



Breaks in Sedentary Time

Beneficial associations with metabolic risk

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OBJECTIVE — Total sedentary (absence of whole-body movement) time is associated with obesity, abnormal glucose metabolism, and the metabolic syndrome. In addition to the effects of total sedentary time, the manner in which it is accumulated may also be important. We examined the association of breaks in objectively measured sedentary time with biological markers of metabolic risk.

RESEARCH DESIGN AND METHODS — Participants ($n = 168$, mean age 53.4 years) for this cross-sectional study were recruited from the 2004–2005 Australian Diabetes, Obesity and Lifestyle study. Sedentary time was measured by an accelerometer (counts/minute⁻¹ < 100) worn during waking hours for seven consecutive days. Each interruption in sedentary time (counts/min ≥ 100) was considered a break. Fasting plasma glucose, 2-h plasma glucose, serum triglycerides, HDL cholesterol, weight, height, waist circumference, and resting blood pressure were measured. MatLab was used to derive the breaks variable; SPSS was used for the statistical analysis.

RESULTS — Independent of total sedentary time and moderate-to-vigorous intensity activity time, increased breaks in sedentary time were beneficially associated with waist circumference (standardized $\beta = -0.16$, 95% CI -0.31 to -0.02 , $P = 0.026$), BMI ($\beta = -0.19$, -0.35 to -0.02 , $P = 0.026$), triglycerides ($\beta = -0.18$, -0.34 to -0.02 , $P = 0.029$), and 2-h plasma glucose ($\beta = -0.18$, -0.34 to -0.02 , $P = 0.025$).

CONCLUSIONS — This study provides evidence of the importance of avoiding prolonged uninterrupted periods of sedentary (primarily sitting) time. These findings suggest new public health recommendations regarding breaking up sedentary time that are complementary to those for physical activity.

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The world is in the grip of a diabetes and obesity epidemic (1). The reasons for this are complex, but modern lifestyles that allow the avoidance of physical activity are thought to have played a major role (2). In response, public health recommendations on participation in moderate-intensity physical activity have been widely promulgated, with the aim of reducing risk of type 2 diabetes, cardiovascular disease, and some cancers (3). However, there is

emerging evidence that another set of behaviors, involving prolonged periods of inactivity and absence of whole body movement, is distinctly related to risk of chronic disease independent of physical activity (4). Self-reported sedentary time (particularly television-viewing time and total sitting time) is associated with obesity, abnormal glucose metabolism, and the metabolic syndrome (5–10). Similarly, three recent studies using objective sedentary time measures have shown sig-

nificant associations with metabolic markers (11–13).

In addition to the effects of total sedentary time, the manner in which it is accumulated may also be important. Single bouts of prolonged inactivity, such as days and weeks of bed rest, decrease insulin sensitivity in healthy humans (14,15), and animal studies have shown rapid alterations in biological pathways affecting lipid metabolism following a single bout of prolonged, uninterrupted sedentary time (16–18). Such extreme prolonged sedentary behavior is rare in free-living healthy adults, but technological and social factors have made prolonged sitting ubiquitous during working, domestic, and recreational time. Occupational and leisure-time factors, as well as inherent individual differences, contribute to variations in how sedentary time is accumulated. Given the strong epidemiological evidence on the deleterious effects of total sedentary time and the experimental evidence on the acute metabolic effects of prolonged sedentary time, there is a need to better understand how the patterns by which free-living sedentary time is accumulated may be associated with metabolic risk.

In Australian adults without diagnosed diabetes, we examined the associations of breaks in objectively assessed sedentary time with adiposity, lipid, blood pressure, and glucose measures. It was hypothesized that, independent of total sedentary time, more frequent breaks in sedentary time would be associated with more favorable metabolic attributes.

