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ABSTRACT

RESTING MECHANOMYOGRAPHY BEFORE AND AFTER AN ACUTE BOUT OF SINGLE LEG RESISTANCE TRAINING

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Mechanomyography (MMG) is the recording of low frequency vibrations along the axis of the axis of the muscle that are detectable at the level of the skin, while electromyography (EMG) detects the electrical action potentials generated by muscles. Previous literature has suggested that resting muscle is inactive due to lack of a resting EMG signal. However, work with sensitive EMG equipment and recent work with MMG during rest has shown resting muscle activity. While the reason for resting muscle activity is largely unknown, it is believed that the activation may have a neural origin. The purpose of the present study is to investigate resting MMG amplitude in the vastus lateralis muscles prior to and following resistance training, and to investigate if a cross-over effect occurs in the non-exercised leg. Ten healthy, college-aged males were measured for resting MMG amplitude 5 minutes before and 30 minutes following an acute bout

of resistance training. The study used two, randomly-ordered visits: a control visit and an exercise visit consisting of an acute bout of resistance training involving six sets of ten repetitions of unilateral leg extensions involving the dominant leg at 65% of their 1 RM max, with 1-min of rest between sets. The results showed no statistically significant differences ($p<0.05$) between resting MMG amplitude for the exercise versus control visits or for the dominant versus non-dominant vastus lateralis muscles. However, taking into consideration the small sample size, visual inspection, and calculation of effect sizes shows the possibility of a decrease in MMG amplitude in the dominant leg following exercise. This suggests that a relaxation effect may occur following resistance exercise.

CHAPTER 1: INTRODUCTION

1.1 Introduction

In 1810, Wollaston demonstrated that contracting muscles produce low frequency sounds [1]. However, muscle studies for the next century were primarily performed with electromyography (EMG), until the work of Gordon and Holbourn [2], who assessed muscle sounds using a new recording "microphone" to listen to what they referred to as muscle sounds. Using the recording microphone in conjunction with EMG, Gordon and Holbourn determined that the sounds generated from the orbicularis oculi were the mechanical counterpart of the electrical signal from the muscle [2]. However, despite this initial breakthrough, very little research was conducted on muscle sounds until 1980 when Oster and Jaffe, using a new sensitive recording device, found that sound amplitude increased with increasing muscle contraction intensity [4]. The works of Oster and Jaffe led to a revival of muscle sound studies.

Since this renewed interest in muscle sound studies by Oster and Jaffe, we now know that contracting muscle produces low frequency vibrations along the long axis of a muscle that are detectable at the level of the skin over the muscle belly [2,3,5]. The activity picked up at the surface of the skin has been referred to as the mechnomyographic (MMG) signal [3,5,6]. MMG activity is the mechanical counterpart to the electrical activity of motor units that is recorded by electromyography (EMG) [2,3,5,6]. The low frequency vibrations are believed to be

generated by the following mechanisms: 1) gross lateral movements created by non-simultaneous activation of muscle fibers at the initiation of contraction, 2) smaller subsequent lateral oscillations at the resonant frequency of the muscle, and 3) dimensional changes of the active muscle fibers [3,5,7,8]. MMG amplitude can be affected by several factors, including: intramuscular pressure, fiber type composition, muscle temperature, stiffness, viscosity of intracellular and extracellular fluids, the firing rates of the active motor units, and the type of muscle actions being performed (concentric vs. eccentric vs. isometric) [3,5,8,10,11].

Very little research has been done on resting MMG, since EMG assessment of resting muscle has generally led researchers to believe that resting muscle is inactive [6]. However, work with sensitive EMG equipment with protective shielding from outside electrical sources has provided some evidence of resting muscle activity, while other studies have shown no changes in EMG activity [6,9]. The reason(s) behind these discrepancies is uncertain, however it could be due to: the sensitivity of available EMG equipment, the percentage of the total number of fibers contracting at rest, and the distance of the active muscle from the sensor [12].

Recent studies have also shown MMG activity in resting muscle [12]. McKay et al. demonstrated an increase in resting MMG activity above baseline values following resistance training and aerobic training sessions [6,12,14]. This post exercise increase in resting MMG activity decays with a time constant that mirrors VO_2 kinetics [12]. However, the mechanism behind why muscles are mechanically activated after exercise is largely unknown. When neuromuscular blockers and

anesthetics are used, research has shown a decrease in MMG activity, suggesting the possibility of a neuromuscular mechanism [10,15,16]. Another possible explanation for increased resting activity is due to contraction of muscle fibers surrounding the vasculature in the muscle to help return blood to the heart, further increased to reduce the pooling of blood post-exercise [17].

Resting muscle mechanical activity may be an important physiological event to study as it may explain one of the reasons why oxygen consumption is increased, as well as if this phenomenon is indeed neurological in nature. Thus, the aim of this study is to investigate the increase in resting muscle MMG activity before and after completion of a resistance training protocol.

1.2 Purpose of the study

The purpose of this study is to investigate whether resting muscle mechnomyographic activity increases in the vastus lateralis of the exercised and non-exercised limbs following a bout of resistance training. Resting muscle was previously believed to be inactive due to a lack of EMG activity [16]. However, early studies with sensitive, electrically shielded EMG indicate that there is some resting electrical activity, thus suggesting that mechanical activity is present [10]. Several studies performed by McKay et al. have found increases in post-exercise resting MMG activity following resistance and aerobic training [12,14]. This research group found a three-fold increase in resting MMG amplitude after a bout of resistance training, and a decay curve that closely matched VO_2 changes post-

exercise. However, no significant EMG activity was detected in the resting muscle [12].

The mechanism responsible for the increase in resting MMG amplitude is largely unknown. McKay's group hypothesized a neuromuscular mechanism, due to a reduction in resting MMG activity following use of a neuromuscular blocker [13-15]. Another theory is that fibers surrounding veins contract to reduce pooling of blood after exercise [17]. If activity is indeed of a neural origin, then a potential crossover effect may be present in the non-exercising control leg.

1.3 Research Questions

The research questions in this study include:

RQ1: Does resistance exercise increase resting muscle mechnomyographic activity relative to baseline values?

RQ2: Does a cross-over effect occur in the non-exercised leg?

1.4 Research Hypothesis

H_R1: Resting muscle mechnomyographic activity will increase relative to baseline following resistance training in the vastus lateralis.

H_R2: The non-exercised vastus lateralis will increase MMG activity relative to baseline values.

1.5 Null Hypothesis

$H_0$1: Resistance training will not increase resting muscle mechnomyographic activity relative to baseline values in the vastus lateralis.

$H_0$2: There will be no increase in resting MMG activity relative to baseline values in the vastus lateralis of the contralateral non-exercised leg.

1.6 Significance of the research problem

Resting muscle MMG activity increases following resistance training and decays at a rate that correlates with VO_2 kinetics [12]. However, there is conflicting evidence with EMG studies related to resting muscle mechanical activity at rest, due to the lack of EMG activity at rest. The mechanism behind resting muscle MMG activity is also uncertain, and little work has been done in this field. Work by Mckay et al. has lead to the notion that the mechanism is neuromuscular in origin [13,16]. By blocking nervous function via an anesthetic, they found a reduction in MMG activity, indicating that this event is neurologically mediated [18]. Another possible theory is that the muscle fibers surrounding the veins within the exercised muscle contract to help return blood to the heart and prevent pooling, which would lead to increased MMG activity due to the signals produced by the contracting fibers around the veins [17].

Studying resting MMG may help explain the reason for excess post-exercise oxygen consumption. Also, if this mechanism is indeed neurological in nature, there could be a crossover effect observed in the contralateral non-exercised leg, which could help identify the phenomenon behind resting muscle mechanical

activity.

1.7 Delimitations of the Study

Delimitations of the study include:

- A control trial will be used to determine experimental treatment effects
- Subjects will be familiarized with experimental procedures.
- Subjects will consist of males aged 18-35 years, regardless of training status.
- Individuals with a known neuromuscular disease and/or a disorder that impairs normal body function will be excluded
- Individuals with a history of a lower limb injury in the tested leg will be excluded.
- Only one muscle group will be assessed (vastus lateralis)
- Subjects will be told to not perform moderate to heavy exercise at least 24 hours before testing

1.8 Limitations of the Study

Limitations of the study include:

- Subjects will be conveniently sampled and may not be representative of the population.
- Subjects may not refrain from physical activity 24 hours before testing time.

- Subjects may not exert maximal effort during the muscle actions
- Training status (untrained vs. trained) may have an effect on resting MMG activity.

1.9 Assumptions of the Study

Assumptions made during this study include:

- Subjects will answer the health questionnaire honestly and accurately
- The MMG sensor and all equipment will be properly calibrated between and across all measurements
- The MMG sensor will be accurately placed over the vastus lateralis according to the recommended guidelines
- The resistance training protocol used in the study will adequately stimulate the vastus lateralis
- Subjects will adhere to the training protocol
- Subjects will perform the training protocol with maximal effort

1.10 Operational definitions:

- Accelerometer: a device used to measure mechanomyogram signals by recording the oscillations produced by contracting muscle
- Dynamic resistance training (isotonic) – external tension remains constant throughout the entire range of motion.

