Adria D. Finch. Neural mechanisms contribute to the age-related increase in metabolic cost of gait. (Under the direction of Tibor Hortobagyi) Department of Exercise and Sport Science, April 2010.

Aging is associated with biomechanical and physiological changes in several organ systems, including neural changes of voluntary movement. One manifestation of age-related changes in neural control of gait is the increased activation of muscles that are antagonist to the prime movers during the stance phase of gait. Another age-related adaptation is the increased metabolic cost of locomotion. Several studies have attempted to link gait mechanics to the increased cost of transport, but none of the mechanical gait variables accounted for the age-related increase in oxygen uptake. Here we hypothesized that the related increase in metabolic cost during gait is mediated by increased antagonist muscle coactivation. EMG and oxygen consumption data were collected during treadmill walking to determine the levels of antagonist muscle coactivation and metabolic cost. The data revealed that old subjects experienced significantly greater levels of both coactivation and metabolic cost. Old subjects had 4-17% greater levels of metabolic cost of gait than young subjects, and 53-61% greater levels of total antagonist muscle coactivation than young subjects. Regression analyses showed that there was a strong association between the level of antagonist muscle coactivation and metabolic cost of gait, suggesting that neural factors contribute to the age-related metabolic adaptations in gait.

NEURAL MECHANISMS CONTRIBUTE TO THE AGE RELATED INCREASE IN METABOLIC COST OF GAIT

A Thesis

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NEURAL MECHANISMS CONTRIBUTE TO THE AGE RELATED INCREASE IN METABOLIC COST OF GAIT

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CHAPTER I: INTRODUCTION

Population distribution in the United States has been drastically changing. Every year the United States has a greater percentage of old individuals within its demographics. In the year 1993 the average life expectancy was 76. By 2050 the average life expectancy is estimated to be somewhere between 82 and 87 years old (US Census Bureau). As the United States population increases in age it is necessary to promote research to address concerns that coincide with aging. This type of research will help physicians and other professionals to provide proper care that older populations require.

Research has shown that as people age, their bodies naturally go through changes involving nearly every anatomical system. One of these physiological changes involves changes in neural control of voluntary movement, specifically increases in antagonist muscle coactivation. This increased muscle coactivation, which is the concurrent activation of pairs of muscles on the opposite side of a joint is caused by both cortical and spinal changes within the nervous system. There are several functional roles of muscle coactivation including increasing joint stiffness, increasing joint stability (Hortobagyi and DeVita 2000), assisting with learning new skills (Person and Roshchina 1958), and decreasing reaction time (Park 2002).

Another change that old adults experience with locomotion is an increase in energy consumption. Old individuals use more oxygen and work at a higher percentage of their maximal oxygen uptake (VO_2max) during gait as compared to younger individuals (Malatesta, D., D.Simar et al. 2004). This occurs when older individuals walk at both absolute and self-selected gait velocities. This increase in metabolic energy could be caused by many factors including changes in mitochondrial function, and muscle sarcopenia, and decreased cardiac output.

It is possible that one of the other physiological changes associated with the increase in energy consumption old people experience is increased antagonist muscle coactivation. When people perform voluntary movements with high levels of muscle coactivation they are activating a larger muscle mass than needed to perform the task, which will in turn increase oxygen and energy demands. Additionally since the antagonist muscles are producing more force, the agonist muscles need to contract more forcefully in order to overcome that force. This should increase the oxygen and energy demands of a person, because greater levels of ATP need to be produced in both agonist and antagonist muscles.

Hypothesis

Several hypotheses were investigated within this study. The global hypothesis was that the age related increase in oxygen consumption was caused by increased antagonist muscle coactivation during normal gait. The first sub-hypothesis was that an age effect would be present in which old adults would experience greater levels of both metabolic cost of gait and antagonist muscle coactivation. The second sub-hypothesis was that as gait velocity increased antagonist muscle coactivation also increased. Finally, the third sub-hypothesis was that there would be a task main effect; decline walking would cause greater levels of coactivation than incline walking.

Purpose

The purpose of this study was to compare the cost of locomotion and EMG muscle activity in antagonist muscles in old and young adults during gait, and to determine if there was a relationship between the metabolic cost of motion and the amount of antagonist muscle coactivation during this motion.

Delimitations

1) All subjects were healthy and free of pain during normal gait. They had no history of lower extremity injury. Additionally they were free of neurological disorders, cardiovascular disease, and musculoskeletal problems.
2) Subjects were men and women, and had a Body Mass Index of <30 kg/m^2
3) Subjects had normal function in activities of daily living.
4) The study looked at EMG activity in one leg during 1 resting condition and 5 walking conditions for six minutes.
5) Young subjects were 18 – 25, and old subjects were 70 – 85.

Limitations

1) The Cosmed mask may not have always completely and perfectly sealed around a subject's face.
2) The EMG signals could have been affected by biological tissue content.
3) Some subjects were not completely comfortable walking on the treadmill, and may have altered their normal gait.
4) The subjects may have felt restricted or weighed down by the Cosmed equipment.
5) The study looked at self selected, slow, fast, incline, and decline walking conditions.

Operational Definitions

Young adult: males and females 18-25 years of age

Old adults: males and females 70-85 years of age

Muscle coactivation: the average antagonist muscle activity level when the agonist is contracting. This value was normalized to the subjects' maximal voluntary contraction.

CHAPTER II: REVIEW OF THE LITERATURE

Cost of Locomotion in Old Adults

Cost of locomotion is defined as how much energy is necessary to move from one place to another. It is dependent on many things including locomotion speed, fitness level, and age. Old individuals use more energy to walk a given distance than young individuals (McCann and Adams 2002; Malatesta, Simar et al. 2003).

One possible reason for increased metabolic cost of gait in old adults may be due to decreased mitochondrial function. The mitochondria are constantly generating reactive oxygen species that eventually damage the mitochondrial DNA (mtDNA) (Harman 2003). These changes in the mtDNA eventually cause dysfunction in the electron transport system which reduces the ATP production of the mitochondria (Dirks, Hofer et al. 2006). Mitochondrial dysfunction may cause cell death which in turn mediates neurodegeneration and sarcopenia (Marzetti and Leeuwenburgh 2006). Mitochondrial dysfunction and cell death means that old individuals yield fewer ATP from the electron transport system. This inefficiency causes old adults to use more oxygen and have greater metabolic cost than young adults.

However, even though mitochondrial dysfunction occurs it isn't the main contributor to increased cost of locomotion in older individuals. As people age the mitochondrial dysfunction they experience mainly occurs in type II muscle fibers, while mitochondria in old people's type I muscle fibers actually become more efficient (Deschenes 2004). Therefore, mitochondrial dysfunction is not the sole cause for age related increases in energy consumption. The exact cause of the increased cost of locomotion that old individuals experience is still debatable. However, the fact that old adults experience increased antagonist muscle coactivation, thereby

activating and using more muscles than young individuals, may in fact play a role in increased metabolic cost of gait.

Mechanical Parameters of the Cost of Locomotion

Some researchers have previously speculated that an increase in metabolic cost of locomotion was due to changes in the mechanics of gait that older individuals experienced. Cross sectional data has indicated that when energy consumption during locomotion increases biomechanical changes in gait also occur Waters, Lunsford et al. 1988). Old adults may alter their patterns of locomotion thereby changing the normal kinetic and potential energies that they utilize, or they may have to use more energy to stabilize their body during locomotion.

Old adults actively change and control their step width more than young adults (Donelan, Shipman et al. 2004). This allows them to alter the level of stability used during gait. Old adults stabilize their joints more during walking, by increasing their step width. They tend to take wider steps and adjust their step width more frequently than young individuals (Owings and Grabiner 2004). Altering step width ultimately requires people to use more mechanical energy and also forces them to consume more oxygen (Donelan, Shipman et al. 2004).

In addition to altering their step width and stability, old adults also change their walking mechanics which may lead to changes in oxygen consumption. Old adults perform more positive work on their center of mass than young individuals during most gait phases (Hernandez, Silder et al. 2009). It has also been shown that as people age they have a shift in joint forces. Old individuals have higher joint torques and powers in their hips and knees than young individuals (DeVita and Hortobagyi 2000).

The mechanical changes that old adults experience may be taxing in terms of energy consumption, however it is unlikely. Mian et al. determined that healthy old individuals do not

experience a significant increase mechanical work during gait. Therefore mechanical changes do not account for the age related increase in metabolic cost of gait. Old adults do however experience a greater amount of antagonist muscle coactivation, which may be the cause of the increased metabolic (Mian, Thom et al. 2006).

Purpose of Muscle Coactivation

Muscles directly involved in producing a desired movement are known as agonist muscles. Those agonists of greatest importance are the prime or principle movers while less important agonist muscles are known as assistant movers or synergists.

Antagonist muscles have actions opposite of the agonist and are generally located on the opposite side of a joint. In order to be metabolically efficient it is desirable to have the antagonist relaxed during the agonist action thereby reducing the amount of active muscle mass (Truijens, Noordermeer et al., 2005). If the antagonist is relaxed, the agonist muscle can exert a full amount of joint torque without having to overcome the force produced by the antagonist. When both the agonist and antagonist muscles are simultaneously contracting, coactivation occurs.

Although muscular coactivation can be metabolically costly it does in fact serve several purposes. In 2000 Hortobagyi and DeVita determined that during downward stepping individuals began coactivating antagonist muscles before ground contact allowing them to prepare for impact. This preparation was viewed as a protective stabilizing mechanism. Antagonist coactivation increases joint stability by increasing the movement control in a joint. Muscular contractions on both sides of a joint make the joint more rigid by creating tension around it. This increases joint stability, especially in motions that require eccentric muscle contractions (Hortobagyi and DeVita 2000). Increased joint stability through coactivation

decreases the risk of injury and can also increase the longevity of a joint. Previous research has also shown that during faster joint movements antagonist muscle activation increases in order to provide better joint stability for individuals (Zijlstra 2004). This study looked at how both the type of muscle contraction during gait, and the velocity during gait affected antagonist muscle coactivation

Coactivation also provides increased coordination. Person et al. found that when learning a new task individuals increased coactivation for better control over the task movement, and then reduced coactivation when they were comfortable with the task. Therefore, when people need to have careful, coordinated movements they are more likely to increase their muscle coactivation (Person and Roshchina 1958).

Coactivation of the antagonist is also used to moderate the force output of the agonist. This is often seen in movements where accuracy is focused on rather than speed. As a result of training or practice, such coactivation is usually reduced (Ives 2006).

One way to decrease response time and increase quickness is to use muscular coactivation. When antagonist muscles are pre-activated or already firing, less time is required to fully activate them. Therefore, it is easier to fully activate the antagonist muscles in order to change direction or to change the type of movement being performed.

Antagonist muscle coactivation exists for many reasons. However when it occurs it could be increasing metabolic cost. Therefore if coactivation is occurring at a time when it is not providing a specific function it is essentially wasting energy for the body. People are more energy efficient when there are minimal levels of muscle coactivation.

Muscle Coactivation in Aging

As people age their bodies go through many changes, including transformations of their nervous systems, and musculoskeletal systems. These modifications force older adults to adopt new ways of performing everyday tasks. One way that old individuals will change is by increasing their antagonist muscle coactivation in both single (Patten and Kamen 2000) and multi-joint tasks (Mian, Thom et al. 2006).

Reciprocal inhibition is a common neuromuscular phenomenon in which an antagonist muscle relaxes while an agonist muscle contracts. It is mediated by the Ia inhibitory interneuron which sends signals to the antagonist muscle telling it to relax. Old adults experience decreased presynaptic inhibition of their Ia inhibitory interneuron (Earles, Vardaxis et al. 2001). This means that a stronger signal from the agonist muscle is required to make the antagonist muscle relax in old individuals compared to young individuals. If a large signal from the agonist muscle does not occur, the antagonist muscle won't be inhibited and will contract, causing muscle coactivation to occur.

Nielsen proposed that another reason for increased muscle coactivation in old individuals was due to afferent and supraspinal pathways converging on the same interneuron. He speculated that interneurons were receiving input from multiple locations which caused them to have a higher resting threshold. Since these interneurons were already slightly excited they required a smaller action potential in order to reach threshold. This means that the interneurons fired more frequently in old individuals than in young individuals. Since interneurons were more likely to reach threshold, agonist and antagonist muscles were more likely to contract at the same time (Nielsen 2004).

Cortical changes in old individuals also seem to alter antagonist muscle coactivation levels. It has been proposed that in old individuals there is a reduced suppression of areas of the brain that control antagonist muscle movement. In other words, areas of the brain are more excited, thereby more likely to cause muscle contractions.

Old adults also experience greater overall cortical stimulation during movement (Hutchinson, Kobayashi et al. 2002). This most likely occurs because the cortical and subcortical structures are reorganized due to age related neurodegradation. This degradation forces old individuals to actively recruit greater areas of their brain. When the brain experiences greater overall activity it is more likely to cause contraction of both agonist and antagonist muscles at the same time.

If older adults are in fact experiencing greater antagonist muscle coactivation they in turn need to supply energy to those antagonist muscles being used. This increase in antagonist muscle activity may be the reason older adults use more energy for everyday tasks.

Summary

It has been proven that old adults coactivate their muscles more than young adults (Hortobagyi and DeVita 2000). This is caused by both spinal and supraspinal mechanisms. Old adults also consume more metabolic energy than young individuals (Mian, Thom et al. 2006). It is important to determine if the age related increase in energy consumption is due to increased antagonist muscle coactivation. This will help researchers and physicians to understand more aspects of aging, which is becoming more and more important as the general population increases in age.

CHAPTER III: METHODOLOGY

Subject Characteristics

Two groups of 12 subjects were recruited. The first group consisted young adults (males: 6, females: 6, age: 20.9± 0.7, BMI: 22.3± 0.4). The second group of subjects was composed of old adults (males: 5, females: 7, age: 77.3± 1.4, BMI: 24.3± 1.0). Table 1 in the Appendix shows the complete subject characteristics.

Inclusion Criteria

1) The subjects fell into one of the desired age categories: young 18-25 years, old 70-90 years.
2) Healthy and mobile during gait and other activities.
3) Free of pain and had no difficulty performing activities of daily living.
4) A BMI less than or equal to 30.0.
5) Were able perform all experimental procedures safely and without difficulty.
6) Provided written informed consent

Exclusion Criteria

1) A history of a serious lower extremity injury
2) Orthopedic problems including osteoporosis, joint replacements, lower extremity or back surgery
3) A history of cardiovascular disease including uncontrolled high blood pressure, a history of heart attacks, coronary artery disease, peripheral artery disease, or a pacemaker placed in their heart
4) Neurological abnormalities including stroke, dementia, Parkinson's disease, etc.
5) Unable to pass the SPPB

6) Could not perform the experimental tasks

7) Smoked cigarettes within the past 6 months

Study Design

Between group comparisons were made to look at energy consumption data, and antagonist EMG data. These determined if any age main effects were present. Specifically, they determined if old adults had higher metabolic energy consumption, and also if old adults had greater antagonist muscle coactivation. Additionally comparisons were made between velocities to determine if a velocity main effect was present for antagonist muscle coactivation levels, and comparisons were made between different tasks to see if there was a condition main effect.

The subjects performed 6 experimental conditions in which their VO2 and muscle activity was measured. There was a resting, a self-selected walking speed, a fast walking speed, a slow walking speed, an incline, and a decline condition. The data collected during these trials was used to determine the subject's energy consumption and antagonist muscle coactivation.

Equipment

Walking was performed on a Simple 9600 Platform Vision (Vision Fitness) treadmill. EMG data was collected by a Bortec EMG system (Bortec Biomedical Ltd.) using 4 channels and a ground. A Cosmed K4b² (Cosmed) VO2 analyzer was worn to collect VO_2 data, and Cosmed software was used to analyze the VO_2 data. Data were collected with Qualisys Track Manager Software (Qualisys, Inc) and analyzed by MatLab (Mathworks).

Experimental Protocol

Subjects were recruited on campus, by word of mouth, and by newspaper advertisement. They participated in a phone interview and went through a movement screening process to determine inclusion in the study. Subjects wore shorts, sneakers, and a heart rate monitor. Before testing began subjects were given an informed consent form explaining the experimental procedures, any risks, and that they could stop the test at any time. The informed consent form was approved by East Carolina University's IRB.

After signing the informed consent form the subjects completed the short physical performance battery protocol and score test (SPPB). This test determined whether or not a person is classified as frail. If they were frail they were excluded from the study. Their height and weight was taken with their shoes on.

The subject's skin was then be prepped for EMG electrodes. All electrodes were placed on the subjects' right leg. The skin was shaved and cleansed with an abrasive lemon scrub and an alcohol pad at five different electrode sites, four muscle sites and one ground site, in order to guarantee good EMG signal conduction. The electrodes were placed on the distal portion of the vastus lateralis (VL), the distal portion of the biceps femoris (BF), the center of the tibialis anterior (TA) muscle belly, and the center of the lateral gastrocnemeus (LG) muscle belly. The ground electrode was located on the fibular head.

Finally the subject wore the Cosmed K4b^2. The correct size mask was determined based upon a blow test. During the blow test the subject had the mask strapped to their face. The experimenter covered up the air hole with their hand and asked the subject to exhale as hard as they could. If air escaped the mask either a different size mask was needed, or adjustments to the fit of the current mask were made. They wore the Cosmed analyzer pack on their back.

After the subject was outfitted for the experiment they performed manually resisted maximal voluntary isometric contractions (MVCs) of each of the four muscles tagged with electrodes. To test the VL subjects sat in a chair with their right knee bent at approximately 60 degrees. The tester held the subject's leg in that position while they pushed against them trying to extend their leg. When testing the BF the tester also held the subject's leg at approximately 60 degrees; the subject tried to bend their knee while the tester resisted the motion. When testing the TA the subject had their knee bent at 90 degrees. They then lifted their toes off the ground while keeping their heel in contact with the floor. The tester tried to push the subject's toes back down to the ground and the subject was forced to resist the motion. The LG was tested by having the tester hold the subject's leg extended out in front of them in the air. There was a slight bend in the subject's knee. The subject tried to plantar flex their foot while the tester pushed against their effort.

The subject was acquainted with the treadmill. They learned how to properly start and stop walking on the treadmill by using its side rails. If the subject was nervous or felt uncomfortable walking on the treadmill they were allowed time to become familiar with walking on it.

There were six conditions each lasting six minutes. The first condition was always the resting condition. The subject stood calmly for the duration of the trial, as EMG and VO_2 data were collected. The second test condition was always walking at a self-selected speed. During this condition the subject walked on a treadmill as it increased in speed. When the subject felt as though they had reached their normal, everyday walking speed they notified the experimenter to leave the treadmill at that speed for the duration of the trial. The remaining four experimental conditions were randomized for each subject. They were level walking at 0.98 m/sec, level

walking at 1.2 m/sec, 6% incline at .98 m/sec, and 6% decline at .98 m/sec. Five minutes into each testing condition the subjects was asked to rate how hard they felt that they were working on a rating of perceived exertion (RPE) chart. They will pick a number between 6 and 20. The subjects will rest 3 minutes between each trial.

Data Analysis

All EMG data will be collected at 960 Hz. The data will first be exported to as a .c3d file to Visual3D (C-Motion), where it was exported as a .mat file to MatLab. There, a program was run to determine the amount of antagonist muscle coactivation for each muscle while its agonist was active. The MatLab program that was used to analyze the data was written in conjunction with Stanislaw Solnik.

In order to determine the amount of antagonist muscle coactivation the program first filtered the data with a butterworth bandpass filter (cut-offs of 20 and 350 Hz), and operated Teager-Kaiser Operator (TKEO) which enhanced the EMG signal by increasing the signal to noise ratio (Solnik, DeVita et al. 2008). This provided a clearer EMG signal which showed the drastic differences between EMG baseline and bursts. The program allowed the baseline to be selected, and then determined exactly when the onset and offset of the agonist muscle activity occured. A muscle burst occurred when EMG activity was greater than 10 standard deviations of the selected baseline, and lasted more than 20 msec. All activity when the agonist muscle was inactive was ignored, and EMG envelopes were placed around the agonist muscle bursts. The intensity of the agonist and antagonist muscle contractions within the EMG envelopes were normalized to the subjects' MVCs. The program then determined the average raw, and average normalized EMG amplitude of both the agonist and antagonist muscles while the agonist muscles were active. This was done for each muscle when it was acting as the agonist. Total

coactivation was expressed as the sum of VL, BF, TA, and LG normalized antagonist coactivation values.

During the test the Cosmed performed a breath by breath analysis of oxygen cost and carbon dioxide expenditure. The data analysis of the metabolic data involved first averaging all the metabolic variables into 15 second intervals. Then the data was averaged by every minute, in order to guarantee that subjects reached steady state. The subjects achieved steady state exercise during minute four. Therefore, the metabolic data in minutes 5 and 6 were averaged together to determine the final values for all the metabolic variables.

The oxygen consumption data was used to establish the metabolic cost by determining the gross metabolic power using the equation

((16.6 * VO2 Task * 1000 / 60) + 4.51 * (VCO2 Task * 1000 / 60)) / Mass (Brockway 1987)

Gross metabolic cost per meter using the equation

(Gross Met Power Task - Gross Met Power Standing) / Velocity.

These were determined for each minute within the 6 minute trial, however when only the steady state values were used in reporting metabolic variables. Once steady state was reached (minute 4), each metabolic variable was averaged across the final 2 minutes of the trial (ACSM Handbook 2008).

Statistical Analysis

The first statistical analyses were of antagonist muscle coactivation and metabolic data during level walking at various velocities. A group (young, old) by velocity (self-selected, .98 m/sec, and 1.2 m/sec) analysis of variance with repeated measures for muscle coactivation and variables for metabolic cost of gait was performed. This determined if there was an age main effect, velocity main effect, or age by velocity interaction effect.

The second statistical analyses were of antagonist muscle coactivation and metabolic data during task dependent walking. A group (young, old) by task (incline, decline) analysis of variance with repeated measures for muscle coactivation and variables for metabolic cost of gait was performed to determine if there was an age main effect, task main effect, or age by task interaction effect.

Tukey's Significantly different post-hoc test $p < 0.05$ was used to determine significane for the ANOVA analysis.

Linear regressions between antagonist muscle coactivation and metabolic cost of gait were also performed to directly test the global hypothesis that the age related increase in metabolic cost of gait was due to increased antagonist muscle coactivation within old individuals.

CHAPTER IV: RESULTS

This chapter is separated into the following sections: 1) Metabolic Results of Level Walking and Task Dependent Walking, 2) Antagonist Muscle Coactivation Results of Level Walking and Task Dependent Walking, 3) Association between Antagonist Muscle Coactivation and Metabolic Cost, and 4) Summary.

The global hypothesis was that the age related increase in metabolic cost was due to increased antagonist muscle coactivation that old individuals experienced. The first sub-hypothesis was that an age effect would be evident in metabolic cost and antagonist muscle coactivation. Old subjects would have higher metabolic cost than young individuals, and old individuals would also have higher antagonist muscle coactivation than young individuals. The second sub-hypothesis was that there would be a velocity effect in antagonist muscle coactivation. As speed increased, antagonist muscle coactivation would also increase. The third sub-hypothesis was based upon the task. Decline walking would elicit greater antagonist muscle coactivation.

Metabolic Results of Level and Task Dependent Walking

This section addresses the first sub hypothesis which states that old adults have higher metabolic cost than young adults.

During level walking old individuals experienced a significantly greater HR ($F = 8.421$, $p = 0.008$), RPE ($F = 13.859$, $p = 0.001$), VO_2 normalized to body weight ($F = 7.883$, $p < 0.001$), gross metabolic power ($F = 6.407$, $p = 0.019$), and gross metabolic energy cost per distance ($F = 4.319$, $p = 0.050$). Young individuals experienced a significantly greater RER ($F = 4.981$, $p = 0.036$). These are shown in Figures 1 through 6, and also represented in Table 2 in the Appendix.

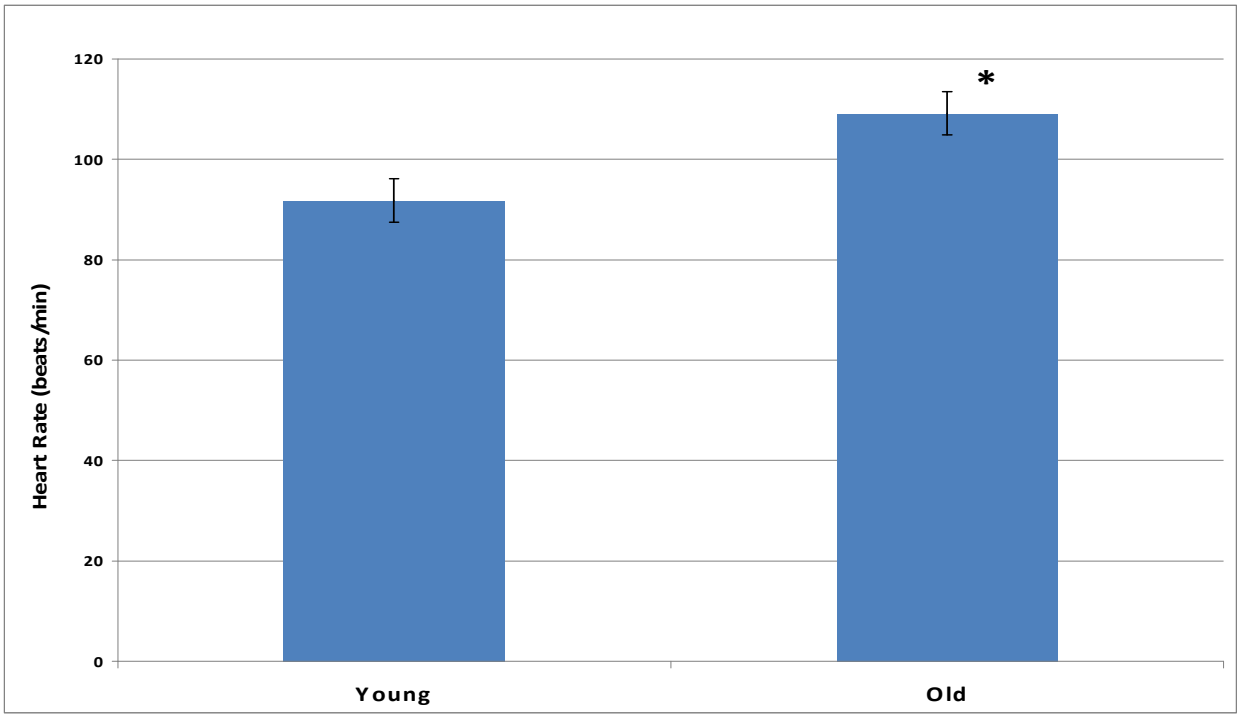

Figure 1. Age main effect of heart rate during level walking. Old adults had a significantly greater HR than young individuals. * denotes an age main effect.

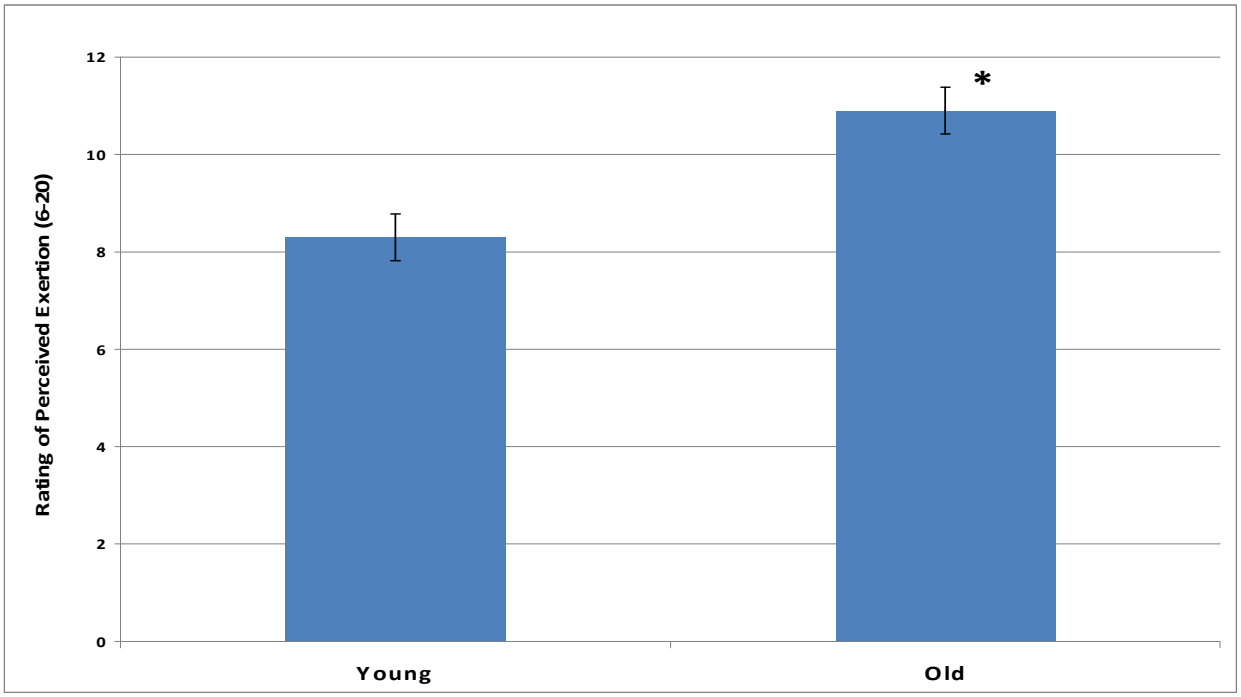

Figure 2. Age main effect of rating of perceived exertion during level walking. Old individuals had a significantly greater RPE than young individuals. * denotes an age main effect.

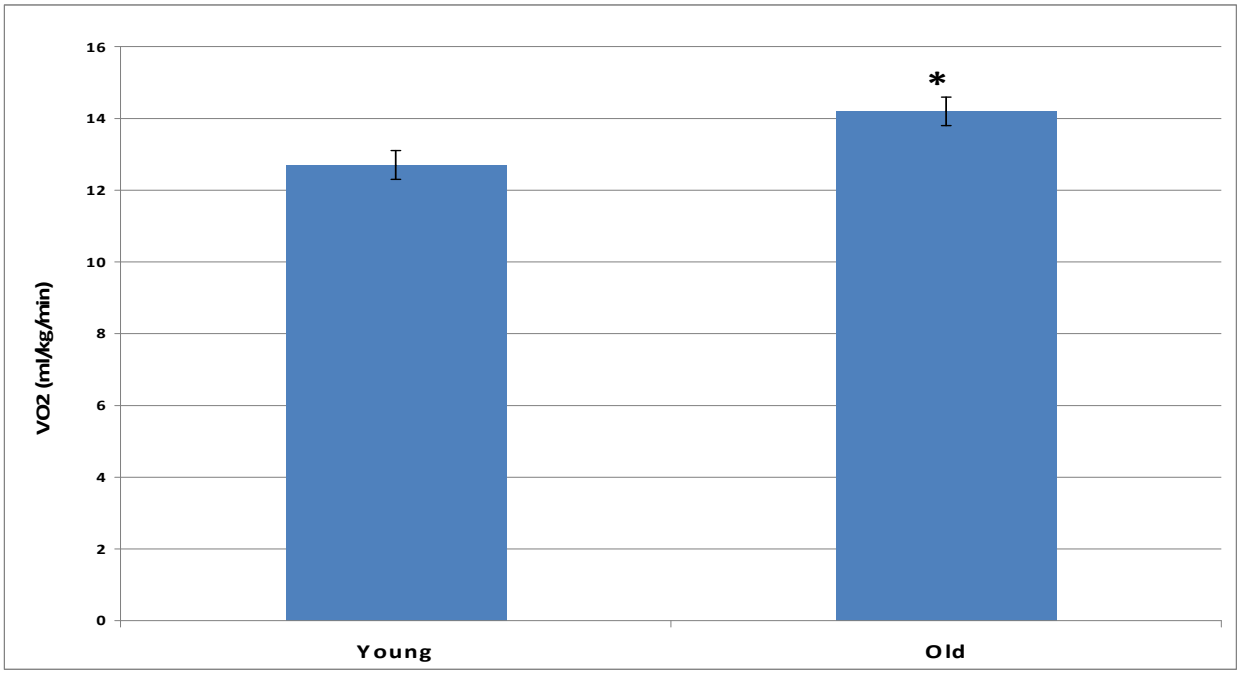

Figure 3. Age main effect of VO_2 normalized to body weight during level walking. Old individuals had a significantly greater VO_2 than young individuals. * denotes an age main effect.

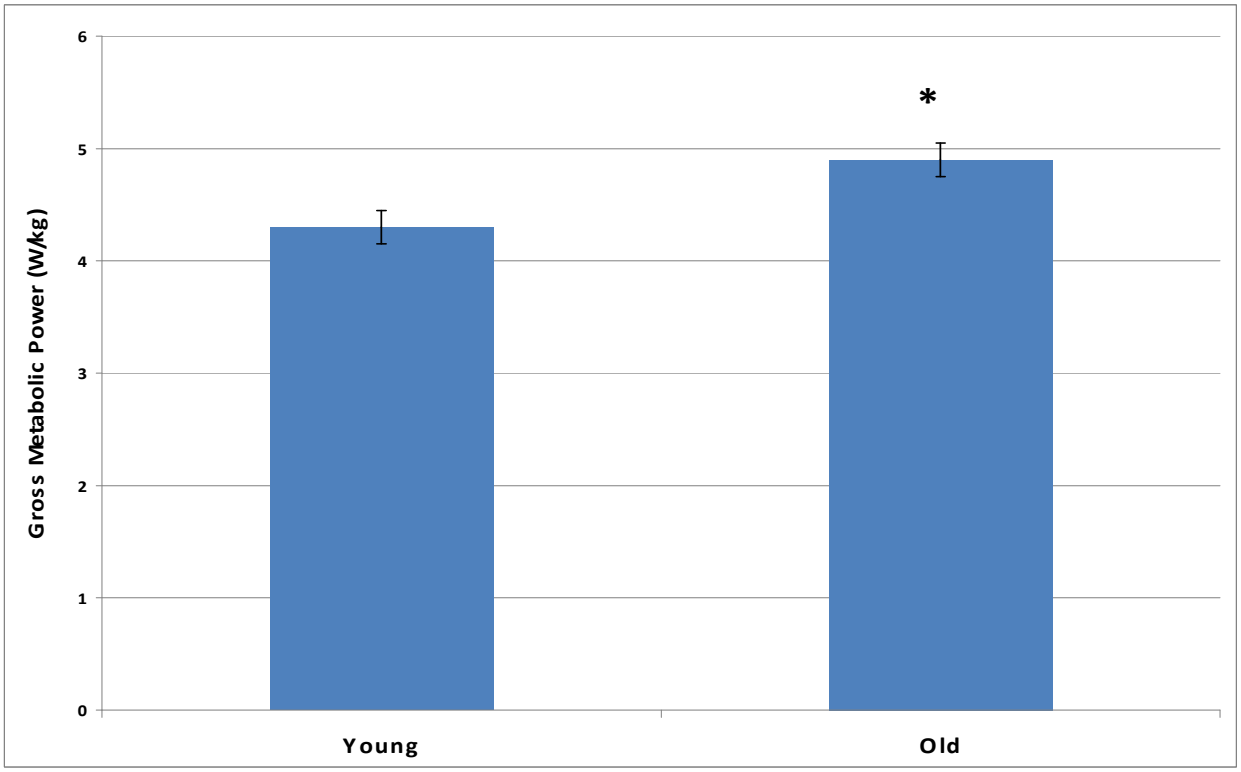

Figure 4. Age main effect of gross metabolic power during level walking. Old individuals had a significantly greater gross metabolic power than young individuals. * denotes an age main effect.

