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Motor-Driven (Passive) Cycling: A Potential Physical Inactivity Countermeasure?

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Abstract

We have previously shown that motor-driven (passive) stationary cycling elevates energy expenditure (EE).

Purpose—To quantify how acute passive cycling affects glucose and insulin responses to an oral glucose tolerance test (OGTT) and basic cognition compared to sitting and moderate-intensity active cycling.

Methods—Twenty-four physically inactive healthy males completed three trials in randomized order involving 30-minute conditions of sitting, passive cycling and moderate-intensity cycling. During each condition, EE was measured and participants performed cognitive tests. Following each condition, a 2-hour OGTT was performed.

Results—EE was significantly higher during the cycling conditions compared to sitting (1.36 ± 0.58 and 6.50 ± 1.73 kcal·min⁻¹ greater than sitting for passive and moderate-intensity, respectively). A significant correlation was found between body fat percentage and post-sitting OGTT 2-h post plasma glucose ($r^2=0.30$, $p<0.05$) so participants were divided into lean ($n=11$) and non-lean ($n=13$) groups. In the non-lean group, compared to sitting, passive cycling lowered 2-h post plasma glucose (7.7 ± 1.3 vs. 6.9 ± 1.6 mmol·L⁻¹, respectively, $p<0.05$) and Matsuda whole body insulin sensitivity index (WBISI) was higher (2.74 ± 0.86 vs. 3.36 ± 1.08 , $p<0.05$). Additionally, passive and moderate-intensity cycling had similar beneficial effects on 2-h post plasma glucose and WBISI. Cognitive performance did not significantly differ between the sitting and passive cycling conditions.

Conclusion—2-h post plasma glucose was lower and WBISI following acute passive cycling was higher in non-lean participants. Given that and the increase in EE without changes in cognitive performance, we propose passive cycling as a promising intervention to counteract some of the deleterious effects of prolonged sitting in the workplace.

Keywords

sitting; sedentary; diabetes; obesity; exercise

Introduction

A plethora of research links prolonged periods of sitting with increased risk of chronic diseases. Prolonged periods of sitting are associated with impaired glucose tolerance (21,22,23,34), obesity (23,35), and even cancer (27). Furthermore, sitting may represent a distinct health risk independent of daily physical activity levels (20). Based on these findings, the latest physical activity guidelines suggest limiting sitting time throughout the day (18). However, there are circumstances when extended sitting cannot be practically avoided. For example, almost half of daily sitting occurs in the workplace (28).

Recent research suggests that interspersed light-intensity physical activity attenuates the negative health effects associated with continuous sitting. For example, Kim et al. (24) found that intermittent light-intensity physical activity reduced postprandial triglyceride and glucose concentrations. Additionally, Dunstan et al. (12) found similar reductions in postprandial glucose and insulin levels when sitting conditions were broken up with either light-intensity or moderate-intensity walking. Thus, there is a growing interest in using light-intensity physical activity to ameliorate the adverse effects of prolonged sitting, as evidenced by the increasing use of treadmill workstations. However, treadmill workstations are large, expensive, and can decrease some aspects of work performance (25,33). A more viable intervention may be motor-driven (passive) cycling. We have previously shown passive cycling increases energy expenditure (EE) to similar levels as light-intensity walking (31,32). Thus, it is reasonable to hypothesize that passive cycling can have similar effects on blood glucose compared to when sitting is interrupted by bouts of light walking, as shown previously (12). Passive cycling may also lower blood glucose to the same extent as light-intensity walking but has yet to be studied. Additionally, because passive cycling requires no volitional muscle activation, it may be less disruptive to work performance.

Before passive cycling can be used in a program to attenuate some of the adverse effects of prolonged sitting, several factors need to be investigated. For example, an ideal intervention should enhance not only EE but also improve cardiometabolic risk factors that are negatively influenced by prolonged conditions of sitting (i.e. blood glucose response to an oral glucose tolerance test, OGTT). Additionally, comparing passive cycling to moderate-intensity cycling, which improves glucose tolerance (11), would provide insight into the effectiveness of passive cycling as an intervention. Lastly, a workplace-based intervention should not impair cognition since this would limit its acceptance in the workplace.

