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## POST-ACTIVATION POTENTIATION: DECAY OR FATIGUE DELAY

By

Ryan L. Meidinger

## THESIS

Submitted to Northern Michigan University In partial fulfillment of the requirements for the degree of

## M.S. EXERCISE SCIENCE

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# SIGNATURE APPROVAL FORM

Post-Activation Potentiation Decay or Fatigue Delay

This thesis by Ryan Meidinger is recommended for approval by the student's Thesis Committee and Department Head in the Department of Health and Human Performance and by the Assistant Provost of Graduate Education and Research.

Committee Chair: Randall L. Jensen

First Reader: Sarah Clarke

Second Reader: Lanae Joubert

Department Head: Elizabeth Wourinen

Interim Director of Graduate Education and Research: Dr. Lisa S. Eckert Date

Date

Date

Date

Date

#### ABSTRACT

## POST-ACTIVATION POTENTIATION DECAY OR FATIGUE DELAY

By

#### Ryan L. Meidinger

Post-activation potentiation has been shown to improve jumping performance and other ballistic activities. The improvements in performance have been attributed to four main mechanisms, but the most important mechanism to the current study is the improvement in neural activity that leads to greater levels of potentiation. Post-activation potentiation has been shown to be stimulated by a maximal activity, called a conditioning contraction, and can be used as a warm up. In studies that have not shown the effects of post-activation potentiation, the proposed reason is fatigue, but the interaction of postactivation potentiation and fatigue have not been thoroughly tested. The purpose of this study was to assess the interaction of fatigue and post-activation potentiation. The present study tested recreational, healthy, lower body resistance trained participants who took part in 3 days of testing (familiarization/baseline testing and 2 fatigue test days). The results of the current study showed no significant difference between the control and experimental days for any of the variables measured. The results of this study demonstrate that the use of a conditioning contraction during a warm up protocol will not be a detriment to performance during repeated jumps and could be used in a warm up. The present study may have been limited by a small number of participants, individual variation, and training status of the participants.

**KEY WORDS**: impulse, conditioning contraction, degradation of performance, synaptic strength

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This thesis follows the format guidelines of Sport Biomechanics.

# PREFACE

The cost of the present study was paid out of pocket from the author of the present study. One credit of the thesis was paid for by an Excellence in Education Grant that will aid the author of the present study to travel to Cologne, Germany and present at the International Society of Biomechanics in Sport annual conference. The equipment used for the present study was made available by Northern Michigan University's School of Health and Human Performance.

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**Figure 1:** Depiction of the sledge used in the current study (used with permission Comyns et al., 2007). The depiction above displays a participant performing a single leg jump, whereas the present study utilized both legs while performing a rebound jump.

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#### **CHAPTER ONE: MANUSCRIPT SUBMISSION**

**Introduction:** Post-activation potentiation consists of both neural and muscular potentiation and fatigue has been shown to be a detriment to mechanisms that effect potentiation of the nervous system and muscle (Tillin & Bishop, 2009). Post-activation potentiation is known to improve performance (Tillin & Bishop, 2009) and fatigue is known to decrease performance (Bishop, 2003a & b). For this reason, many studies have stated that fatigue is present if post-activation potentiation is not present. Muscle can be modeled in a bimodal fashion (unpotentiated through a spectrum of potentiation) (Brown & Loeb, 1998) and neurons can go through a common process (depression and potentiation) (Junge, et al., 2004). As it pertains to the present study, potentiation is a rise in muscular contractile or nervous strength from a prior stimulus (conditioning contraction) (Brown & Loeb, 1998; Junge, et al., 2004). Post-activation potentiation is defined as a rise in potentiation from a prior conditioning contraction, leading to increased force production and rate of force development that improves performance (Brown & Loeb, 1998; Hodgson, Docherty, & Robbins, 2005; Xenofondos, et al., 2010).