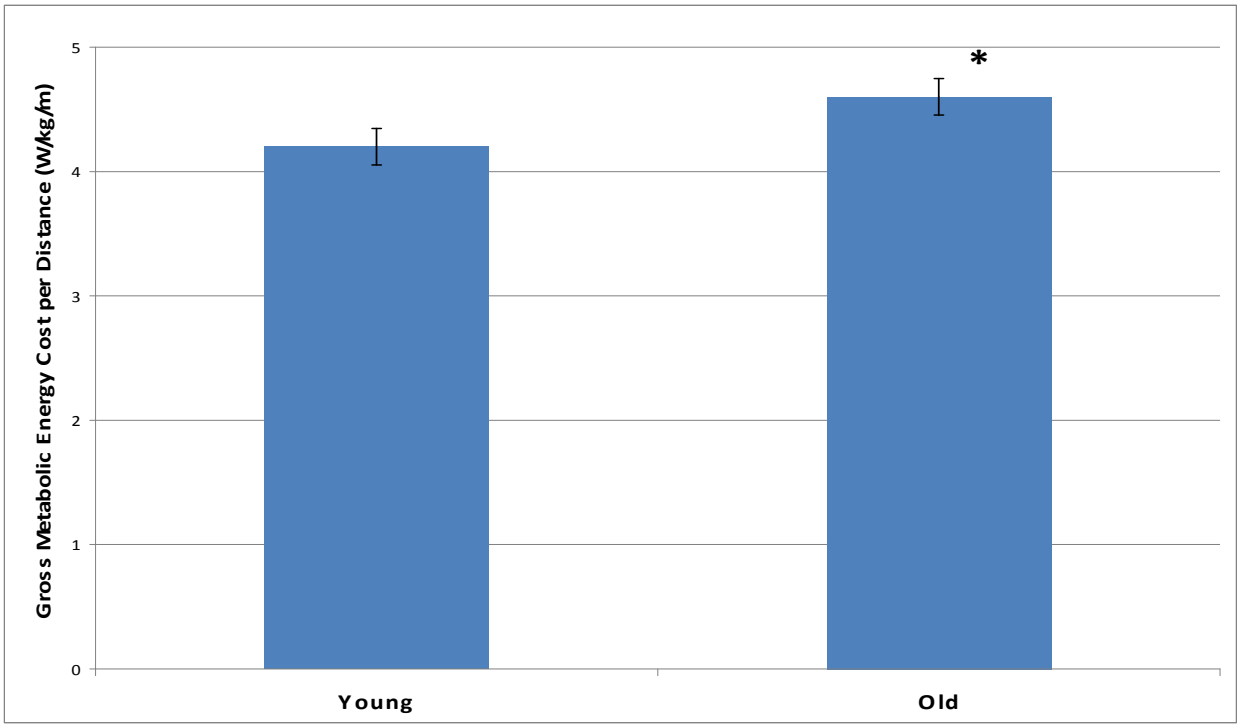

Figure 5. Age main effect of gross metabolic energy cost per distance during level walking. Old individuals had a significantly greater gross metabolic energy cost per distance than young individuals. * denotes an age main effect.

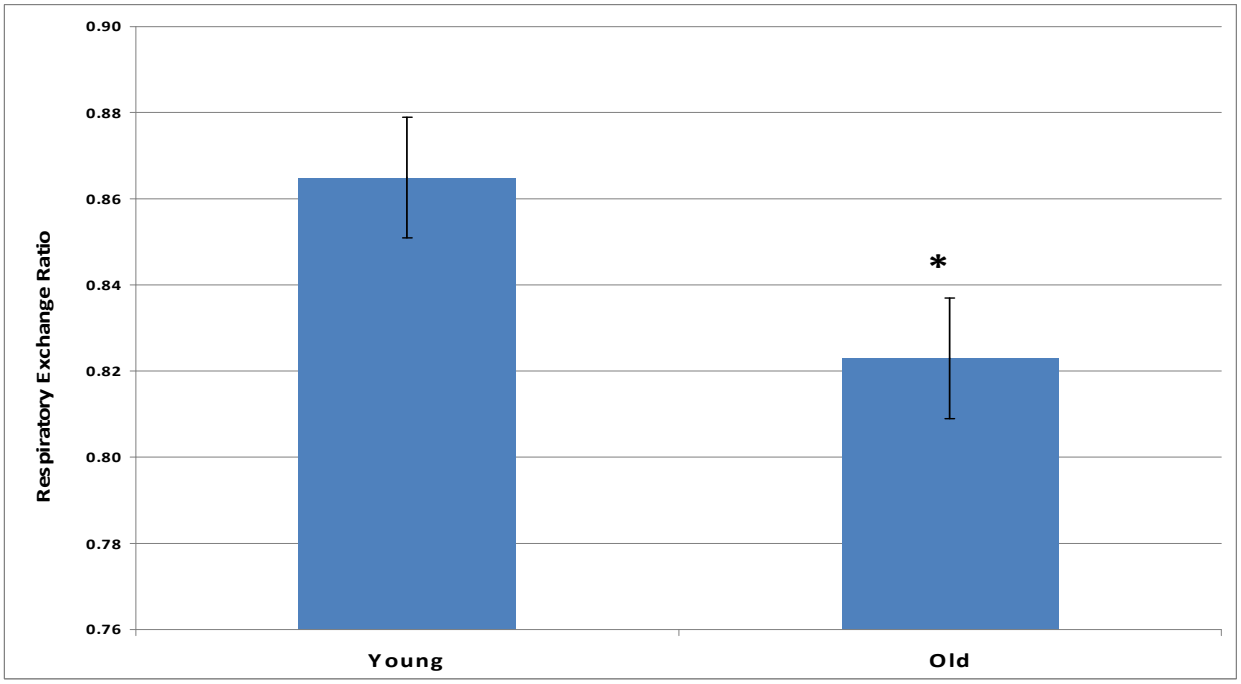

Figure 6. Age main effect of respiratory exchange ratio during level walking. Young individuals had a significantly greater RER than old individuals. * denotes an age main effect.

During task dependent walking conditions old subjects experienced significantly greater HR ($F = 6.047$, $p = 0.005$), RPE ($F = 9.662$, $p = 0.005$), VO_2 normalized to body weight ($F = 4.933$, $p = 0.037$), gross metabolic power ($F = 5.854$, $p = 0.024$), and gross metabolic energy cost per distance ($F = 5.854$, $p = 0.024$). Young subjects experienced a significantly greater RER ($F = 4.472$ $p = 0.046$). These data are shown in Figures 7 through 12, and also in Table 3 in the Appendix.

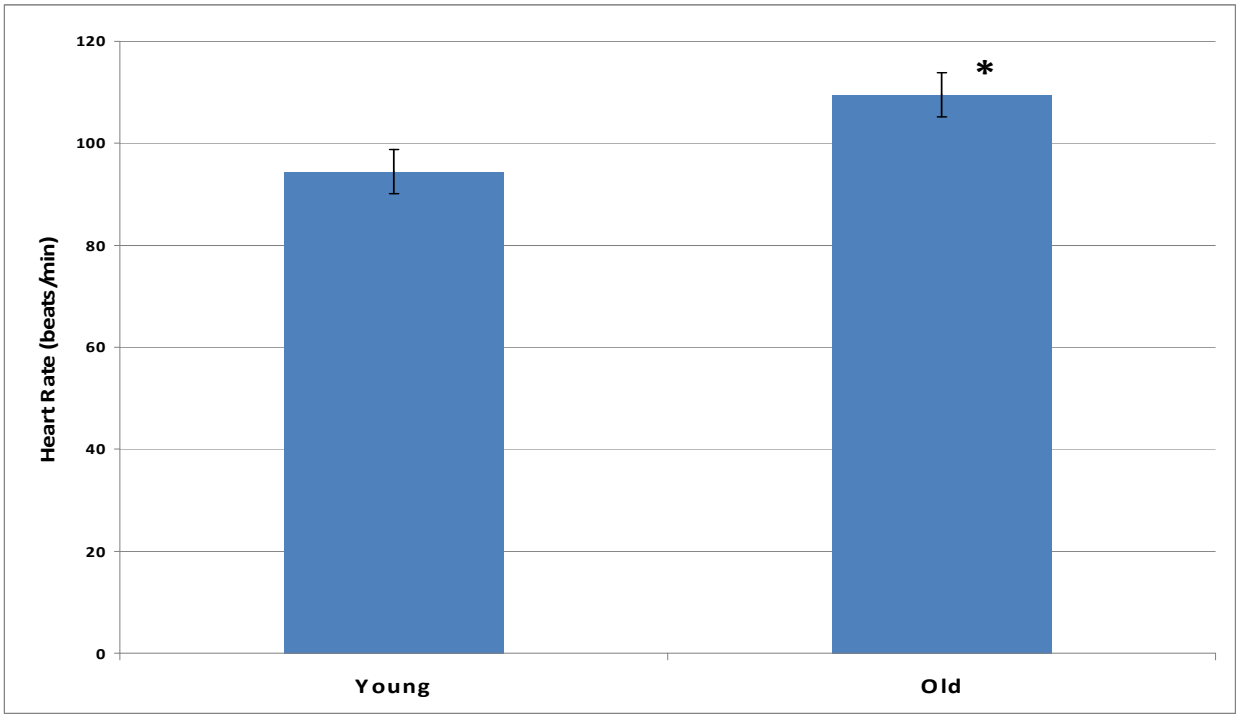

Figure 7. Age main effect of heart rate during incline and decline walking. Old individuals had a significantly greater HR than young individuals. * denotes an age main effect.

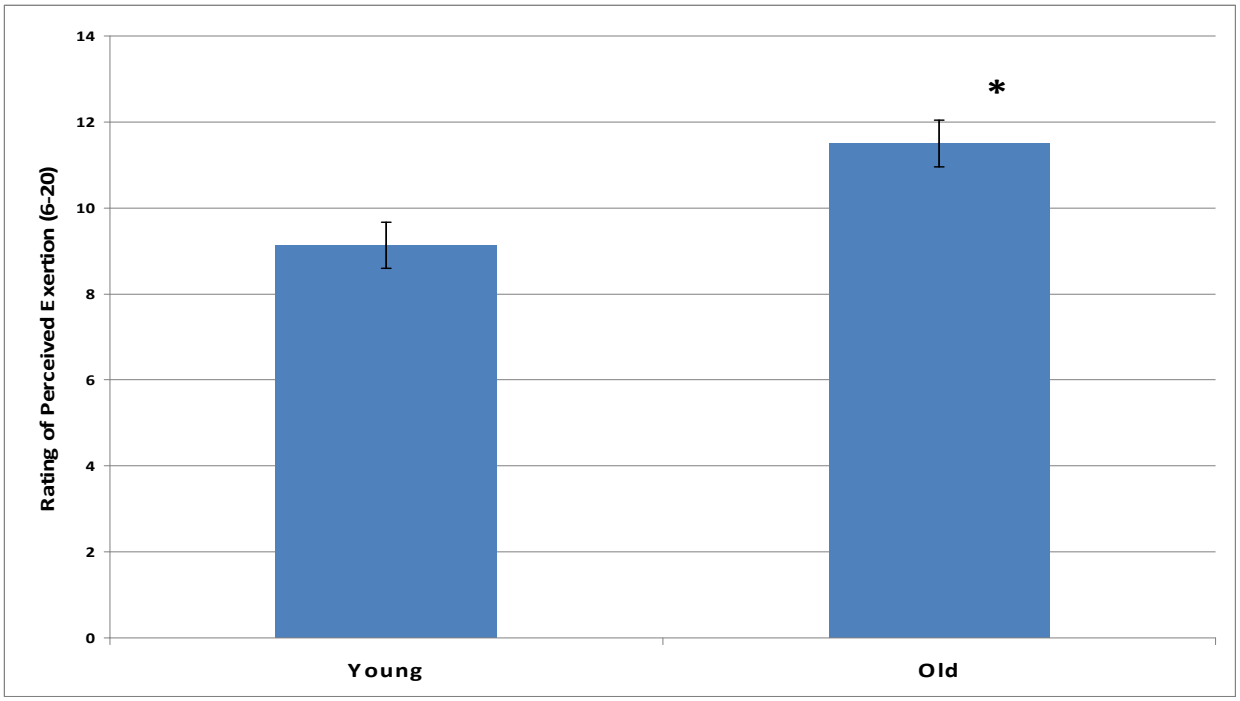

Figure 8. Age main effect of rating of perceived exertion during incline and decline walking. Old individuals had a significantly greater RPE than young individuals. * denotes an age main effect.

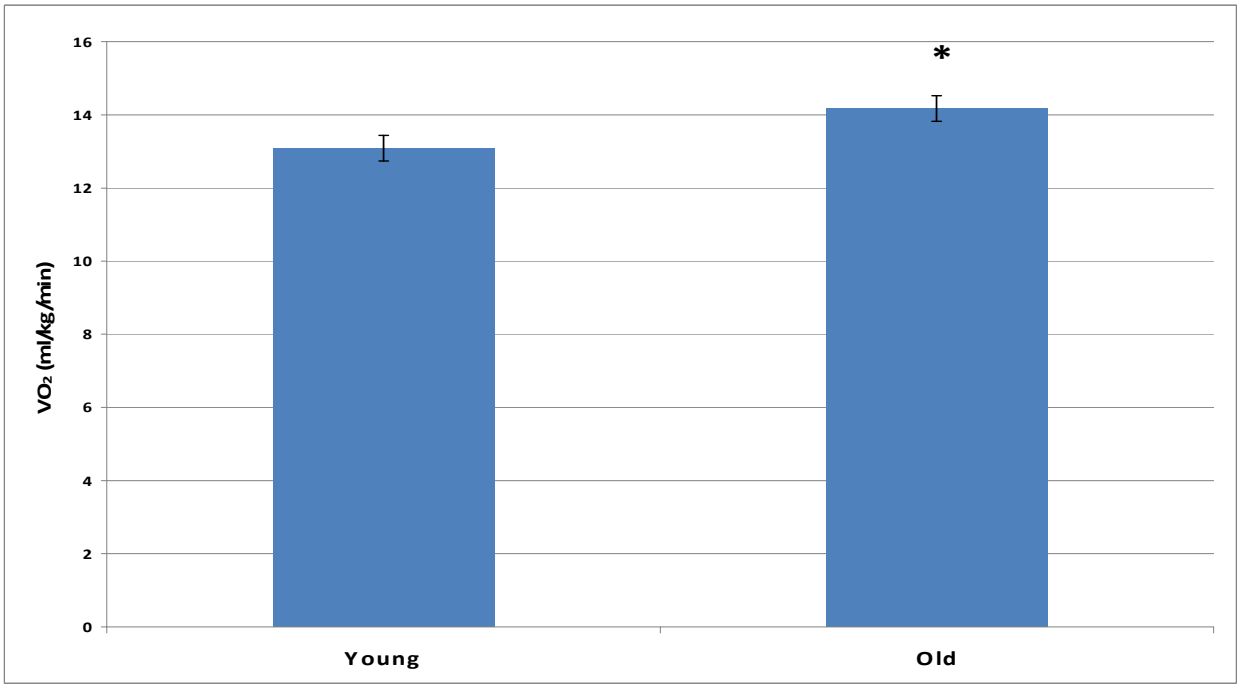

Figure 9. Age main effect of VO2 normalized to body weight during incline and decline walking. Old individuals had a significantly greater VO2 than young individuals. * denotes an age main effect.

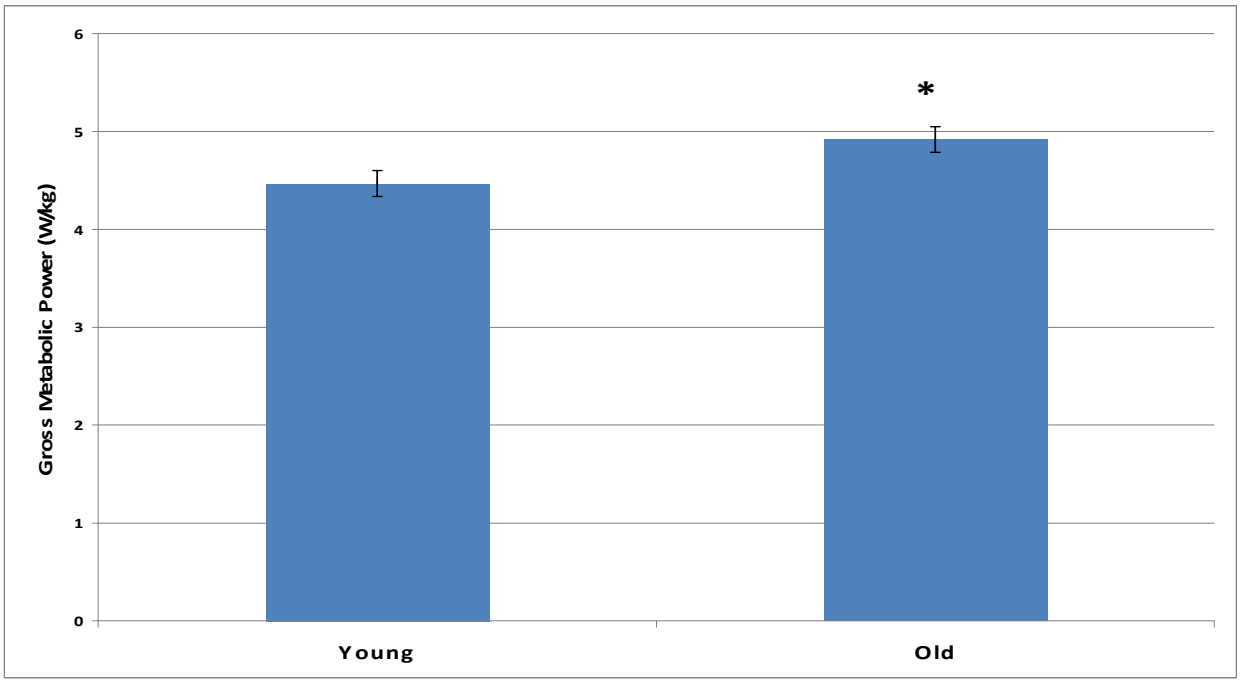

Figure 10. Age main effect of gross metabolic power during incline and decline walking. Old individuals had a significantly greater metabolic power than young individuals. * denotes an age main effect.

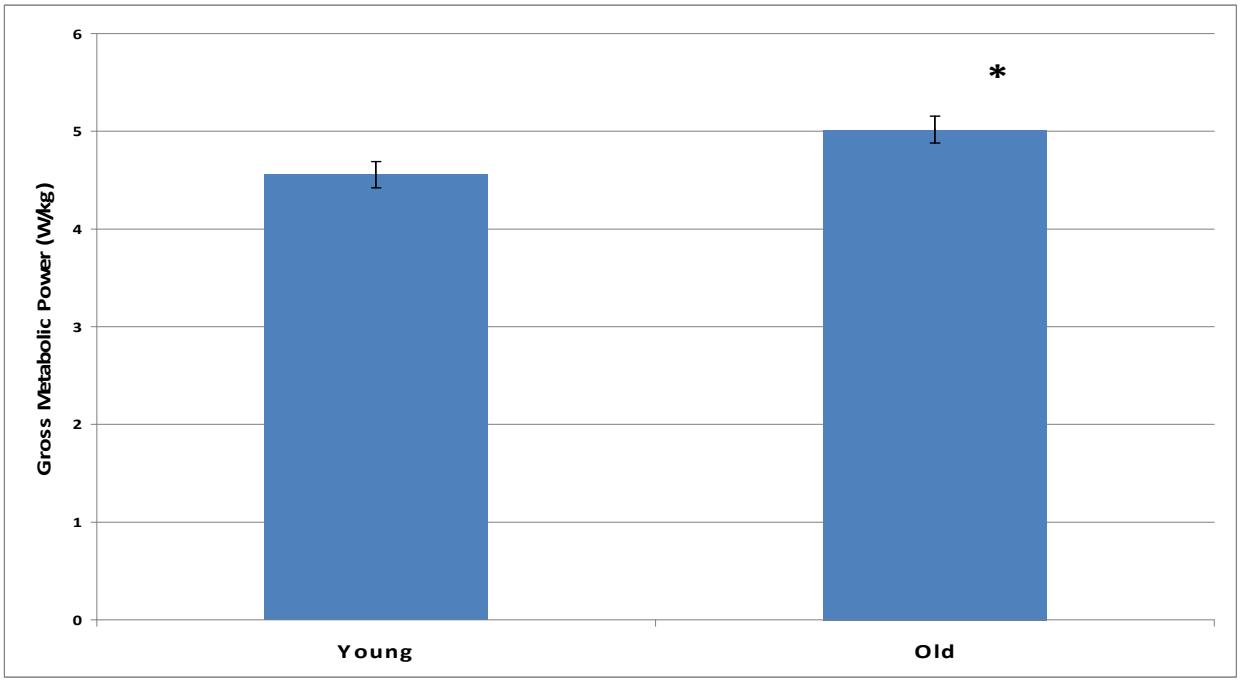

Figure 11. Age main effect of gross metabolic energy cost per distance during incline and decline walking. Old individuals had a significantly greater gross metabolic energy cost per distance than young individuals. * denotes an age main effect.

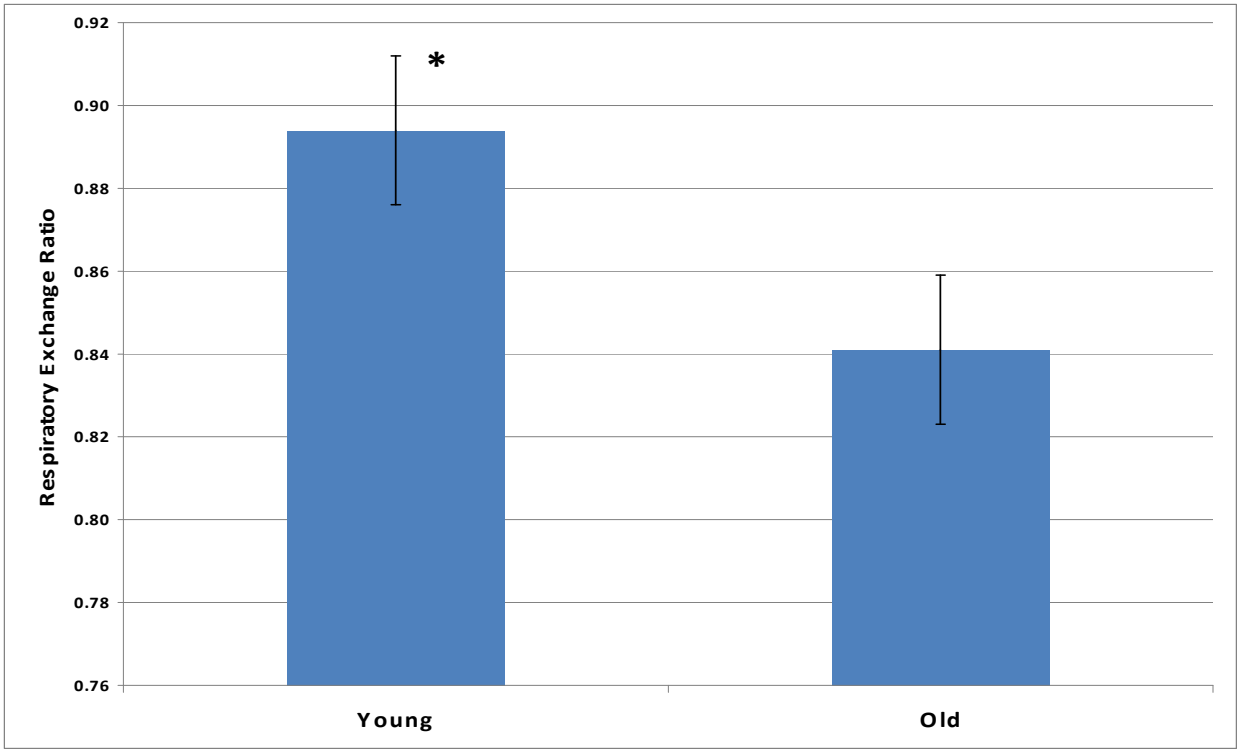

Figure 12. Age main effect of respiratory exchange ratio during incline and decline walking. Young individuals had a significantly greater RER than old individuals. * denotes an age main effect.

Figure 13 shows the percent difference between young and old for each metabolic variable in which old individuals had greater values than young individuals.

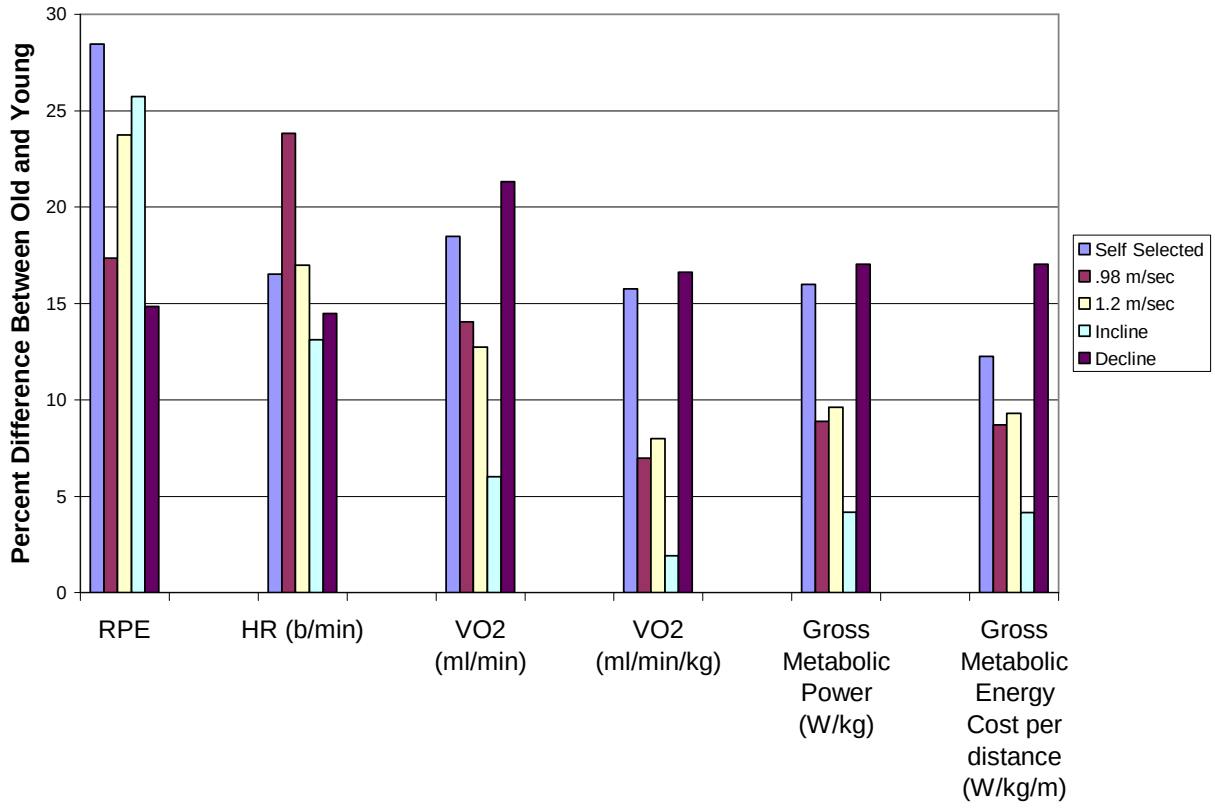

Figure 13. Percent difference between old and young for metabolic variables.

Different velocities also affected metabolic variables. There was a velocity effect in HR between .98 and 1.2 m/sec ($F = 8.997$, $p = 0.001$). RPE was significantly greater at a velocity of 1.2 m/sec compared to both .98 m/sec and self selected velocities ($F = 8.997$, $p = 0.050$). VO2 was greater at a velocity of 1.2 m/sec compared to both .98 m/sec and self selected velocities ($F = 14.279$, $p < 0.001$). When VO2 was normalized to body weight each velocity was significantly different from the others ($F = 13.707$, $p = 0.010$). Gross metabolic power was greater at a velocity of 1.2 compared to both .98 ($F = 12.83$ $p < 0.001$) and self selected ($F = 12.83$ $p = 0.050$) velocities. Gross metabolic energy cost per distance ($F = 11.924$ $p < 0.001$) was significantly lower at a velocity of 1.2 m/sec compared to both .98 m/sec and self selected velocities. These data are shown in Figures 14 through 19, and in Table 2.

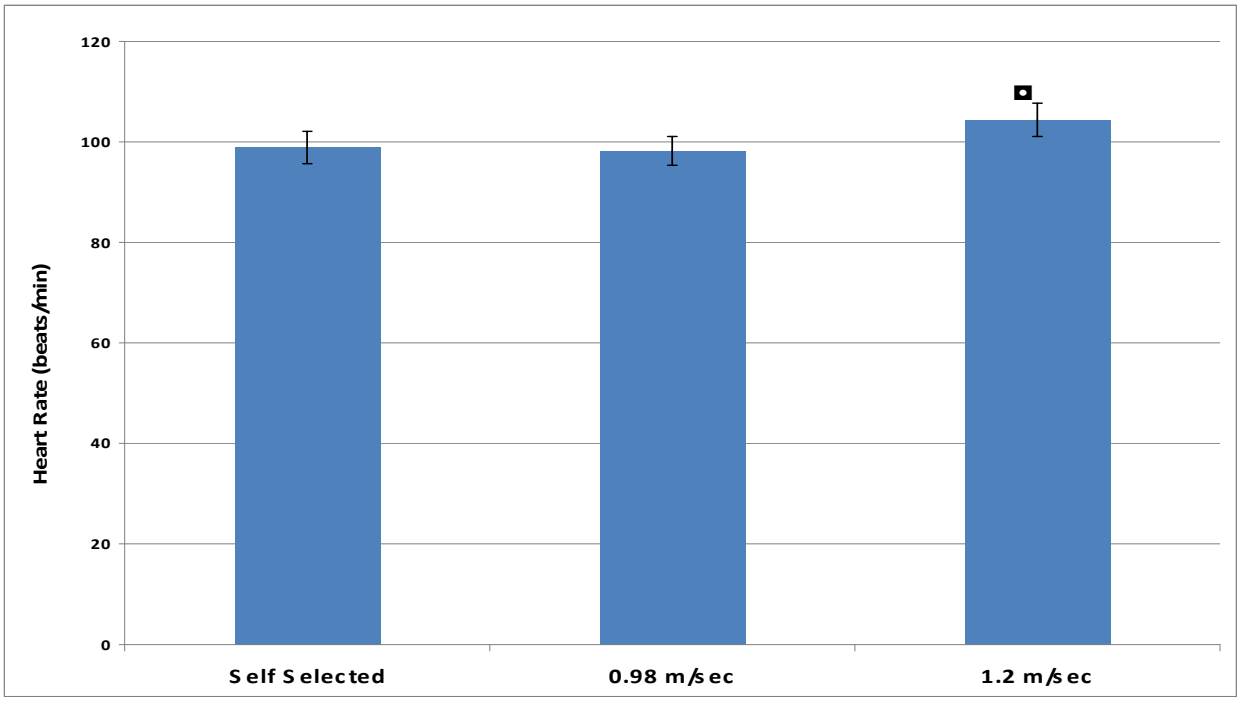

Figure 14. Velocity main effect of Heart Rate in old and young adults. HR during 1.2 m/sec was significantly greater than HR during .98 m/sec. ◘ denotes a velocity main effect.

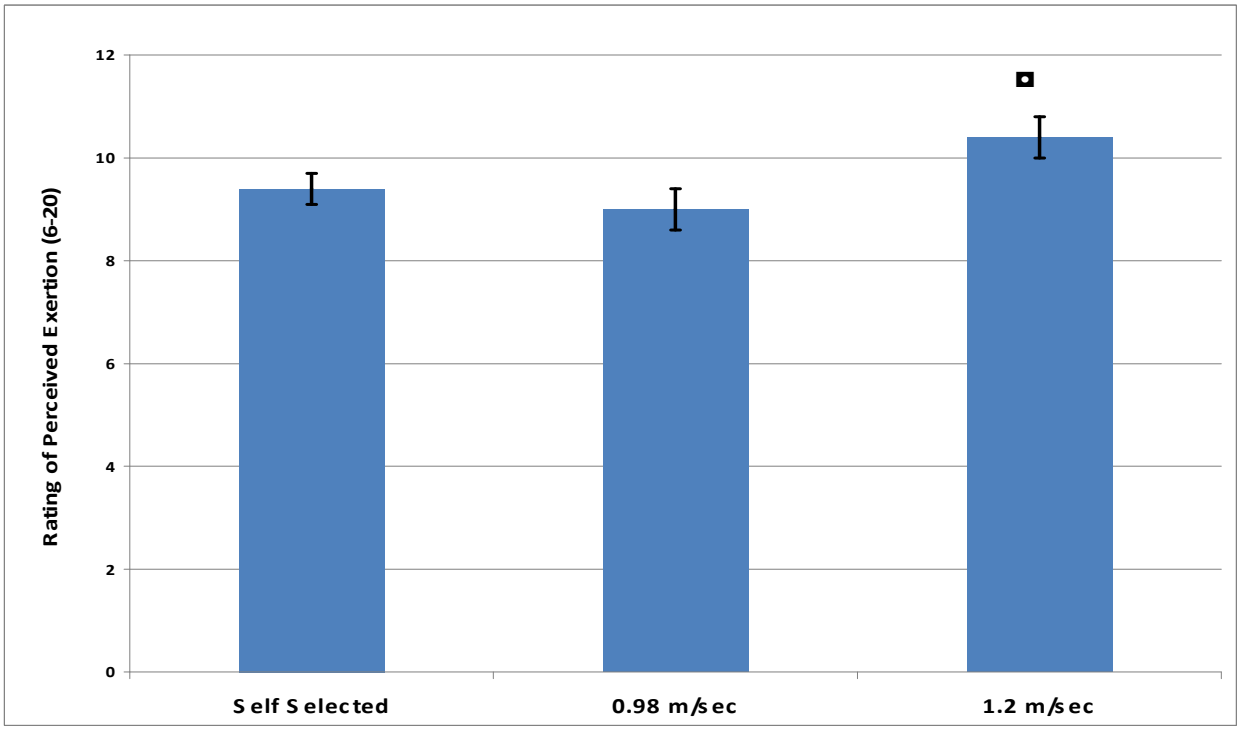

Figure 15. Velocity main effect of rating of perceived exertion in old and young adults. RPE during 1.2 m/sec was significantly greater than RPE during .98 m/sec and self-selected velocities. ◘ denotes a velocity main effect.