Our purpose was to examine how acute passive cycling influences EE, glucose and insulin responses to an OGTT, and cognition. To accomplish this, we compared these variables during and following acute conditions of sitting, passive cycling, and moderate-intensity cycling. We anticipated moderate-intensity cycling would have a higher EE compared to passive cycling, and based on previous research comparing light- and moderate-intensity walking (12), we hypothesized that passive and moderate-intensity cycling would similarly

decrease glucose and insulin responses to an OGTT. Lastly, we hypothesized that passive cycling would minimally impact cognition.

Methods

Overview

The experimental protocol for this study was approved by the University of Colorado Boulder Institutional Review Board. Participants were recruited from the faculty, staff, and student population at the University of Colorado Boulder campus. Participants were informed of study risks and gave written informed consent. Study visits occurred at the University of Colorado Boulder Clinical Translational Research Center over four separate occasions: a screening session followed by three experimental sessions performed in a randomized crossover design. The experimental sessions included 30-minute conditions of either sitting, passive cycling, or moderate-intensity cycling. During each condition, EE was measured and participants performed two cognitive tests (attention switching and a modified version of the psychomotor vigilance task (37)). A 2-hour OGTT was performed following each condition. At least 7 days separated each of the sessions to allow for an adequate washout period.

Participants

Twenty-four physically inactive healthy males aged 19-48 volunteered for the study. Metabolic rate and related metabolic parameters are influenced by menstrual cycle and the small-scale nature of this study limited our ability to control for this confounding variable. As a result, only males were included. Participants were defined as physically inactive if they participated in regular exercise less than 2 hours per week. In addition to regular exercise restrictions, participants self-reported that their occupation did not require significant physical activity. Participants were also required to be free from any acute or chronic physical condition that could influence the outcome measures of the protocol and could not have used any medications (including over-the-counter and herbal products) within the month prior to the first visit that could affect EE, the blood glucose response during an OGTT, or cognition. Lastly, only participants who reported maintaining habitual 6-9 hour/night sleep schedules were included. Participants were 25.5 ± 7.8 years old with an average body mass of 72.0 ± 12.2 kg. Average BMI and body fat percentage were 23.2 ± 3.6 kg·m⁻² and $22.4 \pm 8.9\%$, respectively.

Screening Session

The screening session began with a brief medical history and physical exam to verify that participants were healthy enough to participate. During this time, participants had a fasting blood glucose test via finger stick and analysis using a Medisense Precision PCX glucometer (Abbott Park, Illinois, USA) to confirm they were not diabetic (indicated by fasting levels <7 mg·dL⁻¹). Participants also had a whole body dual energy x-ray absorptiometry (DXA) scan (GE LUNAR DXA system) to determine overall body composition. In addition, the DXA scan was used to estimate leg muscle mass (fat-free and bone-free mass) so we could examine the potential correlation between leg muscle mass and the increase in EE with passive cycling.

During the screening session, participants performed a graded exercise test (GXT) on a treadmill. Although cycling conditions were used in two of the experimental sessions, a GXT on a treadmill was chosen over a cycle ergometer because we find that physically inactive participants are likely to be more comfortable walking/running than cycling and thus more likely to achieve maximal exertion. After a brief warm up period, subjects started the GXT at a moderate intensity with no incline. The grade of the treadmill was then increased every 2 minutes until subjects reached volitional fatigue. For subjects walking/running at speeds $<9.66 \text{ km}\cdot\text{h}^{-1}$, the grade increased 2% while it increased 2.5% for those subjects running at speeds $\geq 9.66 \text{ km}\cdot\text{h}^{-1}$. Estimated maximal rate of oxygen consumption ($\dot{V}\text{O}_{2\text{max}}$) was determined based on treadmill speed, grade, and test duration (1). Maximum heart rate during the GXT was used to set the intensity of the moderate-intensity cycling experimental session.

Participants practiced the two computer tests (37,16) during the screening session. The first test, the attention switching task, required participants to respond to a mannequin mental rotation/spatial orientation question or a three digit addition/subtraction math question. Both questions were shown at the same time on the computer screen and a bar at the bottom of the screen informed participants which question to complete. Performance was measured by analyzing mannequin and math throughput scores. A throughput score is determined based on accuracy and speed of the responses and thus provides data regarding changes in either variable. Participants practiced the switching task a minimum of six times to remove the steep portion of the learning curve as described previously (37). During the second test, the modified psychomotor vigilance task (37), participants pressed a button whenever they saw a yellow circle appear on the black computer screen. The circle appeared randomly over a 10-minute period and measured the reaction time as well as the number of times participants did not press the button within 500 msec ("lapses"). Participants practiced the psychomotor vigilance task once before actual testing as this test does not have a learning curve.