- Electromyography (EMG) – recording technique measuring electrical signals produced by contracting muscles [19].
- Excess Post Exercise Oxygen Consumption (EPOC) – increased oxygen consumption above baseline values following strenuous exercise [20].
- Excess Post Exercise Resting Mechnomyographic Activity (EPERMA): Post-Exercise MMG activity above resting values [6].
- Mechanomyography (MMG) – recording technique measuring low frequency vibrations along the long axis of a muscle [3,5,8]. Mechanical component of motor unit electrical activity [8].
- One repetition maximum (1-RM): maximal amount of force that can be generated in one maximal contraction.
    - VO_2 – Oxygen consumption, rate of oxygen usage by the body [20].

CHAPTER 2: REVIEW OF THE LITERATURE

2.1 Introduction

The purpose of this study is to investigate if resting muscle has increased mechanical activity following an acute bout of lower body resistance training when compared to baseline. A secondary purpose is to determine if this pattern for resting activity is different between the exercised and non-exercised limbs. Resting muscle was previously believed to show no mechanomyographic (MMG) signal, thus being mechanically inactive due to the lack of electromyography (EMG) activity. However, this is contradicted by early studies with sensitive EMG with electrical shielding, which have shown resting muscle activity [9,13].

In the past, studies with mechanomyography (MMG) have mostly been focused on muscle during active contraction, rather than during resting states [3,8, 12]. However, recently MMG studies have suggested that resting muscle is mechanically active both before and following aerobic and anaerobic exercise. Several MMG studies that used spinal anesthetics to paralyze the muscles have found that the muscle activation is likely neurological in nature. However, the phenomenon behind why the muscle is being activated is currently unknown. Thus the aim of the current study is to investigate if MMG activity is increased in resting muscle (relative to baseline) following an acute about of resistance training in the lower limbs.

This chapter will serve as a review of previous research related to utilizing

mechanomyography on resting muscular activity and identifying gaps in the research area. This will be accomplished by presenting studies.

2.2 Literature Related to the Research Problem

De Vries H. (1964)

The purpose of this study was twofold: 1) to provide evidence of very low level of electrical activity recorded by surface EMG overlaying muscle and 2) to determine if postural muscles are electrically silent at a sensitivity level approaching the lower level of electrical noise generated by electrical equipment [9]. EMG recordings were made on five postural muscles: anterior tibialis, gastro-soleus, quadriceps femoris, hamstrings, and erector spinae while subjects stood in an easy standing position and in a relaxed state [9]. The recordings were made on 10 subjects for the anterior tibialis and gastro-soleus, eight subjects for erector spinae, and 15 subjects for the quadriceps and hamstrings [9]. EMG recordings were made with sensitive EMG equipment that is able to detect potentials to levels down to 1.0 μv. Voltage amplitudes were integrated with precision of ±.05 μv at 1.0-μv rms and ±.01 μv at 10- μv rms level [9]. A correction factor was utilized to correct for thermal noise, electrical noise, and amplifier noise. The researchers found significant linear relationship between action potentials at low levels after adjusting for correction factors, suggesting that the smallest potentials recorded from the muscle were most likely muscle action potentials. The results indicated during normal resting conditions, the muscles were not electrically silent (Mean μv

during rest; Gastro-soleus = 0.48; Anterior tibialis = 0.40; quadriceps = .16; Hamstrings = .08). During active standing, EMG activity increased (Mean μv during standing; Gastro-soleus = 5.44; Anterior tibialis = 5.84; quadriceps = 1.46; Hamstrings = 1.12) [9]. The erector spinae and hamstrings group had significantly higher levels of activity when comparing easy standing and rest (Mean μv: hamstrings: rest = .08, standing = 1.12; erector spinae: rest =5.58 and 8.65, standing = 11.32). It was concluded that postural muscles demonstrate electrical activity and the lack of activity found in previous studies may be due to 1) lack of sensitive equipment, 2) poor sampling and, 3) failure to correct for electrical activity generated by amplifier and thermal noise [9].

Joseph J., Nightingale A., and Williams P. L. (1955)

The purpose of this study was to investigate electric potentials over postural muscles while relaxed and during normal posture using sensitive EMG equipment with high amplification. EMG signals were recorded over the quadriceps femoris, hamstrings, tibalis anterior, gastrocnemius, soleus, tibia, and patella in eleven healthy males (18-22 years) while passively standing and while lying on a bed in a relaxed position with the knee and hips slightly flexed. EMG recordings were made with sensitive recording equipment using high amplification and a noise level of 2μV peak to peak. The highest amplification was adjusted to allow amplifier noise to be evidently visible. Both the amplifier and subject were placed in an enclosed screening cage to reduce outside interference. The results indicated

that relaxed muscle demonstrated the following amplitude deflections: 1) background noise up to 2μV present in all recordings, 2) larger potentials between 3-5 μV for an average of 40-50s and, 3) longer duration deflections between 10 to 20μV. The researchers excluded these deflections due to being caused by polarization potentials at the skin-electrode surface point. Similar results were found during the tibia and patella resting measurements. Results during passive standing indicated greater activity in the soleus, gastrocnemius, and tibialis anterior when compared to rest. The quadriceps femoris results were similar to relaxing muscle, while the hamstrings showed no activity while standing. The authors concluded that resting muscle is electrically silent due to the same level of electrical activity observed over the tibia and patella. The researchers hypothesized that if these potentials were generated by motor unit activity, then contraction of fibers of one ore more motor units would yield a larger potential [21]. The researchers also concluded that these potentials probably originated from thermal noise, such as irregular flow of ions through vessels [21].

McKay W, Gregson P, McKay B, and Blanchet T (1998)

The purpose of this study was to investigate if resting muscle in the arm and forearm is mechanically active via MMG, and if this mechanical activity decreases following induction of anesthesia and a neuromuscular junction blocker [13]. The researchers believed that if sound arose, it could be from either contractions due to nerve stimulus, or sound independent of nerve stimulus.

Twenty-one adults aged 16-24 years who were undergoing surgery unrelated to the upper arms and who were administered anesthesia and a neuromuscular blocker, gave consent to undergo MMG measurements. Of these twenty-one volunteers, 10 performed the biceps study (demographics lost) and 12 subjects, 4 female and 8 males (mean ± SD; 50 ± 14 years, 168 ± 10 cm, 72 ± 16 kg), performed the forearm study. MMG recordings were made by a Bruel & Kjaer accelerometer (DK-2850, Naerum, Denmark, frequency 0.2 Hz to 20 kHz) and placed over the belly of the biceps brachii and forearm muscle [13].

Recordings were taken for 30s in the following four stages. (i) Lifting a 2kg weight, (ii) removing the weight and allowing relaxation, (iii) inducing the sodium thiopental anesthesia (5mg/kg), and (iv) after induction of vecuronium, which induces muscle paralysis. Measurements were taken in the same fashion during the forearm study, with the exception of a blood pressure cuff cutting off blood pressure pulsations and choice of muscle relaxant.

The results showed a significant difference in all bicep stages ($p<0.05$), while in the forearm study, only stages (i) and (iv) were significantly different ($p<0.05$). There was also no significant difference ($p>0.05$) in MMG frequency between the forearm and bicep groups. The researchers concluded that resting muscle produces low frequency vibrations that can be detected by MMG and disappear with induction of paralysis [13]. This suggests that MMG signal is neural in nature and the use of paralyzing factors decreases resting muscle activity [13].

2.3 Studies investigating resting MMG during aerobic training.

McKay W, Chilibeck P, Chad K, Daku B (2004)

This study examined whether muscle is mechanically active following aerobic exercise and, if so, how this activity correlates with O2 consumption. The researchers hypothesized that muscle will be mechanically active and may be a factor contributing to excess post exercise oxygen consumption [14]. Ten moderately fit male subjects (mean ± SD; 22.9 ± 2.3 years, 179.1 ± 6.3 cm, 84.2 ± 13.2 kg, Vo2 max = 50.3 ± 7.7 ml • kg $^{-1}$ min $^{-1}$) volunteered to perform 30 minutes of cycling at 70% of their VO_2 max. Muscle mechanical activity and electrical activity was measured via MMG and EMG sensors placed over the rectus femoris during rest [14].

The program consisted of a 30-minute cycle test corresponding to 70% of their VO2 max. Subjects began by warming up at 50% of their VO2max for 5 minutes before beginning the 30-minute stage. Immediately after the cycling test, subjects were to remain sedentary (reading, sitting quietly, watching TV, or studying). Subjects were to remain in a fasted state during this period. MMG and EMG measurements were taken over the rectus femoris, and were measured 2 minutes post exercise and at 30 minute intervals for 90 minutes. After the 90 minutes, MMG and EMG recordings were taken hourly (15 minute each period) for a total duration of 5.5 hours [14]. Subjects were to remain sedentary during these measurements. Maximal oxygen consumption rate measurements were taken prior to the testing protocol during a graded exercise test on a cycle

ergometer to determine each subject's VO_2 max. O_2 uptake was measured every 20 seconds during the test, as well as during the resting MMG and EMG measurements. Subjects also had their RER, VO_2, and VCO_2 measured to determine when steady state had been achieved [14].

The results indicated that post exercise resting muscle activity significantly increased ($p<0.0001$) by 295% and remained elevated for the 5.5 hour measurement time ($p<.05$). MMG amplitude decayed at a similar time constant (τ = 7.2 minutes) as VO_2 kinetics (τ = 7.4 minutes). There were no significant differences between normalized pre-exercise and post-exercise values between EMG amplitude, EMG frequency, and MMG frequency. The researchers concluded that resting muscle is mechanically active following aerobic training, and that it may be a contributing factor to excess post exercise oxygen consumption [14].

