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Second-Year Results of an Obesity Prevention Program at The Dow Chemical Company

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Abstract

Objective—Evaluate innovative, evidence-based approaches to organizational/supportive environmental interventions aimed at reducing the prevalence of obesity among Dow employees after two years of implementation.

Methods—A quasi-experimental study design compared outcomes for two levels of intervention intensity to a control group. Propensity scores were used to weight baseline differences between intervention and control subjects. Difference-in-differences methods and multi-level modeling were used to control for individual and site-level confounders.

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Results—Intervention participants maintained their weight and BMI while control participants gained 1.3 pounds and increased their BMI values by 0.2 over two years. Significant differences in blood pressure and cholesterol values were observed when comparing intervention employees to controls. At higher intensity sites, improvements were more pronounced.

Conclusions—Environmental interventions at the workplace can support weight management and risk reduction after two years.

Keywords

worksite health promotion; weight management; obesity; risk factors; environmental interventions; workplace wellness

INTRODUCTION

To address the epidemic rise in obesity rates (1), many U.S. companies have introduced health promotion and disease prevention programs focused on reducing the prevalence of overweight and obesity among their workers.(2) Traditionally, employers have offered individualized behavior change programs and more recently some have complemented these with environmental interventions that support individual health improvement efforts.(3) Multi-component environmental interventions, defined as “strategies that involve changing the physical surroundings and social, economic, or organizational systems in order to promote individual behavior change,”(4) have not been well evaluated and there is limited knowledge about their ability to achieve long-term behavior change and reduce health risks in employed populations. Of the 47 worksite programs to control overweight and obesity that were reviewed by the CDC Task Force on Community Preventive Services, there were only four studies that examined policy and environmental changes in the worksite. (5)

Environmental interventions are based on a social-ecological model that encourages adoption of healthy behaviors through changes in routine activities. (6) Workplace environmental interventions include offering healthier food choices in cafeterias and vending machines; facilitating physical activity opportunities through promotion of staircase use, creation of marked walking trails, or installation of bike racks on company grounds; and changing company culture by, for example, establishing health improvement goals that align with the organization’s overall mission. (7)

Researchers and practitioners agree that leadership and management support is critical to the success of workplace health promotion programs, especially when aligning organizational and employee health objectives with interventions that modify the physical work environment. (8–12) Experts in the field postulate that interventions that blend individual educational and environmental strategies will produce greater effects than individual approaches alone.(13–19) However, evidence of effectiveness, in terms of increased levels of physical activity, improved eating habits, reduced weight, or reductions in other health risks such as cholesterol, blood pressure and blood glucose levels, has been inconsistent.(4,20,21) This may be due to the dearth of studies employing rigorous research designs, long enough follow-up periods, use of control and comparison groups, and an examination of a broad range of health outcomes. There is also little information on the effects of varying the dose of interventions, for example comparing moderate vs. more intense environmental interventions, on outcomes, and which elements of these environmental interventions achieve the best results.

This paper presents two-year results from a study evaluating the effects of worksite environmental interventions on changes in employee overweight and obesity rates and associated health risks. The study is one of seven funded by the National Heart Lung and Blood Institute (NHLBI) to investigate the health and economic impacts of health improvement

programs that emphasize environmental and social-ecological interventions on the prevention and management of obesity at the workplace.(3)

This is a follow-up analysis to a previous paper that presented interim (year-one) results from this study. (22) Readers are advised to review that manuscript, as well as other associated publications (3,23–26), to learn more about the background of the study, its research design, instrument development and validation, and a detailed description of the interventions employed.

In the year-one analysis, we found a modest but statistically significant treatment effect on weight (1.5 pounds) and body mass index (BMI) (0.2) when comparing employees at intervention (treatment) to control sites, largely because control subjects gained weight. However, no differences were observed in the prevalence or rates of overweight and obesity between treatment and control employees after one year of exposure to environmental interventions. For other health risk factors, intervention effects were noted. Compared to control subjects, intervention site employees had significantly greater decreases in systolic and diastolic blood pressure, no improvement total cholesterol, and an increase in blood glucose levels. We concluded that, in only one year, environmental changes at the workplace can achieve modest improvements in certain health risks.

In this paper, we extend the time horizon for observing the effects of these environmental interventions and present the results for a two-year cohort of employees, some of whom were exposed to environmental interventions and others not. In addition, we report on the differential effects of those exposed to what we termed moderate vs. high intensity environmental interventions.

METHODS

Objectives

With the above interim results as a backdrop, we sought to determine whether two years of exposure to environmental interventions would achieve more pronounced and long-standing changes in employees' health risks and behaviors. Thus, our primary aim was to test whether employees at worksites that implemented environmental interventions, in addition to standard individually-oriented programs, would achieve greater reductions in weight, BMI, and the prevalence of overweight and obesity. We also examined other biometric and behavioral risks typically measured in health promotion programs including blood pressure, total cholesterol, blood glucose, nutrition, physical activity, tobacco use, stress, and alcohol consumption.

A secondary aim was to evaluate the differential effects of intervention dose, or intensity, comparing changes in each of the outcomes for employees at high-intensity and moderate-intensity sites to those of employees at control sites. High-intensity sites provided a combination of individual, environmental, and management commitment programs; moderate-intensity sites provided individual and environmental programs; and control sites only provided individual programs. These are described in further detail below.

Setting

Study participants were employed by The Dow Chemical Company (hereinafter referred to as "Dow"). Dow provides a broad range of products and services to people in 160 countries, including fresh water supplies, food products, pharmaceuticals, paints, packaging, and personal care items. Fifty-four percent of Dow's U.S. employees are laborers, clerical staff, or technical workers. The remaining workers are professionals or managers (44%) or are in sales (2%). Most (75%) of Dow's employees are male, 82% are white, and their average age is 43.

Twelve Dow sites were recruited for the study: nine intervention worksites in Texas (N=8) and Louisiana (N=1), and three control sites in West Virginia (N=1), New Jersey (N=1), and Louisiana (N=1). Eight sites were manufacturing facilities; two focused on research, development, and administrative functions; and two housed manufacturing, as well as research and development, and administrative staff. Most of the sites were large (ranging in size from 57 – 5,000 acres) and operated multiple business units.

Since the interventions were directed at organizational and environmental changes, all of the employees at the study sites were designated as participants in the study. Before the study, Dow had extensive individually-focused employee health promotion programs in place at all the study sites, and these programs continued throughout the study period regardless of treatment or control site designation.

Study Design

Using a quasi-experimental design, we evaluated the effects of environmental interventions implemented at two levels of intensity (intervention sites) in comparison with standard Dow health promotion programs (control sites). Dow's management wanted to assign all 12 sites to the treatment condition but was convinced by study researchers to withhold environmental interventions for two years at control sites so that treatment and comparison site data could be analyzed to determine intervention effects. Thus, three sites were selected as comparison sites by Dow's leadership and site leaders were instructed not to introduce new environmental interventions for two years, although individually-oriented programs were allowed to continue.