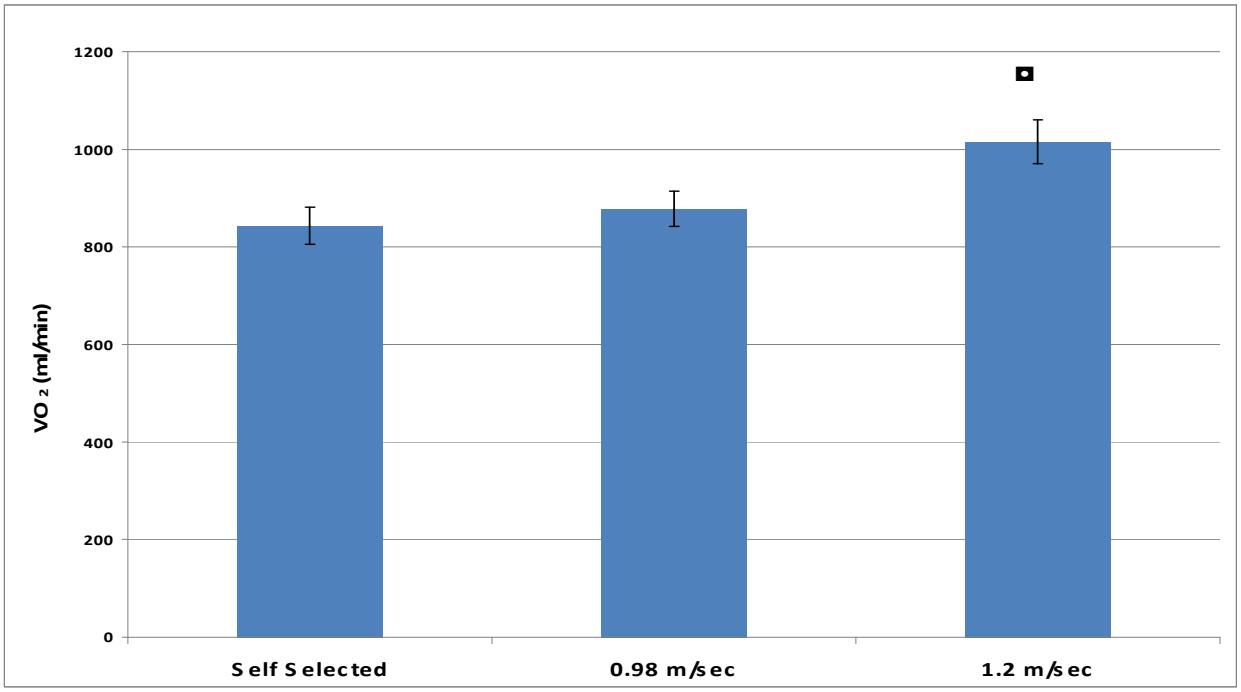

Figure 16. Velocity main effect of VO2. VO2 during 1.2 m/sec was significantly greater than VO2 during .98 m/sec and self-selected velocities. ◘ denotes a velocity main effect.

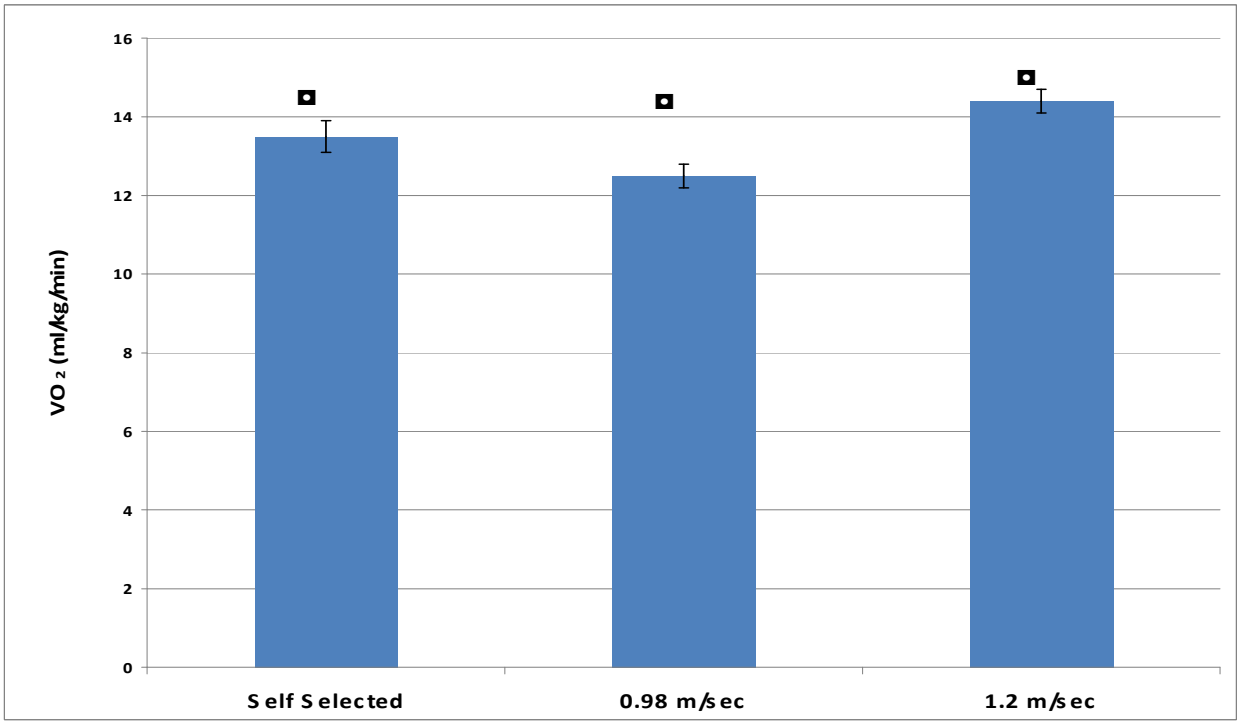

Figure 17. Velocity main effect of VO2 normalized to body weight in old and young adults. VO2 at each velocity was significantly different from the other two velocities. ◘ denotes a velocity main effect

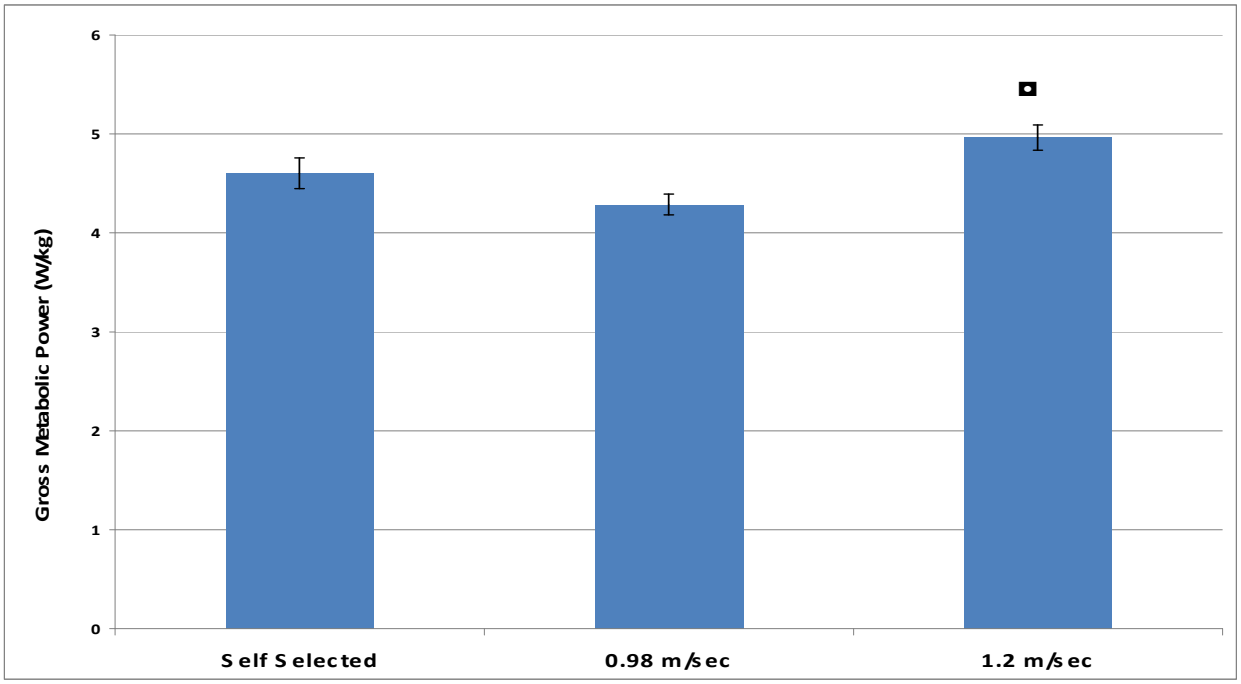

Figure 18. Velocity main effect of gross metabolic power in old and young adults. Gross metabolic power during 1.2 m/sec was significantly greater than metabolic power during .98 m/sec and self-selected velocities. ◘ denotes a velocity main effect.

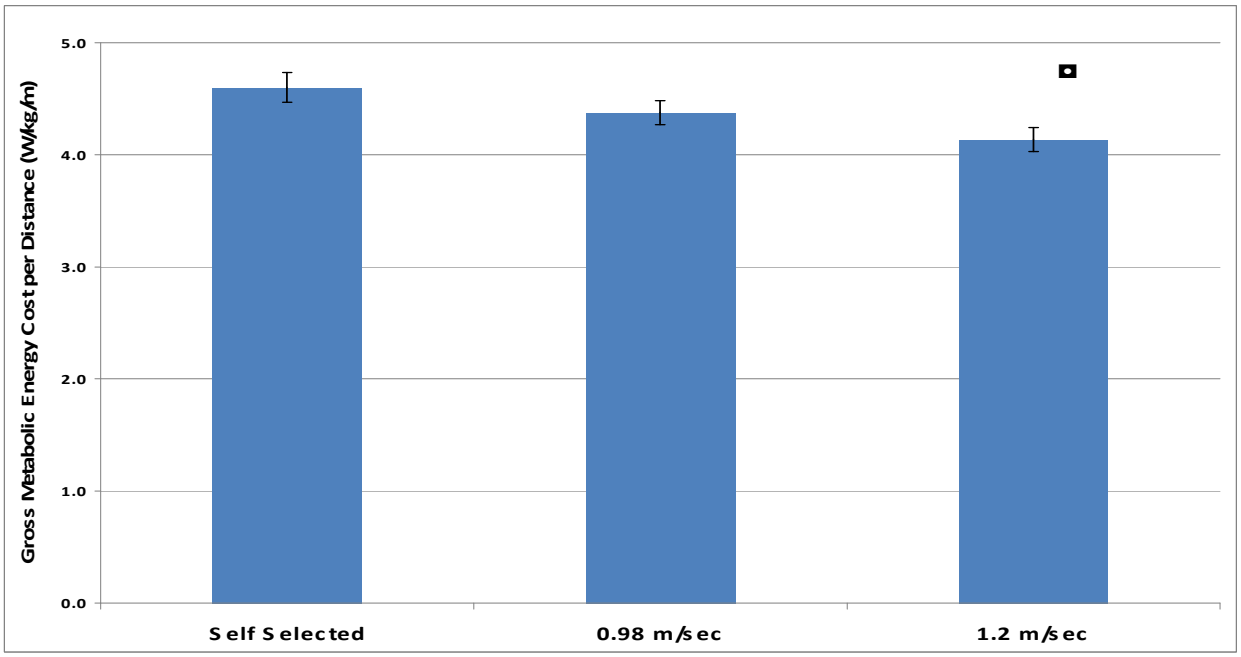

Figure 19. Velocity main effect of gross metabolic energy cost per distance in old and young adults. Metabolic energy cost per distance during 1.2 m/sec was significantly lower than metabolic energy cost per distance during .98 m/sec and self-selected velocities. ◘ denotes a velocity main effect.

During task dependent gait, walking at an incline caused subjects to have a significantly greater HR ($F = 145.734$, $p < 0.001$), RPE ($F = 23.032$, $p < 0.001$), VO2 ($F = 270.542$, $p < 0.001$), VO2 normalized to body weight ($F = 256.835$, $p < 0.001$), gross metabolic power ($F = 236.489$, $p < 0.001$), and gross metabolic energy cost per distance ($F = 236.489$, $p < 0.001$) than when they were walking at a decline. These data are shown in Figures 20 through 25, and Table 3.

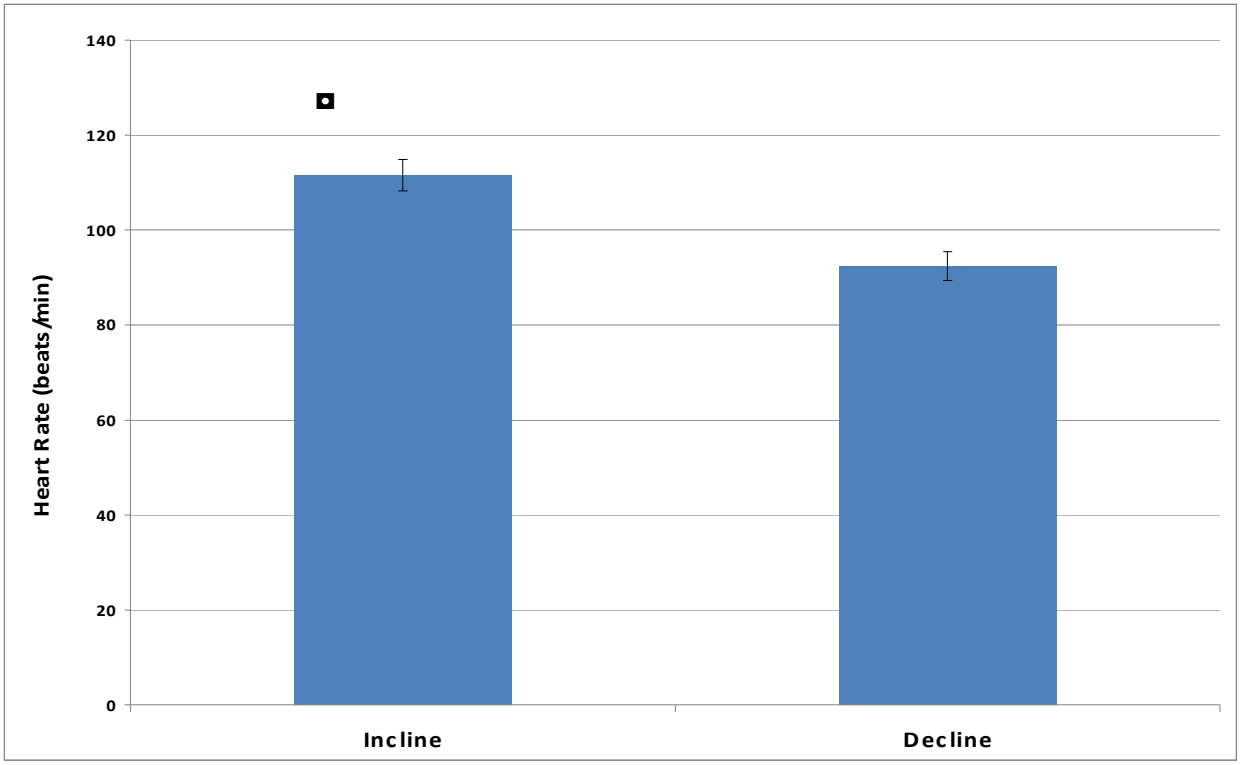

Figure 20. Task main effect of Heart Rate. HR during Incline was significantly greater than HR during decline. ◘ denotes a task main effect.

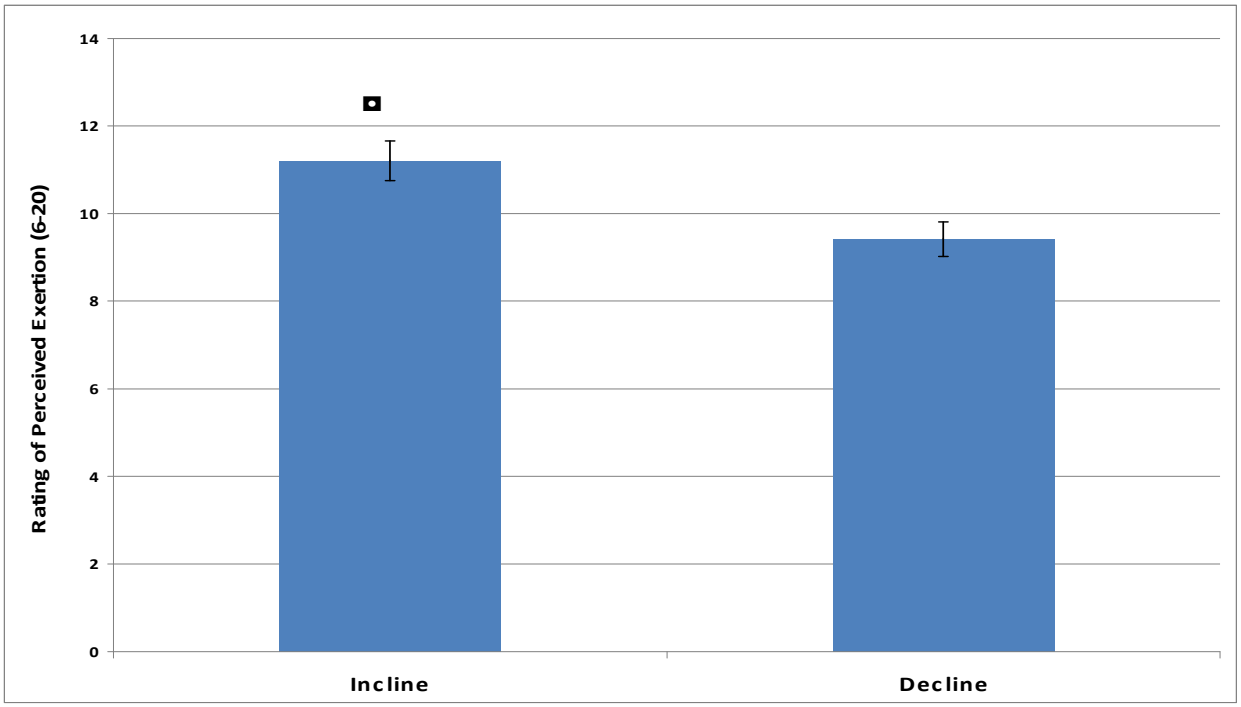

Figure 21. Task main effect of RPE. RPE during Incline was significantly greater than RPE during decline. ◘ denotes a task main effect.

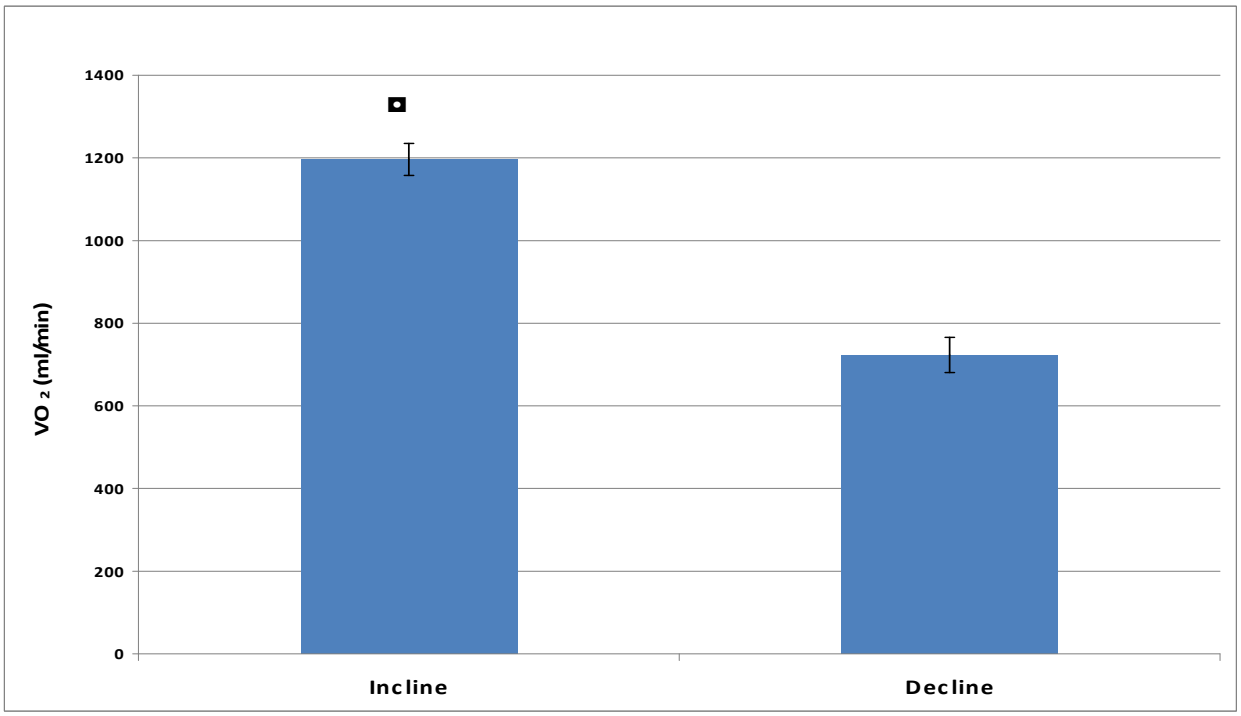

Figure 22. Task main effect of VO_2. VO_2 during incline was significantly greater than VO_2 during decline. ◘ denotes a task main effect.

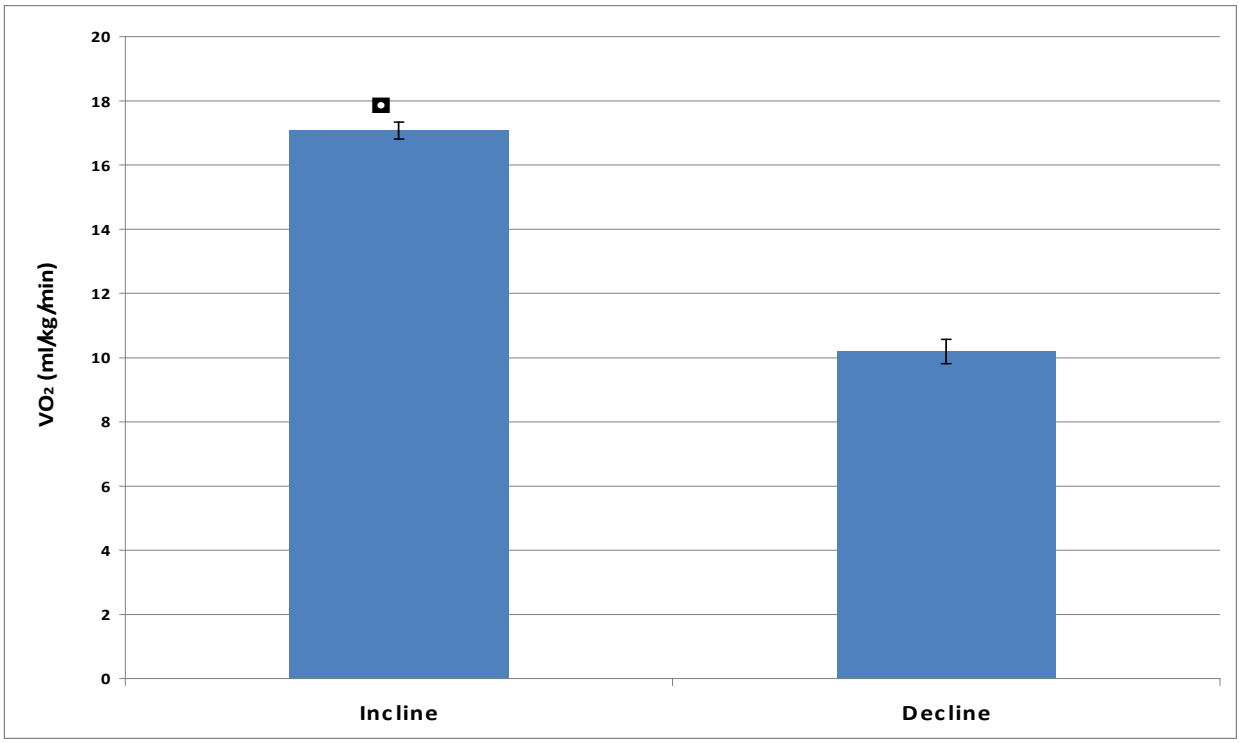

Figure 23. Task main effect of VO_2 normalized to body weight. VO_2 during incline was significantly greater than VO_2 during decline. ◘ denotes a task main effect.

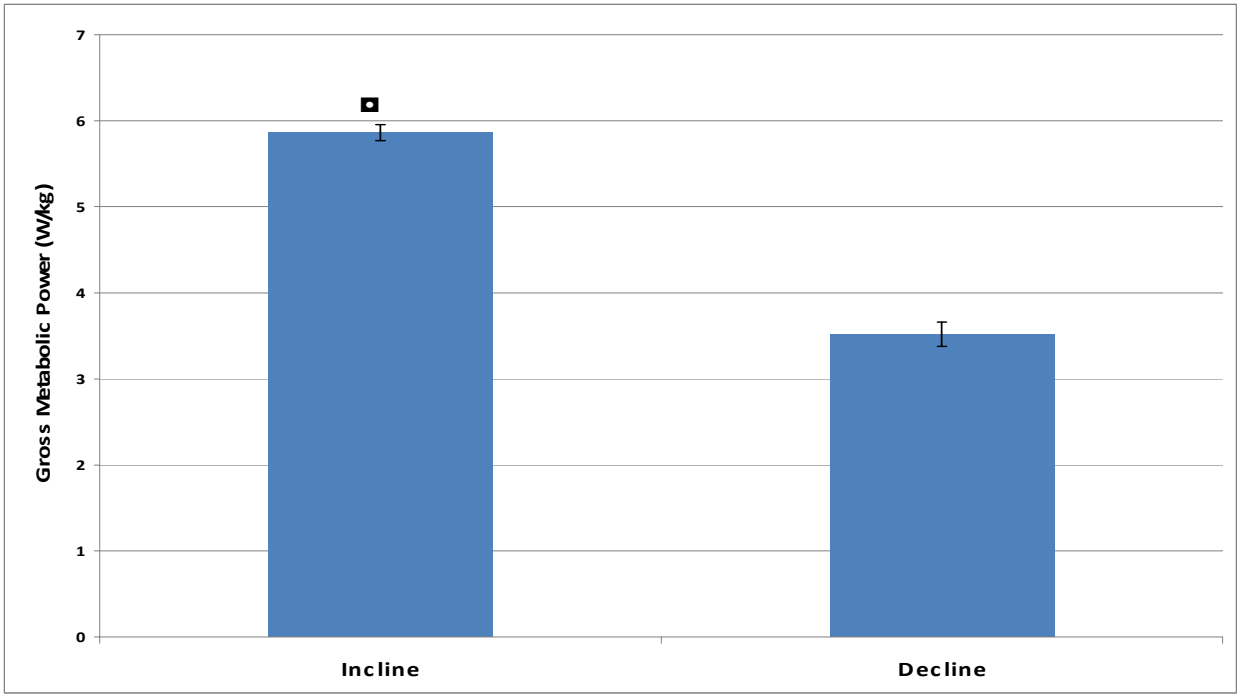

Figure 24. Task main effect of gross metabolic power. Gross metabolic power during incline was significantly greater than metabolic power during decline. ◘ denotes a task main effect.

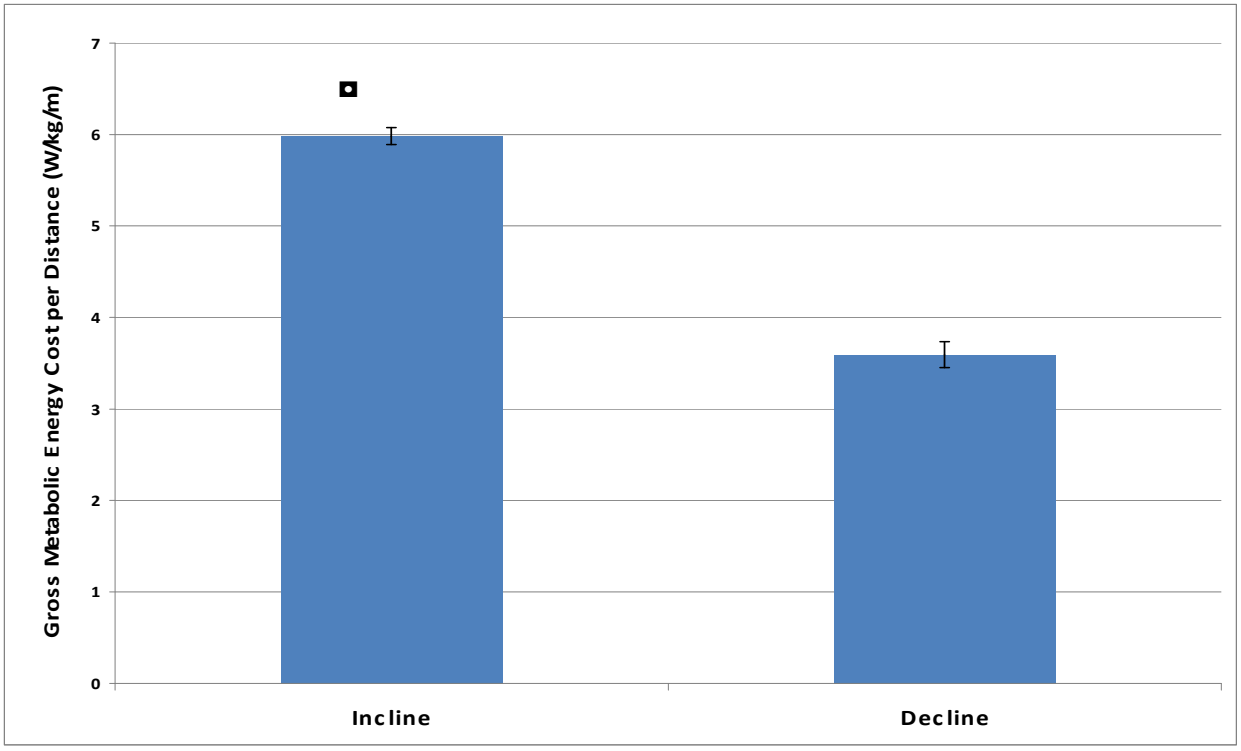

Figure 25. Task main effect of gross metabolic energy cost per distance. Gross metabolic energy per distance was significantly greater during incline than during decline. ◘ denotes a task main effect.

There were no interaction effects for level walking conditions. Figures 50- 56 in the Appendix show the metabolic variable values.

Figure 26 shows the interaction of age and task of RPE. Old subjects had a greater RPE than young subjects in both incline ($F = 5.494$, $p < 0.001$) and decline ($F = 5.494$, $p = 0.050$) conditions. Additionally old subjects had a significantly greater RPE in incline walking compared to their RPE in decline walking ($F = 5.494$, $p < 0.001$).

Figure 27 shows the interaction of age and task of RER. Young individuals experienced greater RPE than old individuals during decline walking ($F = 9.837$, $p < 0.001$).

There were no other significant age and task interaction effects; however the other variables may be seen in Figures 57 through 61 in the Appendix.

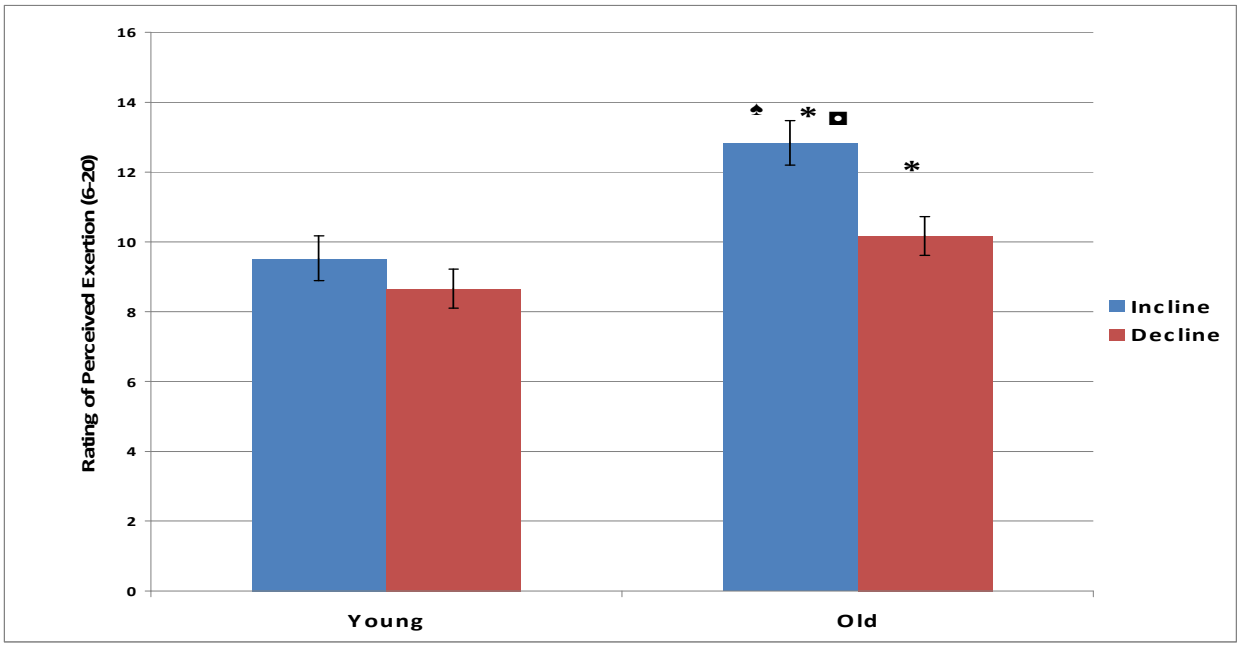

Figure 26. Age by task interaction of RPE. Old incline RPE was significantly greater than young incline RPE, old decline RPE, and young decline RPE. Old decline RPE was significantly greater than young decline RPE. * denotes an age effect. ◘ denotes a task effect. ♠ denotes an age by task interaction.

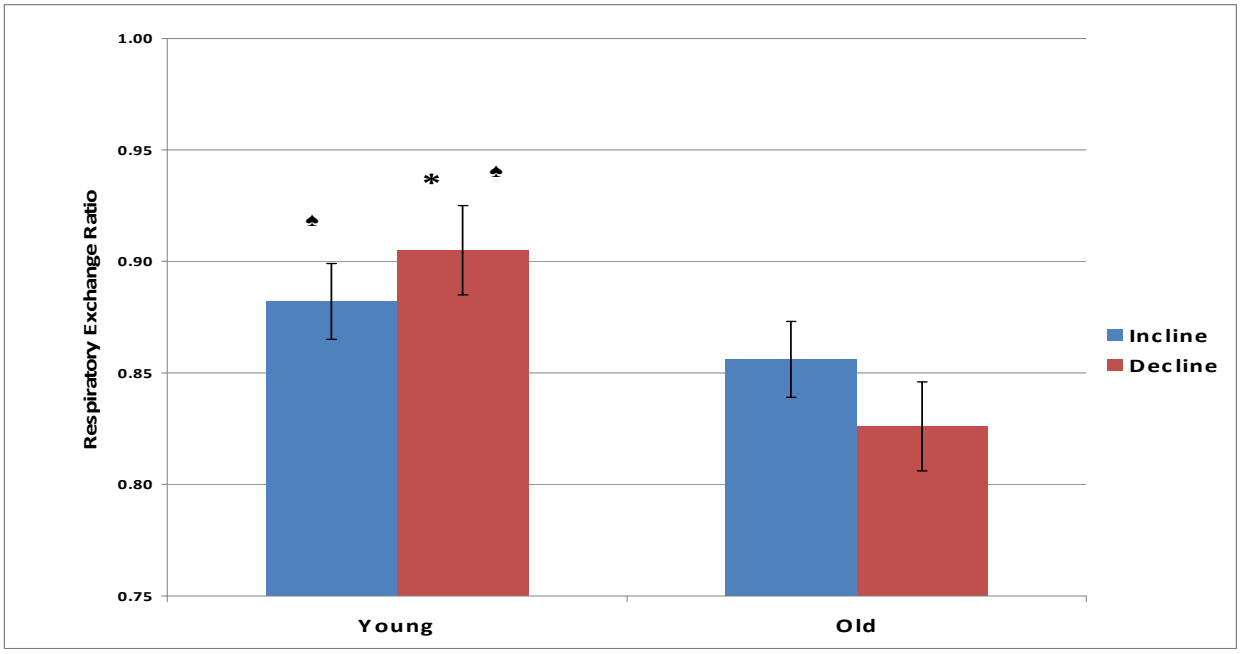

Figure 27. Age by task interaction of RER. Young decline RER was significantly greater than old decline RER and old incline RER. Young incline RER was significantly greater than old decline RER. * denotes an age effect. ♠ denotes an age by task interaction.

