ENERGY EXPENDITURE DURING PASSIVE CYCLING: THE EFFECTS OF LEG MASS, CADENCE, AND ADAPTATION

by

James E. Peterman

B.A., University of Colorado at Boulder, 2007

A thesis submitted to the
Faculty of the Graduate School of the
University of Colorado in partial fulfillment
of the requirement for the degree of
Master of Science
Department of Integrative Physiology
2010

This thesis entitled: Energy Expenditure During Passive Cycling: The Effects of Leg Mass, Cadence, and Adaptation

written by James E. Peterman has been approved for the Department of Integrative Physiology

Rodger Kram, Ph.D.
William C. Byrnes, Ph.D.
Edward L. Melanson, Ph.D.
Data

The final copy of this thesis has been examined by the signatories, and we find that both the content and the form meet acceptable presentation standards of scholarly work in the above mentioned discipline.

IRB protocol # 0509.22

Abstract

Peterman, James E. (M.S., Integrative Physiology)

Energy Expenditure During Passive Cycling: The Effects of Leg Lean Mass, Cadence, and Adaptation

Thesis directed by Associate Professor William C. Byrnes

Passive cycling, during which a motor drives the pedals, increases energy expenditure above rest. However, little is known about the factors that influence energy expenditure during passive cycling. Our purpose was to quantify how leg mass, cycling cadence, and adaptation influence the energy expenditure during passive cycling. Eleven sedentary to recreationally active non-cyclists (5M, 6F, age 18-30) participated. The role of leg mass was studied by comparing one- and two-leg passive cycling. Cycling trials were performed at both 60 and 90 RPM to study the influence of cycling cadence. Adaptation was studied using multiple cycling trials and a 30-minute passive cycling trial. Rest and active (no load) cycling trials were performed for energy cost comparisons. Passive cycling significantly (p<0.03) increased energy expenditure above rest for all cycling trials. The increases in energy expenditure during passive cycling were greater when two legs were compared to one leg (37% vs. 15% at 60 RPM; p=0.008 and 93% vs. 44% at 90 RPM; p=0.001). The increase in energy expenditure was greater for 90 RPM compared to 60 RPM two-leg passive cycling (93% vs. 37%; p<0.001). The increase in energy expenditure was repeatable over multiple trials and was sustained for exercise durations of 30-minutes. Compared to the passive cycling trials, energy expenditure was significantly greater during active (no load) cycling at 60 and 90 RPM.

In conclusion, increases in energy expenditure during passive cycling are directly related to the amount of activated leg mass and cycling cadence and not influenced by adaptation.

Acknowledgements

First off, I would like to thank my committee for taking on the project that is my thesis. I would like to thank Ed Melanson for donating his time and energy to reviewing my thesis. I'd also like to give a big thanks to my two advisors, Dr. William Byrnes and Dr. Rodger Kram. They not only helped guide me through this process but also taught me how to think like a scientist. Their genuine interest in my success was a constant motivator and has furthered my passion for studying physiology. Although the thesis revision process had its rough spots (whether they know it or not), their positive feedback helped me get through it all.

I would also like to thank the folks that helped with this project in some form or another. To the folks that helped with the data collection (Ben Ryan, Eric Homestead, Taylor Schrock, and Paul Walden), I know it wasn't the most exciting protocol but your assistance was greatly appreciated. Thanks also goes out to Micky Reger for showing me how to use the lab equipment and Chris Arellano for helping with a variety of the little things. And of course I would like to thank my subjects for donating their time to be participants in my study. Last but not least, I would like to thank Liz and my family for their support through these past years. Thanks for your words of encouragement and listening when I needed to vent.

Table of Contents

Chapter

I.	Introduction
II.	Methods4
Ш	. Results
IV	Discussion
v.	References
	endix Literature Review29
В.	Between Subjects Comparison

List of Tables

1. Descriptive subject data	5
2. Schedule for experimental session	10
3. Comparison of first and second two-leg trials at both 60 and 90 RPM	13
4. Mean values for indirect calorimetry parameters and heart rate	14
5. Mean values for 30 minute trial	17
6. Cadence and VO ₂ data from previous research	37

List of Figures

1.	Diagram of the cycle ergometer	8
2.	Energy expenditure means for the cycling trials	15
3.	Energy expenditure vs. time during the 30 minute passive cycling trial	18

Introduction

A sedentary lifestyle is a major contributor to obesity and a risk factor for cardiovascular disease and metabolic disorders such as diabetes (1.14.16). Sedentary leisure time activities along with sedentary work environments are exacerbating these problems (26). The American College of Sports Medicine (ACSM) recommends performing moderate-intensity physical activity to help with weight management and overall health (1,16). However, the majority of individuals do not meet these recommendations for physical activity (25). Recent research has also found that individuals who meet the recommendations for physical activity but spend the rest of their time inactive still have increased metabolic risk variables such as increased waist circumference, increased systolic blood pressure, and decreased insulin sensitivity according to 2-h plasma glucose levels. (18,39). Therefore, some researchers have suggested decreasing sedentary time and increasing low-intensity physical activity levels like those classified as non-exercise activity thermogenic (NEAT) (e.g. taking the stairs, walking to the car, fidgeting, etc.) (5,15,17,19,39). Increased levels of NEAT have been linked with healthier blood glucose concentrations (17), increased lipoprotein lipase activity (5), and decreased cardiovascular disease related mortality (11).

In addition to modifying risk factors associated with diabetes and cardiovascular disease, NEAT might also aid in the regulation of body mass. Hill et al. have suggested that an imbalance of only 100 kcal/day is the main driving force behind the obesity epidemic (19). Thus, even small increases in energy expenditure could aid with weight management. For example, the "walking workstation" idea presented by Levine et al. provides a novel way to increase NEAT by allowing individuals to perform a normally

sedentary job in a manner that is more active (29,44). A recent study in our lab (40), found that short duration passive cycling (in which an external motor moves a subject's legs while they contribute no volitional muscle activation) increases energy expenditure by a similar magnitude as the walking workstation. Therefore, passive cycling may be another tool that could be used to increase daily levels of NEAT.

Passive cycling has been studied since at least 1949 (2,38) as a way to examine how peripheral afferent neural input controls ventilation. Most ventilation physiologists however, dislike passive cycling because the increase in VO₂ during passive cycling is seen as a confounding variable (3). Unlike these previous researchers though, we see the increase in VO₂ as a positive because it suggests passive cycling could potentially be used as a novel tool to increase daily levels of NEAT. But little is known about the factors that influence the magnitude of the observed increase in energy expenditure. Specifically, no study to date has carefully examined how leg mass, cycling cadence, or adaptation influence energy expenditure during steady state passive cycling. Previous electromyographic research has demonstrated that despite the lack of volitional activation, the leg muscles are activated during passive cycling, which increases energy expenditure rate (3,40). It would follow that the leg mass involved in passive cycling would quantitatively correspond to the increase in energy expenditure. It has also been suggested that cycling cadence plays a role in the increase in energy expenditure, though that notion was based on non-steady state passive cycling (37). A previous study by Krzeminski et al. found there was no adaptation to repeated trials of passive cycling across two five minute trials separated by 1-2 weeks (24). However, the short duration trials separated by such a long time may have provided enough time to wash out any

potential learning affect. Although one study has examined passive cycling over a duration of 30 minutes, only average values for the entire 30-minute trial were reported giving no indication as to how energy expenditure may change over time (4). Other studies in this area were not designed to examine the increase in energy expenditure and focused on trials lasting no longer than five minutes (2,3,9,37,40,43,45). If passive cycling is to be used as a tool for decreasing chronic disease risk factors and weight management, it is important to understand how the elevation in energy expenditure can be maximized and if this elevation persists over time.