The muscular mechanisms of post-activation potentiation consist of phosphorylation of myosin regulatory light chains and a change in pennation angle (Brown & Loeb, 1998; Hodgson, Docherty, & Robbins, 2005; Reardon, et al., 2014). The neural mechanisms of post-activation potentiation are increased recruitment of muscle fibers from increased synaptic strength (potentiation) and higher order motor neurons (Gullich & Schmidtbleicher, 1996; Hodgson, Docherty, & Robbins, 2005).

The use of a conditioning contraction can stimulate one, if not all of the mechanisms stated above. For this reason, a conditioning contraction can be considered a

type of warm up activity because it is known to potentiate the entire motor unit (Brown & Loeb, 1998; Hodgeson, Docherty, & Robbins, 2005; Tillin & Bishop, 2009; Xenofondos, Laparidis, Galazoulas, Bassa, & Kotzamanidis, 2010; Wilson, et al., 2013), not unlike a warm up (Bishop, 2003a). A warm up has been shown to decrease the effect of fatigue (Bishop, 2003a, 2003b) and there is a possibility that a conditioning contraction could be used to do the same (Andrews, Horodyski, MacLeod, Whitten, & Behm, 2016; Fletcher & Jones, 2004; Rassier & MacIntosh, 2000). If a warm up is supposed to decrease the effect of fatigue and a conditioning contraction can be a warm up, then post-activation potentiation could decrease the effect of fatigue. However, there is minimal research on post-activation potentiation's interaction with fatigue (Andrews, Horodyski, MacLeod, Whitten, & Behm, 2016; Bishop, 2003; Fletcher & Jones, 2004; Rassier & MacIntosh, 2000) and fatigue is often implicated for a lack of improved performance in postactivation potentiation studies (Hodgeson, Docherty, & Robbins, 2005; McCann & Flanagan, 2010; Morana & Perrey, 2009; Sale, 2002; Tillin & Bishop, 2009; Wilson, et al., 2013; Xenofondos, Laparidis, Galazoulas, Bassa, & Kotzamanidis, 2010).

There is evidence of post-activation potentiation's effect on fatigue in low-and high-frequency fatiguing activities during endurance activities (Rassier & MacIntosh, 2000). Rassier and MacIntosh (2000) found that twitch-potentiation, which is postactivation potentiation evoked by electrical stimulus, was able to overcome a lowfrequency fatiguing activity. However, high frequency fatigue was not shown to be overcome by twitch-potentiation (Rassier & MacIntosh, 2000). Andrews et al. (2016) found that post-activation potentiation may overcome the effects of fatigue, but they only assessed it in a unilateral squat to stimulate post-activation potentiation comparing the

conditioned leg and non-conditioned leg. They showed that the leg that did not do the conditioning contraction showed a decrease in performance before the leg that did the conditioning contraction. However, Andrews et al. (2016) only assessed the presence of fatigue over an extended period of time with large rest intervals between attempts. These previously stated limitations lead to the purpose of the present study, which was to assess the effect of post-activation potentiation on the degradation of jumping performance from fatigue during repeated rebound jumps.

#### Methods

#### **Subjects**

The present study included 4 male and 3 female participants with a mean age of 22.7 ( $\pm$ 1.4) years (height: 1.72 $\pm$ 8.7cm, mass: 77.3 $\pm$ 9.1kg), recruited from the student population at Northern Michigan University. The present study was not able to recruit the amount of participants calculated by "G-power" (Faul, Erdfelder, & Lang; 2007) to reach statistical significance (12-14) from time constraints and drop out of participants. The participants were required to have two plus years of lower body resistance training experience to be included in this study. The inclusion criteria were chosen to allow for decreased injury risk and increased chance of having an effect from post-activation potentiation (Sale, 2002). The participants had a mean of 6.4 ( $\pm$ 3.8) years of lower body resistance training experience. Each participant was given a Physical Activity Readiness Questionnaire (PAR-Q) (Thomas, Readings, & Shepard, 2002) to demonstrate that they were healthy and signed an informed consent form. The current study received Institutional Review Board approval (HS17-843) from Northern Michigan University.