Antagonist Muscle Coactivation Results of Level Walking and Task Dependent Walking

To support the first sub-hypothesis old individuals had greater antagonist muscle coactivation during level walking at different velocities. Old adults had significantly greater antagonist muscle coactivation of the BF ($F = 19.052$, $p < 0.001$), VL ($F= 17.536$, $p <0.001$), TA ($F = 4.531$, $p =0.045$), LG ($F = 17.808$, $p <0.001$), and total antagonist muscle coactivation ($F = 31.239$, $p < 0.001$). These age effects are shown in Figures 28 through 32, and also represented in Table 4.

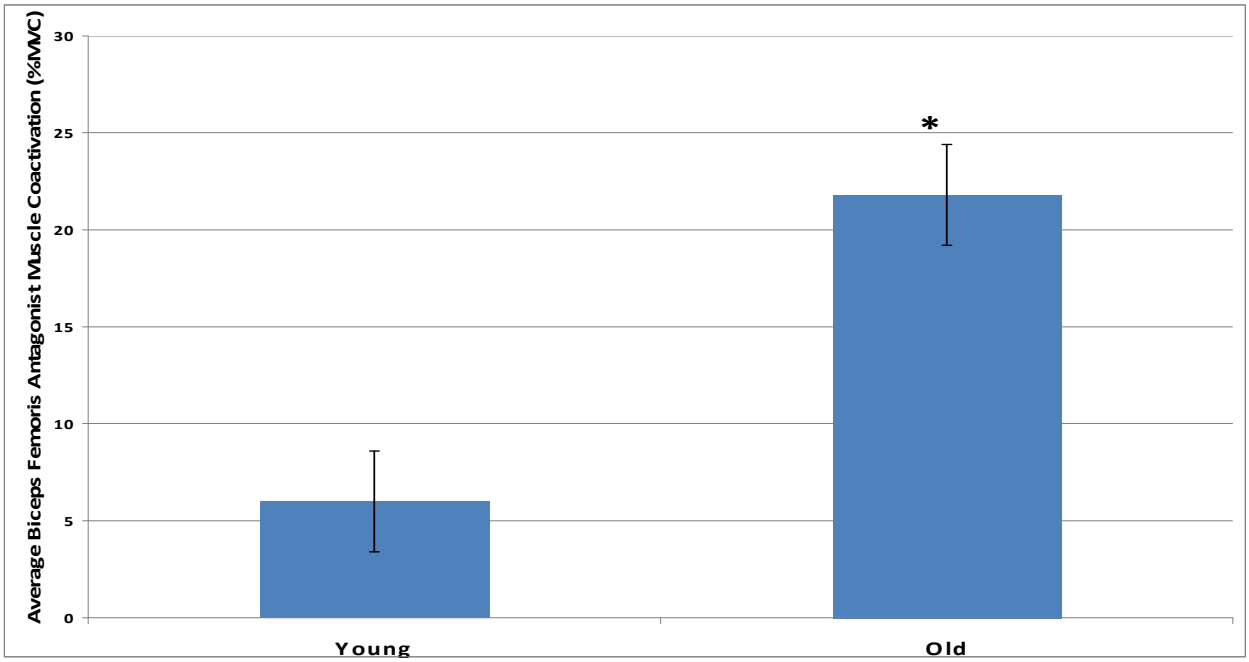

Figure 28. Age main effect of biceps femoris antagonist muscle coactivation during level walking. Old adults had significantly greater BF antagonist muscle coactivation than young adults. * denotes an age main effect.

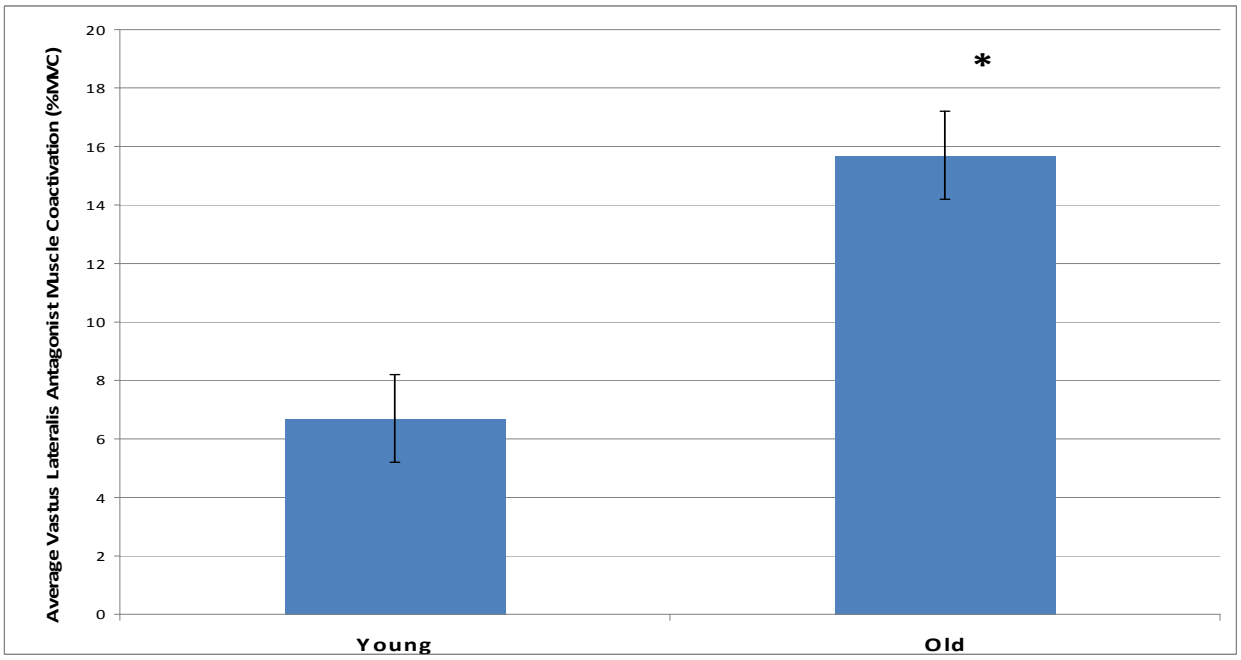

Figure 29. Age main effect of vastus lateralis antagonist muscle coactivation during level walking. Old adults had a significantly greater VL antagonist muscle coactivation than young individuals. * denotes an age main effect

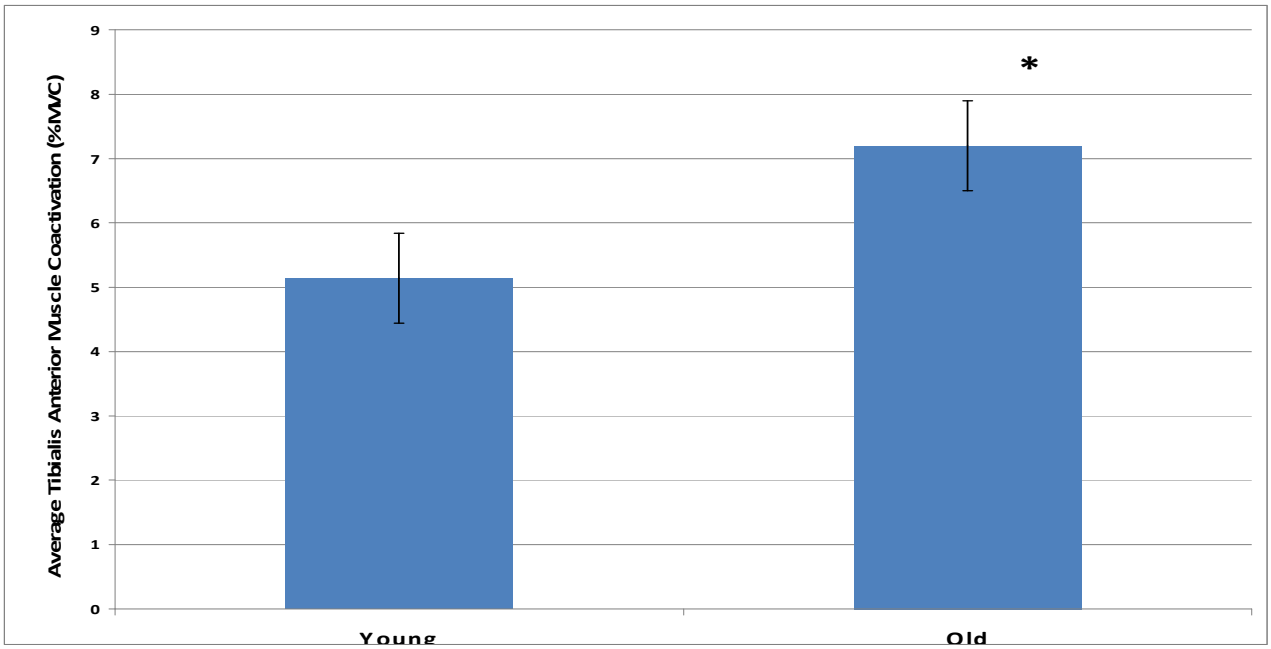

Figure 30. Age main effect of tibialis anterior antagonist muscle coactivation during level walking. Old adults had a significantly greater TA antagonist muscle coactivation than young adults. * denotes an age main effect

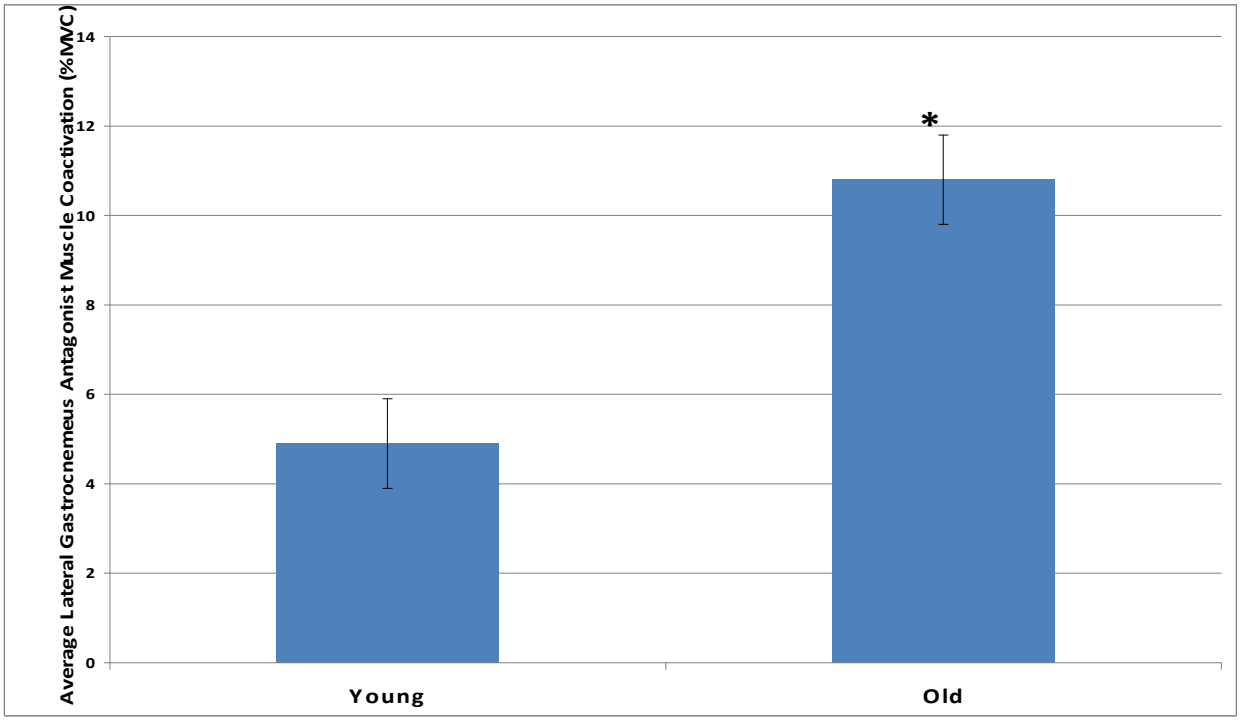

Figure 31. Age main effect of lateral gastrocnemeus antagonist muscle coactivation during level walking. Old adults had a significantly greater LG antagonist muscle coactivation than young individuals. * denotes an age main effect

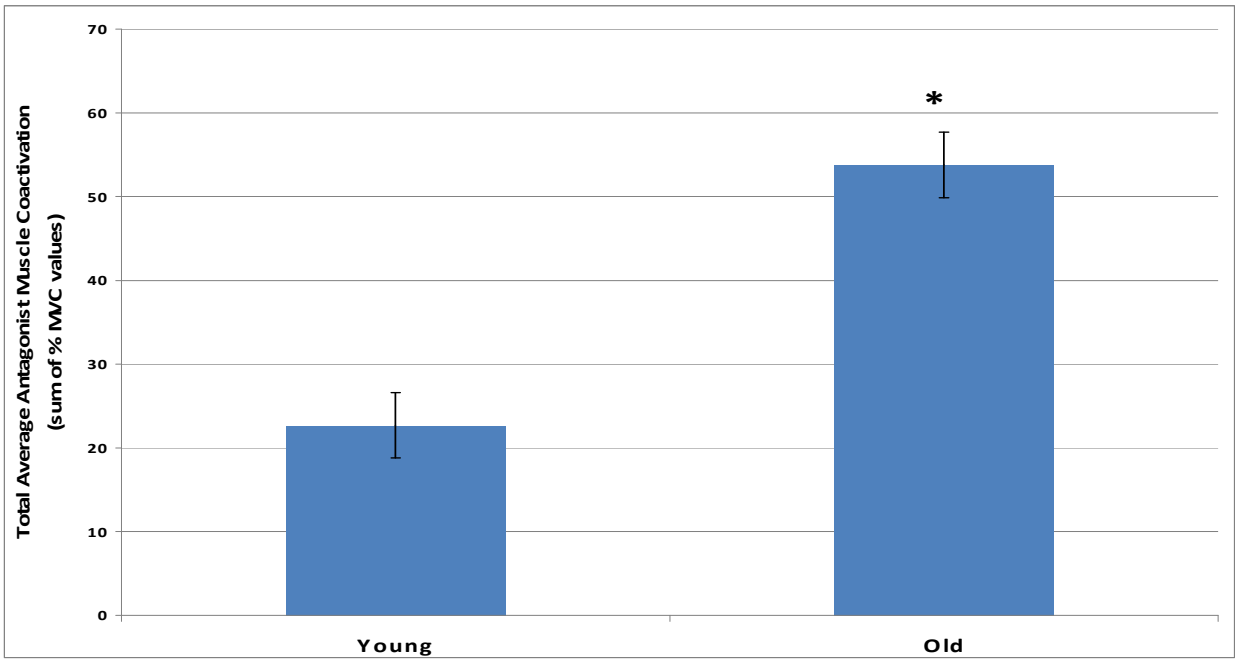

Figure 32. Age main effect of total antagonist muscle coactivation during level walking. The antagonist muscle coactivation of the BF, VL, TA, and LG normalized to their MVC's were added together to determine the total coactivation. Old Adults had a significantly greater total antagonist muscle coactivation than young individuals. * denotes an age main effect

During incline and decline walking old adults had significantly greater antagonist muscle coactivation of their BF ($F = 50.705$, $p < 0.001$), VL ($F = 27.911$, $p < 0.001$), LG ($F = 8.040$, $p = 0.010$), and total antagonist muscle coactivation ($F = 59.103$, $p < 0.001$). These data are shown in Figures 33, 34, 36, and 37 respectively. There was no significant age effect of TA antagonist muscle coactivation. This is shown in Figure 35. Table 5 in the Appendix also shows these values

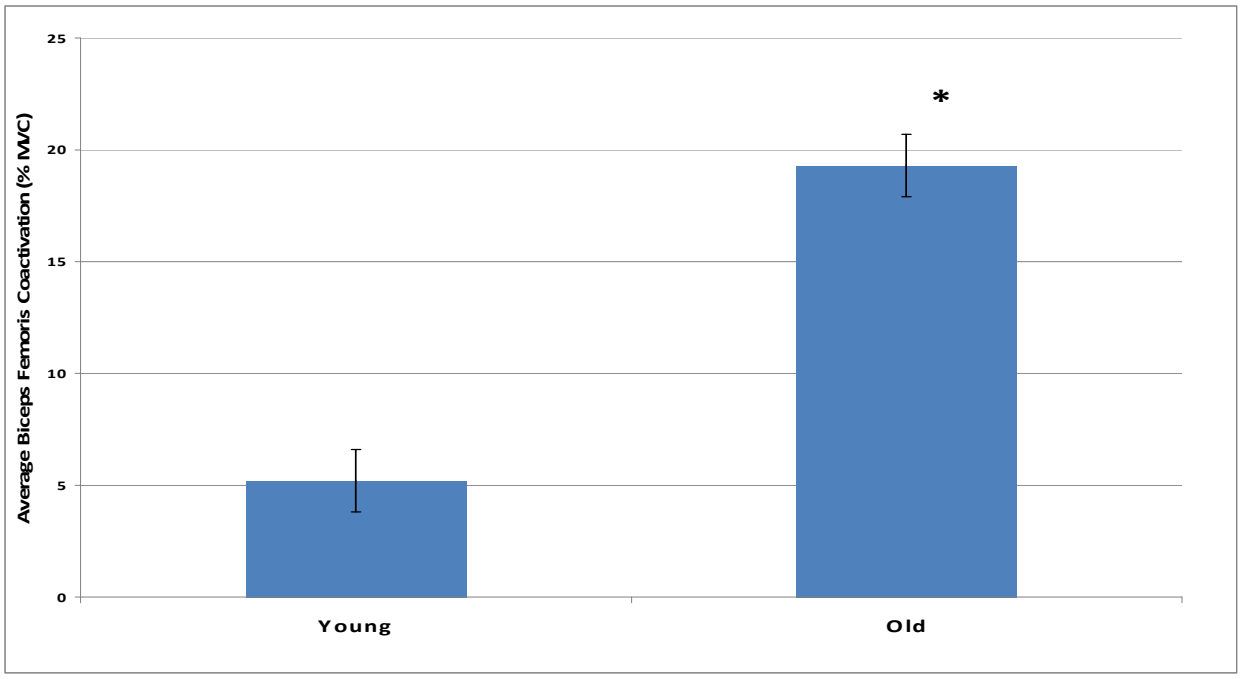

Figure 33. Age main effect of biceps femoris antagonist muscle coactivation during incline and decline walking. Old adults had significantly greater BF antagonist muscle coactivation than young individuals. * denotes an age main effect

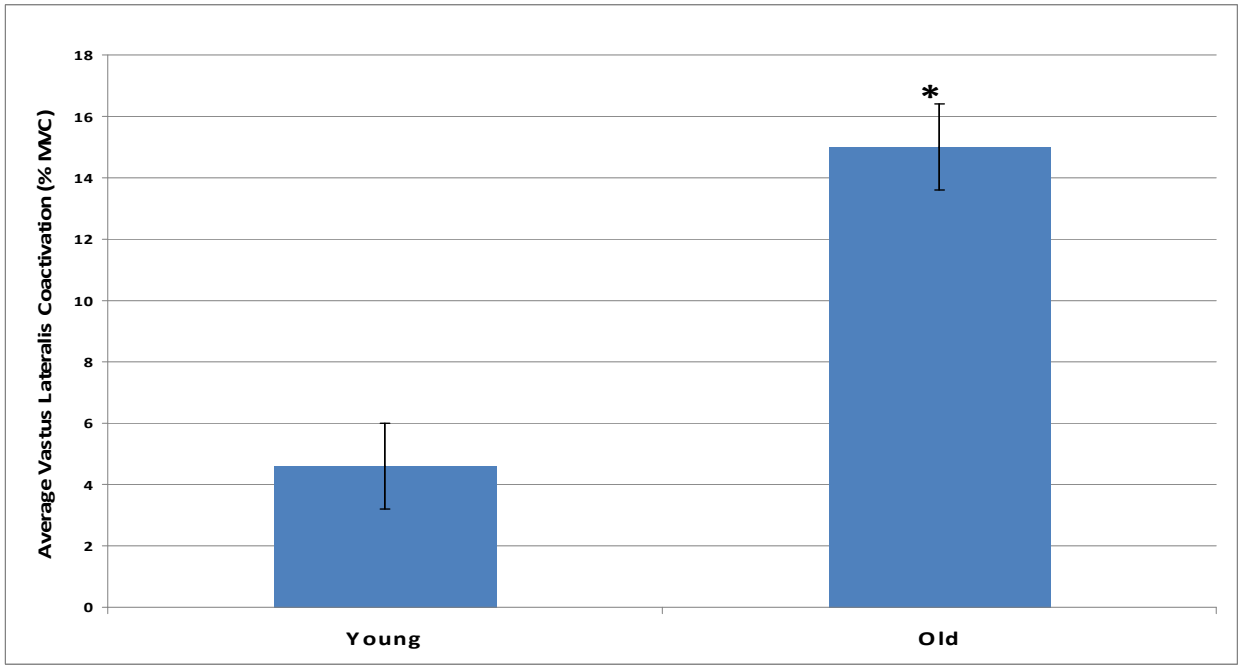

Figure 34. Age main effect of vastus lateralis antagonist muscle coactivation during incline and decline walking. Old adults had significantly greater VL antagonist muscle coactivation than young individuals. * denotes an age main effect

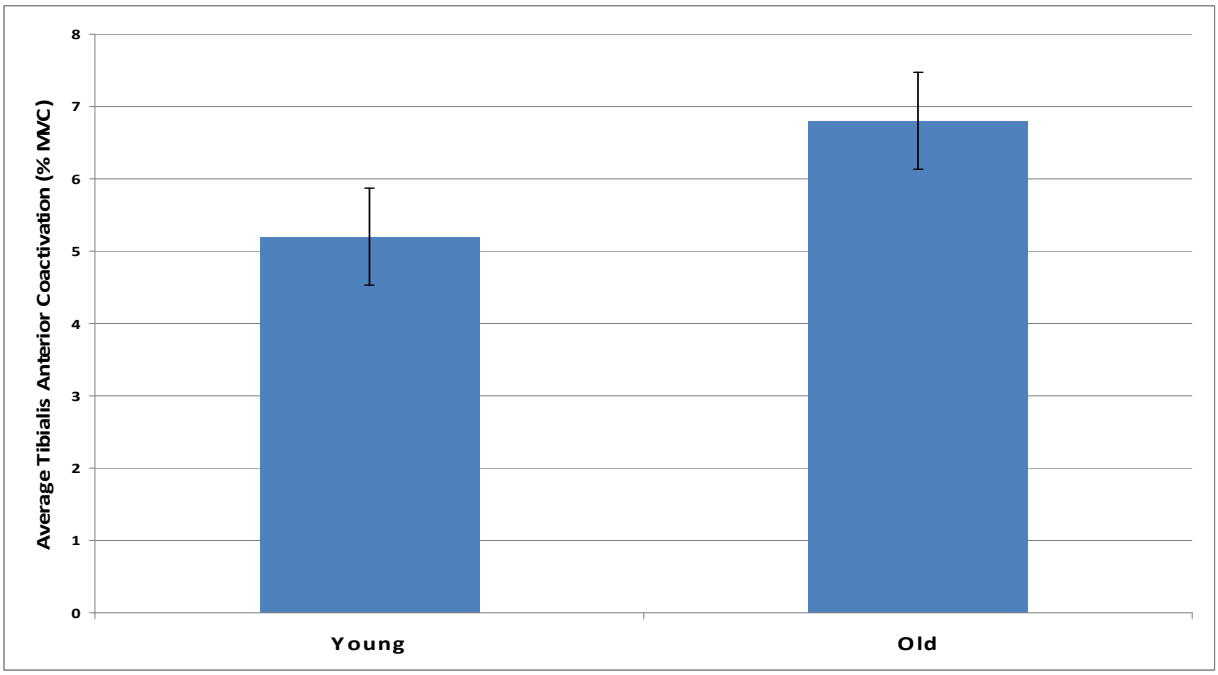

Figure 35. Age main effect of tibialis anterior antagonist muscle coactivation during incline and decline walking. There was no significant age effect present.

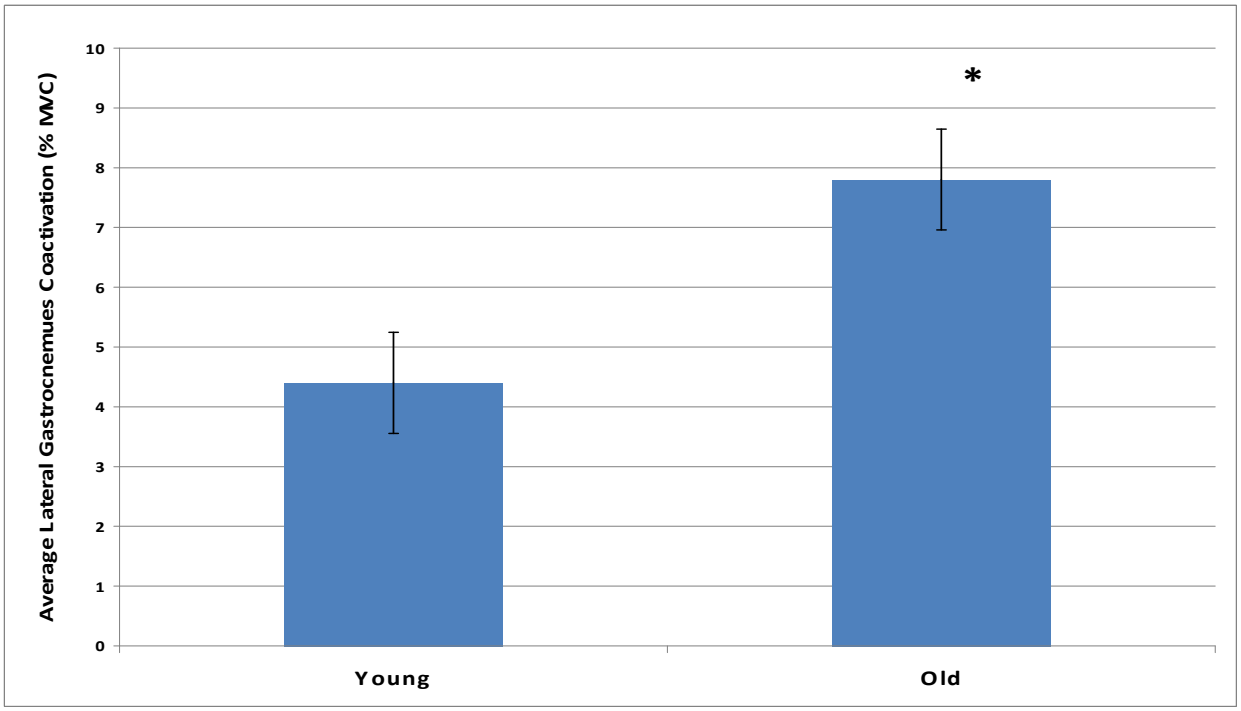

Figure 36. Age main effect of lateral gastrocnemeus antagonist muscle coactivation during incline and decline walking. Old adults had significantly greater LG antagonist muscle coactivation than young individuals. * denotes an age main effect

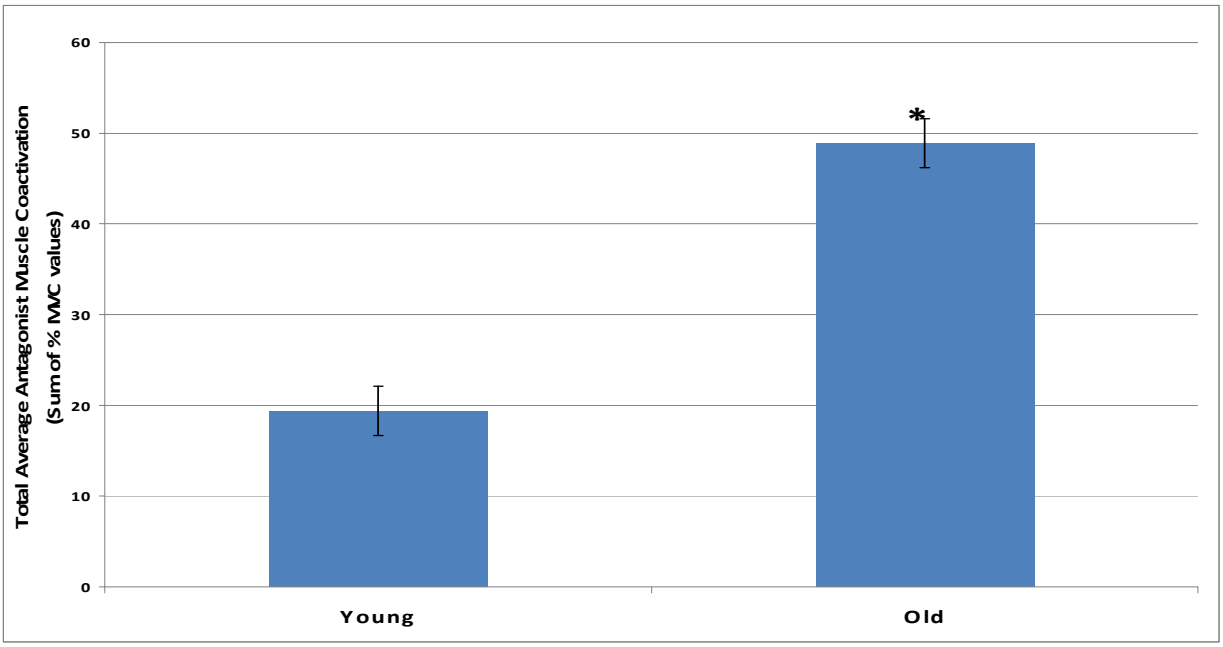

Figure 37. Age main effect of total antagonist muscle coactivation during incline and decline walking. The antagonist muscle coactivation of the BF, VL, TA, and LG normalized to their MVC's were added together to determine the total coactivation. Old adults had a significantly greater total antagonist muscle coactivation than young individuals. * denotes an age main effect

Figure 38 is a summary figure which shows the percent difference between old and young antagonist muscle coactivation in each testing condition. Old individuals had greater levels of antagonist muscle coactivation in all muscles in each condition. These values are also shown in Table 7.

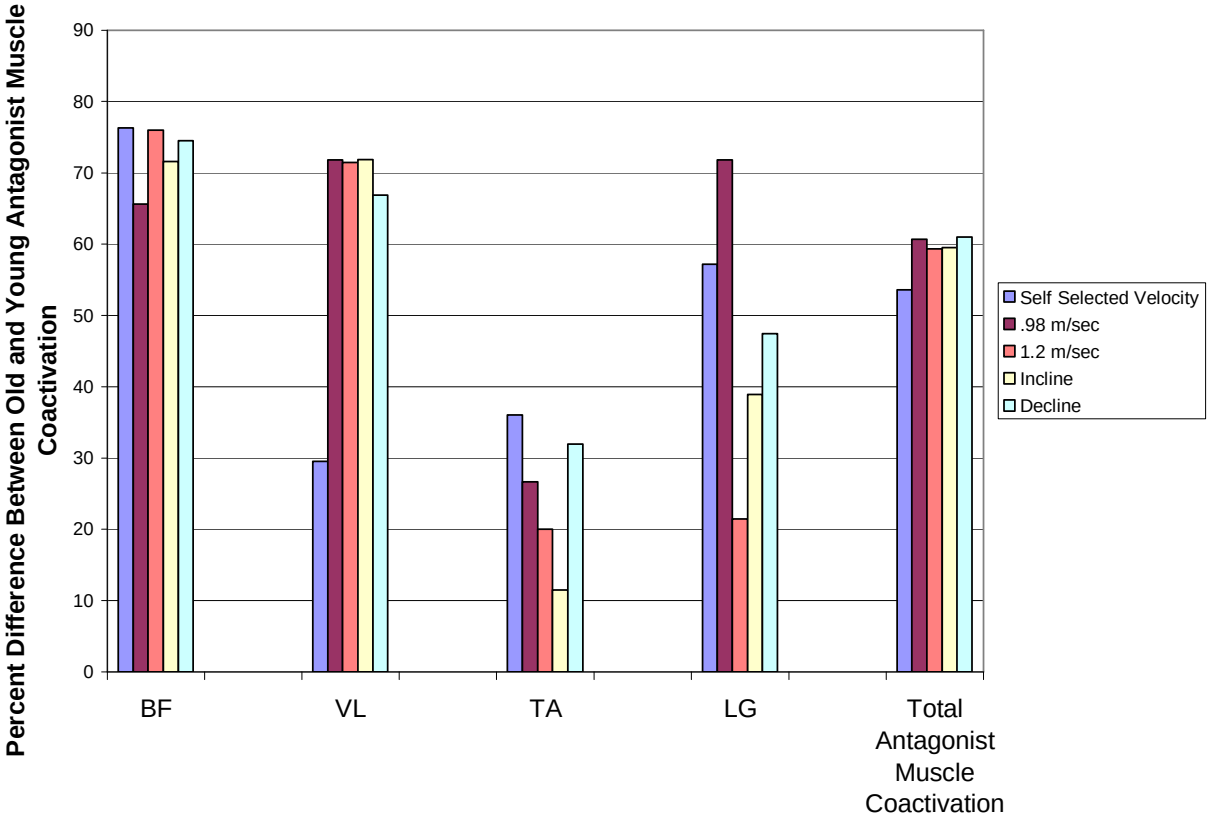

Figure 38. Percent difference between young and old antagonist muscle coactivation. Old had greater antagonist muscle coactivation of all muscles in all conditions.

The second sub-hypothesis was that there would be a velocity main effect of antagonist muscle coactivation. The VL had significantly greater antagonist muscle coactivation when subjects walked at a self selected speed (an average of 1.0 m/sec) compared to walking at .98 m/sec ($F = 6.626$ $p = 0.050$) and walking at 1.2 m/sec ($F = 6.626$, $p < 0.010$). This is shown in Figure 39. Figures 62-65 in the Appendix show the velocity effect of antagonist muscle coactivation of the BF, TA, LG, and total muscle coactivation respectively. Table 4 also shows these values. There was no significant relationship between increasing walking velocity leading to increased antagonist muscle coactivation in these muscles.

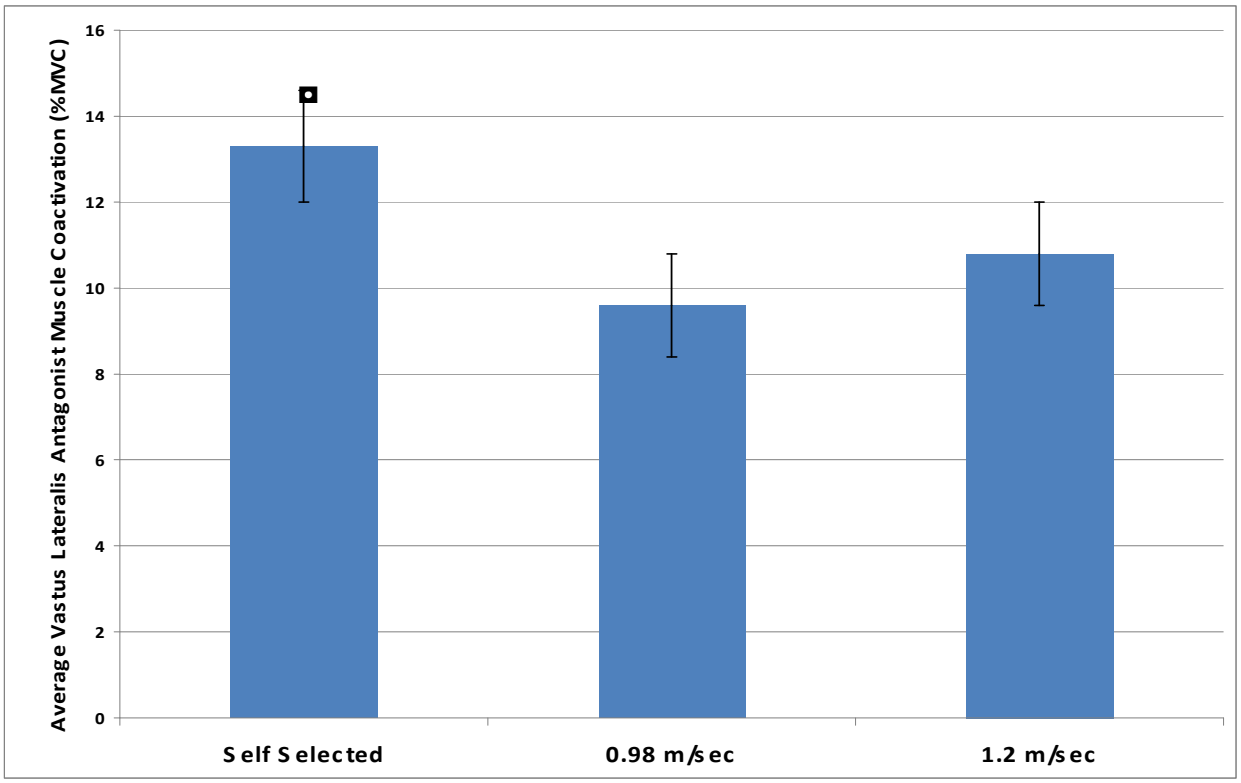

Figure 39. Velocity main effect of vastus lateralis antagonist muscle coactivation in old and young adults. Walking at a self selected velocity caused greater coactivation of the VL than walking at .98 m/sec and walking at 1.2 m/sec. ◘ represents a velocity main effect.