The purpose of this study was to explore the factors that influence energy expenditure during passive cycling. Based on previous research, we chose to investigate leg mass, cycling cadence, and adaptation as potential factors influencing energy expenditure during passive cycling. We hypothesized that energy expenditure during passive cycling would be directly influenced by leg mass and cadence. We also investigated whether or not the body's energy expenditure response adapts to the passive movement of the limbs by using repeated trials and a 30-minute trial. We hypothesized that the rate of energy expenditure would be consistently elevated during repeated and extended passive cycling trials.

Methods

This experiment was designed to examine whether leg mass, cycling cadence, and/or adaptation influence the magnitude of passive cycling energy expenditure. The influence of leg mass was studied by comparing one-leg and two-leg passive cycling since only half of the mass is involved during one-leg passive cycling. The effect of pedaling cadence on energy expenditure was examined by the use of two different cadences during the passive cycling trials. The influence of adaptation was determined by conducting multiple trials and one extended passive cycling trial. Finally, to provide an energy cost comparison to passive cycling, active cycling trials with no resistance were performed.

Subjects

Eleven sedentary to recreationally active healthy subjects (5 males, 6 females) volunteered for this study. A previous study in our lab using passive cycling examined experienced cyclists (40). However, it is possible that training on a bicycle may affect the response to passive cycling. Therefore, we excluded subjects if they trained on a bicycle, recreationally rode a bicycle, and/or commuted by bicycle more than two days per week. Females who were pregnant were excluded from the study because a DXA scan was required which involves a small exposure to radiation. Descriptive data for our subjects are given in Table 1. All subjects were informed of the risks involved with participation in the study and gave written informed consent before participating. The experimental protocol for this study was approved by the University of Colorado at Boulder Institutional Review Board (IRB).

Table 1. Descriptive data for male and female subjects. Values given are mean \pm SD for n = 11 (5 males and 6 females).

	Males	Females
Age (years)	22.7 ± 4.3	22.9 ± 2.4
Height (m)	1.76 ± 0.03	1.66 ± 0.05 *
Body Mass (kg)	65.6 ± 6.0	61.4 ± 10.6
вмі	21.3 ± 1.5	22.3 ± 2.7
Body Fat (%)	20.6 ± 3.4	32.6 ± 9.0 *
Fat Mass (kg)	13.0 ± 2.1	19.8 ± 7.9
Lean Body Mass (kg)	50.4 ± 5.9	39.2 ± 4.9*
Leg Lean Mass (kg)	24.8 ± 3.4	19.7 ± 2.8*

^{*} Significantly different from males

Preliminary Procedures

Subjects came to the laboratory for two separate sessions. The first session began with a whole body DXA scan (GE LUNAR DXA system) to determine the whole body lean mass (fat-free and bone-free mass) of each subject. During the preliminary session, subjects also performed tests for leg dominance. Previous research has suggested that the leg used during passive knee extension affects the ventilation response (21). Therefore, it is possible that other respiratory factors could also be influenced by the leg used during passive cycling. To control for this potential confounding variable, subjects used their dominant limb during the one-leg passive cycling. Three functional tests (20) were used for determining the dominant limb. The first test was balance recovery. The researcher stood behind the subject and applied a light nudge that was strong enough in force that the subject was required to take a step to maintain balance. For the second test, subjects were asked to step up onto a step that was 20 cm high. The third test involved a subject kicking a ball as accurately as possible between two cones placed 1m apart and 10m away from the subject. The three functional tests were performed three times to verify the results with the same sequence of the tests for each subject. The leg most often used to perform each task was considered the dominant limb.

Experimental Procedures

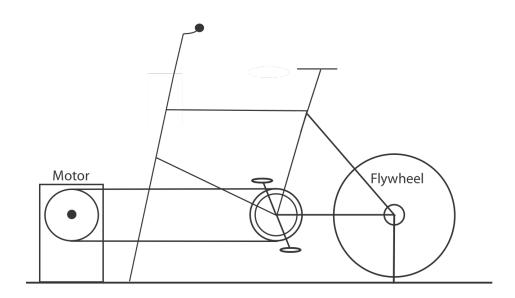
Within two weeks of the preliminary session, subjects reported to the lab to perform the cycling trials. Subjects arrived at least three hours post-prandial to reduce the influence from the thermic effect of food on overall energy expenditure. Because we were interested in examining adaptation, subjects were not allowed to become

familiarized with passive cycling before the experimental procedures. Thus, the first passive cycling trial was the first time that the subjects had ever experienced passive cycling.

A diagram of the modified bicycle ergometer used for the passive and active cycling trials is shown in Figure 1. Toe clips and straps were used to secure the subject's feet on the pedals. During the passive cycling trials, a chain connected the motor to the cranks. For the active cycling trials, this chain was removed and another chain was used to connect the cranks to the flywheel. During the one-leg cycling trials, the pedal for the non-dominant leg was removed, allowing subjects to rest their non-dominant leg on a box placed next to the ergometer. For the active (no load) cycling trials, there was no resistance placed on the flywheel. To help subjects pedal at the desired cadence, an audible metronome was used. A cyclocomputer with a cadence sensor was used by the experimenter to monitor that subjects were pedaling at the appropriate cadence.

The experimental session started with a seated resting trial on the bicycle ergometer with the cranks kept horizontal. Following the resting trial were three passive cycling trials at a cadence of 60 revolutions per minute (rpm): two-leg passive cycling, dominant one-leg passive cycling, and then two-leg passive cycling again. Two active, no-load cycling trials followed. During the first active trial, subjects volitionally pedaled at 60 rpm with no resistance and during the second active trial subjects volitionally pedaled at 90 rpm with no resistance. After the active cycling trials, the passive cycling trials were repeated but at 90 rpm. The trials followed the same order: two-leg passive cycling, one-leg passive cycling, and then two-leg passive cycling. A seated resting trial

Figure 1: Diagram of the modified bicycle ergometer. During active cycling the chain is removed from the motor and attached to the sprocket on the flywheel.



then immediately followed. All of the above trials lasted for five minutes except for the last two-leg passive cycling trial at 90 rpm and the last resting trial. The last passive cycling trial was extended to 30 minutes and the last resting period was extended to 10 minutes. A summary of the trial order is given in Table 2.

During the experimental trials, rate of oxygen consumption (VO₂), rate of carbon dioxide production (VCO₂), expired pulmonary ventilation rate (V_E), tidal volume (V_T), respiratory rate (RR), energy expenditure (EE), respiratory exchange ratio (RER), and heart rate (HR) were measured using a computerized indirect calorimetry system by Parvomedics (Sandy, UT). The indirect calorimetry system was calibrated before each testing session. Gas fractions were calibrated with a primary standard gas mixture within the physiological range (16.01% O₂ and 4.01% CO₂). The volume was calibrated using a 3L syringe at five distinct flow rates within the expected range of the study protocol. Calibration was considered to be complete when recorded volumes were within 1% of the calibration volumes, and gas fractions were within 0.3% of calibration values (e.g. 20.93 \pm 0.06%).

Respiratory and heart rate measurements were averaged every 15 seconds. To ensure that only steady-state values were used, data during the last two minutes of each five minute trial were recorded (minutes 3:00 - 5:00). To determine if elevated energy expenditure persisted during the extended passive cycling trial, measurements were taken for the entire trial and averaged every five minutes. The average values from minutes 3:00 - 5:00 during the extended passive cycling trial were also calculated so that the trial could be compared to the other shorter trials. For the extended resting period, data from minutes 5:00 - 10:00 were averaged.

 Table 2: Schedule for the Experimental Session

	Passive Cycling at 60 RPM				No Load) ling	Passi	ve Cycling at	90 RPM	
Pre- Treatment Rest	First Two- Leg Trial	One-Leg Trial	Second Two-Leg Trial	60 RPM Trial	90 RPM Trial	First Two- Leg Trial	One-Leg Trial	Second Two- Leg Trial/ Prolonged Trial *	Post- Treatment Rest *

^{*} Trials that were extended beyond 5 minutes. The second two-leg passive cycling at 90 rpm was extended to 30 minutes and the post-treatment resting trial lasted for 10 minutes. There was also no break between these last two trials.

