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OPEN

Effects of Reducing Sedentary Time on Glucose Metabolism in Immigrant Pakistani Men

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ABSTRACT

ANDERSEN, E., U. EKELUND, and S. A. ANDERSSEN. Effects of Reducing Sedentary Time on Glucose Metabolism in Immigrant Pakistani Men. *Med. Sci. Sports Exerc.*, Vol. 47, No. 4, pp. 775–781, 2015. **Purpose**: This study aimed to examine the association between changes in objectively measured overall physical activity (PA) and changes in fasting and postprandial plasma insulin, C-peptide, and glucose concentrations in type 2 diabetes-prone immigrant Pakistani men living in Norway and to examine whether this association is explained by changes in moderate and vigorous PA (MVPA) or changes in sedentary time. **Methods**: The current study is a secondary cohort analysis on data collected from the Physical Activity and Minority Health study, a randomized controlled trial aimed at increasing the PA level, and not sedentary time *per se*, in a group of sedentary immigrant Pakistani men (n = 150). For the present analyses, the two groups were merged and a cohort analysis was performed. Overall PA (counts per minute) and its subcomponents, sedentary time and MVPA, were measured with accelerometry. Outcome variables were measured after a 2-h standardized glucose tolerance test. **Results**: Change in overall PA was significantly associated with postprandial log-transformed plasma insulin (β = −0.002; 95% confidence interval (CI), −0.003 to 0.000; P = 0.008), C-peptide (β = −2.7; 95% CI, −4.9 to −0.5; P = 0.01), and glucose concentration (β = −0.006; 95% CI, −0.01 to −0.002; P = 0.002). Change in sedentary time was significantly and beneficially associated with changes in postprandial log-transformed plasma insulin (β = 0.002; 95% CI, 0.001–0.003; P = 0.001), C-peptide (β = 3.7; 95% CI, 1.5–6.0; P = 0.001), and glucose concentration (β = 0.006; 95% CI, 0.002–0.1; P = 0.002), independent of changes in MVPA, waist circumference, and other confounders. **Conclusions**: Increasing overall PA by reducing sedentary time seems as important as increasing time spent at MVPA in relation to postprandial plasma insulin and glucose levels in diabetes-prone immigrant men. **Key Words:** OVERALL PHYSICAL ACTIVITY, CARBOHYDRATE, INSULIN, ETHNIC MINORITY

EPIDEMIOLOGY

The health benefits of engaging in moderate- and vigorous-intensity physical activity (MVPA) have been documented extensively, and more recently, sedentary behavior has been suggested to be associated with a variety of health outcomes (16,28). Furthermore, sedentary behavior has been hypothesized to have deleterious effects on health that are distinct from the lack of MVPA (30). The Australian Diabetes, Obesity, and Lifestyle study observed dose–response associations between television viewing and waist circumference, systolic blood pressure, postprandial glucose (glucose at 2 h), triglycerides, and HDL cholesterol levels in healthy, physically active participants (18). Observational studies have also reported that sitting time or sedentary behavior correlates positively with fasting plasma insulin (11) and glucose levels (17), independent of the time spent in MVPA. In a prospective study, objectively measured time spent sedentary at baseline was associated with insulin resistance at follow-up, and this association was further strengthened after adjusting for MVPA (19). However, the literature is not consistent and prospective observations in high-risk individuals suggest that accelerometer-measured MVPA but not sedentary time predicted insulin resistance at follow-up (13).

On the basis of the previously mentioned studies, some have suggested that sedentary behavior is a paradigm in its own right that is distinctive from that of MVPA (12) and that the physiology of inactivity is different from that of exercise physiology (16). Sedentary behavior should therefore not be used as a synonym for physical inactivity (i.e., not meeting physical activity (PA) recommendations for public health) but rather be defined as "muscular inactivity rather than the absence of exercise" (12), and it refers to activities that involve energy expenditure at the level of <1.5 METs in a sitting or reclining position (i.e., sitting, lying down). By

