

Association of Television Viewing With Fasting and 2-h Postchallenge Plasma Glucose Levels in Adults Without Diagnosed Diabetes

DAVID W. DUNSTAN, PHD¹
JO SALMON, PHD²
GENEVIEVE N. HEALY, MPH³
JONATHAN E. SHAW, MD¹
DAMIEN JOLLEY, MSC⁴

PAUL Z. ZIMMET, MD¹
NEVILLE OWEN, PHD³
ON BEHALF OF THE AUSDIAB STEERING
COMMITTEE

OBJECTIVE — We examined the associations of television viewing time with fasting plasma glucose (FPG) and 2-h postchallenge plasma glucose (2-h PG) levels in Australian adults.

RESEARCH DESIGN AND METHODS — A total of 8,357 adults aged >35 years who were free from diagnosed diabetes and who attended a population-based cross-sectional study (Australian Diabetes, Obesity and Lifestyle Study [AusDiab]) were evaluated. Measures of FPG and 2-h PG were obtained from an oral glucose tolerance test. Self-reported television viewing time (in the previous week) was assessed using an interviewer-administered questionnaire. Homeostasis model assessment (HOMA) of insulin sensitivity (HOMA-%S) and β -cell function (HOMA-%B) were calculated based on fasting glucose and insulin concentrations.

RESULTS — After adjustment for confounders and physical activity time, time spent watching television in women was positively associated with 2-h PG, log fasting insulin, and log HOMA-%B and inversely associated with log HOMA-%S ($P < 0.05$) but not with FPG. No significant associations were observed with glycemic measures in men. The β -coefficients across categories of average hours spent watching television per day (<1.0, 1.0–1.9, 2.0–2.9, 3.0–3.9, and ≥ 4.0) for 2-h PG in women were 0 (reference), 0.009, 0.047, 0.473, and 0.501, respectively (P for trend = 0.02).

CONCLUSIONS — Our findings highlight the unique deleterious relationship of sedentary behavior (indicated by television viewing time) and glycemic measures independent of physical activity time and adiposity status. These relationships differed according to sex and type of glucose measurement, with the 2-h PG measure being more strongly associated with television viewing. The findings suggest an important role for reducing sedentary behavior in the prevention of type 2 diabetes and cardiovascular disease, especially in women.

Diabetes Care 30:516–522, 2007

Physical inactivity increases the risk of many chronic diseases, including type 2 diabetes (1). Recently, several studies have shown that sedentary behav-

ior, as distinct from lack of physical activity (2), may be an important contributor to poor health outcomes (3–8). Television viewing time, which is a major lei-

sure-time sedentary behavior in Australia (9), has been linked to obesity and to type 2 diabetes in adults, independent of physical activity levels (7,8). In addition, our previous work has shown independent positive associations between television viewing time and categories of glucose intolerance. However, since fasting plasma glucose (FPG) and 2-h postchallenge plasma glucose (2-h PG) differ from each other in their respective physiological determinants (10) and in the risks that they carry (11), there is a need to investigate the influence of television viewing on plasma glucose across the glucose continuum from normal to diabetes levels. While some cross-sectional studies have found that television viewing time is positively associated with FPG in adults (3,12), no population-based studies have investigated the extent and strength of the relationship between television viewing time and 2-h PG. Simultaneously studying the relationship between television viewing and both glucose measures is necessary to develop a better understanding of the metabolic pathways through which sedentary behaviors may contribute to an increased risk of diabetes. We examined the associations of plasma glucose measures (FPG and 2-h PG) and fasting insulin with television viewing time, independent of physical activity time and adiposity status, using data from a large population-based study of Australian adults. Furthermore, we examined the dose-response relationships with television viewing time.

RESEARCH DESIGN AND METHODS

The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) was conducted during 1999–2000 using data from a representative national sample of adults (5,6,13). The sample of 11,247 represented 55% of those completing an initial household interview. Insulin assays were conducted only for the population aged >35 years ($n = 9,644$). The present analyses use data from adults (age range 36–91 years) without clini-

From the ¹International Diabetes Institute, Melbourne, Australia; the ²School of Exercise and Nutrition Sciences, Deakin University, Melbourne, Australia; the ³Cancer Prevention Research Centre, School of Population Health, The University of Queensland, Brisbane, Australia; and the ⁴Monash Institute of Health Services Research, Melbourne, Australia.

Address correspondence and reprint requests to Dr. David Dunstan, International Diabetes Institute, 250 Kooyong Rd., Caulfield, Victoria, Australia 3162. E-mail: ddunstan@idi.org.au.

Received for publication 25 September 2006 and accepted in revised form 9 December 2006.

Abbreviations: 2-h PG, 2-h postchallenge plasma glucose; AusDiab, Australian Diabetes, Obesity and Lifestyle Study; FPG, fasting plasma glucose; HOMA, homeostasis model assessment; HOMA-%B, HOMA of β -cell function; HOMA-%S, HOMA of insulin sensitivity.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

DOI: 10.2337/dc06-1996

© 2007 by the American Diabetes Association.