Dow's leadership selected as their control sites locations that were not planning any large-scale health promotion initiatives in the near term that would potentially interfere with the research, and that had basically the same business profile and core health promotion programs at baseline as the interventions sites. This was done to ensure that Dow's core programming would continue to be implemented consistently across all study sites -- intervention and control. Dow considered number of health promotion staff at the sites and their capabilities, site leadership support for health promotion, and any planned site initiatives that might impede implementation. Finally, Dow wanted to make sure that the sample sizes for control sites were adequate.

The nine remaining sites were matched on size and other relevant measures and then randomly assigned to moderate or intense intervention conditions based on a coin flip.

Employees at all study sites were encouraged to participate in the health risk assessment (HRA) and biometric screening programs, although no financial incentives were offered for their participation. The intervention was implemented over a two year period from April 1, 2006 to March 30, 2008.

Baseline data (collected in the first quarter of 2006 using an electronic HRA survey instrument) consisted of employee demographic information and self-reported health behaviors. All HRA participants were offered the opportunity to set up an appointment for biometric screenings but not all of them took advantage of the free service. Biometric screening measures (height, weight, blood pressure, total cholesterol, and blood glucose) were collected by health professionals shortly after HRAs were administered. Employees who participated in the biometric screenings were provided individual written feedback and counseling on their health risks. Follow-up HRA and biometric assessments were then collected during the first quarters of 2007 (year one) and 2008 (year two). The follow-up biometric assessments were only offered to participants who participated in the HRA in the following year (i.e., the researchers did not contact participants who had a biometric screening in 2006 but did not take the HRA in 2007).

Because this study was not a randomized design, we examined baseline demographics to determine whether there were significant differences between the intervention and control participants and controlled for these differences using a propensity weighting method. Methods used to adjust for baseline differences between treatment and control subjects are described later in this paper. When comparing overweight and obesity prevalence between subjects at intervention and control sites, there were no significant differences between groups at baseline. Baseline overweight and obesity prevalence rates at interventions sites were 36.1% and 26.6 %, respectively compared to prevalence rates for overweight and obesity at control sites which were 36.7% and 25.6%, respectively.

Interventions

All sites (intervention and control) offered Dow's standard health promotion and risk reduction programs throughout the study period. These individually-focused health promotion interventions included the following: dissemination of health education materials (newsletters, intranet site, posters, and home mailings); physical activity and weight management counseling; health assessments; on-line behavior change programs; reimbursement for participation in community-based weight management, tobacco cessation, or diabetes education programs; and preventive screening reimbursements.

Moderate-intensity interventions were comprised of two main components: 1) environmental prompts that encouraged employees to make healthy food choices and be physically active; and 2) point-of-choice messages to encourage healthy eating and physical activity, such as strategically placing signs in front of stairwells, vending/;machines, and cafeterias. Other parts of the intervention included modifying vending machine items and cafeteria menus, creating and marking walking paths at all sites, disseminating targeted messages that encouraged healthy eating and physical activity, making available an on-line weight tracking program, offering pedometers to workers, establishing wellness ambassadors at local departments, and developing an employee recognition program for those adopting or encouraging others to adopt healthy lifestyles.

High-intensity treatment sites received all of the above interventions and added elements designed to more directly influence organizational culture and leadership commitment to employee health. At these sites, interventions included: 1) setting health objectives as a component of the sites' management goals, 2) providing management training on health-related topics, 3) compiling and sharing feedback reports to site and senior leaders at corporate headquarters on the sites' achievement of certain program participation targets, and 4) providing additional support and training to the wellness ambassadors. These activities were designed to encourage worksites to explicitly include employee health as an important business objective, and to hold site leadership accountable for employees' engagement in health promotion programs.

All study procedures were reviewed by Institutional Review Boards at Cornell and Emory Universities, Dow's Health Services Review Board, and the NHLBI Data Safety and Monitoring Board.

Outcome Variables

Biometric data were collected using standardized protocols developed by Dow Health Services. Behavioral risk data were collected using standardized instruments developed by the research organizations participating in the NHLBI studies.(3) HRAs were administered online using Dow's established Intranet survey vendor, Valtera Inc.

Biometric measures included height, weight, BMI, total cholesterol, blood pressure (systolic and diastolic), and blood glucose. Blood pressure, cholesterol and blood glucose values were analyzed as continuous variables and were also categorically dichotomized as high vs. low risk based on standard clinical definitions of high risk. BMI was analyzed as both a continuous and categorical variable, i.e., normal (not at risk/low risk, BMI = 18.0 – 24.9), overweight (moderate risk, BMI = 25.0 – 29.9) or obese (high risk, BMI = 30.0+). BMI was calculated from the height and weight measurements collected from participants. Weight was analyzed only as a continuous variable.

During the course of the study, we found that some individuals' height changed from baseline to follow up. While we hypothesized this was due to employees removing their shoes during one measurement and not the other (many of Dow's blue-collar employees wear heavy, durable boots for protection), we needed to control for this difference. Since it was unclear which height was the most accurate (from the first or second assessment), we used the first measure of height and eliminated any participants (N=11) whose height changed by more than six inches from baseline.

Behavioral health risk outcomes, dichotomized as high vs. low risk, were scored using several HRA questions and included indicators for poor nutrition, lack of physical activity, tobacco use, high alcohol use, and high stress. Definitions of health risks for all outcome variables are presented in Table 1.

Statistical Methods

Prior to conducting the comparative analyses, propensity score weights were applied to equalize baseline differences between intervention and control site employees.(27) The propensity score weights were based on the predicted probability of being employed at the intervention sites. Using logistic regression, we modeled the probability of working at an intervention site based on the employee's age, gender, ethnicity, wage status (salaried or hourly), work status (type of job), education, and health risk status (using the Charlson Comorbidity Index [CCI] (28) and the Psychiatric Diagnostic Group [PDG] severity indices). The propensity score weights used in the analysis were the inverse of the predicted probability of being employed at an intervention site (i.e., 1/predicted probability).

To control for employee differences when comparing the results from intense, moderate and control sites, a different set of propensity score weights was created using a multinomial logistic regression model for the same predictor variables used to adjust for intervention and control groups. The model predicted the conditional probability of receiving a particular level of intervention, i.e., being at the intense, moderate or control sites. The propensity weight applied to each of the three study arms was the inverse of the predicted probability of being employed at an intense, moderate or control site.(27)

We first examined the changes in biometric and behavioral risk factors over the two-year period by comparing outcomes for intervention sites (moderate and intense combined) vs. control sites, and then for intense and moderate sites separately compared to control sites. To analyze within group changes in risk factors over time (i.e., whether employees at a given site improved their risks over time), paired t-tests were used for continuous variables (i.e., weight, BMI, blood pressure, and cholesterol) and McNemar chi-square tests were used for categorical variables to compare changes in the proportion of employees at high vs. low risk for the health behaviors of interest.

