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Do Individual Responses to Resistance Exercise Exist to an Extent That Can be Detected Beyond That of Measurement Error/ Random Biological Variability?

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DO INDIVIDUAL RESPONSES TO RESISTANCE EXERCISE EXIST TO AN EXTENT THAT CAN BE DETECTED BEYOND THAT OF MEASUREMENT ERROR/RANDOM BIOLOGICAL VARIABILITY?

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ABSTRACT

Millions of dollars are spent analyzing inter-individual differences in response to resistance exercise, but the lack of a non-exercise control group means they may simply be examining random error. The purpose of this study was to determine whether there are inter-individual differences in response to two distinct resistance exercise protocols. Participants (n=151) were randomly assigned to one of 3 groups as follows: (1) a traditional exercise group performing 4 sets to failure with a load that could be lifted 8-12 times; (2) a one-repetition maximum (1RM) training group performing a 1RM test each visit; and (3) a non-exercise control group. Both exercise groups performed 18 sessions of elbow flexion exercise over 6 weeks. Both 1RM training (2.3kg) and traditional training (2.4kg) increased 1RM strength to a similar extent. Only the 1RM group increased untrained arm 1RM strength (1.5kg) which was greater than both other groups ($p<0.05$). The traditional exercise group also increased ultrasound measured muscle size at all sites (all>0.22cm), each of which were greater than both the control and 1RM group ($p<0.05$). The 1RM group did not increase muscle mass ($p>0.05$). Across both training groups, the only individual responses were found in the change in 1RM strength of the trained arm in the traditional training group (Levene's test $p<0.05$) in which 10 individuals (25%) were classified as responding differently from the mean. The variability in the response to other outcomes did not exceed that of the control group indicating it could not be detected above random error. Other commonly used approaches of classifying differential responders such as clustering analyses, standard deviations above and below the mean, and upper/lower percentiles would produce different results but are not appropriate. These findings demonstrate the importance of

taking into consideration the magnitude of random error when classifying individual responders, and provide possible rationale as to why numerous analyses fail to find/replicate what genes may be responsible for producing more favorable exercise outcomes.

LIST OF ABBREVIATIONS AND SYMBOLS

1RM	One repetition maximum
ACSM	American College of Sports Medicine
ANCOVA	Analysis of covariance
BF	Bayes factor
CI	Confidence interval
DXA	Dual X-ray absorptiometry
HERITAGE	Health, RIsk factors, exercise Training And GEnetics
METRET	Molecular Epidemiology of Resistance Exercise Training
MRI	Magnetic resonance imaging
mRNA	Messenger RNA
MVC	Maximal voluntary contraction
SD	Standard deviation

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CHAPTER 1: INTRODUCTION

Current American College of Sports Medicine (ACSM) guidelines recommend that individuals perform two sessions of resistance exercise and 150 minutes per week of moderate to vigorous aerobic physical activity as a part of a comprehensive exercise program (1). Numerous positive health outcomes are associated with both endurance (2) and resistance (3) exercise which illustrates the potential importance of increasing such low adherence rates (endurance: 41.3% and resistance exercise: 14.7%) (4). While there are established guidelines for prescribing both exercise modalities, it is commonly stated that there is a large variability in response to both endurance (5) and resistance (6) exercise even when individuals complete the same exercise protocol. This has led to a push for personalized exercise programming (7) and expensive genetic analyses to determine the cause of such variability (8).

While a high degree of variability appears to be present in the response to exercise interventions, it may be difficult to tease out whether the variability is caused by the intervention itself, or whether it is caused by measurement error or random biological variability. There are two main ways in which this could be tested which include either (1) having a time-matched control group that details the magnitude of variability existing independent of involvement in the intervention (9); or (2) re-running the same study to test whether these same observations are repeatable (10, 11). Some of the adaptations that occur in response to exercise have long lasting effects, and thus re-running the same study does not seem like an appropriate option, particularly when examining responses to resistance exercise (12). When examining the totality of the current literature there is currently limited evidence that individual responses to exercise interventions

exist and this can be attributed to inadequacies in study designs (i.e. not including a time-matched control group) (13). Additionally, it is currently unknown whether different types of training interventions will produce different magnitudes of variability. Our laboratory has previously shown that repeatedly performing the 1RM test produces large increases in muscle strength (14, 15), but it is unknown if these adaptations are more heterogeneous comparative to traditional training protocols.

Not only is an appropriately designed study needed to assess whether individual responses to exercise interventions exist, but there is also a need to re-evaluate how we are currently classifying individuals as responders or non-responders. While it is common to simply take those who responded one standard deviation above the mean as being high responders (16, 17), any normally distributed data will always result in a similar proportion of the study population being classified as high responders (i.e. 16%) regardless of the true heterogeneity in the data. Another approach involves classifying the top and bottom 15% (18) or 20% (19) of individuals as being high and low responders, respectively, and this has the same limitation of not knowing whether these people are truly differential responders or simply on one end of random error. The ability to classify differential responders should be based on the variability present in the change score of the control group because this allows for the determination of how well individual responders can be detected. If there is no variability in the data set above that of a time-matched control group, this will illustrate that individual responses to exercise interventions cannot be detected with current technology. If there is a high degree of variability we will demonstrate a more informative way to present the data and seek to determine whether there are universally high or low responders across all variables tested. Lastly, we will detail if differences

in training protocols produce different magnitudes of true variability in response to the intervention.

Purpose

The purpose of this study was to determine whether individual responses to resistance exercise could be detected with current technology, while also illustrating the magnitude of variability that was present. In addition, we sought to examine whether there was greater variability depending on the exercise protocol employed (repeated 1RM testing vs. traditional training). One final purpose was to examine differences in muscle strength and muscle size in response to repeated 1RM training compared to that of traditional training.

Research Question

Can individual differences in the response to resistance exercise be detected above and beyond what can be explained simply by measurement error and random biological variability? Furthermore, will the magnitude of individual differences differ when comparing two distinct training protocols?

Hypothesis

With respect to group mean differences, we hypothesized that only the trained arm within the traditional exercise group would increase muscle size and that this increase would exceed that of both the control and 1RM groups. We also hypothesized that both the 1RM and traditional exercise groups would increase 1RM strength and isokinetic strength to a similar extent in the trained arm. For the untrained arm, we hypothesized there would be no changes in muscle size or isokinetic strength in either of the training groups. Additionally, it was hypothesized that 1RM strength of the untrained arm would increase to a similar extent in both the 1RM and traditional exercise groups. As for the presence of individual responders to

exercise, we hypothesized that there would be true individual responses in 1RM strength of both the exercised and non-exercised contralateral limb in both the traditional training and 1RM training groups. With respect to isokinetic strength, we hypothesized individual responders would be present in the trained arm of both the 1RM and traditional exercise groups, but no individual responders would be detectable in the untrained arm. Additionally, we hypothesized there would be no true individual variability with respect to changes in muscle size in either arm for either of the training groups.

Significance of Study

Proposed individual responses to exercise have led to millions of dollars being spent on genome-wide studies to determine what causes these individual responses. These individual responses have also led to the recommendation that exercise prescription should be more personalized. One major limitation of these studies is that we have yet to determine whether these individual responses exist. Furthermore, the current analyses used to assess individual differences are potentially misleading and limit the interpretation.

Assumptions

1. Participants in the traditional training group performed as many repetitions as they could on all sets of exercises.
3. Participants truthfully answered all questions related to meeting the inclusion criteria.
4. Participants adhered to all restriction criteria over the course of the study.
5. Participants gave a maximal effort on all strength measurements.

Delimitations

1. The results of the study are indicative of the effects in untrained individuals.
2. The results of the study are applicable to individuals between the ages of 18-35.

3. The results will only be applicable to adaptations to the elbow flexor muscles.

Limitations

1. Ultrasound was used for measurements of muscle size despite greater reliability reported for magnetic resonance imaging (MRI). Although this is a limitation, we did not have access to MRI, and ultrasound is a common measurement technique used for tracking changes in muscle size and this has been shown to track well with MRI (20).

2. The investigators were not blinded to the individuals group assignment during strength testing, however, the investigators gave the exact same testing instructions to all individuals.

3. We only tested the elbow flexors; however, we felt this is the most appropriate muscle group to test in terms of limiting measurement error and random biological variability. We believe this is true due to the elbow flexors being minimally involved in everyday life comparative to other muscle groups such as muscles of the legs.

Operational Definitions

1. Muscle thickness – The distance between the muscle-fat interface and underlying bone will be measured via B-mode ultrasound.

2. One repetition maximum (1RM) – The maximal load that could be lifted one time with proper form for the dumbbell unilateral elbow flexion exercise.

3. Isokinetic strength – The maximal amount of torque that could be produced against an object moving at a set speed.

4. True variability – The magnitude of variability that could be attributed to the exercise intervention after teasing out measurement error and random biological variability.

CHAPTER 2: LITERATURE REVIEW

Adaptations and benefits of resistance exercise

The most commonly studied outcomes that are associated with engaging in resistance exercise include changes in muscle size and strength. While increases in muscle size are thought to occur through a complex set of molecular events beginning with localized mechanotransduction (21), the causes of increased strength appear to be driven by neurological adaptations, or other local muscular adaptations that are independent of muscle hypertrophy (22–24). This provides important information for exercise programming because individuals looking to get stronger can likely maximize their strength gains by simply performing repeated maximal tests (14, 15). On the contrary, muscle growth occurs independent of the exercise load provided the muscle is fatigued (25–28), and this adaptation is more so related to the magnitude of the muscle being activated and the duration of this activation (29, 30). While muscle size and strength are commonly tested because of their suspected importance for sports performance, resistance training may also be important for attenuating the rate of sarcopenia (age related loss of muscle size and strength) in the general population (31). Other proposed benefits for resistance exercise amongst the general population include decreased adiposity, decreased disease risk, improved cognition and improved self-esteem (32). While engaging in resistance exercise may provide an array of health benefits, it is the outcome associated with resistance exercise (i.e. strength) that seems to be of greater importance (4).

Changes in muscle mass with resistance exercise

Changes in muscle mass are thought to occur through a process of mechanotransduction, in which the mechanical stress of muscle contraction is converted into a chemical signal (21). The effectiveness of a resistance training program for increasing muscle size would appear to be contingent upon activating the mechanotransduction cascade in a sufficient proportion of muscle fibers (29, 30) given that muscle growth at the whole muscle level is thought to occur primarily through an increase in the size of each individual muscle fiber (i.e. muscle hypertrophy) (33). For this reason, it is likely that various resistance training protocols will all induce similar increases in muscle size provided they activate a similar proportion of muscle fibers. Specifically, the exercise load appears to be of little importance provided the exercise is fatiguing enough to activate a large number of muscle fibers and this is apparent when comparing low and high load exercise performed to volitional failure (25–28). The duration in which each muscle fiber is activated also appears to be important considering repeated 1RM training does not increase muscle mass despite presumably high muscle activation (15, 30). Further evidence from our laboratory has shown that even exercising with no external load (i.e. simply flexing through the full range of motion) produces similar increases in muscle size to that of traditional high load exercise (i.e. 70% 1RM) (28). Lastly, increases in muscle size occur via local adaptations in the exercised muscle and are not augmented by exercise-induced systemic hormone production (34). Collectively, these findings illustrate that muscle hypertrophy is a localized process that occurs independent of the exercise load.

Changes in muscle strength with resistance exercise

Early strength gains that occur from resistance exercise have been hypothesized to occur via neural mechanisms before being predominantly driven by increases in muscle size (35).

Despite this original hypothesis, this idea has been recently challenged (24, 36) given that large increases in muscle strength have been observed in the absence of muscle hypertrophy (15). Specifically, our laboratory has examined the effectiveness of repeatedly performing the 1RM strength test in demonstrating that performing the 1RM test produces similar increases in strength to that of a traditional exercise (15). Furthermore, we have shown that adding three sets of exercise to the 1RM test did not further increase muscle strength (14). Therefore, it appears that the most robust stimulus for individuals looking to increase their 1RM strength would be to repeatedly perform the 1RM test. Other studies have illustrated that, while both low and high load training protocols produce similar increases in muscle size, the magnitude of strength increases will usually be greater when higher loads are used (25–28). Therefore, it appears that increases in strength are predominantly driven by the load that is being lifted, and the greatest increases in muscle strength can be achieved by training at or near an individual's 1RM (25–28). Numerous neural mechanisms have been proposed to be responsible for the increases in strength from resistance training including increased firing rates of motor neurons, increased excitability of motor neurons, an increased level of central drive from the motor cortex, and a decrease in excitability of inhibitory neurons (37–39). Although the specific mechanism driving increases in muscle strength are not well understood, further evidence supporting an involvement of the central nervous system exists particularly given increases in muscle strength of contralateral limbs that are not directly trained (and thus do not hypertrophy) (40). Although morphological changes occurring within the exercised muscle cannot be ruled out given the observed increase in specific tension at the fiber level (23), it appears that increases in strength are predominantly driven by adaptations to the nervous system (given that these morphological changes would not be expected to occur in the contralateral limb).

Individual responders to exercise

The idea that individuals respond differently to exercise stimuli has been attributed to rare and common genetic mutations across individuals. This has led to research examining the magnitude of heritability that exists amongst different traits (41). These studies are termed HERITAGE (HEalth, RIsk factors, exercise Training And Genetics) family studies and have concluded that an individual's baseline fitness level (42) and their trainability to exercise (5) are largely inherited. Given that the individual differences in these exercise traits can be attributed to genetics, studies are seeking to determine what genes are associated with more favorable outcomes. For example, as it relates to resistance exercise, it has been proposed that the ACTN3 genotype (43) and the ACE I/D genotype (44) are each associated with more favorable outcomes with respect to muscle size and strength gains. Therefore, if an individual has one of these genotypes it would be assumed that they would respond more favorably to the exact same resistance exercise protocol when compared to an individual with a different genotype. These genotypes may then be linked to different cellular events that occur across individuals. For example, studies have concluded that the amount of muscle size that an individual gains may be related to the rate of satellite cell proliferation (45–47) and these differential rates of satellite cell proliferation can be attributed to genetic differences across individuals. As it pertains to resistance exercise, the Molecular Epidemiology of Resistance Exercise Training (MERET) study was designed to examine how genetics influence adaptations to resistance exercise, but published studies have involved very small sample sizes (e.g. $\leq$ 150 people for a genome study) assessing candidate (pre-specified) genes (48, 49).

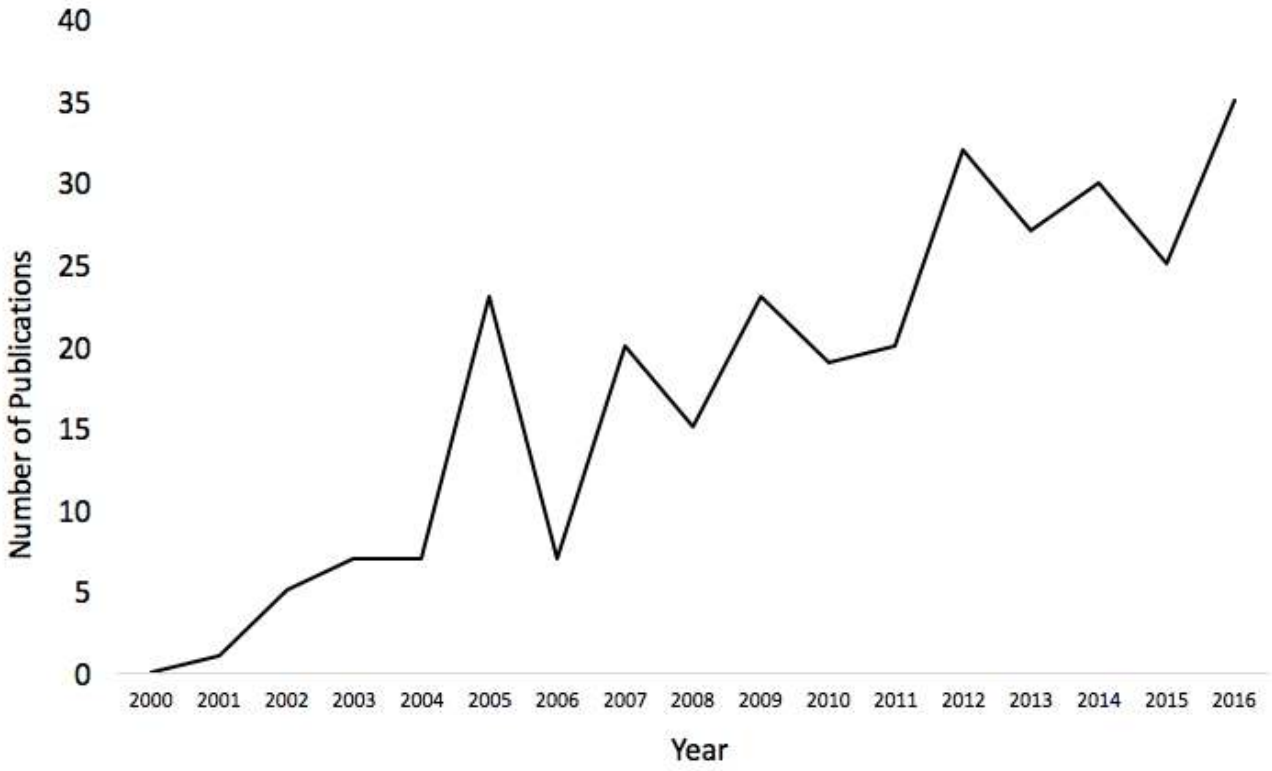
Genome wide association studies

There has been a considerable amount of research focused on deciphering what genetic mutations cause the individual responses to exercise. These types of studies are termed genome wide association studies and incorporate large sample sizes in attempt to link specific single nucleotide polymorphisms (which are common genetic mutations in DNA) with more advantageous/disadvantageous training outcomes. In these studies, gene chips (more technically termed gene chip microarrays) are used to look all messenger RNA (mRNA) transcripts that comprise the human genome. The ability to look at all mRNA transcripts provides an unbiased approach to examining what genes may be responsible for different outcomes because specific candidate genes do not have to be selectively analyzed (50). The ultimate goal is to associate different mRNA transcripts with some type of outcome measure such as different types of diseases (51) or outcomes that occur in response to endurance (52) or resistance training (53) interventions. These studies are quite expensive and can cost upwards of $10 million for a single study (8). Depending on one's perspective it could be argued that these studies have been a major disappointment in failing to link common DNA variants with exercise training outcomes (8). This may be related to the relatively poor reliability that accompanies baseline test-retest measurements of gene expression measures (pearson correlation ranges: 0.21 – 0.85, ICC ranges: 0.13 – 0.74, and CV ranges: 11 – 33%), making it difficult to link unreliable gene expressions with exercise outcomes (54).

To avoid biased and unstable effect size estimates, it has been suggested that sufficient sample sizes must be analyzed (55). Thus, to appropriately design a single study looking at 500,000 single nucleotide polymorphisms, for example, this would require a sample size of approximately 1,200 individuals assuming 5% of the population has the outcome of interest (56).

Therefore, this single study would cost approximately $600,000 to conduct ($500 per chip * 1,200 chips). Given the cost to fund such a study, and recent rise in the number of microarray studies that are specifically centered on its effects related to exercise (Figure 1), it is clear the organizations responsible for funding such studies acknowledge two things: (1) there are individual responses to exercise interventions and (2) it is very important to determine what genes are responsible for these individual responses. Despite this statement, it may not be so clear that individual responses to exercise interventions exist, at least not to an extent that they can be measured with current technology.

Figure 1. Published studies in PubMed using Microarray to analyze exercise adaptations



The number of studies identified using the search terms “microarray” and “exercise” in PubMed. These are expressed as the number of articles within each specific year.

Analyzing individual responses

Examining genetic differences in attempt to link specific genes with different diseases is undoubtedly complex, but this complexity is often minimized by the ability to classify a given disease as a dichotomous variable in that an individual either has the disease or they do not have the disease. This becomes more complex when analyzing individual responses to exercise interventions because there are no established cut points to place individuals into set categories. For example, as it relates to disease, there is often a dichotomous outcome to say any given individual either has cancer or they do not, but as it relates to changes in response to exercise interventions this becomes much more difficult since there are no established cut points to use in determining whether someone responded or did not respond to exercise. While it is common for studies to use the standard deviation of the change between repeated measurements (termed the typical error or standard error of the measurement) (57), this still places individuals into a dichotomous grouping variable (i.e. responders vs. non-responders) (58–60), despite the suggestion that these variables may be more appropriately analyzed on a continuum (61). For example, if the typical error for a given strength test is 5 kg and one individual improves their strength by 15 kg and another individual improves their strength by 50 kg, it would be an oversimplification to just classify them into the same category as responders. Therefore, this makes it difficult to establish groups based on the dependent variable because there is no easy way to group the individuals as they are all likely to have different changes in the outcome variable.

Deciphering true variability from random biological variability and measurement error

Researchers are often attempting to determine the true variability that is present in response to an intervention. In other words, they are seeking to answer the question: Are

differences in how people respond to exercise truly a product of the exercise intervention itself? The ability to measure true variability is impeded by both random biological variability and measurement error. Measurement error may comprise human and/or technological error associated with the measurement and can easily be assessed by using a simple test/retest design. For example, if a researcher were to take an individual's body mass using a standard scale the researcher can simply weigh the individual on two separate occasions if the goal is to truly assess measurement error (measurements that may be biased to having the tester knowing the previous measurement unit may require a longer period between tests). The reliability of the measurement can then be calculated by taking the standard deviation (SD) of the difference between the two measurements, such that a small SD demonstrates a very reliable measurement and a large SD demonstrates poor reliability. This SD of the difference between measures can then be converted to a coefficient of variation (SD divided by the mean) or by multiplying this number by 1.96 to obtain the minimal difference (62). If the researcher does not consider the measurement error that is present, the individual variability in response to an exercise intervention could simply be assessing the magnitude of measurement error. For example, there would be more differential responders with a poor measurement simply because the measurement is not reliable, and thus, it may appear that more individuals are responding differently but this is really a product of a less accurate measurement being used. Therefore, the ability to identify individual responders will be greater when using a measurement that is more accurate, because this will increase the likelihood of an intervention producing individual differences that will exceed that of the measurement error. Having the coefficient of variation or minimal difference is beneficial but is still unlikely to account for all random biological variability that is present unless the two testing sessions are performed over the same duration as the intervention.

Random biological variability can be defined as any type of variability existing in the outcome measure that is not a direct result of the intervention. Using the example used previously in which body mass was weighed twice 1 minute apart, it would be surprising if the two numbers deviated by much, if at all. However, if these two measurements of body mass were taken one day apart, it would be less likely that the exact same body mass values would be obtained because different life events will have occurred in the time spanning the two measurements. Likewise, if these measurements were taken one year apart it would not be surprising if these measurements deviated quite a bit because perhaps the individual had some large life events that occurred (e.g. changing their diet). This example is used to illustrate that it is termed "random biological variability" because it cannot possibly be accounted for in its entirety and illustrates that even though there is no intervention there is still going to inevitably be some level of variability that will exist, and this variability will likely occur to a greater extent if there is a prolonged duration separating the two measurements (63). Therefore, researchers cannot simply have a group of individuals perform an endurance exercise program and conclude that all the variability in the change in body mass was a result of the exercise program because some level of variability would have been present regardless. Thus, to reduce the magnitude of random biological variability that occurs during an exercise intervention, there needs to be some criteria that is given to the participants to ensure they maintain their normal habitual activities as much as possible. Of course, this is still not enough, because as mentioned previously, there still will inevitably be some degree of variability and the only appropriate way to assess the magnitude of true variability is to include a *time matched* control group.