Analysis

Data were analyzed using SPSS software (SPSS Inc., Chicago, IL). Mean values were compared across trials using a repeated measures ANOVA with a LSD post-hoc procedure. Statistical significance was designated at the p=0.05-level. All values are expressed as means ± standard deviation (SD).

Results

Passive cycling increased the rate of energy expenditure significantly compared to rest. Both greater leg mass and faster cycling cadence increased the rate of energy expenditure during passive cycling. During multiple passive cycling trials, the rate of energy expenditure consistently increased above rest. Furthermore, during the 30-minute extended passive cycling trial, the rate of energy expenditure increased above rest and remained above rest for the duration of the trial.

There were no significant differences in energy expenditure rates between the first and second two-leg passive cycling trials for either 60 or 90 RPM (Table 3). Therefore, the values from the two separate two-leg passive cycling trials were averaged and compared to the other trials. There was also no sex effect so the results were pooled for males and females.

Average rate of energy expenditure was 1.28 ± 0.23 kcal/min at rest and was significantly increased during one-leg passive cycling at both 60 RPM (1.49 ± 0.28 kcal/min, p = 0.003) and 90 RPM (1.85 ± 0.25 kcal/min, p<0.001). The rates of energy expenditure during the two-leg passive cycling trials also were greater than rest for both 60 RPM (1.78 ± 0.35 kcal/min, p<0.001) and 90 RPM (2.51 ± 0.58 kcal/min, p<0.001). For the comparison of one- and two-leg passive cycling, the increases in energy expenditures during the one-leg trials (0.21 ± 0.16 and 0.58 ± 0.26 kcal/min at 60 RPM and 90 RPM respectively) were roughly half of the increase measured for two-leg trials (0.50 ± 0.24 and 1.23 ± 0.53 kcal/min at 60 RPM and 90 RPM respectively) (Table 4, Figure 2).

Table 3. Respiratory gas exchange and heart rate during the first and second two-leg trials at both 60 and 90 RPM. Except for heart rate, the first and second trials at 60 RPM did not differ significantly. For passive cycling at 90 RPM, all variables during the first and second trials did not differ significantly. Values given are mean \pm SD for n = 11.

	First Passive 60 RPM Two-Leg Trial	Second Passive 60 RPM Two-Leg Trial	First Passive 90 RPM Two-Leg Trial	Second Passive 90 RPM Two-Leg Trial
$\dot{V}O_2$ (L/min)	0.38 ± 0.08	0.36 ± 0.07	0.52 ± 0.12	0.52 ± 0.14
^{VCO} ₂ (L/min)	0.31 ± 0.06	0.29 ± 0.06	0.43 ± 0.08	0.43 ± 0.10
\dot{V}_{E} (L/min)	12.8 ± 2.7	12.7 ± 2.6	17.3 ± 3.2	17.3 ± 3.3
EE (kcal/min)	1.82 ± 0.38	1.74 ± 0.34	2.51 ± 0.561	2.50 ± 0.63
Ventilatory Equivalent $(\dot{V}_E/\dot{V}O_2)$	34.11 ± 4.71	35.15 ± 4.20	33.67 ± 3.96	33.80 ± 3.47
HR (BPM)	86.7 ± 13.3 *	82.0 ± 11.6	92.0 ± 12.9	89.9 ± 14.0

^{*} Significantly different from the second trial

Table 4. Comparison of the indirect calorimetry parameters and heart rate values during rest, passive cycling, and active (no load) cycling. The first and second two-leg passive cycling trials for both 60 and 90 RPM were averaged because no significant difference was found. The averages are given in the passive two-leg columns. Values given are mean \pm SD for n = 11.

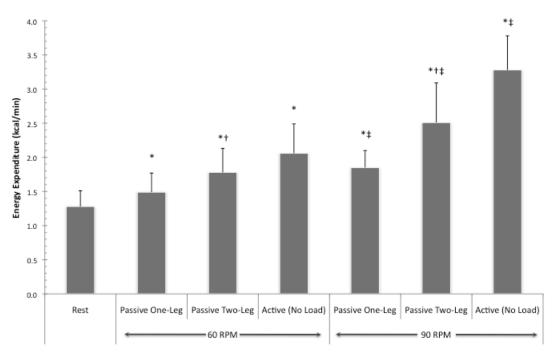
		60 RPM						
	Pre- Treatment Rest	Passive One- Leg	Passive Two-Leg	Active (No Load)	Passive One- Leg	Passive Two-Leg	Active (No Load)	Post-Treatment Rest
$\dot{V}O_2$ (L/min)	0.27 ± 0.05	0.31 ± 0.06 *	0.37 ± 0.07 *†	0.43 ± 0.09 *	0.39 ± 0.06 *‡	0.52 ± 0.13 *†‡	0.68 ± 0.10 *‡	0.28 ± 0.05
$\dot{V}CO_2$ (L/min)	0.22 ± 0.05	0.25 ± 0.05 *	0.30 ± 0.06 *+	0.35 ±0.08 *	0.31 ± 0.04 *‡	0.42 ± 0.09 *†‡	0.56 ± 0.09 *‡	0.23 ± 0.05
V (1 /min)		11.63 ± 2.25	12.79 ± 2.53 *+	14.54 ± 2.74 *	14.27 ± 1.91 *‡	17.31 ±3.14 *†‡	21.16 ± 3.45 *‡	10.48 ± 1.50
EE (kcal/min)	1.28 ± 0.23	1.49 ± 0.28 *	1.78 ± 0.35 *†	2.06 ± 0.43 *	1.85 ± 0.25 *‡	2.51 ± 0.58 *†‡	3.28 ± 0.50 *‡	1.37 ± 0.26
Ventilatory Equivalent $(\dot{V}_E/\dot{V}O_2)$	39.25 ± 5.48	37.62 ± 5.04	34.63 ± 4.29 *†	34.38 ± 4.26 *	37.10 ± 3.46	33.74 ± 3.37 *+	31.05 ± 3.15 *#	37.18 ± 3.20
RER	0.82 ± 0.08	0.81 ± 0.05	0.81 ± 0.4	0.81 ± 0.06	0.82 ± 0.06	0.83 ± 0.06	0.81 ± 0.05	0.82 ± 0.04
HR (BPM)	88.5 ± 17.8	82.4 ± 13.1 *	84.3 ± 12.3	87.2 ± 13.2	84.8 ± 13.9	91.0 ± 13.3 †‡	101.4 ± 15.2 *‡	87.7 ± 20.9

^{*} Significantly different from resting values

[†] Significantly different from one-leg trial

[‡] Significantly different from respective 60 RPM trial

Figure 2: Energy expenditure during the passive and active (no load) cycling trials. All cycling trials resulted in significantly greater energy expenditure rates compared to rest. The passive two-leg cycling trials resulted in a greater energy expenditures compared to the passive one-leg trials. Cycling trials at 90 RPM also had greater energy expenditure rates compared to the respective cycling trials at 60 RPM (mean \pm SD).



^{*} Significantly greater than resting values

[†] Significantly greater than one-leg trial

[‡] Significantly greater than respective 60 RPM trial

Compared to the 60 RPM trials, the energy expenditure rates during the 90 RPM trials were 24%, 41%, and 59% greater during one-leg, two-leg, and active (no load) cycling (Table 4). The rates of energy expenditure during the active (no load) trials compared to the two-leg passive trials were 16% and 31% greater for 60 and 90 RPM respectively (Table 4). During the 30-minute two-leg passive cycling trial, there was a significant increase in the rate of energy expenditure above resting values that persisted for the duration of the trial (Table 5 and Figure 3).