RESEARCH DESIGN AND METHODS

Detailed methods for this cross-sectional, observational study have previously been published (11). In brief, participants were recruited during October–December 2005 from five testing sites of the population-based Australian Diabetes, Obesity and Lifestyle (AusDiab) Study (19,20). AusDiab is the largest Australian longitudinal, population-based study to examine the natural history of diabetes, pre-diabetes, heart disease, and kidney disease. Those with diagnosed diabetes, with visible limitations to mobility, and pregnant women

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Abbreviations: AusDiab, Australian Diabetes, Obesity and Lifestyle.

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were ineligible. Of those available and eligible, all were approached, with the recruitment rate exceeding 80% at each site. Ethics approval was obtained from the International Diabetes Institute; each participant provided written informed consent.

Metabolic, demographic, and behavioral measures

On the day of recruitment, participants underwent biochemical, anthropometric, and behavioral assessments, including an oral glucose tolerance test, as part of the larger set of AusDiab survey procedures (19,20). Fasting and 2-h plasma glucose levels were determined by spectrophotometric-hexokinase methods; fasting serum triglycerides and HDL cholesterol levels were measured by enzymatic methods (Roche Modular, Roche Diagnostics, Indianapolis, IN). Demographic and behavioral attributes were assessed using questionnaires administered by trained interviewers using standard protocols described in earlier papers (19,21). Duplicate waist circumference and triplicate resting blood pressure measurements were conducted by trained personnel. The self-administered validated food frequency questionnaire was used to derive a diet quality score (Diet Quality Index-Revised, scale 1–100 with 100 being high diet quality) with under- and overreporters excluded from the analyses (22–25).

A uniaxial accelerometer (Actigraph model WAM 7164; <http://www.theactigraph.com/>), fitted firmly around the trunk of the participant and placed on the right anterior axillary line was used to measure physical activity. Participants were instructed to wear the accelerometer during all waking hours for a continuous period of seven days, and also to record activity duration, type, and intensity during nonwearing/nonsleep periods. A physical activity diary supplemented the accelerometer data by recording nonambulatory activities, as well as on/off times of the accelerometer. A criterion of at least 20 min of continuous zero counts, as well as diary information, identified nonwearing periods. For their data to be included in the analyses, participants were required to have worn the accelerometer for at least five valid days, including at least one weekend day, where a valid day was at least 10 h of recorded activity (based on both accelerometer and diary data). The majority of the 168 participants (65 men, 103 women) who met the eligibility criteria had seven days of data (80%), with six

(4%) having only five days of valid physical activity data.

Accelerometer data reduction

Accelerometer data were recorded in 1-min epochs. Consistent with previous research (11,12,26), a pragmatic cutoff of <100 counts/min was chosen to classify sedentary time, which typically includes activities such as sitting or working quietly (e.g., reading, typing). Each minute that the accelerometer counts were <100 was considered sedentary time; total sedentary time was the sum of sedentary minutes while the accelerometer was worn. A break was considered as an interruption in sedentary time (minimum 1 min) in which the accelerometer count rose up to or above 100 counts/min. Although the activities that produce accelerometer counts per minute of at least 100 are likely to be different for each individual, they may include activities as light in intensity as standing from a sitting position or walking a step. Mean intensity (reported as accelerometer counts/min) and duration of the breaks were also reported. Accelerometer counts ≥ 100 per minute were classified as active time, with further differentiation to identify separately moderate- to vigorous-intensity activity (≥ 1952 counts/min), and light-intensity activity (100 to 1951 counts/min) (27).