Having the time matched control group is necessary to account for not only measurement error but also random biological variability. Therefore, the only true variability that exists in the

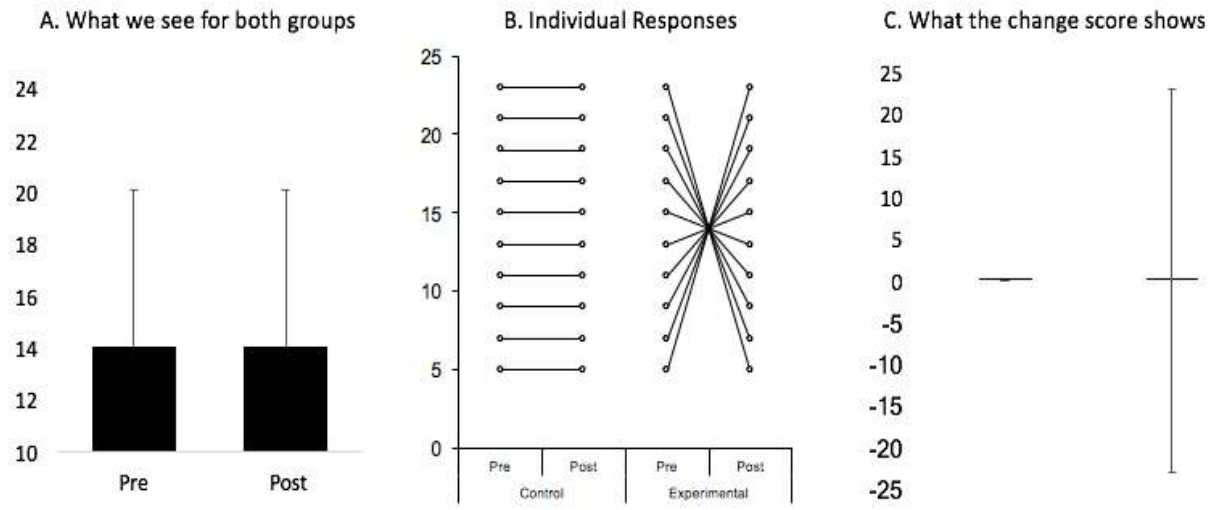
intervention is variability that exists in the experimental group above and beyond that which is present in the control group. This can be calculated by creating a difference score (post value – pre value) for both the control and experimental groups and taking the square root of the difference in squared SD using the following formula: $\sqrt{(SD_{EXP}^2) - (SD_{CON}^2)}$ where SD_{EXP} is the SD of the difference score in the experimental group and SD_{CON} is the SD of the difference score in the control group (9, 64). Therefore, not only must a time matched control group be present to analyze true individual responses, but the SD of the change score must be presented for both the experimental and control groups. While it has been suggested that multiple interventions should be performed to assess whether individual responses are repeatedly observed across studies (10), this is not feasible with various exercise interventions as it is unlikely that certain adaptations will wash out before they are retested (e.g. strength is maintained for long after cessation from resistance training (12).

Reanalyzing studies with time matched control groups

While it is not uncommon for studies to include a time matched control group, studies will often only report that the overall mean of the control group did not change with respect to the outcome measure over the course of the study. Unfortunately, this tells no information about the variability in response to the intervention, and in fact, there can be a large degree of variability within individuals resulting in no change to the overall mean. This is because, as mentioned previously, it is the variability in the *change score* that determines the reliability not whether there is a change in the overall mean. The only reason the mean would increase or decrease would be if there were some type of systematic bias such as a learning effect that occurs with repeated testing. Therefore, studies reporting no changes in the control group do not provide any information on the variability present in the control group.

Similarly, it is very common within the field of exercise science for researchers to report the group SD on the pre and post measures even though the pre and post measurements are taken on the same individuals. While this is appropriate for between subject designs, this provides little meaningful information when testing the same individuals over time because the within subject variability becomes convoluted with the between subject variability that is present. Therefore, providing the pre and post SD provides no information on how individuals responded to the intervention because there is no way to determine which individuals responded in what fashion (Figure 2). Thus, the only way to determine the measurement error/random biological variability that exists over the course of the intervention is to not only include a time matched control group, but also include the variability of the change score within the control group as mentioned previously (9, 64). Not only is this necessary for appropriately examining individual responses, but this practice should also be adopted when reporting pre and post data within the same individuals. After all, people are interested in how variable the intervention is. The variability in the sample to start (the pre SD) and the variability in the sample at the end of the intervention (the post SD) provide little, if any, meaningful information for researchers trying to understand potential differential responses to exercise. Our laboratory has demonstrated this concept previously as it pertains to effect size calculations in that the pooled pre and post test data is highly reflective of the pre SD and in no way reflects the variability of the intervention as this can only be obtained when the variability of the change score is reported (65, 66).

Figure 2. Demonstration of why pre and post standard deviations provide little information



A. Illustrates how the data is commonly represented in manuscripts which may lead many to believe most individuals responded similarly because the standard deviation remains the same. B. An individual response plot illustrating two groups that would both result in the same pre and post graph depicted in A. C. The reporting of the change score and the variability of the change score allows for the reader to not only see the group mean but also how variable the response to the intervention was.

Regression to the mean

The phenomenon of regression to the mean can likely explain a large part of the misreported individual responses to resistance exercise (67, 68). Regression to the mean explains how any fluctuating variable tested within an individual will cause those who score higher or lower on the initial test to then score closer to the group mean on the follow-up test (69). The regression to the mean phenomenon can occur in any variable that fluctuates due to measurement error or natural fluctuations that occur such as circadian rhythms (70). For example, if a group of individuals were to flip a coin 10 times, the group mean with respect to the number of times heads was flipped will likely be five, but there will be some individuals who flip heads nine times and some individuals who flip heads one time. If the same group of individuals were to flip

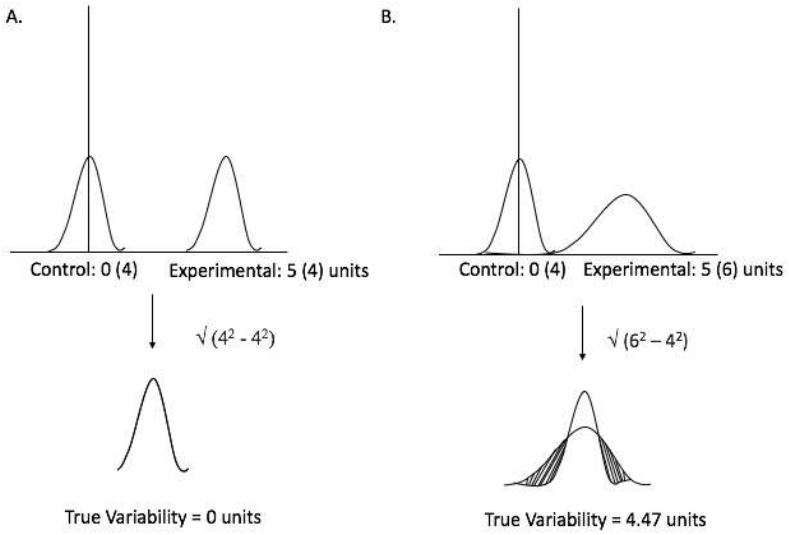
a coin ten times again, it is likely that those who flipped heads once will increase toward the mean, whereas those who flipped heads nine times will decrease toward the group mean. There is no true reason for why these individuals responded any differently from one another other than the fact that they were on one extreme of the measurement during the pre-test measure (in this case the initial coin flip). Examining the data with respect to individual responses would illustrate that some individuals saw large increases (those with low pre-measures) while others had large decreases (those with high pre-measures) and some individuals stayed the same (those who had pre-measures near the group mean). It is important to realize that this has nothing to do with individuals responding differently, but is rather related to fluctuations that occur in random variables that are tested. Therefore, even if the same variable is tested twice without any intervention, it is likely that individuals with the highest pre-value will see the greatest decrease with respect to the outcome variable and those with the lowest pre-value will see the greatest increase. This random variability can be teased out with the inclusion of a time-matched control group.

If there are no individual responders to exercise, what are these studies telling us?

The question can arise as to how there can possibly be an association between different resistance training outcomes and certain genetic variants if there are no true individual responses in the first place. This may be answered by the fact that there are 200,000 mRNA molecules (71) so it is not shocking that some spurious associations may be present. Therefore, it is possible that these associations are simply correlating different mRNA transcripts with measurement error in exercise responses. In other words, it is probable that individuals are indeed getting stronger and increasing muscle mass with training but we cannot detect any difference in how these individuals are responding. Therefore, we are examining changes around the group mean but

these changes are not necessarily real changes, but are rather measurement error or random fluctuations that would have occurred regardless of whether an intervention was employed. Our laboratory has also explained this concept previously in that small changes that occur with resistance training can often result in undistinguishable results from one individual to the next (22). It is again important to focus on the SD in the change that is present rather than simply focusing on the group mean (Figure 3). That is, the mean is irrelevant because it may be that all individuals see similar improvements with respect to exercise outcomes, but these changes cannot be distinguished from one individual to the next. This idea has been demonstrated in a similar fashion previously (10).

Figure 3. The standard deviation of the change score determines individual responders.



A. Illustration of a situation in which the experimental group may increase from baseline with respect to a given outcome measure (e.g. muscle size) but there is no individual variability with respect to the amount of muscle mass gained. Figure B. Illustration of a situation in which the experimental group increases from baselines and there are indeed individual differences in how people respond in the outcome measure (e.g. muscle size). In Figure A the standard deviation curves fit perfectly on top of one another with no differences. In Figure B see the standard deviation curves differ and the striped area illustrates the differential responders that are in the experimental group that were not present in the control group. The focus of this figure is to illustrate that it is the standard deviation, and not the mean, that is important for identifying individual responders.

Current evidence used as support for individual responses to endurance exercise

It should now be understood that any study examining individual responders must include a time-matched control group. Despite this, even the most commonly cited studies (5, 41) (each cited over 600 times) used to support inter-individual variability in response to endurance exercise actually detail a very homogenous response when analyzed appropriately. Using the methods previously discussed for determining individual responders (9, 64), the Bouchard et al. study (5) details that 95% of the sample saw a change in aerobic capacity within ± ~2.5ml/kg/min of one another in response to the same training protocol. This was brought to the forefront in a recent paper (72) in which the author acknowledges in his concluding sentence that, although not difficult, these methods may not be familiar to many authors: "These recommendations will be novel for most researchers, but they are not especially rocket science, and they need to be implemented."

Other commonly cited studies examining individual responses to endurance exercise either fail to have a time-matched control group (42), or do not actually detail any true variability when analyzed appropriately (73). For example, the Hautala et al. study (73) concluded that there is individual variability in the response to changes in aerobic capacity. Here the authors noted an 8% and a 5% increase in endurance capacity following different interventions, but recall the mean is not relevant. The SD of the change in the endurance and resistance training groups respectively were 6% and 5%. The authors reported the SD of the change in the control group to be 6.4% which exceeds that of both experimental groups. Therefore, the entirety of the variability in the change in aerobic capacity can be explained by measurement error and/or random biological variability. For this reason, none of the results from the HERITAGE family studies can be used as support for individual variability in response to training (13). Perhaps

some of the best data available to analyze individual responses to endurance exercise are those not designed to answer this question. This is because any studies that include a time matched control group, and report the variability of the control group, can be re-analyzed to assess the true level of variability present. That is, the magnitude of true variability that exists in a study can be calculated by the reader provided the change scores and SDs are present. A review paper analyzing studies which included the standard deviations of experimental and control groups found that there is little evidence to support a substantial variability in weight loss in response to aerobic exercise interventions (74). This would appear to also hold true across different doses of exercise, such that, more exercise results in more weight loss but the response does not appear to become more variable (75).

To our knowledge, there is only one study that has appropriately assessed the true magnitude of individual variability in response to endurance exercise (76). It was shown that the true variability with respect to improvements in aerobic capacity following high intensity interval training yielded a mean of 254 mL and a standard deviation of 170 mL (76). In other words, the true change in aerobic capacity in response to endurance exercise was 254 mL with 95 percent of the sample falling between -79 mL and 587 mL of oxygen. This is much larger than the magnitude of true variability that could be calculated from previously published studies (as mentioned elsewhere (72)) and may illustrate that different protocols may produce different magnitudes of true individual variability. That is, the estimated true variability in the change in aerobic capacity following traditional endurance exercise in the Bouchard et al. study (5) demonstrated that 95% of the sample fell within ± ~2.5 ml/kg/min, whereas the Phillips et al. study (76) demonstrated that 95% of the sample fell within ± ~3.5 ml/kg/min in response to high intensity interval training. Support for this hypothesis would appear to exist as there are

differential responses to traditional continuous training and sprint interval training performed after a 3 month washout period (77), but we again do not know if these differential responses are simply the result of random error.

Current evidence supporting individual responses to resistance exercise

The most commonly cited study reporting individualized responses to resistance exercise is the aforementioned study by Hubal et al (6). This study details that the entirety of the variability in isometric and isotonic strength could simply be explained by random biological variability and/or measurement error (given the control group expressed the same variability on the change in strength from baseline). The variability in muscle size used in this study could not be appropriately assessed as it was reported as "0.0" in the control group which is unrealistic. When using the percentage change results, the true variability was 0.26% muscle size, 0.74% for isokinetic strength, and 1.14% for one-repetition maximum (1RM) strength. Therefore, the magnitude of variability that can be attributed to the intervention is very small and may be, in part, related to the fact that there were eight collaborating exercise sites. This may have caused a greater variability based on differences employed between testing sites as opposed to true variability in the exercise response between individuals.

Several studies have been conducted to analyze the importance of satellite cell differentiation and its effects on muscle hypertrophy. It was previously shown that the magnitude of muscle mass gained during a resistance exercise intervention was proportional to the magnitude of new myonuclei accrued (45, 46). Furthermore, the same laboratory illustrated that both baseline satellite cell number (46), and increases in myogenin gene expression via resistance exercise (47), were associated with greater training induced increases in muscle mass. Contrarily, differences in diet were not associated with the magnitude of muscle hypertrophy

(78). Other studies comparing high and low responders to exercise have found differential changes in micro RNA expression (19) and myozenin-1 protein levels (79), as well as baseline differences in androgen content (18) and markers of inflammation (80, 81). Therefore, potential differences in how individuals respond to resistance exercise appear to be related to genetic differences as opposed to some extraneous variable that is not accounted for outside of the intervention. While these studies provide a rationale for why there may be individual responders to resistance exercise, it remains unknown how much of the analyzed variability in these studies exceeded measurement error/random biological variability. This may be why it is difficult to replicate some of the findings suggesting that high responders to resistance exercise have different androgen receptor contents and myonuclear content (82).

One study observing individual responses to resistance exercise found greater variability in the squat jump and acceleration when comparing traditional exercise to that of a control group; however measures of muscle size and strength were not assessed (83). Another study which did include a control group concluded that individual responses were present in both maximal aerobic capacity and isometric knee extension force in response to either aerobic training, resistance training, or a combination of both aerobic and resistance training (84). Interestingly, when looking at Figure 1 in the Karavirta et al. paper (84), it would seemingly illustrate a larger variability in the experimental groups relative to the control group, but this again may be deceiving as a look at the reported data reveals that there actually is no variability over that of the control group. For example, the 95% confidence intervals for the three experimental groups spanned 9, 9, and 7 units for males across the experimental groups for aerobic capacity but the control group spanned 11 units (i.e. the control group had a greater variability). For females, the three experimental groups spanned 12, 12 and 9 units, whereas the

control group spanned 12 units, again illustrating no true variability in aerobic capacity. Similarly, with respect to isometric knee extension strength, males in the three experimental groups spanned 9, 9, and 12 units compared to the control group which spanned 11 units. For females, the experimental groups spanned 13, 14, and 18 units, whereas the control group spanned 13 units. Collectively, these results illustrate that if there is some variability in the response to changes in strength with resistance training it is likely difficult to detect above measurement error/random biological variability. This also details that a lot of the variability in the data set that was used to correlate the changes in aerobic capacity with that of strength gains, is probably simply correlating random biological variability/measurement error that is present amongst both variables (our laboratory has explained this concept in further detail elsewhere (22)). While this study presents the data nicely, the conclusion of there being heterogeneity in the exercise response does not match the data, since the same heterogeneity would have been present even if the individuals did not exercise (i.e. it is random error). Other studies have demonstrated that there may be a large degree of true variability in response to strength gains from resistance exercise in young and elderly individuals, but the lack of a time matched control group detailing the meaningfulness of the true variability limits the interpretation of these studies (85, 86).

To our knowledge, there is only one study existing that has provided some reasonable evidence that individual responses to resistance exercise exist (17). The authors report a 21.1% (SD: 11.5%) mean change for muscle strength and a 4.8% (SD: 6.1%) mean change for muscle size in the experimental group. In comparison, the control group demonstrated a 3.5% (SD: 5.9%) mean change in muscle strength and a 0.5% (SD: 4.8%) mean change in muscle size. Therefore, using the methods described previously (9, 64), the true individual variability that can be attributed to the intervention was 9.87% for muscle strength and 3.76% for muscle size. This

illustrates a reasonably large degree of variability particularly given its magnitude relative to the mean changes in the intervention previously mentioned. This study, however, is not without limitations. To obtain a large sample size (n=359) in the Ahtiainen et al. study (17), several original research studies, which used a variety of different measurement techniques for assessing muscle size, were pooled together. While these measurement techniques may track similarly over time (20), there may be differences in the ability to detect muscle growth across measurements. For example, it was previously shown that the ultrasound appears to overestimate the magnitude of muscle growth relative to that of the MRI (87). Therefore, this differential sensitivity to changes in muscle size may have added a degree of variability to the experimental group in the Ahtiainen et al. study (17) because changes in muscle size across all individuals were pooled together using different measurement techniques. Thus, even if muscle mass had increased to the same extent in all individuals, some degree of variability would be introduced in the training group due to differences in the sensitivity of each measurement. This artificially inflated variability in the training group would not be accounted for in the control group given that muscle size would not be expected to change appreciably in the control group, and thus no added variability across measurements would be present (i.e. the differential sensitivity exists in the change in muscle size and would not impact a simple test-retest). This details that the most appropriate assessment of individual responders would likely involve a large sample size in which all individuals are assessed using the same measurement technique. Lastly, it is important to consider that different measurement techniques have different degrees of error. To illustrate, coefficients of variation for muscle size (lean mass for dual X-ray absorptiometry (DXA)) measurements were previously reported as 4.3% for ultrasound, 2.1% for magnetic resonance imaging (MRI) and 1.0% for DXA (17). This is an important consideration as the ability to

detect differential responders is dependent upon measurement error present (i.e. the ability to detect true individual responses will be greater when less measurement error is present).

It is likely that the most informative studies demonstrating potential individual responses to resistance exercise can be found in studies that are not intending to look at individual responses. Clearly an appropriately designed study intending to look at individual responses while incorporating a time matched control group would be best, but many of the studies examining individual responses do not include time matched control groups. Conversely, other resistance training interventions are not purposely designed to examine individual responses but do indeed incorporate time matched control groups. For example, measuring cross sectional area of muscle tissue taken via biopsy revealed that there did appear to be heterogeneity in type I and type II muscle fiber hypertrophy (88). Interestingly, the magnitude of variability appeared to be greater in type I fibers compared to that of type II muscle fibers (when examining the error bars of the change in Figures 3 and 4 of the Hartman et al. study (88)). This information can only be extracted from the paper because the authors report the variability of the change score in both the control and experimental group. Given that this information is rarely reported in resistance training interventions, there are limited studies to analyze, and furthermore, these studies often contain inadequate sample sizes to appropriately assess differential responders as this is not the focus of the research project.

Classifying responders and non-responders

In addition to the lack of currently published studies illustrating true variability (i.e. above that of a time matched control group), the classification of individual responders also tends to be flawed. For example, one study concluded that there are no non-responders to resistance exercise in that each individual responded favorably to at least one of the six variables

measured (89). This has led to the suggestion that individuals will likely respond with some favorable adaptations (90). This is problematic, however, as the authors did not take into context the reliability of their measurement. For example, since it would be rare to get the exact same measurement twice, the probability of an individual not having any magnitude of improvement for at least one of these variables would be very slim, or more precisely the same probability of flipping tails on a coin 6 straight times which equates to 1.5% (0.5^6). Thus, by chance it is likely that every individual would have been a positive responder in at least one variable even if the individuals did not undergo any intervention and were simply tested multiple times. Even when considering the magnitude of measurement error, it has been shown in elderly women that all participants are likely to see a measurable and true improvement in 1RM strength in response to isotonic resistance exercise (91). The same does not hold true, however, when isotonic resistance exercise is performed and strength is assessed using isokinetic dynamometry (85), illustrating the importance of the specific strength measure being used. This is particularly apparent in that the classification of an individual as a responder or non-responder in a given variable (i.e. strength) may differ based on the measurement used (i.e. isokinetic strength vs. dynamic 1RM strength) (86). Conversely, another study reported a range of between 8-13% of individuals responding adversely (>2 standard deviation units on a test-retest spanning a 3 day period) to exercise depending on the outcome being assessed (92). Notably, none of the aforementioned studies took into account the random biological variability present over the same duration of the study (85, 91, 92). These differential findings can at least partially be explained by the variables being tested and the differences in the analyses computed.

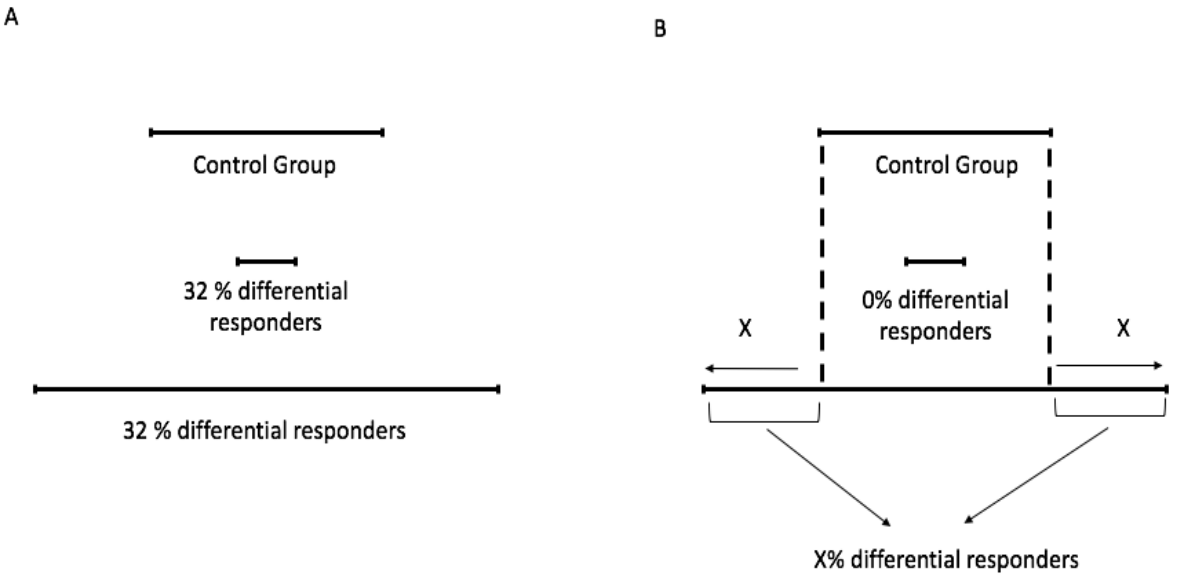
A similar analytic problem exists in another study determining there are no non-responders to endurance exercise (93). The study design only required those who did not exceed

the minimal difference to continue performing higher quantities of exercise and concluded that all individuals are responders to exercise provided a great enough exercise volume is performed. One major limitation with this study (93) is that it did not include those individuals who were responders to exercise after the first six weeks. Therefore, the analysis is subject to the regression to the mean phenomenon mentioned previously in that those individuals whose aerobic capacities were overestimated at the initial post-measure may have been more likely to be classified as responders and did not undergo further testing, yet those whose aerobic capacities were underestimated at the post-training time point were then required to perform additional training. Of course, it is also plausible that the individuals classified as responders would have continued to improve but this is unknown as these individuals were not retained in the analysis.