Both VO_2 and VCO_2 increased during passive cycling and did so nearly in parallel. Thus, the RER did not change significantly compared to rest for any of the trials (Table 4). The consistent RER values throughout the experiment suggest that the blend of carbohydrate and fat metabolism did not change during the experiment. The V_E followed the same trends as the energy expenditure, except that, for passive cycling at 60 RPM with one leg, ventilation did not increase significantly above rest (Table 4).

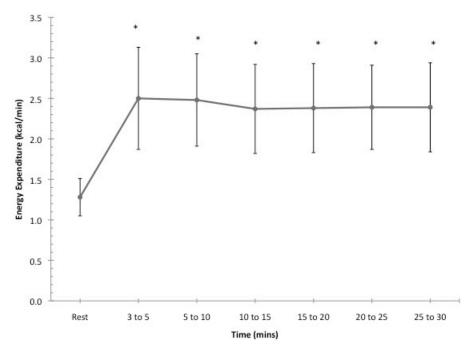
Heart rate showed no general trends. The average heart rates at rest and during two-leg passive cycling at 60 and 90 RPM were 88.5 ± 17.8 BPM, 84.3 ± 12.3 BPM, and 91.0 ± 13.3 BPM respectively. During these trials, the heart rate between subjects ranged from 64.3 - 124.7 BPM, 64.8 - 103.5 BPM, and 64.0 - 105.7 BPM respectively.

Table 5. Comparison of the indirect calorimetry parameters and heart rate values during the extended (30 minute) passive cycling trial at 90 RPM. Values given are mean \pm SD for n = 11.

	Rest	3 to 5 mins	5 to 10 mins	10 to 15 mins	15 to 20 mins	20 to 25 mins	25 to 30 mins
$\dot{V}O_2$ (L/min)	0.27 ± 0.05	0.52 ± 0.14	0.52 ± 0.12	0.49 ± 0.12	0.50 ± 0.12	0.50 ± 0.11	0.50 ± 0.12
VCO₂ (L/min)					0.40 ± 0.09		
	0.22 ± 0.05	0.43 ± 0.10	0.42 ± 0.09	0.41 ± 0.09	*(3-5)	0.40 ± 0.09	0.39 ± 0.09
$\dot{V}_{\scriptscriptstyle E}$ (L/min)	10.37 ± 2.00	17.27 ± 3.24	17.20 ± 2.63	16.61 ± 2.52	16.41 ± 2.94	16.36 ± 2.83	16.51 ± 3.49
				2.37 ± 0.55			
EE (kcal/min)	1.28 ± 0.23	2.50 ± 0.63	2.48 ± 0.57	*(5-10)	2.38 ± 0.55	2.39 ± 0.52	2.39 ± 0.55
Ventilatory							
Equivalent							
$(\dot{V}_E/\dot{V}O_2)$	39.25 ± 5.48	33.80 ± 3.47	33.96 ± 3.84	34.18 ± 3.27	33.44 ± 2.95	33.17 ± 2.86	33.27 ± 4.89
				90.5 ± 14.4			
				*(20-25 &			
HR (BPM)	88.5 ± 17.8	89.9 ± 14.0	90.6 ± 13.6	25-30)	91.8 ± 15.0	92.5 ± 16.0	92.9 ± 15.5

^{*} Significantly different from trial specified in parentheses

Figure 3: The mean \pm SD of energy expenditure during the extended (30 minute) bout of passive cycling at 90 RPM. During the extended trial, the average energy expenditure was 89% greater than rest and consistently remained elevated for the duration of the trial.



^{*} Significantly greater than rest

Discussion

Based on our results, we fail to reject our hypotheses. The rate of energy expenditure during passive cycling was directly related to the amount of leg mass involved with the passive cycling movement. The rate of energy expenditure during passive cycling also increased with cadence. Lastly, there was no adaptation to passive cycling. We found that the elevated energy expenditure rates persisted throughout repeated trials and a 30-minute prolonged trial.

Like previous research, our results show that passive cycling increases the rate of energy expenditure compared to rest. For example, two-leg passive cycling at 60 RPM elicited an increase in VO_2 of 0.11 ± 0.05 L/min. Previous studies utilizing passive cycling at 60 RPM have found that VO_2 increased above rest by 0.08 L/min (4), 0.07 L/min (9), and 0.10 L/min (45). In this study, we also found that two-leg passive cycling at 90 RPM increased VO_2 by 0.26 ± 0.11 L/min, which is similar to the increase of 0.28 L/min found in a previous study in our lab (40).

Our cycling cadence results are also similar to a recent study by Muraki et al. (37) that found VO₂ increased with cadence during passive cycling. We found that at 90 RPM, the energy cost of passive cycling was 29% greater compared to at 60 RPM. In their study, Muraki et al. increased the cycling cadence by 5 RPM every minute from 20 to 70 RPM and concluded that there is a minimum cadence required to elicit a VO₂ response that is roughly 25-40 RPM. However, since the cycling cadence was increased every minute, Muraki et al. likely did not allow subjects to reach a steady-state at each cadence. Therefore, their determination of the VO₂ at each cadence and their estimate of the minimum threshold cadence may be too low. Our results support Muraki et al.'s

general finding that an increase in cadence results in an increase in energy expenditure and allow for a more quantitative assessment of VO_2 at different cadences.

Previous studies measuring oxygen consumption rates during passive cycling (3,4,9,24,38,43,45) have reported VO₂ values ranging from 0.31 to 0.57 L/min for cadences ranging from 45 to 90 RPM. With the exception of one study (43) that utilized a cadence of 45 RPM, this previous research suggests there is a linear increase in VO₂ with cadence. After excluding the study that utilized a cadence of 45 RPM, when the VO₂ is divided by cadence, the cost per crank revolution is roughly constant with a mean $(\pm \text{SD})$ of 6.6 ± 1.0 ml O₂/rev. In our study, the costs were 6.2 ± 1.2 and 5.8 ± 1.4 ml O₂/rev for 60 and 90RPM respectively, with no significant difference between cadences (p=0.134). This constant VO₂ suggests that passive cycling activates the same amount of muscle mass during each revolution, independent of cadence. Cadences faster than 90RPM would likely increase VO₂ further but may not be comfortable/practical.

Our results are also consistent with previous research that found the elevated energy expenditure during passive cycling persists. In our study, subjects performed two separate two-leg passive cycling trials at both 60 and 90 RPM. We found that there was no significant difference in the rate of energy expenditure between the two repeated two-leg passive cycling trials. Our results are thus similar to a previous study by Krzeminski et al. (24) in which no difference was found in subjects that performed two passive cycling trials separated by at least one week.

It should also be noted that although we recorded heart rate in this study, it provided little information. While our data are consistent with one previous passive cycling study that found heart rate does not change significantly (24), others have found

that heart rate does increase (4,8,45). These different findings may be due to the fact that heart rate can be influenced by outside factors (e.g. subject anxiety) at low intensities (6).

While it is possible to compare some of our results to previous research, our study was unique in that we also examined how leg mass influences energy expenditure during passive cycling. In agreement with our hypothesis, we found that the energy expenditure during two-leg passive cycling had roughly double the increase in energy expenditure as one-leg passive cycling (in which only half of the muscle mass is involved). This finding held true for all of the 60 and 90 RPM trials.

Along with finding that leg mass plays a role in increasing the rate of energy expenditure during passive cycling, this study design also allowed us to demonstrate that the legs were the primary source of the increase in energy expenditure. Sitting on a bicycle ergometer is likely to activate lower back and abdominal muscles for stabilizing posture (7). However, since the increase in the rate of energy expenditure during one-leg passive cycling was roughly half of the cost of two-leg passive cycling, we can surmise that the energy expenditure is primarily due to the legs and not the postural muscles.