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definition, a reduction in sedentary time must therefore be associated with an increase in overall PA, and we have previously demonstrated that a social cognitive theory-based multicomponent PA program beneficially influenced PA level (4) and postprandial plasma glucose and insulin concentrations (5) in immigrant Pakistani men living in Norway, a group with high risk of developing type 2 diabetes because of their ethnicity. However, we did not disentangle whether changes in any of the subcomponents of PA, i.e., sedentary time and MVPA, were associated with changes in fasting and postprandial plasma insulin, C-peptide, and glucose concentrations. Understanding the independent associations between changes in sedentary time and MVPA and metabolic outcomes in individuals prone to metabolic diseases is important for public health purposes. We hypothesized that an increase in overall PA induced by a reduction in sedentary time would be associated with favorable changes in postprandial metabolic outcomes, independent of time spent in MVPA.

METHODS

The current study is a secondary cohort analysis based on data collected from the Physical Activity and Minority Health study, a 5-month randomized controlled study aimed at increasing the PA levels of immigrant Pakistani men living in Norway. The design, intervention program, and methods of the Physical Activity and Minority Health study have been described in detail previously (5); a brief description is given as follows. For the original randomized controlled trial, the participants were randomly allocated to either a control or an intervention group. The intervention used a social cognitive theory framework and comprised structured supervised group exercises (twice a week), group lectures (two sessions of 2 h), an individual counseling session, and a telephone call. The intervention aimed to increase total PA level and not MVPA or sedentary time *per se*, so for the present analyses, the two groups were merged and a cohort analysis was performed.

Pakistani men (born in Pakistan or first-generation immigrants) living in Oslo, Norway, age between 25 and 60 yr and not physically active on a regular basis (exercising no more than twice per week at a moderate- or higher-intensity level for 30 min or more at a time or were active commuters (e.g., cycling or walking to work on most days of the week)) were candidates for inclusion in this study. In addition, the following exclusion criteria were applied: known type 2 diabetes before randomization, physical limitations prohibiting participation in organized exercise sessions, and not being able to speak Norwegian.

The recruitment process was carried out in September and October 2008. Briefly, men were recruited at six mosques and at various Muslim festivals in Oslo. Approximately 250 men volunteered to participate, and 182 of these were screened. Of those screened, 32 failed to meet the inclusion criteria, providing a total sample of 150 participants. Seventeen participants were lost to follow-up. The recruitment, exclusions, and participants lost to follow-up are presented in Figure 1. Before participation in the study, a written informed consent was obtained from each participant. The Regional Committee for Medical Research Ethics (Ref. no. S-07300b) and the Norwegian Social Science Data Services (Ref. no. 17212/2/KS) approved the study.

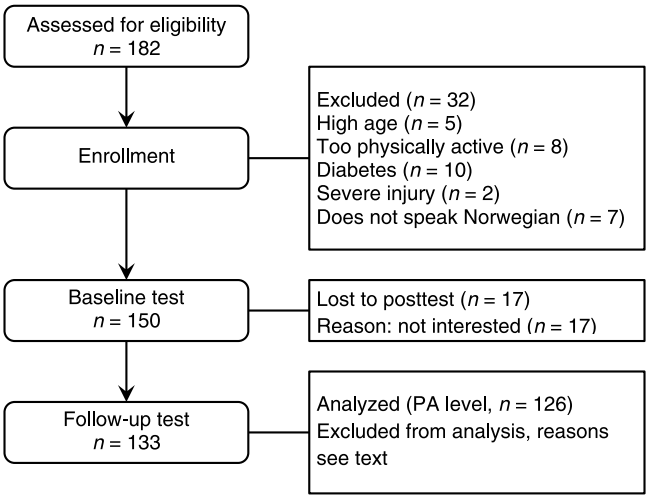

FIGURE 1—Flow of participants through the trial.

PA behavior measured by accelerometry and diabetes risk factors were examined in all participants before and immediately after the 5-month intervention. All testing was conducted at the Norwegian School of Sport Sciences. The baseline test was carried out in October to November 2008, and the follow-up test, in March to May 2009. After a 12-h overnight fast (minimum, 8 h), venous blood samples were drawn from an antecubital vein. An oral glucose tolerance test was performed, i.e., 75 g of glucose in 200 mL of water was ingested, and plasma glucose, insulin, and C-peptide were determined before and 2 h after ingestion of the glucose drink. The samples were centrifuged for 10 min at 2500*g* and analyzed the same day. Blood variables were determined at the Dr. V. Furst Laboratory for Clinical Chemistry, Oslo, Norway. A Modular P Machine (Roche, Japan) was used for measuring glucose (photometry), insulin, and C-peptide (immunoassays).