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

Table 1—Selected characteristics of the study population

	Men	Women
<i>n</i>	3,781	4,576
Age (years)	52.7 (51.7–53.7)	54.1 (52.3–55.9)*
BMI (kg/m ²)	27.0 (26.8–27.3)	26.7 (26.2–27.1)
Waist (cm)	96.8 (95.8–97.8)	85.0 (83.3–86.6)*
Height (m)	1.75 (1.74–1.76)	1.61 (1.60–1.62)*
FPG (mmol/l)	5.62 (5.58–5.67)	5.36 (5.31–5.41)*
2-h PG (mmol/l)	6.22 (6.04–6.40)	6.43 (6.23–6.62)
Fasting serum insulin (pmol/l)	90.0 (88.6–91.4)	85.8 (84.7–87.0)*
HOMA-%S	58.6 (57.7–59.5)	62.0 (61.2–62.9)*
HOMA-%B	109.4 (108.2–110.5)	116.4 (115.4–117.5)*
TV viewing time (h/day)	1.90 (1.77–2.04)	1.71 (1.61–1.81)*
Total physical activity time (h/day)	0.75 (0.69–0.81)	0.53 (0.48–0.58)*
Parental history of diabetes (%)	17.0 (15.5–18.6)	18.4 (16.8–20.1)
Current smoker (%)	16.5 (12.4–20.6)	11.5 (9.4–13.5)*
University/further education (%)	46.5 (41.7–51.4)	34.4 (29.3–39.5)*
Normal glucose tolerance (%)	72.0 (68.5–75.4)	77.4 (74.0–80.8)*
Isolated IFG (%)	10.3 (8.5–12.2)	4.1 (2.7–5.4)*
Isolated IGT (%)	8.7 (7.1–10.2)	11.5 (9.9–13.1)*
Combined IFG/IGT (%)	4.3 (2.9–5.7)	2.5 (1.7–3.3)*
Undiagnosed diabetes (%)	4.7 (3.3–6.1)	4.6 (3.2–5.9)

Data are means (95% CI). Values are weighted to the Australian population, with unadjusted means and percentages. Geometric means are reported for fasting insulin, HOMA-%S, and HOMA-%B. Statistical comparisons are age adjusted. **P* < 0.05 for comparison of men and women. IFG, impaired fasting glucose; IGT, impaired glucose tolerance; TV, television.

cally diagnosed diabetes who did not have missing values for FPG, 2-h PG, fasting insulin, or television viewing time. This included 3,781 men and 4,576 women comprising 8,357 adults. The Ethics Committee of the International Diabetes Institute approved the study. Informed consent was obtained from all participants.

Participants attended the local survey center after an overnight fast (minimum 8 h). The specific measurement procedures (for waist circumference, height, and weight) have been described in earlier publications (13). A 2-h oral glucose tolerance test was performed, with blood specimens centrifuged on-site and transported daily to the central laboratory. Serum samples for insulin were stored at –80°C until assayed. Plasma glucose levels were determined using an Olympus AU600 automated analyzer. Serum insulin was measured using a human insulin-specific radioimmunoassay kit (Linco Research, St. Charles, MO). We also used the homeostatis model assessment (HOMA) computer model to explore the relationships between television viewing and insulin resistance and insulin secretion as potential mechanisms for relationships with blood glucose. This model, which estimates insulin sensitivity (HOMA-%S) and β -cell function (HOMA-%B) from fasting insulin and

glucose concentrations, has been used extensively in epidemiologic studies (14).

Demographic attributes, parental history of diabetes, smoking habits, level of educational attainment (categorized as primary/never attended, some secondary, completed secondary, and university/further education), physical activity (hours/day), and television viewing time were assessed using an interviewer-administered questionnaire. Participants reported their frequency and duration of leisure-time physical activity during the previous week, consisting of walking for recreation or transport, other moderate activity, and vigorous activity (15). They also reported the total time spent watching television or videos in the previous week. This measure has been shown to provide a reliable valid estimate of television time among adults (16). Dietary intake (usual eating habits over the past 12 months) was assessed using a self-administered validated food frequency questionnaire developed by the Anti-Cancer Council of Victoria (17). Calculation of nutrient intake was achieved by multiplying the frequency of food consumption by standard portion weights to obtain weight of food consumed per day. These were then converted to nutrient intakes based on the NUTTAB95 nutrient composition data (18).

Participants reported total time spent watching television or videos in the past week, using a previously validated instrument (16). The total hours of television viewing time per week were divided by seven and used to create five categories of television viewing time (<1.0, 1.0–1.9, 2.0–2.9, 3.0–3.9, and ≥ 4 h/day).