The third sub-hypothesis was that walking at a decline would cause greater muscle coactivation than walking at an incline. Walking at a decline elicited significantly greater antagonist muscle coactivation of the VL ($F = 6.336$, $p = 0.020$). This is shown in Figure 41. During incline walking the BF had greater coactivation ($F = 14.697$, $p = 0.001$). Additionally the total amount of antagonist muscle coactivation was greater in incline walking ($F = 6.450$, $p = 0.019$). These data are shown in Figures 40 and 42 respectively. The task effect of antagonist muscle coactivation of the TA and LG are shown in Figures 66 and 67 in the Appendix. Table 5 also shows the values of antagonist muscle coactivation during incline and decline walking.

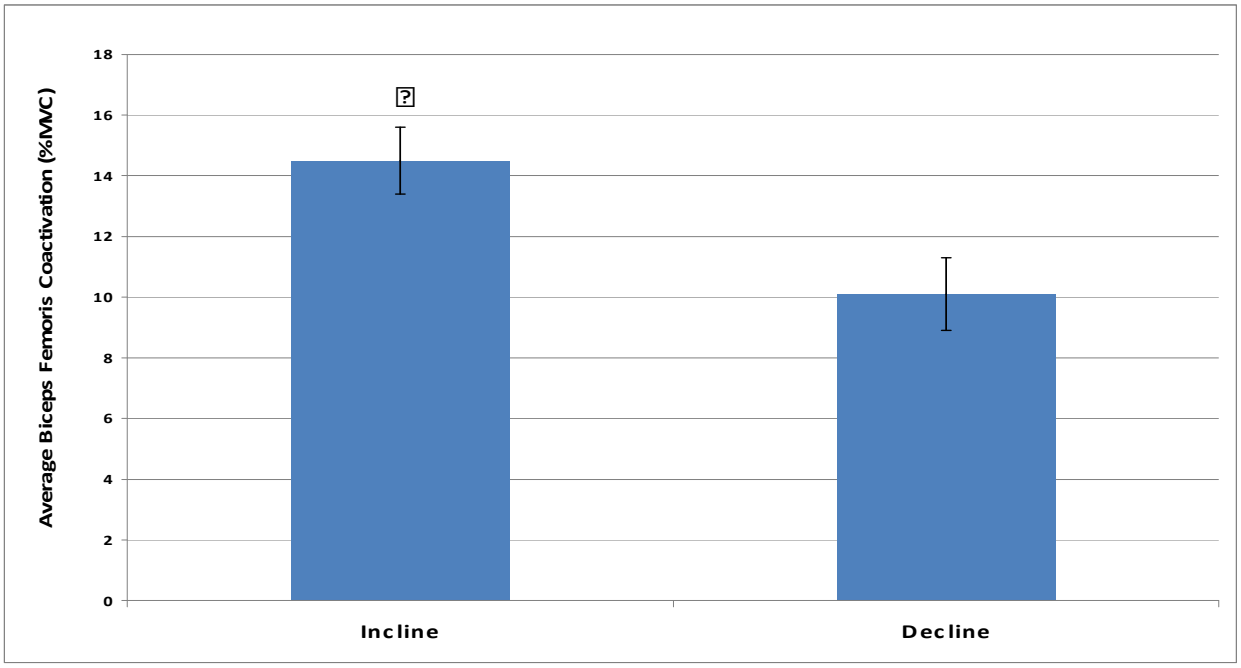

Figure 40. Task main effect of biceps femoris antagonist muscle coactivation in young and old adults. Incline walking caused significantly greater BF antagonist muscle coactivation. ◘ denotes a significant task main effect.

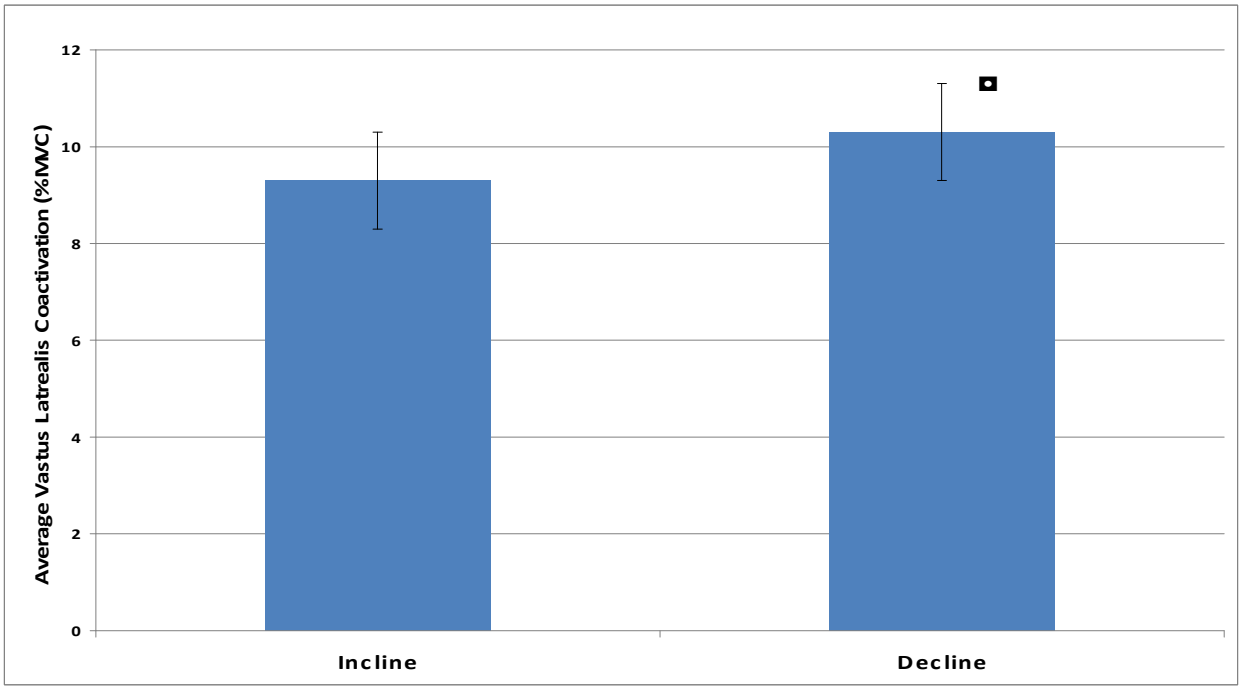

Figure 41. Task main effect of vastus lateralis antagonist muscle coactivation in old and young adults. Decline walking caused significantly greater VL antagonist muscle coactivation. ◘ denotes a significant task main effect.

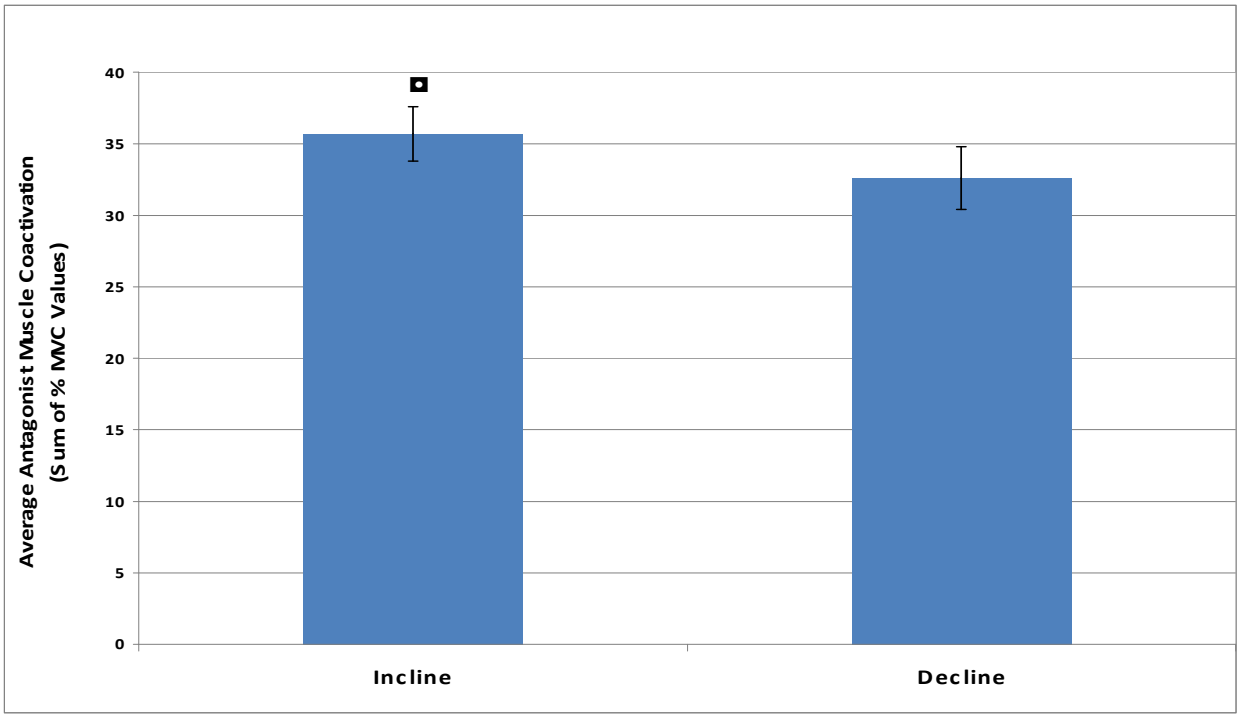

Figure 42. Velocity main effect of total antagonist muscle coactivation in old and young adults. Walking at an incline caused significantly greater total antagonist muscle coactivation. ◘ denotes a significant task main effect.

Figures 43 and 44 show the significant age by gait velocity interaction effects of antagonist muscles. Figure 43 shows that there was an interaction effect of the VL. Old individuals had significantly greater VL antagonist muscle coactivation than young at self selected velocity ($F = 7.075$, $p = 0.050$), .98 m/sec ($F = 7.075$, $p < 0.001$), and 1.2 m/sec ($F = 7.075$, $p < 0.001$). Young subjects had significantly greater coactivation of their VL during the self selected velocity compared to walking at .98 m/sec ($F = 7.075$, $p < 0.001$) and compared to walking at 1.2 m/sec ($F = 7.075$, $p < 0.001$). Figure 44 shows that there was also a significant interaction effect which occurred in the LG while walking. Old individuals had significantly greater LG antagonist muscle coactivation than the young in the self selected condition ($F = 4.892$, $p = 0.050$) and .98 m/sec ($F = 4.892$, $p < .001$). Additionally old subjects had

significantly greater coactivation walking at .98 m/sec compared to walking at 1.2 m/sec (F = 4.892, p = 0.050). Figures 68-70 in the Appendix show the coactivation values of the BF, TA, and total antagonist muscle respectively, which did not exhibit any interaction effects.

During incline and decline walking there were no task by age interaction effects. The antagonist muscle coactivation graphs are shown in Figures 71 through 75 in the Appendix.

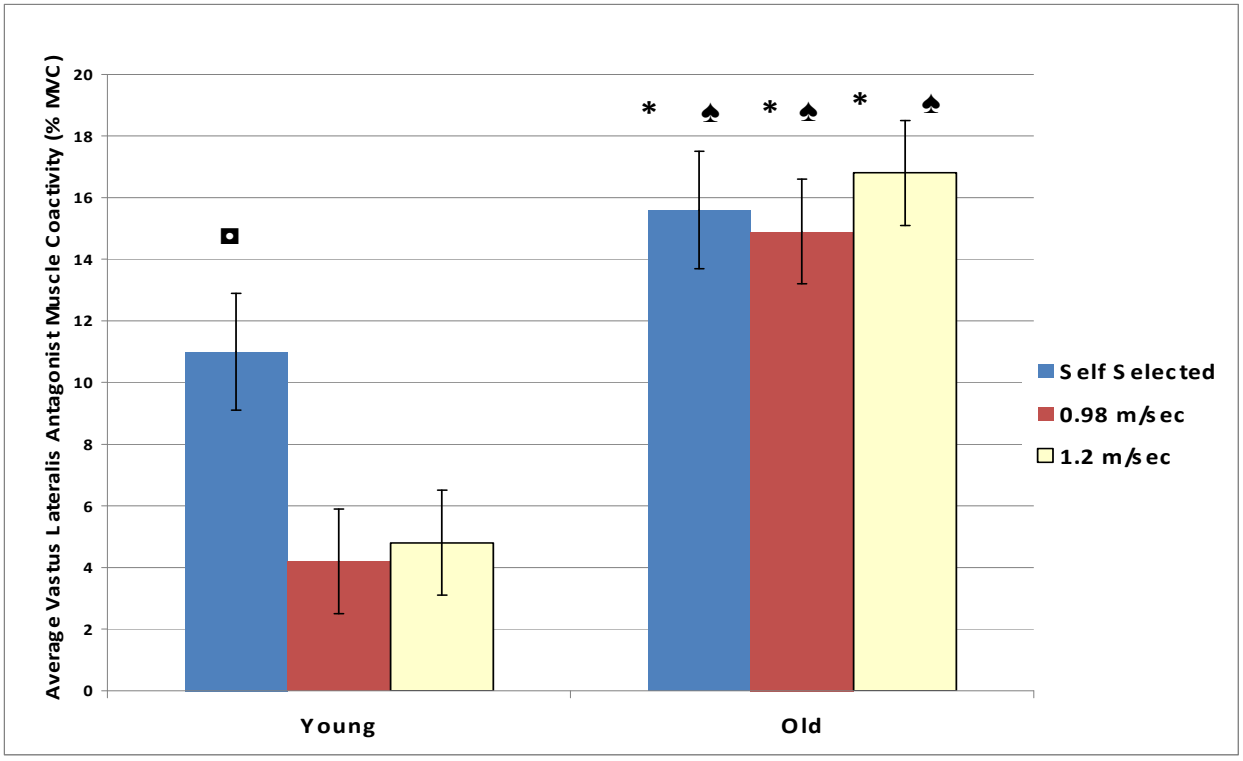

Figure 43. Age by velocity interaction effect of vastus lateralis antagonist muscle coactivation separated into velocity and age. Old walking at a self selected velocity had significantly greater VL coactivation than young individuals walking at self selected velocities, .98 m/sec, and 1.2 m/sec. Old walking at .98 m/sec had significantly greater VL coactivation than young walking at .98 m/sec and 1.2 m/sec. Old walking at 1.2 m/sec had significantly greater VL coactivation than young walking at self selected velocities, .98 m/sec, and 1.2 m/sec. Young walking at self selected velocities had significantly greater antagonist muscle coactivation than young walking at .98 m/sec and 1.2 m/sec. * denotes a difference between young and old walking at the same velocity. ◘ denotes a difference between velocities within either the young or old group. ♠ denotes a difference between a different age group and velocity.

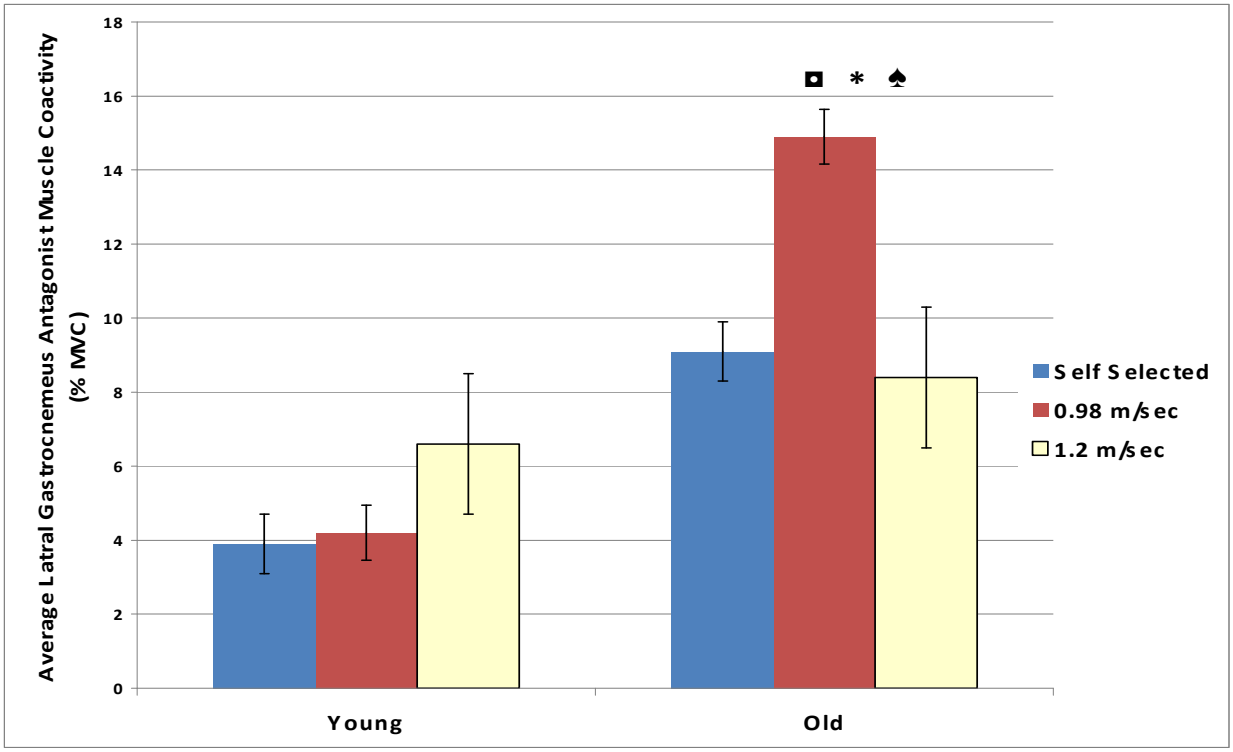

Figure 44. Age by velocity interaction effect of lateral gastrocnemeus antagonist muscle coactivation separated into velocity and age. Old walking at a velocity of .98 m/sec had significantly greater LG coactivation than old walking at 1.2 m/sec. Additionally, old walking at .98 m/sec had significantly greater LG coactivation than young walking at .98 m/sec. Finally old walking at .98 m/sec had significantly greater VL coactivation than young walking at self selected velocities and 1.2 m/sec. ◘ denotes a difference between velocities within either the young or old group. ♠ denotes a difference between a different age group and velocity.

Association between Antagonist Muscle Coactivation and Metabolic Cost

The global hypothesis of this study was that the age related increase in metabolic cost would be due to increased antagonist muscle coactivation. Figure 45 shows that this occurred in the self selected walking speed condition. Old adults tended to have both greater antagonist muscle coactivation and greater metabolic cost than young adults. Additionally a correlation of $r^2 = .2$ could be made between metabolic power and antagonist muscle coactivation showing that that there was a moderate but significant association between the variables. This pattern was

also evident in walking at .98 m/sec r^2 =.36, and walking at 1.2 m/sec r^2 =.42, and is shown in Figures 46 and 47 respectively.

Figure 48 shows that during incline walking old adults exhibited greater amounts of antagonist muscle coactivation; however they did not seem to exhibit greater gross metabolic power. Therefore, during incline walking a strong correlation between total antagonist muscle coactivation and gross metabolic power couldn't be established r^2 =.06.

The relationship between gross metabolic power and total antagonist muscle coactivation during decline walking are shown in Figure 49. This relationship is represented by an r^2 value of .42, showing a high correlation between the two variables.

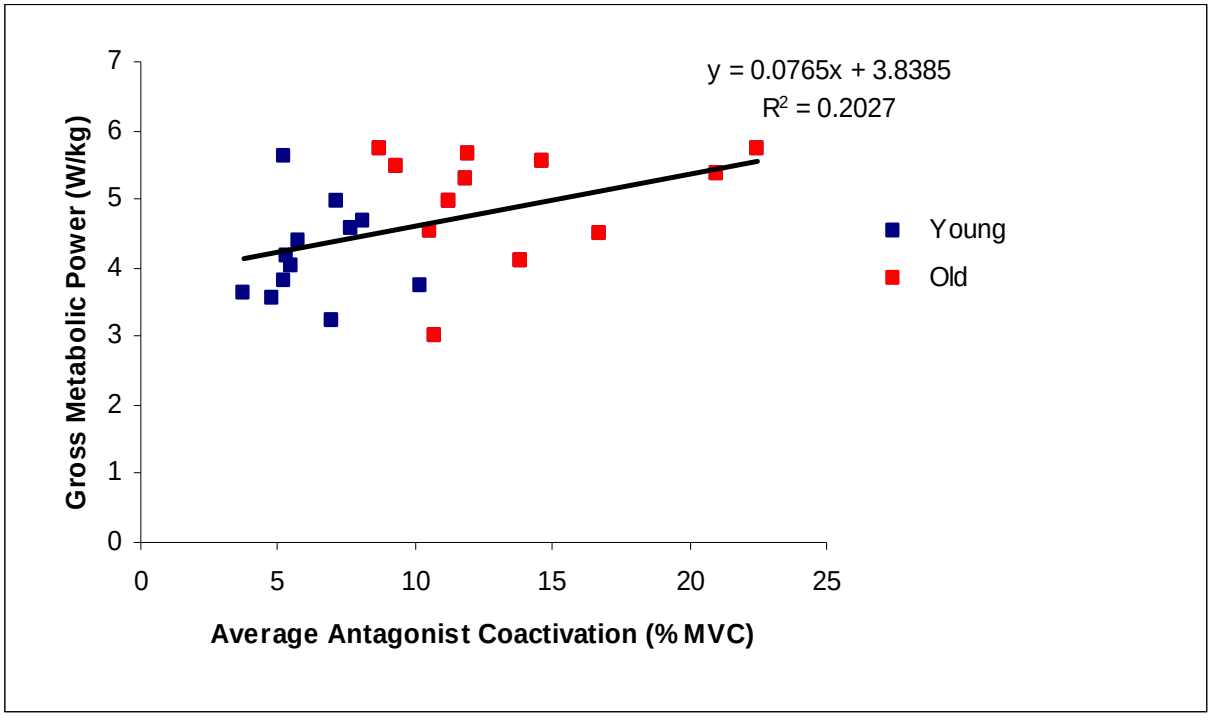

Figure 45. The association of average antagonist muscle coactivation and gross metabolic power in level walking at a self-selected velocity.

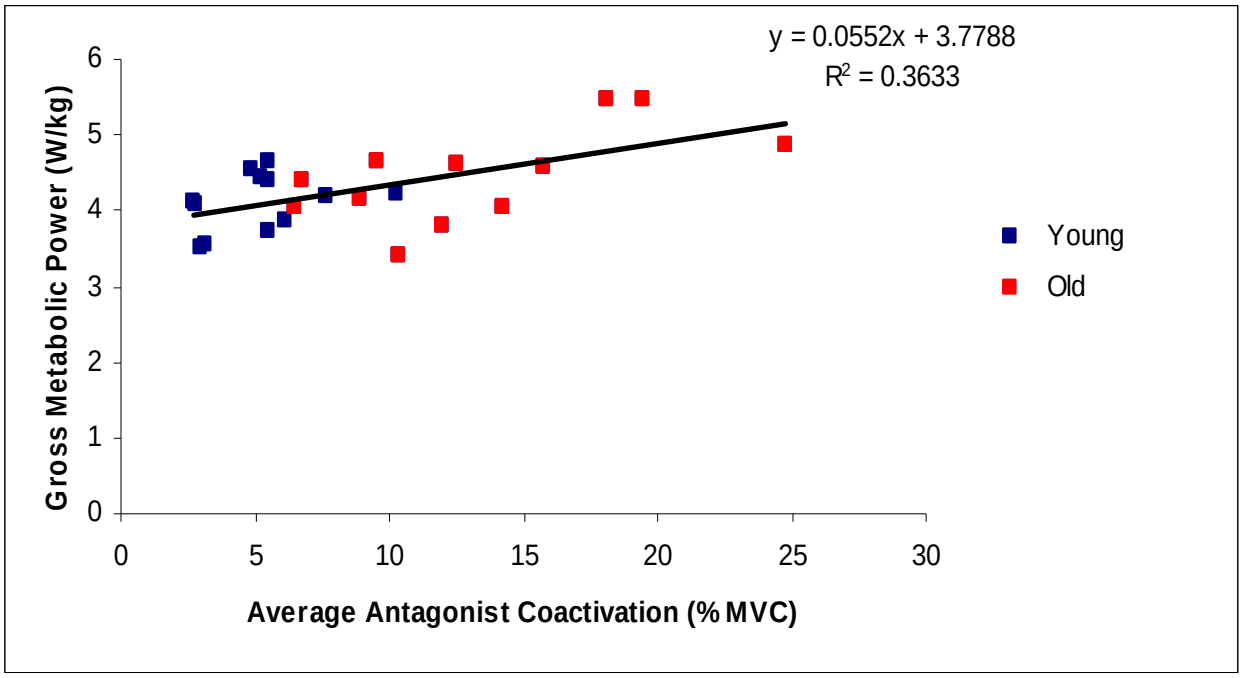

Figure 46. The association between average antagonist muscle coactivation and gross metabolic power in level walking at .98 m/sec. Old individuals again seemed to have greater antagonist muscle coactivation and metabolic power. An r^2 value of .36 was computed showing that there is a pattern between the two variables.

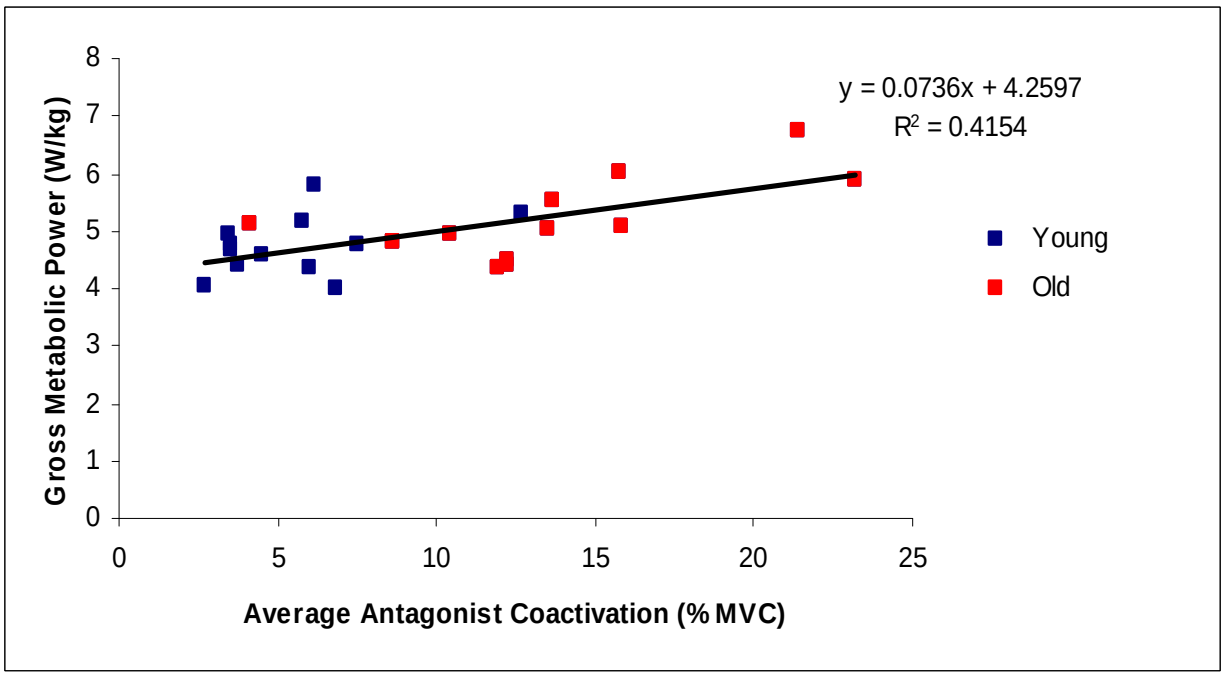

Figure 47. The association between average antagonist muscle coactivation and gross metabolic power in level walking at 1.2 m/sec.

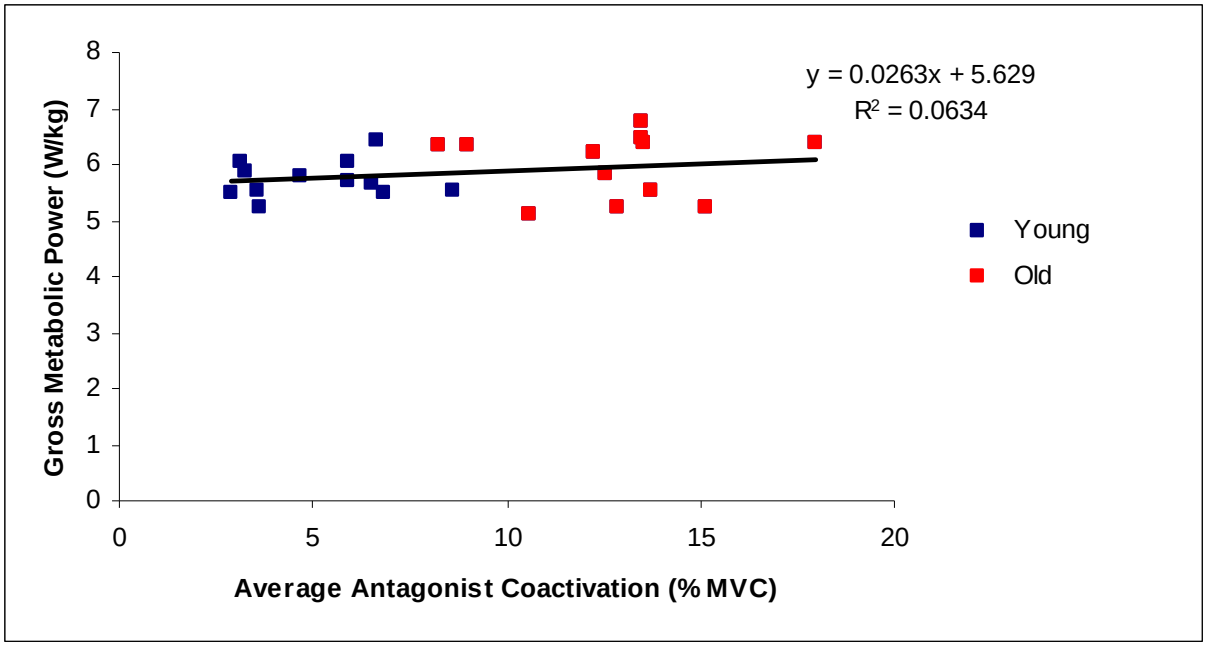

Figure 48. The association between average antagonist muscle coactivation and gross metabolic power in incline walking at a 6% grade at .98 m/sec. There was not a strong correlation between the levels of antagonist muscle coactivation and gross metabolic power during this condition.

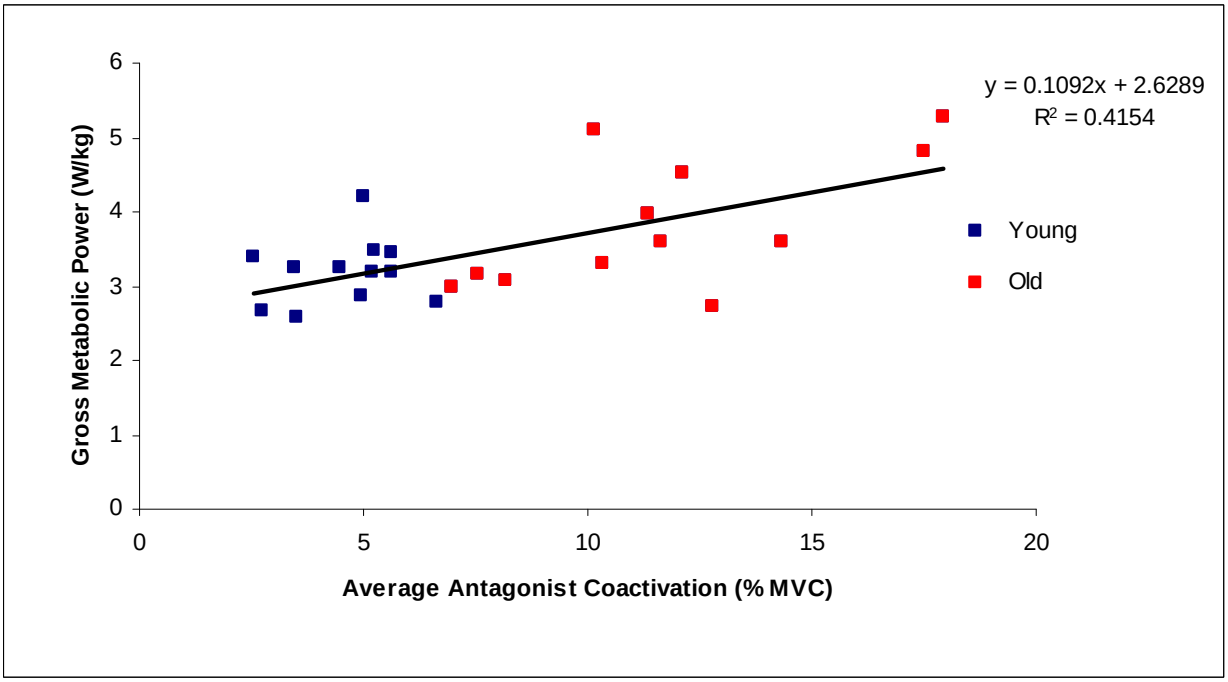

Figure 49. The association between average antagonist muscle coactivation and gross metabolic power in decline walking at a 6% grade at .98 m/sec. During decline walking old adults seemed to experience greater antagonist muscle coactivation and greater metabolic power.

Summary

Statistical analyses have revealed that there are differences between ages with regard to both metabolic energy cost and antagonist muscle coactivation. The current study displayed that old individuals used greater amounts of metabolic energy, and greater amounts of antagonist muscle coactivation during walking in all conditions tested.

Velocity and task main effects also occurred. Velocity main effects were evident in nearly all the metabolic variables tested, and also occurred in VL antagonist muscle coactivation. Task main effects occurred for nearly all metabolic variables tested, and also occurred in the BF, VL, and total antagonist muscle coactivation.

An age by velocity interaction was found in the VL and LG antagonist muscle coactivation levels. While an age by task interaction existed in the RPE, and RER.

Additionally relationships existed between the levels of antagonist muscle coactivation and metabolic cost in level walking at a self selected velocity, level walking at .98 m/sec, level walking at 1.2 m/sec, and decline walking at a 6% at .98 m/sec. During these conditions old adults also appeared to have greater antagonist muscle coactivation and greater gross metabolic power than young individuals, thus supporting our global hypothesis.

