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Association Between Daily Time Spent in Sedentary Behavior and Duration of Hyperglycemia in Type 2 Diabetes

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Abstract

Exercise and sedentary behavior have different physiologic effects, which have yet to be fully explained. Time spent in sedentary behavior has been associated with glucose intolerance in adults at risk for type 2 diabetes, but these data have come largely from cross-sectional studies that have not explored this relationship in adults with diabetes. The specific aim of this study was to examine the relationship between time spent in sedentary behavior and glucose levels in adults diagnosed with type 2 diabetes over 3–5 days. **Methods:** Using continuous and concurrent data gathered from wrist accelerometry and a Continuous Glucose-Monitoring Sensor (CGMS), we conducted a longitudinal, descriptive study involving 86 patients with type 2 diabetes. **Results:** More time spent in sedentary behavior was predictive of significant increases in time spent in hyperglycemia ($B = 0.12$, $p < .05$). **Conclusions:** These findings highlight the relationship between time spent sedentary and time spent in hyperglycemia, as identified through our use of objective, continuous data collection methods for both sedentary behavior and glucose levels across multiple days (Actiwatch, CGMS). For patients with type 2 diabetes, these findings emphasize the need for the development of individualized interventions aimed at decreasing the amount of time spent in hyperglycemia by reducing sedentary time.

Keywords

physical inactivity, health behaviors, real-time data, glucose control

Sedentary behaviors are common among people of all ages and are emerging as a grave threat to global health. This specific class of behaviors is characterized by little physical movement and low energy expenditure ($\leq$1.5 metabolic equivalents; e.g., sitting, lying down, watching television, and driving). Such behaviors may be associated with unique cellular signals and physiologic responses that are not simply the inverse of those associated with physical activity (Hamilton, Hamilton, & Zderic, 2007), but the mechanisms for these responses are not yet understood. For example, among 8,800 adults, time spent in sedentary behaviors such as television viewing was associated with all-cause and cardiovascular mortality even after adjustment for glucose status and other cardiometabolic and health factors (Dunstan, Barr, et al., 2010; Veerman et al., 2012).

In nondiabetic individuals, sedentary behavior has been independently linked to increased risk of abnormal glucose metabolism, including increased insulin secretion and decreased insulin sensitivity (Lahjibi et al., 2013), elevated fasting insulin levels (Ford et al., 2010; Helmerhorst, Wijndaele, Brage, Wareham, & Ekelund, 2009; Thorp et al., 2010), impaired glucose tolerance (Dunstan, Salmon, Owen, et al., 2004, 2005; Dunstan, Salmon, Healy, et al., 2007; Gardiner et al., 2011; Henson et al., 2013; Sisson et al., 2009; Thorp et al., 2010), and type 2 diabetes (T2DM; Dunstan, Salmon, Owen, et al., 2004; Grontved & Hu, 2011; Krishnan, Rosenberg, & Palmer, 2009). These associations were significant even after adjustment for potential confounders, including measures of body composition and time spent in moderate to vigorous physical activity (MVPA; Dunstan, Salmon, Owen, et al., 2004; Dunstan,

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Salmon, Healy, et al., 2007; Ford et al., 2010; Gardiner et al., 2011; Helmerhorst et al., 2009; Henson et al., 2013; Krishnan et al., 2009; Lahjibi et al., 2013; Sisson et al., 2009; Wijndaele et al., 2010). Findings from longitudinal analyses in the AusDiab studies of nondiabetic adults revealed that self-reported increases in television viewing time were linearly associated with increased cardiometabolic risk, independent of baseline physical activity (Hansen et al., 2012; Wijndaele et al., 2010). For every 5-hr increase in television viewing from baseline to 5-year follow-up, markers of glucose homeostasis, including fasting and 2-hr postchallenge plasma glucose, insulin levels, and insulin resistance, were all increased (Hansen et al., 2012; Wijndaele et al., 2010). Even in healthy, active adults who meet the goals set in physical activity guidelines, there is a linear relationship between self-reported time spent in sedentary behaviors and poor cardiometabolic outcomes including fasting plasma glucose in women and waist circumference in both women and men (Healy et al., 2008). In patients at risk for T2DM, sedentary time was independently associated with cardiometabolic risks after the models were adjusted for body mass index (BMI) and MVPA (Henson et al., 2013). Surprisingly, compared to total physical activity and MVPA, time spent in sedentary behavior was a stronger predictor of detrimental metabolic markers, including 2-hr postchallenge glucose, high-density lipoprotein (HDL) cholesterol, and triacylglycerol. Additionally, daily leisure-time sedentary behaviors of $\geq$ 4 hr duration increased the odds of metabolic syndrome in both inactive and active men and inactive women (Sisson et al., 2009). These findings suggest that time spent in sedentary behaviors may cause metabolic dysregulation even in physically active people.