Statistical analysis

Statistical analyses were conducted using Stata Statistical Software release 9.0 (19) survey commands for analyzing complex survey data. Sample weights based on the 1998 estimated residential Australian population aged >35 years were used to account for clustering and stratification in the survey design and for nonresponse. Continuous variables were compared between men and women using linear regression models adjusted for age. Categorical variables were compared between the sexes using age-adjusted logistic regression models. Forced entry multivariable linear regression models were used to examine associations between television viewing and glycemic variables (continuous variables). Separate models were applied for glycemic variables (FPG, 2-h PG, fasting insulin, HOMA-%S, and HOMA-%B) for men and women. The adjusted mean difference (regression coefficient) relative to the reference category (television <1 h/week) was generated for the television viewing categories. We also assessed differences in the effects of television viewing by age and sex using interaction terms in linear regression models (after pooling sex-specific datasets). For fasting plasma insulin and HOMA variables, the natural logarithm was used to correct for skewness in the data.

To control for potential confounding, we adjusted the models for age, height, waist, and dietary covariates (total energy intake, total fat intake, total saturated fat intake, total carbohydrate intake, total sugar intake, total fiber intake, total alcohol intake, and total physical activity) as continuous variables and current smoking status, parental history of diabetes, and university/further education as dichotomous variables. A criterion of *P* < 0.05 was used for statistical significance.

RESULTS— Table 1 shows the sex-stratified characteristics of the 8,357 study participants who were free from clinically diagnosed diabetes. Women were older, spent less time watching television, and were significantly less physi-

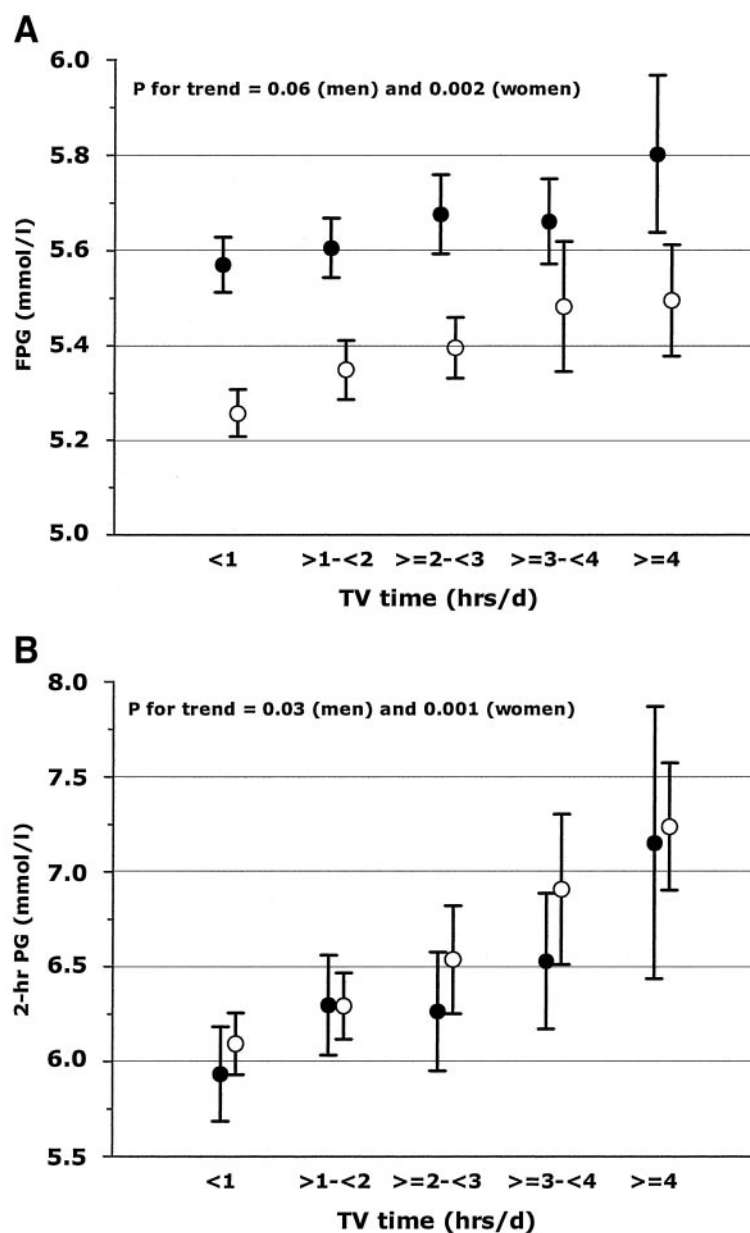


Figure 1—Age-adjusted means (95% CI) for fasting plasma glucose (A) and 2-h PG (B) according to television (TV) watching categories in men (●) and women (○).

cally active than men. FPG and serum insulin levels were lower in women, while there was no significant difference between the sexes for 2-h PG.