Statistical analysis

Forced-entry linear regression models examined the associations of breaks in sedentary time with metabolic risk variables: waist circumference, BMI, serum triglycerides (log transformed to account for high kurtosis), HDL cholesterol, systolic blood pressure, diastolic blood pressure, fasting plasma glucose, and 2-h plasma glucose. As no significant sex interactions were observed, the data for men and women were pooled. Models were adjusted for potential confounders including age (years), sex, alcohol intake (self-reported as none, light, and moderate-to-heavy), employment status (full time, yes/no), education (attended university or further education, yes/no), income (household income $\geq \$1,500$ /week, yes/no), smoking status (current or ex/nonsmoker), family history of diabetes, diet quality, moderate- to vigorous-intensity time (average h/day from both accelerometer and diary data), mean intensity of the breaks, and total sedentary time (hours). Those on hypertensive ($n = 29$) and/or lipid-lowering ($n = 23$) treat-

ment were included in the analysis with treatment considered a covariate. Waist circumference was then included in the model to examine the potential mediating effect of central obesity on the associations. In order to pictorially describe the effect size of the associations, the breaks in sedentary time variable were also categorized into quartiles of breaks in sedentary time. Complete data were available for all variables. Statistical significance was set at $P < 0.05$. The sedentary breaks variable was derived from Matlab version 7.1.0.124 (The Mathworks, Inc.); statistical analyses were conducted using SPSS version 13 (SPSS, Inc. Chicago, IL).

RESULTS — The age range of the participants was 30 to 87 years and the majority (98.8%) spoke English at home. On average, participants spent 57% of their waking hours sedentary, 39% in light-intensity activity, and 4% in moderate- to vigorous-intensity time. The bivariate correlation of total breaks with total sedentary time was moderately low (Pearson $R = 0.20$).

Table 1 reports the sociodemographic, metabolic, and sedentary time-use characteristics of the participants. The number of total breaks reported in Table 1 was the mean total number of breaks across the entire data-collection period. The average intensity of the breaks was 514 counts/min, which is in the light-intensity range (11), while the average duration of the breaks was less than 5 min. Compared to the broader 2004–2005 AusDiab Study population with the same exclusion criteria ($n = 5,449$), participants in this sub-study were younger (53.4 vs. 56.1 years, $P = 0.007$) and had lower systolic blood pressure (120 vs. 123 mmHg, $P = 0.027$), log triglycerides (0.05 vs. 0.10 mmol/L, $P = 0.002$), and fasting plasma glucose (5.21 vs. 5.34 mmol/L, $P = 0.006$). No other statistically significant differences in either the behavioral measures (self-reported physical activity or television viewing time) or the metabolic risk variables were observed.

Table 2 reports the standardized regression coefficients of breaks in sedentary time with metabolic risk. As both the independent (breaks) and dependent (metabolic risk) variables are expressed in the same unit (standard deviations), it is possible to directly compare the magnitude of the effect for the different risk variables. Overall, independent of total sedentary time, the total number of breaks in sedentary time was associated

Table 1—Sociodemographic, metabolic, and sedentary time-use characteristics of participants

Characteristic	
Age (years)	53.4 ± 11.8
Family history of diabetes	22.6
Current smokers	1.8
University/further education	54.2
Moderate/heavy alcohol drinkers	31.6
Full-time employment	54.2
Household income ≥\$1,500 /week	38.1
Diet quality (scale 1–100)	68.8 ± 12.2
Metabolic variables	
Waist circumference (cm)	91.3 ± 12.6
BMI (kg/m ²)	27.2 ± 4.7
Log triglycerides (mmol/l)*	0.05 ± 0.21
HDL cholesterol (mmol/l)*	1.49 ± 0.40
Systolic blood pressure (mmHg)†	120 ± 17
Diastolic blood pressure (mmHg)†	68.2 ± 9.7
Fasting plasma glucose (mmol/l)	5.21 ± 0.49
2-h plasma glucose (mmol/l)	5.72 ± 1.67
Accelerometer-derived variables	
Total sedentary time (h)	56.7 ± 12.1
Moderate- to vigorous-intensity time (h/day)	0.61 ± 0.38
Total breaks (n)	601 ± 115
Intensity of break (accelerometer counts/min)	514 ± 94
Duration of break (min)	4.50 ± 1.05