Another potential issue exists in that some studies determine responders and non-responders through the use of SD units from the mean change in the experimental group (16, 17). This is problematic because by default there will always be approximately 32% of the sample that will be classified as differential responders with 16% being low responders and 16% being high responders (provided the data is normally distributed), and this can be observed in such analyses (16, 17). Thus, even if everyone responds in an almost identical fashion there will still be the same number of high responders as another study which has a large true variability in the response to the intervention. For this reason, it would seemingly be more appropriate to use the variability of the change score in the control group (representing measurement error/random biological variability) for the classification of responders and non-responders. Similar to that of a traditional statistical test, one can then use 1.96 SD units to establish a criteria for what type of change would be unlikely to occur by chance (62). This would then allow for the ability to differentiate responders based on the level of variability that exists in the data set that is

unrelated to the intervention. If there is limited measurement error/random biological variability, then the ability to differentiate the responses from person to person will be great and may exceed 32%. Likewise, if there is a tremendous amount of error/random biological variability (a high SD on the change score of the control group), it may not be possible to declare anyone as an individual responder and this is more appropriate than falsely classifying 32% of the individuals as differential responders. This example is illustrated in Figure 4.

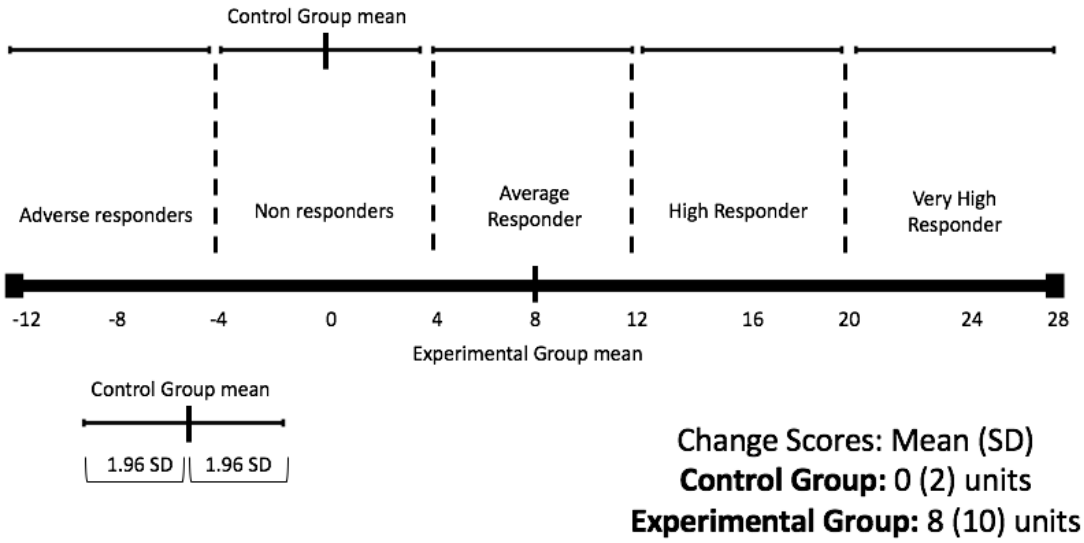
Figure 4. Using standard deviation units to classify individual responders



We propose a more appropriate method for classifying differential responders using a similar concept to what is used for determining the magnitude of variable present (9, 64). A. The commonly employed method of using standard deviation units from the change score in the experimental group. This method appears problematic as there will always be approximately 16% of high responders and 16% of low responders regardless of the variability present in the sample. This method negates the ability to tease out error because the control group is not used. B. The standard deviation of the change score of the control group details a more appropriate method of classifying differential responders because it is dependent on the ability to tease out variability that is unrelated to the intervention. Any individual that responds in a fashion that exceeds 1.96 standard deviation units from the mean of the control group (i.e. the 95% confidence interval) can be properly detected as an individual responder given they are unlikely to exceed the random biological variable/measurement error by chance alone.

If the true variability in the data set is very high, we propose that additional cut points can be established. For example, the equation: [1.96 * SD of change in control group] can be used to establish further cut points into numerous categories that are all each significantly different from one another. That is, any individual response that exceeds 1.96 SD units from a given point can be classified as statistically different. This concept is illustrated in Figure 5.

Figure 5. Classifying individual responders from one another



Responders to exercise can be classified as differential responders from one another. We propose this based on traditional concepts of statistical testing. The standard deviation of the change score in the control group illustrates the magnitude of random biological variable/error. Therefore, two standard deviation units would illustrate a magnitude of change that would be less than 5% likely to occur be explained by random chance. The two standard deviation units in the figure are shown as 95% confidence interval bars on either side of the mean. Given that the change in an outcome mean of the experimental group is likely to be higher than that of the control group, the means are not in alignment. The 1.96 standard deviation units of the change in the control group illustrates the distance by which two points must be separated to be considered different from one another. This allows for the differentiation of responders based on the magnitude of the response present. Note that in this figure all high responders are statistically significantly different than all non-responders, however, not all high responders are statistically significantly different than all average responders. The figure illustrates that any high responder would be statistically significantly different that the mean of the experimental group. This illustrates a gray area in that individuals who are above the group mean but not 1.96 SD from the group mean may not be statistically significantly different that either an average responder or a high responder.

The push for individualized exercise programming

The idea that there are indeed individual responses to being prescribed the same exercise intervention has led many to suggest that exercise programming should be individualized based on genetic differences (7, 60). However, even when individualized exercise programs are prescribed to have individuals train at not only the same relative intensity, but also the most beneficial time day, there still appears to be a large degree of variability in response to the exercise intervention (94). Of course, as previously mentioned, the relevance of this variability is unknown due to the lack of a time matched control group. Therefore, personalizing exercise programming based on differences in how individuals respond to training may not be necessary as this operates under the assumption that these individual differences exist, but the magnitude and meaningfulness of these potential differences has yet to be shown. On the other hand, personalizing exercise programming probably makes sense based on differences in outcomes that are measured at baseline because these differences between individuals are much larger. This can easily be done by prescribing exercise relative to the individual's capability such as a prescribing a percentage of 1RM or percentage of aerobic capacity. While these differences at baseline can be deciphered from one individual to the next, this level of variability is likely to far exceed that which is present in response to training (22). Therefore, while it may be important to individualize exercise programs based on differences at baseline, it may not make sense to individualize exercise programs based on how individuals respond to exercise, since we are again unsure if we can decipher these responses from one another.

Is a high responder in one variable likely to be a high responder in all variables?

Within the aerobic exercise literature it is said that a high responder in one outcome variable may not be a high responder in another outcome variable (58, 95, 96). Within the

resistance training literature a similar conclusion can be drawn in that the change in strength was not correlated with the change in aerobic capacity (84), and similarly, the change in muscle size was not correlated with the change in strength (17). All of the aforementioned studies may have simply been correlating random biological variability/measurement error between two variables, as they did not decipher high responders while taking into account the variability of the control group (as mentioned in Figure 4). Therefore, there currently is no good evidence for or against a universally high responder, but it would seem plausible that some individuals may simply be more adaptable in all aspects relative to others.

Resistance training protocols

Our laboratory has previously recommended that resistance training protocols should always be performed to volitional failure when analyzing individual responses, particularly as it relates to muscle hypertrophy (97). Specifically, there are drastic differences in the number of repetitions that individuals can complete at either low (range: 18-73 repetitions at 35% 1RM) (28) or high (range: 9-18 repetitions at 70% 1RM) (98) loads. Therefore, prescribing a set number of repetitions may result in the protocol providing a different stimulus across individuals as opposed to differences in response to the same stimulus.

Our laboratory has recently been experimenting with repeated 1RM testing in which each training bout consists of simply performing the 1RM test (14, 15). We began using this method of training in response to a study illustrating large increases in strength from performing 1RM testing in addition to five sets of exercise performed for 35 straight days (99). Given large strength increases (12-22%) observed from simply performing the 1RM test (100), we sought to observe whether the findings from Zourdos et al (99) could simply be explained by repeated practice of the 1RM test (14). Indeed, these strength increases from simply performing the 1RM

test have been observed in both trained (101, 102) and untrained individuals (100, 102, 103), and often do not begin to plateau until 8 familiarization sessions have been completed (100). We found similar results in that simply performing the 1RM test resulted in large increases in 1RM strength and this was not augmented by additional exercise volume (an additional 3 sets of exercise) (14). Another study form our laboratory illustrated similar strength increases upon completion of repeated 1RM testing when compared to a more traditional exercise protocol in which individuals trained at 70% of their 1RM (15). It is plausible to hypothesize that repeated 1RM training would result in a more homogeneous strength increase. While the variability of the 1RM and traditional exercise groups did not appear to differ in the previous study (15), the sample size was likely insufficient to make such conclusions. Support for this hypothesis exists in that a previous study demonstrated different intensities of aerobic exercise result in different magnitudes of variability for changes in waist circumference (104), providing some support that the different exercise loads used in the present study may alter the level of true variability present. It has also been suggested that some individuals respond more favorably to different volumes of exercise (105), but we do not know if any of this variability can truly be attributed to different volumes of exercise or simply random error.

CHAPTER 3: METHODS

Participants

A total of 158 individuals (60 males and 98 females) were recruited for participation in this study. Inclusion criteria was as follows: (1) must be between the ages of 18-35 years; (2) cannot have any orthopedic injuries preventing individuals from performing elbow flexion exercise; (3) must not be regularly engaging in resistance exercise within the previous six months; and (4) cannot use tobacco products. This sample size that was recruited was larger than what is typically used to detect a significant group difference because we were attempting to discern individual variability in the exercise response. The sample of 50 individuals per group was sufficient to detect a ratio of variance equal to 2.24, assuming an alpha of 0.05 and power of 0.8. This meant that the SD of the intervention group would be significant if it was 1.5 times that of the control group ($\sqrt{2.24}$). This was reasonable to expect given the results of a previous study (17) noted a ratio of variance of 3.80 (ratio of SD was 1.95) for muscle strength and 1.61 for muscle size (ratio of SD was 1.32). We additionally used Bayesian inference to help determine if any lack of statistical significance was due to an insufficient sample size (and thus an ambiguous Bayes factor) or if this was due to the null hypothesis being a better fit for the data. For a secondary aim of the present study, which was designed to compare means across groups, a sensitivity analysis revealed that, with a power of 0.8 and alpha of 0.05, the present sample of 50 individuals in each group was sufficient to detect an effect size (f) of 0.25 (i.e. a moderate effect for the ANOVA). This is a conservative sensitivity analysis as the ANCOVA

employed helped to reduce unexplained error variance given the large correlation between pre and post test measures of the dependent variables.

Study design

Participants in the experimental group reported to the laboratory on 20 separate occasions (18 training, 1 pre-testing and 1 post-testing); whereas the control group completed 2 total sessions (1 pre-testing and 1 post-testing). On visit one participants filled out initial paperwork to ensure they were eligible for participation. Following, the completion of the pre-testing visits, participants were assigned to one of three groups: (1) a traditional training group; (2) a 1RM training group; and (3) a non-exercise control group. The allocation of group assignment was done using covariate adaptive randomization to ensure all groups had similar pre-testing values with respect to 1RM strength. The allocation of individuals to each group was done using a computer program supplied at request to the authors of a previously published paper (106). Briefly, the first 12 males and females were each randomly assigned to a group by using a random number generator. Based on strength data from these individuals, we then categorized all subsequent individuals into quartiles of strength by using 0.675 z scores above and below the mean of this sample as this z score corresponds to the 25^{th} and 75^{th} percentile, respectively, when the data are normally distributed. That is, the 1^{st} strength quartile was classified as being <0.675 standard deviations below the mean, the 2^{nd} strength quartile was classified as being below the mean but no less than 0.675 standard deviations below the mean, the 3^{rd} strength quartile was classified as being above the mean but no more than 0.675 standard deviations above the mean, and the 4^{th} strength quartile was classified as being at least 0.675 standard deviations above the mean. This randomization was done after individuals completed their pre-testing visit to ensure that neither the researchers nor the participants performed differently because of their group

assignment. The pre and post testing visits consisted of measuring height, body mass, muscle thickness, isotonic strength, and isokinetic strength.

Muscle thickness

Ultrasound measurements (Logiq e, General Electric, Fairfield, CT) of muscle thickness were made at the front of the participant's upper arm. The probe (Logiq e, L4-12t probe, General Electric, Fairfield, CT) was coated with gel and held transversely against the skin. Measurements were taken in duplicate and saved for later analysis in a blinded fashion. Measurements were taken at each 50, 60 and 70% the distance between the acromion process and lateral epicondyle of the arm.

One repetition maximum (1RM) strength

Maximum concentric strength (the heaviest weight that can be lifted one time) of the participants' arms using the unilateral elbow flexion exercise completed with dumbbells was tested. This measurement was made to set the workload for the exercise bouts and also to assess pre and post strength changes. The participant completed the same protocol on the both arms. All 1RM attempts were separated by 90 seconds of rest and were performed with the individuals back and heels against a wall to ensure strict form. All 1RMs were measured to the nearest 0.2 kg and were usually obtained in around 5 attempts. In a randomized fashion, the 1RM was completed in its entirety on one arm, before continuing to test 1RM strength in the contralateral arm.

Isokinetic strength

Participants were seated on a dynamometer (Biodex Medical Systems, Shirley, New York, USA) with the seat and lever arm adjusted appropriately and the settings recorded and standardized for all future tests. After weighing the individuals arm to correct for gravity,

participants then perform three consecutive isokinetic contractions at 60°/s and then rested for 60 seconds before performing another three contractions at the same speed. The range of motion for the isokinetic test was performed from 10° to 100° of elbow flexion, with 0° representing full elbow extension. During all contractions, the individuals were provided with verbal and visual feedback to encourage them to contract as hard as possible. The highest torque value the individual could produce was be recorded as their peak torque value for analysis. This was performed on both arms.

Training protocol

The training visits occurred three times per week on non-consecutive days and were separated by at least 24 hours. Each session consisted of performing elbow flexion exercise of the dominant arm. The traditional training group performed four sets to volitional failure using a load initially corresponding to 70% of their pre-determined 1RM. After the first set of exercises were completed on the first training visit, the load was adjusted to ensure individuals would reach volitional failure between 8-12 repetitions on all subsequent sets for the duration of the training intervention. Ninety seconds of rest was allotted between sets. The 1RM training group performed up to 5 individual repetitions each visit, starting with a load corresponding to approximately 85% of the individual's 1RM. The load was then progressively increased until individuals either failed on one of the attempts, or they completed 5 successful attempts. The goal was to try and match their previous 1RM on the fourth attempt and then exceed this value on the fifth attempt. Each of the repetitions were separated by 90 seconds of rest. The dominant arm (assessed as the hand individuals write with) was chosen to be trained in order to also observe the magnitude of the cross-over effect which has been proposed to occur to a greater extent in the non-dominant arm (107).

Frequentist statistical analysis

All frequentist statistics were performed using SPSS 22.0 statistical software package (IBM, SPSS Inc.; Chicago, IL). For inter-individual responses, a variable was created as the change score (post-pre) for all outcomes, and the standard deviation of this change was compared between the control and each experimental group to detail the true inter-individual response. We chose to examine the true level of individual variability only if there was a significant equality of variance test (i.e. Levene's test) indicating that the variability differed between the intervention groups and the control group. The true inter-individual variability was calculated using the formula $[\sqrt{SD_I^2 - SD_C^2}]$, where SD_I^2 represents the squared standard deviation of the change score in the intervention group, and SD_C^2 represents the squared standard deviation of the change score in the control group. Furthermore, we assessed the number of individuals who exceed the variability present in the control group with the goal of determining if there are universal high/low responders, and to compare this value to other commonly used methods. High and low responders were classified as those in the experimental group who had a change score exceeding 1.96 SD of the control group (i.e. they have a true differential response exceeding measurement error/random biological variability). Depending on the magnitude of true inter-individual variability that is present, we chose, a prori, to create multiple groups each consisting of a further 1.96 SD unit increment apart if there are a lot of individuals that are found to be 1.96 SD away from the mean. Additionally, two separate one way ANCOVA's were performed with baseline values as a covariate to determine whether the changes in variables (e.g. muscle thickness, 1RM strength and isokinetic strength) differed by group. One model was used to test the dominant arm and another model was used to test the non-dominant arm. We additionally tested whether the difference in strength gains (i.e. change scores from baseline)

differed between the trained and untrained arms within each group using paired t tests. This was done to assess whether the untrained arm increased strength as much as the trained arm within each the control, 1RM, and traditional exercise groups. Statistical significance was set at an alpha level of 0.05.

Bayesian statistical analysis

In addition to the frequentist statistics, all statistical analyses were also performed using an analogous test with a Bayesian statistical approach. Bayesian inference quantifies evidence for or against the null hypothesis using Bayes factors (BF). One of the main benefits of Bayesian statistics, particularly in the present study, is that support can be provided for the null hypothesis which cannot be done using a traditional frequentist approach. This is of importance, because a non-significant test does not necessarily indicate equality across groups being compared, it simply means that the groups are unlikely to be different. It is entirely plausible to have the results of a study provide no support for the null or alternative hypothesis, indicating that the results of the study are ambiguous and require larger sample sizes to make a definitive conclusion for which model fits the data better. Thus, the Bayesian analysis helped us determine whether any insignificant tests (particularly Levene's tests) were not statistically significant due to an insufficient sample size (resulting in an ambiguous BF) or because the null hypothesis was supported.

All Bayesian statistical analyses were performed using JASP version 0.9.2 (JASP Team (2018); Amsterdam, The Netherlands). Bayes factors were used to provide support for the null ($BF \leq 0.33$) or alternate hypotheses ($BF \geq 3$) such that the BF is calculated by dividing the support for the alternative hypothesis by the support for the null hypothesis. A BF in in the range of 0.34 to 2.99 represents ambiguity. For those unfamiliar with Bayesian statistics, a BF of 3, for

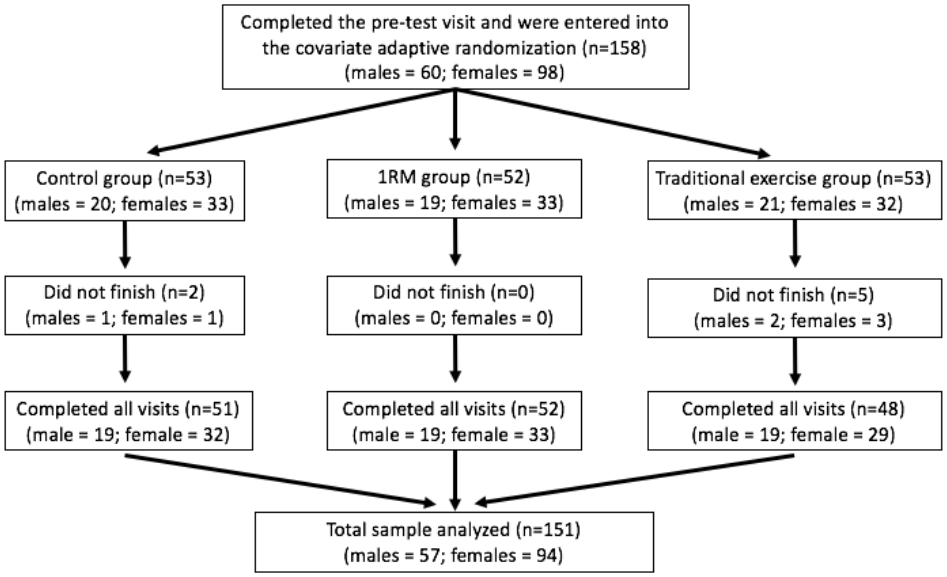
example, indicates the observed data are 3 times more likely under the alternative than the null hypothesis, while a BF of 0.33, for example, indicates the observed data are 3 times more likely (synonymous with saying the alternative is 0.33 times as likely) under the null than the alternative hypothesis. All Bayesian analyses were conducted using uninformed priors as recommended previously (108). To obtain a BF for the Levene's tests, the F value obtained from the Levene's test in SPSS was converted to a t statistic and then this information was used in JASP to obtain a BF. For differences in mean values, Bayesian ANCOVAs were used to test for group (control, 1RM, traditional) differences with respect to the change scores for muscle thickness and muscle strength while adjusting for the pre-test values. Here the covariate was added to the null hypothesis to assess the impact of group after accounting for the covariate. We additionally compared the magnitude of change in the trained and untrained arm within each group using Bayesian paired t tests. For the Bayesian ANCOVAs that were performed, the uninformed prior was r=0.5 for fixed effects and r=0.354 for the baseline covariate. For Bayesian paired and unpaired t tests, an uninformed prior width of 0.707 which was centered around 0 was used.

CHAPTER 4: RESULTS

Sample size and descriptive statistics

A total of 151 out of the recruited 158 individuals (96%) completed the study. There were 3 males (2 in the traditional exercise group and 1 in the control group) and 4 females (3 in the traditional exercise group and 1 in the control group) that dropped out of the study for unrelated reasons. A flow chart illustrating the number of individuals who were recruited and those who completed the study is shown in Figure 6. A breakdown of the number of males and females within each group is shown in Table 1. Overall, there were slightly more males than females, but each group had a similar proportion of males and females.

Figure 6. Flow chart showing individuals who completed the study



A total of 151 of the initial 158 individuals that completed the pre-test visit were included in the final analysis. Only those individuals that completed the pre-test and post-test visits were included.

Table 1. Participants in each group

	Control	1RM	Traditional	Total
Male	19	19	19	57
Female	32	33	29	94
Total	51	52	48	151

The table indicates individuals that completed all measurements. There were 3 males and 4 females who did not complete the post-testing visit. This resulted in 5 fewer people in the traditional exercise group and 2 fewer people in the control group.

Descriptive statistics are shown in Table 2. While these groups were randomized according to initial 1RM strength of the dominant arm, there were no apparent differences in any of the baseline variables. We did not test for any baseline difference across groups since the groups were randomized, and thus, by definition, any differences would be the result of random chance making any statistically significant findings a type 1 error (109).

Exercise volume and repetitions completed

The total repetitions of exercise completed by the training groups are shown in Table 3 and Figure 6, while the total volume completed is shown in Table 4 and Figure 7. By design the total repetitions completed and exercise volume was greatest in the traditional exercise group. Specifically, the average number of repetitions completed by the traditional exercise group was 732 (SD: 72), while it was 76 (SD: 6) for the 1RM group. As for the exercise volume, the traditional exercise group completed an average of 7,244 (SD: 2,150) kg, while the 1RM training group completed an average of 1,008 (SD: 390) kg. When broken down by individual exercise session, the traditional exercise group averaged 40.6 (SD: 4.0) repetitions and 402 (SD: 119) kg of volume, while the 1RM training group averaged 4.2 (SD: 0.3) repetitions and 56 (SD: 21) kg

of volume. The control group is not included in the tables and figures displaying repetitions and exercise volume since they did not exercise and these values would simply be 0. No statistical analyses were computed to compare repetitions and exercise volume as these values are meant to be used as descriptive (as opposed to inferential) statistics.