Finally, a nutritionist instructed participants on how to record and follow a prescribed diet for the three days leading up to each experimental session. Participants were instructed to eat at least 150 grams of carbohydrate each of the three days and refrain from any caffeine or alcohol consumption. In addition, participants were asked to eat the same foods for the three days prior to each of the three different experimental sessions.

Experimental Sessions

To control for potential confounding variables, a number of restrictions were placed on participants for the three days prior to each experimental session. Participants followed the prescribed diet, recorded and maintained their normal sleep schedule, and minimized physical activity. To monitor physical activity during the three days prior to each of the experimental sessions, participants logged their activities and also wore Actigraph GT3XPlus activity monitors (Actigraph, Pensacola, FL, USA). Accelerometer data were recorded in one-minute epochs. Physical activity intensity was divided into two categories: sedentary ($<100 \text{ counts}\cdot\text{min}^{-1}$) and moderate-vigorous intensity ($>1952 \text{ counts}\cdot\text{min}^{-1}$) (15). On the day of the experimental sessions, participants fasted overnight and minimized

physical activity on the way to the lab. Participants either drove to the lab or were driven by a member of the research team.

The experimental sessions were performed in a randomized crossover manner but each followed the same format and was performed at the same time of day (Figure 1). Upon arrival to the lab, participants sat quietly for 70 minutes. This sitting time allowed the dependent measures to stabilize. During this 70 minutes of sitting, participants practiced each computer task one additional time. A nurse placed a catheter in a vein of the subject's arm or hand in preparation for the blood draws during the (OGTT). Once the catheter was placed, a resting, pre-activity blood sample was collected.

Following the 70 minutes of seated rest, participants performed one of the three randomized conditions: sitting, passive cycling, or moderate-intensity cycling. For the sitting condition, participants sat still for 30 minutes on the passive cycling ergometer. The 30 minutes of sitting was replaced with either passive cycling or moderate-intensity cycling for the other respective conditions. The passive cycling was performed at 80 RPM on a custom-built stationary cycle (Figure 2). The moderate-intensity cycling was performed at 64-76% of maximum heart rate determined from the GXT (18) at the subject's preferred cadence. Due to the unique design of the custom-built passive cycling device, a different stationary recumbent cycle (5002b, SportsArt, Woodinville, WA) was utilized for the moderate-intensity cycling condition.

Respiratory gas exchange was measured continuously during each condition using a Parvomedics TrueOne 2400 computerized indirect calorimetry system (Sandy, Utah, USA). Heart rate was measured using radio telemetry (Polar®) that was synced with the ParvoMedics calorimetry system and collected the heart rate data concurrently with the respiratory variables. Prior to each experimental session, the indirect calorimetry system was calibrated according to the manufacturer specifications. Respiratory and heart rate measurements were averaged every 15 seconds. Average EE was determined over the last 27 minutes of each condition (minutes 3 – 30).

During each condition, participants also performed the attention switching and psychomotor vigilance tasks. These tests were administered at minutes 5 and 15 of each 30-minute condition.

Immediately following the 30-minute test condition, a fasting blood sample was obtained and a 2-h OGTT was administered. Participants were instructed to consume a 296 mL beverage containing 75g of glucose (Azer Scientific, Morgantown, PA, USA) within 2 minutes. Once the glucose beverage was finished, a timer started (time = 0) and blood samples were taken at time points 10, 20, 30, 45, 60, 75, 90, 105, and 120 minutes. At each time point, approximately 2 mL of blood was collected from the catheter. The plasma was then separated by spinning the whole blood at 3700 RPM for 10 minutes. From the plasma, duplicate samples were drawn for the determination of plasma glucose. For each duplicate, 25 µL of plasma was mixed with a “cocktail” containing 50 µL of a buffer, lysing (Triton XL-100), and anti-glycolytic (sodium fluoride) solution. Each duplicate sample prepared according to the manufacturer's instructions was then analyzed using a YSI 2300 glucose

analyzer (YSI, Yellow Springs, OH USA). Plasma insulin levels were determined using a competitive radioimmunoassay kit (Millipore Corporation, Billerica, MA, USA).