Habitual PA was assessed with an MTI ActiGraph accelerometer (MTI model 7164; Manufacturing Technology, Inc., Fort Walton Beach, FL). The monitor measures acceleration in the vertical plane and provides a summary measure "count" for a predefined epoch. The participants were instructed to wear the accelerometer for 7 d on the right hip during all waking hours, except while swimming and bathing. Accelerometers were programmed to start recording at 6:00 a.m. the day after the participants received their accelerometer. The epoch length was set to 1 min. In the analysis of accelerometer data, epoch periods with a value of zero (with two exceptions) for 60 min or longer were interpreted as "accelerometer not worn" and were excluded

TABLE 1. Descriptive characteristic of participants at baseline and follow-up.

Characteristics	Baseline (n = 144–150)	Follow-up (n = 117–130)
Weight (kg)	83.8 ± 13.0	82.4 ± 12.7*
Body mass index (kg·m^{-2})	27.2 ± 3.6	26.8 ± 3.6
Waist circumference (cm)	98.2 ± 10.1	97.1 ± 10.2
Fasting insulin (pmol·L^{-1})	103 ± 56	85 ± 60*
Postprandial insulin (pmol·L^{-1})	796 ± 587	607 ± 545*
Fasting C-peptide (pmol·L^{-1})	1003 ± 316	953 ± 358
Postprandial C-peptide (pmol·L^{-1})	3835 ± 1363	3488 ± 1372*
Fasting glucose (mmol·L^{-1})	5.4 ± 0.9	5.3 ± 1.0
Postprandial glucose (mmol·L^{-1})	6.9 ± 2.9	6.1 ± 2.3*
Overall PA level (counts·min^{-1})	308 ± 131	370 ± 147*
MVPA (min·d^{-1})	32.3 ± 20.8	41.5 ± 23.4*
Sedentary time (h·d^{-1})	8.6 ± 1.6	8.3 ± 1.7

Data are means ± SD.
*P < 0.01 for baseline versus follow-up.

from the analysis (13,31). PA data were included if the participant had accumulated a minimum of 480 min·d^{-1} of activity data for at least 2 d regardless of the type of day (work day or weekday). There were no differences in overall PA between those who wore the monitor for 2 d and those who wore the monitor for 3 d or longer (data not shown). For that reason, it was decided to also include those participants who had only worn the monitor for 2 d (baseline, n = 7; follow-up, n = 3). On average (±SD), participants wore the monitor for 6.3 ± 1.8 d at baseline and 6.1 ± 1.5 d at the follow-up assessment. Average wearing time (±SD) was 13.5 ± 1.5 h·d^{-1} at baseline and 13.6 ± 1.6 h·d^{-1} at the follow-up assessment. Accelerometer data were processed and analyzed using the SAS-based (version 9) software program (SAS Institute, Inc., Cary, NC) called CSA analyzer (http://csa.svenssonsport.dk). One hundred and forty-two participants had valid recordings at baseline (95%). Four lost their monitor, and four had less than two valid days of recordings. One hundred and twenty-six participants provided valid PA recordings at posttest. Seventeen were lost to follow-up, five had less than 2 d of recordings, and two did not return their accelerometer. Sedentary behavior (sedentary time) was defined as ≤100 counts per minute (24), light-intensity activity was defined as 101–1951 counts per minute, and MVPA was defined as ≥1952 counts per minute (14). These cut points are widely used and show good correlation (r = 0.88) with direct $\dot{V}O_2$ measurement (14). Overall PA was calculated as total counts/wear time.

Waist circumference was measured at the end of a gentle expiration, midway between the lower rib margin and the iliac crest. Weight was measured without shoes in light clothing by a SECA electronic scale (model 767; SECA, Germany) to the nearest 0.5 kg. Height was measured without shoes with a transportable stadiometer (Harpenden; Holtain, Crymych, GB) and was set to the nearest 0.5 cm.