CHAPTER V: DISCUSSION

The purpose of this study was to compare the cost of locomotion and muscle activity in antagonist muscles in old and young adults during gait. Additionally it was to determine if there was a relationship between the metabolic cost of gait and the amount of antagonist muscle coactivation that people experience. Previous studies have determined that old individuals experience higher amounts of both metabolic cost and antagonist muscle coactivation; however the two variables have not yet been linked.

This chapter is divided into the following sections: 1) Development of the Hypothesis, 2) The Effects of Aging on Metabolic Cost 3) Neurological Changes which Elicit Increased Antagonist Muscle Coactivation, 4) The Velocity and Task Effect on Antagonist Muscle Coactivation, 5) Age Related Increased Metabolic Cost is Mediated by Increased Antagonist Muscle Coactivation, 6) Conclusions

Development of the Hypothesis

Physiological aging causes ample changes in both metabolic and neuromuscular systems. These alterations result in increases of both cost of transport during locomotion and antagonist muscle coactivation as hypothesized by sub hypothesis one, which stated that old individuals would experience greater levels of metabolic cost of gait and antagonist muscle coactivation. Previous research has always looked at these two variables as separate entities. However according to the global hypothesis of this study, metabolic cost of gait, and antagonist muscle coactivation appear linked and dependent upon each other.

The Effects of Aging on Metabolic Cost

According to the results in this study, older adults experienced greater metabolic cost than young adults in all walking conditions. This age main effect supports the first sub

hypothesis of this study and also matches the results of nearly all previous research regarding aging to date.

The majority of previous research has linked age related increase in metabolic cost to decreased mitochondrial function. As mitochondria work to provide energy to cells they produce some byproducts in addition to ATP and other metabolic compounds. These byproducts are reactive oxygen species and are most likely created within the electron transport system (Sanz, Stefanastos et al. 2010). Specifically this occurs when reverse electron transport occurs (Schofeld and Wojtczak 2007) or electrons leak out of the chain. These mitochondrial reactive oxygen species not only cause aging related changes but have also been linked to several age-related diseases such as Parkinson's, cancer, and diabetes. An increase in these free radicals has been shown to reduce life expectancy in old individuals (Sanz, Pamplona et al. 2006).

Mitochondrial reactive oxygen species cause mutations to occur within mitochondrial DNA (Harman 2003). It is still unclear how exactly they cause these mutations, however it has been determined that the reactive oxygen species cause mtDNA to fracture and break apart thereby contributing to their degradation and mutations (Shokolenko, Venediktova et al. 2009). Overtime, if the mitochondria have undergone mutations they tend to cause dysfunction in the electron transport system thereby yielding fewer ATP molecules (Dirks, Hofer et al. 2006). Since older people have experienced greater levels of mitochondrial reactive oxygen species they will have more mutated mitochondria. If their mitochondria are functioning below their optimal level old individuals must increase their metabolic cost in order to perform tasks that otherwise would have required less metabolic energy.

Even though the mitochondrial dysfunction theory is related to age mediated increases in metabolic cost it does not necessarily explain why old adults in this particular study had greater

metabolic cost. Although older adults do experience mitochondrial dysfunction it occurs mostly in type II muscle fibers, while type I muscle fibers actually become more efficient as people age (Deschenes 2004). Although each muscle is different large motor units and fast twitch muscle fibers are not normally activated until at least 60% of max strength (Ives 2005). As shown in Table 8 the old individuals in this study were activating their muscles at levels much lower than 60% of their max. Typical values for old normalized muscle strength ranged between 10% and 30% of their MVCs. Therefore, the old subjects were clearly utilizing type I muscle fibers while walking which means the mitochondria they were using were highly functional. This forces a new conclusion to be made as to why the older individuals experienced greater metabolic cost.

The increased metabolic cost that the older individuals experienced in this study was most likely caused by higher raw agonist muscle activation, and higher raw antagonist muscle activation in old subjects. Additionally old subjects experienced greater normalized agonist and antagonist muscle activation. When individuals activate their muscles at higher levels they are recruiting more motor units, recruiting larger motor units, or increasing the motor units' firing rate (Ives 2005). As previously mentioned, the old adults in this experiment were most likely not using type II muscle fibers, therefore they must have been recruiting more small motor units than the young, or they simply had higher motor unit firing rate than the young. According to Layec et al., if individuals recruit more or larger motor units they increase their oxidative cost (Layec, Bringard et al. 2009). Therefore when older individuals have higher muscle activation they will in turn have a higher oxidative cost, thereby possibly explaining their greater cost of locomotion seen in this study.

Neurological Changes which Elicit Increased Antagonist Muscle Coactivation

Old adults experienced significantly greater antagonist muscle coactivation than young adults in nearly all muscles in every walking condition examined. This phenomenon may be explained by several different possible mechanisms occurring at both the spinal and supraspinal regions.

As people age they experience decreased reciprocal inhibition. Reciprocal inhibition is controlled at the spinal level through Ia inhibitory motor neurons. Kido et al. found that in both the tibialis anterior and the soleus in older individuals had decreased reciprocal inhibition (Kido, Tanaka et al. 2004). When someone has decreased reciprocal inhibition their interneuron actually has a higher resting potential. In other words, the interneuron and consequentially motor neuron and muscle fibers that it innervates will require less electrical activity to become stimulated, it is closer to motor threshold. Therefore, it is easier for old individuals to cause an action potential to fire and stimulate an antagonist muscle while the agonist is firing, thus increasing the likelihood of antagonist muscle coactivation.

Another explanation of increased antagonist muscle coactivation is that old individuals experience cortical changes that reduce the suppression of brain areas that control antagonist muscle movement. By reducing suppression of these brain areas antagonist muscles are more likely to fire in old individuals. In fact these muscles are likely to begin firing even before movement is necessary. Hortobagyi and DeVita found that in a downward stepping task, old individuals not only had greater levels of both agonist and antagonist muscle activity, but they also had greater antagonist muscle activity before the task even started (Hortobagyi and DeVita 2000). The action potentials in antagonist muscles that old people experience prior to beginning a task most likely come from the brain as a way to prepare the body for what is about to

experience. Although the current study didn't look at the duration and timing of the antagonist muscle contraction it is safe to assume that it was longer in old than young subjects as a way to prepare old individuals for the task at hand.

Another supra-spinal change that old individuals experience involves the reorganization of the brain due to age related neurodegredation. As people age, they experience changes in their dendrites and brain synapse (Levenez, Garland et al. 2008). The reorganization of the brain causes more areas of the brain to be active in old people than in young people during a simple task (Capaday, Devanne et al. 1998). Since more areas of the brain are active in old individuals it is possible that they are unintentionally recruiting areas that are not necessary for the task at hand. The cortical connections in older brains could be causing antagonist muscles to activate, when really they only need their agonist muscles to fire. Hutchinson, Kobayashi, et al. found that old individuals used significantly more of their brain than young individuals for simple tasks such as moving their index finger (Hutchinson, Kobayashi et al. 2002). The increased brain activity old individuals experienced will result in greater muscle activation all around, including antagonist muscle activation.

These neurological and physiological changes that occur as people age are inventible. Even though they weren't evaluated in this particular study we can assume that they were the reasons that old subjects experienced greater levels of antagonist muscle coactivation than young subjects.

Velocity and Task Effect on Antagonist Muscle Coactivation

The second and third sub hypotheses were that faster walking velocities would cause greater antagonist muscle coactivation and that decline walking would cause greater muscle coactivation than incline walking.

The only significant velocity main effect that was present in this particular study was found in the vastus lateralis during self selected walking speed. This value was significantly lower than vastus lateralis coactivation when walking at both .98 and 1.2 m/sec. Since the average self selected walking speed was 1.0 m/sec it was assumed that antagonist muscle coactivation of the .98 m/sec condition would have been nearly the same as the self-selected speed, and if anything else slightly lower than the self selected condition. This however was not the case.

Hortobagyi et al. previously found that as walking velocity increased antagonist muscle coactivation also increased (Hortobagyi, Solnik et al. 2009). Although it wasn't significant, the current study found that when walking at 1.2 m/sec subjects experienced greater VL, TA, and total coactivation than when walking at .98 m/sec. One possible reason that these values weren't significant could be that neither speed took subject's out of their comfort zone of walking. In other words, the walking tasks could have been too similar or simple to elicit major velocity main effects.

Although the values weren't significant, walking at a self selected velocity elicited the greater amounts of antagonist muscle coactivation in the VL, TA, and total muscle than both the .98 m/sec and 1.2 m/sec conditions. Most previous research has shown that while walking at a self selected velocity the body naturally picks a pace that elicits the least amount of work (Jones, Waters et al. 2009). In other words the body chooses the path of least resistance. This was not the case in the present study.

One possible explanation for this phenomenon has to do with testing protocol. The first walking condition that subjects always participated in was walking at a self-selected speed. Besides the resting measurement, this was the only condition which was not randomized. Person

once showed that antagonist muscle coactivation is higher in tasks that are unfamiliar to people, then decreases as individuals become more familiar with the task they are performing their antagonist muscle coactivation decreases (Person and Roshchina 1958). It is quite possible that when the subject's first started to walk on the treadmill they weren't given enough time to acclimate to it and were still in the learning phase of treadmill walking during the self selected condition.

The third sub-hypothesis was that there would be a condition effect in which walking at a decline would cause greater antagonist muscle coactivation than walking at an incline. This was assumed because one of the purposes of antagonist muscle coactivation is to provide support and stabilization to joints (Hortobagyi and DeVita 2000). Additionally during decline walking the downward lowering motion that occurs requires greater stabilization in order to promote joint longevity and health (Granata, Lee et al. 2005).

The results of this study showed that decline walking caused significantly greater VL coactivation than incline walking. It also showed that walking at an incline actually produced greater antagonist muscle coactivation than walking at a decline in the BF and in total antagonist muscle coactivation. This may have occurred for several different reasons. Some scientists have looked at individuals with previous knee injuries and determined that during inclined treadmill walking their hamstring muscles activate significantly earlier. It is speculated that this is a method to increase knee stability during walking (Kalund, Sinkjaer et al. 1990). Subjects within this study, specifically those who had never walked on a treadmill before could have been activating their BF more in order to increase their stability in this new task.

Another possible reason for this refuted sub-hypothesis could have been that the subjects were walking on the treadmill itself. Walking on the treadmill could have elicited different

muscle pattern than what would have otherwise been seen in overground walking. Lee and Hidler found that when walking on a treadmill adults experienced greater muscle activation of the hamstring during the swing phase of gait. This could also explain why subjects had greater BF coactivation during incline walking (Lee and Hidler 2008).

A final reason for increased total coactivation during the incline condition could have to do with the intensity level at which the subjects were walking. As shown in Figure 21, subjects were working at a significantly higher workload during the incline condition. Since the intensity was greater during the incline condition it could have forced subjects to have greater antagonist muscle coactivation compared to walking at a decline when the workload was significantly less. Previous research has shown that greater levels of coactivation exist within motions that are more intense (Caty, Aujouannet et al. 2007). Additionally research has shown that as individuals fatigue antagonist muscle coactivation also increases (Psek and Cafarelli 1993). Since the subjects within the present study were working at a higher intensity during the incline condition they are more likely to demonstrate fatigue in this condition, thereby, leading to their increased antagonist muscle coactivation.

Age Related Increased Metabolic Cost is Mediated by Increased Antagonist Muscle Coactivation

As previously mentioned there was a significant age effect in both metabolic cost and antagonist muscle coactivation. Old individuals experienced greater levels of both gross metabolic power and antagonist muscle coactivation. Regression lines were able to be placed in Figures 45 through 49. It appears as though in all conditions except the incline condition there is a relationship between the two variables.

The aging process causes inevitable alterations in the body's neurological, physiological, and anatomical systems. One set of changes already discussed involves increased levels of antagonist muscle activity. As individuals age they experience alterations in their cortical and sub cortical nervous system. These changes include decreased reciprocal inhibition, decreased cortical inhibition, a reconfiguration of the brain, and increased brain activity. All these variables lead to age related increases in antagonist muscle coactivation. They will increase the likelihood of the antagonist muscle firing.

When antagonist muscles fire they elicit opposing force against the agonist muscles. This opposing force causes the agonist muscles to have to contract stronger in order to overcome the power being produced by the antagonist. Therefore, not only is extra effort put into making the antagonist muscle contract, but extra exertion is also used to make the agonist overcome the antagonist muscle. All this extra effort costs the body metabolically. When greater levels of muscle mass are recruited for movement greater amounts of metabolic energy are needed fuel the bodies ATP demands (Truijens, Noordermeer et al. 2005).

This age related demand in metabolic energy explains why old individuals constantly use more energy to complete everyday tasks and activities of daily living (ADL). Old individuals execute tasks at a greater level relative to their maximum amount of effort (Hortobagyi, Mizelle et al. 2003). This is caused by their inefficient movements using relatively high levels of antagonist muscle coactivation.

This pattern was evident in walking at a self selected speed, walking at .98 m/sec, walking at 1.2 m/sec, and walking at a decline. It was not very apparent in the incline walking condition. As shown in Figure 48, old adults still experienced greater levels of antagonist

muscle coactivation; however they didn't experience an increase in metabolic power of the same magnitude.

The old subjects used for this study were incredibly healthy and highly active for their age. Ten out of the twelve older subjects exercised for at least an hour everyday. Oftentimes the old subjects were more active then the young subjects that were tested. Five out of the twelve young subject's tested exercised at least an hour a day. If normal, less active old subjects or more active young subjects were used, more drastic results would have been seen, and a stronger correlation between antagonist muscle coactivation and gross metabolic power would have been evident. Training status also influences the level of antagonist muscle coactivation present in individuals. Both old and young subjects who were involved in exercise programs experienced lower amounts of antagonist muscle coactivation after several weeks of training (LaRoche, Roy et al. 2008).

The findings of the present study are incredibly important for the medical and fitness worlds. Now that we've shown that the age related increase in antagonist muscle coactivation causes or at least adds to the increased metabolic cost old people experience physicians, researchers, and other health professionals can prescribe exercises and fitness routines that will reduce antagonist muscle coactivation. This will in turn reduce metabolic energy demands and allow old individuals to complete ADLs at energy levels lower to their maximum capacity. Old adults may be able to increase their overall level of function.

Conclusions

Age was found to affect antagonist muscle coactivation levels in all muscles evaluated in both different tasks and at different velocities during gait. Additionally age affected the amount of metabolic energy used in all tasks and velocities during gait. Old adults experienced

significantly greater amounts of antagonist muscle coactivation and metabolic cost than young adults. After using a regression analysis it was observed that the two variables were related. The age related increases in antagonist muscle coactivation seem to contribute to the age related increases in metabolic cost. This suggests that the neural changes associated with aging and changes in muscle control ultimately effect old individuals' energy levels and general ability to function at the highest level possible.

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APPENDIX-A

Table 1. Subject characteristics

Characteristic	Young			Old		
	Mean		SE	Mean		SE
Age (years)	*20.9	±	0.7	* 77.3	±	1.4
Height (cm)	175.0	±	2.5	169.9	±	2.5
Mass (kg)	68.4	±	2.0	70.4	±	13.5
BMI	22.3	±	0.4	24.3	±	1.0
Self Selected Walking Speed (m/sec)	1.0	±	0.1	1.0	±	0.1
Resting HR (beats/min)	84.1	±	4.2	80.3	±	3.3
Resting VO2 (ml/min)	302.5	±	21.8	295.9	±	21.5
Resting VO2 (ml/min/kg)	4.4	±	0.9	4.1	±	0.6

Values represented as mean ± standard error.

* represents an age effect between young and old subjects

Table 2. Metabolic variables for level walking conditions

Metabolic Variable	Age	Velocity						Age		
		Self Selected			.98 m/sec			1.2 m/sec					
		Mean		SE	Mean		SE				Mean		SE
RPE	Young	7.8	±	0.5	8.1	±	0.6	9.0	±	0.6	*8.3	±	0.5
	Old	10.9	±	0.5	9.8	±	0.6	11.8	±	0.6	*10.9	±	0.5
	Velocity	9.4	±	0.3	9.0	±	0.4	◘10.4	±	0.4			
HR (b/min)	Young	90.0	±	4.5	80.6	±	4.1	94.7	±	4.6	*91.8	±	4.3
	Old	107.8	±	4.5	105.8	±	4.1	114.1	±	4.6	*109.2	±	4.3
	Velocity	98.9	±	3.2	93.2	±	2.9	◘104.4	±	3.3			
RER	Young	0.86	±	0.02	0.88	±	0.02	0.86	±	0.01	*0.87	±	0.01
	Old	0.83	±	0.02	0.81	±	0.02	0.83	±	0.01	*0.82	±	0.01
	Velocity	0.84	±	0.01	0.84	±	0.01	0.85	±	0.01			
VO2 (ml/min)	Young	847.3	±	53.2	825.5	±	50.9	946.6	±	63.5	873.1	±	52.1
	Old	1039.3	±	53.2	960.4	±	50.9	1084.7	±	63.5	1018.1	±	52.1
	Velocity	843.3	±	37.7	877.9	±	36.0	◘1015.6	±	44.9			
VO2 (ml/min/kg)	Young	12.3	±	0.6	12.0	±	0.4	13.8	±	0.4	*12.7	±	0.4
	Old	14.6	±	0.6	12.9	±	0.4	15.0	±	0.4	*14.2	±	0.4
	Velocity	◘13.5	±	0.4	◘12.5	±	0.3	◘14.4	±	0.3			
Gross Metabolic Power (W/kg)	Young	4.2	±	0.2	4.1	±	0.1	4.7	±	0.2	*4.3	±	0.1
	Old	5.0	±	0.2	4.5	±	0.1	5.2	±	0.2	*4.9	±	0.1
	Velocity	4.6	±	0.2	4.3	±	0.1	◘5.0	±	0.1			
Gross Metabolic Energy Cost per distance (W/kg/m)	Young	4.3	±	0.2	4.2	±	0.2	3.9	±	0.2	*4.2	±	0.1
	Old	4.9	±	0.2	4.6	±	0.2	4.3	±	0.2	*4.6	±	0.1
	Condition	4.6	±	0.1	4.4	±	0.1	◘4.1	±	0.1			

Variables are expressed as mean ± standard error.

* represents an age effect between young and old subjects

◘ represents a velocity effect

Table 3. Metabolic variables for task dependent walking conditions

Metabolic Variable	Age	Task						Age		
		Incline 6%			Decline 6%					
		Mean		SE	Mean		SE	Mean		SE
RPE	Young	9.5	±	0.6	8.7	±	0.6	*9.1	±	0.5
	Old	♠12.8	±	0.6	♠10.2	±	0.6	*11.5	±	0.5
	Condition	◘11.2	±	0.5	◘9.4	±	0.4			
HR (b/min)	Young	103.7	±	4.6	85.2	±	4.3	*94.5	±	4.3
	Old	119.4	±	4.6	99.6	±	4.3	*109.5	±	4.3
	Condition	◘111.6	±	3.3	◘92.4	±	3.1			
RER	Young	0.88	±	0.02	♠0.91	±	0.02	*0.89	±	0.02
	Old	0.86	±	0.02	♠0.83	±	0.02	*0.84	±	0.02
	Condition	0.87	±	0.01	0.87	±	0.01			
VO2 (ml/min)	Young	1159.4	±	55.1	637.0	±	60.1	898.0	±	54.0
	Old	1233.6	±	55.1	809.6	±	60.1	1021.6	±	54.0
	Condition	◘1196.5	±	39.0	◘723.3	±	42.5			
VO2 (ml/min/kg)	Young	16.9	±	0.4	9.3	±	0.5	*13.1	±	0.3
	Old	17.2	±	0.4	11.1	±	0.5	*14.2	±	0.3
	Condition	◘17.1	±	0.3	◘10.2	±	0.4			
Gross Metabolic Power (W/kg)	Young	5.7	±	0.1	3.2	±	0.2	*4.47	±	0.13
	Old	6.0	±	0.1	3.8	±	0.2	*4.92	±	0.13
	Condition	◘5.9	±	0.1	◘3.5	±	0.1			
Gross Metabolic Energy Cost per distance (W/kg/m)	Young	5.9	±	0.1	3.3	±	0.2	*4.56	±	0.14
	Old	6.1	±	0.1	3.9	±	0.2	*5.02	±	0.14
	Condition	◘6.0	±	0.1	◘3.6	±	0.1			

Variables are expressed as mean ± standard error.

* represents an age effect between young and old subjects

◘ represents a condition effect

♠ represents an age by condition interaction

Table 4. Average antagonist muscle coactivation during level walking conditions

Muscle	Age	Gait Velocity (m/sec)						Age	
		Self Selected		0.98 m/sec		1.2 m/sec			
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
BF	Young	5.1 ±	2.5	7.5 ±	3.0	5.3 ±	3.0	*6.0 ±	2.6
	Old	21.7 ±	2.5	21.8 ±	3.0	22.1 ±	3.0	*21.8 ±	2.6
	Velocity	13.4 ±	1.8	14.7 ±	2.1	13.7 ±	2.1		
VL	Young	♠11.0 ±	1.9	4.2 ±	1.7	4.8 ±	1.7	*6.7 ±	1.5
	Old	♠15.6 ±	1.9	♠14.9 ±	1.7	♠16.8 ±	1.7	*15.7 ±	1.5
	Velocity	◘13.3 ±	1.3	9.6 ±	1.2	10.8 ±	1.2		
TA	Young	5.1 ±	0.8	4.9 ±	0.7	5.6 ±	0.8	*5.1 ±	0.7
	Old	8.0 ±	0.8	6.7 ±	0.7	7.0 ±	0.8	*7.2 ±	0.7
	Velocity	6.5 ±	0.6	6.0 ±	0.5	6.2 ±	0.6		
LG	Young	3.9 ±	0.8	4.2 ±	0.8	6.6 ±	1.9	*4.9 ±	1.0
	Old	9.1 ±	0.8	♠14.9 ±	1.7	8.4 ±	1.9	*10.8 ±	1.0
	Velocity	6.5 ±	0.6	9.6 ±	1.2	7.5 ±	1.3		
Total	Young	25.2 ±	3.9	20.8 ±	4.9	22.1 ±	4.8	*22.7 ±	3.9
	Old	54.3 ±	3.9	52.9 ±	4.9	54.3 ±	4.8	*53.8 ±	3.9
	Velocity	39.7 ±	2.8	36.9 ±	3.4	38.2 ±	3.4		

The average antagonist muscle coactivation is represented by % of the muscles' maximal voluntary contraction. It is represented by mean ± standard error

* represents an age effect between young and old subjects

◘ represents a velocity effect

♠ represents an age by condition interaction

Table 5. Average antagonist muscle coactivation based upon type of task

Muscle	Age	Task						Age		
		Incline 6%			Decline 6%					
		Mean		SE	Mean		SE	Mean		SE
BF	Young	6.4	±	1.5	4.1	±	1.7	*5.2	±	1.4
	Old	22.5	±	1.5	16.1	±	1.7	*19.3	±	1.4
	Condition	◘14.5	±	1.1	◘10.1	±	1.2			
VL	Young	4.1	±	1.5	5.1	±	1.4	*4.6	±	1.4
	Old	14.6	±	1.5	15.4	±	1.4	*15.0	±	1.4
	Condition	◘9.3	±	1.0	◘10.3	±	1.0			
TA	Young	5.4	±	0.7	5.1	±	0.8	5.2	±	0.7
	Old	6.1	±	0.7	7.5	±	0.8	6.8	±	0.7
	Condition	5.7	±	0.5	6.3	±	0.6			
LG	Young	4.7	±	0.9	4.1	±	0.8	*4.4	±	0.8
	Old	7.7	±	0.9	7.8	±	0.8	*7.8	±	0.8
	Condition	6.2	±	0.6	6.0	±	0.6			
Total	Young	20.6	±	2.6	18.3	±	3.1	*19.4	±	2.7
	Old	50.9	±	2.6	46.9	±	3.1	*48.9	±	2.7
	Condition	◘35.7	±	1.9	◘32.6	±	2.2			

The average antagonist muscle coactivation is represented by % of the muscles' maximal voluntary contraction. It is represented by mean ± standard error

* represents an age effect between young and old subjects

◘ represents a condition effect

Table 6. Percent difference between young and old metabolic variables

Metabolic Variable	Condition				
	Self Selected	0.98 m/sec	1.2 m/sec	Incline	Decline
RPE	28.4	17.3	23.7	25.7	14.8
HR (b/min)	16.5	23.8	17.0	13.1	14.5
VO2 (ml/min)	18.5	14.0	12.7	6.0	21.3
VO2 (ml/min/kg)	15.8	7.0	8.0	1.9	16.6
Gross Metabolic Power (W/kg)	16.0	8.9	9.6	4.2	17.0
Gross Metabolic Energy Cost per distance (W/kg/m)	12.2	8.7	9.3	4.2	17.0

Old individuals had higher values of all the variables represented in this table. The values here are the percent differences between young and old.

Table 7. Percent difference between young and old coactivation values

Muscle	Condition				
	Self Selected	0.98 m/sec	1.2 m/sec	Incline	Decline
BF	76.3	65.6	76.0	71.6	74.5
VL	29.5	71.8	71.4	71.9	66.9
TA	36.0	26.6	20.0	11.5	32.0
LG	57.1	71.8	21.4	39.0	47.4
Total Antagonist Muscle Coactivation	53.6	60.7	59.3	59.5	61.0

Old individuals had greater coactivation values in all muscles in all conditions. The values represent the difference between old and young coactivation expressed as a percent.

Table 8. Normalized agonist EMG values Expressed as % MVC.

Muscle	Age	Condition				
		Self Selected	0.98 m/sec	1.2 m/sec	Incline	Decline
BF	Young	5.4	8.1	9.5	8.2	10.2
	Old	29.0	26.1	28.3	27.3	22.5
VL	Young	8.0	5.0	16.8	5.4	7.9
	Old	22.9	17.5	5.4	20.2	20.0
TA	Young	12.4	10.9	11.5	11.6	10.4
	Old	18.2	18.0	20.8	18.7	17.2
LG	Young	11.1	13.2	14.8	20.6	8.7
	Old	25.3	29.7	29.5	31.3	17.9

Old individuals experienced greater normalized agonist EMG activity than young individuals.

Table 9. Raw agonist EMG values

Muscle	Age	Condition				
		Self Selected	0.98 m/sec	1.2 m/sec	Incline	Decline
BF	Young	0.028	0.029	0.033	0.028	0.036
	Old	0.034	0.031	0.034	0.032	0.026
VL	Young	0.020	0.019	0.022	0.019	0.026
	Old	0.041	0.032	0.025	0.040	0.036
TA	Young	0.073	0.069	0.074	0.077	0.066
	Old	0.082	0.080	0.092	0.084	0.075
LG	Young	0.027	0.030	0.035	0.051	0.020
	Old	0.041	0.048	0.048	0.050	0.029

Mean raw EMG activity for young and old subjects. Old individuals had greater levels of activation in all conditions and muscles except for incline LG and decline BF.

Table 10. Raw antagonist EMG values

Muscle	Age	Condition				
		Self Selected	0.98 m/sec	1.2 m/sec	Incline	Decline
BF	Young	0.018	0.025	0.018	0.020	0.014
	Old	0.026	0.026	0.027	0.030	0.019
VL	Young	0.014	0.014	0.019	0.015	0.018
	Old	0.029	0.028	0.031	0.028	0.029
TA	Young	0.029	0.029	0.032	0.032	0.033
	Old	0.036	0.031	0.031	0.029	0.034
LG	Young	0.009	0.009	0.014	0.011	0.009
	Old	0.014	0.015	0.013	0.012	0.012

Mean raw EMG activity for young and old adults. Old individuals had greater levels of activation in all conditions other than 1.2 m/sec TA and LG.

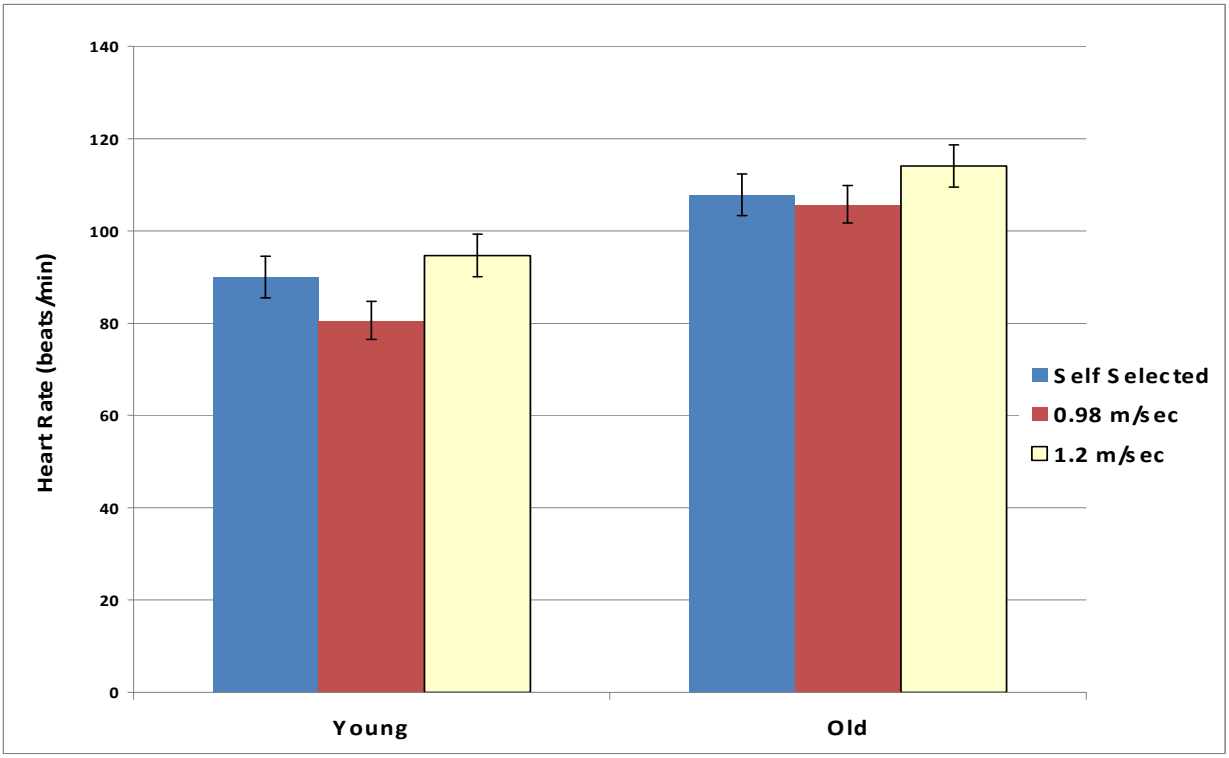

Figure 50. Age and velocity interaction of heart rate. There were no significant interactions.

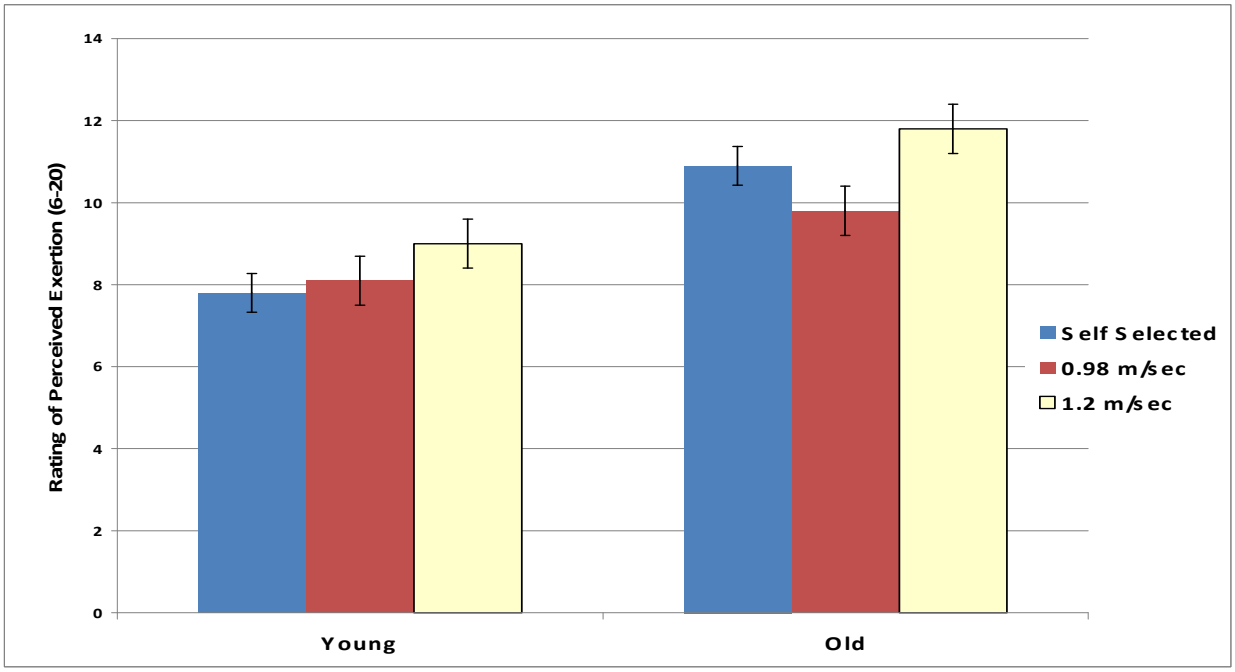

Figure 51. Age and velocity interaction of rating of perceived exertion. There were no significant interactions.

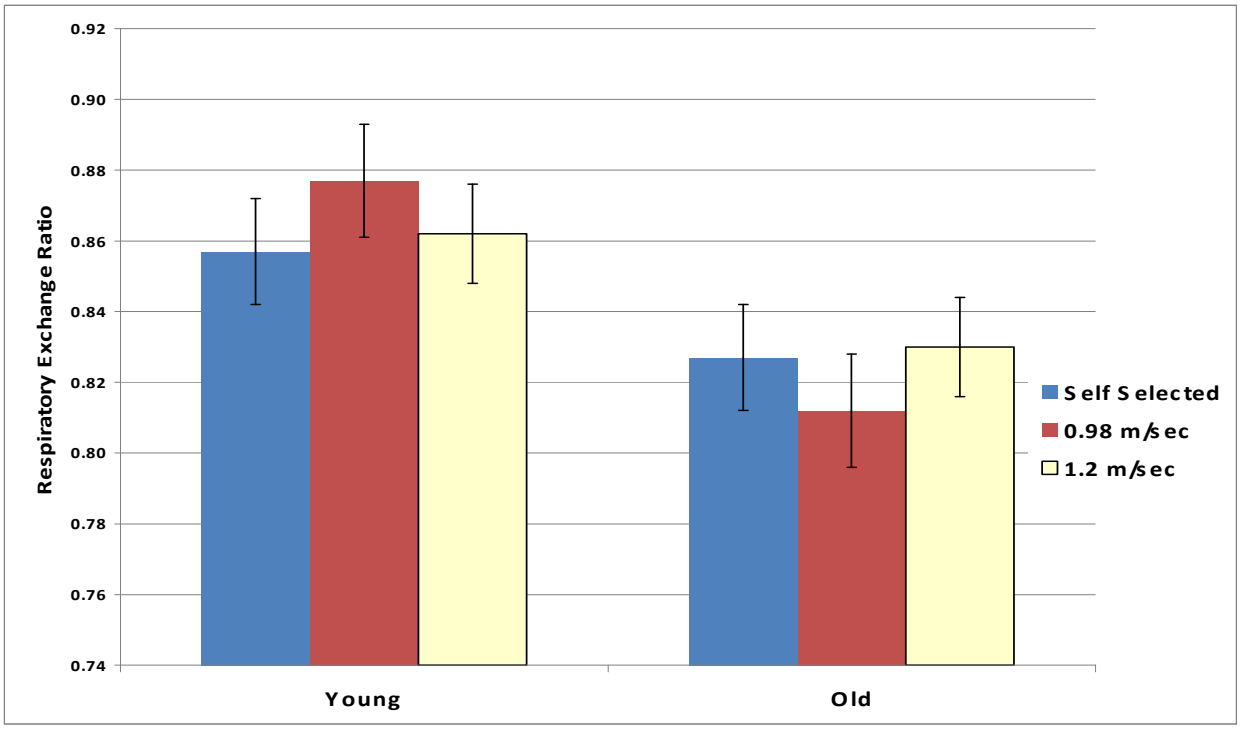

Figure 52. Age and velocity interaction of respiratory exchange ratio. There were no significant interactions.