The age-adjusted means of FPG and 2-h PG are presented for the five categories for television viewing time in Fig. 1. After adjustment for age, we found a significant positive association between television viewing and FPG in women ($P = 0.002$) and a positive association that approached statistical significance in men ($P = 0.06$). A positive association between television viewing and 2-h PG was observed in both men ($P = 0.03$) and women ($P = 0.001$). In age-adjusted re-

gression models, each 1 h/day increase in television time increased FPG by 0.02 mmol/l (95% CI 0.001–0.04, $P = 0.04$) in men and 0.04 mmol/l (0.02–0.06, $P = 0.001$) in women, while the increase in 2-h PG was 0.11 mmol/l (–0.002 to 0.23, $P = 0.06$) and 0.16 mmol/l (0.08–0.25, $P = 0.001$) in men and women, respectively.

Using multiple regression analysis, we estimated the independent associations across categories of television viewing time for glycemic measures (Table 2). All models were adjusted for age, waist circumference, height, education, smoking, parental history of diabetes, physical activ-

ity, and dietary covariates. In women, television viewing time had a significant positive association with 2-h PG, such that the mean 2-h PG was 0.5 mmol/l higher in those watching >3 h of television per day compared with those watching <1 h. No such relationship was seen with FPG. Television viewing time was also positively associated with log fasting insulin and log HOMA-%B and inversely associated with HOMA-%S in women. In general, a significant increase in the regression coefficients compared with the reference category (<1 h/day) was observed in women who watched >3 h of television per day. Although the positive association with 2-h PG approached statistical significance in men ($P = 0.06$), no significant associations were observed between television viewing time and glycemic variables in men. No significant sex interactions were observed for FPG, 2-h PG, or log HOMA-%B; however, the interaction approached significance for log insulin and log HOMA-%S ($P = 0.06$ for both). No age interactions were observed for any of the glycemic measures.

CONCLUSIONS— Sedentary behavior (particularly television viewing time) has been linked to several adverse health outcomes, including obesity and type 2 diabetes (7,8), abnormal glucose metabolism (5), the metabolic syndrome (3,4,6), high blood pressure (20), and cardiovascular disease (12). Our study extends these previous findings by examining the associations between hours of television watched per day and continuous glycemic variables in adults without diagnosed diabetes. We have shown in a large population-based study that television viewing was positively associated with plasma glucose, independent of physical activity and body habitus, with the 2-h PG having a stronger association with television viewing time than did FPG, in analyses adjusted for potential confounders, including waist circumference. Furthermore, in contrast to men, in whom a modest association was observed with 2-h PG, significant independent associations of television viewing with 2-h PG, insulin sensitivity, and β -cell function were demonstrated in women.

Associations between television viewing time and blood glucose concentrations have not been intensively investigated in cross-sectional studies. In the population-based National Heart, Blood, and Lung Institute Family Heart Study of 1,778 Caucasian adults, signifi-

cant positive associations between television viewing time and FPG levels were observed in both men and women (12). Similar observations were made in another cross-sectional study of middle-aged French adults (3). However, unlike our study, only the univariate associations were reported in these studies. It is important to take into account potential confounders such as weight status when examining associations with glucose metabolism, since overweight not only affects insulin sensitivity, but also can lead to an adaptation of the pancreatic β -cell function (21,22). While we observed significant dose-response relationships between age-adjusted FPG levels in women, which also approached statistical significance in men, these associations were no longer evident once we adjusted for potential confounders, including waist circumference. This finding is consistent with the observations made among young adults in the Bogalusa Heart Study (23). In contrast, the association we observed between 2-h PG and increasing television viewing time persisted in multivariable analyses in women and, to a lesser extent, in men, suggesting that physiological processes other than the effect of being overweight may be involved.

Our findings extend the literature showing that, in adults without diabetes, prolonged television viewing is associated with significant deleterious effects on glycemic measures, independent of physical activity levels and adiposity status. Independent associations with television viewing have also been reported from the Health Professionals Follow-Up Study (24) in relation to diabetes risk. This suggests that sedentary behavior, as indicated by television viewing time, is an important health behavior that is distinct from physical activity and may have a unique role in the disruption of normal metabolic function. These findings could have important implications for public health in relation to reducing the prevalence of hyperglycemia in adults, suggesting that it may not only be important to increase participation in physical activity, but also to reduce the time spent watching television (and possibly other sedentary behaviors).