Means (±SD) were used to describe baseline and follow-up data. Paired-samples t-test was used for testing differences

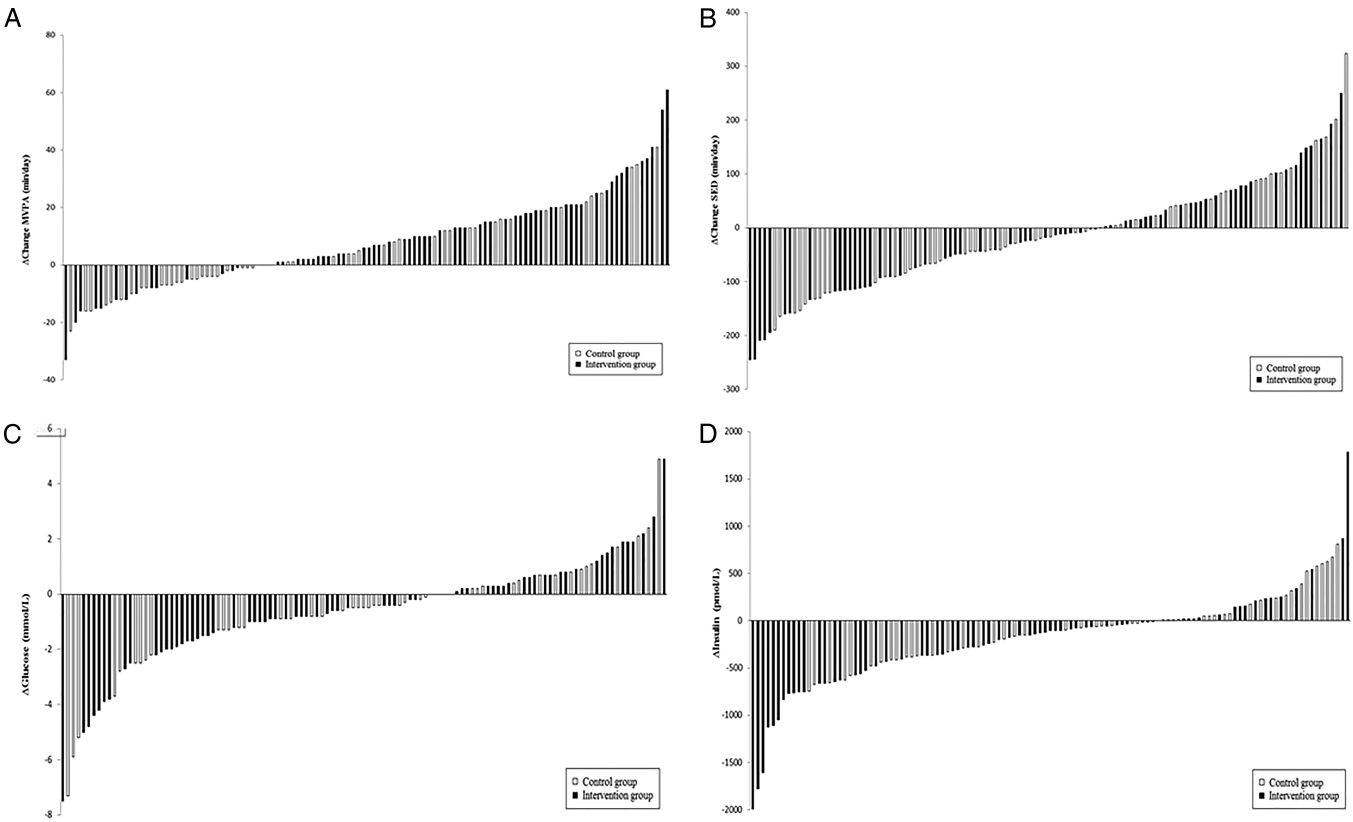

FIGURE 2—Individual changes (follow-up − baseline) in MVPA (A), sedentary time (B), glucose (C), and insulin (D). Participants originally randomized to the intervention group are represented by *black bars*, and the control group is represented by *white bars*. SED, sedentary.

