for randomization, and whether the merger had been announced at the time of the study for IT. Moderation analyses include additional 3-way interactions between time, intervention, and baseline CRS. The sample size was 1946 (973 per time point) for long-term care and 1110 (555 per time point) for IT. Negative values indicate a decrease in CRS. Lastly, ICCs of participants in workgroups were estimated for all 4 models and are reported here (overall: long-term care ICC = 0.016, IT ICC = 0.064; moderation analysis: long-term care ICC = 0.009, IT ICC < 0.001).

baseline CRS (e.g., younger workers), there may have been floor effects, indicating that it would be very challenging to further reduce the CRS. The strong effect of age on the increase in the 10-year hard cardiovascular disease event is well established.[14,32] Thus, the results indicate that the workplace intervention reduced increases in CRS in more vulnerable workers.

Strengths and Limitations

There are a number of issues that may have affected results. First, although we conducted an intention-to-treat analysis including all those with follow-up data, we included employees who completed the baseline survey and either left the workplace or refused to participate in follow-up using values with last observation carried forward, which yielded consistent moderated intervention effects regardless of analytic approach. Our analysis of attritors suggests that they were not differentially lost to follow-up by treatment assignment. Second, as with all intention-to-treat analyses, we included those who did not participate in intervention sessions or did not receive enough intervention in analyses. When only a fraction of employees and supervisors fully participated, this served to dilute any effects.

The use of a summary cardiometabolic score as a primary outcome has both advantages and disadvantages. The advantages are embedded in additive impacts of small changes in risk across each risk factor. As in the Framingham risk factor score, the summary score is a potent and reliable indicator of future cardiometabolic morbidity and mortality. As with the original score, our score is designed to reflect age- and sex-specific risks. The disadvantage of such a summary score is that it does not enable one to see whether selective risk factors responded differentially to the intervention. A major strength of our study was that interviewers were blind to intervention status, and results relied primarily on biomarkers collected and without the researcher knowing which group respondents were in. We validated CRS outcome on the independent Framingham Offspring cohort.[23]

The pathways by which a social intervention such as ours might influence cardiometabolic risk are multiple and include psychosocial mechanisms, biological pathways, and health behaviors. Our intervention did not include a health behavior change component but focused on work conditions hypothesized to be determinants of health. In this model, more proximate factors may directly mediate relationships between a workplace intervention and health outcomes. In previous publications, we reported that the intervention influenced both distress and depressive symptoms and sleep.[33–35]

This study has limitations. The follow-up period was limited to 12 months, which may not be long enough to assess whether the effects of the intervention were sustained. CRS measures