In one of the few studies on the effects of sedentary behavior involving patients with diagnosed T2DM, researchers examined sedentary time in relationship to inflammatory markers, including interleukin-6, adiponectin, and C-reactive protein (CRP) over a 6-month period. Sedentary time at baseline was associated with interleukin-6 in men and women. For each hour increase in sedentary time, interleukin-6 increased by 8% (95% confidence interval [CI] = [0, 15]) in men and by 12% (95% CI [0, 24]) in women, but this relationship was no longer significant when the models were adjusted for waist circumference. There were no associations between baseline measures of sedentary time and adiponectin or CRP; however, after 6 months, every hour reduction in sedentary time from baseline was associated with a 24% reduction in CRP (95% CI [1.0, 48.0]) in women only (Falconer et al., 2014). In cross-sectional studies of men and women with new-onset T2DM, time spent sedentary was independently associated with clustered metabolic risk profiles (A. J. Cooper et al., 2014) and individual metabolic risk factors (waist circumference, insulin, insulin resistance, lower HDL; A. R. Cooper et al., 2012), even when findings were adjusted for MVPA expenditure (A. J. Cooper et al., 2014; A. R. Cooper et al., 2012).

Research has shown that adults with T2DM accrue significantly higher amounts of sedentary time than healthy controls (Cichosz et al., 2013; Hamer, Bostock, Hackett, & Steptoe, 2013), yet the majority of data on the effects of sedentary time on metabolic markers come largely from studies in nondiabetic participants. Additionally, much of the data have been gathered using cross-sectional methods. Consequently, there remains a large gap in our knowledge of the immediate and dynamic interplay between time spent sedentary and blood glucose levels across time in free-living adults with T2DM.

The specific aim of the present study was to examine the relationship between time spent in sedentary behavior and glucose levels in adults with T2DM over 3–5 days using continuous and concurrent data gathered from wrist accelerometry and a Continuous Glucose-Monitoring Sensor (CGMS). Use of accelerometry and CGMS allowed us an opportunity to obtain objective measurements of sedentary behaviors and glucose levels across multiple days in these participants.

Method and Material

Research Design and Sample

We conducted a longitudinal, descriptive study of objectively measured physical activity and glucose levels among free-living ambulatory adults with T2DM. We recruited participants in a large Midwestern city in the United States through flyer distribution and Internet-based bulletin boards between September 2012 and December 2013. Adults were eligible for inclusion if they were aged 45 years or older with a history of diagnosed T2DM for $\geq$6 months and were able to read and write in English. They were excluded if they had any condition that prohibited them from walking.

Participants completed three data collection visits over 6 days to a dedicated clinical research center within the College of Nursing at a large Midwestern university or at a Department of Veteran's Affairs VA hospital within the Physical Performance Laboratory. During the first visit, we obtained demographic information, anthropometric measurements (height, weight, and waist circumference), and a diabetes-focused health history and placed a CGMS on the abdomen and a wrist accelerometer on the nondominant wrist of each participant. Participants wore these devices throughout the 6 days of the study. We instructed participants to complete daily sleep and activity diaries and to perform three to four self-monitored blood glucose tests per day for CGMS calibration. During Visit 2 (Day 3 of data collection), we replaced the CGMS sensor with a fresh sensor per the manufacturer's instructions. At Visit 3 (Day 6 of data collection), participants returned the diaries and devices, from which we downloaded the data immediately. We checked these data for missing values and accuracy. Participants were reimbursed US$100.00 for their efforts. The institutional review boards of the participating institutions approved all study methods, and participants provided informed consent.

Glucose Measurement

For evaluation of glucose control, we measured hemoglobin A1C (HbA_{1c}) using the A1CNow+™ from Bayer Healthcare (Whippany, NJ). The National Glycohemoglobin Standardization Program has certified the A1CNow+, and results obtained

from the A1CNow+™ are comparable to those from laboratory methods using high-performance liquid chromatography (Bode, Irvin, Pierce, Allen, & Clark, 2007).