Our study design also allowed us to examine the influence of cycling cadence on energy expenditure. As hypothesized, we found that when the cadence during passive cycling was increased from 60 to 90 RPM, the energy expenditure also increased. This finding makes sense considering that we found leg mass also positively influences energy expenditure during passive cycling. At a faster cadence, the same muscle mass is activated more frequently thus increasing the rate of energy expenditure. As a result, our leg mass and cycling cadence results both show that energy expenditure during passive cycling is determined by the muscle mass involved.

The finding that energy expenditure during passive cycling is influenced by the leg mass involved supports the idea that muscle is active during passive cycling. Using electromyography (EMG), one previous study found no increase in muscle activity during passive cycling (8), but two other studies have found significant increases (3,43). Researchers have proposed that the increase in muscle activity during passive cycling is due to subjects involuntarily pedaling during the passive cycling trials (2,3,24,38,45). It is also possible that the passive cycling movement is eliciting stretch reflexes that activate the muscle. However, more research is needed to understand the exact mechanism responsible for the increased energy expenditure during passive cycling.

The third and final factor that we examined in our study was how adaptation influences the rate of energy expenditure during passive cycling. In agreement with our hypothesis, we found no adaptation, i.e. there was a consistently elevated energy expenditure across multiple trials and during the prolonged 30-minute passive cycling trial. When subjects performed two separate two-leg passive cycling trials at both 60 and 90 RPM, we found no significant difference in energy expenditure. During a prolonged 30-minute passive cycling trial, we also found that the elevated energy expenditure persisted. Based on these findings, it appears that the body does not adapt to the passive cycling movement, which suggests it could be used repeatedly and for extended periods of time to increase energy expenditure.

Since passive cycling produces a consistent and reproducible increase in energy expenditure, we can foresee passive cycling being explored as a tool for decreasing risk for cardiovascular and metabolic diseases such as diabetes and for weight management.

Based on our results, an individual could expend 100 kcal by undertaking approximately

90 minutes of passive cycling at 90 RPM, which could easily be achieved in a sedentary work environment or during normally sedentary lifestyle activities like watching television. Since the increase in energy expenditure during passive cycling is similar to that seen with NEAT and NEAT has been linked with decreased risk factors for chronic diseases, it follows that passive cycling could be used as a tool to modify chronic disease risk factors.

If passive cycling could be used as a tool to improve health, it could allow individuals to exercise while seated. Therefore, unlike the walking workstation, passive cycling would not pose a risk for people with balance issues. Because passive cycling requires no volitional movement, individuals who are otherwise unable to exercise may also be able to conveniently increase energy expenditure relatively easily. For example, people with paraplegia or individuals with peripheral arterial disease (PAD) might benefit from passive cycling that requires no volitional muscle contraction but which increases energy expenditure.

Although we have established that there is potential for passive cycling to be used as a tool to improve health, more research is needed before it can be used in a clinical setting. Our subject population included only healthy, lean subjects riding a modified cycle ergometer that required balancing on a bicycle seat. However, individuals most likely to receive the greatest health benefits from passive cycling include those in the overweight/obese population and those with chronic disease risk factors. Also, while our one- vs. two-leg results suggest that balancing on a bicycle seat did not influence energy expenditure during passive cycling, there is still a possibility that the rate would change if a recumbent style seat were used. In addition, increased levels of NEAT have been

linked with lower blood glucose concentrations (17) and decreased insulin sensitivity according to 2-h plasma glucose levels. (18). Therefore, it is logical that passive cycling, which is energetically similar to NEAT, may improve insulin sensitivity (a major determinant of blood glucose concentration). It is also important to verify our adaptation results using repeated bouts of prolonged passive cycling.

In conclusion, energy expended during passive cycling is positively influenced by leg mass and cycling cadence. These factors could be used to maximize the benefits of passive cycling. The persistent and sustained increase in energy expenditure over multiple trials and an extended 30-minute trial suggests that it is possible that passive cycling could be used as a tool to increase daily levels of NEAT and thus improve health. Future research is needed to determine if there are other potential health benefits of passive cycling.

References

- 1. **Albright A, Franz M, Hornsby G, et al.** Exercise and type 2 diabetes. *Med Sci Sports Exerc* 32(7): 1345-1360, 2000.
- 2. **Bahnson ER, Horvath SM, Comroe JH**. Effects of active and 'passive' limb movements upon respiration and O₂ consumption in man. *J Appl Physiol* 2: 169-173, 1949.
- 3. **Bell HJ, Ramsaroop DM, Duffin J**. The respiratory effects of two modes of passive exercise. *Eur J Appl Physiol* 88(6): 544–552, 2003.
- 4. **Benjamin FB, Peyser L**. Physiological effects of active and passive exercise. *J Appl Physiol* 19: 1212-1214, 1964.
- 5. **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(Pt 2): 673-682, 2003.
- 6. **Brooks GA, Fahey TD, Baldwin KM.** *Exercise Physiology: Human Bioenergetics and Its Applications.* New York: McGraw Hill, 2005, p. 345.
- 7. **Burnett AF, Cornelius MW, Dankaerts W, O'sullivan PB**. Spinal kinematics and trunk muscle activity in cyclists: a comparison between healthy controls and non-specific chronic low back pain subjects-a pilot investigation. *Man Ther* 9(4): 211-219, 2004.
- 8. **De Meersman RE, Zion AS, Weir JP, Lieberman JS, Downey JA**. Mechanoreceptors and autonomic responses to movement in humans. *Clin Auton Res* 8(4): 201–205, 1998.
- 9. **Dixon ME, Stewart PB, Mills FC, Varvis CJ, Bates DV**. Respiratory consequences of passive body movements. *J Appl Physiol* 16: 30-34, 1961.
- 10. **Donnelly JE, Blair SN, Jakicic JM, Manore MM, Rankin JW, Smith BK.** American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc.* 41(2):459-471, 2009.
- 11. **Dunstan DW, Barr EL, Healy GN, et al.** Television viewing time and mortality: the Australian Diabetes, Obesity, and Lifestyle Study (AusDiab). *Circulation* 121(3): 384-391, 2010.
- 12. **Ehrman JK,** ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription. Baltimore, MD: American College of Sports Medicine, 2009.

- 13. **Figoni SF, Rodgers MM, Glaser RM, et al.** Physiologic responses of paraplegics and quadriplegics to passive and active leg cycle ergometry, *J Am Paraplegia Soc.* 13(3): 33–39, 1990.
- 14. **Hamilton MT, Hamilton DG, Zderic TW.** Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. *Diabetes* 56: 2655-2667, 2007.
- 15. Hamilton MT, Healy GN, Dunstan DW, Zderic TW, Owen N. Too little exercise and too much sitting: inactivity physiology and the need for new recommendations on sedentary behavior. *Curr Cardiovasc Risk Rep* 2: 292-298, 2008.
- 16. **Haskell WL, Lee I, Russell RP, et al.** Physical activity and public health: updated recommendations for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc* 39(8): 1423-1434, 2007.
- 17. **Healy GN, Dunstan DW, Salmon J, et al.** Objectively measured light-intensity physical activity is independently associated with 2-h plasma glucose. *Diabetes Care* 30(6): 1384-1390, 2007.
- 18. Healy GN, Dunstan DW, Salmon J, Shaw JE, Zimmet PZ, Owen N. Television viewing time and continuous metabolic risk in physically active adults. *Med Sci Sports Exerc* 40(4): 639-645, 2008.
- 19. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? *Science* 299(5608): 853-855, 2003.
- 20. **Hoffman M, Schrader J, Applegate T, Koceja D.** Unilateral postural control of the functionally dominant and nondominant extremities of healthy subjects. *J Athl Train* 33(4): 319-322, 1998.
- 21. Hotta N, Yamamoto K, Sato K, Katayama K, Fukuoka Y, Ishida K. Ventilatory and circulatory responses at the onset of dominant and non-dominant limb exercise. *Eur J Appl Physiol* 101: 347-358, 2007.
- 22. **Knikou M, Kay E, Rymer WZ.** Modulation of flexion reflex induced by hip angle changes in human spinal cord injury. *Exp Brain Res.* 168: 577-586, 2006.
- 23. **Knikou M**. Hip-phase-dependent flexion reflex modulation and expression of spasms in patients with spinal cord injury. *Exp Neurol*. 204(1): 171-181, 2007.