between baseline and follow-up. Changes in exposure and outcome variables were calculated as follow-up minus baseline. Change in insulin-2h was normalized ($\log_{10}$); glucose and C-peptide were normally distributed. Univariate and multivariate linear regression analyses were used to examine the association between change in the exposure variables (overall PA (counts per minute), time spent sedentary and in MVPA) and change in fasting and postprandial insulin, C-peptide, and glucose. The potential confounders included in the multivariate models were waist circumference, age, smoking, and intervention group. Furthermore, associations between changes in time spent sedentary and in MVPA were mutually adjusted to examine whether these subcomponents of overall PA were independently associated with the outcomes. Colinearity was assessed using the variance inflation factor. We analyzed all data using the Statistical Package for the Social Sciences (SPSS; IBM, Inc., Chicago, IL) version 21, and statistical significance was set at $P \leq 0.05$.

RESULTS

The men were, on average, 37.4 ± 7.7 yr old at baseline. Of the 150 men included in the study, 11 were diagnosed with type 2 diabetes at the baseline test, two had impaired fasting glucose, 27 had impaired glucose tolerance, and 75 had the metabolic syndrome; in addition, the values referring to CHO metabolism in Table 1 indicate that the participants were prone to diabetes.

At follow-up, there was a significant reduction in fasting insulin and postprandial insulin, C-peptide, and glucose but not in fasting glucose and C-peptide (Table 1). Overall PA increased by 20% (95% confidence interval (CI), 13–27) between baseline and follow-up. On average, sedentary time was reduced by 13.8 min·d^{-1} (95% CI, −32 to 5) and ranged from −245 to 324 min·d^{-1} (Fig. 2). Time spent in MVPA increased by 7.6 min·d^{-1} (95% CI, 4–10) and ranged from 61 to −33 min·d^{-1} (Fig. 2). Change in sedentary time from baseline to follow-up was not significantly correlated with change in MVPA. Change in sedentary time was correlated with changes in postprandial insulin, C-peptide, and glucose (r = 0.32, 0.30, and 0.33, respectively; $P < 0.01$), whereas change in MVPA was only significantly correlated with change in waist circumference ($r = -0.30$; $P < 0.01$).

Changes in overall PA and changes in sedentary time were significantly associated with changes in postprandial plasma insulin, C-peptide, and glucose concentrations in univariate analyses (Table 2). The unadjusted associations between MVPA and postprandial plasma insulin, C-peptide, and glucose were not statistically significant, although the relation with glucose approached statistical significance. Model 1 (adjusting for changes in waist circumference, smoking, age, and intervention group) and model 2 (additionally adjusting sedentary time for MVPA and *vice versa*) showed virtually no change in the magnitude or direction of the association for sedentary time. Adjusting for baseline waist circumference produced results similar to those of adjusting for changes in waist circumference (data not shown). The results in model 2 imply that reducing sedentary time by, for example, 30 min·d^{-1} will reduce postprandial C-peptide by 111 pmol·L^{-1} and glucose by 0.18 mmol·L^{-1}. In sensitivity analyses, we also adjusted for the baseline values of the outcomes and the results for overall PA and sedentary time were materially unchanged (data not shown). Change in overall PA (counts per minute) and its subcomponents was not significantly associated with change in fasting insulin (data not shown).

TABLE 2. Association between changes in objectively measured overall PA (counts per minute), sedentary time, and MVPA with changes in postprandial insulin, C-peptide, and glucose in diabetes-prone immigrant Pakistani men living in Norway (n = 116).