We conducted continuous glucose measurement using samples from interstitial fluid that were collected over two 3-day periods using the Medtronic iPro CGMS (iPro; Medtronic, Northridge, CA). Glucose data were downloaded into the Medtronic CareLink Management Software (Northridge, CA) as 5-min averages and then exported as Excel data files for use in the analysis. The iPro system does not provide visual reports of real-time glucose values; thus we chose this system to prevent participants from making aggressive changes in their diabetes management during the time they were wearing the monitor. Prior research confirmed the accuracy of the Medtronic MiniMed iPro Continuous Glucose-Monitoring Sensor (CGMS) through a home-based multicenter trial of 415 sensors (Gross et al., 2000). Median daily correlation coefficients with capillary blood glucose results were 0.92, with 75% of readings above 0.75. The CGMS was able to correctly identify episodes of glycemic excursion (fluctuation) outside the target range.

We derived daily average blood glucose levels from the CGMS data. For this study, we defined hyperglycemia as time in minutes during waking hours with blood glucose levels >8.9 mmol/mol (>160 mg/dl). This cut point corresponds to the mean glucose level necessary for achieving HbA_{1c} levels close to the goal range of <7% (<53 mmol/mol; Inzucchi et al., 2012).

Sedentary Behavior and Physical Activity Measurement

We measured sedentary behavior and physical activity continuously over 6 days using the Actiwatch-Score wrist accelerometer and Respironics Actiware software Version 5.70.1 (Philips Respironics, Bend, OR). The device was sensitive to motion in all directions. Epochs were recorded in 30-s intervals. Recent studies involving patients with diabetes have reported successful use of wrist actigraphy to quantify activity (Allen, Fain, Braun, & Chipkin, 2008). In a trial of 60 overweight women, wrist-worn accelerometry (Actiwatch AW64) was correlated with activity energy expenditure using whole-room indirect calorimetry ($r = .73$, $p < .05$; Chen et al., 2003). We compared accelerometer data with daily diaries of self-reported sleep, wake, and nonwear times. Data were excluded for diary self-reported nonwear time. We did not include sleep time as part of sedentary time.

For the present study, we defined sedentary behavior as time spent in activity at levels <100 activity counts (ACs) per 30-s epoch (<200 AC/min), light physical activity 101–900 AC/30-s epoch (202–1,800 AC/min) and MVPA as AC > 900/30-s epoch (>1,800 AC/min). There is little consensus regarding the choice of sedentary behavior AC thresholds and no relevant published data from studies in which wrist accelerometers were used in adults. In studies reporting sedentary behavior measured by hip-worn accelerometers, 100 AC/min is the usual upper limit for ACs for denoting sedentary behavior. However, Lopes, Magalhaes, Bragada, and Vasques (2009) found that a threshold of 100 AC/min significantly underestimated sedentary time and that a sedentary score threshold of <200 AC/min was a better gauge of sedentary behavior in adults who were overweight/obese or who had type 2 diabetes. For this reason, we used a threshold of 200 AC/min as our upper limit of sedentary activity. We identified nonwear time as any bout of consecutive counts of 0 AC/min lasting ≥90 min that was not classified as sleep/rest time by the software. Data were considered valid if wear time was ≥600 min/day.

Data Analysis

We conducted statistical analyses using SPSS, Version 22 (Chicago, IL) and STATA, Version 13 (College Station, TX). Descriptive data analyses were conducted to present sample characteristics of participants. Generalized estimating equations (GEEs) were used to examine the association of daily sedentary time with daily duration of hyperglycemia over 5 consecutive days. We chose the GEE method because it allows for analyzing longitudinal and continuous data while accounting for within-subject correlations, nonnormal distributions, and missing data. The GEE model was estimated with age, gender, BMI, and time spent in light activity for all analyses. The GEE model statistic B is akin to the R^2 coefficient in a regression: it describes the strength of the relationship and how much of the dependent variable (hyperglycemia) is explained by the independent variable (sedentary time). The sample size for the daily repeated-measure GEE model in this study was calculated with the power analysis software R longpower (Donohue, Gamst, & Edland, 2013) and the Monte Carlo simulation method based on the pilot data analysis. The expected effect size of sedentary time to hyperglycemia was assumed to be 0.15 and α as .05. The sample size of 86 subjects with 5 days of measurements gave us adequate power of 0.80.