Malek M, Coburn J, Housh T, Rana S. (2011)

The purpose of this study was to investigate the relationship between MMG activity and excess post-exercise oxygen consumption (EPOC) following incremental cycle ergometer exercise in the quadriceps [22]. Twelve adult males (mean ± SD; 22.6 ± 0.9 yrs, VO_2 Max 42.8 ± 1.95 ml/kg/min) volunteered to perform an incremental cycle test to exhaustion using a cycle ergometer. The maximal cycle ergometer test consisted of cycling at 50W for two minutes and increased by 30W every two minutes until completion of the test. MMG and EMG measurements were recorded over the vastus lateralis, rectus femoris, and vastus

medialis 30 minutes before, 8-12 minutes during the test, and for 60 minutes after the incremental cycle test. EPOC measures were taken both before and after the incremental cycle test [22]. The results showed that significant τ values for VO_2 (τ = 14.0, R^2 = 0.954) and for MMG amplitude for all three muscles (vastus lateralis, τ = 4.8, R^2 = .0944; rectus femoris, τ= 4.8 R^2 = .944; and vastus medialis τ= 4.6 R^2 = .943) during the 60 minutes of post-exercise recovery. EMG showed no significant decay patterns for all three muscle groups during the 60-minute recovery. A one-way repeated measures ANOVA indicated that a significant mean difference between τ values for VO_2 and MMG amplitude existed, however no significant difference existed between the τ value and all three muscle groups. The MMG amplitude for the rectus femoris that was the only muscle that significantly correlated with VO_2 (r=.57) [22]. The researchers concluded that increased MMG activity following exercise does not completely explain or correlate with increased oxygen consumption following exercise [22].

2.4 *Studies investigating resting MMG during resistance training*

McKay W, Jacobson P, Chilibeck P, Daku B (2006)

The purpose of this study was to determine if there is a minimum threshold necessary to induce post exercise resting muscle activity as demonstrated by mechanomyography (MMG). A secondary purpose was to determine if a relationship exists between exercise level and resting muscle activity. A third purpose was to determine if a cross-over effect occurs. A final purpose was to see

how different muscle lengths affect MMG activity [5]. Ten (6 males, 4 females) healthy, moderately fit individuals (Mean ± SD; 33 ± 13 years, 176 ± 8 cm, 75.6 ± 13 kg) volunteered to perform isokinetic leg extensions in sets consisting of 1, 5, 10, 20, and 30 repetitions on a Biodex dynamometer at 60 °/s. MMG measurements were taken via an accelerometer placed over the mid-rectus femoris of both the left and right legs in both the flexed and extended positions [6].

Prior to testing, the MMG sensor was calibrated for each subject. During the protocol, each subject was placed in the dynamometer and told to relax. Once the subjects were seated, recordings were made prior to beginning exercise. Recordings were performed during two-minutes (12 second intervals) while the leg was extended and another two minutes with the leg flexed. During exercise, MMG recordings were made immediately before and after each set of the exercise test. After exercise, recordings were made immediately at the conclusion of exercise with the subject relaxed, legs flexed, and legs extended [6].

The researchers found no significant exercise threshold needed to induce muscle activity. However, an earlier study found an increase in muscle activity following enforced rest. The researchers also found a significant increase in MMG amplitude in the right leg only after the set consisting of 30 repetitions. Additionally, a linear correlation existed between MMG amplitude and total work performed in both the right and left legs (Right: R = .61; Left R = .67). The non-exercised leg, increased in MMG activity by half the amount of the exercised leg demonstrating a cross over effect. The researchers theorize the evidence of a

crossover effect suggests a neural origin behind MMG activity at rest [6]. A positive correlation also existed between the two exercised legs ($r=.62$ $p<0.01$). The researchers also found greater MMG activity when the leg was extended (shorter length), possibly due to the shorter length creating a more compliant muscle-tendon unit for detection of oscillations by MMG [6]. The researchers concluded that the higher resting MMG in the short muscle was due to a more compliant musculotendinous unit [6].

McKay W, Chilibeck P, Daku B (2007)

This study examined mechanomyographic (MMG), electromyography (EMG), and oxygen consumption in resting muscle before and after an acute bout of resistance training [12]. Ten males (mean ± SD; 22.3 ± 2.3 years, 179 ± 6 cm, 84 ± 13 kg) volunteered to perform 30 minutes of resistance training, consisting of the following: (1) one set of ten reps at a weight equal to 50% 1-RM for both leg press and leg extension and (2) five sets of eight reps at a weight equal to 75% 1-RM for both leg press and leg extension [12]. Subjects were given one-minute of rest between each exercise set [12].

A metabolic cart (VMAX 29 series, Sensormedics, Yorba Linda, CA) was used to measure O_2 consumption and CO_2 production rates. MMG activity was measured by an accelerometer (B&K #4381), amplified by a B&K charge amplifier, and recorded by a Vetter #3000 recorder. EMG activity was measured via a bipolar electrode arrangment. MMG amplitude was expressed in terms of

mean absolute value. Arterial pulses were factored out to prevent disruptions in the signal. MMG frequency was also detected from the power spectrum of the MMG signal [12].

A preliminary visit was used to determine leg press and knee extension 1-RM. The next visit consisted of the resistance training protocol, with EMG and MMG measures taken immediately following the training. Prior to this, the subjects remained rested in a room for 30 minutes while RMR was measured 5 times until a steady state was achieved. After achievement of this steady state, the subjects performed the resistance training protocol. EMG, MMG, and metabolic measures were taken during 15 minute periods, at 30 minute intervals for 90 minutes, and hourly for 5.5 hours following exercise, while subjects remained relaxed with little to no movement [12].

The researchers found that MMG activity significantly increased (pre, 3.0 ± 0.99 mm • s^{-1} to 10.1 ± 4.5 mm • s^{-1}; p=0.001) following the exercise protocol, which corresponded to an average increase ranging from 1.8 to 7.7 mm • s^{-1} for all subjects [12]. EMG activity also increased following exercise (p=0.03). However, all but four values exceeded the lower detection resolution of the equipment. The researchers also found that MMG amplitude and VO_2 decayed at a similar time constant (7.5±2.2 and 7.2 ±1.0) [12]. In conclusion, the researchers found an increase in resting muscle MMG activity following exercise. MMG activity also decayed with a time constant similar to VO_2 kinetics, indicating it may play a role in excess post exercise oxygen consumption. The researchers also found an

increase in resting muscle EMG activity, however, the instruments were not sensitive enough to detect it. The authors believed that the lack of EMG activity may be due to a relatively small number of muscle fibers contracting at a given time, and these fibers are spread throughout the muscle, which would not cause a strong enough signal to be detected [12].

Wages N, Beck T, Ye Xin, Hofford C (2013)

This study investigated resting mechnomyographic (MMG) activity in the erector spinae and trapezius muscles following resistance exercise [23]. Twenty males (mean ± SD; 21 ± 1.6 y, 178.6 ± 6.4 cm, 84.3 ± 13 kg) performed three sets of ten reps of the deadlift, bent-over row, and lat pull-down exercises[23]. MMG activity was measured with an accelerometer (Entran EGAS FT-10, Measurment Specialties, Hamton, VA) and placed over the erector spinae and lower trapezius. The MMG signals were sampled at a rate of 1000 samples/sec and digitized with pass frequencies between 5 and 100 Hz [23]. Recordings were made prior to exercise and 30 minutes following exercise in 10-second intervals [23].

The researchers found a 10% decrease in resting normalized MMG amplitude in the erector spinae and a 15% decrease in normalized MMG amplitude in the trapezius. The authors concluded that there was a decrease in MMG amplitude following a moderate bout of resistance exercise, indicating a relaxation effect that may be useful in treatment of back pain [23].

2.5 Studies investigating resting MMG relation to oxygen

consumption

McKay W, Lett B, Chilibeck P, Daku B (2009)

The purpose of this study was to investigate if paralyzing the muscles below the waist reduced resting metabolic rate (RMR) and resting MMG. The authors believed that if resting MMG is indeed neurological in nature and relates to O_2 consumption, then paralyzing the muscles should decrease following induced paralysis [15]. Ten subjects (7 female, 3 male; mean ± SD; 55 ± 8.6 years, 166 ± 11 cm, and 84± 16 kg) who were undergoing surgery and having a spinal anesthetic were utilized in the study. MMG recordings were collected with an accelerometer (Bruel & Kjaer model #4381 accelerometer) and amplified (B&K model #2635 amplifier). The accelerometer was placed over the mid-point of the quadriceps. MMG recordings were expressed in mean absolute acceleration units, and oxygen consumption and VO_2 measures were recorded with K4B2 indirect calorimetry system (COSMED Sr1, Rome). Electrocardiograms (ECG) were also used to assess cardiac rhythm. Prior to undergoing surgery, each subject had RER, MMG, and ECG measured for 2 minutes before entering the surgery room. Subjects were then measured for 20 minutes while being administered the anesthetic. MMG, ECG, and metabolic measurements were recorded both before paralysis (2 min) and during paralysis (20 min) to determine if paralysis reduced RER and MMG activity during rest [15].

The researchers found decreases in oxygen uptake and mean MMG amplitude of 25% and 37%, respectively, following induction of the spinal

anesthetic [15]. The decrease in O_2 consumption correlated with the decreasing MMG activity (R= .624) [15]. The researchers concluded that metabolic rate and MMG activity both decreased following induction of a muscle-paralyzing factor. They believed that a number of factors could lead to this decrease, including: (1) an anxiety-related increase in breathing, (2) an increase in organ work, (3) cellular metabolism, and (4) resting muscular mechanical work [15]. However, the authors also believed the latter to be the case due to the relationship between reduced mechanical activity and RMR. Although more work in the area needs to be done, the researchers concluded that the resting muscle activity is indeed likely neurological in nature [15].