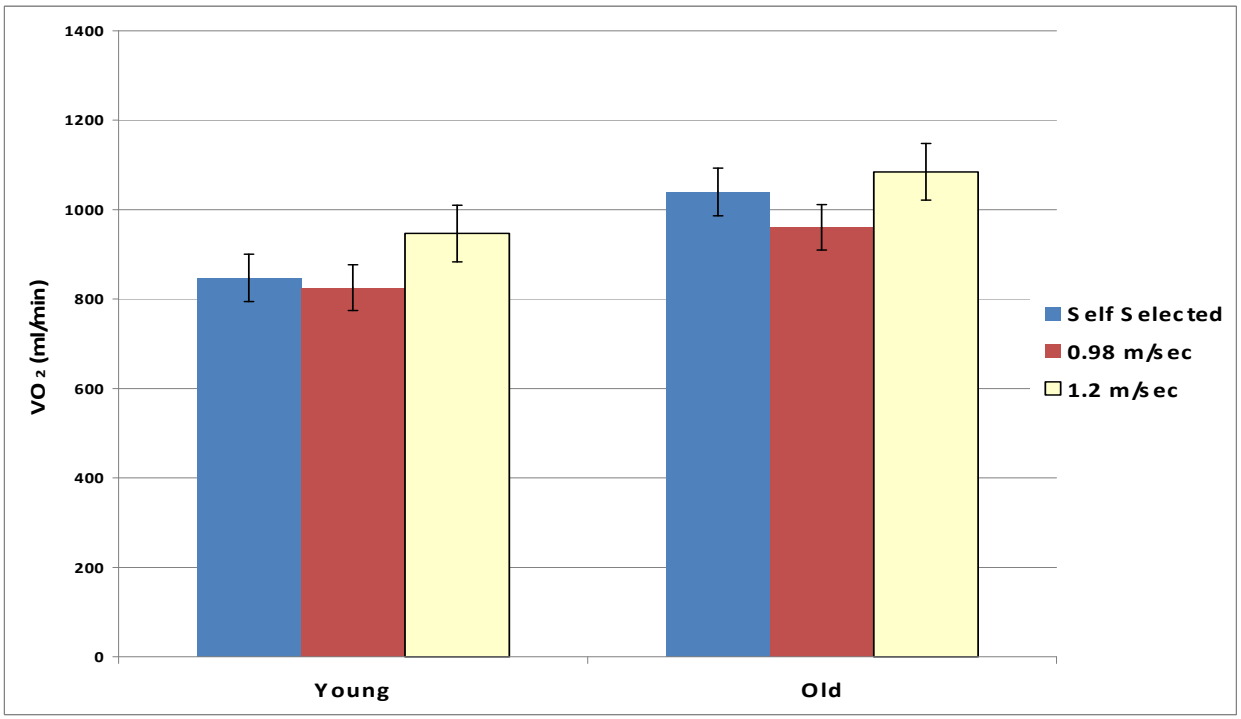

Figure 53. Age and velocity interaction of VO2 separated into velocity and age. There were no significant interactions.

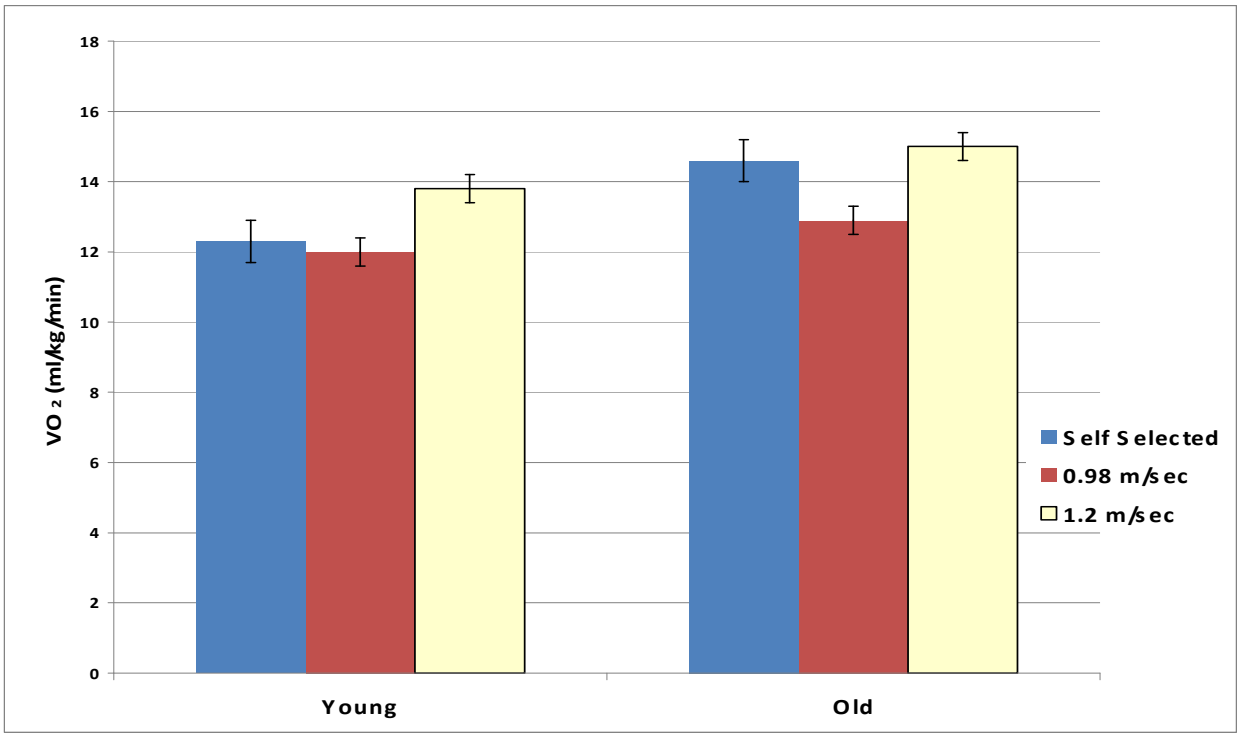

Figure 54. Age and velocity interaction of VO2 normalized to body weight. There were no significant interactions.

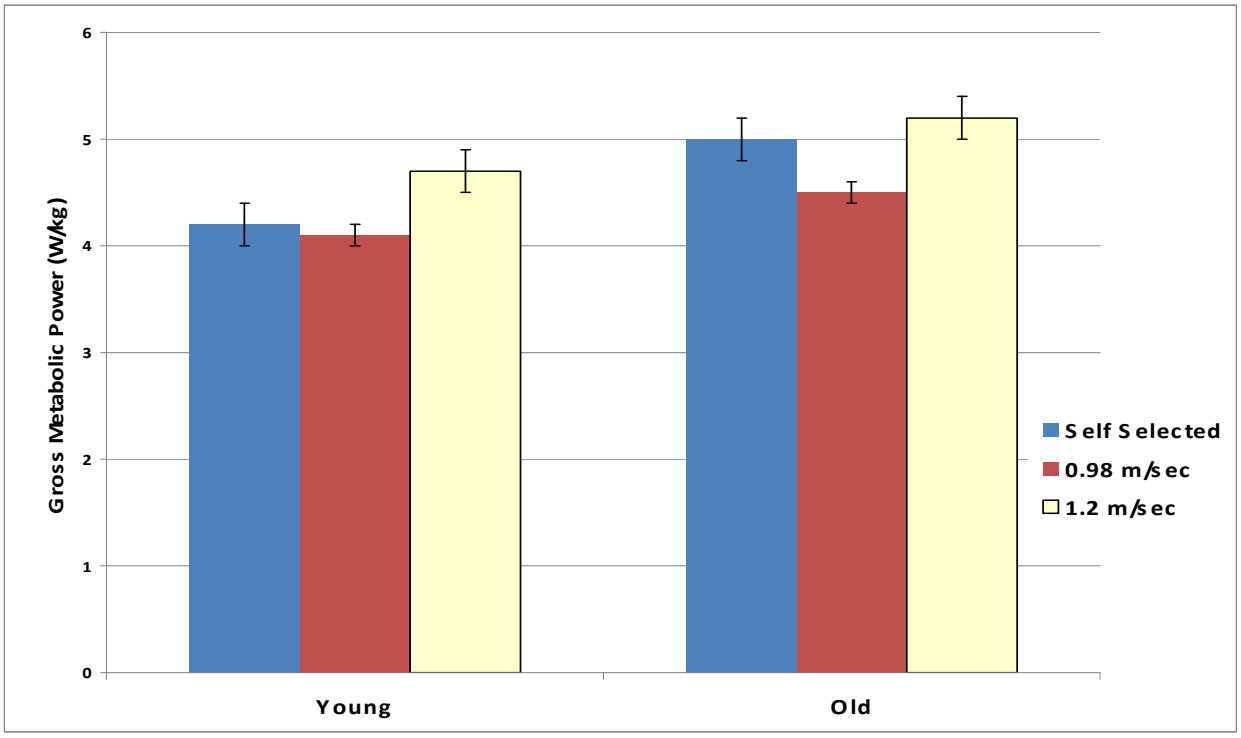

Figure 55. Age and velocity interaction of gross metabolic power. There were no significant interactions.

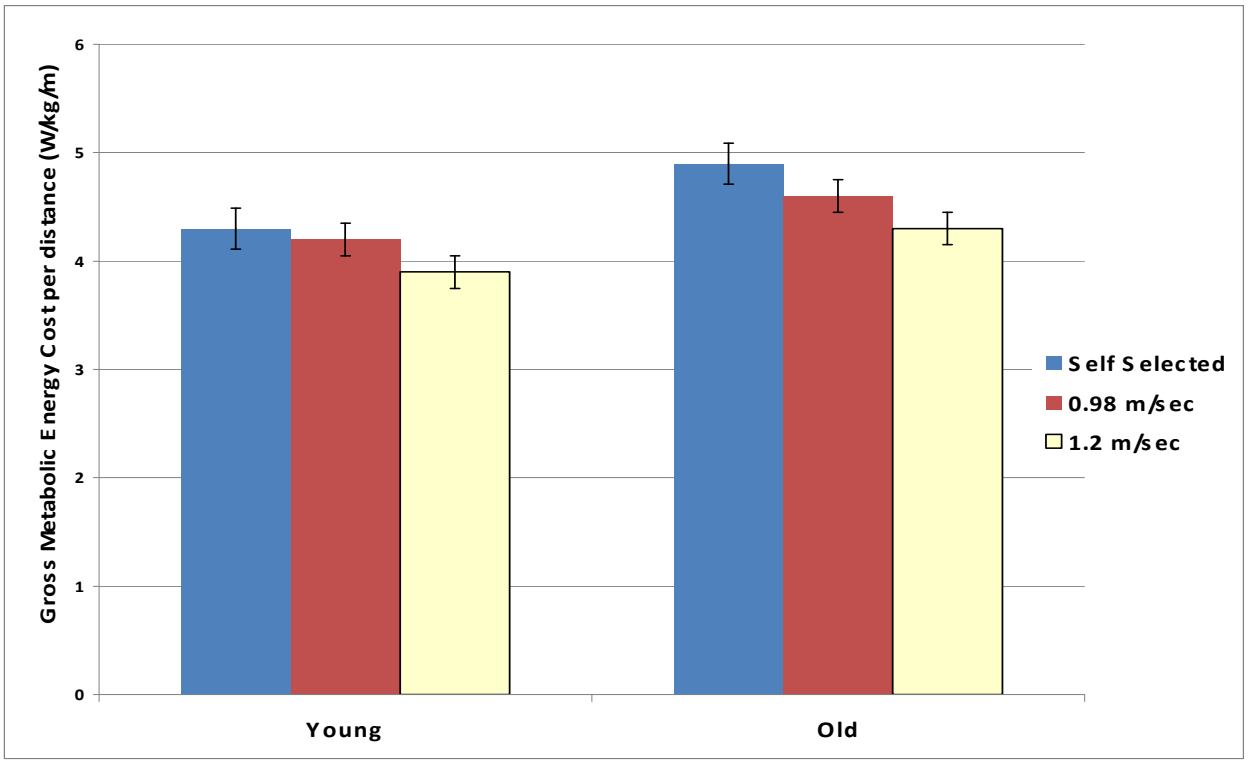

Figure 56. Age and velocity interaction of gross metabolic energy cost per distance. There were no significant interactions.

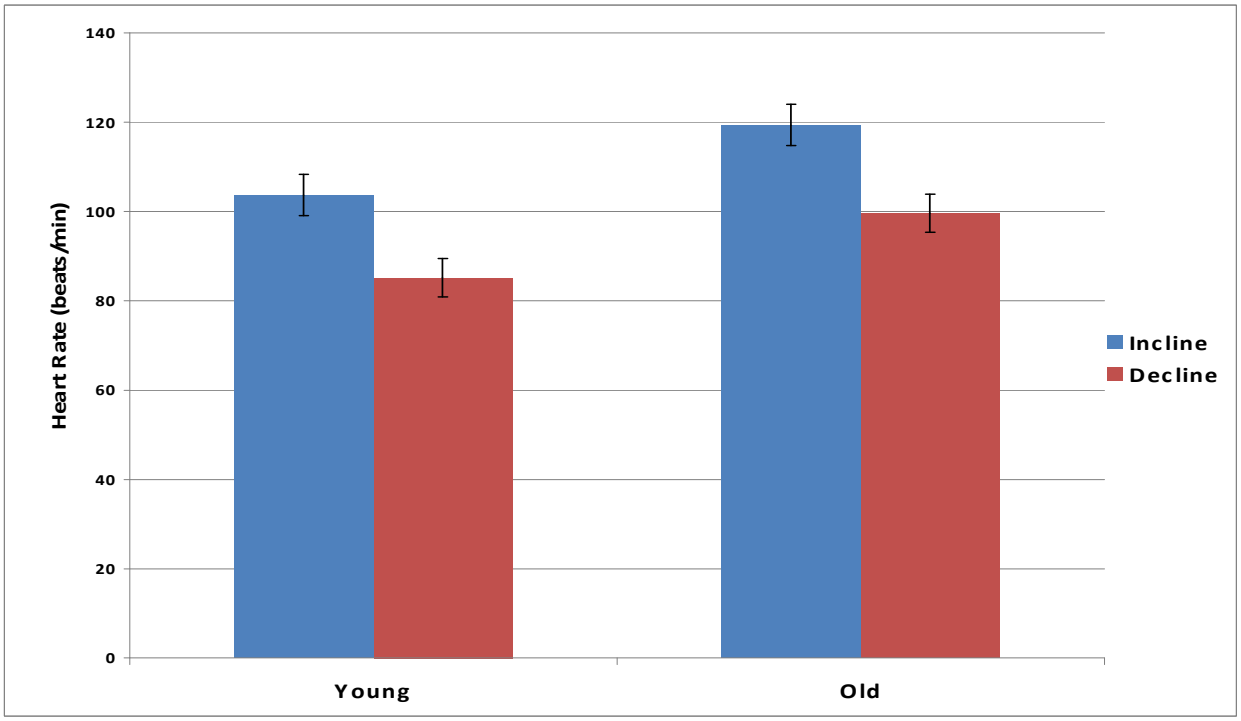

Figure 57. Age by task interaction of HR . There were no significant interactions.

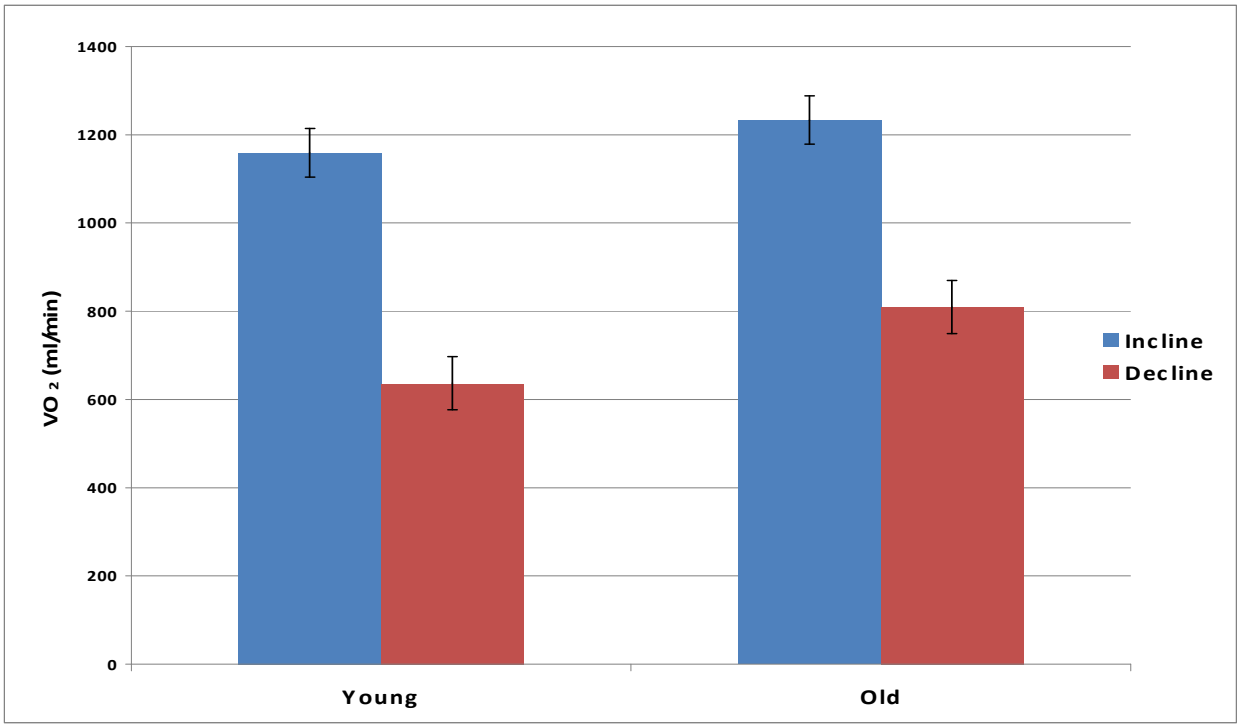

Figure 58. Age by task interaction of VO2. There were no significant interactions.

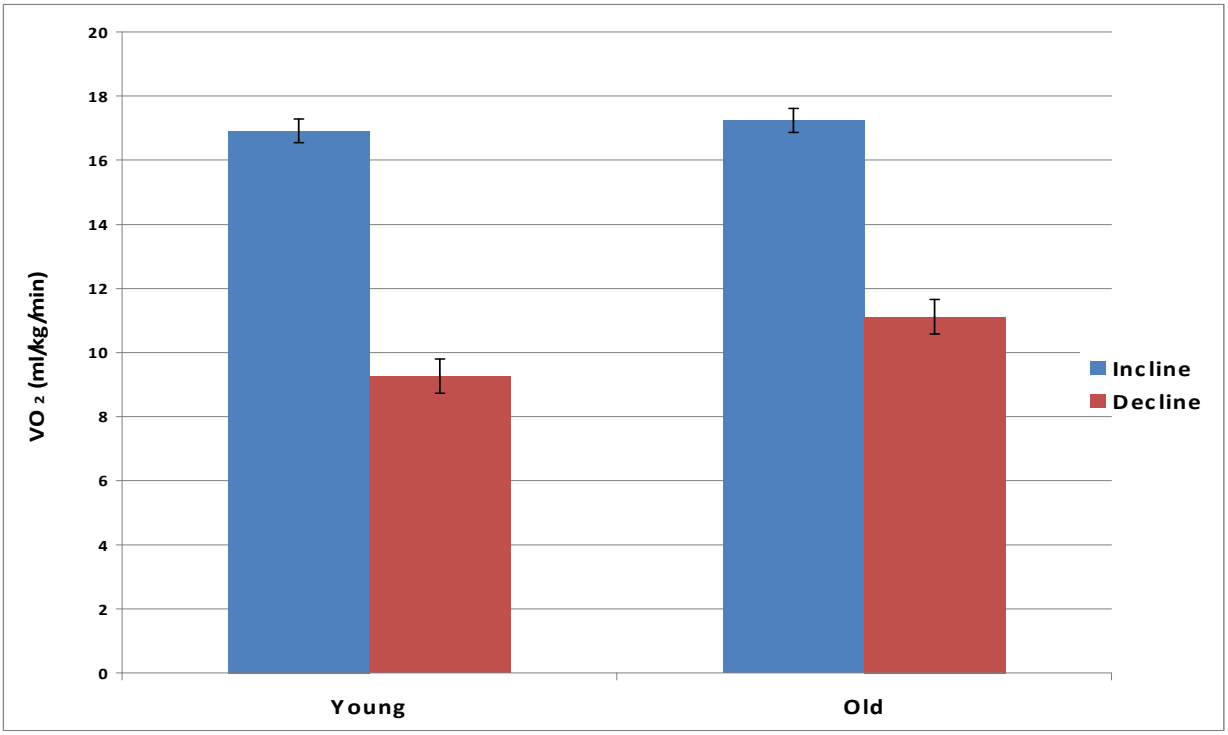

Figure 59. Age by task interaction of VO2 normalized to body weight. There were no significant interactions.

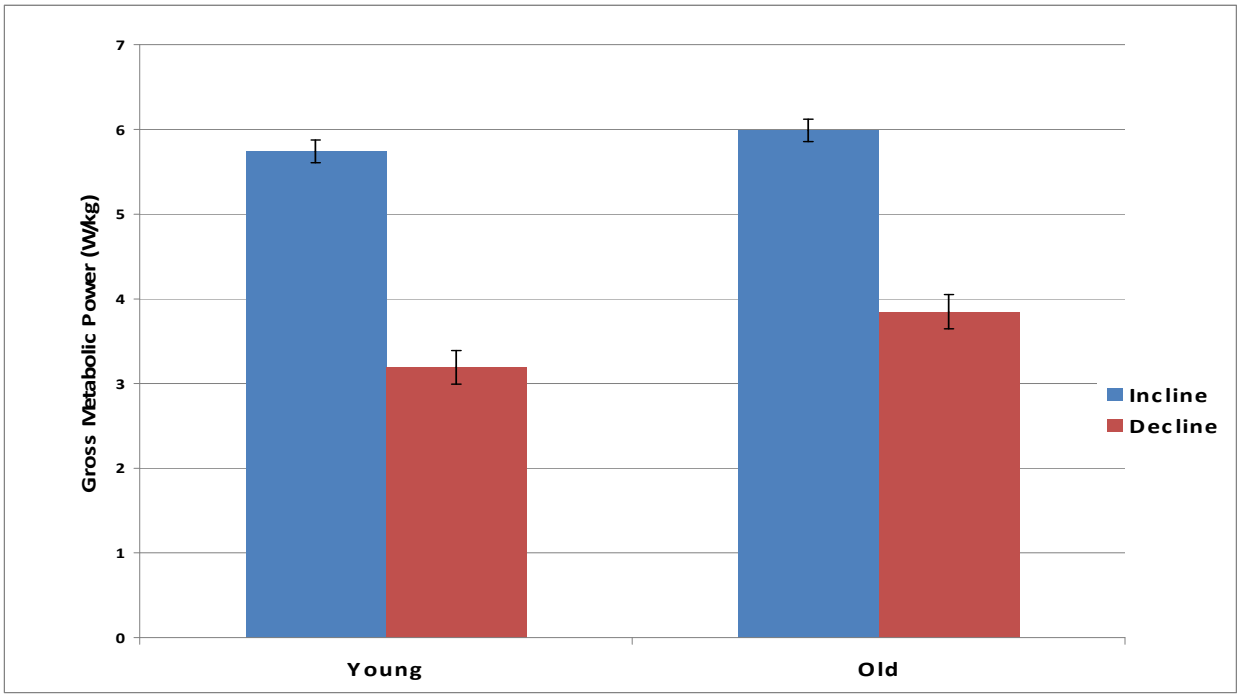

Figure 60. Age by task interaction of gross metabolic power . There were no significant interactions.

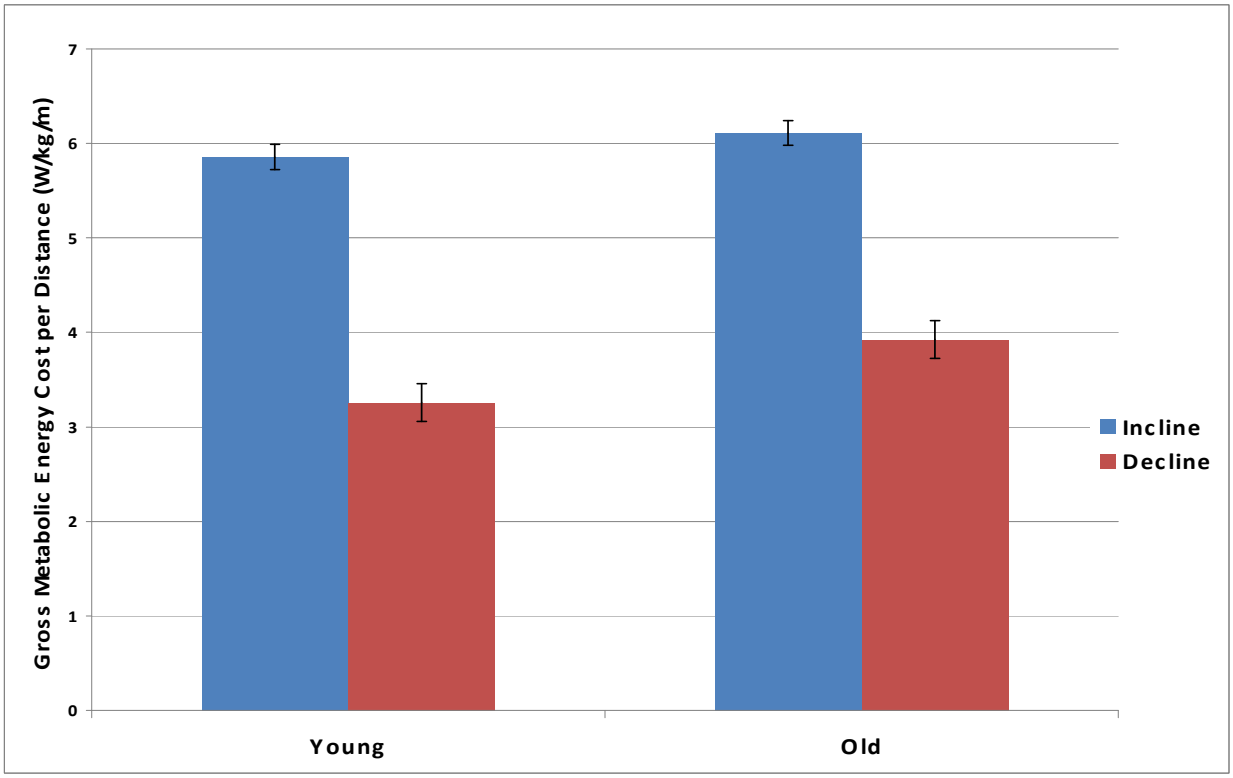

Figure 61. Age by task interaction of gross metabolic energy cost per distance . There were no significant interactions.

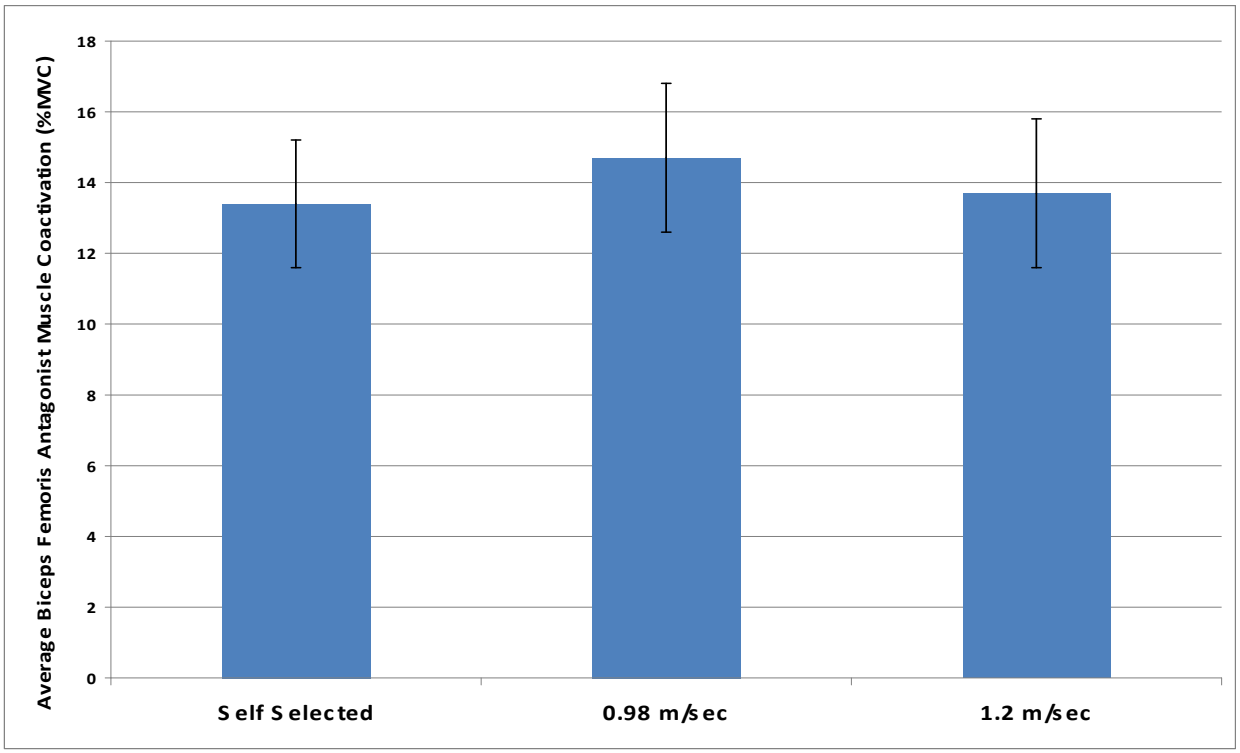

Figure 62. Velocity main effect of biceps femoris antagonist muscle coactivation in old and young adults. There was no velocity effect for this muscle.

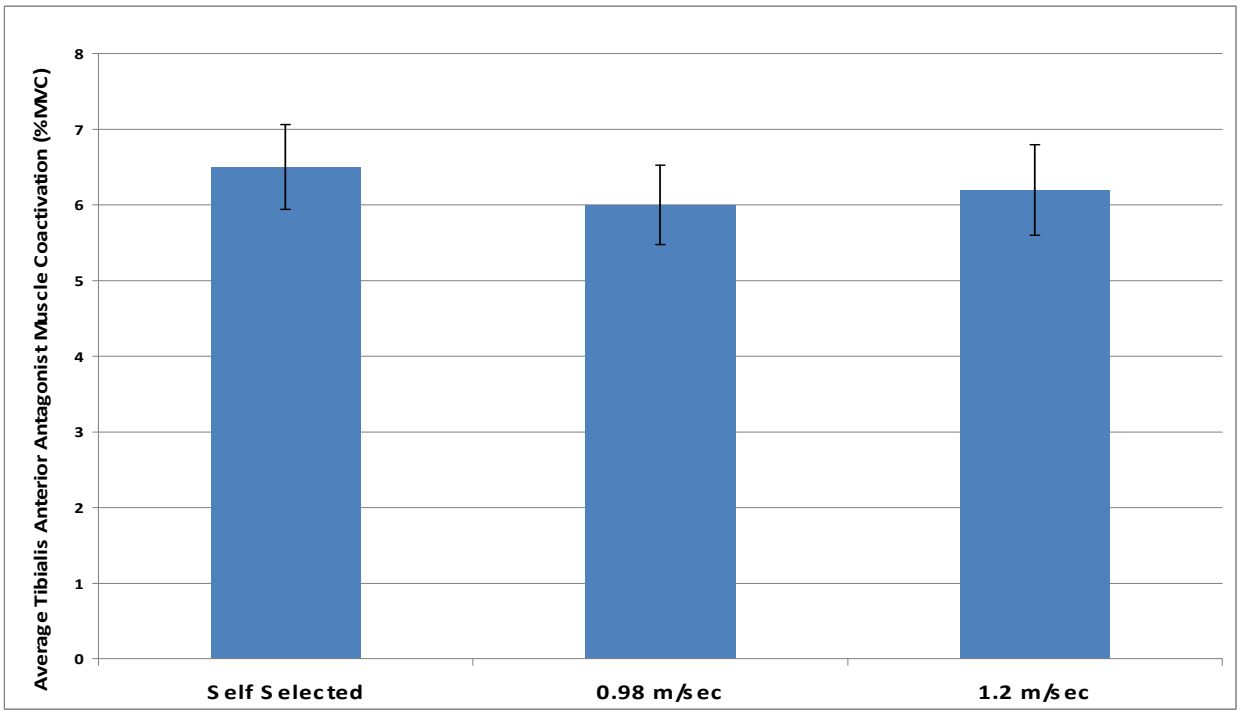

Figure 63. Velocity main effect of tibialis anterior antagonist muscle coactivation in old and young adults. There was no velocity effect for this muscle.

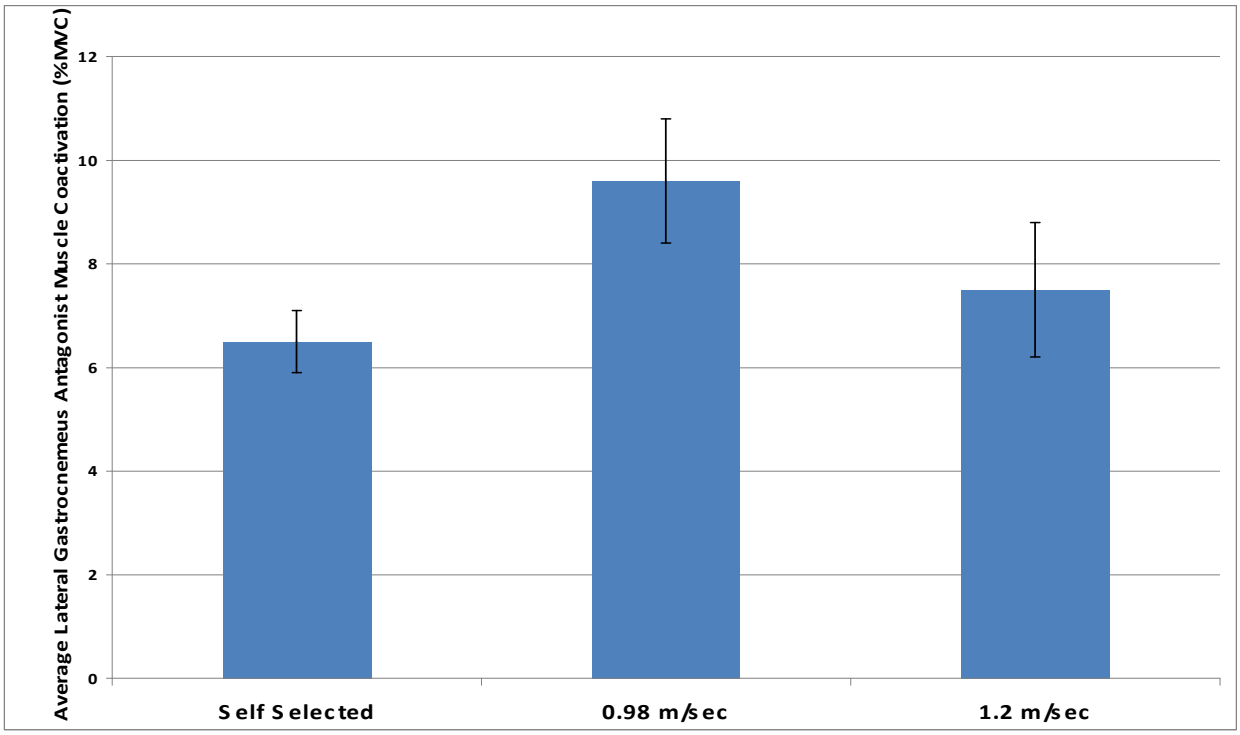

Figure 64. Velocity main effect of lateral gastrocnemeus antagonist muscle coactivation in old and young adults. There was no velocity effect for this muscle.