The participants were instructed to avoid caffeine the day of testing (from waking to testing) and alcohol for 48 hours prior to their test to reduce the effect of these two substances on the central nervous system. Caffeine and alcohol were avoided in the present study as they could affect the central nervous system (Eckart, et al., 1998; Davis et al., 2003). No exact assessment of caffeine dosage was taken, but the participants in the current study stated a range of 0 to 4 cups of coffee a day (mean  $1.4\pm1.4$ ) on a regular basis. Participants were asked to eat 2 to 4 hours prior to testing and were rescheduled or excluded if they stated they had not eaten within this time frame prior to testing. Participants were allowed to hydrate in any way they saw fit during the testing protocols and were asked to hydrate the day of the protocols. Participants were rescheduled or excluded if they stated that they had not followed the diet and hydration guidelines.

#### Procedures

The present study took place in the Exercise Science Lab at Northern Michigan University. Testing consisted of three days of roughly three hours of total testing, as follows: 15-20 minutes familiarization/baseline testing, and 30-45 minutes on each control and experimental testing days. The counter movement jumps were performed in the sledge, as presented by Comyns and colleagues (2007), and shown in Figure 1. The countermovement was used in the present study because it can be used to estimate drop height, which has been shown to be most effective at 75% of countermovement height, as unpublished data has shown that this is the optimal drop jump height (Song, et al., In Press). After the countermovement jumps, the participants were placed in the sledge and dropped from an estimated 75% of max countermovement jump height. The participants

performed three to five rebound jumps to familiarize themselves with jumping on the sledge.

The order of participation in the experimental or control testing days was achieved through a randomized block design. On the control day the participants were fitted with the electromyography (EMG) electrode on the rectus femoris, which was placed half way between the anterior superior iliac spine and the superior portion of the patella. The participants then warmed up on a cycle ergometer for 5 minutes similar to that of the familiarization/baseline testing day. After their warm up, participants had a five-minute break where they were placed into the chair of the sledge (see Figure 1). The participants were placed in position with a knee angle similar to their countermovement jump loading position. The knee angle of the loading position was measured at 240 Hz (Casio High Speed Exilim, Tokyo, Japan) from the sagittal view on the familiarization day during the three countermovement jumps. The mean value was taken to determine knee angle used for the Maximal Voluntary Isometric Contractions (MVIC). This position was used for a five second MVIC, used to normalize EMG amplitude. After the MVIC the participants rested for 18 minutes to allow the effects of post-activation potentiation to subside (Chui, et al., 2003; Wilson, et al., 2013). One minute prior to the end of the 18 minute rest, lactate was measured via finger stick technique (Lactate Scout+, EKF, Penarth, England). At the end of the 18 minute rest, participants started their fatigue protocol. Lactate was measured in the present study to estimate effect of the anaerobic metabolism during the fatigue protocol (Bishop, 2003b).

The fatigue protocol consisted of the participants being dropped from 75% of their best countermovement jump height and performed repeated rebound jumps until

they could not jump to 75% of their best countermovement jump height, or they decided to stop. During the fatigue protocol, participants were instructed to hold onto the shoulder straps of the chair and jump off the force plate as fast and as high as possible. After the participants stopped jumping, they remained in the chair for 5 minutes until the postfatigue protocol lactate was measured. The fatigue protocol and lactate measurement protocol were measured at consistent times, in the same fashion, on both testing days.

On the experimental day the participants did the same warm up, rest, and MVIC as on the control day. However, after the MVIC participants rested for six minutes prior to performing three MVIC conditioning contractions, each separated by five seconds. (Chui, et al., 2003; Wilson, et al., 2013) Lactate measurements, drop height, the fatigue protocol, and stoppage were all conducted the same as on the control day. A representation of the familiarization/baseline testing, control, and experimental days is shown in Figure 2. On both experimental and control days, total number of repetitions until the subjects stopped themselves or were stopped by the researchers, were counted (stated as repetitions to cutoff in Table 1).