Mckay W, Chilibeck P, Daku B, Lett B (2010)

The purpose of this study was to determine how much resting muscular activity contributes to overall metabolic activity by measuring acceleration of the leg and MMG activity before and after induction of a muscular paralyzing agent. The researchers believe that a paralyzed leg will fall slower than the unparalyzed leg, and from this, force and power can be determined and the amount it contributes to metabolic activity [16]. Four questions were investigated in this study, including: (1) how much force is related to MMG activity, (2) how much force is produced by resting muscular activity, (3) how much energy is produced by resting muscle activity, and (4) is this a significant source of calorie consumption [16].

Ten subjects (8 F, 2M; Mean ± SD; 47 ± 11 years, 166.8 ± 9.4 cm, 77.4 ± 15.8 kg) participated in the study. All subjects were undergoing general surgery unrelated to the lower right leg and under general anesthesia. Each subjects' right leg was placed into a strap load cell (Interface Model SML-5). MMG activity was detected by a Bruel & Kjaer #4381 accelerometer (B &K, Naerum, Denmark), amplified (B&K #2635 amplified), and calibrated by a B&K charge amplifier set to .2-100 Hz. An MMG accelerometer was placed over the mid point of the rectus femoris to record muscle activity [16].

Each subject underwent a Wartenberg test, which allows a supported object to free-fall under the force of gravity, before and after induction of general anesthesia. Acceleration was determined for the free falling leg, and then used to calculate muscle force and power by subtracting the acceleration of free-falling leg pre- from that during the post-muscle paralysis [16].

The researchers found a significant increase in acceleration after induction of paralysis (7.65 ± 1.51 m s^{-2}) compared to pre-paralysis (6.99 ± 1.51 m s^{-2}) and a decrease in MMG activity after induction of paralysis (4.2 ± 2.6 mm s^{-2}) compared to pre-paralysis (10.6 ± 3.7 mm s^{-2}) [16]. Force produced by the resting quadriceps equaled 22.6 ± 16.8 N with a power of 0.34 ± 0.17 W [16]. Calculated daily caloric expenditure for this muscle equaled 7.0 ± 3.6 kcal day^{-1} and 205kcal day^{-1} of total skeletal muscle caloric expenditure due to resting muscle [16]. The researchers believed that due to the nature of resting muscle activity, research into resting muscle activity may help us understand some aspects of resting metabolism [16].

2.6 *Studies investigating effects of temperature on resting MMG*

Mckay W, Vargo M, Chilibeck P, Daku B (2013)

This study investigated whether resting muscle is mechanically active in order to maintain body temperature with decreasing ambient temperature [18]. Twenty subjects (7 F; 29 ± 10.6 y, 1.6 ± .1 m, 61.3 ± 10.8 kg, 13M; 26.1 ± 6.1 y, 1.8 ± .1 m, 83.3 ± 16.1 kg) volunteered to lay down, rest, and be cooled from 40° C to 12°C over a time course of 65 minutes [18]. Cooling subjects had the following recorded: (1) temperature, (2) mechanomyography (MMG), (3) electromyography (EMG), and (4) VO_2. MMG was recorded with a Bruel and Kjaer accelerometer (4381 B &K, Denmark), amplified between 2-100 Hz, and sampled at 1000 Hz. The MMG accelerometer was placed over the midpoint of the anterior thigh [18]. Surface EMG was taken with a Delsys bipolar electrode (Delsys Inc. Boston, Mass) placed near the MMG accelerometer. Subjects were equipped with a Cosmed K4B2 indirect calorimtry system for metabolic measurements.

After having fasted for 2-4 hours, the subjects laid down supine wearing shorts and a t-shirt while ambient temperature was lowered by .5°C until 7.5°C was reached, or until subjects began shivering [18]. During this time EMG, MMG, temperature, and metabolic activity were recorded.

The researchers found a significant increase in both MMG and EMG when ambient temperatures reached 21.5°C [18]. When comparing men to women,

women achieved a significant increase in MMG amplitude at a higher temperature (25°C) compared to men (17°C), indicating that women regulate temperature better than males [18]. Resting muscle MMG activity was correlated with oxygen consumption (R =.65 p =.01), indicating a potential relationship between the two variables [18]. In conclusion, the researchers believed that resting muscular mechanical activity might play a role in regulating temperature [18].

2.7 Summary of the literature

The referenced literature helps us understand the current research in the area of resting muscle activity as detected by mechanomyography. It also illustrates how little research has been performed in this field, and the lack of knowledge of why the muscle is being activated.

Resting muscle was previously believed to be mechanically inactive due to an apparent lack of electrical activity. However, in 1965, De Vries used sensitive EMG equipment with high amplification and electrical shielding to show the presence of electrical activity in resting muscle [9]. These findings disputed those of Joseph, who, in 1955, found similar EMG amplitude in resting muscle as that in bony prominences. Joseph concluded the small nature of the marked potentials were not large enough to be considered motor unit activity, and attributed the potentials to irregular ion flow [22].

Mckay et al. (1998) performed the first study investigating whether resting muscle is mechanically active [13]. This group examined mechanomyography in

the biceps and forearm, both before and after a local anesthetic as well as and after lifting a small weight. They found that resting muscle is indeed mechanically active, and this activity diminishes with induction of a neuromuscular blocker [13].

Resting muscle mechanical activity also increases following resistance training and aerobic training [6, 12-14]. Mckay et al (2004) investigated resting muscle mechanical activity following 30 minutes of aerobic exercise at 70% of VO_2 peak [14]. The researchers measured resting muscle activity by MMG, as well as oxygen consumption before and for 5.5 hours following exercise [14]. They found an increase in MMG activity, relative to baseline, following vigorous aerobic exercise, as well similar decay time constant with that of the oxygen consumption rate [14]. Mckay et al (2006) investigated graded levels of exercise on resting muscle mechanomyography, the cross over effect, and the effect of muscle length on MMG activity [6]. The researchers found no definite threshold for MMG activity, a linear correlation between resting MMG activity and workload, a cross-over effect, and increased resting MMG when the muscle was shortened [6]. Mckay et al (2007) investigated resting MMG, EMG, and VO_2 before and after a vigorous bout of lower body resistance exercise [12]. A significant increase in MMG and EMG was observed relative to baseline, however EMG amplitude was below the resolution of the instruments [12]. The researchers also found similar decay time constants between VO_2 and MMG over the 5.5h duration. Wages et al (2013) investigated resting MMG following back resistance training [23]. This study found a decrease in MMG indicating a relaxation effect [23].

The mechanism and reasons for why resting muscle is activated is still not completely understood. However, several studies have investigated the mechanism of resting muscle activity using neuromuscular blockers and general anesthesia [14-17]. These studies found a decrease in MMG activity following induction of anesthesia and a neuromuscular blocker, indicating that resting muscular activity is neurological in nature [12,14-17]. Mckay et al (2013) investigated a possible mechanism as to why resting muscle is mechanically active following cooling. They theorized that resting muscle was mechanically active in part to regulate temperature. Another possible theory is that MMG is detecting contracting muscle fibers surrounding the veins, thereby pushing blood back to the heart following exercise [14,16,17].

Overall, there are several gaps in knowledge and a lack of clear understanding when it comes to resting muscle activity, both before and following exercise. One such gap arises when looking at VO_2 kinetics and MMG activity following resistance training and aerobic training. Aerobic training reaches peak MMG activity immediately following training, while peak MMG activity is delayed following resistance training. Another gap in the literature is the discrepancies between MMG activity and EMG activity. MMG activity is usually detected, but at times it seems EMG activity increases, but is below the resolution of the equipment. Although it seems that resting muscle activity is indeed neurological in nature, the reason behind why the muscle is being activated is unknown.

CHAPTER 3: METHODOLOGY

3.1 Introduction

The purpose of the present study is to investigate if resting muscle demonstrates MMG activity following an acute bout of lower body resistance training relative to baseline levels. Resting muscle was previously believed to show no MMG signal, due to EMG activity being absent during rest. However, this is contradicted by early studies with sensitive EMG equipment that used electrical shielding, which have shown resting muscle activity [9,11,21].

Previous studies with MMG have mostly been focused on muscle during active contraction, rather than during resting states [17,23]. However, recent studies with MMG have confirmed that resting muscle is mechanically active both before and following aerobic and anaerobic exercise. Several MMG studies working with neuromuscular nerve blockers and spinal anesthetics have pointed that the muscle activation is likely neurological in nature. However, the phenomenon behind why the muscle is being activated remains mostly unknown. Thus, the aim of the current study is to investigate if MMG activity is increased in resting muscle (relative to baseline) following an acute about of dynamic resistance training in the rectus femoris. In addition, if it is active, are there differences between the exercised and non-exercised leg?

This chapter will serve to explain the materials, methods, and instruments utilized in the study. This chapter will include the following: (1) description of the sample, (2) instrumentation and measurement protocols, (3) research design, (4)

data collection procedures, and (5) data management and analysis.

3.2 Sample

Ten healthy male subjects (24.5 ± 4.25 yrs) participated in the study. Subjects were conveniently sampled from the University of Oklahoma, Norman campus, and included both trained and untrained males. All subjects were required to sign a written informed consent form and complete a health status questionnaire. To be eligible to participate in the study, the subject had to indicate no current no signs/symptoms of lower limb pain or previous lower limb injuries, no lower limb surgery, and no neuromuscular or musculoskeletal problems. Participants also had to refrain from physical activity at least 24 hours prior to testing. The University of Oklahoma's Institutional Review Board approved the study.