Difference-in-differences (DID) methods were then used to compare between group changes in outcomes over time (Time 3 minus Time 1), i.e., whether changes in risks were more pronounced at intervention sites compared to control sites. The DID analysis also allowed us

to control for baseline values that may be due to long-standing differences in the demographic and health risk profile of employees at any given site. All DID analyses were adjusted using the propensity score weights described previously.

Finally, we needed to control for the variability across workplaces caused by outside forces other than the intervention. At some sites, leaders and program champions were more aggressive in implementing programs than at others. Size of the site influenced the degree to which interventions could be put in place since some sites were quite small (about 100 employees) while others quite large (over 4,000 employees). Some sites had cafeterias while others did not, and some offered fitness facilities or access to community facilities and others did not. In some sites, leadership was relatively stable, and in others leaders often changed. In our study, some sites experienced layoffs during the course of the study, were slated for closing, or were sold off to another company.

To control for the effects of being at a worksite that received the intervention and the likely correlation of measures among employees within the worksite, we applied statistical methods widely applied when conducting a clustered randomized trial. (29) The worksite's influence on outcomes was evaluated by including a site-level variable in the predictive models, using either a fixed effect in the model for categorical (binomial) outcomes or a random effect in the model for continuous outcomes.

The analysis exploring site level effects on outcomes was conducted alongside the main analysis that only considered intervention effects on individual employees, without regard to site-specific influences. The two analyses were performed to address the debate among researchers regarding the need to control for site-level effects when sites, rather than individuals, are randomized into treatment and control conditions. Thus, results are presented with and without a site-level adjustment. Binomial outcomes were modeled using the SAS GLIMMIX procedure, and the continuous outcomes were modeled using the SAS MIXED procedure. All statistical analyses were conducted using the SAS 9.1 software package.

Missing Value Calculation

Study attrition in analysis of biometric data was controlled for using a non-response weighting procedure.(30–32) A logistic regression model was constructed to predict the probability of not participating in a follow-up assessment for employees contributing baseline data. The model based its predictions on the employee's age, gender, CCI score, education, work status, and assignment to an intervention or control condition. A non-response weight was calculated as the inverse of the predicted probability of not having a missing response (i.e., 1/predicted probability). The non-response weight was further multiplied by the intervention group propensity score as described above.

Additionally, a mean-based imputation procedure was used to account for missing biometric data for the cohort group of employees (missing data were reported for 0.0%–1.7% of participants for each outcome variable). This involved imputing missing values based on the mean value of the variable taken from the control group. For example, if a participant's BMI was missing at follow-up, the missing value was replaced with the "average BMI" of subjects in the control sites at follow up. Outcomes with missing data were then re-calculated based on these imputed values.

Results presented below are for the main analyses, not accounting for missing data. Overall, our findings were unaffected by missing data and so those alternative results are not shown in this paper and are available upon request.

RESULTS

Participation

There were 10,281 employees (8,013 at intervention sites and 2,268 at control sites) who were eligible to participate in the study. Our target goal was to recruit 6,000 employees from this pool of eligible employees (i.e., 60% participation rate). At Time 1, 5,124 employees participated in the HRA (49.8% participation rate). Of the Time 1 HRA participants, 3,504 also enrolled for the biometric screenings (68.4% of HRA participants). The final cohort of HRA participants consisted of 2,431 employees who participation in both Time 1 and Time3, of which 1,521 also provided biometric data. Additional information regarding participation in the various treatment arms is displayed in Figure 1.

Comparisons between Intervention and Control Groups

Table 2 shows the baseline demographic and health status comparisons between intervention and control group employees before and after propensity score adjustment. Before adjustment, intervention group subjects were younger, had a higher proportion of minorities, and were more educated. Intervention group employees also consisted of more hourly-wage employees, and were more likely to be operatives, laborers, and service workers. Gender and health status were similar for the intervention and control groups even before adjustment.

After propensity score adjustment, all differences between intervention and control groups were no longer statistically significant showing that the propensity score weighting process was successful. All subsequent analyses comparing intervention and control subjects used propensity score-weighted groups.

Changes in Weight and BMI

Table 3 displays the propensity score adjusted changes in employee weight and BMI for intervention and control group employees. As shown, average weight and BMI was unchanged at the intervention sites but increased significantly at the control sites ($p<.01$). However, the proportion of overweight employees increased significantly at the intervention sites ($p<.01$) and the proportion of obese employees decreased significantly at the control sites ($p<.01$).

In the DID analysis presented in Table 4, a net 1.6 pound difference between intervention and control group employees was observed in favor of the intervention group ($p<.01$). The difference between groups was not due to intervention group employees losing weight, but rather due to control group employees gaining weight by an average of 1.3 pounds. Similarly, a 0.3 differential in BMI between intervention and control groups occurred because control group subjects increased their BMI significantly without a corresponding decrease in BMI for intervention group employees. These results were upheld even after controlling for autocorrelation among employees within site (i.e., controlling for site effects). However, no differences were observed for changes in overweight and obesity prevalence when comparing treatment and control subjects.

Results for the three-group comparisons are presented in Tables 5 and 6. The high and moderate-intensity groups maintained their weight and BMI, while the control group employees gained an average of 1.3 pounds ($p<.01$) and their BMI increased an average of 0.2 points ($p<.01$). While the net difference in average weight between the high-intensity and control groups was significant at 1.5 pounds ($p<.05$), the net difference between the moderate-intensity and control group subjects' weight of 1.3 pounds was not significant. After controlling for site effects, the net difference in average weight and BMI was significant ($p<.05$) for both the high and moderate-intensity employees compared to controls. No significant impact on

rates of overweight and obesity was found for either the moderate or intense group subjects, with and without controlling for site effects.

Changes in Blood Pressure, Total Cholesterol, and Blood Glucose

Examining changes in other biometric values, intervention group subjects experienced greater net improvements than control group subjects in blood pressure (systolic and diastolic) ($p<.01$) and total cholesterol ($p<.05$). Blood glucose levels increased for both intervention and control subjects and the net differences between groups were not significant. After controlling for site effects, the significance levels for all these findings remained unchanged. In terms of high risk prevalence, the only significant net reductions between intervention and control groups were found in blood pressure, but these differences were no longer significant after controlling for site effects. Results are presented in Tables 7 and 8.

Comparing outcomes for these biometric values by intensity of treatment, effects were generally more pronounced at the high-intensity sites when compared to control sites. As shown in Tables 9 and 10, both systolic blood pressure and cholesterol levels were reduced to a greater extent at high-intensity sites than moderate-intensity sites compared to control sites. We observed an impact by treatment intensity where the high intensity sites compared to control sites showed a significant net difference in cholesterol ($p<.05$), whereas the net difference between moderate-intensity sites and controls was non-significant. In terms of risk prevalence, we found a treatment intensity impact in favor of the high intensity sites for blood pressure when compared to control sites ($p<.01$). Most of these results were supported after controlling for site effects except for comparisons of high-intensity vs. control sites for high blood pressure risk which became non-significant, and moderate-intensity vs. control comparisons for diastolic blood pressure which became significant ($p<.05$).