Data are means ± SD or %. *Means adjusted for lipid-lowering medication (*n* = 23); †means adjusted for hypertensive medication (*n* = 29). Sedentary time (<100 accelerometer counts/min); moderate- to vigorous-intensity time (≥1,952 accelerometer counts/min and diary information).

with significantly lower waist circumference, BMI, triglycerides, and 2-h plasma glucose (Table 2). The associations with triglycerides (β = −0.13, 95%CI −0.29 to 0.03, *P* = 0.097) and 2-h plasma glucose (β = −0.14, 95%CI −0.29 to 0.02, *P* = 0.082) were attenuated when waist circumference was included in the model. To provide a visual representation of the effect size of the associations, Fig. 1 shows the estimated marginal means for the associations of quartiles of breaks in sedentary time with waist circumference, BMI, triglycerides, and 2-h plasma glucose. Compared to those in the lowest quartile of breaks in sedentary time, those in the highest quartile had, on average, a 5.95 cm lower waist circumference (*P* = 0.025) and a 0.88 mmol/L lower 2-h plasma glucose (*P* = 0.019).

CONCLUSIONS— Total sedentary time, both self-reported and objectively-assessed, has been shown to be detrimentally associated with health outcomes and with markers of metabolic risk across diverse population groups (5–13). We report the first study to examine the associations of breaks in objectively-assessed sedentary time with metabolic outcomes. We found that independent of

total sedentary time, moderate-to-vigorous intensity time, and mean intensity of the breaks, more interruptions in sedentary time were beneficially associated with metabolic risk variables, particularly adiposity measures, triglycerides, and 2-h plasma glucose. These findings suggest that it is not only the amount of sedentary time that is important, but also the manner in which it is accumulated.

These findings have important methodological and public health implications. As sedentary time comprises a large proportion of waking hours (>50% for

the majority of participants), small but important changes in sedentary behavior may go undetected using total sedentary time measures—particularly when self-report measures are used. We have described an objective method that may provide additional insight into important nuances of how adults' metabolic health is determined by how they spend time in sedentary and physically active behaviors. The accelerometer also enables assessment of difficult-to-capture light-intensity physical activity, which is typically incidental in nature. As beneficial metabolic associations were observed with breaks that were relatively short in duration and light in intensity, advice to regularly break up or interrupt sustained sedentary time may be feasible to implement across numerous settings, including the workplace. In practice, this might be as simple as getting up during television advertising breaks, or taking brief ambulatory breaks during prolonged periods of sitting at work.

Mechanisms underlying these findings are likely to be complex. Insights may be gained from experimental studies examining the acute effects of prolonged inactivity. However, given the ethical issues associated with these studies, including the possibility of deep vein thrombosis, the majority of work has been conducted using animal models. Time-course studies in animals have reported substantial decreases in lipoprotein lipase activity and impaired triglyceride and HDL cholesterol metabolism measured after prolonged inactivity (4,16–18). As skeletal muscle is one of the major sites for the clearance of both plasma triglycerides (16) and an oral glucose load from plasma (28), one potential mechanism for these detrimental changes is the absence of skeletal muscle contractions.

Table 2—Standardized regression coefficients of total breaks with metabolic risk variables

Metabolic risk variable	β (95% CI)	<i>P</i>	Adjusted <i>R</i> ²
Waist circumference (cm)	−0.16 (−0.31 to −0.02)	0.027	0.26
BMI (kg/m ²)	−0.19 (−0.35 to −0.02)	0.026	0.05
Triglycerides (log) (mmol/l)*	−0.18 (−0.34 to −0.02)	0.029	0.13
HDL cholesterol (mmol/l)*	0.03 (−0.12 to 0.18)	0.685	0.25
Systolic blood pressure (mmHg)†	−0.03 (−0.18 to 0.12)	0.697	0.21
Diastolic blood pressure (mmHg)†	0.03 (−0.12 to 0.19)	0.670	0.19
Fasting plasma glucose (mmol/l)	−0.09 (−0.25 to 0.07)	0.287	0.09
2-h plasma glucose (mmol/l)	−0.18 (−0.34 to −0.02)	0.025	0.14

Adjusted for age, sex, employment, alcohol intake, income, education, smoking, family history of diabetes, diet quality, moderate- to vigorous-intensity time, mean intensity of breaks, and total sedentary time.