3.3 Instrumentation and Procedures

3.3.1 Familiarization

Each subject visited the laboratory on three separate occasions. The first visit included filling out consent forms/health status questionnaires, familiarization, and one repetition maximum (1-RM) testing. The purpose of the familiarization session was to let each subject become comfortable and familiar with the testing equipment and protocols used, as well as to minimize any potential learning effect. Upon arrival to the lab, each subject completed an informed

consent form and health status questionnaire while the researcher explained the study in detail. During this time, the subjects were free to ask any questions that they might have about the nature of the study. If the subject consented that he/she wanted to participate in the study, and he/she met all inclusion criteria, then he/she performed 15-25 unilateral leg extensions of the right leg, with no weight added on the leg extension machine. The subject's non-dominant leg was placed onto a raised platform to disallow movement. Following this warm-up procedure, the subjects underwent testing for their respective 1-RM. 1-RM was determined by the maximal amount that each subject could lift one time during a maximal leg extension within 2-3 attempts.

3.3.2 Testing and Control Sessions

The second and third visit consisted of the dynamic resistance training protocol and the control session, each separated by at least 48 hours. These sessions were randomly ordered. During the resistance training protocol, the subjects performed 10 reps at 65% of their 1-RM for 6 sets using the dominant leg, with one minute of rest between sets. Prior to these work sets, the subjects performed a warm-up of 10 submaximal leg extensions. For all repetitions, the subjects were seated upright in the leg extension machine with the dominant leg resting against a padded surface and the non-dominant leg bound in place to allow minimal movement. During the control session, the subjects sat rested on the leg extension machine in a similar fashion as the experimental visit without

performing any activity, for a duration identical to the testing session.

*3.3.3 Mechanomyography (MMG) Procedure*s:

Surface MMG sensors (Entran EGAS FT-10, Measurement Specialties, Hampton, VA) were placed over the mid anterior portion of the vastus lateralis as described by the SENIAM project. Sensor placement was determined by measuring the point two-thirds of the distance between the anterior superior iliac spine and the lateral side of the patella [24]. To ensure proper sensor placement, the leg was extended as the researcher applied pressure while palpating the muscle. Each MMG accelerometer was secured over the vastus lateralis by double-sided adhesive tape. Prior to placement, the MMG sensor placement sites were prepared by careful shaving and cleansing with rubbing alcohol to remove surface dirt and oils.

MMG measurements were taken during the control and resistance training sessions. During the control visit, MMG signals were recorded over the vastus lateralis of the exercised and non-exercised legs every 10 seconds for 30 minutes to achieve normal resting baseline values. During this time, the subjects were relaxed sitting in an upright position on a table with both legs extended. Subjects were free to read or watch television during the measurements. The initial resistance training measures were recorded once every 10 seconds for 5 minutes to achieve a baseline value to normalize the MMG amplitudes values. However, immediately after the subject performed their last resistance training set, the

subjects returned to the upright position with legs extended. During this time, MMG signals were recorded in the exercised and non-exercised vastus lateralis muscles every 10 seconds for 30 minutes in a manner similar to the control visit.

3.3.4 Signal processing:

All data were processed using a custom program built with LabVIEW (version 7.1, National Instruments, Austin, TX) programming software. All MMG signals were digitized at 1000 samples s^{-1} and, stored in a personal computer (Macintosh Macbook pro 2011, Apple Computer, Inc., Cupertino, CA) for further analysis. All MMG signals were band-pass filtered (fourth-order Butterworth) between frequencies of 5Hz and 100Hz. MMG amplitude (Root mean square) was calculated and normalized to control values and pre-exercise values.

3.4 Research Design

The study required a total of three separate visits to the research lab. The first visit included a familiarization session, 1-RM, and filling out health status questionnaire forms. The next two visits were randomized between the resistance training protocol and the control visit. The resistance training protocol consisted of performing single leg resistance exercise of the dominant leg for the purpose of fatiguing the vastus lateralis. Each visit was separated by a minimum of 48 hours. The study utilized a pre-test/post-test design, with each subject acting as his own control. All MMG data were normalized to pre-testing values.

3.5 Data Collection and Data management Procedures:

All data were collected, stored, and analyzed by the principal investigator. This data included subject demographics (age, height, weight), MMG amplitude values, and resistance exercise values (1-RM and reps completed). All data was encrypted, coded, and stored on a password-protected computer, which only the researcher had access to. Each subject was assigned a personal randomized code identifier so that the researcher did not know whose data belonged to which subject.

3.6 Statistical Analysis:

All statistical analyses were performed using SPSS version 19, using a critical alpha level of $p \leq 0.05$. MMG data were analyzed using a three-way (limb × visit × time) repeated measures analysis of variance (AVOVA) with Bonferroni post-hoc comparisons. When relevant, follow up one-way ANOVAs and paired sample t-tests were used. Finally, Cohen's d was used to determine effect size based on the interpretation of Cohen (1988) using .2 (small), .5 (medium), and .8 (large) effect.[25] Cohen's d was found by using M2= Dominant leg or Testing visit, M1 = Non-dominant leg or Control visit, and pooled standard deviation = standard deviation pooled at each time point. Cohen's d and pooled standard deviation were calculated using the following formula:

$$\text{Cohen's D} = \frac{\text{Mean Difference}}{\text{Standard Deviation}} \quad \text{Or} \quad \frac{\text{M2-M1}}{\text{Pooled Standard Deviation}}$$

$$\text{Pooled Standard Deviation} = \sqrt{[(s_1^2 + s_2^2) / 2]}$$

CHAPTER 4: RESULTS

4.1 Introduction

The purpose of the current chapter is to describe the results of the study. Initial data includes subject demographics and descriptive information. Next are the results of the three-way repeated measures ANOVA with post hoc comparisons. Finally, Cohen's d is reported to illustrate the effect sizes of the differences between visits and leg at each specific time point.

4.2 Results

4.2.1 Descriptive data

Table 1 illustrates subject characteristics defined by mean and standard deviations. A total of 10 subjects were used in the study, all of which were male participants.

Table 1:
Subject Descriptive Data (n=10)

Variable	Means ± SD
Age (yrs)	24.5 ± 4.3

4.2.2 Three-Way Mixed Factorial ANOVA for MMG Amplitude

The results from the three-way ANOVA indicated that there was a significant visit × leg × time interaction. . As described by Keppel (1991), decomposition of the three-way interaction is at the discretion of the researcher, since the simple effects can only be examined for two factors at any given time point. [26] Despite the finding that there was a significant three-way interaction, follow-up Bonferroni post-hoc comparisons indicated that there were no significant pairwise differences amongst the mean MMG amplitude values across any of the factors. It is highly likely that this result is due to the slightly underpowered nature of the study. Specifically, the study had enough power to identify a significant three-way interaction, but with the observed effect sizes, was slightly underpowered when conducting the pairwise comparisons. Figure 1 represents the mean normalized MMG amplitude values for the dominant and non-dominant vastus lateralis muscles across time during the control visit. Figure 2 expresses the same data, but during the testing visit. Figure 3 represents the mean normalized MMG amplitude values for the dominant leg across time during the control vs. testing visit, while figure 4 shows the same data, but for the non-dominant leg. All raw and normalized MMG amplitude data can be found in Appendix A.

4.2.3 Cohen's d effect size

Findings from calculation of Cohen's d are represented in tables 2-5. Table 2 reports the effect size of dominant vs. non-dominant legs across time during the control visit, while table 3 reports the same data during the testing visit. Tables 4

and 5 represent the effect sizes of the normalized MMG amplitude values for the dominant and non-dominant legs across time during the control vs. testing visits.

4.2.4 Interpretation of Figure 1 and table 2

Figure 1 represents normalized MMG amplitude for the dominant vs. non-dominant vastus lateralis across time during the control visit. The results from the 3-way repeated measures ANOVA showed no statistically significant ($p < 0.05$) differences between legs during the control visit. The x-axis on figure 1 represents the 30 minutes of post-exercise data collection. Time point 0 represents the 5 minutes of pre-exercise resting conditions normalized to 100%. By visual inspection, the non-dominant leg had an overall decrease in MMG amplitude during the 30 minutes of relaxation.

Table 2 represents the results from Cohen's d on effect size of the dominant vs. non-dominant MMG amplitude across time. The results indicate that there is a moderate effect at minute 20 ($d = 0.47$) and 25 ($d = 0.42$) and a moderate to large effect at minute 30 ($d = 0.62$). These results indicate that there was a moderate (although not statistically significant) decrease in MMG amplitude in the non-dominant leg compared to the dominant leg during minutes 20-30.