In contrast to the fasted state, the major sites for the disposal of an oral glucose load are muscle, brain, and splanchnic tissues, together accounting for at least 80% of the load (25,26). In our study, television viewing time was more strongly associated with 2-h PG, suggesting that

Table 2—Adjusted mean differences in glycemic variables according to categories of television watching in men and women (multivariate linear regression)*

	n	Glycemic variable				
		FPG	2-h PG	InHOMA-%S†	InHOMA-%St	InHOMA-%B†
Men						
TV watching (h/day)						
<1	872	Ref.	Ref.	Ref.	Ref.	Ref.
1.0–1.9	1,126	−0.002 (−0.07 to 0.07)	0.162 (−0.13 to 0.46)	0.011 (−0.04 to 0.06)	−0.011 (−0.06 to 0.04)	0.009 (−0.03 to 0.05)
2.0–2.9	978	0.018 (−0.05 to 0.09)	0.000 (−0.39 to 0.39)	−0.022 (−0.08 to 0.03)	0.022 (−0.03 to 0.08)	−0.019 (−0.05 to 0.01)
3.0–3.9	479	−0.023 (−0.10 to 0.06)	0.195 (−0.10 to 0.49)	0.017 (−0.07 to 0.11)	−0.017 (−0.11 to 0.07)	0.016 (−0.06 to 0.09)
≥4	326	0.069 (−0.16 to 0.29)	0.547 (−0.05 to 1.15)	0.015 (−0.03 to 0.08)	−0.016 (−0.08 to 0.04)	0.002 (−0.05 to 0.06)
P for trend		0.58	0.06	0.89	0.87	0.96
Women						
TV watching (h/day)						
<1	1,377	Ref.	Ref.	Ref.	Ref.	Ref.
1.0–1.9	1,272	0.034 (−0.05 to 0.11)	0.009 (−0.17 to 0.19)	0.030 (−0.02 to 0.07)	−0.030 (−0.08 to 0.02)	0.010 (−0.02 to 0.04)
2.0–2.9	1,057	0.023 (−0.05 to 0.10)	0.047 (−0.31 to 0.41)	0.020 (−0.02 to 0.06)	−0.021 (−0.06 to 0.02)	0.003 (−0.03 to 0.03)
3.0–3.9	514	0.073 (−0.05 to 0.20)	0.473 (0.06 to 0.88)	0.083 (0.03 to 0.14)	−0.083 (−0.14 to −0.03)	0.033 (−0.07 to 0.08)
≥4	356	0.065 (−0.04 to 0.17)	0.501 (0.12 to 0.88)	0.098 (0.01 to 0.19)	−0.097 (−0.18 to −0.01)	0.044 (−0.02 to 0.11)
P		0.15	0.02	0.0001	0.0001	0.04

Data are regression coefficients (95% CI). *Each model shows the mean difference in glycemic measures for increasing categories of television (TV) watching relative to the reference (Ref.) category (<1 h). All models are adjusted for age, height, waist, education, smoking, dietary covariates (total energy, total fat, total carbohydrate, total sugars, fiber, and alcohol), and physical activity time. †Fasting serum insulin and HOMA variables were logarithmically transformed before inclusion in the model. Coefficients and 95% CIs reflect transformed values.

sedentary behavior may contribute to reduced glucose disposal in these tissues, although this will need to be confirmed by using direct measures of insulin resistance, such as a euglycemic-hyperinsulinemic clamp. Nevertheless, since skeletal muscle is an important site for the clearance of an oral glucose load from plasma, it is possible that the associations we have observed between television viewing and 2-h PG could at least be partly explained by the acute and/or cumulative absence of muscle contraction through inactivity, a defining characteristic of sedentary pursuits. This is further supported by our recent observation that 2-h PG appears to be more sensitive than FPG to the beneficial effects of leisure-time physical activity (27). Collectively, our findings suggest that in order to reduce the prevalence of diabetes and prediabetes, it may not only be important to increase participation in physical activity, but also to reduce time spent watching television (and possibly other sedentary behaviors).

An important finding of this study is the differential relationship with television viewing according to sex. This is consistent with our earlier findings showing that the associations between television viewing and abnormal glucose metabolism (as represented by impaired fasting glucose, impaired glucose tolerance, and previously undiagnosed diabetes) and the metabolic syndrome were stronger in women once adjustment was made for confounders (5,6). A similar sex differential has been reported in middle-aged French adults (3) and among U.S. adults (4) with respect to the risk of having the metabolic syndrome. At this point, we can only speculate on possible mechanisms to explain the sex differential in our findings to date. Differences in biology between men and women may be implicated, since there is evidence that the physiological benefits of exercise differ between men and women (28). Furthermore, at the other end of the activity continuum, sex differences in fuel homeostasis have been observed after 7 days of head-down bed rest, whereby insulin resistance was observed at the muscular level for men but at both the muscle and liver levels in women (29). These sex differences, in response to what could be considered as extreme sedentary behavior (bed rest), may provide some insight into the sex differential we observed in insulin sensitivity estimated from HOMA, since this is believed to re-

fect both hepatic and peripheral insulin sensitivity (30).