*Additional adjustment for lipid-lowering medication; †additional adjustment for hypertensive medication. Adjusted *R*² gives an estimate of explained variance, taking into account the sample size.

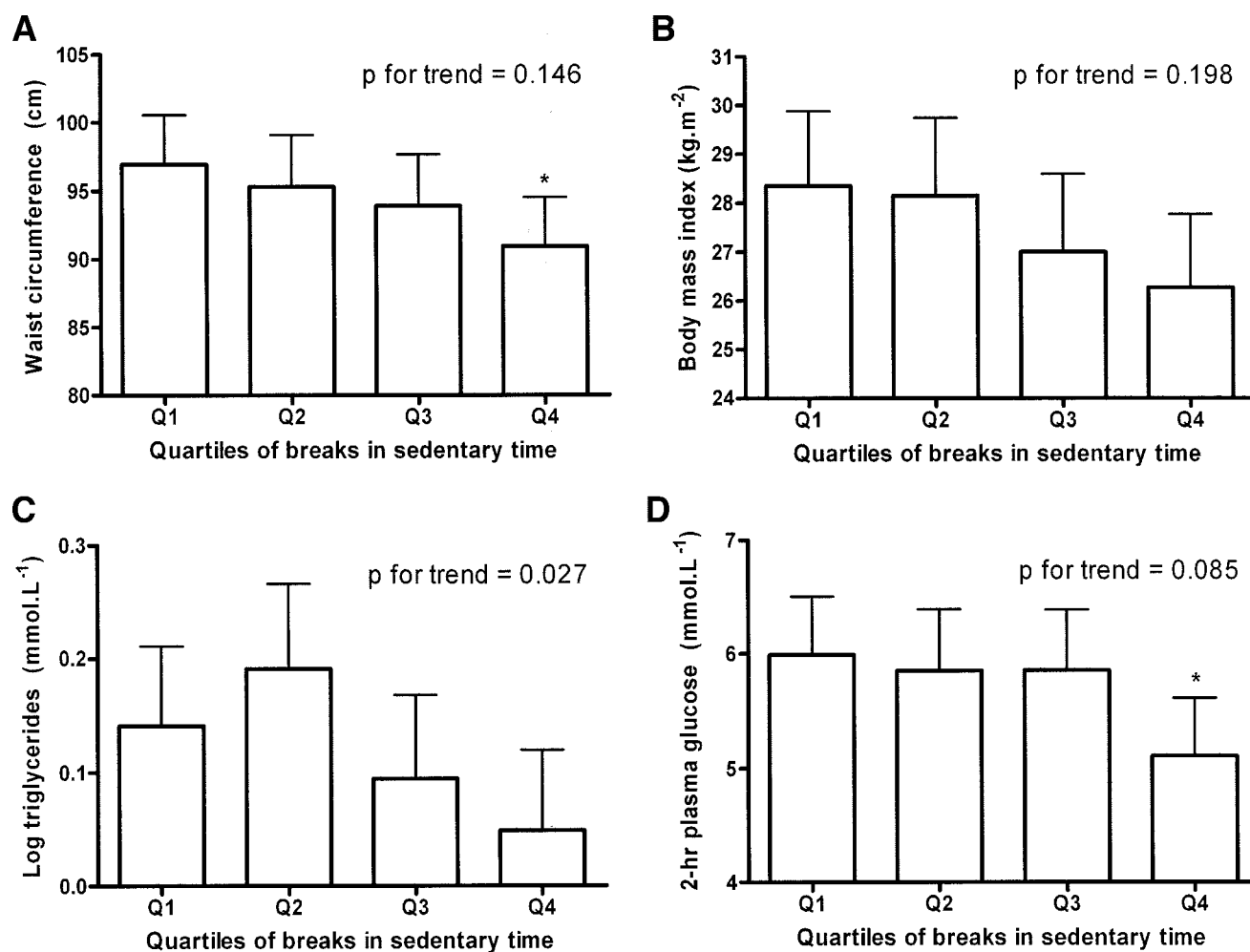


Figure 1—Quartiles of breaks in sedentary time with metabolic risk variables: waist circumference (A), BMI (B), triglycerides (C), and 2-h plasma glucose (D). Estimated marginal means (SE) adjusted for age, sex, employment, alcohol intake, income, education, smoking, family history of diabetes, diet quality, moderate- to vigorous-intensity time, mean intensity of breaks, and total sedentary time. Triglycerides (log) are additionally adjusted for lipid-lowering medication. Cut points for quartiles were 506, 612, and 673 breaks; * $P < 0.05$ compared to quartile 1.

In rats, plasma triglyceride and HDL cholesterol clearance was impaired locally in leg skeletal muscles during a day of reduced low-intensity standing/spontaneous cage ambulation of movement (16). This was associated with a ~90% reduction in the skeletal muscle lipoprotein lipase activity locally in the legs (16). These detrimental changes were physiologically corrected with light intensity ambulatory activities (16). However, the long term consequences of repeated bouts of prolonged inactivity have not been determined. The increased risk associated with fewer breaks in sedentary time observed in our study may reflect a chronic metabolic adaptation to repeated bouts of prolonged inactivity, though this inference requires further investigation.

The beneficial association of breaks in sedentary time with metabolic markers may also reflect higher total energy ex-

penditure in those with more frequent breaks. Even activities as minimal as standing, rather than sitting, have been shown to result in substantial increases in total daily energy expenditure and resistance to fat gain (29–31). It was not possible to adjust for total energy expenditure, as accelerometers were not worn for 24 h per day. However, we did adjust for the mean intensity of the breaks, as well as total moderate-to-vigorous intensity time.