Changes in Behavioral Risk Factors

Within treatment conditions, significant improvements were noted in nutrition and physical activity ($p <.01$), and improvement in stress levels approached significance ($p = .054$) for the intervention group. For the control group, significant improvements were observed for nutrition, tobacco use, and stress ($p<.01$). Risks for poor nutrition and physical activity showed a net improvement of 6.4% ($p<.01$) and 3.2% ($p<.05$), respectively, for the intervention group in comparison to the control group. However, after controlling for site effects, these differences were no longer statistically significant. No intervention effects were found for tobacco use, high alcohol use or high stress.

Comparing outcomes for intervention vs. control groups by level of intervention intensity, both moderate- and high-intensity group employees achieved significant net risk reductions in poor nutrition compared to control sites ($p<.05$ and $p<.01$, respectively), although these were not significant after controlling for site effects. An intensity impact was found for poor physical activity in favor of the high-intensity sites, where the high-intensity sites had a significant net risk reduction ($p<.05$) compared to controls, while no such significant net difference was found for the moderate-intensity sites. Controlling for site effects yielded non-significant net differences for both of the intervention arms compared to controls for poor physical activity. No intervention effects were found on tobacco use, high alcohol use, and stress.

DISCUSSION

Our study sought to determine whether environmental and social-ecological interventions introduced at the workplace, alongside individually-oriented interventions, would produce additional health benefits when compared to individually-oriented programs alone. Over a two-year period, a cohort of Dow employees were exposed to two levels of environmental and

social-ecological interventions at the workplace, in addition to individual interventions, and their experience was compared to a control group of employees who only received individual interventions.

Nine worksites received environmental interventions and three served as controls. Of the nine intervention sites, four received what was termed moderate interventions, primarily focused on providing greater access to, and information about, healthy eating and physical activity. Five high-intensity sites built upon the moderate-intensity interventions by seeking to increase local leadership engagement in health promotion and weight reduction initiatives. This was done by setting site goals related to participation and engagement in programs, aligning department and site goals, offering more leadership training and feedback, and putting in place reporting mechanisms related to program accomplishments for senior leadership. On-line weight tracking programs, where employees could monitor their weight gains or losses, and enhanced employee recognition programs, were also put in place at all intervention sites. The three control sites continued to deliver individually-oriented health promotion programs largely consisting of counseling and coaching services directed at employees prepared to make behavior change.

Over the course of the two-year study, intervention site employees maintained their baseline weight while control site employees gained weight an average of 1.3 pounds and 0.2 BMI points. However, there was no intervention effect on the overall prevalence of overweight and obesity for intervention group employees. These findings were consistent even after controlling for site effects.

By its nature and design, environmental intervention programs are more diffused than those that target high risk individuals directly. Thus, it is expected that effects on weight and BMI would be smaller than in individually focused interventions and that all employees, not just those at high risk, would be affected. While our results are not be considered clinically significant at the individual level, the incremental effect of these environmental interventions at intervention sites compared to individually-focused interventions alone at control sites is promising.

Previous research has shown that worksite health promotion programs achieve a modest impact on overweight and obesity and most of these programs are individually-focused. In a recent review of 47 worksite obesity management programs by the CDC Community Guide, (5) six individually-focused behavioral programs reduced workers' BMI by an average of 0.5 points, which is somewhat greater than the 0.3 BMI differential found between treatment and control site workers in this study. While the study effects found here are modest in the near term, if sustained, they can potentially translate to long-term clinical gains. Helping employees prevent age-related weight gain may be as important as supporting their weight loss efforts, given that increased adiposity and weight gain in mid-life impacts one's health at an older age.(33,34)

Participants at intervention sites also experienced a net improvement in their biometric values for blood pressure and total cholesterol compared to the control group. These results were upheld after adjusting for site-level effects. Intervention sites showed improved levels of blood pressure and total cholesterol while control sites showed increases or no changes in these biometric measures, respectively. In general, the net differences between intervention and control subjects for biometric values were greater at high-intensity than moderate-intensity sites, demonstrating an intervention dose effect. Despite these improvements in biometric values at the intervention sites, the significant reduction in the percent at risk for high blood pressure was no longer significant after controlling for site level effects. However, it is important to note that across measures (especially for blood pressure) and study arms, risk prevalence was fairly low to begin with and baseline measures were generally well within the

normal range. Thus a floor effect may be one explanation for the lack of significant risk reduction. This was also relevant to our analysis of blood glucose levels where high risk prevalence was relatively low before and after the intervention, for all employee groups.

Prior to controlling for site level effects, all three treatment arms significantly reduced their risk for poor nutrition, with the intervention group experiencing a significant net improvement compared to controls. The intervention sites (primarily the high-intensity group) also demonstrated significant net improvement in physical activity compared to controls. However, these findings became non-significant after controlling for site-level effects. These results may partly explain why we did not find an effect on overweight and obesity prevalence. A longer time horizon may be needed to determine if weight loss can be achieved through adoption of healthy lifestyle habits. Three other behavioral risk factors, tobacco use, stress and excess alcohol consumption, did not improve, nor were they expected to, since the programs did not explicitly target these health risks.

Compared to our first year results, the weight difference between intervention and control site subjects was nearly identical and of no clinical significance: a net difference of −1.5 pounds after one year and −1.6 pounds after two years. The net difference in BMI (−0.2 and −0.3) was also similar for both study periods. Thus, while no net difference in weight change was achieved after an additional year of intervention, participants in the high and moderate intensity treatment sites were able to maintain their weight and BMI while control site subjects experienced increases in both weight and BMI over the two year period. The prevalence of overweight increased more at the intervention sites (3.1%) after two years than after one year (1.7%). This increase can be explained by the simultaneous change in obesity prevalence; as obese employees lost weight, they were re-categorized as overweight. Obesity rates were reduced at intervention sites after two years (−1.0%) compared to an increase (0.6%) after one year, indicating an overall positive trend of reducing obesity rates as subjects shifted down to the overweight category. An important benefit of environmental interventions is that, once in place, they continue to influence behaviors with little additional cost and effort.

A larger net effect was observed for blood pressure and total cholesterol when comparing year one and year two results, although no statistical tests were conducted to determine whether the differences from one year to another were statistically significant.[1] In year one, net reductions in average systolic blood pressure, diastolic blood pressure and total cholesterol levels were −1.6, −1.2, and −1.1, respectively. After two years, these measures were reduced by −7.0, −1.6, and −3.6, respectively. Also, after two years, a potential effect was found on poor nutrition and physical activity which was not apparent in year one.

LIMITATIONS

There are many limitations worth noting. First, a major concern when conducting environmental worksite interventions is determining the unit of analysis. The question often asked is whether the intervention is measured by comparing average values for a given set of employees at a site, or values for each individual at that site. If the site is the unit of analysis, and only average values are used in statistical analyses, then large N's (and degrees of freedom) are required to determine statistical significance, and many sites are needed to establish a treatment effect.