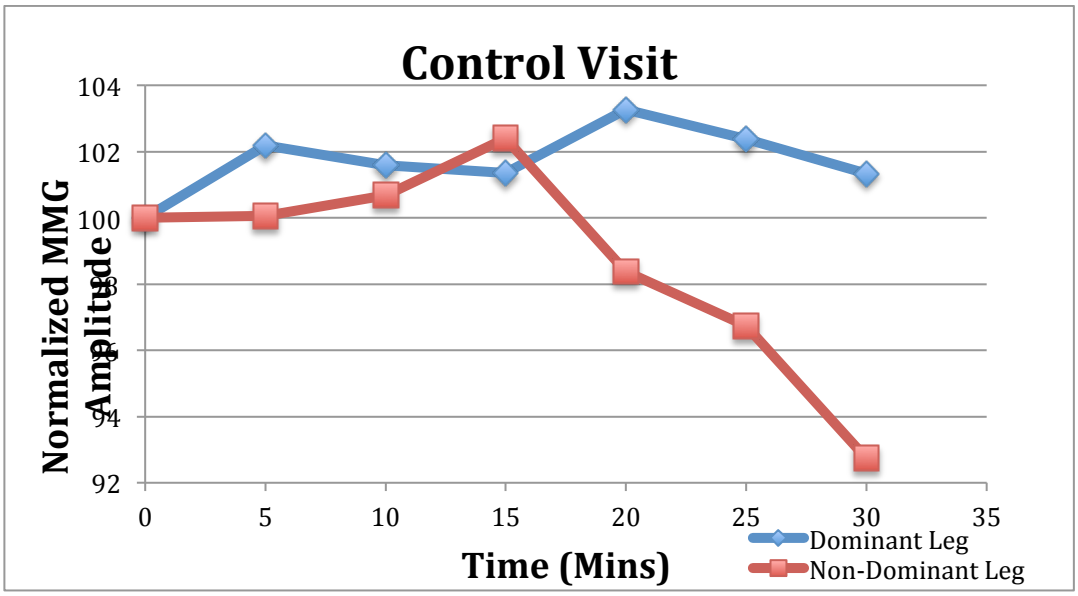

Figure 1. Vastus lateralis Dominant vs. Non-dominant MMG amplitude across time during control visit. Time point 0 indicates the 5 minutes pre-exercise resting conditions normalized to 100%.

Table 2. Effect Size of dominant vs. Non-dominant across time during control visit. A positive value indicates a larger MMG amplitude in the dominant leg in relation to the non-dominant leg during that time point. A negative value indicates a larger non-dominant MMG amplitude value in relation to the dominant leg.

Time	Effect Size
	N/a
Pre 0.5 minutes	
Post 5 minutes	0.187948782
Post 10 minutes	0.071490988
Post 15 minutes	-0.086132446
Post 20 minutes	0.471988018
Post 25 minutes	0.429087981
Post 30 minutes	0.629436707

4.2.5 Interpretation of Figure 2 and table 3

Figure 2 represents the mean normalized MMG amplitude values in the dominant vs. non-dominant vastus lateralis during the testing visit. The results

from the 3-way repeated measures ANOVA showed no significant statistically significant ($p > 0.05$) differences between legs during the testing visit.

Table 3 reports the results of Cohen's D effect size between dominant and non-dominant legs during the testing intervention. A small to moderate effect size occurred during minute 15 (d= -0.44) and a moderate effect size occurred between minutes 20 (d= -0.499) and 30 (d= -0.47). These findings suggest that the dominant leg had an overall decrease in MMG amplitude during minutes 15, 20, and 30 when compared to the dominant leg.

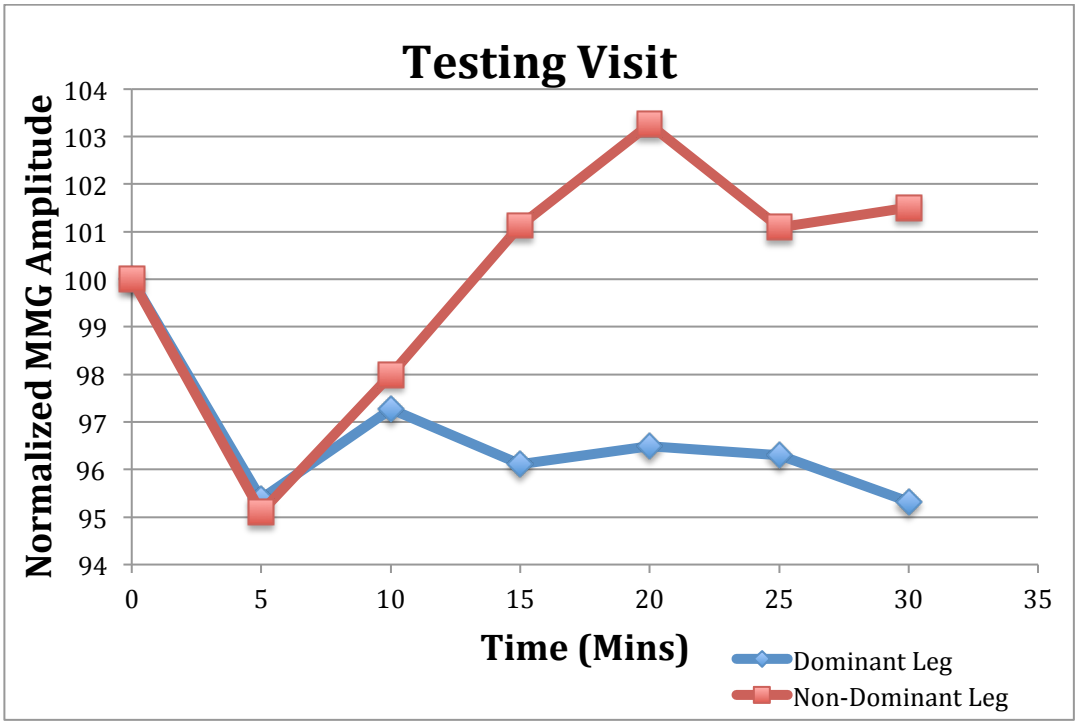

Figure 2. Vastus lateralis Dominant vs. Non-dominant MMG amplitude across time during testing visit. Time point 0 indicates the 5 minutes pre-exercise resting conditions normalized to 100%.

Table 3. Effect Size of dominant vs. Non-dominant across time during testing visit. A positive value indicates a larger MMG amplitude in the dominant leg in relation to the non-dominant leg during that time point. A negative value indicates a larger non-dominant MMG amplitude value in relation to the dominant leg.

Time	Effect Size
Pre 0.5 minutes	N/a
Post 5 minutes	0.025919085
Post 10 minutes	-0.07552212
Post 15 minutes	-0.445335929
Post 20 minutes	-0.498888511
Post 25 minutes	-0.302579177
Post 30 minutes	-0.472753488

4.2.6 Interpretation of Figure 3 and table 4

Figure 3 demonstrates the mean normalized MMG amplitude in the dominant vastus lateralis during the testing vs. control visits. ANOVA results showed no statistically significant ($p > 0.05$) difference between leg, visit, and time.

Table 4 shows the results of Cohen's d for effect size in the dominant leg during control vs. testing visits. A large effect size existed at minute 5 ($d = -0.78$), minute 15 ($d = -0.89$), and minute 30 ($d = -0.72$). A medium to large effect size existed at minutes 20 ($d= -0.67$) and 25 ($d= -0.66$). A medium effect size existed at minute 10 ($d = -0.44$). These findings suggest that there was an overall decrease in normalized MMG amplitude in the dominant leg during the testing visit when compared to the control visit.

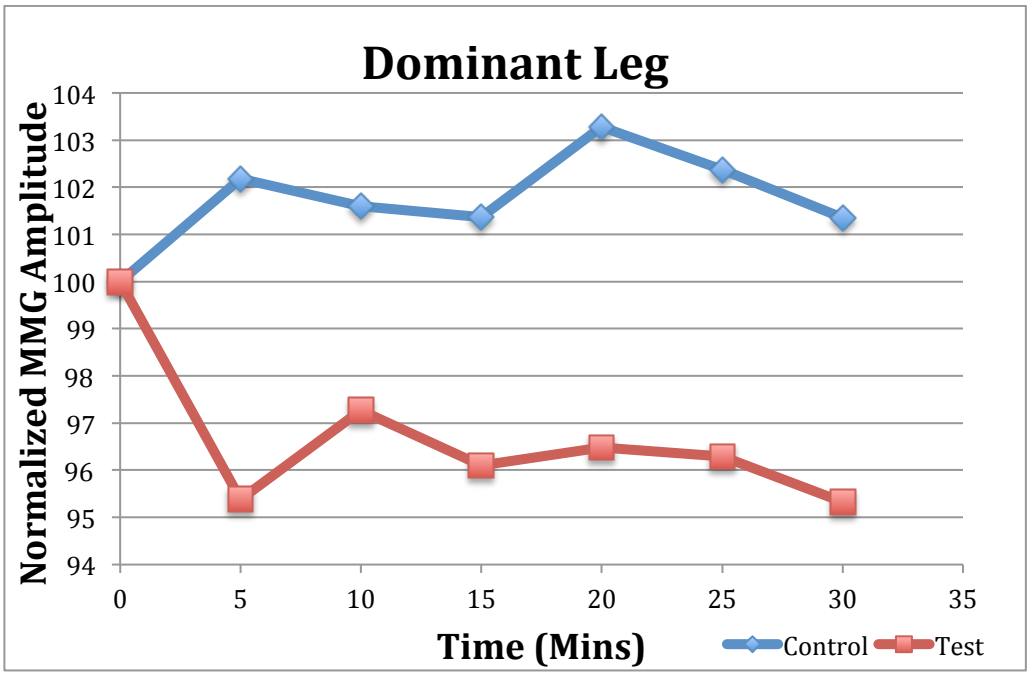

Figure 3. Vastus lateralis Dominant MMG amplitude during control and testing visit. Time point 0 indicates the 5 minutes pre-exercise resting conditions normalized to 100%

Table 4. Effect Size of dominant leg across time during control vs. testing visit. A positive value indicates a larger MMG amplitude during the testing visit compared to the control visit while a negative value indicates a larger MMG amplitude value during the control visit compared to the testing visit.