This study was conducted in a non-clinical sample that was similar to the broader AusDiab Study population, using objective measures of sedentary and physical activity time. Even following adjustment for several potential confounding factors, including total sedentary and moderate-to-vigorous intensity time, the effect sizes of the associations with the metabolic risk markers were clinically sig-

nificant. For example, those in the highest quartile of breaks in sedentary time had, on average, a 5.9 cm lower waist circumference than those in the lowest quartile. However, this is an observational study using cross-sectional data, and further investigations are required to determine possible causal associations. Summarizing accelerometer counts can be problematic. Counts as low as 100 and as short as 1 min were considered as “active” and thus as breaks in sedentary time. Although we can report the average duration and intensity of the breaks, it is not possible to determine in this observational study whether there is a minimum amount of time and/or intensity for interrupting sedentary time that may be required for beneficial metabolic effects. Future experimental research is needed to examine the effects of manipulating interruptions to sedentary time on acute met-

abolic changes. Additionally, given that sleep duration has been associated with metabolic risk (32), future studies on the effects of sedentary time accumulation in free-living populations should also include a measure of sleep duration. In this study, we report the association of breaks in sedentary time with metabolic risk adjusted for total sedentary time. However, although statistically appropriate for assessing the independent association of breaks in sedentary time with metabolic risk, this measure is one that is neither clinically nor intuitively easy to interpret. For the purposes of characterizing how sedentary time is broken up, a composite measure, such as breaks per sedentary hour, may provide more meaningful information.