In this study, when we evaluated program impact using the site as a control variable in our multilevel modeling, we were at times unable to find a significant worksite-level effect, which

[1]We did not statistically compare the Time 3 – Time 1 difference with the Time 2 – Time 1 difference because it was not central to our analysis and because it would have been complex to do so. Standard statistical tests would have been inappropriate given the dependence between the two differences and given the two sets of propensity score weights accorded to each difference.

was most likely due to our small sample size of 12 worksites. Mass and Hox (35) concluded that a minimum sample size of 50 is needed when conducting multilevel studies, and that fewer numbers often lead to biased estimates of the second-level standard errors. Thus, large multi-site studies with 50 or more worksites would be needed to test the effects of environmental interventions using site as the unit of analysis. This is hard to achieve in workplace studies since very few employers have enough sites to allow for such experiments, and even if they did, they would be reluctant to withhold interventions for large numbers of employees located at control sites for an extended time period.

We struggled with this issue and in the end decided to focus our analyses on employees and consider them the unit of analysis, taking pains to equalize intervention and control subjects on their baseline demographic and health status characteristics so that statistically similar individuals could be followed from Time 1 to Time 3. Further, to account for the reality that employees at any given site were exposed to a similar intervention, we controlled for site effects in our regression analysis, which then eliminated many statistically significant findings. This was not the case, however, for our primary outcomes of weight and BMI, where statistically significant results remained even after controlling for the site variable. Given the debate among researchers as to whether individual or site level results should be presented when reporting findings from environmental interventions, we decided to present both sets of results.

A second important potential bias could arise from the fact that the employee cohort examined in the analysis of Time 1–Time 3 results is demonstrably different from the group of employees from whom baseline data were collected. We compared baseline employees to those in the cohort and found that, compared to those who completed HRAs at baseline but not at follow-up, Time 1–Time 3 cohort members were more highly educated, more likely to be salaried workers and have white collar jobs, and more likely to be female. (Data not shown.) We controlled for these variables in our multivariate models but acknowledge that there may be other unmeasured variables that could influence the outcomes.

Coupled with the above limitation, we experienced a relatively high attrition rate over the course of the study (52.6% overall). By study arm, attrition was 54.3% and 45.1% for the intervention and control group, respectively. This level of attrition is not unusual for worksite studies (compared to clinical trials) since employees were not compensated for participating in the study. In real-world studies of workplace obesity programs, employers may experience even higher attrition rates (as high as 76.4%) in just one year, despite offering financial rewards (36).

To address the issue of missing data, we applied several statistical approaches to adjust for the potential bias due to attrition and as a way of performing sensitivity analyses to determine whether alternative methods would produce different conclusions. We applied mean-based and weight-based imputation methods and these produced results which supported our original findings. The weight-based approach we used is preferred by many researchers because it reduces the bias that non-response may cause in the estimates (Imbens, 2000). Using this method, we were able to include more participants in the analysis, increasing the sample size and improving the representiveness of the results. The weighting procedure corrected for the demographic characteristics of participants with missing values. Thus, the imputed values produced were expected to be similar to those of non-responders. Nonetheless, it may be the case that the weighting approach may increase variances of the estimates to a certain extent (Imbens, 2000). The methodological trade-off in using this approach is between bias reductions and increased variance. Since our goal was to improve the representativeness of the sample and balance the data between different arms of the study, the weighting procedure was preferred.

The second method considered to control for missing data is referred to as baseline observation carried forward (BOCF). This approach assumes that subjects who drop out of a study will maintain their baseline values. However, as shown in our analysis, as employees aged, they gained weight in the control group and maintained weight in the intervention group. Thus, we did not use the BOCF method since we concluded it would introduce more, not less, bias to our results.

A third method of handling missing data, mean-based imputation, was applied to the analysis of biometric values for cohort participants. Since only a small proportion of subjects had missing data (0.0% – 1.7%), the mean-based imputation method was considered an appropriate approach. However, as previously mentioned, our main findings were not affected by the missing data after we applied this method, thus we presented our results without accounting for the small amount of missing data.

A third limitation is related to attribution of outcomes to the intervention. While we observed modest improvements in blood pressure and total cholesterol for intervention site employees, we cannot rule out the possibility that these employees may have achieved these results because of pharmacological interventions rather than environmental changes. Since we did not observe significant reductions in weight or BMI, it is entirely possible that the improvements were due to other factors such as changes in medication use. However, because we did not analyze pharmacy data in this analysis, we are unable to control for this potential bias.

A fourth limitation relates to the quasi-experimental design of the study. Although moderate and high intensity sites were randomly assigned, treatment and control sites were not. Since the majority of the intervention sites were located in Texas and the control sites were located in Louisiana, West Virginia and New Jersey, factors such as geography, culture or other unmeasured variables may have influenced the results. To determine whether we needed to control for “state” effect, we examined the overweight and obesity prevalence rates for the four states included in the study using data from the National Center for Chronic Disease Prevention and Health Promotion’s Behavioral Risk Factor Surveillance System (37). We found that the prevalence rates for overweight and obesity in New Jersey, West Virginia and Louisiana at baseline (2006) for overweight and obesity were 36.7% and 25.6%, respectively. In Texas and Louisiana, the prevalence was similar at 36.1% and 26.6% for overweight and obesity, respectively. Since the prevalence rates were similar across treatment and control states, we concluded that there was no need to control for a state fixed effect.

Besides these limitations, there were others that may have influenced our results. Some sites were slow to implement the interventions. Thus, we may not have had enough data to observe any long-term changes associated with weight loss or other health risks due to differential length of exposure to the interventions. That said, we did include a site effect control variable to account for this and other various site level variations as previously explained. However, as noted above, because of our small sample size, when controlling for site effects we may have produced biased estimates favoring a lack of effect from the intervention. For example, although at an individual level we observed significant changes in diet and physical activity favoring intervention subjects, these results were no longer significant when controlling for site. Further studies examining the effects of program fidelity and dose on employee participation in and awareness of program features, and their combined effects on outcomes, are needed and may provide greater insight into the effects of environmental interventions.

Finally, results may have been attenuated by differences in those who dropped out after baseline data collection and those remaining in the study at time of final follow-up. We note that our results after two years of exposure to the interventions are different than our one year results and this may be explained by the different cohorts that were followed for each study period

(i.e., the Time 1–Time 3 cohort [N=2,431] was smaller than the Time 1–Time 2 cohort [n=3,152]). The remaining participants were proportionally better-educated and more likely to be in white-collar jobs, and were also less likely to be obese or use tobacco products than those who dropped out. Thus, any improvements in outcomes due to the intervention may have been muted because there was less room for improvement in the remaining cohort. On the other hand, cohort members had a proportionally greater high cholesterol risk than non-cohort employees, and the mean value for this risk factor did improve significantly from baseline.