Results

A total of 144 individuals inquired about the study. We screened 139 individuals for eligibility, and 135 met the inclusion criteria. Of those, 25 decided not to participate due to travel distance, requirements of the study, or amount of reimbursement. Additionally, 3 participants did not show up for their study visits, and 21 had incomplete data sets and were not included in the analyses. Thus, 86 adults (aged 58 ± 8.7 years) with T2DM met the criteria for inclusion in the analyses. The sample characteristics of these subjects are detailed in Table 1. Almost half of the participants were female (46%) and more than half were African American (57.3%). The mean duration of T2DM was 8.6 years, with less than 35% of participants on insulin therapy. On average, the participants were overweight or obese (BMI 33.3 kg/m^2 ± 6.9). Most participants had HbA_{1c} values slightly higher than those recommended by the American Diabetes Association and European Association for the Study of Diabetes (7.6% ± 2.0 mmol/mol and 60 ± 22.0 mmol/mol). We found a significant gender difference in BMI

Table 1. Participant Characteristics ($N = 86$) and Study Variables.

Variable	Mean ± *SD* (Range) or *n* (%)
Age (years)	58.1 ± 8.81 (45–81)
Education (years)	14.5 ± 2.7 (7–24)
Female	42 (48.8)
Race	
White	29 (33.7)
African American	48 (55.8)
Asian American	3 (3.5)
Native American/Pacific Islander	1 (1.2)
Other	5 (5.8)
Insulin therapy	28 (32.9)
DM duration (years)	8.6 ± 6.9 (0.8–30)
HbA_{1c} (%)	7.6 ± 2.0
HbA_{1c} (mmol/mol)	59.3 ± 21.3
BMI (kg/m^2)	33.3 ± 6.9 (20.1–67.3)
Mean hyperglycemia time (min/day)	548.6 ± 411.9 (20–1427)
Mean sedentary time (min/day)	516.0 ± 121.3 (183.6–848.7)
Mean light physical activity (min/day)	422.0 ± 108.8 (125.7-637.2)
Mean moderate to vigorous physical activity (min/day)	5.5 ± 6.8 (0–32.4)

Note. BMI = body mass index.

(males mean BMI = 30.9 ± 5.3 kg/m^2, females 35.9 ± 7.6 kg/m^2, $t = 13.549$, $p = .001$). There were no other significant differences between genders.

Wear time for both the Actiwatch and the CGMS ranged from 2 to 5 days, with most participants wearing the devices for 5 days (Actiwatch 84.2% and CGMS 68.4%). Across all days that the devices were worn, the mean time spent with blood glucose levels ≥160 mg/dl (hyperglycemia) was 549 min/day. Mean time spent in sedentary behavior was 511 min/day during waking hours, while time spent in MVPA was <6 min/day (Table 1).

More time spent in sedentary behavior was predictive of significant increases in time spent in hyperglycemia. The relationship remained significant after controlling for age, gender, diabetes duration, insulin use, BMI, and light physical activity ($B = 0.14$, $SE = 0.05$, $p = .003$; Table 2). Use of any insulin therapy was also a significant predictor of time spent in hyperglycemia.

Table 2. Generalized Estimating Equation (GEE) of Duration of Hyperglycemia by Sedentary Time.

	Duration of Hyperglycemia by Sedentary Time	
Parameter	*B*	*SE*
Sedentary time	0.14**	0.05
Light-intensity physical activity time	0.01	0.05
Age	−5.87	5.07
Gender (female)	91.00	404.53
Diabetes duration (years)	6.82	7.48
Insulin use (no)	−237.91*	107.63
BMI	−1.47	9.96
Gender (female) × BMI	−0.97	12.24

Note. $N = 86$. BMI = body mass index.
*$p < .05$. **$p < .01$.

Discussion

Our participants accumulated a large amount (>8 hr) of objectively measured sedentary time each day during waking hours. While sleep is considered a sedentary behavior, it imparts health benefits; thus, we decided not to include sleep in our models. In one of the few studies comparing objectively measured sedentary behavior in adults with and without T2DM, Hamer, Bostock, Hackett, and Steptoe (2013) reported the subjects with T2DM accrued over 11 hr of sedentary time each day, which was a significantly higher amount than the healthy controls accrued. The investigator attributed this finding to the fact that the subjects with T2DM were significantly more overweight and the excess weight may act as a barrier to physical activity.

Similar to findings from studies of nondiabetic adults, our findings suggest that the total amount of time spent sedentary is associated with higher blood glucose levels, even when adjusted for time spent in light physical activity, gender, and BMI. With our use of continuous objective measures, we were able to see how the total daily amount of time spent sedentary predicted the daily amount of time a subject would spend with hyperglycemic blood glucose values ≥8.9 mmol/mol (160 mg/dl) across 5 days. The *B* statistic of 0.12 suggests that every 1-min increase in sedentary time results in 0.12 min increase in time spent in hyperglycemia. For example an additional 60 min of sedentary time translates to an additional 7.4 min in hyperglycemia. These findings are important because mean glucose levels above this cut point correspond to increases in HbA_{1c} level, placing patients at greater risks for microvascular complications (Stratton et al., 2000). Additionally, chronic hyperglycemia is associated with lower muscle strength with aging, putting aging adults with T2DM at higher risks for poor physical functioning (Kalyani, Metter, Egan, Golden, & Ferrucci, 2015).