Peak vertical ground reaction forces, rate of force development, impulse and flight time were measured on a force platform (OR6-2000 Advanced Mechanical Technology, INC. [AMTI], Watertown, MA, USA). Peak vertical ground reaction forces were measured as the maximum recorded vertical force recorded by the force platform. Impulse was calculated as the area under the force curve. When the participant was in contact with the force plate ground reaction forces were measured and the time between contacts were used to assess flight time (the main variable of jumping performance). The

number of rebound jumping repetitions was measured during each fatigue protocol to assess differences between days.

The current study assessed rate of force development as peak force divided by the time to peak force (Haff, Ruben, Lider, Twine, & Cormie, 2015). Impulse and flight time were used to quantify the degradation of performance through a fatigue index ((peak<sub>measurement</sub>-lowest<sub>measurement</sub>)/peak<sub>measurement</sub>) (Naharudin & Yusof, 2013). Dal Pupo and colleagues (2014) validated rebound jumps as a measure of anaerobic fatigue in the same fashion as the present study. However, to the best of the authors' knowledge impulse has not been used as a measurement to assess fatigue.

Muscular activity of the rectus femoris was assessed via EMG with electrodes (Noraxon, Scottsdale, AZ, USA) attached to the participant's skin (Cram, Kasman, Holtz, 1998). The participant's skin was wiped and scrubbed with an alcohol swab to decrease impedance. The EMG amplitude was rectified, band pass filtered at 10 and 450 HZ, and normalized to the MVIC for EMG amplitude. In addition, Fast Fourier Transformation was used to determine the mean frequency (Noraxon myoFORCE EMG Analysis Software; Noraxon, Scottsdale, AZ, USA). EMG was assessed during contact with the force plate. EMG mean frequency was used to assess the rate of decline in strength of the peripheral nervous system, measured during loading for the rebound jumps. The rate of decline was assessed by using the slope of a best fit line of the mean frequency. Mean frequency can show a decrease in the frequency of activity of the nervous system as it fatigues (Gerdle, Larsson, & Karlsson; 2000).

#### Statistical Analysis

A repeated measures ANOVA was performed to determine differences between the conditions for the following variables: accumulation of impulse, impulse fatigue index, peak impulse, rate of force development, flight time fatigue index, slope of electromyography amplitude, slope of mean frequency, accumulation of lactate, ground reaction forces, repetitions to cut off, flight time, and impulse fatigue index. Accumulation of impulse was calculated by adding all impulses from each jump together for each participant. The fatigue index was calculated, as follows: peak of the variable minus the minimum, all divided by the peak of the variable. Effect size was assessed as described by Hopkins (2002) with a scale based on *f*-values converted to a partial eta square  $(f=(n_p^{-2/}(1-n_p^{-2}))^{0.5}$ . Effect size values were noted as trivial (<0.04), small (0.041-0.249), medium or moderate (0.25=-0.549), large (0.55-0.799), and very large (>0.8) (Hopkins, 2002).

#### Results

None of the results from the present study were significantly different between the control and experimental days. The accumulation of lactate neared significance with a p-value of 0.071 and had a moderate effect size of 0.445. The rest of the variables were not close to significance, the next closest being peak impulse (p = 0.104). The means and standard deviations, as well as results from the repeated measures ANOVA, are displayed in Table 1. Next the participants were divided into a group that increased accumulated impulse (deemed responders) and decreased accumulated impulse (non-responders) to attempt to further explain these results (see Table 2). The average training ages of the responders were  $8.5\pm3.7$  and non-responders were  $3.7\pm1.2$  years.

# Discussion

The results of the present study could not confirm nor deny that post-activation potentiation was present during the fatigue protocols, which limited the ability to verify the interaction of post-activation potentiation and fatigue. The reason the presence of post-activation potentiation could not be confirmed or denied in all participants was because some participants presented with the effect and other did not (see Table 2). This was consistent with Comyns and colleagues (2007), who stated that individualized protocols may be important to stimulating post-activation potentiation. Post-activation potentiation may have been present in some participants and not in others. The small group of participants, possible individual variation, and participants' training status likely played a role in the lack of significant changes in the present study. However, the present study does show some promise.