Table 2. Descriptive Statistics

	Control	1RM	Traditional
Age (years)	21 (3)	20 (1)	21 (2)
Height (cm)	168 (9)	169 (13)	169 (9)
Weight (kg)	72.2 (15.7)	74.6 (22.0)	72.6 (17.2)
Muscle thickness dominant 50% (cm)	2.66 (0.57)	2.72 (0.57)	2.79 (0.61)
Muscle thickness dominant 60% (cm)	2.88 (0.56)	2.92 (0.56)	2.99 (0.57)
Muscle thickness dominant 70% (cm)	3.22 (0.57)	3.27 (0.60)	3.32 (0.61)
Muscle thickness non-dominant 50% (cm)	2.61 (0.60)	2.65 (0.54)	2.71 (0.66)
Muscle thickness non-dominant 60% (cm)	2.78 (0.58)	2.83 (0.55)	2.89 (0.62)
Muscle thickness non-dominant 70% (cm)	3.10 (0.60)	3.18 (0.59)	3.22 (0.61)
1RM dominant (kg)	13.5 (4.6)	13.3 (4.5)	13.6 (5.2)
1RM non-dominant (kg)	12.9 (4.4)	12.7 (4.5)	12.9 (4.9)
Isokinetic dominant (Nm)	35.4 (12.7)	36.9 (14.2)	36.6 (12.4)
Isokinetic non-dominant (Nm)	32.4 (12.6)	34.9 (14.2)	34.5 (12.2)

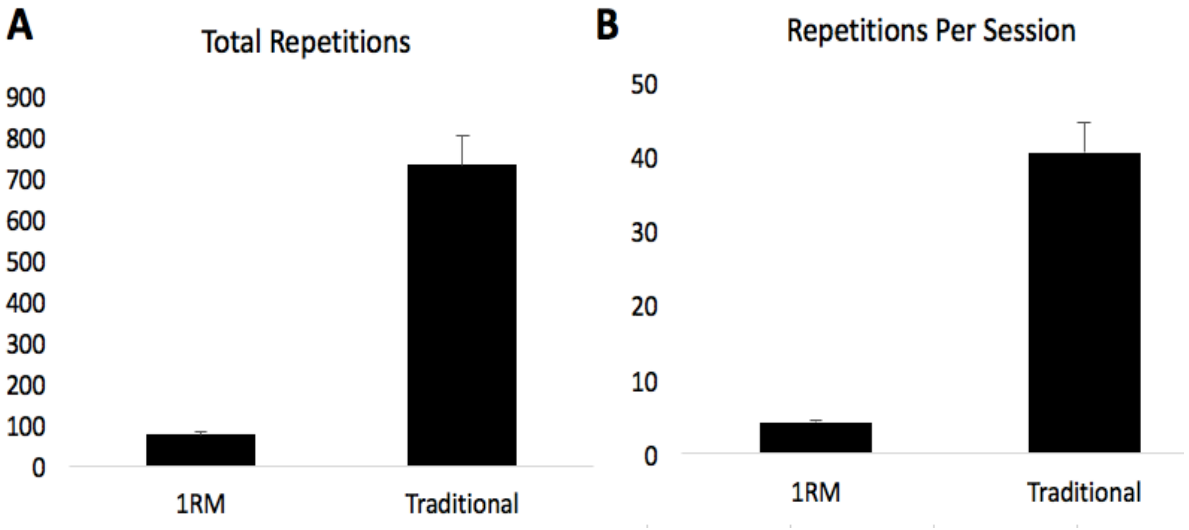
Data expressed as means and standard deviations.

Table 3. Repetitions completed

	Traditional	1RM
Total repetitions	732 (72)	76 (6)
Males	710 (80)	77 (5)
Females	746 (64)	76 (6)
Total repetitions per session	40.6 (4.0)	4.2 (0.3)
Males	39.4 (4.4)	4.2 (0.3)
Females	41.4 (3.6)	4.2 (0.3)

Values are expressed as means and standard deviations.

Figure 7. Repetitions completed



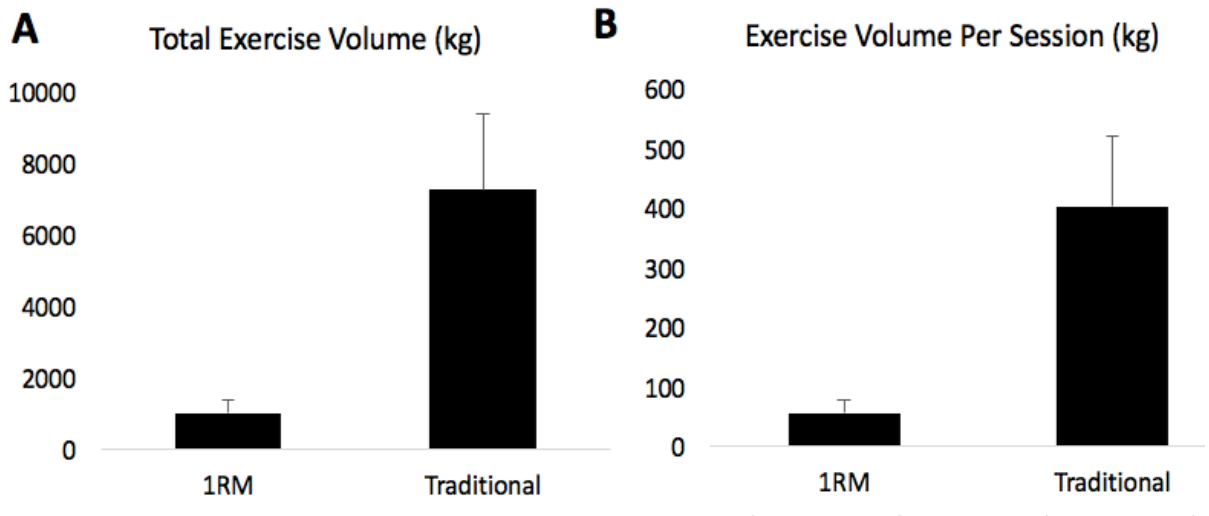
Values are expressed as means and standard deviations. A) Total repetitions completed over the entirety of the training study. B) Average number of repetitions completed per individual training session. The control group is not presented because no exercise was performed and these values would simply be 0. No statistical analyses were computed as these values are intended to be used as descriptive statistics.

Table 4. Exercise volume

	Traditional	1RM
Total volume (kg)	7,244 (2,150)	1,008 (390)
Males	8,795 (2,181)	1,383 (344)
Females	6,227 (1,413)	791 (211)
Total volume per session (kg)	402 (119)	56 (21)
Males	488 (121)	76 (19)
Females	345 (78)	43 (11)

Values are expressed as means and standard deviations.

Figure 8. Exercise volume



Values are expressed as means and standard deviations. A) Total volume completed over the entirety of the training study. B) Average volume completed per individual training session. The control group is not presented because no exercise was performed and these values would simply be 0. No statistical analyses were computed as these values are intended to be used as descriptive statistics.

Mean changes in muscle size of the trained arm

The absolute changes in muscle size of the dominant arm are shown in Table 5 and Figure 8, while the relative changes are shown in Table 6 and Figure 9. When examining the 50% site, the results of the frequentist ANCOVA (i.e. independent variable = group, dependent variable = 50% site change, covariate = 50% site pre-value) revealed there was a significant difference between groups ($p<0.001$). Follow-up comparisons detailed that the traditional exercise group had greater muscle growth compared to both the control ($p<0.001$) and 1RM ($p<0.001$) training groups. There was no increase in muscle thickness above that of the control group for the 1RM training group ($p=0.190$). Adjusted change scores and 95% confidence intervals for muscle thickness at the 50% site for each of the groups were as follows: control = 0.00 (95% CI: -0.05, 0.06) cm; 1RM = 0.06 (95% CI: 0.00, 0.12) cm; and traditional = 0.23 (95% CI: 0.17, 0.29) cm. The results of the Bayesian ANCOVA with an uninformed prior at the 50% site (i.e. independent variable = group, dependent variable = 50% site change, covariate = 50% site pre-value) detailed support for the alternative hypothesis (BF = 7,482). Post-hoc comparisons found strong support for a greater increase in muscle thickness in the traditional exercise group relative to that of the control (BF = 3,733) and 1RM (BF = 62) training groups. The results were ambiguous when examining changes in muscle size between the 1RM and control group (BF = 0.646). Thus, for the 50% site the frequentist and Bayesian statistical approaches yield similar results in illustrating that only the traditional training group increased muscle thickness.

At the 60% site, the results of the frequentist ANCOVA (i.e. independent variable = group, dependent variable = 60% site change, covariate = 60% site pre-value) revealed there was a significant difference between groups ($p<0.001$). Pairwise comparisons showed that the

traditional exercise group increased muscle size above that of both the control ($p<0.001$) and 1RM ($p=0.001$) groups. There was no difference between the control and 1RM training groups ($p=0.0503$). Specifically, the adjusted change scores from baseline at the 60% site were as follows: control = 0.02 (95% CI: -0.03, 0.08) cm; 1RM = 0.11 (95% CI: 0.05, 0.17) cm; traditional = 0.26 (95% CI: 0.20, 0.32) cm. The results of the Bayesian ANCOVA with an uninformed prior at the 60% site (i.e. independent variable = group, dependent variable = 60% site change, covariate = 60% site pre-value) detailed support for the alternative hypothesis (BF = 13,743). Follow-up comparisons showed that the traditional exercise group increased above that of the control (BF = 7,184) and 1RM (BF = 26.0) groups. There results for the comparison of muscle growth between the 1RM training group and the control group were ambiguous (BF = 2.32). Similar to that of the 50% site, muscle growth was only present in the traditional training group when compared to the control group. The interpretation of the results did not differ between the Bayesian and frequentist statistical approaches.

At the 70% site, the results of the frequentist ANCOVA (i.e. independent variable = group, dependent variable = 70% site change, covariate = 70% site pre-value) revealed there was a significant difference between groups ($p<0.001$). Pairwise comparisons showed that the traditional exercise group increased muscle mass above that of the control ($p<0.001$) and 1RM ($p=0.005$) groups. There was no difference in muscle growth between the 1RM training group and the control group ($p=0.085$). Specifically, the adjusted change scores were as follows: control = 0.04 (95% CI: -0.02, 0.11) cm; 1RM = 0.12 (95% CI: 0.06, 0.19) cm; and traditional = 0.26 (95% CI: 0.19, 0.33) cm. The results of the Bayesian ANCOVA with an uninformed prior at the 70% site (i.e. independent variable = group, dependent variable = 70% site change, covariate = 70% site pre-value) detailed support for the alternative hypothesis (BF = 332), in that the

traditional exercise group increased muscle size to a greater extent than both the control (BF = 224) and 1RM (BF = 5.66) groups. The results for the difference in muscle growth between the control and 1RM training groups were ambiguous (BF = 1.30). Therefore, both the Bayesian and frequentist statistical approached demonstrate that muscle growth only present in the traditional exercise group, and this increase was greater than that observed in the control and 1RM groups.

Table 5. Absolute changes in muscle size of the trained arm

	Control			1RM			Traditional		
	Pre	Post	Δ	Pre	Post	Δ	Pre	Post	Δ
50% site (cm)									
Total	2.66	2.67	0.00 (0.21)	2.72	2.78	0.06 (0.20)	2.79	3.02	0.22 (0.25)
Males	3.23	3.18	-0.04 (0.24)	3.29	3.35	0.05 (0.23)	3.35	3.67	0.32 (0.29)
Females	2.32	2.36	0.04 (0.17)	2.39	2.45	0.06 (0.19)	2.43	2.60	0.16 (0.20)
60% site (cm)									
Total	2.88	2.91	0.02 (0.20)	2.92	3.03	0.11 (0.19)	2.99	3.25	0.26 (0.25)
Males	3.44	3.44	0.00 (0.24)	3.50	3.62	0.12 (0.18)	3.51	3.88	0.36 (0.29)
Females	2.55	2.59	0.04 (0.18)	2.59	2.69	0.10 (0.19)	2.64	2.83	0.19 (0.20)
70% site (cm)									
Total	3.22	3.27	0.04 (0.23)	3.27	3.40	0.12 (0.18)	3.32	3.58	0.25 (0.29)
Males	3.79	3.87	0.07 (0.28)	3.92	4.07	0.15 (0.16)	3.88	4.25	0.37 (0.37)
Females	2.89	2.92	0.02 (0.19)	2.90	3.01	0.10 (0.19)	2.95	3.14	0.18 (0.20)

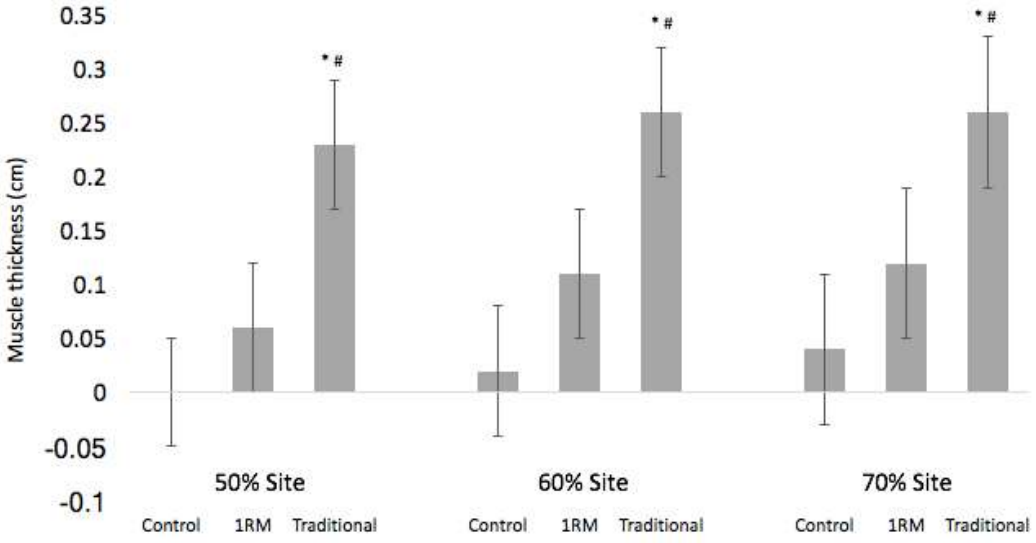
Values are expressed as means and standard deviations.

Table 6. Relative changes in muscle size of the trained arm

	Control Δ%	1RM Δ%	Traditional Δ%
Muscle thickness 50% (cm)			
Total	1.2 (7.9)	2.8 (7.8)	8.2 (9.3)
Males	-1.1 (7.6)	2.1 (7.0)	9.9 (10.1)
Females	2.6 (7.8)	3.2 (8.3)	7.0 (8.8)
Muscle thickness 60% (cm)			
Total	1.5 (7.0)	4.2 (6.8)	8.6 (8.1)
Males	0.6 (7.0)	3.7 (5.3)	10.6 (8.2)
Females	2.0 (7.0)	4.5 (7.5)	7.3 (7.9)
Muscle thickness 70% (cm)			
Total	1.7 (7.1)	4.1 (6.0)	7.9 (8.5)
Males	2.5 (7.7)	4.2 (4.2)	10.2 (10.0)
Females	1.2 (6.8)	4.0 (6.8)	6.4 (7.1)

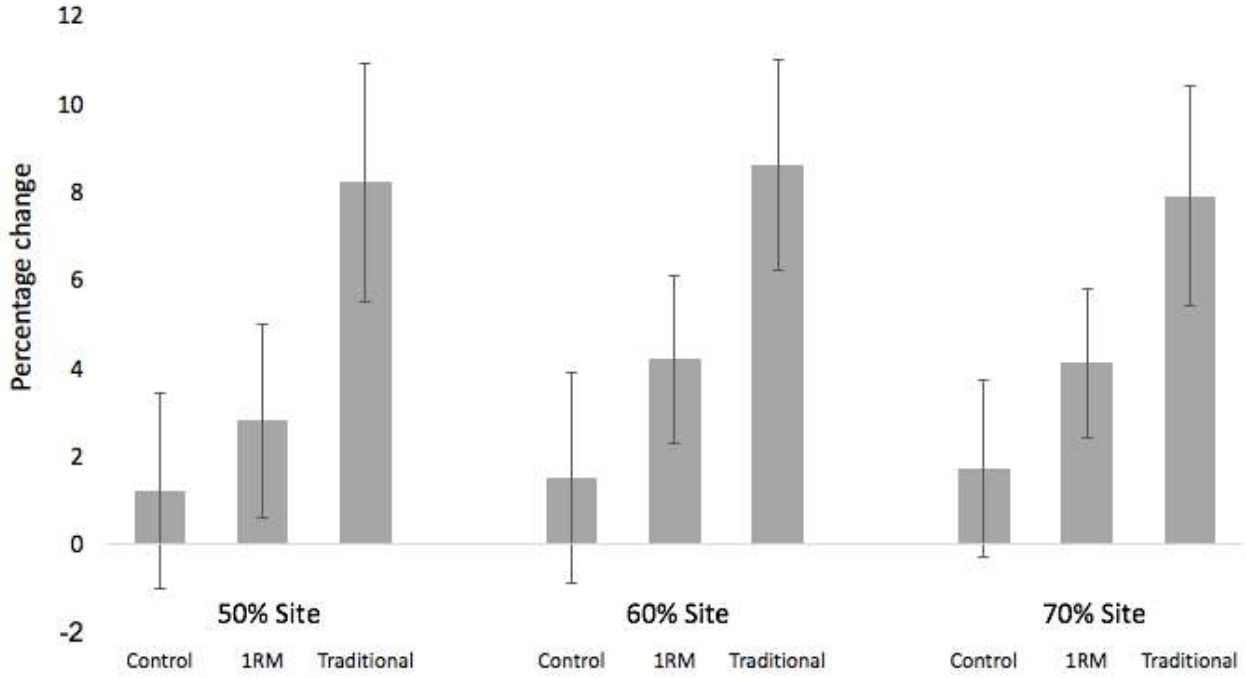
Values are expressed as mean percentage changes and standard deviations.

Figure 9. Absolute changes in muscle size of the trained arm



Values are expressed as adjusted means and 95% confidence intervals. *significantly different from the control group within each measurement site, # statistically different from the 1RM training group within each measurement site.

Figure 10. Relative changes in muscle size of the trained arm



Values are expressed as mean percentage changes and 95% confidence intervals. Statistics were not computed on the relative values, but are shown on the raw values in Figure 8.

Mean changes in muscle size of the untrained arm

The absolute changes in muscle size of the untrained arm are shown in Table 7 and Figure 10, while the relative changes are shown in Table 8 and Figure 11. The results of the ANCOVA for the 50% site of the untrained non-dominant arm (independent variable = group, dependent variable = 50% site change score, covariate = 50% site pre-value) illustrated no differences between any of the groups (p=0.827). Specifically, adjusted change scores from baseline were as follows: control = 0.01 (95% CI: -0.04, 0.06) cm, 1RM = 0.00 (95% CI: -0.04, 0.06) cm, and traditional = 0.03 (95% CI: -0.02, 0.08) cm. The results of the Bayesian ANCOVA provided strong support for the null hypothesis (BF = 0.070) indicating no differences were present between groups in the change in muscle size of the non-training arm at the 50% site. Thus, the results of the Bayesian and frequentist statistical approaches yield similar results in

suggesting that the non-training arm did not increase muscle mass at the 50% site in either of the training groups relative to the control.

The results of the ANCOVA for the 60% site of the untrained non-dominant arm (independent variable = group, dependent variable = 60% site change score, covariate = 60% site pre-value) illustrated no differences between any of the groups (p=0.963). Specifically, adjusted changes from baseline for each of the groups were as follows: control = 0.02 (95% CI: -0.03, 0.07) cm, 1RM = 0.01 (95% CI: -0.03, 0.06) cm, and traditional = 0.01 (95% CI: -0.04, 0.06) cm. The results of the Bayesian ANCOVA provided strong support for the null hypothesis (BF = 0.067) indicating no differences were present between groups in the change in muscle size of the non-training arm at the 60% site. Thus, the results of the Bayesian and frequentist statistical approaches yield similar results in suggesting that the non-training arm did not increase muscle mass at the 60% site in either of the training groups relative to the control.

The results of the ANCOVA for the 70% site of the untrained non-dominant arm (independent variable = group, dependent variable = 70% site change score, covariate = 70% site pre-value) illustrated no differences between any of the groups (p=0.758). Specifically, adjusted change scores from baseline were as follows: control = 0.05 (95% CI: 0.00, 0.10) cm, traditional = 0.04 (95% CI: 0.00, 0.09) cm, and traditional = 0.02 (95% CI: -0.03, 0.07) cm. The results of the Bayesian ANCOVA provided strong support for the null hypothesis (BF = 0.086) indicating no differences were present between groups in the change in muscle size of the non-training arm at the 70% site. Thus, the results of the Bayesian and frequentist statistical approaches yield similar results in suggesting that the non-training arm did not increase muscle mass at the 70% site in either of the training groups relative to the control. Collectively, across all three of the

sites measured on the untrained arm, there was no change in muscle mass occurring for either of the training groups.

Table 7. Absolute changes in muscle size of the untrained arm

	Control			1RM			Traditional		
	Pre	Post	Δ	Pre	Post	Δ	Pre	Post	Δ
50% site (cm)									
Total	2.61	2.63	0.01 (0.23)	2.65	2.65	0.00 (0.20)	2.71	2.73	0.02 (0.16)
Males	3.15	3.14	0.00 (0.30)	3.19	3.20	0.00 (0.23)	3.25	3.30	0.04 (0.16)
Females	2.29	2.32	0.03 (0.19)	2.33	2.34	0.00 (0.19)	2.35	2.36	0.01 (0.16)
60% site (cm)									
Total	2.78	2.80	0.02 (0.21)	2.83	2.84	0.01 (0.19)	2.89	2.90	0.00 (0.16)
Males	3.30	3.32	0.01 (0.25)	3.40	3.41	0.01 (0.21)	3.39	3.45	0.05 (0.13)
Females	2.47	2.50	0.03 (0.20)	2.50	2.51	0.01 (0.18)	2.56	2.53	-0.02 (0.17)
70% site (cm)									
Total	3.10	3.15	0.05 (0.21)	3.18	3.22	0.04 (0.17)	3.22	3.24	0.01 (0.18)
Males	3.67	3.74	0.07 (0.27)	3.82	3.83	0.01 (0.18)	3.75	3.81	0.05 (0.21)
Females	2.76	2.81	0.04 (0.17)	2.81	2.87	0.05 (0.16)	2.87	2.86	0.00 (0.16)

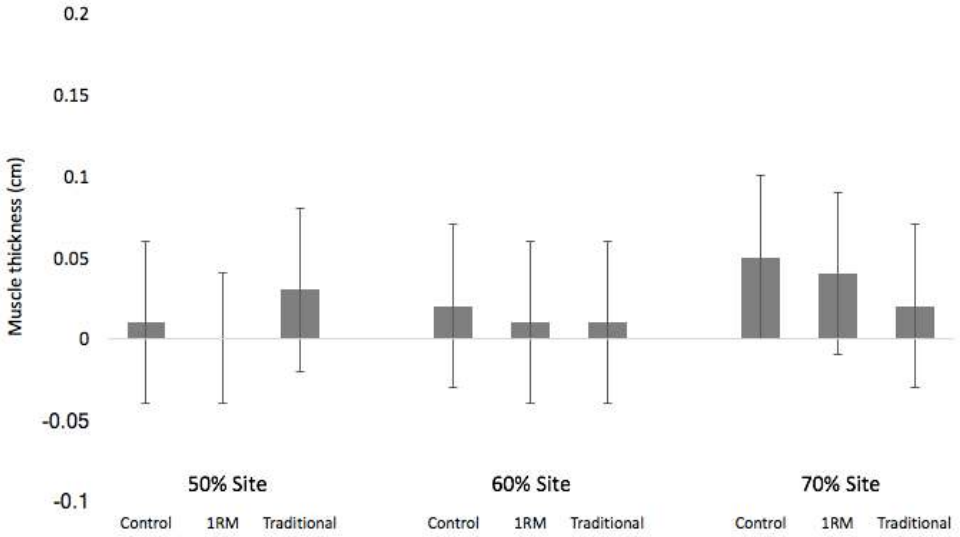
Values are expressed as means and standard deviations.