Other authors have reported relationships between time spent sedentary and glucose levels. Healy was among the first to use objective activity measures (i.e., waist-mounted accelerometers) in cross-sectional analyses of sedentary time and measures of glycemia, but these measures were conducted in nondiabetic individuals among a subset of subjects in the AusDiab study (Healy et al., 2007). Healy et al. reported that higher sedentary time was associated with significantly higher 2-hr postchallenge plasma glucose levels but not fasting plasma glucose levels. Additionally, more time spent in light-intensity physical activity was significantly associated with lower 2-hr postchallenge plasma glucose levels, even after adjustment for time spent in MVPA. Henson et al. (2013) also reported a linear association between accelerometer-derived sedentary time and 2-hr postchallenge plasma glucose in individuals at-risk for T2DM. Similar to our study, these authors adjusted for age and

measures of adiposity. While researchers in these previous studies adjusted for MVPA, we chose to use light-intensity activity in our models, as our subjects were so sedentary. Few studies have included subjects with T2DM. A. J. Cooper et al. (2014) measured physical activity in adults with T2DM over 4 consecutive days. While average daily sedentary time was associated with clustered metabolic risks, the authors did not find a direct association with HbA_{1c}. While these previous studies all used objective measures of sedentary behavior, they used only static measures of glucose as their outcome and were thus unable to see the longitudinal relationships between time spent sedentary and time spent in hyperglycemia. Our choice of using concurrent and continuous measures of both sedentary behavior and glucose levels revealed a relationship that previous studies may have missed by using only static measures of glucose control.

Our finding that those subjects who did not use insulin spent less time in hyperglycemia was surprising but may be explained by the fact that many adults with T2DM are not started on insulin therapy until their blood glucose control is severely compromised. In fact, when we revisited the data, we found the use of any insulin therapy to be positively associated with HbA_{1c} levels ($r = .332$, $p < .01$, not reported), suggesting those on insulin therapy also had the poorest diabetes control.

There are limitations to our study. We used a wrist-worn accelerometer to measure time in sedentary behavior, which may have missed some leg movement that a hip-worn device would have picked up. We were also unable to measure time spent standing versus sitting or lying due to the inability of the study accelerometer to detect postural changes. Theoretically, more skeletal muscle is required for standing than for sitting or lying, thus standing promotes glucose lowering. We did not collect dietary information. Collecting such data might have allowed us to see what role the amounts or types of food eaten played in the relationships measured. Although we collected use of medication at baseline, we did not monitor its ongoing use. We wanted to keep as true as possible to the ''free-living'' nature of the study and thus did not ask participants to maintain food diaries or medication documentation, which might have caused them to change normal behaviors. Additionally, it is possible that the relationship between sedentary time and glucose levels is bidirectional, though were not able to interpret directionality from our analyses. We studied the association between time spent in sedentary behavior and hyperglycemia at five time points over the study week. These analyses used daily averages of each variable rather than within-day averages of hourly or day-section epochs. In the future, the use of multilevel modeling of shorter time periods of data may help to add temporal direction to the findings. Other measures of glucose, such as measures of glucose variability (fluctuation in glucose levels throughout the day) or postprandial glucose excursions (the rise in glucose after a meal) may add to our understanding of the relationships between time spent sedentary and glucose levels in T2DM.

Conclusion

To our knowledge, this study is among the first to use objective and continuous measures of sedentary behavior and glucose levels in a group of adults with T2DM over multiple days. Our findings add to the literature about the relationship between time spent sedentary and time spent in hyperglycemia. These findings are of special interest because recent evidence suggests that patients with T2DM are more sedentary than their nondiabetic age- and sex-matched counterparts (Cichosz et al., 2013). For patients with T2DM, our findings offer considerable possibilities for the development of individualized interventions aimed at decreasing the amount of time spent in hyperglycemia by reducing sedentary time.