It is also possible, however, that the sex differences observed in the relationships between television viewing and glycemic variables reflect the different behavior patterns of men and women. Consistent with previous reports (31), we have recently shown a significant dose-response association between physical activity of at least moderate intensity and 2-h PG (27). In the present study, we found, as is consistently the case in other studies (20), that men were more physically active but watched slightly more television than women. Thus, it is possible that the physical activity of men, who typically engage in more vigorous activities (32), is protective against the effects of television viewing time on glycemic variables. While our analyses controlled for the effects of total physical activity, our measures did not differentiate intensities or patterns of activity. Although total volume of physical activity has been shown to have a greater influence on insulin sensitivity than does the intensity of activity (33), when total volume is similar, vigorous-intensity activity provides more enduring benefits to insulin action compared with moderate-intensity activity (34).

Alternatively, the sex differences in the association between television viewing time and glycemic variables may reflect underlying differences in television viewing time as a marker of overall sedentary behavior. Sedentary behaviors constitute a large proportion of the activities undertaken during leisure time in Australians (9). Television viewing time is only one component of overall sedentary time, and the extent to which television viewing time is representative of total sedentary behavior is unknown. Previous research has indicated that various sedentary behaviors are not alike in their relationship to health outcomes (3,7). Although television viewing is typically the sedentary behavior most strongly associated with the health outcome (7), the sex differences observed between television viewing time and glycemic variables may indicate that television viewing time is a stronger marker of overall sedentary behavior in women compared with men. Further research is required to measure sedentary time in men and women across a spectrum of behaviors, including occupational sedentary behavior and automobile use.

There is a strong case for considering

the underlying sedentary or inactivity physiology as a distinct research problem, since a better understanding of the biochemical, molecular, and cellular mechanisms of sedentary living could assist with the development of primary prevention strategies for many chronic diseases (1,39). For example, it has been recently demonstrated in animal models that the activity of lipoprotein lipase (an enzyme that binds to circulating lipoproteins when present on the vascular endothelium) in the muscle is essential for hydrolysis of triglyceride contained in lipoproteins. This is greatly reduced within several hours of sustained inactivity (40). Loss of lipoprotein lipase activity at the vascular endothelium impairs optimal tissue-specific uptake of lipoprotein fatty acids (39), which could in turn affect glucose homeostasis. It is well established that elevated blood free fatty acid levels play a key role in the development of insulin resistance in obesity and type 2 diabetes (41). Indeed, even in healthy humans, elevated plasma free fatty acid levels have been shown to inhibit insulin-stimulated glucose oxidation within 1–2 h, followed by inhibition of glucose uptake and glycogen synthesis within 3–4 h (41). Moreover, a recent study (42) has shown that insulin sensitivity decreases in as little as 2 days of physical inactivity in rats, mediated through alterations in insulin receptor signaling. We are unable to elucidate whether the associations with glycemic measures we have observed with television viewing reflect an acute or chronic response to sedentary behavior. Future research should focus on a better understanding of the biological pathways involved and the timing of these responses, which could inform the design of intervention programs aimed at reducing sedentary behavior.

The strengths of this study are its large sample size across a wide age range in both sexes and the use of continuous measures of glycemia. Its limitations include the assessment of just one aspect of sedentary living (television viewing) and the cross-sectional study design. Therefore, it is difficult to determine the extent to which participants were in higher television viewing time categories because of their health status; however, considering that those with known diabetes were excluded from the study, it is unlikely that their blood glucose level influenced their television viewing habits. Although the measurement of television viewing was obtained from a validated instrument

(16), considering the problems inherent in self-report, future research should aim to include objective measures of sedentary behavior. Additionally, as previously mentioned, television viewing is only one component of overall sedentary behavior, and the extent to which it is a marker of total sedentary time may differ for men and women. While the HOMA method has been widely used to assess β -cell function and insulin resistance (14), this method is derived from a mathematical assessment of the interaction between β -cell function and insulin resistance using fasting glucose and insulin concentrations. As such, HOMA is less informative than direct measures of glucose disposal (hyperinsulinemic-euglycemic clamp or the intravenous glucose tolerance test), since it is dependent on both peripheral and hepatic insulin sensitivity and does not accurately describe whole-body glucose uptake in those with minimal elevations of FPG, such as those with impaired fasting glucose and impaired glucose tolerance (30).

Ours is the first report from a population-based study to show associations with television viewing time across a range of glycemic variables. The findings reinforce the case for a strong focus in diabetes and obesity research on sedentary behaviors, such as television viewing, in addition to the now well-established base of evidence on the importance of increasing physical activity (5,24,27,31). It may also be the case that other sedentary behaviors have an additive effect on risk, in that television viewing may be a marker for a broader pattern of sedentary lifestyle that includes a variety of other forms of sitting time. Further research is required to examine such questions, addressing the structure and the determinants of sedentary behavior patterns in adult populations. With an increasingly strong body of evidence on deleterious health effects and possible underlying mechanisms, there is the need to develop more precise (ideally objective) measures of sedentary behavior and also to better understand the modifiable determinants of sedentary behavior (2). Such studies would inform the interventions needed to reduce sedentary behavior, particularly among those who are at increased risk of type 2 diabetes.