Plasma glucose values 2-h post-glucose load were determined. In addition, for both plasma glucose and insulin, the incremental area under the curve (iAUC) was calculated using the values immediately prior to the ingestion of the glucose beverage as the baseline. Insulin and glucose values throughout the OGTT were used to determine insulin sensitivity using the whole body insulin sensitivity index (WBISI) developed by Matsuda and DeFronzo (26). The WBISI is an estimate of whole body insulin sensitivity using both plasma glucose and insulin data with higher WBISI values indicating increased insulin sensitivity.

Analysis

The analysis plan used a general linear model for repeated measures. The general linear model accounts for the correlation between measured outcome variables across different exercise conditions and also allows for a flexible covariance structure (unstructured). All models were fit using the SPSS Mixed procedure (SPSS Inc. version 20, Chicago, IL, USA). For the within group comparisons, the experimental sessions were treated as a factor, using the sitting activity as the reference group such that parameter estimates for passive and moderate-intensity cycling were in comparison to the control condition. Between group comparisons added adiposity group as a factor. Previous research has shown physical activity lowers blood glucose and raises EE (11) and our *a priori* hypotheses were for these same trends. Thus, planned *post-hoc* comparisons for plasma glucose and insulin variables and EE were performed as one-way analyses. All other comparisons were performed as two-way analyses. Data are presented throughout the paper as mean $\pm$ SD.

Results

Compared to sitting (1.25 ± 0.17 kcal $\cdot$ min $^{-1}$), EE was significantly higher during passive cycling (2.61 ± 0.65 kcal $\cdot$ min $^{-1}$, $p < 0.05$) and moderate-intensity cycling (7.75 ± 1.78 kcal $\cdot$ min $^{-1}$, $p < 0.05$). EE was also significantly higher during moderate-intensity cycling compared to passive cycling ($p < 0.05$). The increase in EE during passive cycling was not related to the between-subject variance in leg muscle mass ($R^2 = 0.06$, $p = 0.245$). Heart rate followed the same patterns as EE (Table 1).

A significant correlation was found between body fat percentage and 2-h post plasma glucose following the sitting condition ($r^2 = 0.30$, $p < 0.05$). As a result, participants were split into two groups: lean and non-lean. Participants were placed in the non-lean group if they had a body fat percentage $> 19\%$ (if under 40 years old) or $> 22\%$ (if older than 40) (17). Based on body fat percentage, 11 participants were placed in the lean group and 13 in the non-lean group. Descriptive data for the groups are given in Table 2. In addition to differences in body fat, lean participants had a significantly greater estimated $\dot{V}O_2$ max compared to the non-lean participants (48.9 ± 7.9 and 38.7 ± 4.3 mL $\cdot$ kg $^{-1}$ min $^{-1}$ for lean and non-lean, respectively, $p < 0.05$). Although not significant, there was a trend for fasting glucose levels being lower in the lean group (4.8 ± 0.3 and 5.1 ± 0.3 mmol $\cdot$ L $^{-1}$ for lean and non-lean, respectively, $p = 0.068$). No difference was found between groups or across days for

physical activity levels leading up to the experimental sessions (Table 1). Additionally, no difference was found between groups for EE during the trials (Figure 3).

In the non-lean group, 2-h post OGTT plasma glucose concentrations following passive cycling ($6.9 \pm 1.6 \text{ mmol} \cdot \text{L}^{-1}$, $p < 0.05$) and moderate-intensity cycling ($6.8 \pm 1.2 \text{ mmol} \cdot \text{L}^{-1}$, $p < 0.05$) were lower when compared to the continuous sitting condition ($7.7 \pm 1.3 \text{ mmol} \cdot \text{L}^{-1}$) (Figure 3). Compared to sitting, plasma glucose iAUC was significantly lower only for the moderate-intensity cycling condition (363 ± 87 and $303 \pm 12 \text{ mmol} \cdot \text{L}^{-1} \cdot \text{min}$, respectively, $p < 0.05$). For plasma insulin iAUC, neither cycling condition was different from sitting, but a significant difference was found between passive cycling and moderate-intensity cycling ($59,927 \pm 36,689$ and $51,463 \pm 34,062 \text{ pmol} \cdot \text{L}^{-1}$, respectively, $p < 0.05$). WBISI was significantly higher following passive cycling (3.36 ± 1.08 , $p < 0.05$) and moderate-intensity cycling (3.41 ± 1.38 , $p < 0.05$) compared to sitting (2.74 ± 0.86) (Figure 3). No significant differences were found between passive and moderate-intensity cycling for 2-h post plasma glucose, plasma glucose iAUC, and WBISI.