Table 8. Relative changes in muscle size of the untrained arm

	Control Δ%	1RM Δ%	Traditional Δ%
Muscle thickness 50% (cm)			
Total	1.3 (7.9)	0.5 (7.9)	1.3 (6.7)
Males	0.5 (7.6)	0.6 (7.3)	1.4 (5.1)
Females	1.7 (8.2)	0.5 (8.2)	1.2 (7.6)
Muscle thickness 60% (cm)			
Total	1.4 (7.4)	0.6 (6.7)	0.3 (6.1)
Males	0.9 (6.9)	0.7 (6.6)	1.6 (4.0)
Females	1.7 (7.8)	0.6 (6.9)	-0.4 (7.0)
Muscle thickness 70% (cm)			
Total	2.1 (6.4)	1.6 (5.6)	0.7 (5.9)
Males	2.6 (6.7)	0.6 (4.8)	1.8 (6.3)
Females	1.8 (6.4)	2.1 (6.0)	0.1 (5.6)

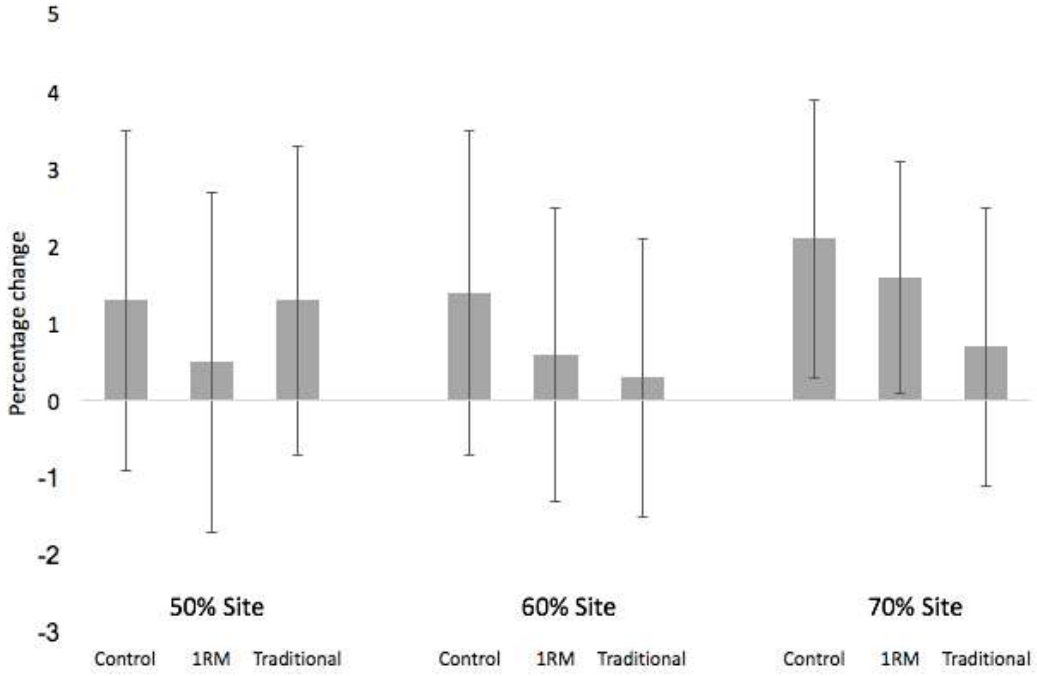
Values are expressed as mean percentage changes and standard deviations.

Figure 11. Absolute changes in muscle size of the untrained arm



Values are expressed as adjusted mean changes and 95% confidence intervals. None of the values were statistically significantly across groups.

Figure 12. Relative changes in muscle size of the untrained arm



Values are expressed as mean percentage changes and 95% confidence intervals.

Mean changes in strength of the trained arm

The absolute changes in muscle strength of the trained arm are shown in Table 9 and in Figures 12 (for isotonic strength) and 13 (for isokinetic strength). The relative changes are shown in Table 10 and Figure 14. The results of the ANCOVA on the training arm (independent variable = group, dependent variable = change in 1RM strength, covariate = pre-test 1RM strength) showed a significant difference in strength gains between groups ($p<0.001$). Follow-up tests demonstrated that both the 1RM ($p<0.001$) and traditional ($p<0.001$) training groups increased strength when compared to the control group. There were no differences in the increase in 1RM strength between the 1RM and traditional training groups in the trained arm ($p=0.842$). Specifically, the adjusted changes from baseline were as follows: control = 0.45 (95% CI: -0.04, 0.95) kg, 1RM = 2.34 (95% CI: 1.85, 2.83) kg, and traditional = 2.41 (95% CI: 1.90, 2.93) kg. The results of the Bayesian ANCOVA demonstrated strong support for the alternative

hypothesis (BF = 405,502). Follow-up tests demonstrated that both the 1RM (BF = 4.791e+6) and traditional (BF = 11,915) exercise groups increased 1RM strength above that of the control group. When comparing the change in 1RM strength between the 1RM and traditional training groups, there was support for the null hypothesis (BF = 0.218) suggesting that both groups increased strength to a similar extent. Thus, the results for both the Bayesian and frequentist statistical approaches yield similar conclusions in that both the 1RM and traditional exercise group increase strength of the trained arm to a similar extent above that of an untrained control group.

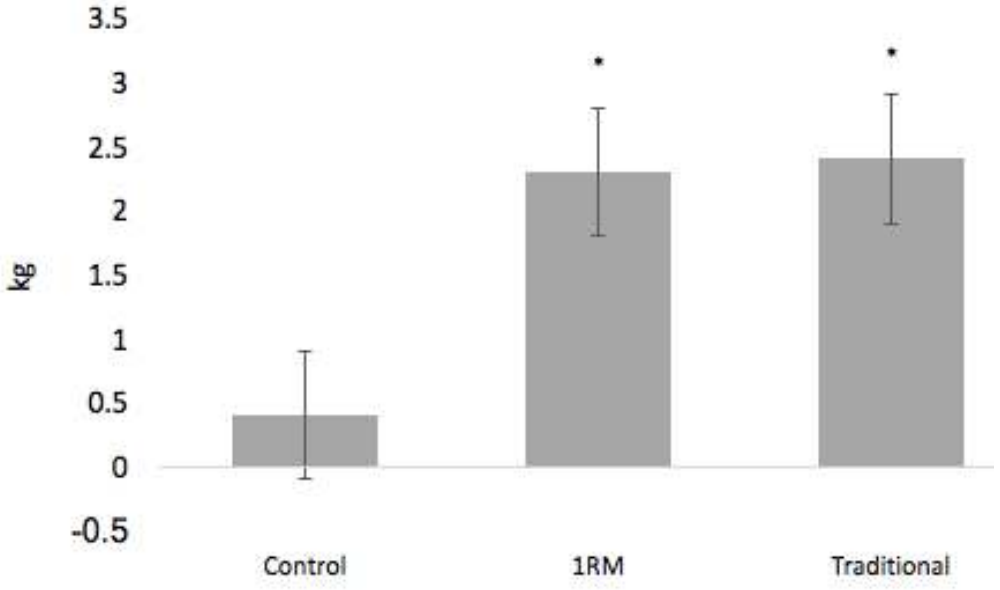
With respect to the ANCOVA on isokinetic strength of the trained arm (independent variable = group, dependent variable = change in isokinetic strength, covariate = pre-test isokinetic strength), there was no effect of group (p=0.299) indicating the training groups did not increase isokinetic strength when made relative to the control group. Specifically, the adjusted changes from baseline were as follows: control = -0.5 (95% CI: -1.5, 0.4) nm, 1RM = 0.4 (95 % CI = -0.5, 1.4) nm, and traditional = 0.4 (95% CI: -0.6, 1.4) nm. The results of the Bayesian ANCOVA demonstrated support for the null hypothesis (BF = 0.181) further suggesting that the training groups did not increase isokinetic strength.

Table 9. Absolute changes in strength of the trained arm

	Control			1RM			Traditional		
	Pre	Post	Δ	Pre	Post	Δ	Pre	Post	Δ
1RM (kg)									
Total	13.5	14.0	0.4 (1.4)	13.3	15.6	2.3 (1.5)	13.6	16.0	2.4 (2.3)
Males	18.4	19.4	1.0 (1.9)	17.9	20.7	2.7 (1.5)	18.7	20.5	1.8 (2.7)
Females	10.6	10.7	0.0 (0.8)	10.6	12.7	2.1 (1.5)	10.3	13.1	2.7 (1.9)
Isokinetic (Nm)									
Total	35.4	34.9	-0.5 (3.2)	36.9	37.3	0.3 (3.3)	36.6	37.0	0.3 (4.6)
Males	47.8	47.1	-0.7 (3.7)	51.6	51.9	0.3 (3.4)	48.2	47.3	-0.9 (6.1)
Females	28.1	27.7	-0.3 (2.9)	28.4	28.9	0.4 (3.3)	29.0	30.2	1.2 (3.1)

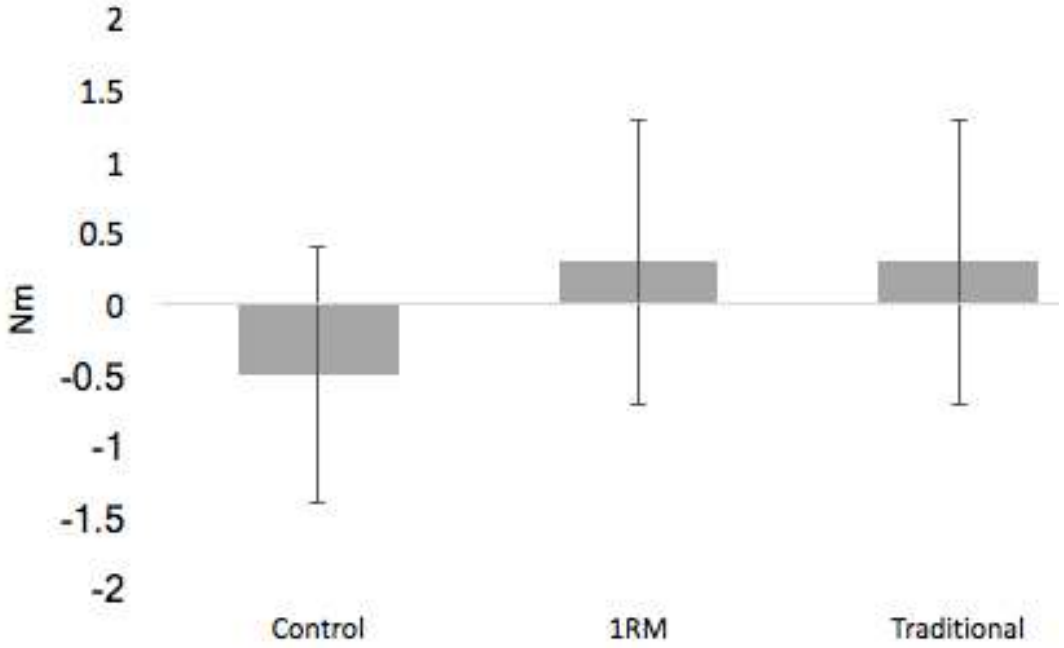
Values are expressed as means and standard deviations.

Figure 13. Absolute changes in 1RM strength of the trained arm



Values are expressed as adjusted mean changes and 95% confidence intervals. * indicates a significant difference from the control group.

Figure 14. Absolute changes in isokinetic strength of the trained arm



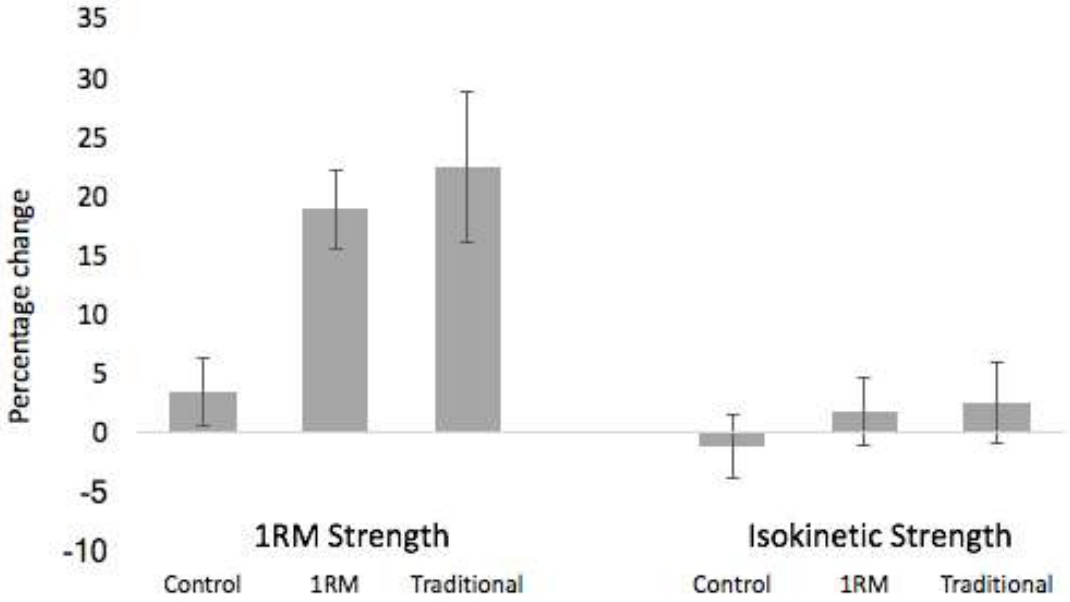
Values are expressed as adjusted mean changes and 95% confidence intervals. None of the group changes were statistically different from one another.

Table 10. Relative changes in muscle strength of the trained arm

	Control Δ%	1RM Δ%	Traditional Δ%
1RM			
Total	3.4 (10.2)	18.8 (12.4)	22.4 (21.7)
Males	6.4 (12.6)	16.2 (9.5)	12.0 (17.9)
Females	1.6 (8.2)	20.2 (13.6)	29.2 (21.6)
Isokinetic			
Total	-1.2 (9.1)	1.7 (10.4)	2.5 (11.6)
Males	-0.7 (7.6)	1.1 (7.0)	-0.4 (13.5)
Females	-1.5 (10.0)	2.0 (12.1)	4.4 (10.0)

Values are expressed as mean percentage changes and standard deviations.

Figure 15. Relative changes in muscle strength of the trained arm



Values are expressed as mean percentage changes and 95% confidence intervals. No statistical analyses were computed for percentage changes.

Mean changes in strength of the untrained arm

The absolute changes in muscle strength of the non-dominant untrained arm are shown in Table 11 and in Figures 15 (for isotonic strength) and 16 (for isokinetic strength). The relative changes are shown in Table 12 and Figure 17. The ANCOVA on 1RM strength of the untrained arm (independent variable = group, dependent variable = change in 1RM strength, covariate = pre-test 1RM strength) was statistically significant ($p<0.001$). Follow-up comparisons showed that the 1RM group increased strength of the untrained arm above that of the control ($p<0.001$) and traditional ($p=0.012$) groups. The traditional exercise group did not increase untrained arm strength above that of the control group ($p=0.154$). Specifically, the adjusted changes in 1RM strength of the untrained arm for each group were as follows: control = 0.45 (95% CI: 0.07, 0.83) kg, 1RM = 1.54 (95% CI: 1.17, 1.92) kg, traditional = 0.85 (95% CI: 0.45, 1.24) kg. The results

of the Bayesian ANCOVA also provided support that the untrained arm changed 1RM strength differently across groups (BF = 73.5). Follow-up comparisons demonstrated the 1RM training group increased untrained arm 1RM strength to a greater extent than both the control (BF = 271) and traditional (BF = 3.00) exercise groups. The results of the comparison between the traditional exercise group and control group were ambiguous (BF = 0.530) but provided weak support for the null hypothesis.

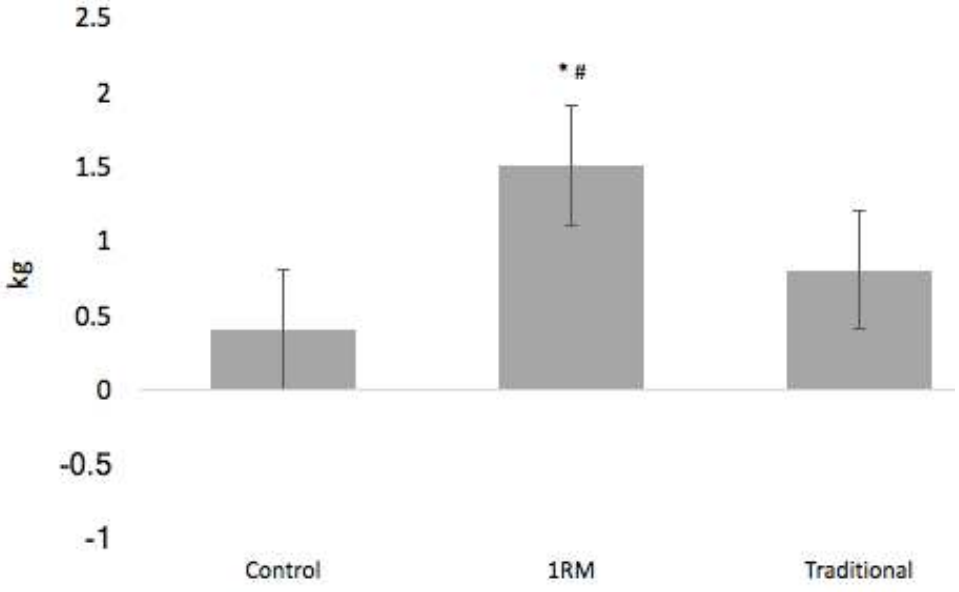
When examining the results of the ANCOVA for isokinetic strength of the untrained arm, there was no difference in strength changes across groups (p=0.241). Specifically, the adjusted changes from baseline in isokinetic strength were as follows: control = 1.1 (95% CI: 0.0, 2.3) nm, 1RM = -0.1 (95% CI: -1.3, 0.9) nm, and traditional = 0.7 (95% CI: -0.3, 1.9) nm. The results of the Bayesian ANCOVA demonstrated support for the null hypothesis (BF = 0.21) providing further support that there was so impact of training on the change in isokinetic strength of the untrained arm. Therefore, the results of both the frequentist and Bayesian approaches yield similar results in suggesting that only the 1RM training group increased 1RM strength of the untrained arm when made relative to the control group. Additionally, neither of the training groups increased isokinetic strength of the untrained arm when made relative to the control group.

Table 11. Absolute changes in strength of the untrained arm

	Control			1RM			Traditional		
	Pre	Post	Δ	Pre	Post	Δ	Pre	Post	Δ
1RM (kg)									
Total	12.9	13.4	0.4 (1.3)	12.7	14.2	1.5 (1.3)	12.9	13.7	0.8 (1.4)
Males	17.2	18.2	0.9 (1.4)	17.5	19.0	1.5 (1.1)	17.4	18.4	1.0 (1.7)
Females	10.4	10.5	0.1 (1.1)	9.9	11.5	1.5 (1.5)	9.9	10.7	0.7 (1.1)
Isokinetic (Nm)									
Total	32.4	33.6	1.2 (3.5)	34.9	34.7	-0.2 (4.0)	34.5	35.3	0.7 (4.8)
Males	45.0	46.0	0.9 (4.0)	49.8	48.8	-0.9 (5.0)	45.4	47.1	1.6 (6.5)
Females	24.9	26.2	1.3 (3.3)	26.4	26.6	0.2 (3.3)	27.4	27.6	0.1 (3.1)

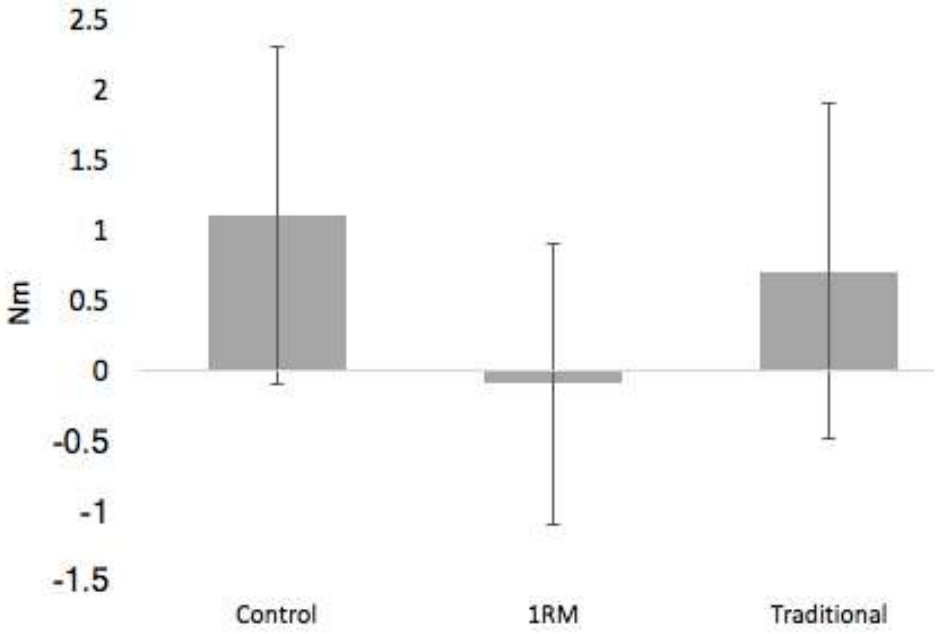
Values are expressed as means and standard deviations

Figure 16. Absolute changes in 1RM strength of the untrained arm



Values are expressed as adjusted means changes and 95% confidence intervals. *statistically different from control. # statistically different from traditional exercise.

Figure 17. Absolute changes in isokinetic strength of the untrained arm



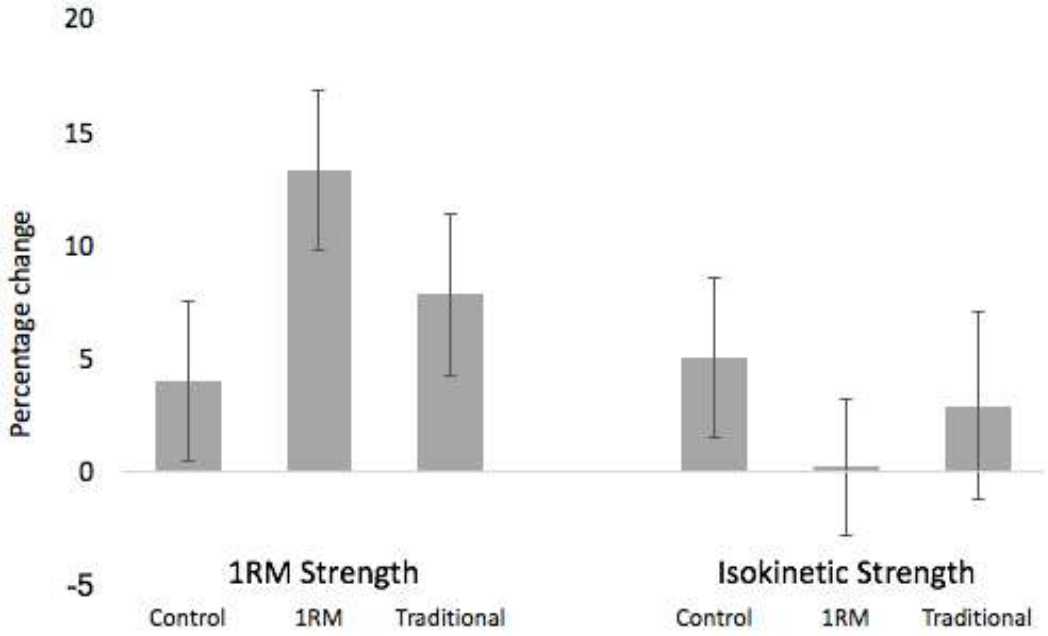
Values are expressed as means changes and 95% confidence intervals. No statistical analyses were computed on the percentage changes.

Table 12. Relative changes in muscle strength of the untrained arm

	Control Δ%	1RM Δ%	Traditional Δ%
1RM			
Total	4.0 (12.2)	13.3 (12.6)	7.8 (12.2)
Males	6.2 (9.4)	9.3 (7.0)	6.4 (10.5)
Females	2.7 (13.6)	15.6 (14.5)	8.7 (13.3)
Isokinetic			
Total	5.0 (12.4)	0.0 (10.9)	2.9 (14.0)
Males	2.9 (10.5)	-1.7 (9.5)	4.4 (15.3)
Females	6.2 (13.5)	0.9 (11.6)	1.9 (13.2)

Values are expressed as mean percentage changes and standard deviations

Figure 18. Relative changes in muscle strength of the untrained arm



Values are expressed as percentage changes and 95% confidence intervals. No statistical analyses were computed on the relative changes.