We provide preliminary evidence that breaking up sedentary time may provide beneficial metabolic effects in addition to the beneficial effects of reducing total sedentary time and increasing time spent in moderate- to vigorous-intensity physical activity. The possible causal nature of these associations, and the biological and behavioral mechanisms that may underlie them, require further investigation. Our findings add to the case for public health recommendations on sedentary behavior and health (33), which are complementary to those for physical activity (3).

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References

1. Naser KA, Gruber A, Thomson GA: The emerging pandemic of obesity and diabetes: are we doing enough to prevent a disaster? *Int J Clin Pract* 60:1093–1097, 2006
2. Zimmet P, Alberti KG, Shaw J: Global and societal implications of the diabetes epidemic. *Nature* 414:782–787, 2001
3. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, Buchner D, Ettinger W, Heath GW, King AC, et al.: Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 273:402–407, 1995
4. Hamilton MT, Hamilton DG, Zderic TW: The role of low energy expenditure and sitting on obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. *Diabetes* 56:2655–2667, 2007
5. Dunstan DW, Salmon J, Owen N, Armstrong T, Zimmet PZ, Welborn TA, Cameron AJ, Dwyer T, Jolley D, Shaw JE: Physical activity and television viewing in relation to risk of undiagnosed abnormal glucose metabolism in adults. *Diabetes Care* 27:2603–2609, 2004
6. Dunstan DW, Salmon J, Healy GN, Shaw JE, Jolley D, Zimmet PZ, Owen N: Association of television viewing with fasting and 2-hr post-challenge plasma glucose

levels in adults without diagnosed diabetes. *Diabetes Care* 30:516–522, 2007

7. Dunstan DW, Salmon J, Owen N, Armstrong T, Zimmet PZ, Welborn TA, Cameron AJ, Dwyer T, Jolley D, Shaw JE: Associations of TV viewing and physical activity with the metabolic syndrome in Australian adults. *Diabetologia* 48:2254–2261, 2005
8. Foster JA, Gore SA, West DS: Altering TV viewing habits: an unexplored strategy for adult obesity intervention? *Am J Health Behav* 30:3–14, 2006
9. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE: Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA* 289:1785–1791, 2003
10. Jakes RW, Day NE, Khaw KT, Luben R, Oakes S, Welch A, Bingham S, Wareham NJ: Television viewing and low participation in vigorous recreation are independently associated with obesity and markers of cardiovascular disease risk: EPIC-Norfolk population-based study. *Eur J Clin Nutr* 57:1089–1096, 2003
11. Healy GN, Dunstan DW, Salmon J, Cerin E, Shaw JE, Zimmet PZ, Owen N: Objectively measured light-intensity physical activity is independently associated with 2-h plasma glucose. *Diabetes Care* 30:1384–1389, 2007
12. Ekelund U, Griffin SJ, Wareham NJ: Physical activity and metabolic risk in individuals with a family history of type 2 diabetes. *Diabetes Care* 30:337–342, 2007
13. Healy GN, Wijndaele K, Dunstan DW, Shaw JE, Salmon J, Zimmet PZ, Owen N: Objectively measured sedentary time, physical activity, and metabolic risk: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). 31:369–371
14. Jennings G, Nelson L, Nestel P, Esler M, Korner P, Burton D, Bazelmans J: The effects of changes in physical activity on major cardiovascular risk factors, hemodynamics, sympathetic function, and glucose utilization in man: a controlled study of four levels of activity. *Circulation* 73:30–40, 1986
15. Lipman RL, Raskin P, Love T, Triebwasser J, Lecocq FR, Schnure JJ: Glucose intolerance during decreased physical activity in man. *Diabetes* 21:101–107, 1972
16. Bey L, Hamilton MT: Suppression of skeletal muscle lipoprotein lipase activity during physical inactivity: a molecular reason to maintain daily low-intensity activity. *J Physiol* 551:673–682, 2003
17. Hamilton MT, Etienne J, McClure WC, Pavey BS, Holloway AK: Role of local contractile activity and muscle fiber type on LPL regulation during exercise. *Am J Physiol* 275:E1016–E1022, 1998
18. Zderic TW, Hamilton MT: Physical inactivity amplifies the sensitivity of skeletal