For the lean participants, glucose and insulin responses to the OGTT were not different between conditions except for plasma glucose iAUC (Table 1). Unexpectedly, plasma glucose iAUC following passive cycling was significantly higher compared to the sitting condition (233 ± 111 and $164 \pm 114 \text{ mmol} \cdot \text{L}^{-1} \cdot \text{min}$, respectively, $p < 0.05$).

Cognitive performance was analyzed with the lean and non-lean groups combined. Psychomotor vigilance task performance while cycling at a moderate-intensity showed faster reaction times compared to the passive cycling trial (332.3 ± 27.2 and $344.1 \pm 31.0 \text{ ms}$, respectively, $p < 0.05$). There was a trend for reaction time during moderate intensity cycling to also be faster compared to the sitting condition ($340.0 \pm 28.8 \text{ ms}$, $p = 0.06$). No significant differences were found between conditions for attention switching task throughput performance (Table 1). No difference was found between groups for cognitive performance during the trials.

Discussion

As expected, EE during passive cycling and moderate-intensity cycling was significantly higher compared to sitting across all participants. In the non-lean group, we found that 2-h plasma glucose levels were significantly lower and WBISI was significantly higher following both the passive and active moderate-intensity cycling conditions compared with the sitting condition. Additionally, in the non-lean group, these changes in 2-h post plasma glucose and WBISI were comparable following both passive and moderate cycling despite the substantial differences in EE (moderate-intensity cycling EE was more than $5 \text{ kcal} \cdot \text{min}^{-1}$ greater than passive cycling). This finding is similar to previous research comparing light and moderate-intensity walking (12) and suggests that even small increases in EE can contribute to lower postprandial blood glucose response. When designing the study, moderate-intensity cycling was included so that we could compare passive cycling to an intervention that is known to lower blood glucose levels and our results suggest moderate-intensity cycling can be an effective intervention. However, passive cycling is associated

with less physical exertion, so it is likely to be more easily incorporated into a work environment. Therefore, passive cycling will be the focus throughout the discussion.

While previous research has shown that sitting worsens cardiometabolic risk factors independent of adiposity (21,22,34), it should not be surprising that we found different glucose and WBISI responses between our adiposity groups. Adipose tissue contributes to impairments in glucose tolerance (19) and the significantly greater 2-h post plasma glucose, greater plasma glucose iAUC, and lower WBISI following the sitting trial suggest our non-lean participants had impaired glucose tolerance compared to the lean participants. Additionally, despite similar levels of physical activity between groups, the lean group had a significantly greater estimated $\dot{V}O_2\text{max}$ compared to the non-lean group. $\dot{V}O_2\text{max}$ has a significant genetic as well as physical activity component. Since the groups were similarly low in physical activity levels, the differences in $\dot{V}O_2\text{max}$ may be the result of a stronger genetic influence. Genetic fitness levels, independent of physical activity levels, are associated with decreased cardiovascular risk (36), which suggests our lean participants may have had a healthier genetic profile. Because of these differences in cardiometabolic profiles between groups, it is possible the acute passive and moderate intensity cycling interventions did not provide enough of a stimulus to further lower blood glucose in our lean group.

Of note, when participants were divided into either lean or non-lean groups, body fat percentage was used instead of BMI. This resulted in one subject with a non-lean BMI but lean body fat percentage and six participants with a lean BMI but a non-lean body fat percentage. Our decision to split groups by body fat percentage was based on previous research showing body fat percentage is better related to cardiovascular disease risk compared to BMI (10). The current study supports this previous research and demonstrates that within a group with a predominately lean BMI, percent body fat influences the glucose and insulin response following physical activity. For the lean group, while there was no difference in the 2-h post plasma glucose levels, there was an unexpected increase in plasma glucose iAUC following the passive cycling trial compared with the sitting trial. However, because the study was not powered *a priori* to detect differences between groups, it is possible that this is due to a type II error.