Comparison of the strength in the trained and untrained arms

In addition to examining differences in strength changes across groups, we also examined if there were differences in strength between the trained and untrained arm within each group using paired t tests. The purpose of this analysis was to test whether any strength increases in the trained arm exceed that of the untrained arm, or whether the strength increases were comparable between the trained and untrained limbs. Within the control group, there was no statistically significant difference in the change in strength between arms (mean = 0.00; 95% CI: -0.36, 0.35; p=0.981). Within the 1RM group, the trained arm increased 0.80 (95% CI: 0.43, 1.17) kg more than the untrained arm which was statistically significant (p<0.001). The trained arm of the traditional exercise group increased 1.55 (95% CI: 1.05, 2.06) more than the control arm which was also statistically significant (p<0.001). When examining the results of the Bayesian paired t

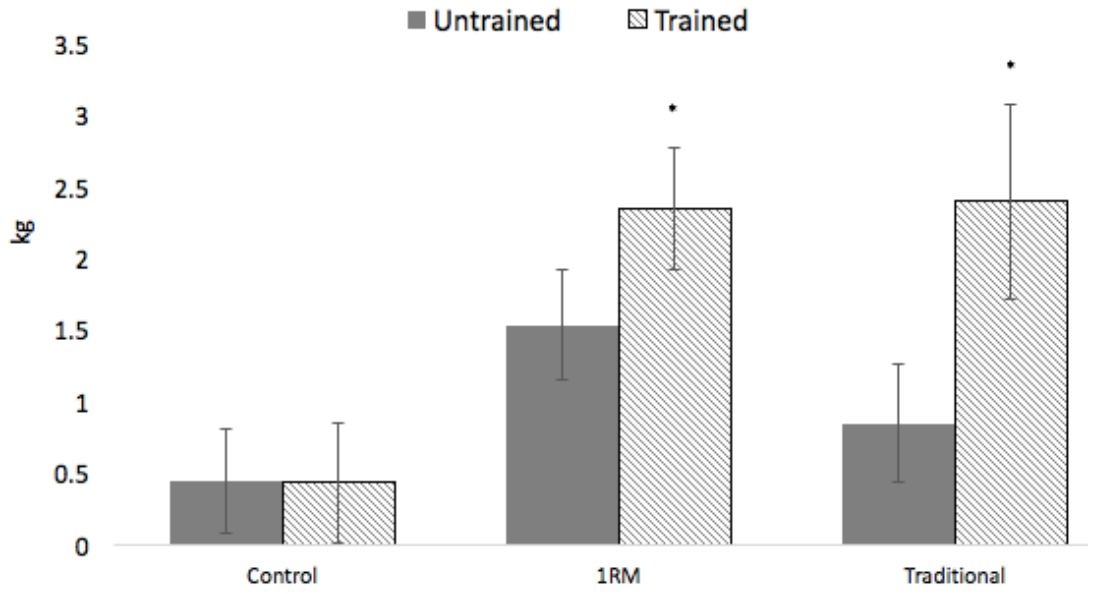
tests, the difference in the control group demonstrated support for the null hypothesis (BF = 0.152), while both the 1RM group (BF = 357) and traditional exercise (BF = 103,656) groups demonstrated support for the alternative hypothesis. Thus, the results of the frequentist and Bayesian statistical analyses yielded similar results. Even though the 1RM group increased strength of the contralateral arm more so than the traditional exercise group, both training groups saw larger strength increases in the trained limbs.

Table 13. Differences in strength gains between arms

	Δ 1RM strength (kg)	Δ Isokinetic strength (Nm)
Control		
Non-dominant arm	0.45 (1.31)	1.21 (3.56)
Dominant arm	0.44 (1.46)	-0.50 (3.22)
Difference	0.00 (1.29)	-1.72 (3.50)
1RM		
Non-dominant arm	1.54 (1.38)	-0.20 (4.01)
Dominant arm	2.35 (1.54)	0.38 (3.35)
Difference	0.80 (1.32)	0.59 (4.97)
Traditional		
Non-dominant arm	0.85 (1.41)	0.78 (4.80)
Dominant arm	2.40 (2.32)	0.38 (4.67)
Difference	1.55 (1.74)	-0.38 (5.92)

Values for the changes are expressed as means and standard deviations. The difference scores are calculated using the formula: dominant arm change – non-dominant arm change.

Figure 19. 1RM strength changes in each arm across groups



Values are expressed as mean changes and 95% confidence intervals. * indicates significantly different from the untrained limb within each group.

Variability of muscle size and strength of the trained arm

With respect to the variability present at the 50% site, the Levene's tests were not statistically significant for either the 1RM (p=0.623, BF=0.232) or traditional (p=0.558; BF=0.247) training groups. For the 60% site, the Levene's tests were also not statistically significant for either the 1RM (p=0.537; BF=0.247) or traditional (p=0.063; BF=1.006) training groups. The same held true for the 70% site, in which the Levene's test for both the 1RM (p=0.177; BF=0.473) and traditional (p=0.155; BF=0.526) training groups were not statistically significant. Thus, both the frequentist and Bayesian statistical analyses provided support that there were no individuals responders to resistance exercise that could be detected above that of random error with respect to the outcome of muscle size of the dominant arm.

With respect to the variability present in the trained arm for 1RM strength, the Levene's test was not statistically significant for the 1RM training group (p=0.114; BF=0.643), but it was for the traditional (p=0.008; BF=5.381) training group. The true variability in 1RM strength of the traditional exercise group equated to 1.8 kg after the removal of random error (Table 15). There was no difference in the variability in response to isokinetic strength for either the 1RM (p=0.502, BF= 0.255) or traditional (p=0.067, BF= 0.959) training groups.

When re-running all analyses using the variability of the relative changes (i.e. the standard deviation of the percentage changes), the only value that changed was that of 1RM strength in the 1RM group which became statistically significant (p=0.015; BF=3.057). When using the percentage change values, the standard deviations of each group were as follows: control = 10.28, 1RM = 12.40, traditional = 21.7, with each group being statistically different from one another. That is, while the 1RM group was more variable than the control group, the traditional exercise group was more variable than the 1RM group (p=0.002, BF=19.84). Expressed as a percentage, the true variability after removal of random error of the 1RM and traditional exercise groups were as follows: 1RM = 6.9% ($\sqrt{(12.40^2 - 10.28^2)}$) and traditional = 19.1% ($\sqrt{(21.70^2 - 10.28^2)}$).

Table 14. Variability in muscle size and strength of the trained arm

	Control	1RM	Traditional
Muscle thickness 50%			
Total	0.21	0.20	0.25
Males	0.24	0.23	0.29
Females	0.17	0.19	0.20
Muscle thickness 60%			
Total	0.20	0.19	0.25
Males	0.24	0.18	0.29
Females	0.18	0.19	0.20
Muscle thickness 70%			
Total	0.23	0.18	0.29
Males	0.28	0.16	0.37
Females	0.19	0.19	0.20
Isotonic (1RM)			
Total	1.46	1.54	2.32
Males	1.98	1.50	2.78
Females	0.89	1.53	1.93
Isokinetic			
Total	3.22	3.35	4.67
Males	3.71	3.43	6.18
Females	2.96	3.36	3.19

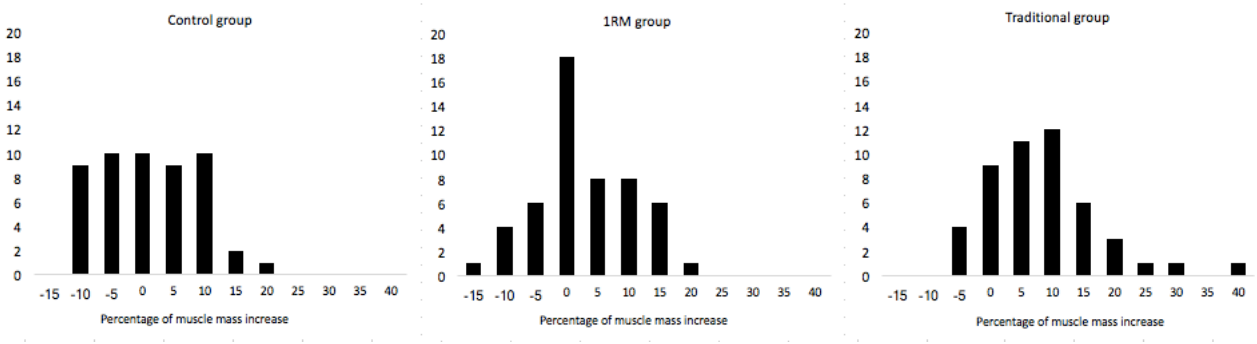
Values are expressed as change score standard deviations.

Table 15. Variability of the trained arm after the removal of random error

	Computation of true variability	True Variability
Muscle thickness 50%		
1RM	$\sqrt{(0.20^2 - 0.21^2)}$	0
Traditional	$\sqrt{(0.25^2 - 0.21^2)}$	0.13
Muscle thickness 60%		
1RM	$\sqrt{(0.19^2 - 0.20^2)}$	0
Traditional	$\sqrt{(0.25^2 - 0.20^2)}$	0.15
Muscle thickness 70%		
1RM	$\sqrt{(0.18^2 - 0.23^2)}$	0
Traditional	$\sqrt{(0.29^2 - 0.23^2)}$	0.17
Isotonic (1RM)		
1RM	$\sqrt{(1.54^2 - 1.46^2)}$	0.48
Traditional	$\sqrt{(2.32^2 - 1.46^2)}$	1.80*
Isokinetic		
1RM	$\sqrt{(3.35^2 - 3.22^2)}$	0.92
Traditional	$\sqrt{(4.67^2 - 3.22^2)}$	3.38

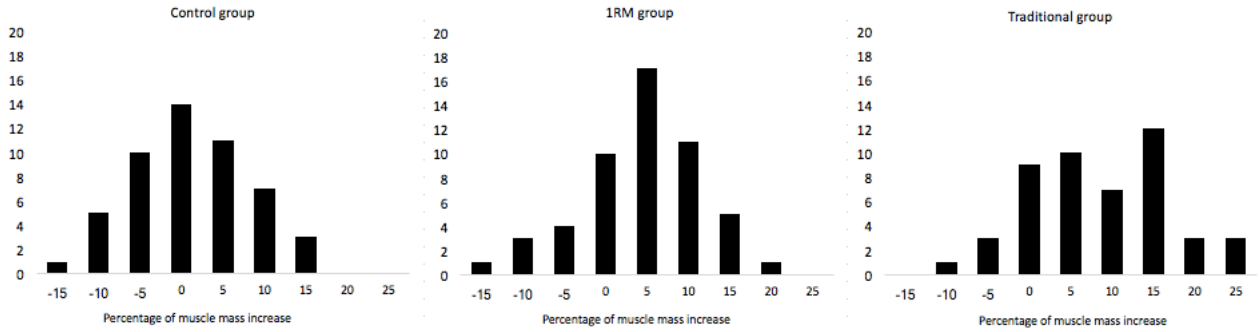
To compute the true variability that could be attributed to the exercise intervention, the following formula was used: $\sqrt{SDI^2 - SDC^2}$]. In this formula, SDI is the standard deviation of the change score in the intervention group and SDC is the standard deviation of the change score in the control group. Values of negative variability were simply reported as "0" variability being present after the removal of random error. * indicates a statistically significant variability that differed from that of the control group.

Figure 20. Changes in muscle thickness of the trained arm at the 50% site



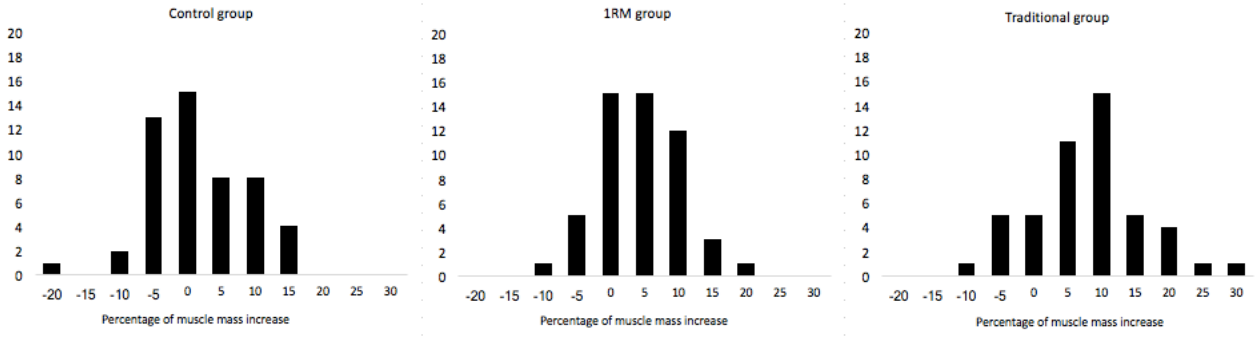
The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 0.21, 1RM = 0.20, and traditional = 0.25.

Figure 21. Changes in muscle thickness of the trained arm at the 60% site



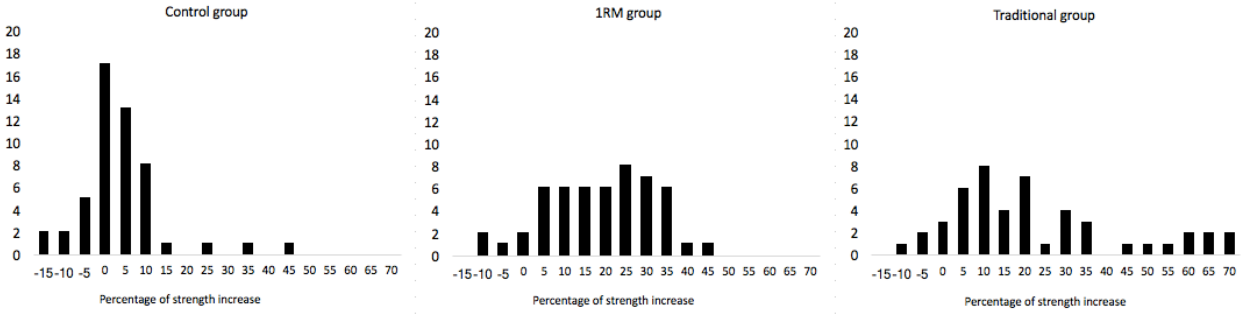
The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 0.20, 1RM = 0.19, and traditional = 0.25.

Figure 22. Changes in muscle thickness of the trained arm at the 70% site



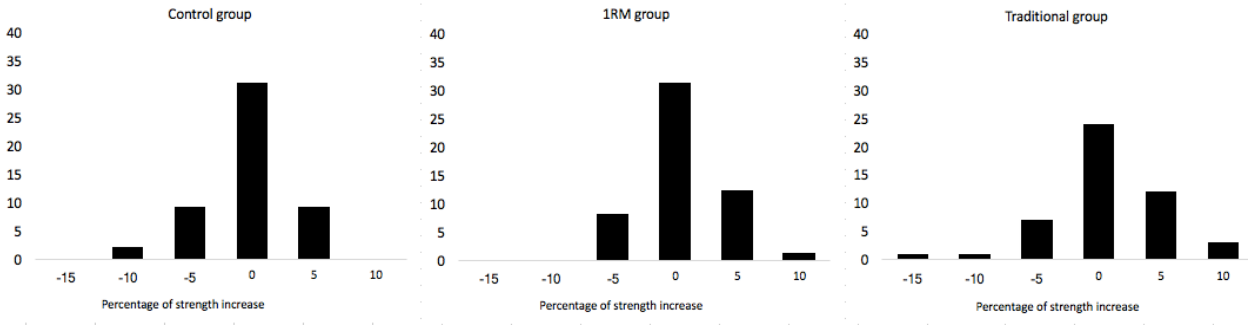
The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 0.23, 1RM = 0.18, and traditional = 0.29.

Figure 23. Changes in isotonic (1RM) strength of the trained arm



The results of the Levene's tests indicated that only the traditional exercise group resulted in true individual responses above that of measurement error. The standard deviations for each of the groups were as follows: control = 1.46, 1RM = 1.54, and traditional = 2.32. This resulted in a true SD of 1.8 kg that could be attributed to the actual intervention after the removal of random error.

Figure 24. Changes in isokinetic strength of the trained arm



The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 3.22, 1RM = 3.35, and traditional = 4.67.

Variability of muscle size and strength of the untrained arm

When examining the variability of muscle size and strength of the non-dominant arm, none of the variables were statistically significant and all provided support for the null hypothesis using Bayesian statistics. The results of the Levene's tests for the 1RM group were as follows: 50% site (p=0.604, BF=0.235), 60% site (p=0.846, BF=0.212), 70% site (p=0.489, BF=0.258), 1RM strength (p=0.436, BF=0.273), and isokinetic strength (p=0.738, BF=0.219). The results of the Levene's tests for the traditional training group were as follows: 50% site (p=0.403, BF=0.29), 60% site (p=0.246, BF=0.387), 70% site (p=0.591, BF=0.241), 1RM strength (p=0.782, BF=0.219), and isokinetic strength (p=0.110, BF=0.667). Thus, there were no differences in how individuals responded to adaptations in muscle size of the non-dominant arm.

Table 16. Variability in muscle size and strength of the untrained arm

	Control	1RM	Traditional
Muscle thickness 50%			
Total	0.23	0.20	0.16
Males	0.30	0.23	0.16
Females	0.19	0.19	0.16
Muscle thickness 60%			
Total	0.21	0.19	0.16
Males	0.25	0.21	0.13
Females	0.20	0.18	0.17
Muscle thickness 70%			
Total	0.21	0.17	0.18
Males	0.27	0.18	0.21
Females	0.17	0.16	0.16
Isotonic (1RM)			
Total	1.31	1.38	1.41
Males	1.48	1.19	1.73
Females	1.11	1.50	1.18
Isokinetic			
Total	3.56	4.01	4.80
Males	4.03	5.01	6.58
Females	3.31	3.31	3.15

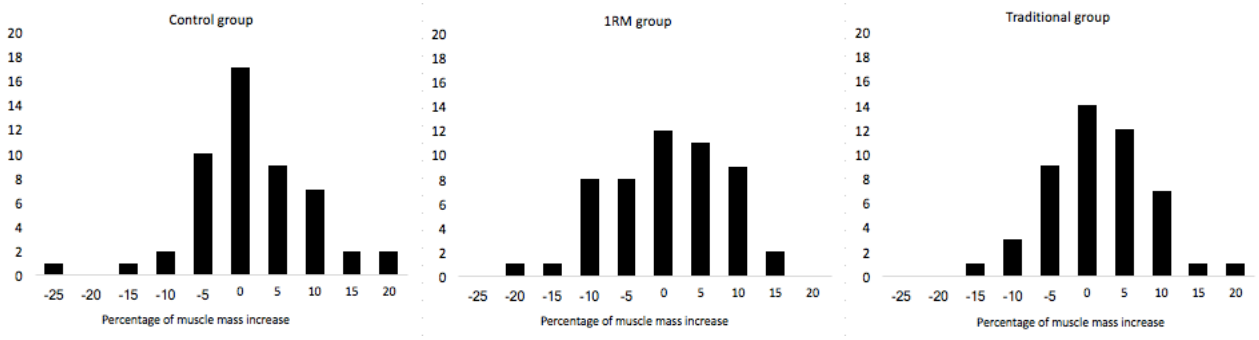
Values are expressed as change score standard deviations.

Table 17. Variability of the untrained arm after the removal of random error

	Computation of true variability	True variability
Muscle thickness 50%		
1RM	$\sqrt{(0.20^2 - 0.23^2)}$	0
Traditional	$\sqrt{(0.16^2 - 0.23^2)}$	0
Muscle thickness 60%		
1RM	$\sqrt{(0.19^2 - 0.21^2)}$	0
Traditional	$\sqrt{(0.16^2 - 0.21^2)}$	0
Muscle thickness 70%		
1RM	$\sqrt{(0.17^2 - 0.21^2)}$	0
Traditional	$\sqrt{(0.18^2 - 0.21^2)}$	0
Isotonic (1RM)		
1RM	$\sqrt{(1.38^2 - 1.31^2)}$	0.43
Traditional	$\sqrt{(1.41^2 - 1.31^2)}$	0.52
Isokinetic		
1RM	$\sqrt{(4.01^2 - 3.56^2)}$	1.84
Traditional	$\sqrt{(4.80^2 - 3.56^2)}$	3.21

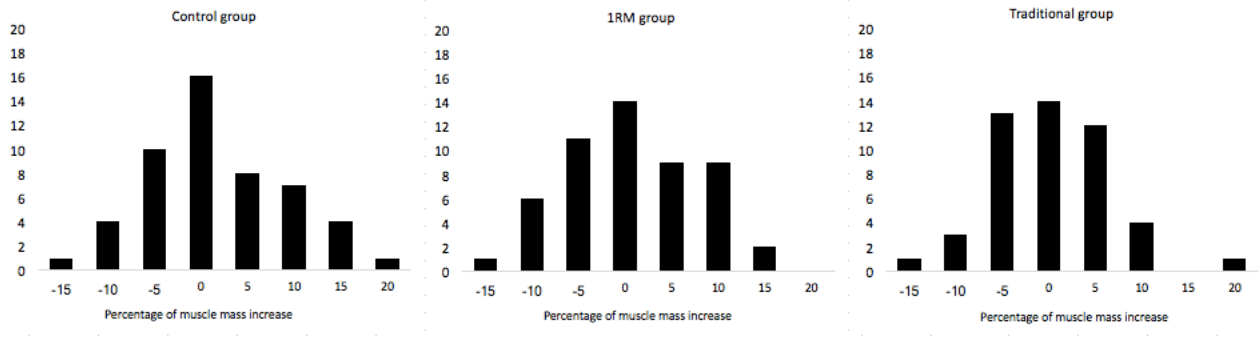
To compute the true variability that could be attributed to the exercise intervention, the following formula was used: $\sqrt{SDI^2 - SDC^2}$]. In this formula, SDI is the standard deviation of the change score in the intervention group and SDC is the standard deviation of the change score in the control group. Values of negative variability were simply reported as "0" variability being present after the removal of random error. None of the true variability values listed above were statistically different from the random error of the control group.

Figure 25. Changes in muscle thickness of the untrained arm at the 50% site



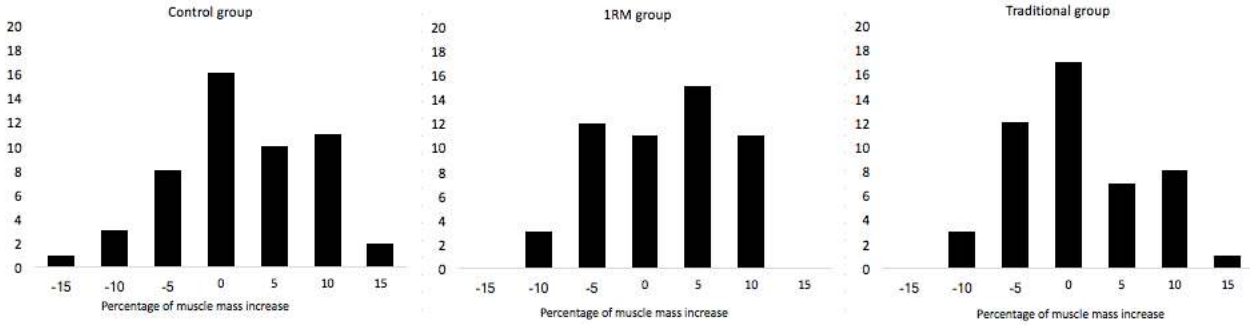
The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 0.23, 1RM = 0.20, and traditional = 0.16.

Figure 26. Changes in muscle thickness of the untrained arm at the 60% site



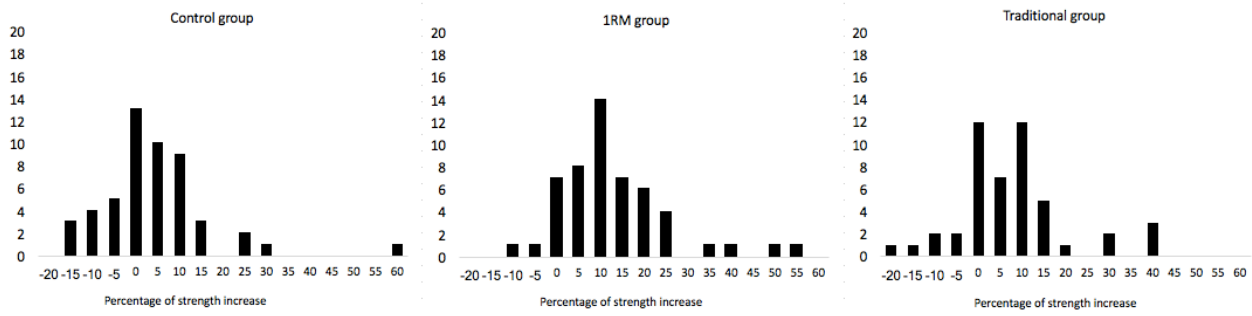
The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 0.21, 1RM = 0.19, and traditional = 0.16.