Acknowledgments—D.W.D. and J.S. are supported by a Victorian Health Promotion Foundation Public Health Research Fellowship. N.O. is supported by a program grant

(no. 301200) from the National Health and Medical Research Council and by a Research Infrastructure Grant from Queensland Health. G.N.H. is supported by an Australian Postgraduate Award and by Smart State funding from the Queensland Government.

We are most grateful to the following for their support of the study: the Commonwealth Department of Health and Aged Care, Abbott Australasia, Alphapharm, Aventis Pharmaceutical, AstraZeneca, Bristol-Myers Squibb Pharmaceuticals, Eli Lilly (Aust), GlaxoSmithKline, Janssen-Cilag (Aust), Merck Lipha, Merck Sharp & Dohme (Aust), Novartis Pharmaceutical (Aust), Novo Nordisk Pharmaceutical, Pharmacia and Upjohn, Pfizer, Roche Diagnostics, Sanofi Synthelabo (Aust), Servier Laboratories (Aust), BioRad Laboratories, HITECH Pathology, the Australian Kidney Foundation, Diabetes Australia, Diabetes Australia (Northern Territory), Queensland Health, South Australian Department of Human Services, Tasmanian Department of Health and Human Services, Territory Health Services, and the Victorian Department of Human Services and Health Department of Western Australia. Also, for their invaluable contribution to the setup and field activities of AusDiab, we are enormously grateful to A. Allman, B. Atkins, S. Bennett, S. Chadban, S. Colagiuri, M. de Courten, M. Dalton, M. D'Emben, T. Dwyer, D. Jolley, I. Kemp, P. Magnus, J. Mathews, D. McCarty, A. Meehan, K. O'Dea, P. Phillips, P. Popplewell, C. Reid, A. Stewart, R. Tapp, H. Taylor, T. Welborn, and F. Wilson.

References

- Booth FW, Chakravarthy MV, Gordon SE, Spangenburg EE: Waging war on physical inactivity: using modern molecular ammunition against an ancient enemy. *J Appl Physiol* 93:3–30, 2002
- Owen N, Leslie E, Salmon J, Fotheringham MJ: Environmental determinants of physical activity and sedentary behavior. *Exercise Sports Sci Rev* 28:153–158, 2000
- Bertrais S, Beyeme-Ondoua JP, Czernichow S, Galan P, Hercberg S, Oppert JM: Sedentary behaviors, physical activity, and metabolic syndrome in middle-aged French subjects. *Obes Res* 13:936–944, 2005
- Ford ES, Kohl HW 3rd, Mokdad AH, Ajani UA: Sedentary behavior, physical activity, and the metabolic syndrome among U.S. adults. *Obes Res* 13:608–614, 2005
- 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
- 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
- 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
- Salmon J, Bauman A, Crawford D, Timperio A, Owen N: The association between television viewing and overweight among Australian adults participating in varying levels of leisure-time physical activity. *Int J Obes* 24:600–606, 2000
- Australian Bureau of Statistics: *How Australians Use Their Time*. Canberra, Australia, Commonwealth of Australia, 1998
- Abdul-Ghani MA, Tripathy D, DeFronzo RA: Contributions of β -cell dysfunction and insulin resistance to the pathogenesis of impaired glucose tolerance and impaired fasting glucose. *Diabetes Care* 29:1130–1139, 2006
- Unwin N, Shaw J, Zimmet P, Alberti KGMM: Impaired glucose tolerance and impaired fasting glycaemia: the current status on definition and intervention. *Diabet Med* 19:708–723, 2002
- Kronenberg F, Pereira MA, Schmitz MK, Arnett DK, Evenson KR, Crapo RO, Jensen RL, Burke GL, Sholinsky P, Ellison RC, Hunt SC: Influence of leisure time physical activity and television watching on atherosclerosis risk factors in the NHLBI Family Heart Study. *Atherosclerosis* 153:433–443, 2000
- 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
- Wallace TM, Levy JC, Matthews DR: Use and abuse of HOMA modeling. *Diabetes Care* 27:1487–1495, 2004
- Australian Institute of Health and Welfare (AIHW): *The Active Australia Survey: A Guide and Manual for Implementation, Analysis and Reporting*. Canberra, Australia, AIHW, 2003
- Salmon J, Owen N, Crawford D, Bauman A, Sallis JF: Physical activity and sedentary behavior: a population-based study of barriers, enjoyment, and preference. *Health Psychol* 22:178–188, 2003
- Ireland P, Jolley D, Giles G, O'Dea K, Powles J, Rutishauser I, Wahlqvist M, Williams J: Development of the Melbourne FFQ: a food frequency questionnaire for use in an Australian prospective study involving an ethnically diverse cohort. *Asia Pac J Clin Nutr* 3:19–31, 1994
- Lewis J, Milligan G, Hunt A: *NUTTAB95 Nutrient Data Table for Use in Australia*. Canberra, Australia, Australian Govern-