Many previous researchers have utilized study designs in which participants performed an intervention immediately following the consumption of a test drink/meal (12,29). With this study design, both light- and moderate-intensity walking have been shown to lower plasma glucose levels to a similar extent (12). It is likely we would have found comparable results for passive and moderate-intensity cycling if we had also utilized this study design. However, in the present study, that order was reversed with participants performing a single acute condition of passive cycling and then performing the OGTT while sitting allowing for the determination of how glucose metabolism is influenced by passive cycling once the cycling bout is complete. Despite our conservative study design, changes in 2-h post plasma glucose and WBISI were still found following passive and moderate-intensity cycling in the non-lean group. A potential mechanism for these changes may be the higher muscle activity that occurs during passive cycling which has been shown in previous studies using electromyography (EMG) (4,32). Muscle activity elicits insulin-independent pathways that promote the uptake of glucose (9). Such changes are only temporary unless repeated bouts

of physical activity are performed (8) so repeated bouts of passive cycling likely are needed to maintain the observed glucose and insulin responses reported in our study. Since it has been suggested that lean participants need a greater stimulus to attain significant decreases in cardiometabolic risk factors (5), it is possible that a longer term passive cycling intervention could result in measurable improvements in lean participants.

Increased muscle activity during passive cycling may also play a role in improving other risk factors. For example, increased muscle activity is the likely source for the increase in EE. This increase in EE is significant in that it can help combat diseases like obesity. In fact, passive cycling increases EE more than other commonly proposed sitting interventions like standing or sitting on an exercise ball (3). Furthermore, muscle activity is associated with changes to other cardiometabolic risk factors like HDL and triglycerides (30), lipoprotein lipase (7), and genes involved with overall homeostasis (38). It is possible that passive cycling may lower these other cardiometabolic risk factors and help to further counteract the deleterious effects of sitting; something that should be examined in longer term follow-up studies.

In addition to improving cardiometabolic risk factors, another important consideration for an intervention is that it should not negatively affect workplace productivity. No measureable cognitive deficits were observed during passive cycling compared to sitting. Findings from our measures of cognition suggest either passive or moderate-intensity cycling could be used in a workplace environment. This is in agreement with previous research that found a cycling workstation did not influence typing speed (14) although others have found work performance was lower during light-intensity cycling (33). Conflicting data exist for many interventions regarding work productivity and may be related to the tasks and measures being utilized (2,5,6,14,25,33). Thus, our findings suggest that passive cycling will not negatively influence workplace productivity, however, measures of work specific tasks are needed to determine whether passive cycling influences actual work productivity.

There are a variety of ways to implement passive cycling in the workplace environment. For example, continuous passive cycling could be performed to persistently increase EE. Another option would be to use passive cycling intermittently to break up prolonged sitting much like other studies have used walking (12,29). To break up sitting with walking requires an expensive treadmill workstation or requires individuals to leave their workstation. Passive cycling on the other hand, would allow for individuals to remain at their desk resulting in less disruption to work productivity.

There are some limitations to the present study. Only males were studied and there is some evidence that prolonged sitting negatively impacts females to a greater extent compared to males (13). We have previously found no difference in males and females regarding the EE response to passive cycling (31,32) but it is possible that passive cycling may result in a different blood glucose response in females compared to males so females should be included in future research. Also, the average age of our participants was only 25.5 ± 7.7 and older individuals may not respond in the same way to passive cycling. Lastly, physical activity was only measured for three days prior to the lab visits and it is possible these three days were not representative of the weekly physical activity of participants. Although no

differences were found in physical activity for the three measured days, the lean participants could have higher weekly physical activity and this could explain the differences in $\dot{V}O_2\text{max}$ and plasma glucose between adiposity groups.

In conclusion, across all physically inactive participants, passive cycling doubled EE compared to sitting. Further, 2-h post plasma glucose was lower and WBISI higher in the non-lean participants following passive cycling compared to sitting. Lean participants did not show changes in these variables perhaps due to differences in their cardiometabolic profile. Additionally, passive cycling did not significantly impact cognition. Thus, we propose that passive cycling is a feasible intervention to decrease sedentary sitting in the workplace. Future research is needed to determine feasibility and long-term effectiveness in a real world workplace environment.

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Figure 1.
Overview of the experimental sessions

Figure 2.
Diagram of the stationary passive cycle.

Figure 3.

The mean $\pm$ SD of select physiological results for the two adiposity groups. While energy expenditure was higher during moderate-intensity cycling compared to passive cycling, both cycling conditions resulted in similar changes for WBISI and 2-h post plasma glucose. Open bars/symbols represent the lean group while filled bars represent the non-lean group. A: energy expenditure. B: whole body insulin sensitivity index (WBISI). C: plasma glucose incremental area under the curve (iAUC). Squares represent the sitting trial, circles the passive cycling trial, and triangles the moderate-intensity cycling trial. D: 2-h post plasma glucose. * Significantly different than lean group. † Significantly different than the sitting condition. ‡ Significantly different than the passive cycling condition.