Figure 27. Changes in muscle thickness of the untrained arm at the 70% site



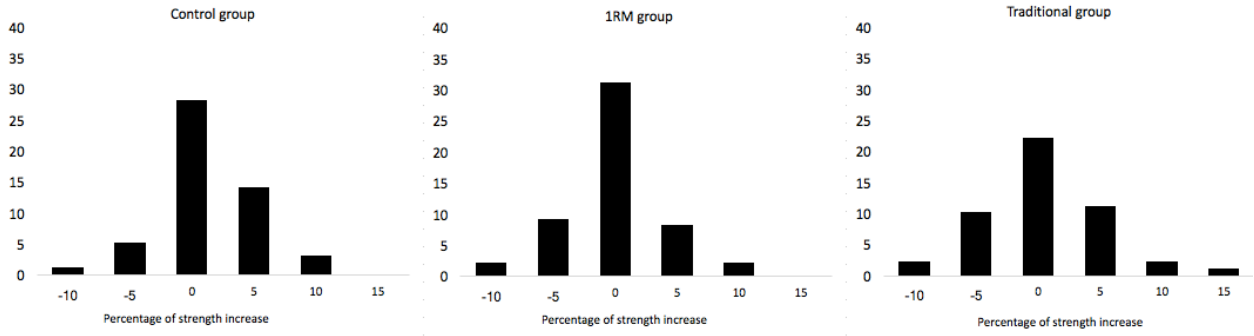
The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 0.21, 1RM = 0.17, and traditional = 0.18.

Figure 28. Changes in isotonic (1RM) strength of the untrained arm



The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 1.31, 1RM = 1.38, and traditional = 1.41.

Figure 29. Changes in isokinetic strength of the untrained arm



The results of the Levene's tests were not statistically significant indicating that no individual responders were present in either the 1RM group or the traditional exercise groups. The standard deviations for each of the groups were as follows: control = 3.56, 1RM = 4.01, and traditional = 4.80.

Analysis of differential responders

Since there were individual responders in 1RM strength of the trained arm that could be identified in the traditional exercise group, we then examined how many of these individuals could confidently be defined as being differential responders from the mean. This was done by examining which individuals responded in a fashion that exceeded the magnitude of random error obtained from the control group (i.e. responding greater than 1.96 standard deviations from the mean response of the traditional exercise group). Of the 48 individuals in the traditional exercise group, 38 (79.2%) could not be identified as responding differently from the mean, and 10 individuals (20.8%) could confidently be identified as differential responders. None of the individuals could be classified as extreme responders, meaning that no one responded greater than 5.88 standard deviations (1.96 * 3) above the mean (1.96 standard deviations above the mean would be the lower bound of the high responder group, another 1.96 standard deviations would be the mean of the high responder group, and another 1.96 standard deviations would be the upper bound of the high responder group). Had we used other statistical approaches we

would have concluded that we had a different number of individual responders as shown in Table 18 for the traditional exercise group and Table 19 for the 1RM group.

Table 18. Individual responders in the traditional exercise group depending on approach

	Current approach	K-means cluster	1 SD above or below mean
1RM Strength			
Differential Responders	10 (20.8%)	20 (41.7%)	12 (25.0%)
High Responders	6 (12.5%)	8 (16.7%)	8 (16.7%)
Low Responders	4 (8.3%)	12 (25.0%)	4 (8.3%)
Isokinetic Strength			
Differential Responders	0 (0%)	23 (47.9%)	11 (22.9%)
High Responders	0 (0%)	17 (35.4%)	5 (10.4%)
Low Responders	0 (0%)	6 (12.5%)	6 (12.5%)
50% Muscle Thickness Site			
Differential Responders	0 (0%)	27 (56.3%)	13 (27.1%)
High Responders	0 (0%)	1 (2.1%)	7 (14.6%)
Low Responders	0 (0%)	26 (54.1%)	6 (12.5%)
60% Muscle Thickness Site			
Differential Responders	0 (0%)	26 (54.2%)	15 (31.3%)
High Responders	0 (0%)	20 (41.7%)	9 (18.8%)
Low Responders	0 (0%)	6 (12.5%)	6 (12.5%)
70% Muscle Thickness Site			
Differential Responders	0 (0%)	23 (47.9%)	14 (29.2%)
High Responders	0 (0%)	12 (25.0%)	8 (16.7%)
Low Responders	0 (0%)	11 (22.9%)	6 (12.5%)

Values are expressed as the number of individuals in the traditional exercise group that would have met the specific responder classification based on the method employed. In parentheses is the percentage of the traditional exercise group (n=48) that met each classification. SD= standard deviation. The k-mean cluster analysis broke individuals up into three clusters (low responders, normal responders, and high responders).

Table 19. Individual responders in the 1RM exercise group depending on approach

	Current approach	K-means cluster	1 SD above or below mean
1RM Strength			
Differential Responders	0 (0%)	25 (48.1%)	17 (32.7%)
High Responders	0 (0%)	14 (26.9%)	9 (17.3%)
Low Responders	0 (0%)	11 (21.2%)	8 (15.4%)
Isokinetic Strength			
Differential Responders	0 (0%)	26 (50%)	18 (34.6%)
High Responders	0 (0%)	12 (23.1%)	11 (21.1%)
Low Responders	0 (0%)	14 (26.9%)	7 (13.5%)
50% Muscle Thickness Site			
Differential Responders	0 (0%)	24 (46.2%)	18 (34.6%)
High Responders	0 (0%)	15 (28.9%)	10 (19.2%)
Low Responders	0 (0%)	9 (17.3%)	8 (15.4%)
60% Muscle Thickness Site			
Differential Responders	0 (0%)	31 (59.6%)	15 (28.9%)
High Responders	0 (0%)	21 (40.4%)	6 (11.6%)
Low Responders	0 (0%)	10 (19.2%)	9 (17.3%)
70% Muscle Thickness Site			
Differential Responders	0 (0%)	30 (57.7%)	17 (32.7%)
High Responders	0 (0%)	22 (42.3%)	9 (17.3%)
Low Responders	0 (0%)	8 (15.4%)	8 (15.4%)

Values are expressed as the number of individuals in the traditional exercise group that would have met the specific responder classification based on the method employed. In parentheses is the percentage of the traditional exercise group (n=52) that met each classification. SD= standard deviation. The k-mean cluster analysis broke individuals up into three clusters (low responders, normal responders, and high responders).

CHAPTER 5: DISCUSSION

Main findings

The main findings from the present study can be broken down into two categories, one of which involved mean differences between the three groups and the other involved the analysis of individual responders. With respect to the mean differences between groups, the main findings were as follows: (1) measurable muscle hypertrophy was only present in the traditional exercise group; (2) 1RM strength increased to a similar extent in both the traditional training group and the 1RM training group; (3) untrained arm strength only increased in the 1RM training group; and (4) there were no increases in isokinetic strength in either of the training groups for either the trained or untrained arm. With respect to the assessment of individual responders to resistance exercise, our main findings were as follows: (1) there were no individual responders to changes in muscle size in either the 1RM or traditional training groups; (2) there were no individual responders with respect to changes in isokinetic strength in either the 1RM or traditional training group; (3); only the traditional exercise group demonstrated individual responders in 1RM strength of the trained arm; and (4) there were no individual responders with respect to changes in 1RM strength of the untrained arm.

Mean changes in muscle size

At each of the three sites measured, muscle growth was only present in the traditional exercise group when made relative to the control group. The increase in muscle size of the traditional exercise group was also greater than that of the 1RM group. Our results support the findings of previous work from our laboratory suggesting that repeatedly performing the 1RM

test does not result in measurable muscle hypertrophy (15). This can likely be explained by an insufficient duration in which the activated motor units are firing as opposed to an insufficient recruitment of motor units, given that the maximal contractions during the 1RM test would be likely to activate at or near 100% of the muscle fibers (30). Therefore, in addition to activating a larger number of motor units, these motor units must be activated for a sufficient duration to induce the mechanotransduction cascade. The duration component required to increase muscle mass may possibly be related to increases in intracellular calcium (110) or phosphatidic acid (111), both of which are upstream targets of the mechanistic target of rapamycin complex 1 (mTORC1) thought to be important for muscle growth (112, 113).

The changes in muscle size that were present appeared to be similar for both males and females, with the only difference being that females in the traditional exercise group appeared to gain slightly less absolute muscle mass when compared to males in the traditional exercise group. When expressed as a relative percentage of muscle mass gained in response to training, the discrepancy between males and females was much smaller. These findings have been observed previously (6, 114) in which males and females gain a similar relative amount of muscle mass, but males tend to gain more muscle mass when expressed in absolute terms. There did not appear to be any sex differences in muscle growth in response to 1RM training and this was likely due to the stimulus being insufficient to induce muscle growth independent of sex. As for the untrained arm, there were no changes in muscle mass at any of the sites for either of the training groups. This supports the idea that muscle growth is a locally driven process and only the muscles being contracted will increase in size.

Mean changes in strength of the trained arm

Our finding that 1RM muscle strength increased to a similar extent in both the repeated 1RM training group and the traditional exercise group supports previous research from our laboratory (15). These results provide strong support for the principle of specificity and detail that large strength gains can be observed from simply performing the 1RM test. This also would seemingly refute the hypothesis that more exercise volume results in greater strength gains (115–117), given that the traditional exercise group completed substantially more volume than the 1RM group but this added volume did not augment strength. It is known that lifting heavier loads result in greater increases in 1RM strength (25, 26), and this presents a possible limitation to the present study as the loads were not matched across groups. Even so, it is unknown if the difference in loads in the present study (i.e. 70% vs. 100% 1RM) would result in differential strength increases. It is also possible that exercise volume is important for increasing muscle strength, but the volume of exercise performed by the 1RM test alone was sufficient to exceed the level of volume necessary to increase muscle strength. That is, we do not know if we would have seen as large of strength increases had we just performed one maximal repetition each visit. We could not test this, however, because completing a 1RM test, by definition, requires at least two attempts. One way to overcome this potential limitation would be to use dynamometry where a maximal strength test can be performed with only one contraction lasting just a few seconds. This may provide an avenue for future research.

The specific mechanisms by which muscle strength is increased with resistance exercise are largely unknown. Given that muscle size only increased in the traditional exercise group, yet comparable increases in 1RM strength were observed between the 1RM and traditional exercise groups, this would suggest that muscle hypertrophy is playing little if any role with respect to

increasing 1RM strength. Thus, the increases in strength are likely due to neural mechanisms or possibly adaptations to muscle that are independent of muscle hypertrophy. Some of these proposed neural mechanisms for increasing strength include increased firing rates of motor neurons, increased excitability of motor neurons, an increased level of central drive from the motor cortex, and a decrease in excitability of inhibitory neurons (36–38). Some of the local muscular changes that may increase strength include shifts in fiber types (118), increases in calcium sensitivity (119), and increases in strongly bound cross-bridges (120). Of note, we did not measure any potential mechanisms responsible for the increases in strength and are simply providing some possible explanations.

With respect to the lack of change in isokinetic strength, this can likely be explained by the fact that individuals trained with isotonic contractions, and strength increases tend to be greater when the test more closely resembles the training modality (121). Nonetheless, we did notice increases in isokinetic strength at the same speed (60º per second) when examining responses to the same training regimens performed in the knee extensors (15). The reasoning for the differential findings is unknown, but could be related to differences between the upper and lower body musculature, or the lack of a time matched control group in the previous study (15). Additionally, it seems likely that isotonic strength is more adaptable because there is a greater skill component involved in performing isotonic contractions relative to isokinetic contractions that move through a set range of motion. Support for this exists in that we previously observed large increases in 1RM strength with repeated 1RM testing, but did not observe any changes in isometric strength after repeated isometric strength testing (14).

Cross-over effect of strength to the untrained arm

When examining the differences in strength adaptations of the untrained arm, only the 1RM group increased 1RM strength above that of the control group. This increase was also greater than that of the traditional exercise group. Mechanistically, the reasoning as to why the 1RM group responded to a greater extent than the traditional exercise group is difficult to explain considering little is known on the specific neurological adaptations that occur to the training arm let alone the contralateral untrained arm (40). It is also particularly difficult to explain considering there were no differences in strength of the trained arm between the 1RM and traditional exercise groups. A recent meta-analysis concluded that the contralateral effect of strength may be more pronounced when a greater exercise volume is performed, while the exercise load is of less importance (122). At a first glance, this would appear to contradict our findings since the 1RM group performed a lower total volume and exercised with a heavier load. However, the 1RM group did perform a greater volume of exercise that directly resembled that of the strength test, which may be an important factor to consider. It should also be mentioned that there is substantial heterogeneity present amongst the 10 studies included in this meta-analysis (123), which may limit the interpretation of these findings. Nonetheless, it seems likely that whatever neural mechanisms are contributing to strength increases of the trained arm (mentioned in the previous section) are also likely present in the untrained arm within the 1RM group.

As for the lack of increase in strength of the non-dominant arm in the traditional exercise group, this may be largely related to the fact that it was compared to a non-exercised control group. A previous study that included a large sample size and a control group, did not find any changes of exercise above that of a time matched control group (124). Therefore, it seems that

there are two possible conclusions: (1) just performing the 1RM test one time produces increases in strength that are maintained for a sufficient duration (i.e. 2-3 months), and/or (2) there is substantial error around the change in 1RM strength that occurs over time making it difficult to detect small changes above and beyond this error. A meta-analysis including only those studies which compared the cross-over effect relative to a control group observed about a 7% greater increase in contralateral strength in response to training (40, 125). Notably, almost all individual studies included in these meta-analyses did not observe statistical significance as was the case in the present study. Therefore, it seems reasonable to conclude that if there is a cross-over effect to traditional training (although not observed in the present study) it is likely very small.

When examining adaptations of the untrained arm, there were no changes in isokinetic strength across any of the groups. This is not surprising given there were no changes in isokinetic strength present in the arm that was directly trained. This again can likely can be explained by the principle of specificity that is also present in the cross-over effect. It is unknown if there would have been a cross-over effect in isokinetic strength had individuals been training with isokinetic contractions, and this may provide an avenue of future research.

Individual responders to resistance exercise

The only variable that we could identify individual responders to resistance exercise above that of random error was 1RM strength in the trained arm. There was a true variability of 1.8 kg which was quite large when compared to the mean change of 2.4 kg. When expressed in relative terms, the true variability of 19.1% also appeared to be quite large relative to the mean change of 22.4%. When using our method of identifying those who exceeded that of time matched random error, we could identify 10 of the individuals (~20%) in the traditional exercise group as responding differently than the group mean. A total of 6 of these individuals responded

more favorably, while 4 individuals responded less favorably. Had we used more traditional approaches such as classifying individuals as those more or less than one standard deviation from the mean (16, 17), those in the top or bottom 15% (18) or 20% (19) of responses, or cluster analysis (19, 46, 47, 78, 79, 81, 82, 126), we would have made a drastically different conclusion on the number of individual responders.

Within the 1RM group there were no individual responders, yet we would have grouped numerous individuals into different responder categories simply stratified based on measurement error, had we used more traditional approaches. This would have then led us to inappropriately look at what variables were associated with high or low responses to exercise, despite there being no differential responders to exercise in the first place. The same also held true for the traditional training group aside from changes in 1RM strength. For the 1RM strength variable, only those individuals who are classified as differential responders above and beyond that of time matched random error should be analyzed. Thus, numerous analyses that are computed are likely correlating a large degree of random error (as opposed to true individual differences) with different mRNA transcripts. When you combine this fact with the poor reliability of mRNA transcripts (pearson correlation average across 6 gene expressions = 0.43) (54), it seems reasonable that even if there was a gene responsible for causing individuals to respond differently to exercise it would be very hard to detect given there is a large degree of random error (1) in the training responses and (2) in the measurement of mRNA transcripts.

The reason as to why there were individual responders to 1RM strength in response to traditional exercise but not 1RM training is unknown. Notably, the variability in the traditional exercise group was also greater than that of the 1RM group suggesting this is not simply artifact (i.e. the training groups were directly compared, and we are not just referring to a difference in

statistical differences). One possible explanation is that the 8-12 repetition maximum that was performed by all individuals in the traditional exercise group may have resulted in a different relative percentage of 1RM performed across individuals. Thus, those individuals with better strength endurance may have been able to perform 8-12 repetitions with a higher relative percentage of 1RM as compared to those with poorer strength endurance. Even so, it seems unlikely that the magnitude of difference in relative 1RM load being lifted across individuals in the traditional exercise group would be large enough to result in differential strength increases. However, it could be argued that the repetition range itself establishes an exercise intensity in which all individuals exercised at the same relative intensity. An additional explanation as to why there was more variability in the traditional exercise group may be that repeatedly performing the 1RM test caused all individuals in the 1RM group to perform roughly the same amount of near maximal/maximal repetitions (4-5) each visit. On the other hand, individuals in the traditional exercise group may have performed a different number of near maximal/maximal repetitions depending on their determination and perseverance to continue exercising when approaching failure. One final explanation provided is that some physiological mechanisms responsible for increasing strength [e.g. fiber type shifts (118), changes in calcium kinetics (119), etc.] require a greater exercise volume, and the change in these variables may have differed across individuals in the traditional exercise group. This hypothesis, however, would seemingly imply that different mechanisms are responsible for increasing strength following repeated 1RM testing as compared to traditional exercise (since there were no individual responders in the 1RM group), and this requires additional research.

There were no differences in how individuals responded to adaptations in muscle size and strength of the untrained arm. This lack of individual differences in muscle size of the untrained

arm is not surprising given the only value that changed across any of the groups was 1RM strength in the 1RM training group. Nonetheless, the lack of change for all variables in the untrained arm provides support that there were no differences in how individuals responded to the cross-over effect of strength. Combined with the mean results, this details that the cross-over effect of 1RM strength was more pronounced in the 1RM group, but individuals responded in a similar fashion depending on their group assignment. As mentioned previously, we could have used a variety of alternative approaches, but this would not be appropriate since the variability would have been present even if there was no intervention (i.e. the variability in the training groups was the same as that of the control group).

Limitations

As with all experimental studies, this study is not without limitations. We used ultrasound for our measurement of muscle size and the reliability of this device is more heavily dependent upon the skill of the technician operating the machine in comparison to other measures of muscle size (e.g. MRI). As such, the quantification of random error (provided by the 6 week control group) does not necessarily generalize to all ultrasound technicians. Additionally, the reliability of ultrasound tends to be slightly worse than that of MRI (17), which may make it more difficult to detect true differential responders in muscle growth. Of course, the reliability is also dependent upon the technician performing the measurements so this may not necessarily be true across all technicians. Additionally, we measured strength using a relatively simple movement in the elbow flexion exercise, and it is entirely possible that more differential responders could be detected had there been a larger skill component involved. Nonetheless, one of the most cited studies within our field as support for large degrees of differential responders to exercise primarily focused on the elbow flexor muscles (6). This also allowed us to test a muscle that is

relatively inactive throughout the day and less likely to be altered by extraneous activity (as compared to the legs, for example). Lastly, our results are specific to resistance exercise, and the health benefits accompanying exercise appear more pronounced in response to endurance exercise. Even so, there are purported benefits of performing resistance exercise and numerous studies continue to analyze differential responders to resistance exercise.

CHAPTER 6: CONLCUSION

Main findings

The purpose of this study was to determine whether individual responses to resistance exercise could be detected with current technology, while also illustrating the magnitude of true variability present. We also sought to examine whether the magnitude of variability present depended on the exercise protocol employed (repeated 1RM testing vs. traditional training). One final purpose was to examine differences in muscle size and strength in response to repeated 1RM training compared to that of traditional training. The main findings from this study were as follows: (1) measurable muscle hypertrophy was only present in the traditional exercise group; (2) 1RM strength increased to a similar extent in both the traditional training group and the 1RM training group; (3) untrained arm strength only increased in the 1RM training group; (4) there were no increases in isokinetic strength in either of the training groups for either the trained and untrained arm; (5) there were no individual responders to changes in muscle size in either the 1RM or traditional training groups; (6) there were no individual responders with respect to changes in isokinetic strength in either the 1RM or traditional training group; (7); only the traditional exercise group demonstrated individual responders in 1RM strength of the trained arm; and (8) there were no individual responders with respect to changes in 1RM strength of the untrained arm.

Hypotheses

1.We hypothesized that only the trained arm within the traditional exercise group would increase muscle size and that this increase would exceed that of both the control and 1RM groups. This hypothesis was correct for all three of the measurement sites.

2. We hypothesized that both the 1RM and traditional exercise groups would increase 1RM strength and isokinetic strength to a similar extent in the trained arm. This hypothesis was partially correct as the 1RM strength increased to a similar extent in both the 1RM and traditional exercise group. We were incorrect, however, with our hypothesis on isokinetic strength, as isokinetic strength did not increase in either of the training groups.

3. We hypothesized that, for the untrained arm, there would not be any changes in muscle size or isokinetic strength in either of the training groups. This hypothesis was correct as neither muscle size nor isokinetic strength changed in the untrained arm for either group.

4. We hypothesized that 1RM strength of the untrained arm would increase to a similar extent in both the 1RM and traditional exercise groups. This hypothesis was not correct as only the 1RM training group increased 1RM strength and this increase was greater than that of both the control group and the traditional exercise group.

5. As for the presence of individual responders to exercise, we hypothesized that there would be true individual responses in 1RM strength of both the exercised and non-exercised contralateral limb in both the traditional training and 1RM training groups. This hypothesis was mostly incorrect, as the only individual responders we could detect involved that of the trained arm in the traditional exercise group. No other individual responses could be detected for changes in 1RM strength.

6. With respect to isokinetic strength, we hypothesized individual responders would be present in the trained arm of both the 1RM and traditional exercise groups, but no individual responders would be detectable in the untrained arm. This hypothesis was only partially correct as there were no individual responders in isokinetic strength among either group for both the trained and untrained arms.

7. Lastly, we hypothesized there would be no true individual variability with respect to changes in muscle size in either arm for either of the training groups. This hypothesis was correct as no individual responders could be detected for changes in muscle size.

Significance of findings

The results of the present study provide a more appropriate way to analyze individual responders to resistance exercise and detail the importance of including a time matched control group to detail the magnitude of random error present over time. Additionally, we show that, when analyzed appropriately, only about 20% of individuals performing traditional resistance exercise could be classified as differential responders in 1RM strength; whereas no one could be detected as a differential responder from the group mean with respect to muscle growth or isokinetic strength. Future studies examining differential responders to resistance exercise should include a time matched control group and only analyze those individuals that can truly be classified as responding differently from the group mean. This can be done using the degree of random error that occurs over a similar time frame (i.e. based off the standard deviation of the change score in a time matched control group). This is of extreme importance because studies analyzing the molecular causes of individual responses to exercise can cost upwards of $10 million for a single study (8). Even at such high costs, these studies often provide little to no information on why people respond differently to exercise since they may be largely analyzing

random error as opposed to true differences in the exercise response. We propose an alternative way of analyzing this data, and this alternative approach may help explain some of the null or nonreplicable findings.

Future Research

The findings of our present study provide an avenue for future research questions. Some of the questions that may warrant explanation are as follows: (1) Are the mechanisms that increase strength following repeated 1RM testing the same as those which increase strength following traditional exercise? (2) Does exercise volume become more important for increasing strength when performing exercises that involve less of a skill component (i.e. isokinetic/isometric training)? (3) Would the same increase in strength be observed had we performed even less exercise volume in the 1RM group, while also matching the relative exercise intensity? For example, would strength increases be similar when comparing the effects of one maximal isokinetic contraction lasting only seconds as compared to multiple sets of isokinetic exercises? (4) Would there be a different number of individual responders had we measured muscle size using an alternative measurement technique or alternative ultrasound technician? (5) Are there any genes that can be linked to more favorable exercise outcomes when only assessing those who truly respond differently from the group mean? (6) Would the number of differential responders to exercise be different if the training apparatus made the training protocol even more homogenous across individuals (i.e. using an identical range of motion and speed being performed at maximal effort with isokinetic testing via dynamometry)? (7) Would it be easier to detect individual responders to strength if both the training and strength test were performed using a more objective assessment such as dynamometry? These questions remain to be answered and provide avenues for future research.