- 24. Krzeminski K, Kruk B, Nazar K, Ziemba AW, Cybulski G, Niewiadomski W. Cardiovascular, metabolic and plasma catecholine responses to passive and active exercises. *J Physiol Pharmacol* 51: 267-278, 2000.
- 25. **Kruger J, Yore MM, Kohl HW 3rd.** Leisure-time physical activity patterns by weight control status: 1999-2002 NHANES. *Med Sci Sports Exerc* 39(5):788-795, 2007.
- 26. Lanningham-Foster L, Nysse LJ, Levine JA. Labor saved, calories lost: the energetic impact of domestic labor-saving devices. *Obes Res* 11(10):1178-1181, 2003.
- 27. Levine JA, Eberhardt NL, Jensen MD. Role of nonexercise activity thermogenesis in resistance to fat gain in humans. *Science*. 283(5399):212-214, 1999.
- 28. Levine J, Melanson EL, Westerterp KR, Hill JO. Measurement of the components of nonexercise activity thermogenesis. *Am J Physiol, Endocrinol Metab.* 281:670-675, 2001.
- 29. **Levine JA, Miller JM.** The energy expenditure of using a "walk-and-work" desk for office workers with obesity. *Br J Sports Med* 41: 558-561, 2007.
- 30. Levine JA, Lanningham-Foster LM, McCrady SK, et al. Interindividual variation in posture allocation: possible role in human obesity. *Science*, 307(5709):584-586, 2005.
- 31. Levine JA, McCrady SK, Lanningham-Foster LM, Kane PH, Foster RC, Manohar CU. The role of free-living daily walking in human weight gain and obesity. *Diabetes*. 57(3): 548-554, 2008.
- 32. McCrady SK, Levine JA. Sedentariness at work: how much do we really sit? *Obesity*. 17(11):2103-2105, 2009.
- 33. **Miyamura M, Ishida K, Yasuda Y.** Ventilatory response to the onset of passive and active exercise in human subjects, *Jpn J Physiol* 42(4): 607–615, 1992.
- 34. **Miyamura M, Ishida K, Hashimoto I, Yuza N.** Ventilatory response at the onset of voluntary exercise and passive movement in endurance runners, *Eur J Appl Physiol* 76: 221-229, 1997.
- 35. **Morikawa T, Ono Y, Sasaki K, et al.**, Afferent and cardio-dynamic drives in the early phase of exercise hyperpnea in humans, *J Appl Physiol* 67(5): 2006–2013, 1989.

- 36. **Muraki S, Ehara Y, Yamasaki M.** Cardiovascular responses at the onset of passive leg cycling exercise in paraplegics with spinal cord injury, *Eur J Appl Physiol* 81(4): 271–274, 2000.
- 37. **Muraki S, Tsunawake N.** Relationship between pedaling rate and physiological responses during passive leg cycling. *Isokinetics and Exercise Science* 16: 19-24, 2008.
- 38. **Otis AB**. Application of Gray's theory of respiratory control to the hypernea produced by passive movements of the limbs. *J Appl Physiol* 1(11):732-751, 1949.
- 39. Owen N, Healy GN, Matthews CE, Dunstan DW. Too much sitting: the population health science of sedentary behavior. *Exerc Sport Sci Rev.* 38(3): 105-113, 2010.
- 40. **Reger M, Kram R, Byrnes W.** Estimates of cycling efficiency at low power outputs. *Med. Sci. Sports Exerc.* 41:85, 2009.
- 41. Sato Y, Katayama K, Ishida K, Miyamura M. Ventilatory and circulatory responses at the onset of voluntary exercise and passive movement in children, *Eur J Appl Physiol* 83: 516-523, 2000.
- 42. Sato K, Matsuo H, Katayama K, et al. Ventilatory and circulatory responses at the onset of voluntary exercise and passive movement in sprinters, *Eur J Appl Physiol* 92: 196-203, 2004.
- 43. Scott AC, Francis DP, Davies LC, Ponikowski P, Coats AJS, Pieploi MF. Contribution of skletal muscle 'ergoreceptors' in the human leg to respiratory control in chronic heart failure. *J Physiol* 529: 863-870, 2000.
- 44. **Thompson WG, Foster RC, Elde DS, Levine JA**. Feasibility of a walking workstation to increase daily walking. *Br J Sports Med* 42(3): 225-228, 2008.
- 45. **Waisbren SJ, Whiting CS, Nadel ER.** Effects of passive limb movement on pulmonary ventilation. *Yale J Biol Med* 63: 549-556, 1990.

Appendix A – Literature Review

Passive movement involves an external device moving a subject's limbs while they are instructed to remain as relaxed as possible. Since at least 1949 (2,38), ventilation physiologists have been using passive movements as a way to determine how peripheral afferent input contributes to increases in ventilation (4). Early ventilation research found that depending on the type of passive movement used, there can be an increase in the metabolic rate as measured via oxygen consumption (VO₂)(2,38). Ventilation physiologists consider this increase in VO₂ a confounding variable in the study of ventilation and they intentionally choose passive movements that do not produce a VO₂ response (42). Because of this lack of interest, there has been little research regarding the energetics of passive movement. However, what little research exists suggests that the energetics of passive movement deserve closer attention. A simple task that requires no volitional muscle activation but increases energy expenditure may promote the benefits of physical activity in populations that cannot or do not want to become physically active.

Ventilation physiologists have investigated a variety of passive movements. The two most common types of passive movement have been passive knee extension (referred to as passive leg extension in previous studies) and passive cycling. Numerous passive movement studies have found that during both types of passive movement there is an increase in ventilation. However, with respect to energetics, the passive movements differ. In two separate studies, Bell et al. and Otis et al. compared the main types of passive movement and found that there were significant increases in VO₂ only during passive cycling (3,38). These studies found that the increase in ventilation that occurs with passive cycling is matched with an increase in VO₂ representing an increase in

energy expenditure. During passive knee extension, Bell et al. and Otis et al. found there was not an increase in VO_2 , which means that during passive leg extension there is hyperventilation (i.e. a V_E/VO_2 mis-match).

A comparison of other studies that have measured VO_2 supports the idea that passive cycling is the only type of passive movement that causes an increase in VO_2 . A variety of passive cycling studies have found significant increases in VO_2 (3,8,24,37,40,43). A comparison to passive knee extension is difficult because only a minority of passive leg extension studies have measured VO_2 . However, Sato et al. found no increase in VO_2 during passive knee extension (41). Morikawa et al. found no increase in VO_2 for subjects with traumatic spinal cord transection but they did find an increase in VO_2 for healthy subjects (35). Both of these passive knee extension studies focused on the phase I ventilation response (the rapid increase in ventilation that occurs during the intitial 20 seconds of exercise), though, so subjects performed only short duration passive trials (20 seconds – 1 minute). These short trials preclude any information about energy expenditure because a steady state is not reached in 1 minute. Overall, the research indicates that passive cycling results in an increased energy expenditure while passive knee extension does not.

Reasons for Changes in Energy Expenditure During Passive Cycling

Although there are multiple studies showing significant increases in VO₂ during passive cycling, the mechanisms behind the increased VO₂ have yet to be determined. One possible mechanism is that with passive cycling there is an increase in ventilation and this increase in ventilation requires more activation of respiratory muscles (8,37). It

is suggested that passive cycling causes stimulation of mechanoreceptors in the muscles and/or joints, which then causes an increase in ventilation (8). However, there is a major shortcoming with this argument because ventilation also increases with passive knee extension. If increased ventilation alone caused the increases in VO₂ during passive cycling then it should also cause increases during passive knee extension. That however, is not the case.