Despite these limitations, this study contains some notable strengths. Unlike many studies conducted in workplaces, we employed a prospective, quasi-experimental design whereby employees at some sites received the interventions and others at control sites received only standard programs for a significant time period – in our case, two years. Also, this study collected multiple outcomes in addition to weight, enabling us to examine the effect of environmental and organizational interventions not just on weight, but also on other health risks of employees. Finally, this study examined the differential effects of two levels of intervention intensity, and the added value of engaging leadership in environmental and other health promotion interventions.

CONCLUSIONS

Overall, we observed a modest intervention impact on study outcomes in terms of weight and BMI, blood pressure and cholesterol. The more intense interventions produced better results when compared to controls than did moderate interventions. Over the course of two years, participants at the intervention sites (high intensity and moderate intensity sites combined) were able to maintain their weight and BMI while control subjects experienced increases in weight and BMI. These are encouraging findings. Given that national obesity rates are on the rise (1), and that people tend to gain weight as they age (38), the small but significant effects of environmental interventions at the worksite are notable. Stemming age-related weight gain is just as much a part of the solution for reducing the prevalence of overweight and obesity as helping people lose weight.

We also found a modest intervention effect for blood pressure values in favor of the intervention group and intensity effects for total cholesterol values in favor of high intensity sites, compared to the control sites. As for behavioral risk factors, when not controlling for site-level effects, intervention site participants showed greater improvements in diet and physical activity compared to controls. These findings were no longer statistically significant after controlling for site-level effects.

Changing employees' behaviors and modifying their health risks requires focused time and attention. Environmental and social ecological interventions often require engaging leadership support, changing the work culture, and modifying organizational policies, all of which can involve lengthy administrative approval processes. In addition, environmental interventions, compared to individually-focused ones, may take longer to implement or be noticed by employees. Thus, the full effects of environmental interventions may not be observable within even a two-year time horizon. Furthermore, environmental interventions are directed at all employees, not just those at high risk. Additional research, with more worksites, longer follow-up periods, and different risk groups is necessary to gain a better understanding of the broad range of environmental interventions available at the worksite and their impact on employee health risks.

Our findings suggest that it may be worthwhile for an organization to consider low-cost environmental interventions as complementary to individual approaches for weight management. While the effects are small in the near term, they can potentially translate to long

term clinical gains, especially if comprehensive programs that include both environmental and individual components are sustained over time.

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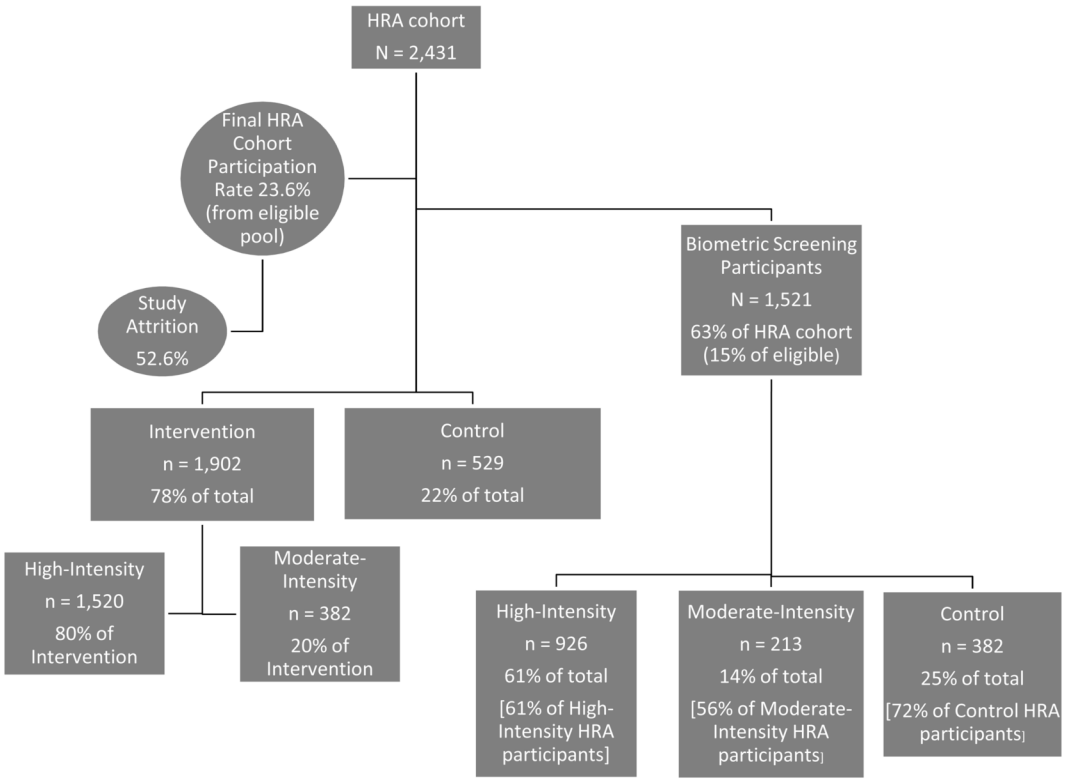

Figure 1.
Participation rates for HRA cohort and biometric screening cohort participants.

Table 1

Definitions of Health Risk Outcomes Groups

Biometric Risk Factors	
Normal weight	BMI† 15 – 24.9*
Overweight	BMI 25.0 – 29.9
Obese	BMI ≥ to 30
High blood pressure	Blood Pressure 160/100 or higher (systolic/diastolic)
High cholesterol	Total cholesterol 240 mg/dl or higher
High blood glucose	Blood glucose 126 mg/dl or higher
Behavioral Risk Factors	
Poor nutrition	4 or more fast food meals per week OR
	2 or more sweetened beverages per day OR
	3 or fewer fruit and vegetable servings per day
Lack of physical activity	Does not engage in any moderate or strenuous physical activity at least once per week
Tobacco use	Currently using tobacco
High alcohol use	Men: 3+ drinks per day OR 15+ drinks per week
	Women: 2+ drinks per day OR 8+ drinks per week
High stress	Reported experiencing high stress over the past four weeks and poor ability to deal with stress

†BMI: Body Mass Index

Based on consultation with medical directors at Thomson Reuters, any employee with a BMI<15 was removed from this analysis as these values were considered to be potentially unreliable, inaccurate, or illogical.