- muscle to the lipid-induced downregulation of lipoprotein lipase activity. *J Appl Physiol* 100:249–257, 2006
19. Dunstan DW, Zimmet PZ, Welborn TA, Cameron AJ, Shaw J, de Courten M, Jolley D, McCarty DJ: The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)—methods and response rates. *Diabetes Res Clin Pract* 57:119–129, 2002
20. Barr ELM, Magliano DJ, Zimmet PZ, Polkinghorne KR, Atkins RC, Dunstan DW, Murray SG, Shaw JE: AusDiab 2005: The Australian Diabetes, Obesity and Lifestyle Study. Tracking the Accelerating Epidemic: its Causes and Outcomes [article online], 2006. Melbourne, Australia, International Diabetes Institute. Available from http://diabetes.com.au/pdf/AUSDIAB_Report_Final.pdf. Accessed 11 June 2007
21. Dunstan DW, Zimmet PZ, Welborn TA, De Courten MP, Cameron AJ, Sicree RA, Dwyer T, Colagiuri S, Jolley D, Knuiman M, Atkins R, Shaw JE: The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study. *Diabetes Care* 25: 829–834, 2002
22. Ireland P, Jolley D, Giles D, O'Dea K, Powles J, Rautishauser I, Wahlqvist ML, Williams J: Development of the Melbourne FFQ: a food frequency questionnaire for use in an Australian prospective study involving an ethnically diverse cohort. *Asia Pacific J Clin Nutr* 3:19–31, 1994
23. Haines PS, Siega-Riz AM, Popkin BM: The Diet Quality Index revised: a measurement instrument for populations. *J Am Diet Assoc* 99:697–704, 1999
24. Newby PK, Hu FB, Rimm EB, Smith-Warner SA, Feskanich D, Sampson L, Willett WC: Reproducibility and validity of the Diet Quality Index Revised as assessed by use of a food-frequency questionnaire. *Am J Clin Nutr* 78:941–949, 2003
25. Willett W: *Nutritional Epidemiology*. New York, Oxford University Press, 1998
26. Craig CL, Marshall AL, Sjostrom M, Bauman AE, Booth ML, Ainsworth BE, Pratt M, Ekelund U, Yngve A, Sallis JF, Oja P: International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc* 35:1381–1395, 2003
27. Freedson PS, Melanson E, Sirard J: Calibration of the Computer Science and Applications, Inc. accelerometer. *Med Sci Sports Exerc* 30:777–781, 1998
28. DeFronzo RA, Bonadonna RC, Ferrannini E: Pathogenesis of NIDDM: a balanced review. *Diabetes Care* 15:318–368, 1992
29. Levine JA: Nonexercise activity thermogenesis (NEAT): environment and biology. *Am J Physiol Endocrinol Metab* 286: E675–E685, 2004
30. Levine JA, Eberhardt NL, Jensen MD: Role of nonexercise activity thermogenesis in resistance to fat gain in humans. *Science* 283:212–214, 1999
31. Levine JA, Lanningham-Foster LM, McCrady SK, Krizan AC, Olson LR, Kane PH, Jensen MD, Clark MM: Interindividual variation in posture allocation: possible role in human obesity. *Science* 307:584–586, 2005
32. Bjorvatn B, Sagen IM, Oyane N, Waage S, Fetveit A, Pallesen S, Ursin R: The association between sleep duration, body mass index and metabolic measures in the Hordaland Health Study. *J Sleep Res* 16: 66–76, 2007
33. Owen N, Leslie E, Salmon J, Fotheringham MJ: Environmental determinants of physical activity and sedentary behavior. *Exerc Sport Sci Rev* 28:153–158, 2000