Another proposed mechanism for the increase in VO₂ during passive cycling is that there is an increased return of pooled venous blood. When the legs are moved, the muscles act as pumps to move the venous blood back to the heart. This pooled venous blood is deficient in O₂ and has an excess of CO₂. Otis et al. suggest that this returning blood stimulates an increase in ventilation and also increases VO₂ (38). However, this argument does not explain why there is a difference in VO₂ between passive cycling and passive knee extension. During passive knee extension, the muscles presumably also act to increase venous return. As a result, there should be an increase in VO₂ during passive knee extension but there is not. Another problem is that this venous blood argument would only apply for short passive trials in which a steady state was not reached. Once steady state is reached it can be assumed that the venous blood in the legs has returned to the heart, which means VO₂ should return to resting values. However, during steady state passive cycling, VO₂ remains elevated (3,8,24,40,43). Thus, a return of venous blood does not provide a satisfactory explanation as to why passive cycling results in an increased VO₂.

Leg muscle activity measurements (electromyography, EMG) have greatly influenced the way people think about the increase in VO₂ during passive cycling.

Although only a few studies have measured muscle activity, it appears that during passive cycling there is significant muscle activity but during passive knee extension there is not. For the passive cycling studies that have measured EMG (3,8,24,40) both De Meersman et al. and Krzeminski et al. found no significant increases in muscle activity (8,24). However, De Meersman et al. only measured the quadriceps muscle, which makes it hard to generalize to the entire leg. Also, Krezminski et al. found increases in EMG activity but these increases did not reach statistical significance and may be due to statistical power issues. Two other passive cycling studies have found increases in muscle activity (3,39). But, of these two, Bell et al. measured only the vastus lateralis (3). For passive knee extension, the results are more straightforward. Two passive knee extension studies found no increase in EMG activity (33,35). Thus these few studies suggest that passive cycling increases muscle activity but passive knee extension does not, which corresponds with the VO₂ results.

In agreement with the research that has found increased muscle activity levels during passive cycling, the majority of researchers have proposed that passive cycling is not entirely passive (2,3,24,38,45). In other words, many researchers have suggested that during passive cycling, subjects are unintentionally and/or actively assisting the movement. This would explain why there is an increase in VO₂ during passive cycling and not during passive leg extension. However, the increased muscle activity levels may not be from subjects assisting the movement but may instead be from stretch/flexion reflexes. When Knikou et al. stimulated sensory receptors of subjects with spinal cord injury, they found that there was a flexion response similar to what is required for locomotion (22). That study, along with a follow up study by Knikou et al. (23), suggest

that hip joint proprioceptors play a major role in this flexion response. It was found that a flexed hip diminishes the flexion response. These studies may help explain the reasons for an increased VO₂ seen with passive cycling. During passive cycling, the sensory receptors involved with the stretch reflex along with the hip proprioceptors may be eliciting a flexion response. Thus, EMG recordings may be detecting the muscle activation that is due to this flexion response and not voluntary muscle activation. This increased muscle activation would also explain the increased VO₂ that occurs during passive cycling.

Potential Factors Influencing Energy Expenditure During Passive Cycling

Little is known about the factors that influence VO₂ during steady state passive cycling since the majority of research has investigated only factors that influence ventilation during passive knee extension. Because the VO₂ response is not similar between passive cycling and passive knee extension, it is not possible to use previous research to determine if factors that influence ventilation during passive knee extension also influence VO₂ during passive cycling. However, both passive cycling and passive knee extension result in an increase in ventilation. The main difference is that during passive cycling, this increase in ventilation is matched by an increase in VO₂. Therefore, one can assume that if a factor influences the ventilation during passive knee extension, that same factor may also influence ventilation and thus VO₂ during passive cycling. With this assumption in mind, what follows is a discussion of four potential factors that may influence VO₂ during passive cycling.

Training Status

Ventilation physiologists have investigated how the training status of subjects influences the ventilatory response during passive knee extension. In the first study by a group in Japan, endurance trained subjects were compared to untrained subjects (34). Miyamura et al. found that the endurance trained subjects had an increase in ventilation but that the response was attenuated compared to controls. The next study compared track and field sprinters to untrained subjects (42). Again it was found that the trained sprinters had an attenuated response. The last study in this group compared dominant to non-dominant limbs (21). The idea behind this was that throughout a day, a person chooses their dominant limb over the non-dominant limb resulting in light training all day for that limb. The study found that the dominant limb also had a reduced ventilation response to passive knee extension. All three of these studies suggest that training has an effect on the ventilation response seen with passive knee extension, however, they were unable to reach a satisfactory conclusion as to why this happens.

It is important to note that these training status studies may not reflect what would occur during passive cycling. The above studies used only short duration trials in which a steady state was not reached and also did not measure VO₂. Another issue with these studies is that only passive knee extension was used. As discussed above, passive knee extension does not result in the same VO₂ response as passive cycling. Because of these issues, it is not possible to determine if training status affects energy expenditure during passive cycling. However, these training status studies suggest that trained individuals have a different ventilation response during passive knee extension and this response may also indicate an attenuated VO₂ response during passive cycling.

Muscle Mass

It would seem that if muscle were being activated during passive cycling, as most studies involving EMG have suggested, then the muscle mass of that subject would determine their energy expenditure. This might then provide a reason why a study by Figoni et al. using paraplegics and quadriplegics (13) did not find significant increases in VO₂ during passive cycling. Paraplegics and quadriplegics are expected to have less muscle mass in their legs due to atrophy. If muscle mass is a factor that determines the changes in energy expenditure, it follows that the energy expenditure would not have increased very much in these subjects. As a result, it would be hard to find significant changes in energy expenditure compared to rest. Figoni et al., however, did not measure muscle mass values for the subjects so it is not possible to determine if this idea is valid. We are aware of no studies that have compared muscle mass values to the changes in VO₂. As a result, more research needs to be done in this area.

While no research has investigated the role of muscle mass during passive cycling, the ventilation research has examined how it affects passive knee extension. A study by Miyamura et al. compared the ventilation response for one-leg and two-leg passive knee extension as a way to investigate the role of muscle mass (33). The reasoning was that one-leg passive knee extension involves half the muscle mass of two-leg knee extension so the ventilation response should increase additively. What they found though, was that the ventilation response was not additive. The response to one-leg passive movement was higher than expected and was more than half of the two-leg response. One must be careful, though, when attempting to say that this study by

Miyamura et al. disproves the role muscle mass may play in increasing VO_2 during passive cycling because VO_2 was not measured. The study also used passive knee extension and trials that were only 30 seconds long. As a result, Miyamura et al. only provide a glimpse into what may happen to energy expenditure during one-leg or two-leg passive cycling.

Cycling Cadence

Throughout the literature, passive cycling studies have used a variety of pedal cadences. This variation presents an opportunity for comparison across multiple studies. When the VO_2 is plotted with the cycling cadence used in the study, there is a clear linear relationship between VO_2 and cadence. Previous studies can also be compared by dividing the VO_2 by the cadence that was used. When this is done across multiple passive cycling studies (3,4,9,24,38,43,45), it is seen that there is a roughly constant VO_2 cost per pedal rotation (Table 6). This constant VO_2 cost is approximately 6.6 mL O_2 /pedal rotation.

If there is a cost per pedal rotation during passive cycling, one would expect to see a linear increase in a subject's VO₂ as cadence is increased. One study looking at various pedal cadences found just that (37). Muraki et al. increased the pedal cadence by 5 rpm every minute starting at 20 rpm and ending at 70 rpm. They found that there was a linear increase in VO₂ as the passive cycling cadence increased. However, Muraki et al. did not publish their VO₂ values for each cadence so it cannot be determined if VO₂ increased by the general cost of 6.6 mL O₂/pedal rotation as described above. Also, there were methodological issues in this study, most importantly a steady state was not reached

Table 6: Cadence and VO_2 data from previous studies on passive cycling. The average VO_2 /pedal rotation is 6.6 ± 1.0 mL O_2 /rot.