Time	Effect Size
Pre 0.5 minutes	N/a
Post 5 minutes	-0.785725019
Post 10 minutes	-0.447040282
Post 15 minutes	-0.897033822
Post 20 minutes	-0.67938679
Post 25 minutes	-0.668992508
Post 30 minutes	-0.729216408

4.2.7 Interpretation of Figure 4 and table 5

Figure 4 shows the differences in normalized MMG amplitude between the non-dominant leg during control and testing visits. The results from the 3-way repeated measures ANOVA showed no statistically significant ($p > 0.05$) differences between leg, visit, and time.

Table 5 reports the results of Cohen's d effect size in the non-dominant legs between visits. A moderate effect exists at minute 30 ($d = 0.51$). A small effect exists at minutes 5 ($d = -0.38$) and 20 ($d = 0.31$).

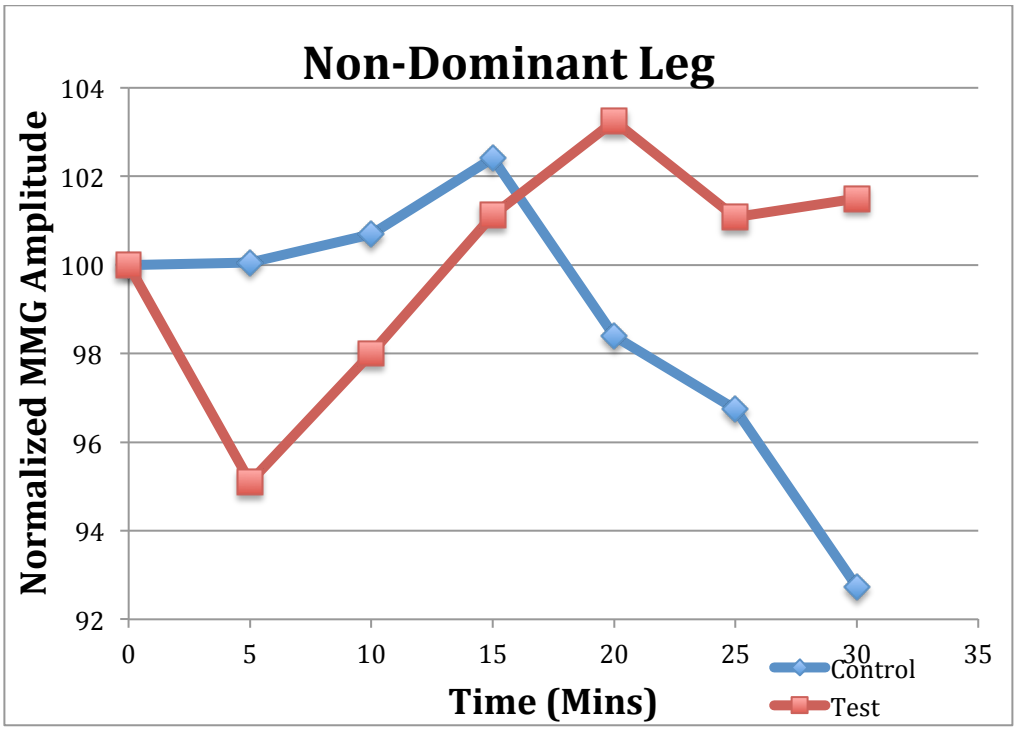

Figure 4. Vastus lateralis Non-dominant normalized MMG amplitude during control and testing visit. Time point 0 indicates the 5 minutes pre-exercise resting conditions normalized to 100%

Table 5. Effect Size of Non-dominant leg across time during control vs. testing visit. A positive value indicates a larger MMG amplitude during the testing visit compared to the control visit while a negative value indicates a larger MMG amplitude value during the control visit compared to the testing visit.

Time	Effect Size
Pre 0.5 minutes	N/a
Post 5 minutes	-0.383285235
Post 10 minutes	-0.19477383
Post 15 minutes	-0.082789046
Post 20 minutes	0.351484962
Post 25 minutes	0.235426904
Post 30 minutes	0.515019831

CHAPTER V: DISCUSSIONS AND CONCLUSIONS

5.1 Introduction

The purpose of this study was to investigate if resting muscle activity demonstrates increased mechanical activity via MMG during an acute bout of single leg resistance training when compared to normalized baseline values. A secondary research question was to determine if this resting activity is different between the exercised and non-exercised legs. Originally, it was suggested that resting muscles showed no MMG activity due to lack of EMG activity at rest [6]. However, this is contradicted by more recent studies showing some resting MMG activity following both aerobic and anaerobic exercise [6,12,14]

The content of this chapter will focus on discussion and interpretation of the results presented in the previous chapter. This will include explanations of the significance of the findings and conclusions that can be inferred from the findings. Additionally, suggestions for future research will be discussed.

5.2 Findings

The present study found no significant mean differences ($p > 0.05$) between MMG amplitude values from the exercise and control visits for the vastus lateralis muscle at any time point. These findings must be interpreted with great caution, given the fact that there were moderate and even large effect sizes observed for some of the pairwise differences. It is not the prerogative of this study to debate the use of effect sizes versus statistical significance testing. Both

have their value in research. However, the findings from this study provided support for the contention that the 10 research subjects that were used provided enough statistical power to find a significant 3-way interaction, but not enough to result in any pairwise mean differences (given the observed effect sizes).

Figures 2 and 3 demonstrated moderate mean differences, and taking into consideration the effect sizes shown in tables 3 and 4 suggest that there may have been an overall decrease in normalized MMG amplitude in the dominant leg during exercise when compared to the control visit. These findings are similar to results by Wages (2013), who found 10% and 15% decreases in normalized MMG amplitude following resistance exercise in the erector spinae and trapezius muscles [23]. They suggested that the decrease in MMG amplitude was due to a relaxation effect in the exercised muscles following resistance training. Additionally, these findings are similar to those from de Vries (1965, 1968), who found a decrease in EMG amplitude following an acute bout of moderate intensity resistance training in the quadriceps femoris [27]. This suggests that there was a slight relaxation effect in the dominant leg following resistance training.

As shown in figures 2 and 4, the non-dominant leg had an initial decrease in MMG amplitude 5 minutes post exercise. Although not statistically significant, there is a small-moderate effect (-0.38) at 5 minutes post-exercise. This suggests that there may be a slight relaxation effect in the non-dominant leg following resistance training that quickly went back to baseline.

5.3 Significance of the results

The present study found no significant changes in post-exercise MMG amplitude across time for either the dominant or non-dominant vastus lateralis. However, the aforementioned problem with borderline statistical power and the effect sizes shown in figures 2 and 4 show an overall decrease (although not statistically significant) in MMG amplitude in the dominant leg following exercise. These findings are supported by research performed by Wages (2013) and De Vries (1965, 1968), who respectively found a decrease in MMG and EMG amplitudes following an acute bout of resistance training [9,27].

McKay et al. (2004, 2006, 2007) have previously examined MMG amplitude responses following both aerobic and resistance exercise [6,12,14]. McKay et al (2006) reported an increase in MMG mean amplitude in the rectus femoris following right leg extensions on an isokinetic dynamometer. The authors found that resting MMG amplitude increased linearly with work rate and that a cross over effect occurred in the non-exercised leg [6]. McKay et al (2007) found an increase in resting MMG amplitude in the rectus femoris following an acute bout of resistance training involving the leg press and leg extension at 75% of their 1RM (5 sets of 8 repetitions) [12]. Additionally, the authors (McKay et al 2007) found an increase in EMG amplitude following the same exercise [12]. Similarly, McKay et al (2004) found an increase in resting MMG amplitude in the rectus femoris following 30 minutes of aerobic training on a cycle ergometer at 70% peak VO_2[14]. The findings from the current study differed from the results of McKay et

al [6,12,14], as well as differed in the muscle groups measured and exercise intensities. McKay et al (2004, 2007) also demonstrated that MMG amplitude decayed with a time constant similar to VO_2 kinetics, suggesting that this activity may have contributed to EPOC. While the increase in activity is largely unknown, it is suggested that the activity comes from blood vessels near the muscle that help return blood back to the heart following exercise [17].

The findings from the present study are important in the sense that they exhibited a relaxation effect following resistance exercise. These findings were similar to results from Wages (2013), who found 10% and 15% decreases in normalized resting MMG amplitude in the erector spinae and trapezius muscles, respectively [23]. Wages (2013) suggested that the relaxation effect might be useful for alleviating pain in populations that suffer from chronic lower back issues. Additionally, the present results are similar to De Vries (1968) who found a decrease in resting EMG amplitude in the quadriceps femoris muscles following resistance training.

5.4 Conclusion

In conclusion, the results of the present study did not show any statistically significant ($p > 0.05$) differences between resting MMG amplitude for the exercise and control visits in the dominant and non-dominant limbs. While no significance was demonstrated, visual inspection of figures 2 and 3 and the main effects shown in tables 3 and 4 show a probable overall decrease in resting normalized MMG

amplitude following resistance training in the vastus lateralis. This suggests that a relaxation effect existed following an acute bout of single leg resistance training. While no statistically significant difference ($p>0.05$) occurred in the non-dominant leg, a small-moderate relaxation effect was demonstrated in the non-dominant leg 5 minutes post exercise (figure 4, table 5).