- ment Publishing Service, 1995
19. StataCorp: *Stata Statistical Software: Release 9.0*. College Station, TX, StataCorp LP, 2005
 20. 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
 21. Jones CN, Pei D, Staris P, Polonsky KS, Chen YD, Reaven GM: Alterations in the glucose-stimulated insulin secretory dose-response curve and in insulin clearance in nondiabetic insulin-resistant individuals. *J Clin Endocrinol Metab* 82:1834–1838, 1997
 22. Tayek JA, Manglik S, Abemayor E: Insulin secretion, glucose production, and insulin sensitivity in underweight and normal-weight volunteers, and in underweight and normal-weight cancer patients: a Clinical Research Center study. *Metabolism* 46:140–145, 1997
 23. Gustat J, Srinivasan SR, Elkasabany A, Berenson GS: Relation of self-rated measures of physical activity to multiple risk factors of insulin resistance syndrome in young adults: the Bogalusa Heart Study. *J Clin Epidemiol* 55:997–1006, 2002
 24. Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB: Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. *Arch Intern Med* 161:1542–1548, 2001
 25. Ferrannini E, Bjorkman O, Reichard GA Jr, Pilo A, Olsson M, Wahren J, DeFronzo RA: The disposal of an oral glucose load in healthy subjects: a quantitative study. *Diabetes* 34:580–588, 1985
 26. Kelley D, Mitrakou A, Marsh H, Schwenk F, Benn J, Sonnenberg G, Arcangeli M, Aoki T, Sorensen J, Berger M, et al.: Skeletal muscle glycolysis, oxidation, and storage of an oral glucose load. *J Clin Invest* 81:1563–1571, 1988
 27. Healy GN, Dunstan DW, Shaw JE, Zimmet PZ, Owen N: Beneficial associations of physical activity with 2-h but not fasting blood glucose in Australian adults: the AusDiab Study. *Diabetes Care* 29:2598–2604, 2006
 28. Wilmore JH: Dose-response: variation with age, sex, and health status. *Med Sci Sports Exerc* 33:S622–S634, 2001
 29. Le Blanc J, Nadeau A, Boulay M, Rousseau-Mignerot S: Effects of physical training and adiposity on glucose metabolism and I-Insulin binding. *J Appl Physiol* 11: 424–427, 1979
 30. Tripathy D, Almgren P, Tuomi T, Groop L: Contribution of insulin-stimulated glucose uptake and basal hepatic insulin sensitivity to surrogate measures of insulin sensitivity. *Diabetes Care* 27:2204–2210, 2004
 31. Van Dam RM, Schuit AJ, Feskens EJ, Seidell JC, Kromhout D: Physical activity and glucose tolerance in elderly men: the Zutphen Elderly study. *Med Sci Sports Exerc* 34:1132–1136, 2002
 32. Macera CA, Ham SA, Yore MM, Jones DA, Ainsworth BE, Kimsey CD, Kohl HW 3rd: Prevalence of physical activity in the United States: behavioral risk factor surveillance system, 2001. *Prev Chronic Dis* 2:A17, 2005
 33. Houmard JA, Tanner CJ, Slentz CA, Duscha BD, McCartney JS, Kraus WE: Effect of the volume and intensity of exercise training on insulin sensitivity. *J Appl Physiol* 96:101–106, 2004
 34. DiPietro L, Dziura J, Yeckel CW, Neuffer PD: Exercise and improved insulin sensitivity in older women: evidence of the enduring benefits of higher intensity training. *J Appl Physiol* 100:142–149, 2006
 35. Levitan EB, Song Y, Ford ES, Liu S: Is nondiabetic hyperglycemia a risk factor for cardiovascular disease? A meta-analysis of prospective studies. *Arch Intern Med* 164:2147–2155, 2004
 36. The DECODE Study Group: Is the current definition for diabetes relevant to mortality risk from all causes and cardiovascular and noncardiovascular diseases? *Diabetes Care* 26:688–696, 2003
 37. Hu G: Gender difference in all-cause and cardiovascular mortality related to hyperglycaemia and newly-diagnosed diabetes. *Diabetologia* 46:608–617, 2003
 38. Legato MJ, Gelzer A, Goland R, Ebner SA, Rajan S, Villagra V, Kosowski M: Gender-specific care of the patient with diabetes: review and recommendations. *Gen Med* 3:131–158, 2006
 39. Hamilton MT, Hamilton DG, Zderic TW: Exercise physiology versus inactivity physiology: an essential concept for understanding lipoprotein lipase regulation. *Exerc Sport Sci Rev* 32:161–166, 2004
 40. 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
 41. Boden G: Role of fatty acids in the pathogenesis of insulin resistance and NIDDM. *Diabetes* 46:3–10, 1997
 42. Kump DS, Booth FW: Alterations in insulin receptor signalling in the rat epitrochlearis muscle upon cessation of voluntary exercise. *J Physiol* 562:829–838, 2005