Table 2

Baseline demographics characteristics of the HRA cohort

	Unadjusted*			Adjusted*		
	Intervention (n=1,902)	**Control (n=529)**	***P***	**Intervention (n=1,902)**	**Control (n=529)**	***P***
Average Age†	43.9	46.4	<.0001	44.6	44.8	0.5313
% Female‡	27.30%	27.40%	0.9552	27.10%	27.00%	0.8862
Ethnicity‡			0.0001			0.0953
White	75.10%	83.20%		77.10%	79.10%	
Education‡			<.0001			0.3606
Not Indicated	24.50%	51.40%		30.40%	30.40%	
Less than Bachelor Degree	28.80%	8.10%		24.50%	22.80%	
Bachelor Degree	35.50%	23.80%		32.90%	35.00%	
Masters or Doctorate	11.20%	16.60%		12.20%	11.80%	
Wage Status			<.0001			0.3594
Salaried	63.20%	74.10%		65.10%	63.90%	
Hourly	36.80%	25.90%		34.90%	36.20%	
Work Status‡			<.0001			0.2063
Officials and Managers, Professionals, & Sales	49.00%	51.40%		49.40%	49.60%	
Technicians, Office and Clerical, & Craft Workers (skilled)	35.30%	40.60%		36.60%	38.10%	
Operatives (semi-skilled), Laborers (unskilled), & Service Workers	15.70%	7.90%		14.00%	12.40%	
CCI‡	0.27	0.26	0.8038	0.26	0.24	0.4152
NPDG‡	0.16	0.15	0.5027	0.16	0.15	0.7337

*"Unadjusted" refers to analyses conducted without using propensity score weighting methodology, while "Adjusted" indicates that propensity score weighting methodology was used to account for covariates

†P-values are the results of t-test between the control and intervention sites.

‡P-values are the results of chi-square analyses between the control and intervention sites

Table 3

Biometric cohort outcomes for primary hypotheses by intervention and control with propensity score weighting

	Intervention (n=1,139)				**Control (n=382)**			
	% at High Risk			***P***	% at High Risk			***P***
	2006	2008	Δ		2006	2008	Δ	
Obese	31.0%	30.0%	−1.0%	0.2339	33.8%	31.7%	−2.1%	0.0013
Overweight	39.8%	42.9%	3.1%	0.0032	41.0%	40.9%	−0.1%	0.9143
	Average Value			***P***	Average Value			***P***
	2006	2008	Δ		2006	2008	Δ	
Weight	189.3	189.0	−0.3	0.3801	187.9	189.2	1.3	0.008
BMI	28.3	28.2	−0.1	0.3348	28.0	28.2	0.2	0.005

Table 4

Comparison of the Difference-in-differences analysis not controlling for site level effects to the Difference-in-differences analysis controlling for site level effects

	Not Controlling for Site Effects		Controlling for Site Level Effects	
	Intervention v. Control		Intervention v. Control	
	† Δ b/t Treatment	*P*	† Δ b/t Treatment	*P*
Primary Hypotheses				
Obese	1.1%	0.4274	0.5%	0.9216
Overweight	3.2%	0.1232	5.2%	0.2252
Weight	−1.6	0.0051	−1.6	0.0050
BMI	−0.3	0.0028	−0.3	0.0027

Table 5

Biometric cohort outcomes for primary hypotheses by intensity with propensity score weighting

	High-intensity (n=926)				**Moderate-intensity (n=213)**				**Control (n=382)**			
	% at High Risk			***P***	% at High Risk			***P***	% at High Risk			***P***
	2006	2008	Δ		2006	2008	Δ		2006	2008	Δ	
Obese	30.4%	29.3%	−1.1%	0.1634	29.4%	31.1%	1.7%	0.0489	33.7%	31.6%	−2.1%	0.0014
Overweight	39.5%	43.2%	3.7%	0.0005	43.3%	45.4%	2.1%	0.0587	41.1%	40.9%	−0.2%	0.8609
	Average Value			***P***	Average Value			***P***	Average Value			***P***
	2006	2008	Δ		2006	2008	Δ		2006	2008	Δ	
Weight	188.9	188.7	−0.2	0.6399	187.9	187.9	0.0	0.9827	188.0	189.3	1.3	0.0071
BMI	28.2	28.2	0.0	0.5604	28.3	28.3	0.0	0.8326	28.0	28.2	0.2	0.0045

Table 6

Comparison of the Difference-in-differences analysis not controlling for site level effects to the Difference-in-differences analysis controlling for site level effects

	Not Controlling for Site Effects				Controlling for Site Effects			
	High-intensity v. Control		Moderate-intensity v. Control		High-intensity v. Control		Moderate-intensity v. Control	
	†Δ b/t treatment	*P*	†Δ b/t treatment	*P*	†Δ b/t treatment	*P*	†Δ b/t treatment	*P*
Primary Hypotheses								
Obese	1.0%	0.5315	3.8%	0.1193	0.3%	0.9486	0.1%	0.8780
Overweight	3.9%	0.0857	2.3%	0.5087	5.5%	0.2246	4.4%	0.4714
Weight	−1.5	0.0163	−1.3	0.1454	−1.5	0.0148	−2.1	0.0333
BMI	−0.2	0.0089	−0.2	0.1817	−0.2	0.0075	−0.3	0.0341

Table 7

Biometric cohort outcomes for secondary hypotheses by intervention and control with propensity score weighting

	Intervention (n=1,139)				**Control (n=382)**			
	% at High Risk				**% at High Risk**			
	2006	2008	Δ	***P***	2006	2008	Δ	***P***
Health Risk								
Biometric Screening Risk								
High Blood Pressure	3.9%	2.2%	−1.7%	0.0068	1.3%	2.4%	1.1%	0.0361
High Cholesterol	10.1%	9.1%	−1.0%	0.2429	12.0%	12.0%	0.0%	0.9762
High Blood Glucose	3.9%	4.2%	0.3%	0.4690	3.2%	3.8%	0.6%	0.1417
	Intervention (n=1,139)				Control (n=382)			
Biometric	Average Value				Average Value			
Screening Value	2006	2008	Δ	***P***	2006	2008	Δ	***P***
BP Systolic	124.4	122.3	−2.1	<.0001	123.2	128.1	4.9	<.0001
BP Diastolic	80.3	78.2	−2.1	<.0001	79.6	79.1	−0.5	0.3862
Cholesterol	196.0	192.8	−3.2	0.0006	193.3	193.7	0.4	0.792
Blood Glucose	94.3	96.2	1.9	<.0001	95.1	95.8	0.7	0.3881

Table 8

Comparison of the Difference-in-differences analysis not controlling for site level effects to the Difference-in-differences analysis controlling for site level effects

	Not Controlling for Site Effects		Controlling for Site Level Effects	
	Intervention v. Control		Intervention v. Control	
	†Δ b/t Treatment	*P*	†Δ b/t Treatment	*P*
Biometric Screening Risk				
High Blood Pressure	−2.8%	0.0156	−2.5%	0.1351
High Cholesterol	−1.0%	0.5548	−0.9%	0.7095
High Blood Glucose	−0.3%	0.7040	−0.2%	0.7999
Biometric Screening Value				
BP Systolic	−7.0	<.0001	−7.0	<.0001
BP Diastolic	−1.6	0.0015	−1.7	0.0014
Cholesterol	−3.6	0.0205	−3.6	0.0205
Blood Glucose	1.2	0.1409	1.2	0.1456

†"Δ b/w Treatments" was calculated as Intervention-Control, Δ indicates either the percent at risk or the average value

Table 9

Biometric cohort outcomes for secondary hypotheses by intensity with propensity scores weighting