Four of the individuals (2 male and 2 female) were able to increase accumulation of impulse on the experimental day while three decreased accumulated impulse (see Figure 3). The four participants that increased impulse on the experimental day had an average increase of 17.6±6.8 percent while the other participants had a decrease of 10.9±4.3 percent. The participants who increased in impulse were evenly separated in terms of the order of participating in the testing protocols. These four individuals also had an increase in ground reaction forces and flight time on the experimental testing days. Comyns and colleagues (2006) noted the possibility of individuals responding to postactivation potentiation at different time frames, which could explain the two divergent reactions to the conditioning contractions. However, to examine this effect, a larger sample size than was used in the current study would be needed. The present study's

participants were comprised of recreationally trained individuals and prior research has shown minimal, if no, positive results from this population (Chui, et al., 2003; Hamada, Sale, & MacDougall, 2000; Paasuke, et al., 2007).

Hamada and coworkers (2000) found that more experienced participants had greater levels of post-activation potentiation in the muscles they often train. However, Hamada and colleagues (2000) also found that even in recreationally active individuals, performance was enhanced, but to a lesser extent compared to athletes. The authors of the present study attempted to find recreationally active athletes that would have the highest opportunity to present with post-activation potentiation, but this could have been a limitation. A large standard deviation was present in the current study for both performance variables (flight time and impulse), which shows that there could be an effect, but it was overshadowed by the high degree of variability. The large standard deviation could be a product of individual variation that has been shown to be an issue in other post-activation potentiation studies (Comyns, Harrison Hennessy, & Jensen, 2006; Rixon, Lamont, & Bemben, 2007; Tillin & Bishop, 2009; Wilson, et al., 2013; Xenofondos, Laparidis, Galazoulas, Bassa, & Kotzamanidis, 2010).

Comyns and colleagues (2006) stated that in complex training (the use of a conditioning contraction added to a warm up), individual variation was an issue in weightlifting and plyometric trained individuals. The results of the present study echo this finding as some individuals responded to the conditioning contractions and some did not (see Figure 3). However, the present study did not have the sample size needed to assess for responders and non-responders.

Andrews and colleagues (2016) was the article most similar to the present study, but varied in certain methodological aspects. The authors of the present study set out to assess the effect of a conditioning contraction, which may elicit post-activation potentiation, on a fatigue protocol. Andrews and coworkers (2016), set out to assess the effect of a unilateral conditioning contraction on fatigue in the contralateral leg; and examine how fatigue interacts with post-activation potentiation. The latter was the purpose of the present study, as the interaction between post-activation potentiation and fatigue needs to be researched in more depth. Andrews and colleagues (2016) utilized unilateral jumps on the dominant or non-dominant leg on three occasions with a unilateral split squat measuring: the dominant (conditioned) leg, non-dominant (nonconditioned) leg, and non-dominant (non-conditioned) leg without any conditioning on the other leg (control). After the conditioning contraction, the participants performed drop and counter movement jumps on the leg being measured on each of the three days. Andrews et al. (2016) tested the aforementioned jumps at one, five and ten minutes after the conditioning contractions. The authors of the present study allowed 10 minutes rest after the conditioning contraction.

Andrews and colleagues (2016) showed a slight decrease in improvement in the conditioned leg and suggested that a ten minute rest may be too long, as shown by the peak in performance increase at five minutes. Andrews et al. (2016) did not assess this as a rest, but a peak in performance at 5 minutes could allude to the effect of post-activation potentiation peaking at five minutes instead of 10. The performance improvements were shown to peak at the five minute mark and return below the one minute improvement in the ten minute test. This could show a limitation in the rest interval utilized in the current

study (ten minutes) before the fatigue protocol. The limitation of the rest interval could be a reason that Andrews and colleagues (2016) were able to verify an interaction of post-activation potentiation and fatigue, whereas the current authors were unable to verify this interaction. However, improvements in performance may have peaked at 5 minutes, but the tests at these rest intervals were not separated. The tests being done one, five and ten minutes apart after the conditioning contraction shows that after a conditioning contraction and 4 jumps (2 drop and 2 countermovement jumps), post-activation potentiation will peak and improve jump performance within 4 minutes.