5.5 Recommendations

Several recommendations can be made for further research. First, the sample size of the present study was limited at 10 participants. It would be beneficial to include more participants to further investigate this issue. Secondly, MMG amplitude in the dominant leg never reached baseline values during the 30 minutes of post-exercise measurements. It may be beneficial then to extend the time to an hour or longer of post-exercise recording. This could potentially be beneficial for investigating the length of time delay for MMG amplitude in relation to EPOC. Finally, using the same study but only resistance-trained subjects might be beneficial for further investigating the effect that training status has on resting muscle activity.

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APPENDICES

A1: Institutional Review Board

The UNIVERSITY of OKLAHOMA

Institutional Review Board for the Protection of Human Subjects

Approval of Initial Submission – Expedited Review – AP01

Date: June 25, 2014 **IRB#:** 4406

Principal Investigator: Cody A. Miller **Approval Date:** 06/25/2014

Expiration Date: 05/31/2015

Study Title: Resting mechanomyography after an acute bout of single leg resistance exercise.

Expedited Category: 4

Collection/Use of PHI: No

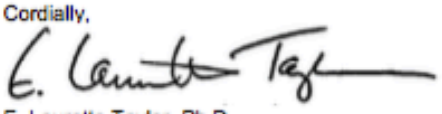
On behalf of the Institutional Review Board (IRB), I have reviewed and granted expedited approval of the above-referenced research study. To view the documents approved for this submission, open this study from the *My Studies* option, go to *Submission History*, go to *Completed Submissions* tab and then click the *Details* icon.

As principal investigator of this research study, you are responsible to:

- Conduct the research study in a manner consistent with the requirements of the IRB and federal regulations 45 CFR 46.
- Obtain informed consent and research privacy authorization using the currently approved, stamped forms and retain all original, signed forms, if applicable.
- Request approval from the IRB prior to implementing any/all modifications.
- Promptly report to the IRB any harm experienced by a participant that is both unanticipated and related per IRB policy.
- Maintain accurate and complete study records for evaluation by the HRPP Quality Improvement Program and, if applicable, inspection by regulatory agencies and/or the study sponsor.
- Promptly submit continuing review documents to the IRB upon notification approximately 60 days prior to the expiration date indicated above.
- Submit a final closure report at the completion of the project.

If you have questions about this notification or using iRIS, contact the IRB @ 405-325-8110 or irb@ou.edu.

Cordially,



E. Laurette Taylor, Ph.D.
Chair, Institutional Review Board

A2: Recruitment Flier

Resting Mechanomyography before and after an acute bout of single leg resistance exercise

Purpose: Investigate if resting muscle mechanical activity increases relative to baseline after an acute bout of single-legged resistance exercise as well as compare resting muscle mechanical activity with the contralateral leg.

What you will be asked to do:

- Perform single leg resistance training (70% of max)
- Have resting mechanomyography sensors placed over the leg and measurements taken while resting
- Three mandatory visits separated by 24 hours (~45 mins ea.)

Requirements:

- Healthy College aged males, ages 18-35
- No history of lower limb injury or surgery
- No known metabolic or neuromuscular disease

Injury:

****Participating in this study may induce muscle soreness, with possible risk of muscle injury or muscle tear.

Cody Miller
Phone: (903) 456 - 9281
Email: cody.a.miller-1@ou.edu

A3: Health Questionnaire

PRE-EXERCISE TESTING HEALTH & EXERCISE STATUS QUESTIONNAIRE

The University of Oklahoma
DEPARTMENT OF HEALTH AND EXERCISE SCIENCE

Name _____ Date_____

Home Address_____

Phone _____

Person to contact in case of emergency _____

Emergency Contact Phone _____ Birthday (mm/dd/yy)____/_____/_____

Gender_____ Age _____(yrs) Height _____(ft)_____(in) Weight_____(lbs)

Does the above weight indicate: a gain____ a loss____ no change____ in the past year?
If a change, how many pounds?_____(lbs)

A. JOINT-MUSCLE STATUS (✓Check areas where you currently have problems)

Joint Areas	Muscle Areas
( ) Wrists	( ) Arms
( ) Elbows	( ) Shoulders
( ) Shoulders	( ) Chest
( ) Upper Spine & Neck	( ) Upper Back & Neck
( ) Lower Spine	( ) Abdominal Regions
( ) Hips	( ) Lower Back
( ) Knees	( ) Buttocks
( ) Ankles	( ) Thighs
( ) Feet	( ) Lower Leg
( ) Other_____	( ) Feet
	( ) Other_____

B. HEALTH STATUS (✓Check if you currently have any of the following conditions)

( ) High Blood Pressure	( ) Acute Infection
( ) Heart Disease or Dysfunction	( ) Diabetes or Blood Sugar Level Abnormality
( ) Peripheral Circulatory Disorder	( ) Anemia
( ) Lung Disease or Dysfunction	( ) Hernias
( ) Arthritis or Gout	( ) Thyroid Dysfunction
( ) Edema	( ) Pancreas Dysfunction
( ) Epilepsy	( ) Liver Dysfunction
( ) Multiply Sclerosis	( ) Kidney Dysfunction
( ) High Blood Cholesterol or Triglyceride Levels	( ) Phenylketonuria (PKU)
	( ) Loss of Consciousness
( ) Allergic reactions to rubbing alcohol	

* *NOTE: If any of these conditions are checked, then a physician's health clearance will be required.*

C. PHYSICAL EXAMINATION HISTORY

Approximate date of your last physical examination_____

Physical problems noted at that time_____

Has a physician ever made any recommendations relative to limiting your level of physical exertion? _____YES _____NO
If YES, what limitations were recommended?_____
_____

D. CURRENT MEDICATION USAGE (List the drug name and the condition being managed)

MEDICATION	CONDITION
_____	_____
_____	_____
_____	_____

E. PHYSICAL PERCEPTIONS (Indicate any unusual sensations or perceptions. ✔Check if you have recently experienced any of the following during or soon after *physical activity* (PA); or during *sedentary periods* (SED))

PA	SED		PA	SED	
( )	( )	Chest Pain	( )	( )	Nausea
( )	( )	Heart Palpitations	( )	( )	Light Headedness
( )	( )	Unusually Rapid Breathing	( )	( )	Loss of Consciousness
( )	( )	Overheating	( )	( )	Loss of Balance
( )	( )	Muscle Cramping	( )	( )	Loss of Coordination
( )	( )	Muscle Pain	( )	( )	Extreme Weakness
( )	( )	Joint Pain	( )	( )	Numbness
( )	( )	Other_____	( )	( )	Mental Confusion

F. EXERCISE STATUS

Do you regularly engage in aerobic forms of exercise (i.e., jogging, cycling, walking, etc.)? **YES** **NO**

How long have you engaged in this form of exercise? _____ years _____ months

How many hours per week do you spend for this type of exercise? _____ hours

Do you regularly lift weights? **YES** **NO**

How long have you engaged in this form of exercise? _____ years _____ months

How many hours per week do you spend for this type of exercise? _____ hours

Do you regularly play recreational sports (i.e., basketball, racquetball, volleyball, etc.)? **YES** **NO**

How long have you engaged in this form of exercise? _____ years _____ months

How many hours per week do you spend for this type of exercise? _____ hours

A4: Informed Consent

University of Oklahoma
Institutional Review Board
Informed Consent to Participate in a Research Study

Project Title: Resting mechanomyography after an acute bout of single leg resistance exercise
Principal Investigator: Cody Miller
Department: Health and Exercise Science

You are being asked to volunteer for this research study. This study is being conducted at the Collums Building (Department of Health and Exercise Science, Norman campus). You were selected as a possible participant because you are a healthy male between the ages of 18-35.

Please read this form and ask any questions that you may have before agreeing to take part in this study.

Purpose of the Research Study

The purpose of this study is to investigate if low-frequency vibrations in resting muscle increase relative to baseline after an acute bout of single-legged resistance exercise as well as compare if vibrations in the non-exercised leg increase.

Number of Participants

About 25 healthy, college aged men will participate in this study.

Procedures

If you agree to this study you will be ask to make three separate visits to the Collums building of the Department of Health and Exercise Science. Each visit will last no more than 45 minutes. The first visit will include familiarization with the equipment and muscle actions that will be performed. The first visit will also include one-repetition maximum (1-RM) testing, which will test the maximum weight that you can lift in one attempt following a brief warm-up. The second and third visit will be randomized between a control visit and a testing visit. During the control visit, you will be asked to sit, relaxed while a mechanomyography (MMG) sensors are placed over both thighs to record mechanical activity of your muscles while relaxing. The MMG sensors are small sensors that detect small movements under the skin in a noninvasive manor. During the experimental visit, you will be asked to perform six sets of 10 repetitions at 70% of 1-RM for single-legged leg extension with one minute of rest between sets. After completion of exercise, you will be asked to relax while MMG sensors are placed over both thighs to record mechanical activity of your muscles. Prior to placement of the MMG sensors, your skin will be shaved and cleaned with alcohol for MMG sensor placement.

Length of Participation

Participation in this study requires a total of three visits to the research laboratory. These visits must each be separated by at least 24 hours. Each visit will take approximately 45 minutes.

IRB NUMBER: 4406
IRB APPROVAL DATE: 05/19/2019
IRB EXPIRATION DATE: 04/30/20

you cannot reach the research team, you may contact the University of Oklahoma – Norman Campus Institutional Review Board (OU-NC IRB) at 405-325-8110 or irb@ou.edu.

You will be given a copy of this information to keep for your records. If you are not given a copy of this consent form, please request one.

Statement of Consent

I have read the above information. I have asked questions and have received satisfactory answers. I consent to participate in the study.

Signature Date