	β Coefficient (±95% CI)	P value	R^2
Insulin at 2 h (pmol·L^{-1})[a]			
Unadjusted			
Sedentary time	0.002 (0.001 to 0.003)	0.001	0.10
MVPA	−0.007 (−0.015 to 0.002)	0.13	0.02
Overall PA	−0.002 (−0.003 to 0.000)	0.008	0.06
Model 1			
Sedentary time	0.002 (0.001 to 0.003)	0.001	0.14
MVPA	−0.004 (−0.014 to 0.005)	0.35	0.05
Overall PA	−0.002 (−0.003 to 0.000)	0.02	0.09
Model 2			
Sedentary time[b]	0.002 (0.001 to 0.003)	0.001	0.14
MVPA[c]	−0.001 (−0.011 to 0.007)	0.75	0.14
C-peptide at 2 h (pmol·L^{-1})			
Unadjusted			
Sedentary time	3.6 (1.5 to 5.7)	0.001	0.10
MVPA	−3.3 (−18 to 11)	0.6	0.002
Overall PA	−2.7 (−4.9 to −0.5)	0.01	0.05
Model 1			
Sedentary time	3.5 (1.4 to 5.5)	0.001	0.15
MVPA	0.7 (−14 to 16)	0.9	0.06
Overall PA	−2.2 (−4.5 to 0.08)	0.06	0.09
Model 2			
Sedentary time[b]	3.7 (1.5 to 5.8)	0.001	0.16
MVPA[c]	5.5 (−9 to 20)	0.4	0.16
Glucose at 2 h (mmol·L^{-1})			
Unadjusted			
Sedentary time	0.006 (0.003 to 0.01)	0.001	0.11
MVPA	−0.025 (−0.05 to 0.001)	0.056	0.03
Overall PA	−0.006 (−0.01 to −0.002)	0.002	0.08
Model 1			
Sedentary time	0.007 (0.003 to 0.10)	0.001	0.12
MVPA	−0.031 (−0.057 to −0.004)	0.02	0.05
Overall PA	−0.007 (−0.011 to −0.003)	0.001	0.11
Model 2			
Sedentary time[b]	0.006 (0.002 to 0.10)	0.001	0.15
MVPA[c]	−0.022 (−0.048 to 0.003)	0.08	0.15

Data are unstandardized regression coefficients (95% CI).
Models 1 and 2 are adjusted for changes in waist circumference, smoking, group, age, and wear time.
[a]$\log_{10}$.
[b]Additionally adjusted for MVPA.
[c]Additionally adjusted for sedentary time.

DISCUSSION

The results of the present study suggested that an increase in overall PA, likely explained by a reduction in sedentary time rather than an increase in MVPA, was associated with beneficial changes in postprandial plasma insulin, C-peptide, and glucose concentrations in immigrant Pakistani men living in Norway with a high risk of developing type 2 diabetes. The association between change in sedentary time and metabolic outcomes was independent of changes in MVPA, waist circumference, smoking, and other confounders. Our results

suggest that an increase in overall PA by reducing sedentary time seems beneficial for glucose metabolism in immigrant Pakistani men living in Norway. Our results suggest that a change in MVPA of 10 $min \cdot d^{-1}$ or a change in sedentary time of 30 $min \cdot d^{-1}$ is associated with a similar magnitude of change in 2-h glucose of approximately 0.2 $mmol \cdot L^{-1}$. This change is lower compared with that observed after an extensive lifestyle intervention in the Finnish Diabetes Prevention Study (33) but may be clinically relevant.

Our results extend previous cross-sectional observations suggesting that sedentary time is positively associated and time spent in light-intensity activity is negatively associated with postprandial glucose (17). Others have suggested that time in MVPA is prospectively and inversely associated with insulin resistance, independent of time spent sedentary in individuals at high risk of developing type 2 diabetes because of family history (13). A recent previous 5-yr follow-up of that study also observed that a 5-yr change in MVPA was inversely associated with change in fasting insulin, although this association was attenuated after adjusting for time spent sedentary (34). In contrast, change in time spent sedentary was not associated with change in fasting insulin (34). Differences between the findings of Wijndaele et al. (34) and ours may be explained by differences in risk status between groups. We examined immigrant Pakistani men, a high-risk group because of their ethnicity (6). Furthermore, 50% of our sample was categorized as having the metabolic syndrome (2) and 76% were obese according to their body mass index (35) compared with 29% in the study of Wijndaele et al (34), and the fasting insulin levels were remarkably higher in our sample. Thus, in high- or very high-risk individuals, substituting sedentary time with light-intensity PA seems beneficial in relation to glucose metabolism, whereas in those with lower risk, an increase in moderate-intensity activity may be needed to positively influence metabolic parameters. However, future randomized studies are needed to determine the minimal dose of activity needed to reduce the metabolic risk in high-risk individuals. Finally, different results between the study of Wijndaele et al. (34) and our study may be explained by a differential association between sedentary time and postprandial glucose metabolism compared with fasting levels of glucose and insulin. However, this hypothesis needs confirmation in future studies.