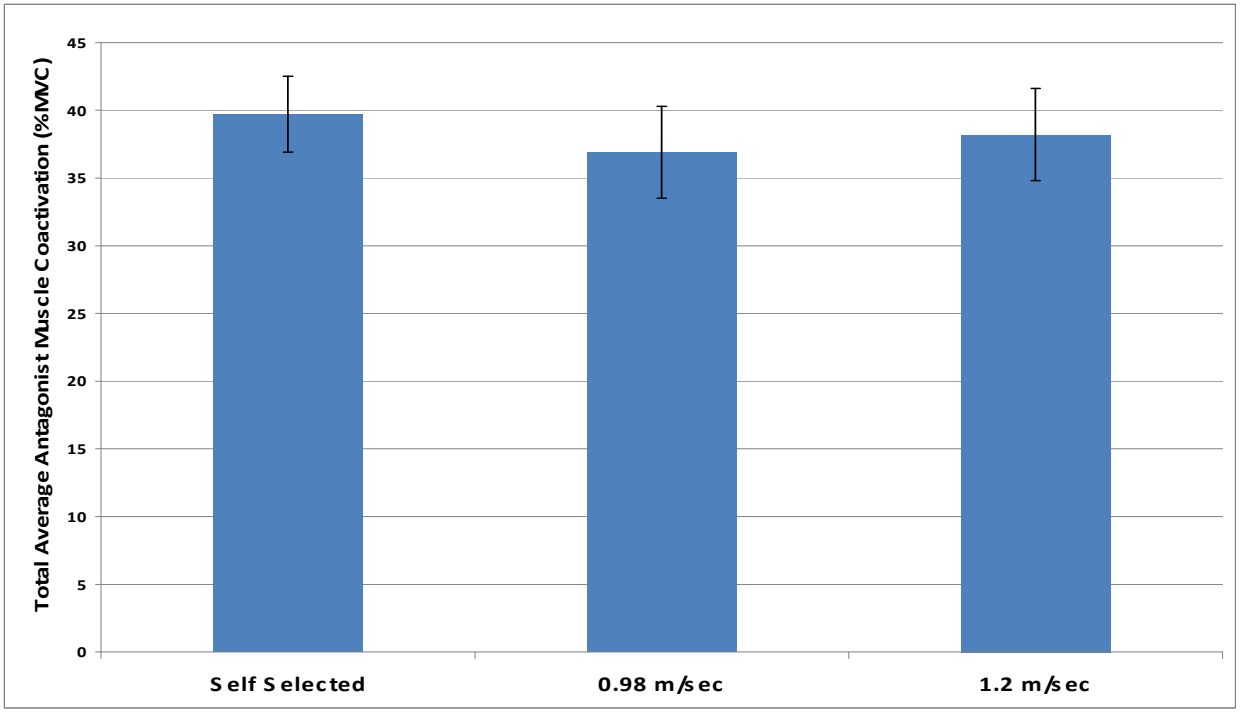

Figure 65. Velocity main effect of total antagonist muscle coactivation in old and young adults. There was no velocity effect.

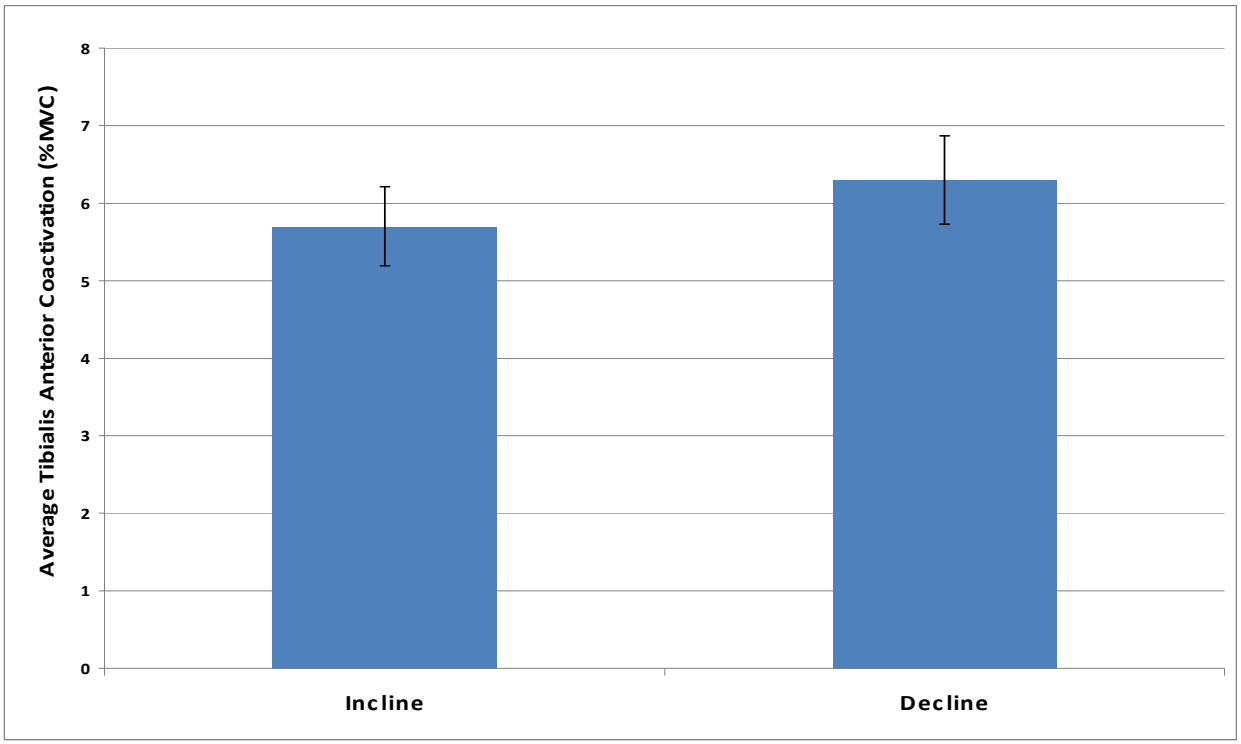

Figure 66. Task main effect of tibialis anterior antagonist muscle coactivation in old and young adults. There was no task main effect.

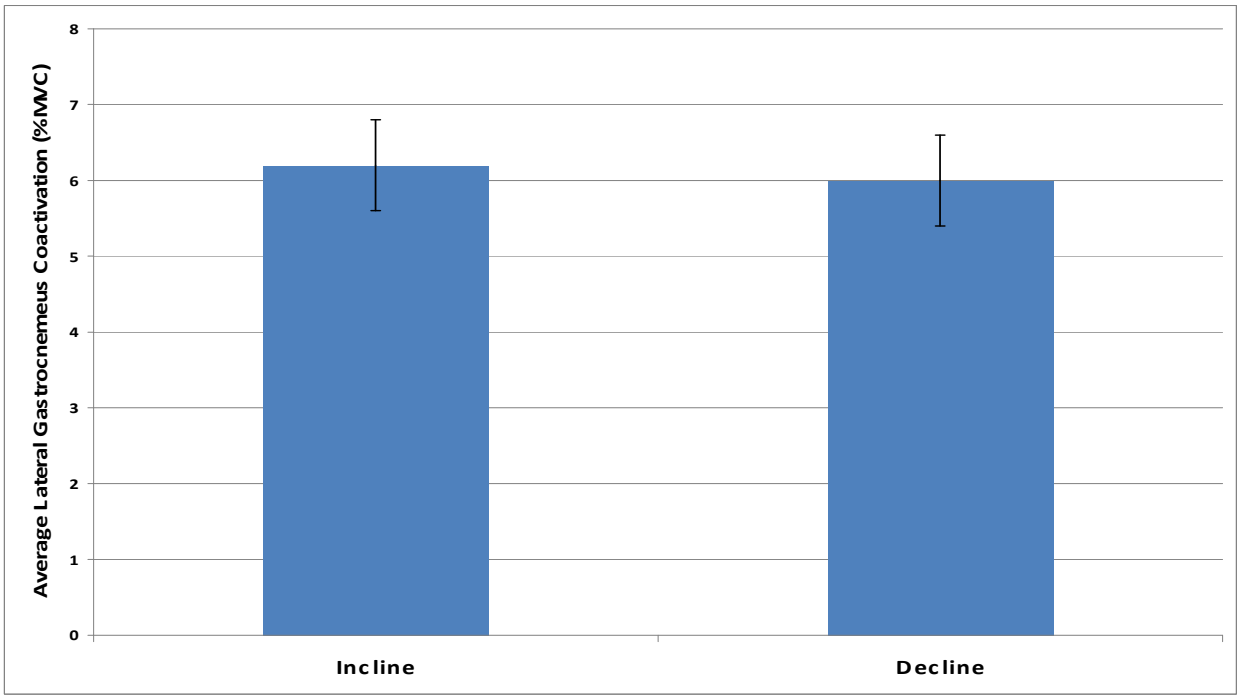

Figure 67. Task main effect of lateral gastrocnemeus antagonist muscle coactivation in old and young adults. There was no task main effect.

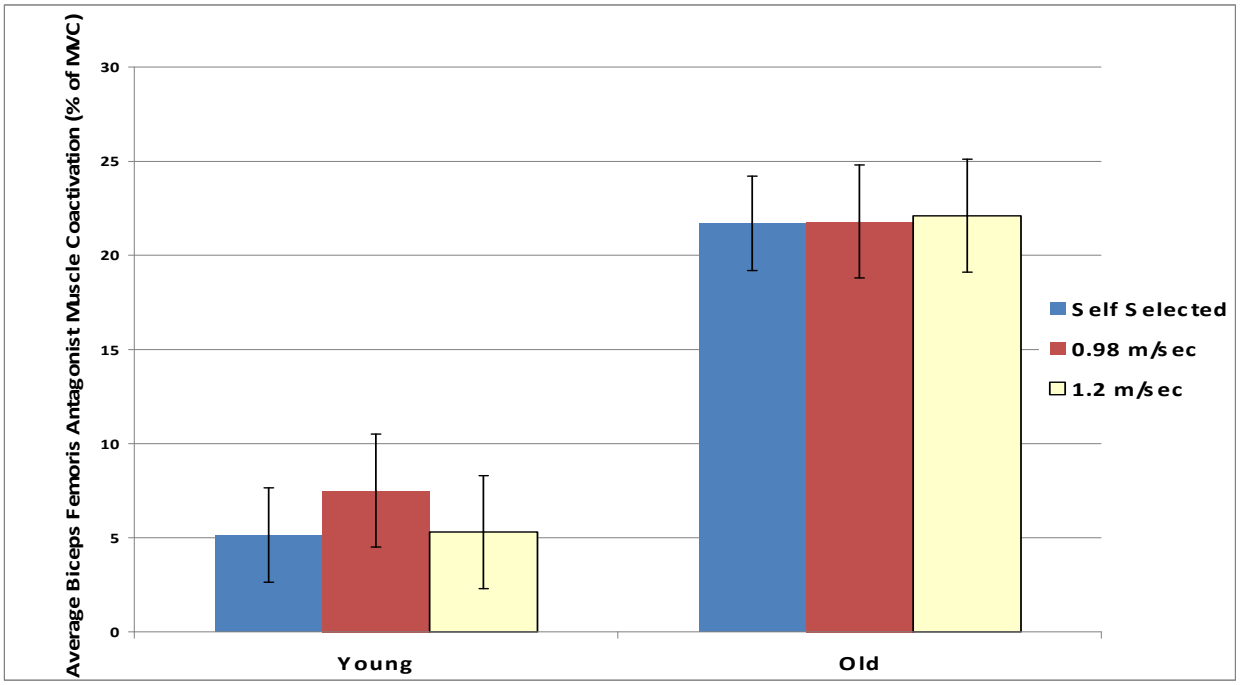

Figure 68. Age by velocity interaction effect of biceps femoris antagonist muscle coactivation. There was no significant interaction effect.

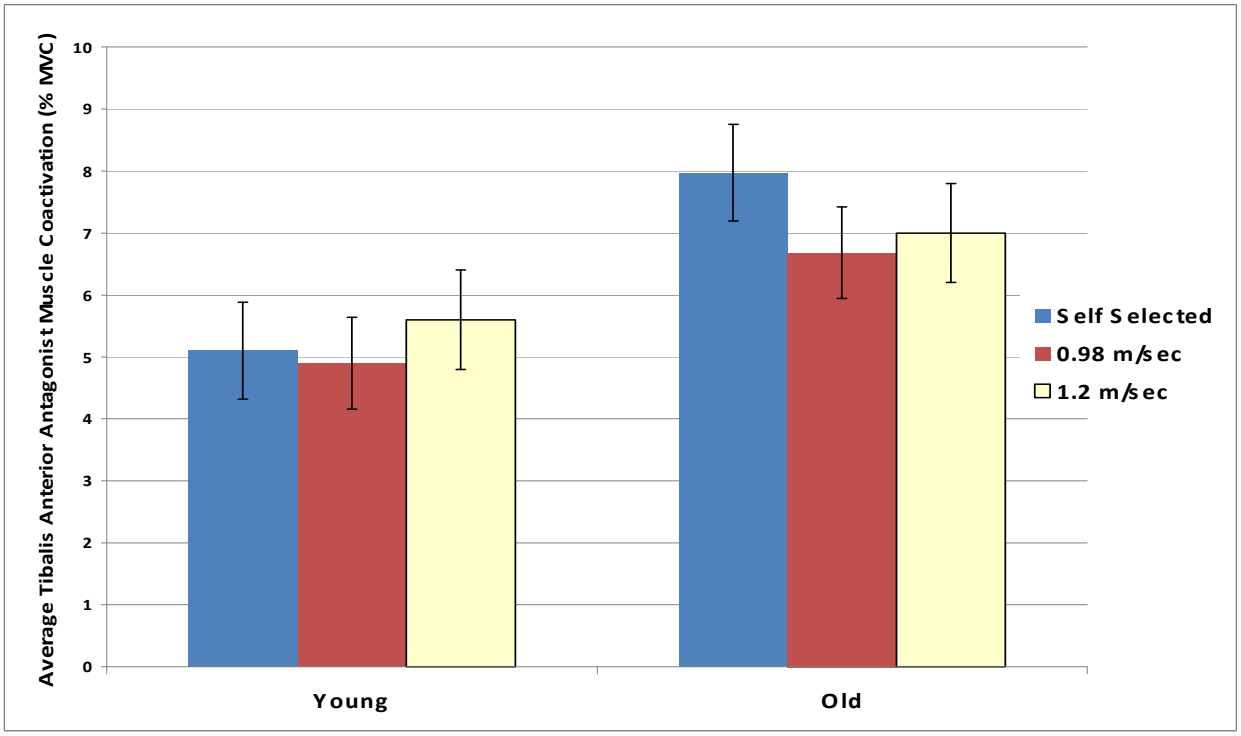

Figure 69. Age by velocity interaction effect of tibialis anterior antagonist muscle coactivation. There was no significant interaction effect.

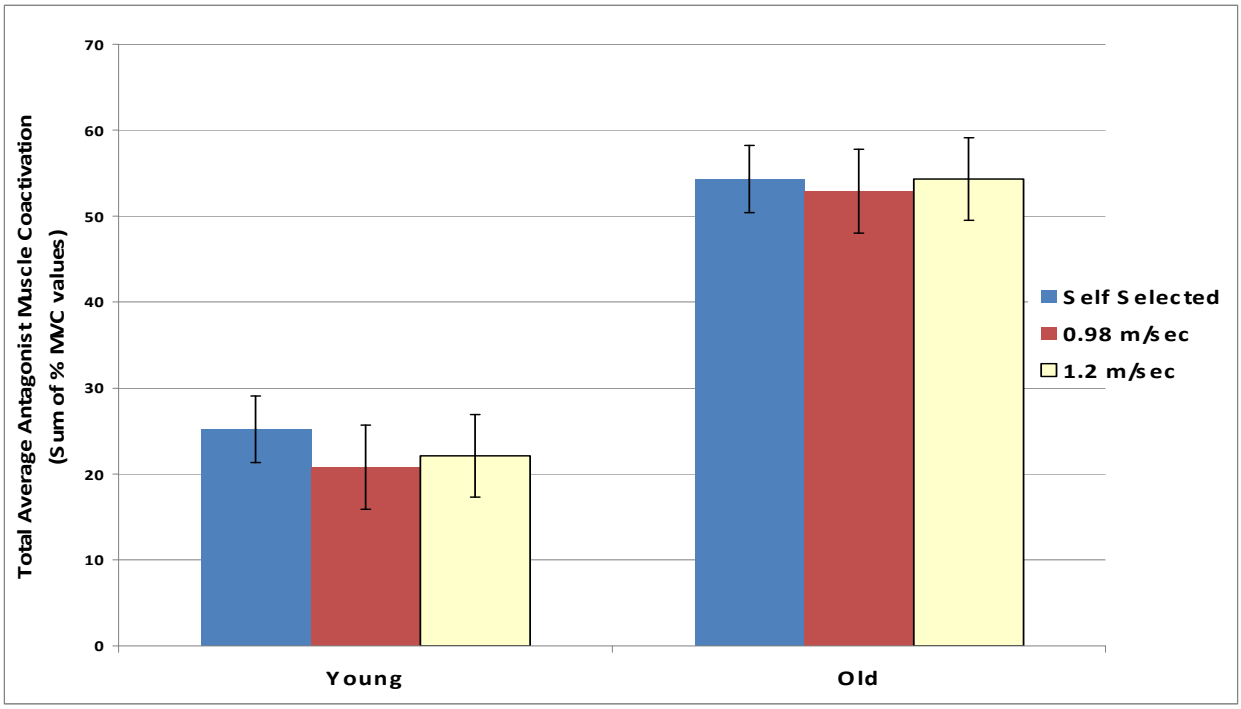

Figure 70. Age by velocity interaction effect of total antagonist muscle coactivation. There was no significant interaction effect.

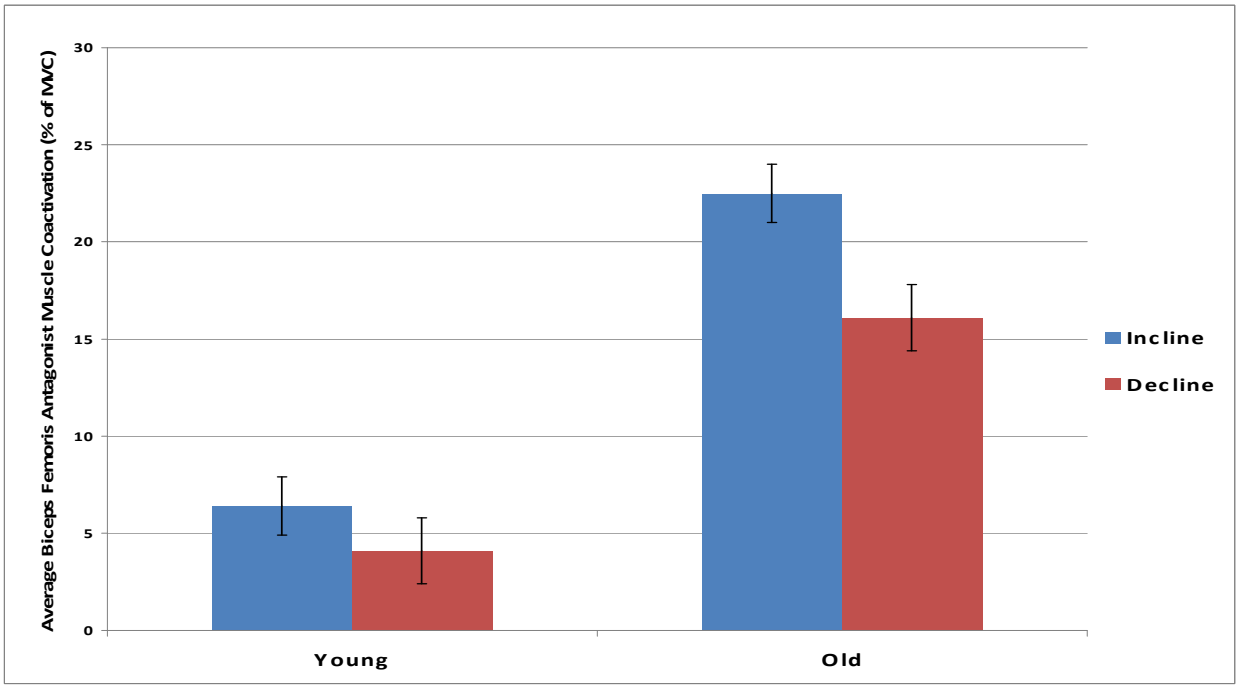

Figure 71. Age by task interaction effect of biceps femoris antagonist muscle coactivation. There was no significant interaction effect.

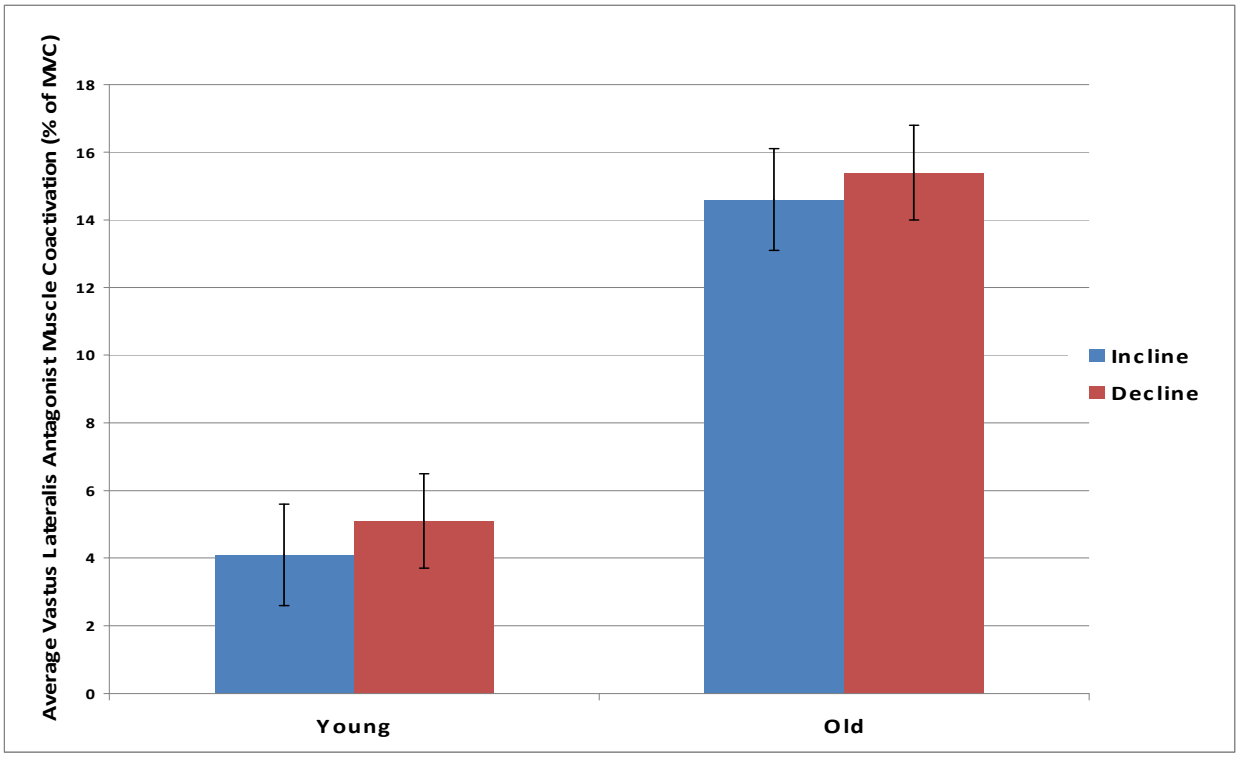

Figure 72. Age by task interaction effect of vastus lateralis antagonist muscle coactivation. There was no significant interaction effect.

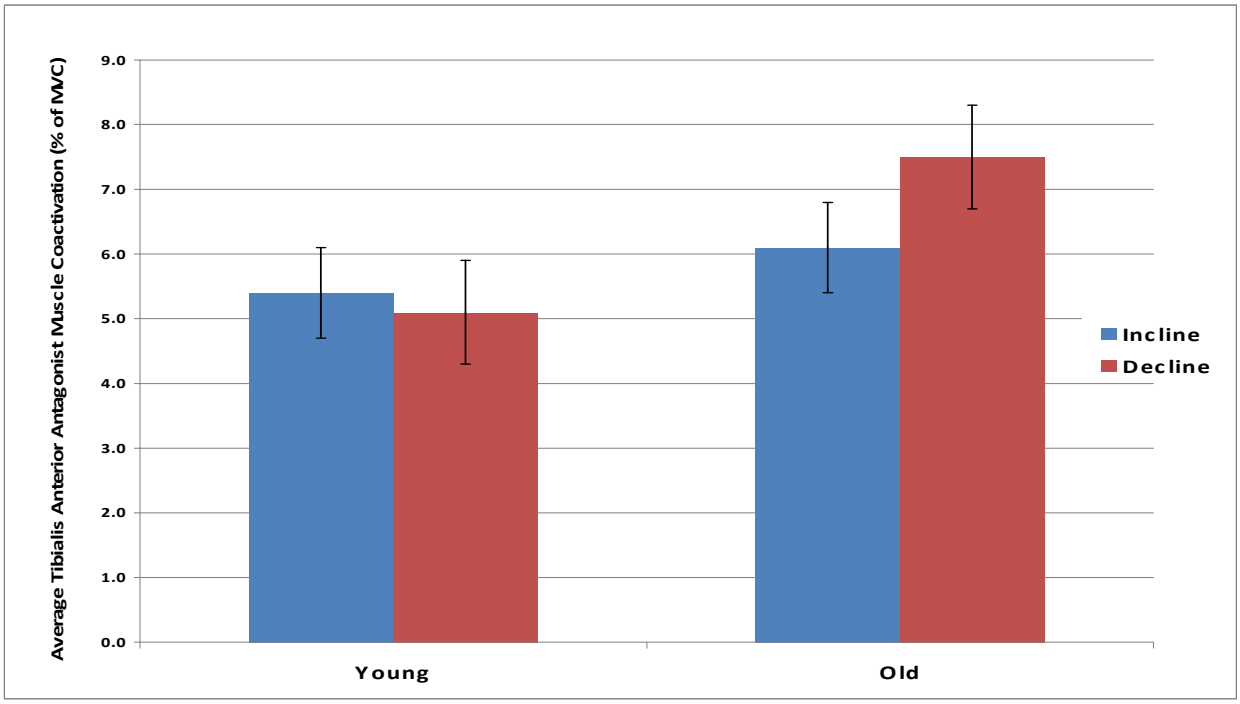

Figure 73. Age by task interaction effect of tibialis anterior antagonist muscle coactivation . There was no significant interaction effect.

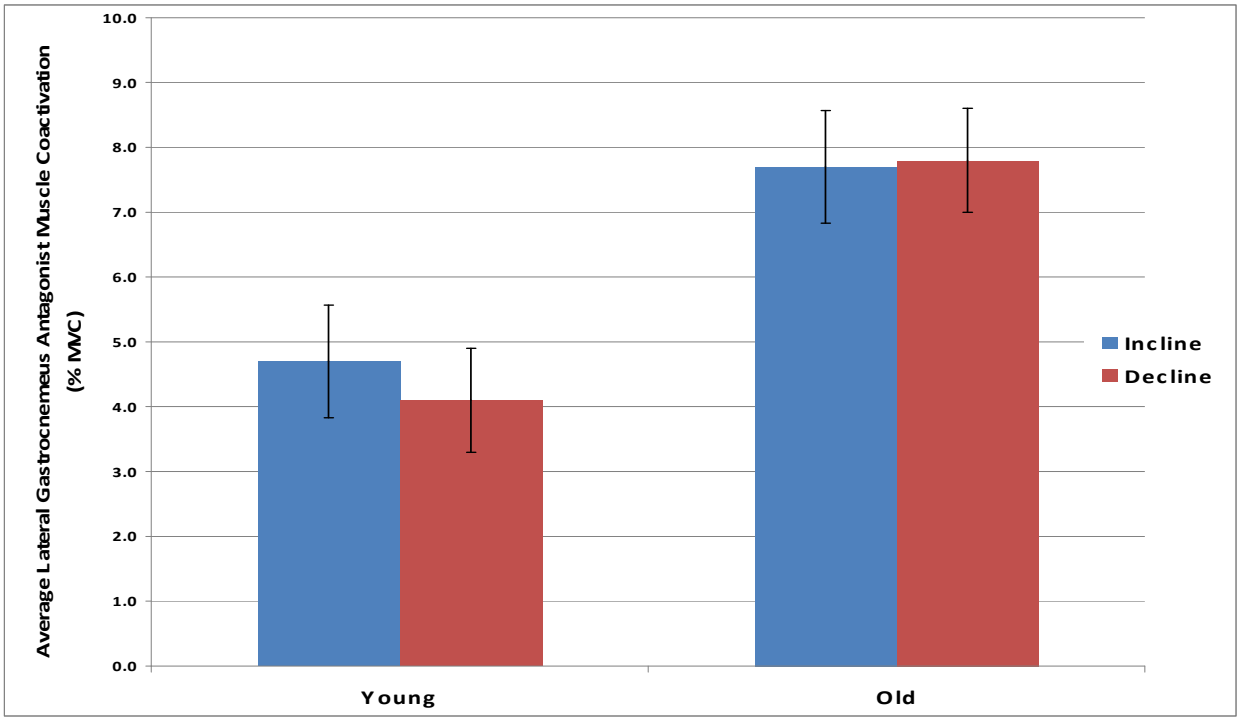

Figure 74. Age by task interaction effect of lateral gastrocnemeus antagonist muscle coactivation. There was no significant interaction effect.

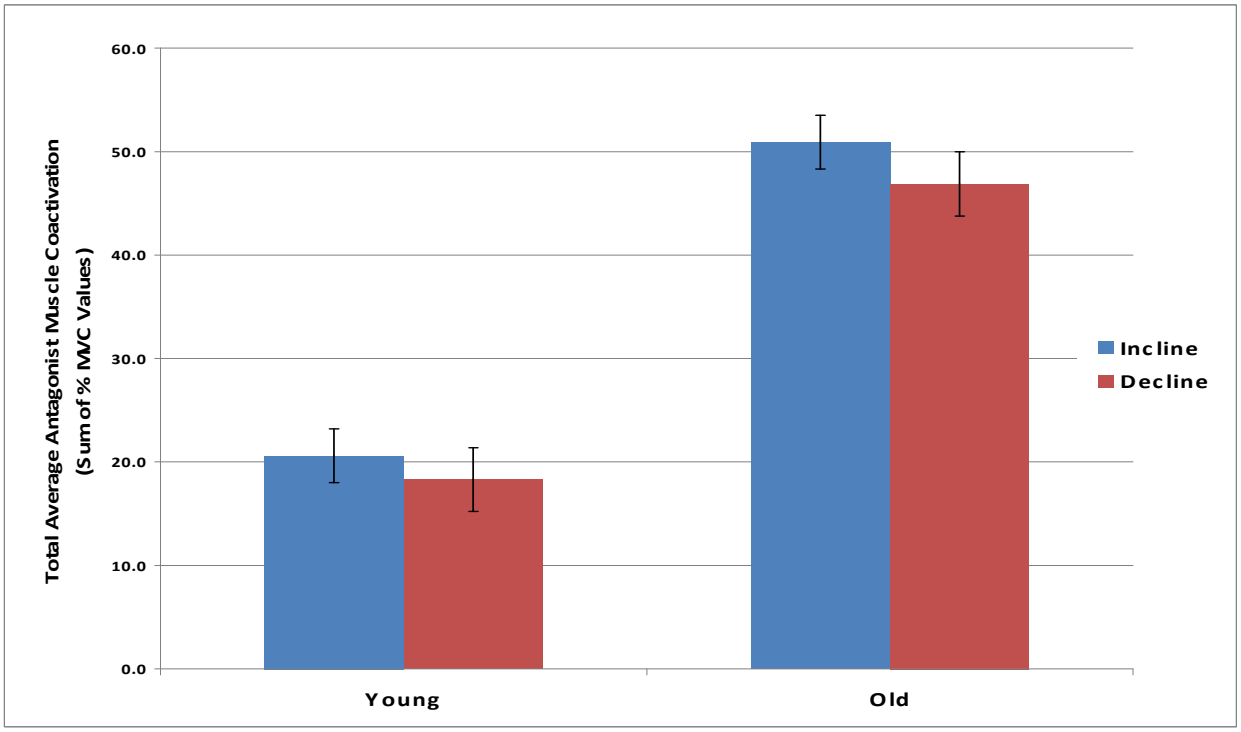

Figure 75. Age by task interaction effect of total antagonist muscle coactivation. There was no significant interaction effect.

University and Medical Center Institutional Review Board
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Chair and Director of Biomedical IRB: L. Wiley Nifong, MD
Chair and Director of Behavioral and Social Science IRB: Susan L. McCammon, PhD

TO: Paul DeVita, PhD, Dept. of EXSS, ECU

FROM: UMCIRB

DATE: September 10, 2009

RE: Expedited Continuing Review of a Research Study

TITLE: "Oxygen Consumption During Incline Walking"

UMCIRB #05-0273

The above referenced research study was initially reviewed and approved by expedited review on 5/11/05. This research study has undergone a subsequent continuing review using expedited review on 9/8/09. This research study is eligible for expedited review because it is a collection of data through noninvasive procedures (not involving general anesthesia or sedation) routinely employed in clinical practice, excluding procedures involving x-rays or microwaves. Where medical devices are employed, they must be cleared/approved for marketing. (Studies intended to evaluate the safety and effectiveness of the medical device are not generally eligible for expedited review, including studies of cleared medical devices for new indications.) Examples: (a) physical sensors that are applied either to the surface of the body or at a distance and do not involve input of significant amounts of energy into the subject or an invasion of the subject's privacy; (b) weighing or testing sensory acuity; (c) magnetic resonance imaging; (d) electrocardiography, electroencephalography, thermography, detection of naturally occurring radioactivity, electroretinography, ultrasound, diagnostic infrared imaging, doppler blood flow, and echocardiography; (e) moderate exercise, muscular strength testing, body composition assessment, and flexibility testing where appropriate given the age, weight, and health of the individual. The Chairperson (or designee) deemed this **unfunded** study **no more than minimal risk** requiring a continuing review in **12 months**. Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminate an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a continuing review/closure application to the UMCIRB prior to the date of study expiration. The investigator must adhere to all reporting requirements for this study.

The above referenced research study has been given approval for the period of **9/8/09** to **9/7/10**. The approval includes the following items:

- Continuing Review Form (dated 9/2/09)
- Protocol Summary
- Informed consent (version date 5/10/05)

The Chairperson (or designee) does not have a conflict of interest on this study.

The UMCIRB applies 45 CFR 46, Subparts A-D, to all research reviewed by the UMCIRB regardless of the funding source. 21 CFR 50 and 21 CFR 56 are applied to all research studies under the Food and Drug Administration regulation. The UMCIRB follows applicable International Conference on Harmonisation Good Clinical Practice guidelines.