0.0	VIII (1)		
Study	Cadence Used in Study	VO ₂ During Passive Cycling (L)	VO ₂ /pedal rotation (mL O ₂ /rot)
Bell et al.	65	0.43	6.6
Benjamin et al.	60	0.50	8.3
Dixon et al.	60	0.39	6.5
Krzeminski et al.	50	0.32	6.4
Waisbren et al.	60	0.31	5.2
Reger et al.	90	0.57	6.3

for each pedal cadence. One might speculate that if a steady state was reached, the linear trend would still remain. Overall, studies of passive cycling suggest that cadence is another aspect that can be manipulated to maximize VO₂ differences between trials although no study has directly measured cadence affects during steady state passive cycling.

Adaptation

While passive cycling for an extended period of time, the body may adapt to the passive movement of the limbs and VO₂ may begin to return to resting values. Thus, adaptation is another aspect that may influence changes in metabolism seen during passive cycling. To date only Krzeminski et al. have investigated adaptation during passive cycling (24). In the Krzeminski et al. study, subjects performed a 5 minute passive cycling trial and then one to two weeks later, subjects returned to the lab and performed the trial again. The researchers found that in both passive cycling trials the VO₂ was elevated above resting values with no significant difference between trials. These findings would suggest that there is no adaptation that occurs during passive cycling. However, if passive cycling were to be used in a clinical setting, it would be used for longer than five minutes. Therefore it is important to know what happens during a single extended passive cycling trial.

Other studies have indirectly examined adaptation to passive cycling. In some studies (3,24,36), subjects were allowed to try passive cycling before the actual testing protocol. This "practice" trial was used so that subjects could learn to be as passive as possible and served as a time to adapt to passive cycling. Even after subjects have been

given time to adapt to passive cycling, the researchers in these studies still found that there was an increase in VO₂ during passive cycling. Other studies have indirectly examined adaptation to passive cycling by using 30 minute trials (4) or by using multiple trials (45). However, neither of these studies present the data in a way that allows for interpretation as to whether there is adaptation taking place or not. As a result, the previous research seems to suggest that there is no adaptation taking place during passive cycling, however, more well designed research is still needed to determine if this is truly the case.

Energy Cost Comparisons

When discussing the energetics of passive cycling, it is important to have an understanding of how the energy costs compare to non-passive movements. One way this can be done is by comparing passive cycling to no-load cycling performed actively by the subject. In the passive cycling literature there are a variety of findings. Three studies have found that the increases in VO₂ during passive cycling are similar to those during active cycling against no load (2,8,43). But Reger et al. recently reported that the VO₂ during passive cycling is significantly less than during active cycling (40). There is no clear reason for the different findings between these passive cycling studies.

Another and perhaps better way to consider the magnitude of the metabolic rate during passive cycling is to compare passive cycling to non-cycling related activities.

This can be done by comparing passive cycling to a component of daily energy expenditure. Daily energy expenditure is made up of three main components: basal metabolic rate, thermic effect of food, and physical activity. Physical activity can be

further divided into two groups. The first group is physical activity that is planned such as running or hiking. The second group is nonexercise activity thermogenesis (NEAT), which includes any physical activity not included in the first group (27). Studying the energetics of NEAT is difficult, though, because it must be somehow separated from purposeful exercise. NEAT also involves activities like fidgeting which are hard to measure (28). Isolating one aspect of NEAT provides a good way to estimate its overall energetic cost. Walking makes up the majority of NEAT (31) and the energy expenditure during walking is relatively easy to measure. Walking at 1 MPH has been shown to double resting energy expenditure (28). This magnitude is very similar to what has been found during passive cycling at 90 rpm (40). Therefore it seems that passive cycling could be energetically similar to NEAT in magnitude.

Applications

The similar magnitudes of passive cycling and NEAT emphasize why the VO₂ changes during passive cycling deserve more attention. Obesity has become a major problem in developed countries with 66% of the U.S. population over weight and/or obese (10,31). For the majority of the population, this weight gain has been attributed to an imbalance of only 100 kcals/day (19). One of the suggestions for these excess kcals has been to increase NEAT (10,27,30). The reason for this is that individuals only need to make small changes to create an energy imbalance. Thus, even low intensity activities like NEAT could prove beneficial for helping with weight management. The finding that passive cycling is comparable in magnitude to NEAT highlights the possibility that passive cycling may be useful for weight management.

Passive cycling may also be beneficial for overall health. Recent research has found that individuals who spend the majority of their time inactive but still meet the ACSM guidelines for physical activity have increased metabolic risk variables (18). Increased light activity time (i.e. increased NEAT) and thus decreased sedentary time have been linked with healthier blood glucose (17) healthier lipoprotein lipase activity (5), and decreased cardiovascular disease related mortality (11). Thus, passive cycling, which requires no volitional movement but still increases energy expenditure, may be another way to increase activity levels and thus enhance overall health.

It is possible that passive cycling could be incorporated into the work place. Many individuals spend the majority of the day sitting at a desk in an office. Making these individuals more active could promote weight loss/control (32) and improve overall health. Levine et al. have suggested a "walking workstation" as a way to increase energy expenditure at work (44). This technique involves a person standing and walking at 1 MPH on a treadmill while performing his or her normal work routine. Passive cycling has been found to increase energy expenditure in a similar manner as that found when using the "walking workstation" (40,44). Thus, passive cycling may provide many of these same benefits but allow individuals to remain seated and not put forth any effort. Passive cycling could then turn a relatively sedentary office environment into a more active environment that improves worker's well being. Because passive cycling requires no intentional movement, individuals that are unable to exercise may also be able to increase energy expenditure relatively easy. For example, paraplegics and individuals with peripheral arterial disease (PAD) could benefit from a task that requires no intentional muscle contraction but which increases energy cost.

Summary

The potential benefits of passive cycling have largely gone unnoticed. This is because the focus of passive movement research has been on ventilation and not energy expenditure. A review of passive cycling and passive knee extension studies suggests that only passive cycling may provide a significant increase in energy expenditure required for improving overall health. Although there has been limited research on this topic, previous research suggests that pedaling cadence, training status, muscle mass, and adaptation may influence the energy expenditure during passive cycling. However, no research to date has directly investigated how these factors actually influence energy expenditure during steady state passive cycling.

The fact that passive cycling can elevate metabolic rate with no intentional muscle contractions highlights its potential to be used as a tool to prevent obesity, cardiovascular disease, and metabolic disorders such as diabetes. To optimize the use of passive cycling in a clinical setting, more needs to be known about the factors that influence the increase in metabolism seen with passive cycling.

Appendix B – The Influence of Muscle Mass Between Subjects

Our study examined the influence of leg lean mass on energy expenditure during passive cycling by comparing within subjects. However, since our study protocol involved the use of a DXA scan, we were also able to examine the influence of leg lean mass between subjects. This between subjects comparison was accomplished by calculating the correlation between each subject's leg lean mass (via DXA) and energy expenditure during passive cycling. In contrast with our within subjects (one- vs. two-leg) comparison, our between subjects comparison did not indicate that individual leg lean mass influences energy expenditure during passive cycling. The magnitude of the increase in energy expenditure did not correlate with the between subject variation in lean mass at 60 RPM (R²=0.10, p=0.34) or at 90 RPM (R²=0.22, p=0.15).

Our between subjects findings may be explained by the fact that at a fixed absolute power output, subjects riding on a cycle ergometer have VO_2 (L/min) values that vary by $\pm 5\%$ independent of their lean body mass (12). Therefore, the fact that an individual's VO_2 during cycling at a set absolute power output is not determined by their total lean body mass suggests that between subjects we should not expect a significant correlation between leg lean mass and energy expenditure during passive cycling.