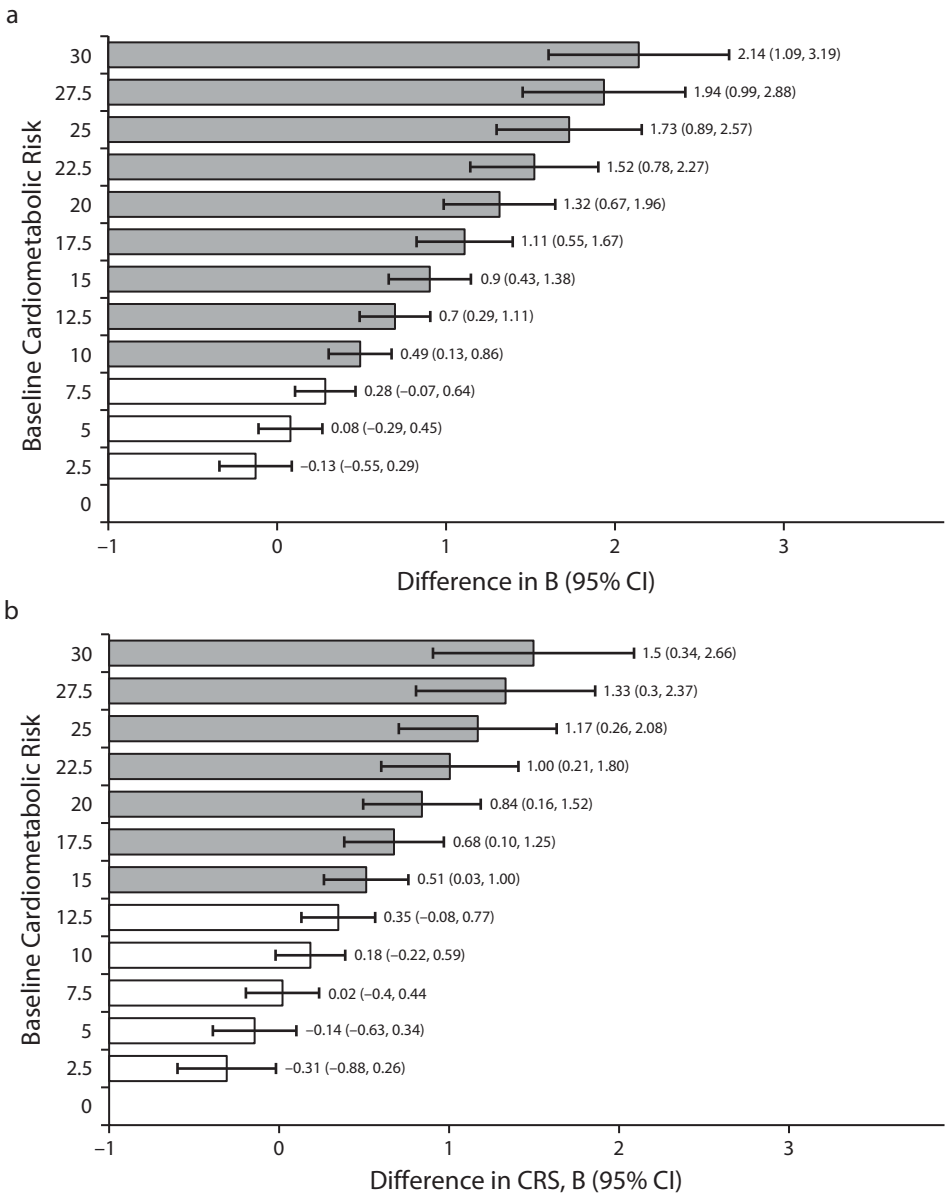

FIGURE 2— Workplace Intervention Effects on Estimated 10-Year Cardiometabolic Risk Score (CRS) for (a) Long-Term Care Industry Employees and (b) IT Industry Employees: United States, 2009–2013

Note. CI = confidence interval; CRS = risk of 10-year cardiovascular disease event. Difference in CRS = intervention change minus usual practice change. Shading (darker bars) indicates estimates significantly different from 0 at $P < .05$. Intervention effects on estimated 10-year CRS by industry were calculated using multilevel models, controlling for number of employees in cluster for long-term care and controlling for whether cluster was assigned rather than randomized, number of employees in cluster, the function (core or support) used for randomization, and whether the merger had been announced at the time of the study for IT. The sample size was 1946 (973 per time point) for long-term care and 1110 (555 per time point) for IT. Differences in intervention effects by baseline CRS were statistically significant at $P < .05$. Estimates are shown in Table A (available as a supplement to the online version of this article at http://www.ajph.org).

did not include clinical evaluation or medical records. Although the study included employees from numerous locations in 2 industries, future studies are needed to improve generalizability. Of note, we developed the sex-specific CRS algorithms[36] we used as the outcome with a cohort that was predominately non-Hispanic White. Given that at least 30% of our study participants were of races/ethnicities other than non-Hispanic White, there is a potential risk to validity. However, if there is bias or measurement error, the randomization balanced the proportion of race/ethnicity by intervention groups, and the findings should be relatively unaffected by this error. Nevertheless, future studies should

consider recalibrated measures of the CRS for other racial/ethnic groups.[37]

Conclusions

Few randomized trials have been conducted to assess whether changing the work environment can affect cardiovascular risk. Ours is among the first of the trials to do so. In light of recent findings reporting weak or null effects of workplace wellness programs,[7,38] it is important to identify work conditions per se that may affect employee health. Our goal was to change workplace conditions we hypothesized were influencing employee health rather than ask employees to adapt to stressful working conditions via programs that did not change the environment. Our findings suggest that older workers and those with higher cardiovascular risk will benefit from such interventions. We followed a social determinants of health framework reviewed in earlier work in *AJPH*,[8] and we emphasize the need to conduct well-designed experimental workplace redesign interventions aimed at improving health and well-being. Earlier work from the WFHN reported effects on psychological distress, sleep, and a number of safety and organizational behaviors. To our knowledge, this is the first to report the impact on cardiometabolic risk.

Our findings align with much recent work identifying health risks related to low schedule control, long work hours, and lean or just-in-time operations, especially for low- and middle-wage earners.[39] Flexible work policies and practices were increasingly seen during the COVID-19 pandemic as essential for maintaining worker health as well as the health of family and community members. Our findings support the direct impact of workplace organization on the health of workers. AJPH

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CONFLICTS OF INTEREST

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HUMAN PARTICIPANT PROTECTION

Study methods were approved by the Harvard T.H. Chan School of Public Health institutional review board (IRB) and the IRBs of other study centers and was conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent before randomization.

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