Current guidelines from the American Diabetes Association and the American College of Sports Medicine recommend 150 min/week of MVPA spread out over at least 3 days (Colberg et al., 2010). The American Diabetes Association only recently added recommendations for decreasing sedentary behavior as part of their guidelines for physical activity (American Diabetes Association, 2015). Traditionally, physical activity recommendations by health care providers have emphasized MVPA. It is important that health care providers incorporate the recent recommendations of decreasing sedentary time, especially for those patients who meet the current guidelines for physical activity. These patients may feel that they are decreasing their risk for poor cardiometabolic outcomes despite the possibility that they may still have extended periods of sedentary time. Additionally, in patients who have high sedentary time due to diabetes-related complications such as neuropathic pain or low exercise tolerance, prescribing simple measures to decrease sedentary time could offer these patients a means of decreasing glucose levels in the absence of higher intensity activity levels. Findings from our study add to the justification for including recommendations for decreasing sedentary time as part of current guidelines for physical activity in diabetes prevention and management. Nurses, with their focus on optimization of health and illness prevention, are especially well positioned to incorporate these findings into their clinical practices at the bedside and in the community.

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Author Contribution

C. Fritschi contributed to conception, design, acquisition, analysis, and interpretation; drafted the manuscript; critically revised the manuscript; gave final approval; and agrees to be accountable for all aspects of work ensuring integrity and accuracy. H. Park contributed to the analysis and interpretation, drafted manuscript, gave final approval, and agrees to be accountable for all aspects of work ensuring integrity and accuracy. A. Richardson contributed to the conception, acquisition, and analysis; drafted the manuscript, gave final approval, and agrees to be accountable for all aspects of work ensuring integrity and accuracy. E. G. Collins contributed to interpretation, critically revised the manuscript, gave final approval, and agrees to be accountable for

all aspects of work ensuring integrity and accuracy. R. Mermelstein contributed to interpretation, critically revised the manuscript, gave final approval and agrees to be accountable for all aspects of work ensuring integrity and accuracy. L. Riesche contributed to acquisition, critically revised the manuscript, gave final approval, and agrees to be accountable for all aspects of work ensuring integrity and accuracy. L. Quinn contributed to conception, acquisition, and interpretation; drafted the manuscript; critically revised the manuscript, gave final approval and agrees to be accountable for all aspects of work ensuring integrity and accuracy.

Authors' Note

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008 (5).

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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References

Allen, N. A., Fain, J. A., Braun, B., & Chipkin, S. R. (2008). Continuous glucose monitoring counseling improves physical activity behaviors of individuals with Type 2 diabetes: A randomized clinical trial. *Diabetes Research and Clinical Practice*, *80*, 371–379. doi:10.1016/j.diabres.2008.01.006

American Diabetes Association. (2015). Foundations of care: Education, nutrition, physical activity, smoking cessation, psychosocial care, and immunization. Sect. 4. in standards of medical care in diabetes—2015. *Diabetes Care*, *38*, s20–s30. doi:10.2337/dc15-S007

Bode, B. W., Irvin, B. R., Pierce, J. A., Allen, M., & Clark, A. L. (2007). Advances in hemoglobin A1c point of care technology. *Journal of Diabetes Science and Technology*, *1*, 405–411.

Chen, K. Y., Acra, S. A., Majchrzak, K., Donahue, C. L., Baker, L., Clemens, L., . . . Buchowski, M. S. (2003). Predicting energy expenditure of physical activity using hip- and wrist-worn accelerometers. *Diabetes Technology & Therapeutics*, *5*, 1023–1033. doi:10.1089/152091503322641088

Cichosz, S. L., Fleischer, J., Hoeyem, P., Laugesen, E., Poulsen, P. L., Christiansen, J. S., & Hansen, T. K. (2013). Objective measurements of activity patterns in people with newly diagnosed Type 2 diabetes demonstrate a sedentary lifestyle. *Diabetic Medicine*, *30*, 1063–1066. doi:10.1111/dme.12199

Colberg, S. R., Sigal, R. J., Fernhall, B., Regensteiner, J. G., Blissmer, B. J., Rubin, R. R., & the American Diabetes Association. (2010). Exercise and type 2 diabetes: The American College of Sports Medicine and the American Diabetes Association: Joint position statement. *Diabetes Care*, *33*, e147–e167. doi:10.2337/dc10-9990; 10.2337/dc10-9990

Cooper, A. J., Brage, S., Ekelund, U., Wareham, N. J., Griffin, S. J., & Simmons, R. K. (2014). Association between objectively assessed sedentary time and physical activity with metabolic risk factors among people with recently diagnosed Type 2 diabetes. *Diabetologia*, *57*, 73–82. doi:10.1007/s00125-013-3069-8