In the present study the only near significant effect was in the accumulated lactate between the experimental and control days. The near significant effect of the conditioning contraction on accumulated lactate shows that a conditioning contraction could be incorporated into a standard warm up, in an attempt to aid in clearance of lactic acid. The "mobilization" hypothesis states that lactate may be blunted after an active warm up, which may attenuate anaerobic energy production (Bishop, 2003a). However, Bishop (2003) stated that this attenuation may only last up to five minutes after a heavy warm up. The present study utilized a 10 minute break after three maximal contractions of the muscles used in jumping. If the muscle was allowed to return to anaerobic based energy production, the result of the present study would show no significant difference between experimental and control days. However, it could be argued that the opposite was found in the present study, which could mean that the effect of the conditioning contractions extended the attenuation of anaerobic energy production or aided in lactate clearance. For this reason, it could be beneficial to use a conditioning contraction during a warm up to decrease lactate accumulation.

Lactic acid is a marker of anaerobic metabolism (Bishop, 2003a), but it does not have a direct effect on fatigue. However, anaerobic metabolism produces lactic acid, which is lactate and a hydrogen ion. An accumulation of hydrogen ions can lead to more acidic blood, which can stimulate group III/IV afferent fibers and cause central or peripheral nervous fatigue (Amann, Sidhu, Weavil, Mangum, &Venturelli, 2015). In concert with the improved lactate clearance or "mobilization" phenomena, mean frequency muscle activity for the rectus femoris decreased at a more rapid rate, although not significantly on the experimental day. An improvement in mean frequency and lactate accumulation could imply that there were fewer metabolites present in the blood, which could lead to less negative feedback by group III/IV afferent fibers (Amann, Sidhu, Weavil, Mangum, &Venturelli, 2015). However, the present study was unable to support the changes in lactate along with changes in the rate of mean frequency decline. The present study shows evidence of another variable being present contributing to fatigue after a conditioning contraction. The paradox shown in the present study demonstrates that there may be an unknown variable effecting fatigue after conditioning contractions.

# Conclusion

The present study could not confirm nor deny that post-activation potentiation and fatigue interacted during a rebound jump, fatigue protocol in across participants. The results of the present study show that in some individuals there may be an improvement in accumulated impulse, ground reaction forces, flight time, EMG amplitude, accumulated lactate and maintained repetitions across testing days. The present study was limited by the small number of participants, possible individual variations, and the effect of the participants' past training experience. Future research should gather a larger

number of well-trained athletes, and allow for an assessment of individual variation. The results of the present study were that a conditioning contraction used to elicit postactivation potentiation could serve as a portion of a warm up in some individuals. However, the results could not show evidence of the interaction of post-activation potentiation and fatigue.



Figure 1: Depiction of the sledge used in the current study (used with permission Comyns et al., 2007). The depiction above displays a participant performing a single leg jump, whereas the present study utilized both legs while performing a rebound jump.