	High-intensity (n=926)				Moderate-intensity (n=213)				Control (n=382)			
	% at High Risk				% at High Risk				% at High Risk			
	2006	2008	Δ	*P*	2006	2008	Δ	*P*	2006	2008	Δ	*P*
Biometric Screening Risk												
High Blood Pressure	3.8%	1.6%	−2.2%	<.0001	5.8%	2.9%	−2.9%	0.0002	1.3%	2.4%	1.2%	0.0346
High Cholesterol	9.7%	8.1%	−1.6%	0.0595	8.2%	10.7%	2.6%	0.0019	11.9%	11.9%	0.0%	0.9819
High Blood Glucose	3.5%	4.3%	0.8%	0.0594	3.3%	2.1%	−1.2%	0.0007	3.1%	3.8%	0.6%	0.1436
	Average Value				Average Value				Average Value			
	2006	2008	Δ	***P***	2006	2008	Δ	***P***	2006	2008	Δ	***P***
Biometric Screening Value												
BP Systolic	125.4	122.7	−2.6	<.0001	120.9	120.7	−0.2	0.8465	123.1	128.0	4.9	<.0001
BP Diastolic	80.5	78.4	−2.1	<.0001	79.4	77.2	−2.2	0.007	79.5	79.1	−0.4	0.4205
Cholesterol	195.2	191.0	−4.2	<.0001	197.1	197.7	0.6	0.7744	193.2	193.8	0.5	0.7200
Blood Glucose	94.2	96.0	1.8	0.0003	93.8	94.8	1.0	0.2537	95.1	95.7	0.6	0.4706

Table 10

Comparison of the Difference-in-differences analysis not controlling for site level effects to the Difference-in-differences analysis controlling for site level effects

	Not Controlling for Site Effects				Controlling for Site Effects			
	High-intensity v. Control		Moderate-intensity v. Control		High-intensity v. Control		Moderate-intensity v. Control	
	† Δ b/t treatment	*P*	† Δ b/t treatment	*P*	† Δ b/t treatment	*P*	† Δ b/t treatment	*P*
Biometric Screening Risk								
High Blood Pressure	−3.4%	0.0047	−4.1%	0.066	−2.9%	0.0675	−0.8%	0.6543
High Cholesterol	−1.6%	0.3821	2.6%	0.3488	−1.2%	0.6109	0.5%	0.9069
High Blood Glucose	0.2%	0.8714	−1.8%	0.1459	0.3%	0.9799	−2.2%	0.3349
Biometric Screening Value								
BP Systolic	−7.5	<.0001	−5.1	<.0001	−7.4	<.0001	−5.4	<.0001
BP Diastolic	−1.7	0.0016	−1.8	0.0566	−1.6	0.0033	−1.9	0.0311
Cholesterol	−4.7	0.0049	0.1	0.9829	−4.3	0.0107	−1.2	0.6424
Blood Glucose	1.2	0.1524	0.4	0.7184	1.3	0.1400	0.83	0.5516

† "Δ b/w Treatments" was calculated as Intervention-Control, Δ indicates either the percent at risk or the average value

Table 11

HRA cohort outcomes for secondary hypotheses by intervention and control with propensity scores

	Intervention (n=1,902)				Control (n=529)			
	% at High Risk				% at High Risk			
	2006	2008	Δ	*P*	2006	2008	Δ	*P*
Health Risk								
Poor Nutrition	78.3%	69.3%	−9.0%	<.0001	74.2%	71.6%	−2.6%	<.0025
Poor Physical Activity	10.2%	7.1%	−3.1%	<.0001	5.3%	5.4%	0.1%	0.8719
Tobacco Use	11.7%	11.5%	−0.2%	0.5635	7.4%	8.3%	0.9%	0.0183
High Alcohol Use	6.4%	5.7%	−0.7%	0.2142	2.4%	2.4%	0.0%	0.9585
High Stress	2.7%	2.0%	−0.7%	0.0544	2.0%	0.8%	−1.2%	0.0008

Table 12

Comparison of the Difference-in-differences analysis controlling for site level effects to the Difference-in-differences analysis without site level effects

	Not Controlling for Site Effects		Controlling for Site Level Effects	
	Intervention v. Control		Intervention v. Control	
	† Δ b/t Treatment	*P*	† Δ b/t Treatment	*P*
Health Risk				
Poor Nutrition	−6.4%	0.0005	−5.3%	0.0943
Poor Physical Activity	−3.2%	0.0147	−0.9%	0.9659
Tobacco Use	−1.1%	0.1419	−1.5%	0.5117
High Alcohol Use	−0.7%	0.4789	−1.6%	0.441
High Stress	0.5%	0.5136	−0.6%	0.7258

Table 13

HRA cohort outcomes for secondary hypotheses by intensity with propensity scores

	High-intensity (n=1,520)				Moderate-intensity (n=382)				Control (n=529)			
	% at High Risk				% at High Risk				% at High Risk			
	2006	2008	Δ	*P*	2006	2008	Δ	*P*	2006	2008	Δ	*P*
Health Risk												
Poor Nutrition	77.6%	69.8%	−7.8%	<.0001	78.3%	69.2%	−9.1%	<.0001	74.4%	71.8%	−2.6%	0.0027
Poor Phys. Activity	9.9%	7.1%	−2.8%	<.0001	10.6%	6.8%	−3.8%	<.0001	5.4%	5.5%	0.1%	0.9055
Tobacco Use	12.0%	11.9%	−0.1%	0.7908	10.9%	10.3%	−0.6%	0.0731	7.5%	8.4%	0.9%	0.0223
High Alcohol Use	6.1%	6.1%	0.0%	0.9860	7.3%	5.7%	−1.6%	0.0056	2.4%	2.4%	0.0%	0.9450
High Stress	2.6%	1.9%	−0.7%	0.0479	1.8%	2.0%	0.2%	0.5172	2.0%	0.8%	−1.2%	0.0007

Table 14

Comparison of the Difference-in-differences analysis controlling for site level effects to the Difference-indifferences analysis

	Not Controlling for Site Effects				Controlling for Site Effects			
	High-intensity v. Control		Moderate-intensity v. Control		High-intensity v. Control		Moderate-intensity v. Control	
	† Δ b/t treatment	*P*	† Δ b/t treatment	*P*	† Δ b/t treatment	*P*	† Δ b/t treatment	*P*
Health Risk								
Poor Nutrition	−5.2%	0.0089	−6.5%	0.0228	−4.6%	0.1554	−7.7%	0.0683
Poor Phys. Activity	−2.9%	0.037	−3.9%	0.0758	−0.7%	0.8909	−1.6%	0.7664
Tobacco Use	−1.0%	0.2622	−1.5%	0.2075	−1.6%	0.492	−1.1%	0.6896
High Alcohol Use	0.0%	0.9904	−1.6%	0.3189	−1.0%	0.5851	−3.9%	0.3060
High Stress	0.5%	0.5429	1.5%	0.2236	−0.5%	0.7045	−0.6%	0.8039