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VITA

EDUCATION

May 2019 Anticipated — Ph.D. Health and Kinesiology, The University of Mississippi

May 2016 — M.S. Exercise Science, The University of Mississippi

May 2012 — B.A. Health and Physical Education, Rowan University

PROFESSIONAL EXPERIENCE

2014-Present — Graduate Teaching Assistant at The University of Mississippi; Oxford, MS

Spring 2012 — Health and Physical Education Teaching Assistant at Deptford High School; Deptford, NJ

Fall 2011 — Health and Physical Education Teaching Assistant at Lake Tract Elementary School; Deptford, NJ

UNIVERSITY TEACHING EXPERIENCE

1) Exercise Testing and Prescription Lab (ES 457)
2) Exercise Physiology Lab (ES 349)
3) Measurement and Statistics in Exercise Science (ES 351)
4) Trends and Topics in Exercise Science (ES 391)
5) Personal and Community Health (HP 191)
6) First Aid and CPR (HP 203)
7) Tennis (EL 147)
8) Weight Lifting (EL 151)
9) Jogging (EL 156)

GRANTS

1) Loenneke JP on behalf of **Dankel SJ**. Principal Investigator (2015). The effects of metabolic stress on muscle size and strength. The Biolayne Foundation. $10,000 (FUNDED). Intellectually contributed to the study design and drafting of the grant.

2) Loenneke JP. Principal Investigator (2015). Can muscle growth occur through resistance training with no external load? American College of Sports Medicine $8,950 (Not Funded). Intellectually contributed to the contents of the grant.
3) Loenneke JP. Principal Investigator (2017). The muscular and vascular effects of very low loads with and without different levels of blood flow restriction." American College of Sports Medicine $10,000 (Not Funded). Intellectually contributed to the contents of the grant.
4) Loenneke JP. Principal Investigator (2017). "Have improper analyses cost us millions: reassessing inter-individual responses to exercise." National Institutes of Aging. $300,000 (Not Funded). Intellectually contributed to the study design and drafting of the grant.
5) Loenneke JP. Principal Investigator (2018). Are there individual responses to two distinct resistance exercise protocols: Or is it all just measurement error?" American College of Sports Medicine $10,000 (Not Funded). Intellectually contributed to the study design and drafting of the grant.
6) Loenneke JP. Principal Investigator (2018). Change in force is dictated by contraction history. American Physiological Society $30,000 (Under Review). Intellectually contributed to the contents of the grant.
7) Loenneke JP. Principal Investigator (2019). "Does inter-repetition rest augment adaptation when effort is matched?" American College of Sports Medicine $10,000 (Under Review). Intellectually contributed to the contents of the grant.

PEER-REVIEWED PUBLICATIONS

1) Abe T, Counts BR, Barnett BE, **Dankel SJ**, Lee K, and Loenneke JP. Associations between Handgrip Strength and Ultrasound-Measured Muscle Thickness of the Hand and Forearm in Young Men and Women. Ultrasound in medicine & biology. 2015;41(8):2125-2130.
2) Barnett BE, **Dankel SJ**, Counts BR, Nooe AL, Abe T, and Loenneke JP. Blood flow occlusion pressure at rest and immediately after a bout of low load exercise. Clinical physiology and functional imaging. 2016;36(6):436-440.
3) Buckner SL, Abe T, Counts BR, **Dankel SJ**, Barnett BE, and Loenneke JP. Muscle and fat mapping of the trunk: a case study. Journal of Ultrasound. 2015;18(4):399-405.
4) Counts BR, **Dankel SJ**, Barnett BE, Kim D, Mouser JG, Allen KM, Thiebaud RS, Abe T, Bemben MG, and Loenneke, JP. The influence of relative blood flow restriction pressure on muscle activation and muscle adaptation. Muscle & nerve. 2016;53(3):438-445.
5) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Participation in muscle strengthening activities as an alternative method for the prevention of multimorbidity. Preventive Medicine. 2015;81:54-57.
6) **Dankel SJ,** Loenneke JP, and Loprinzi PD. The impact of overweight/obesity duration on the association between physical activity and cardiovascular disease risk: an application of the "fat but fit" paradigm. International Journal of Cardiology. 2015;201:88-89.
7) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Physical activity and diet on quality of life and mortality: The importance of meeting one specific or both behaviors. International Journal of Cardiology. 2016;202:328-330.

8) **Dankel SJ**, Jessee MB, Abe T, Loenneke JP. The Effects of Blood Flow Restriction on Upper-Body Musculature Located Distal and Proximal to Applied Pressure. Sports Medicine. 2016;46(1):23-33.
9) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Does the fat-but-fit paradigm hold true for all-cause mortality when considering the duration of overweight/obesity? Analyzing the WATCH (Weight, Activity and Time Contributes to Health) Paradigm. Preventive Medicine. 2016;83:37-40.
10) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Determining the Importance of Meeting Muscle-Strengthening Activity Guidelines: Is the Behavior or the Outcome of the Behavior (Strength) a More Important Determinant of All-cause Mortality?. Mayo Clinic Proceedings. 2016;91(2):166-174.
11) Buckner SL, **Dankel SJ,** Counts BR, Barnett BE, Jessee MB, Mouser JG, Halliday TM, and Loenneke JP. Do rhythms exist in elbow flexor torque, oral temperature and muscle thickness during normal waking hours? Physiology and Behavior. 2016;160:12-17.
12) Buckner SL, **Dankel SJ,** Mattocks KT, Jessee MB, Mouser JG, Counts BR. and Loenneke JP. The problem of muscle hypertrophy: revisited. Muscle & Nerve. 2016;54(6):1012-1014.
13) Buckner SL, **Dankel SJ**, Counts BR, Jessee MB, Mouser JG, Mattocks KT, Laurentino GC, Abe T, and Loenneke JP. Influence of Cuff Material on the Blood Flow Restriction Stimulus in the Upper Body. The Journal of Physiological Sciences. 2017;67(1):207-215.
14) Buckner SL, **Dankel SJ,** Counts BR, Barnett BE, Jessee MB, Mouser JG, Halliday TM, and Loenneke JP. Does the time of your health screening alter your "health"? International Journal of Cardiology. 2016;220:524-526.
15) Buckner SL, Jessee MB, Mattocks KT, Mouser JG, Counts BR, **Dankel SJ**, and Loenneke JP. Determining strength: A case for multiple methods of measurement. Sports Medicine. 2017;47(2):193-195.
16) Buckner SL, Mouser JG, Jessee MB, **Dankel SJ**, Mattocks KT, and Loenneke JP. What does individual strength say about resistance training status? Muscle & nerve. 2017;55(4):455-457.
17) Mattocks KT, **Dankel SJ**, Mouser JG, Buckner SL, Jessee MB, Counts BR, Laurentino GC, Loenneke JP. Periodization: What is it good for? Journal of Trainology. 2016;5(1):6-12.
18) Counts BR, Rossow LM, Mattocks KT, Mouser JG, Jessee MB, Buckner SL, **Dankel SJ**, Loenneke JP. Let's Talk About Sex: Where are the Young Females in Blood Flow Restriction Research. Clinical Physiology and Functional Imaging. 2018;38(1):1-3.
19) Counts BR, Buckner SL, **Dankel SJ**, Jessee MB, Mattocks KT, Mouser JG, Laurentino GC, and Loenneke JP. The acute and chronic effects of "NO LOAD" resistance training. Physiology and Behavior. 2016;164(Pt A):345-352.
20) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Combined Associations of Muscle-Strengthening Activities and Accelerometer-Assessed Physical Activity on Multimorbidity: Findings From NHANES. American Journal of Health Promotion. 2017;31(4):274-277.
21) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Mild depressive symptoms amongst Americans in relation to physical activity, current overweight/obesity, and self-reported history of overweight/obesity. International Journal of Behavioral Medicine. 2016;23(5):553-60.

22) **Dankel SJ,** Loenneke JP, and Loprinzi PD. The WATCH (Weight Activity and Time Contributes to Health) paradigm and quality of life: the impact of overweight/obesity duration on the association between physical activity and health-related quality of life. The International Journal of Clinical Practice. 2016; 70(5):409-15.
23) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Dose-Dependent Association between Muscle Strengthening Activities and All-Cause Mortality: Prospective Cohort Study Among a National Sample of Adults in the USA. Archives of Cardiovascular Disease. 2016;109(11):626-633.
24) **Dankel SJ,** Loenneke JP, and Loprinzi PD. The Individual, Joint, and Additive Interaction Associations of Aerobic-Based Physical Activity and Muscle Strengthening Activities on Metabolic Syndrome. International Journal of Behavioral Medicine. 2016;23(6):707-713.
25) **Dankel SJ**, Buckner SL, Jessee MB, Mattocks KT, Mouser JG, Counts BR, Laurentino GC, Abe T, Loenneke JP. Post-exercise blood flow restriction attenuates muscle hypertrophy. European Journal of Applied Physiology. 2016; 116(10):1955-63.
26) **Dankel SJ**, Jessee MB, Mattocks KT, Mouser JG, Counts BR, Buckner SL, Loenneke JP. Training to fatigue: The answer for standardization when assessing muscle hypertrophy?. Sports Medicine. 2017;47(6):1021-1027.
27) **Dankel SJ**, Mattocks KT, Jessee MB, Buckner SL, Mouser JG, Counts BR, Laurentino GC, Loenneke JP. Frequency: The Overlooked Resistance Training Variable for Inducing Muscle Hypertrophy?. Sports Medicine. 2017;47(5):799-805.
28) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Health Outcomes in Relation to Physical Activity Status, Overweight/Obesity, and History of Overweight/Obesity: A Review of the WATCH Paradigm. Sports Medicine. 2017;47(6):1029-1034.
29) **Dankel SJ**, Mouser JG, Mattocks KT, Counts BR, Jessee MB, Buckner SL, Loprinzi PD, and Loenneke JP. The widespread misuse of effect sizes. Journal of Science and Medicine in Sport. 2017;20(5):446-450.
30) **Dankel SJ,** Loenneke JP, and Loprinzi PD. The Impact of Overweight/Obesity Duration and Physical Activity on Telomere Length: An Application of the WATCH Paradigm. Obesity Research and Clinical Practice. 2017;11(2):247-252.
31) Jessee MB, Buckner SL, **Dankel SJ**, Counts BR, Abe T, and Loenneke JP. The Influence of Cuff Width, Sex, and Race on Arterial Occlusion: Implications for Blood Flow Restriction Research. Sports Medicine. 2016;46(6):913-21.
32) Buckner SL, **Dankel SJ**, Mattocks KT, Jessee MB, Mouser JG, Counts BR, Laurentino GC, and Loenneke JP. Differentiating Swelling and Hypertrophy Through Indirect Assessment of Muscle Damage in Untrained Men Following Repeated Bouts of Resistance Exercise. European Journal of Applied Physiology. 2017;117(1):213-224.
33) Buckner SL, Mouser JG, **Dankel SJ**, Jessee MB, Mattocks KT, and Loenneke JP. The General Adaptation Syndrome: Potential Misapplications to Resistance Exercise. Journal of Science and Medicine in Sport. 2017;20(11):1015-1017.
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35) Buckner SL, **Dankel SJ**, Mouser JG, Mattocks KT, Jessee MB, Loenneke JP. Chasing the Top Quartile of Cross-Sectional Data: Is it Possible with Resistance Training? Medical Hypotheses. 2017;108:63-68.
36) Counts BR, Buckner SL, Mouser JG, **Dankel SJ**, Jessee MB, Mattocks KT, Loenneke JP. Muscle growth: To infinity and beyond? Muscle and Nerve. 2017;56(6):1022-1030.
37) **Dankel SJ**, Counts BR, Barnett BE, Bucker SL, Abe T, Loenneke JP. Muscle adaptations following 21 consecutive days of strength test familiarization compared with traditional training. Muscle & Nerve. 2017; 56(2):307-314.
38) **Dankel SJ**, Buckner SL, Jessee MB, Mattocks KT, Mouser JG, Counts BR, Laurentino GC, Loenneke JP. Can Blood Flow Restriction Augment Muscle Activation During High Load Training? Clinical Physiology and Functional Imaging. 2018;38(2):291-295.
39) **Dankel SJ**, Buckner SL, Counts BR, Jessee MB, Mouser JG, Mattocks KT, Laurentino GC, Abe T, Loenneke JP. The Acute Muscular Response to Two Distinct Blood Flow Restriction Protocols. Physiology International. 2017;104(1):64-76.
40) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Answer to the letter of Reza Pakzad and Saeid Safiri. Archives of Cardiovascular Disease. 2017;110(4):274.
41) **Dankel SJ**, Mouser JG, Jessee MB, Mattocks KT, Buckner SL, Loenneke JP. Post-exercise blood flow restriction attenuates hyperemia similarly in males and females. European Journal of Applied Physiology. 2017;117(8):1707-1712.
42) **Dankel SJ**, Jessee MB, Buckner SL, Mouser JG, Mattocks KT, Loenneke JP. Are higher blood flow restriction pressures more beneficial when lower loads are used? Physiology International. 2017;104(3):247-257.
43) **Dankel SJ**, Mattocks KT, Jessee MB, Buckner SL, Mouser JG, Loenneke JP. Do Metabolites that are Produced During Resistance Exercise Enhance Muscle Hypertrophy? European Journal of Applied Physiology. 2017;117(11):2125-2135.
44) **Dankel SJ,** Loenneke JP, and Loprinzi PD. Cancer-specific Mortality Relative to Engagement in Muscle Strengthening Activities and Lower Extremity Strength. Journal of Physical Activity & Health. 2018;15(2):144-149.
45) **Dankel SJ**, Buckner SL, Jessee MB, Mouser JG, Mattocks KT, Abe T, Loenneke JP. Correlations do not show cause and effect: Not even for changes in muscle size and strength. Sports Medicine. 2018;48(1):1-6.
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SCIENTIFIC ABSTRACTS/ORAL PRESENTATIONS

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2) Counts BR, **Dankel SJ**, Barnett BE, Abe T, Loenneke JP. High relative pressures do not augment changes in early phase muscular adaptations during blood flow restricted exercise. ACSM National Conference, May 2015, San Diego, California
3) **Dankel SJ**, Barnett BE, Counts BR, Nooe AL, Abe T, Loenneke JP. Blood flow occlusion pressure at rest and immediately after a bout of low load exercise. ACSM National Conference, May 2015, San Diego, California.
4) Counts BR, Buckner SL, **Dankel SJ**, Jessee MB, Mattocks KT, Mouser JG, Laurentino GC, and Loenneke JP. The Acute Response to No Load Exercise: Is it Sufficient? ACSM National Conference, May 2016, Boston, Massachusetts.
5) Barnett BE, Buckner SL, **Dankel SJ**, Counts BR, Jessee MB, Mouser JG, Halliday TM and Loenneke JP. Circadian Rhythms in Blood Glucose and Blood Pressure: Are they Reproducible? ACSM National Conference, May 2016, Boston, Massachusetts.

6) Mouser JG, Buckner SL, Counts BR, **Dankel SJ**, Jessee MB, Mattocks KT, Laurentino GC, and Loenneke JP. Venous versus Arterial Blood Flow Restriction: The Impact of Cuff Width. ACSM National Conference, May 2016, Boston, Massachusetts.
7) Ingram JW, Buckner SL, **Dankel SJ**, Counts BR, Mouser JG, Abe T, Laurentino GC, and Loenneke JP. The influence of time on determining blood flow restriction pressure. ACSM National Conference, May 2016, Boston, Massachusetts.
8) Mattocks KT, Buckner SL, **Dankel SJ**, Counts BR, Jessee MB, Mouser JG, Laurentinio GC, Abe T, and Loenneke JP. The Influence of Cuff Material on the Blood Flow Restriction Stimulus in the Upper Body. ACSM National Conference, May 2016, Boston, Massachusetts.
9) Laurentino GC, Mouser JG, Buckner SL, Counts BR, **Dankel SJ**, Jessee MB, Mattocks KT, Loenneke JP, Tricoli V. The influence of cuff width on regional muscle growth: Implications for Blood Flow Restriction Training. ACSM National Conference, May 2016, Boston, Massachusetts.
10) Jessee MB, Buckner SL, **Dankel SJ**, Counts BR, Abe T, and Loenneke JP. The Influence of Cuff Width and Sex on Arterial Occlusion: Implications for Blood Flow Restriction Research. ACSM National Conference, May 2016, Boston, Massachusetts.
11) Loenneke JP, Buckner SL, **Dankel SJ**, Jessee MB, Counts BR, Mouser JG, Mattocks KT, Laurentino GC, and Abe T. The Influence of Cuff Material on the Acute Muscular Response to Blood Flow Restricted Exercise in the Upper Body. ACSM National Conference, May 2016, Boston, Massachusetts.
12) Buckner SL, **Dankel SJ**, Counts BR, Barnett BE, Jessee MB, Mouser JG, Halliday TM, and Loenneke JP. The Influence of Circadian Rhythms on Upper Body Isometric Strength, Muscle Thickness and Body Temperature. ACSM National Conference, May 2016, Boston, Massachusetts.
13) **Dankel SJ**, Counts BR, Barnett BE, Buckner SL, Abe T, Zourdos MC, and Loenneke JP. Muscle adaptation to 21 Straight Days of Elbow Flexor Exercise in Trained Individuals. ACSM National Conference, May 2016, Boston, Massachusetts.
14) Mouser JG, Laurentino GC, **Dankel SJ**, Buckner SL, Jessee MB, Counts BR, Mattocks KT, Loenneke JP. Blood Flow in Humans During Low-Load Exercise with and without Blood Flow Restriction. ACSM National Conference, May 2017, Denver, Colorado.
15) Mattocks KT, Jessee MB, Counts BR, Buckner SL, Mouser JG, **Dankel SJ**, Laurentino GC, Loenneke JP. Effects of Different Levels of Blood Flow Restriction on Arterial Occlusion Pressure and Perceptual Responses. ACSM National Conference, May 2017, Denver, Colorado.
16) Loenneke JP, **Dankel SJ**, Jessee MB, Buckner SL, Mouser JG, Mattocks KT. Are Higher Blood Flow Restriction Pressures More Beneficial When Lower Loads Are Used?. ACSM National Conference, May 2017, Denver, Colorado.
17) Jessee MB, Mattocks KT, Counts BR, Buckner SL, Mouser JG, **Dankel SJ**, Laurentino GC, Loenneke JP. The Acute Muscular Responses to Blood Flow Restricted Exercise Using Low and High Relative Pressures. ACSM National Conference, May 2017, Denver, Colorado.

18) Buckner SL, **Dankel SJ**, Mattocks KT, Jessee MB, Mouser JG, Counts BR, Laurentino GC, Loenneke JP. Differentiating Swelling and Hypertrophy Following Repeated Bouts of Resistance Exercise. ACSM National Conference, May 2017, Denver, Colorado.
19) **Dankel SJ**, Jessee MB, Buckner SL, Mouser JG, Mattocks KT, Loenneke JP. Cardiovascular and Perceptual Responses to Various Blood Flow Restriction Pressures and Exercise Loads. ACSM National Conference, May 2017, Denver, Colorado.
20) **Dankel SJ**, Mouser JG, Mattocks, KT, Jessee MB, Buckner SL, Abe T, Loenneke JP. The effects of cuff width on hemodynamics in the legs during blood flow restriction. ACSM National Conference, May 2018, Minneapolis, Minnesota.
21) Mouser JG, Mattocks KT, **Dankel, SJ**, Buckner SL, Jessee MB, Bell ZW, Abe T, Loenneke JP. Cardiovascular Responses to Blood Flow Restriction and Very Low Load Resistance Exercise in the Upper Body. ACSM National Conference, May 2018, Minneapolis, Minnesota.
22) Jessee MB, Buckner SL, Mattocks KT, Mouser JG, **Dankel SJ**, Bell ZW, Abe T, Loenneke JP. Very Low Load Resistance Exercise Is Augmented By Blood Flow Restriction In The Lower Body. ACSM National Conference, May 2018, Minneapolis, Minnesota.
23) Mattocks KT, Mouser JG, Jessee MB, **Dankel SJ**, Buckner SL, Bell ZW, Abe T, Loenneke JP. Acute Hemodynamic Response to Very Low Load Resistance Exercise With or Without Blood Flow Restriction. ACSM National Conference, May 2018, Minneapolis, Minnesota.
24) Buckner SL, Jessee MB, **Dankel SJ**, Mouser JG, Mattocks KT, Bell ZW, Abe T, Loenneke JP. Muscular responses to very low load resistance exercise with blood flow restriction in the upper body. ACSM National Conference, May 2018, Minneapolis, Minnesota.
25) Bell ZW, Buckner SL, Jessee MB, Mouser JG, Mattocks KT, **Dankel SJ**, Abe T, Loenneke JP. Perceptual And Cardiovascular Responses To Very Low Load Exercise With And Without Blood Flow Restriction. ACSM National Conference, May 2018, Minneapolis, Minnesota.

PUBLIC PRESS RELEASES

1) Lower Extremity Review (lermagazine): Strength drives survival: But benefits of training appear complex. October 2017.
2) University of Mississippi News: UM Researchers Make Waves in Blood Pressure Research. March 2019.

REFEREED JOURNALS

2016 – Present Muscle and Nerve
2016 – Present Journal of Trainology
2016 – Present Plos One
2017 – Present JAMA Pediatrics
2017 – Present AIMS Public Health
2018 – Present Rejuvenation Research
2018 – Present PeerJ
2018 – Present International Journal of Sports Physiology and Performance
2018 – Present Journal for the Measurement of Physical Behaviour
2018 – Present Research Quarterly for Exercise and Sport

2018 – Present International Journal of Sports Medicine
2018 – Present BMC Medicine
2018 – Present Obesity Research & Clinical Practice
2019 – Present Heliyon
2019 – Present Frontiers in Physiology
2019 – Present Journal of Physical Activity and Health
2019 – Present Preventive Medicine Reports

HONORS AND AWARDS

2015 J. Robert Blackburn Graduate Student Award in Exercise Science
2017 J. Robert Blackburn Graduate Student Award in Exercise Science
2018 Graduate Student Award in Applied Sciences
2019 J. Robert Blackburn Graduate Student Award in Exercise Science
2019 HESRM American Kinesiology Association (AKA) Doctoral Scholar Award
2019 American Kinesiology Association (AKA) National Doctoral Student Award
2019 Interdisciplinary Certificate in Applied Statistics
2019 1st Place in thee Graduate Student Council Research Symposium

MENTORSHIP

Jeremy Loenneke, Ph.D.
The University of Mississippi (2014 – 2019)

SERVICE

2015 Rebel Man Sprint Triathlon volunteer: Bike Route Coordinator
2015 ACSM National Conference - Student Help Desk volunteer
2015 Amtrykes in Action race volunteer
2015 University of Mississippi Junior Orientation Representative from Exercise Science
2016 Rebel Man Sprint Triathlon volunteer: Bike Route Coordinator
2016 University of Mississippi Junior Orientation Representative from Exercise Science
2016 ACSM National Conference: Registration Desk volunteer
2016 Special Olympics Turkey Bowl volunteer
2017 University of Mississippi Graduate Program Representative from Exercise Science
2017 Volunteer Judge for Mississippi Region VII Science Fair
2018 Volunteer Judge for Mississippi Region VII Science Fair
2018 Faculty Hiring Committee Student Member
2018 Rebel Man Sprint Triathlon volunteer: Supervisor
2018 Student Advisor
2019 Volunteer Judge for Mississippi Region VII Science Fair
2019 Rebel Man Sprint Triathlon volunteer: First Aid Station