Cooper, A. R., Sebire, S., Montgomery, A. A., Peters, T. J., Sharp, D. J., Jackson, N., & Andrews, R. C. (2012). Sedentary time, breaks in sedentary time and metabolic variables in people with newly diagnosed Type 2 diabetes. *Diabetologia*, *55*, 589–599. doi:10.1007/s00125-011-2408-x

Donohue, M. C., Gamst, A. C., & Edland, S. D. (2013). *R longpower: Sample size calculations for longitudinal data*. Retrieved from https://bitbucket.org/mdonohue/longpower

Dunstan, D. W., Barr, E. L., Healy, G. N., Salmon, J., Shaw, J. E., Balkau, B., & Owen, N. (2010). Television viewing time and mortality: The Australian diabetes, obesity and lifestyle study (AusDiab). *Circulation*, *121*, 384–391. doi:10.1161/CIRCULATIONAHA.109.894824

Dunstan, D. W., Salmon, J., Healy, G. N., Shaw, J. E., Jolley, D., Zimmet, P. Z., . . . AusDiab Steering Committee. (2007). Association of television viewing with fasting and 2-h postchallenge plasma glucose levels in adults without diagnosed diabetes. *Diabetes Care*, *30*, 516–522. doi:10.2337/dc06-1996

Dunstan, D. W., Salmon, J., Owen, N., Armstrong, T., Zimmet, P. Z., Welborn, T. A., & AusDiab Steering Committee. (2004). Physical activity and television viewing in relation to risk of undiagnosed abnormal glucose metabolism in adults. *Diabetes Care*, *27*, 2603–2609.

Dunstan, D. W., Salmon, J., Owen, N., Armstrong, T., Zimmet, P. Z., Welborn, T. A., & AusDiab Steering Committee. (2005). Associations of TV viewing and physical activity with the metabolic syndrome in Australian adults. *Diabetologia*, *48*, 2254–2261. doi:10.1007/s00125-005-1963-4

Falconer, C. L., Cooper, A. R., Walhin, J. P., Thompson, D., Page, A. S., Peters, T. J., & Andrews, R. C. (2014). Sedentary time and markers of inflammation in people with newly diagnosed Type 2 diabetes. *Nutrition, Metabolism, and Cardiovascular Diseases*, *24*, 956–962. doi:10.1016/j.numecd.2014.03.009

Ford, E. S., Li, C., Zhao, G., Pearson, W. S., Tsai, J., & Churilla, J. R. (2010). Sedentary behavior, physical activity, and concentrations of insulin among US adults. *Metabolism*, *59*, 1268–1275. doi:10.1016/j.metabol.2009.11.020

Gardiner, P. A., Healy, G. N., Eakin, E. G., Clark, B. K., Dunstan, D. W., Shaw, J. E., . . . Owen, N. (2011). Associations between television viewing time and overall sitting time with the metabolic syndrome in older men and women: The Australian Diabetes, Obesity and Lifestyle study. *Journal of the American Geriatrics Society*, *59*, 788–796. doi:10.1111/j.1532-5415.2011.03390.x

Grontved, A., & Hu, F. B. (2011). Television viewing and risk of Type 2 diabetes, cardiovascular disease, and all-cause mortality: A meta-analysis. *Journal of the American Medical Association*, *305*, 2448–2455. doi:10.1001/jama.2011.812

Gross, T. M., Bode, B. W., Einhorn, D., Kayne, D. M., Reed, J. H., White, N. H., & Mastrototaro, J. J. (2000). Performance evaluation

of the MiniMed continuous glucose monitoring system during patient home use. *Diabetes Technology & Therapeutics*, *2*, 49–56.

Hamer, M., Bostock, S., Hackett, R., & Steptoe, A. (2013). Objectively assessed sedentary time and Type 2 diabetes mellitus: A case-control study. *Diabetologia*, *56*, 2761–2762.

Hamilton, M. T., Hamilton, D. G., & Zderic, T. W. (2007). Role of low energy expenditure and sitting in obesity, metabolic syndrome, Type 2 diabetes, and cardiovascular disease. *Diabetes*, *56*, 2655–2667. doi:10.2337/db07-0882

Hansen, A. L., Wijndaele, K., Owen, N., Magliano, D. J., Thorp, A. A., Shaw, J. E., & Dunstan, D. W. (2012). Adverse associations of increases in television viewing time with 5-year changes in glucose homoeostasis markers: The AusDiab study. *Diabetic Medicine*, *29*, 918–925. doi:10.1111/j.1464-5491.2012.03656.x