Our study has some limitations that need to be addressed. First, this study is a secondary analysis of data from a randomized controlled trial, focusing on increasing PA; however, for the present analyses, the two groups were merged and a cohort analysis was performed. Thus, the extent to which causality can be inferred is less than that for a randomized controlled trial. However, the precise measure of exposure and outcome variables and the possibility to control for changes in waist circumference and other possible confounders reduce the likelihood that our observations are explained by chance or bias. Second, we cannot rule out the possibility that our observations are explained by unmeasured confounders such as dietary intake and pharmaceutical drugs (medicine). Television viewing is positively associated with intake of high-energy snack foods (29); therefore, those who reduced their sedentary time (including television time) could also have reduced their intake of these snacks more than those who did not reduce their sedentary time. Finally, care should be taken when trying to apply the results to other populations that differ in PA level, glucose and insulin levels, and ethnic background. This is due to the physiological differences between Caucasians and South Asians; for example, Southeast Asians have greater tendency to deposit intraabdominal fat (25) and less muscle mass in the lower extremities (9), which may explain the higher prevalence of insulin resistance among South Asians. Underlying ethnic differences in the physiological responses to PA might also be considered. For example, the association between exercise capacity and all-cause mortality has been shown to be lower in African-Americans compared with that in Caucasians, and pedometer studies suggest that the association between PA and adiposity is weaker in Japanese populations than that in Caucasians (8,21,26).

The observed association between sedentary time and postprandial glucose, insulin, and C-peptide may have important clinical implications. In response to prolonged insulin resistance and the resultant hyperinsulinemia, progressive beta cell failure may occur, which in turn can lead to hyperglycemia and, finally, type 2 diabetes (3). Reductions in 2-h insulin levels, induced by change in sedentary time, may reduce the constraint on the pancreatic beta cells. Although fasting insulin is more commonly used as a surrogate measure of insulin resistance because of the high correlation with the euglycemic clamp technique (22), it may be criticized because it reflects insulin action in an unstimulated or basal state, whereas in everyday life, the insulin action is postprandial in most part. A reduction in sedentary time by 1 $h \cdot d^{-1}$ could theoretically reduce postprandial glucose levels by 0.36 $mmol \cdot L^{-1}$ (≈5%). To put in perspective, data from the Finnish Diabetes Prevention Study showed that an intensive lifestyle intervention reduced the 2-h glucose levels by an average of six percentage points in the intervention group compared with that in controls and reduced their risk of developing diabetes by 58% (32). Furthermore, two meta-analyses provided evidence that hyperglycemia in the nondiabetic range is associated with increased risk of cardiovascular morbidity and mortality and that cardiovascular disease events increased linearly with postprandial glucose levels (10,23), suggesting that any reduction in postprandial glucose levels seems beneficial for metabolic health.

The mechanisms for the independent role of time spent sedentary on health is not known, but research on animals (7,15,16,36) has suggested that fewer muscular contractions may result in reduced lipoprotein lipase activity and clearance of blood lipids and glucose and less insulin secretion. Sedentary behavior may also be an independent risk factor for type 2 diabetes and cardiovascular diseases simply on the basis of low energy expenditure, resulting in overweight or obesity (27). Furthermore, an acute bout of

light-intensity PA has been shown to blunt the rise in postprandial plasma glucose (20) and insulin concentrations (1). Reducing sedentary time and substituting this with time spent in light- and moderate-intensity PA could thus improve CHO metabolism and increase energy expenditure.

In summary, our results suggest that increasing overall PA by reducing sedentary time rather than increasing time spent in MVPA is associated with beneficial changes in postprandial plasma insulin, C-peptide, and glucose concentrations in a diabetes-prone immigrant population. Importantly, these associations were independent of change in abdominal obesity. Substituting sedentary time with time in light-intensity PA (e.g., slow walk) and thereby increasing overall PA energy expenditure may be a preventive strategy for high- or very high-risk individuals who may lack motivation for increasing MVPA.

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The authors have no conflict of interest to disclose.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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