Table 1
Mean±SD for the sitting, passive cycling, and moderate-intensity cycling conditions for the two adiposity groups.

	Lean			Non-Lean		
	Sitting	Passive Cycling	Moderate-Intensity Cycling	Sitting	Passive Cycling	Moderate-Intensity Cycling
Physiological Measures	2-h Post Plasma Glucose (mmol·L ⁻¹)	5.9 ± 1.2	6.1 ± 1.5	5.8 ± 0.8	7.7 ± 1.3 *	6.8 ± 1.2 **
	Plasma Glucose iAUC (mmol·L ⁻¹ ·min)	164 ± 114	233 ± 111 †	183 ± 86	363 ± 87 *	303 ± 112 **
	Insulin iAUC (mmol·L ⁻¹ ·min)	33297 ± 16851	36843 ± 10714	39135 ± 16367	56516 ± 34809	51463 ± 34062 †
	WBISI	5.57±2.19	5.54±2.72	5.30 ± 1.6	2.74±0.86 *	3.41 ± 1.38 **
	Energy Expenditure (kcal·min ⁻¹)	1.25 ± 0.19	2.68 ± 0.71 †	8.17 ± 1.83 ††	1.24 ± 0.16	7.39 ± 1.73 ††
	Heart Rate (BPM)	69.0 ± 9.9	78.7 ± 8.6 †	137.3±7.6 ††	72.9 ± 6.9	134.6±9.0 ††
	Step Count	5617 ± 2604	5714 ± 2149	5467 ± 2348	6204 ± 1815	6331 ± 1580
Average Physical Activity Measures for the 3 Days Prior to the Trials	Time Spent in Moderate to Vigorous Physical Activity (min)	32.3 ± 26.2	30.4 ± 16.5	28.1 ± 21.2	35.6 ± 20.0	38.5 ± 19.2
	Time Spent Sedentary (min)	534.9 ± 102.4	523.1 ± 115.4	539.6 ± 148.1	570.0 ± 127.9	599.5 ± 130.0
	Activity Monitor Wear Time (hr)	12.1 ± 2.0	12.2 ± 2.3	12.3 ± 3.0	13.0 ± 2.5	13.8 ± 2.4
Cognitive Measures		Sitting		Passive Cycling	Moderate-Intensity Cycling	
	PVT Reaction Time (ms)	340.0 ± 28.8		344.1 ± 31.0	332.3 ± 27.2 †	
	PVT Lapses	1.2 ± 1.7		1.1 ± 2.0	1.0 ± 1.1	
	Mannequin Throughput Score	26.4 ± 8.4		25.9 ± 8.2	26.6 ± 8.6	
	Math Throughput Score	19.7 ± 3.5		19.1 ± 5.2	20.3 ± 5.7	

* Significantly different than the lean group

† Significantly different than the sitting trial

‡ Significantly different than the passive trial

Table 2

Descriptive data for the two adiposity groups. Values given are mean $\pm$ SD for n = 24 (11 lean and 13 non-lean).

	Lean (n=11)	Non-Lean (n=13)
Age (years)	22.9 $\pm$ 4.1	27.8 $\pm$ 9.3
Weight (kg)	66.2 $\pm$ 8.1	77.0 $\pm$ 12.7 *
BMI (kg·m ⁻²)	20.7 $\pm$ 2.2	25.3 $\pm$ 3.1 *
Body Fat (%)	14.0 $\pm$ 3.1	29.5 $\pm$ 4.7 *
Total Leg Lean Mass (kg)	26.1 $\pm$ 3.7	24.4 $\pm$ 4.2
Estimated VO ₂ max (mL·kg ⁻¹ ·min ⁻¹)	48.9 $\pm$ 7.9	38.7 $\pm$ 4.3 *
Maximum Heart Rate (beats·min ⁻¹)	195.8 $\pm$ 7.6	191.6 $\pm$ 10.9
Fasting Plasma Glucose (mmol·L ⁻¹)	4.8 $\pm$ 0.3	5.1 $\pm$ 0.3

* Significantly different from the lean group.