Healy, G. N., Dunstan, D. W., Salmon, J., Cerin, E., Shaw, J. E., Zimmet, P. Z., & Owen, N. (2007). Objectively measured light-intensity physical activity is independently associated with 2-h plasma glucose. *Diabetes Care*, *30*, 1384–1389. doi:10.2337/dc07-0114

Healy, G. N., Dunstan, D. W., Salmon, J., Shaw, J. E., Zimmet, P. Z., & Owen, N. (2008). Television time and continuous metabolic risk in physically active adults. *Medicine and Science in Sports and Exercise*, *40*, 639–645. doi:10.1249/MSS.0b013e3181607421

Helmerhorst, H. J., Wijndaele, K., Brage, S., Wareham, N. J., & Ekelund, U. (2009). Objectively measured sedentary time may predict insulin resistance independent of moderate- and vigorous-intensity physical activity. *Diabetes*, *58*, 1776–1779. doi:10.2337/db08-1773

Henson, J., Yates, T., Biddle, S. J., Edwardson, C. L., Khunti, K., Wilmot, E. G., & Davies, M. J. (2013). Associations of objectively measured sedentary behaviour and physical activity with markers of cardiometabolic health. *Diabetologia*, *56*, 1012–1020. doi:10.1007/s00125-013-2845-9

Inzucchi, S. E., Bergenstal, R. M., Buse, J. B., Diamant, M., Ferrannini, E., Nauck, M., … Matthews, D. R. (2012). Management of hyperglycemia in Type 2 diabetes: A patient-centered approach: Position statement of the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). *Diabetes Care*, *35*, 1364–1379. doi:10.2337/dc12-0413

Kalyani, R. R., Metter, E. J., Egan, J., Golden, S. H., & Ferrucci, L. (2015). Hyperglycemia predicts persistently lower muscle strength with aging. *Diabetes Care*, *38*, 82–90. doi:10.2337/dc14-1166

Krishnan, S., Rosenberg, L., & Palmer, J. R. (2009). Physical activity and television watching in relation to risk of Type 2 diabetes: The Black women's health study. *American Journal of Epidemiology*, *169*, 428–434. doi:10.1093/aje/kwn344

Lahjibi, E., Heude, B., Dekker, J. M., Højlund, K., Laville, M., Nolan, J., & Balkau, B. (2013). Impact of objectively measured sedentary behaviour on changes in insulin resistance and secretion over 3 years in the RISC study: Interaction with weight gain. *Diabetes and Metabolism*, *39*, 217–225. doi:10.1016/j.diabet.2012.12.006

Lopes, V. P., Magalhaes, P., Bragada, J., & Vasques, C. (2009). Actigraph calibration in obese/overweight and Type 2 diabetes mellitus middle-aged to old adult patients. *Journal of Physical Activity & Health*, *6*, S133–S140.

Sisson, S. B., Camhi, S. M., Church, T. S., Martin, C. K., Tudor-Locke, C., Bouchard, C., & Katzmarzyk, P. T. (2009). Leisure time sedentary behavior, occupational/domestic physical activity, and metabolic syndrome in U.S. men and women. *Metabolic Syndrome and Related Disorders*, *7*, 529–536. doi:10.1089/met.2009.0023

Stratton, I. M., Adler, A. I., Neil, H. A., Matthews, D. R., Manley, S. E., Cull, C. A., & Holman, R. R. (2000). Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): Prospective observational study. *British Medical Journal (Clinical Research Ed.)*, *321*, 405–412.

Thorp, A. A., Healy, G. N., Owen, N., Salmon, J., Ball, K., Shaw, J. E., & Dunstan, D. W. (2010). Deleterious associations of sitting time and television viewing time with cardiometabolic risk biomarkers: Australian Diabetes, Obesity and Lifestyle (AusDiab) study 2004–2005. *Diabetes Care*, *33*, 327–334. doi:10.2337/dc09-0493

Veerman, J. L., Healy, G. N., Cobiac, L. J., Vos, T., Winkler, E. A., Owen, N., & Dunstan, D. W. (2012). Television viewing time and reduced life expectancy: A life table analysis. *British Journal of Sports Medicine*, *46*, 927–930. doi:10.1136/bjsports-2011-085662

Wijndaele, K., Healy, G. N., Dunstan, D. W., Barnett, A. G., Salmon, J., Shaw, J. E., … Owen, N. (2010). Increased cardio-metabolic risk is associated with increased TV viewing time. *Medicine and Science in Sports and Exercise*, *42*, 1511–1518. doi:10.1249/MSS.0b013e3181d322ac