# Familiarization/baseline testing

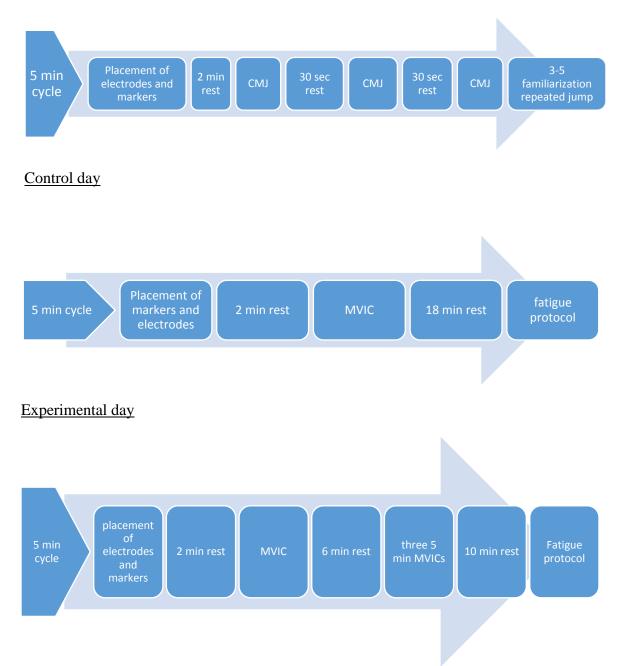


Figure 2: A representation of the protocol conducted on the testing and familiarization days in the current study. Lactate was measured 1 minute prior and 5 minutes post fatigue protocol on the control and experimental days.

# Table 1: Displayed are the means and standard deviations of all of the measuredvariables for Control and Experimental conditions; as well as the probability and

effect size of the comparisons between condi
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	Control		Experimental		Comparison	
	Mean	Standard Deviation	Mean	Standard Deviation	P-value	Partial eta square
Accumulation of Impulse (Ns)	44295.5	20508.3	45618.7	18492.4	0.722	0.023
Impulse Fatigue Index (Ns)	7947.7	4123.9	8222.1	4936.3	0.601	0.048
Peak Impulse (Ns)	687.3	119.0	760.6	128.1	0.104	0.379
Rate of Force Development (N/s)	5908.5	1244.7	5527.6	1413.1	0.292	0.182
Flight Time Fatigue Index (s)	0.308	0.084	0.320	0.112	0.710	0.025
Electromyography Amplitude Slope (Mv)	-0.072	0.527	0.058	0.148	0.536	0.067
Mean Frequency Slope (Mv)	-0.028	0.239	-0.148	0.311	0.294	0.180
Peak Ground Reaction Forces (N)	1310.5	315.0	1346.6	335.3	0.736	0.020
Flight Time (s)	0.809	0.105	0.780	0.099	0.112	0.366
Repetitions to Cutoff	68.7	26.8	64.0	22.7	0.229	0.230

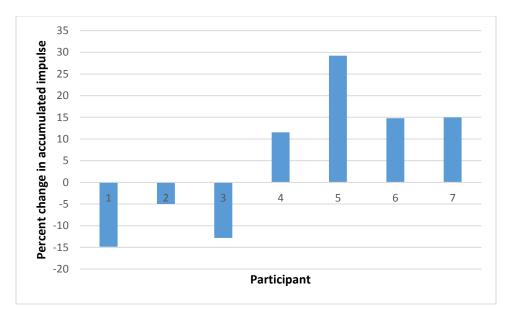


Figure 3: Displayed are the percent changes in accumulated impulse between the control and experimental days of all participants in the current study. The present figure also shows that 3 of the participants had decreases in accumulated impulse while 4 had increases.

Table 2: The mean difference (±SD) of Responders and Non-Responders between the experimental and control days for the variables studied. All data in the table below represent the difference in the means between the control and experimental testing days for the responders and non-responders in the current study. A negative means that there was a decrease in the variable from the control to the experimental testing days.

	Responder (n=4)	Non-Responders
		(n=3)
Flight Time	0.05±0.05	0.02±0.01
Accumulated Impulse	8980.31±6466.75	-2404.13±3903.65
Peak GRF	154.79±203.12	-52.86±245.22
RFD	-557.04±775.57	-159.97±783.39
EMG Amplitude slope	-0.101±0.29	0.184±0.63
Lactate	-2.3±0.2	-0.7±0.2
EMG Mean Frequency	0.26±0.01	0.26±0.30
slope		
Repetitions	-0.8±6.1	-10±8.6

#### **Chapter II: Literature Review**

As it pertains to post-activation potentiation, potentiation is a rise in the strength of the muscle or the synapse from a prior contractile history (i.e. a conditioning contraction). In post-activation potentiation research, the part of the contractile history that pertains to performance is termed a conditioning contraction, which is an activity used to stimulate the effects of post-activation potentiation (Tillin & Bishop, 2009). Potentiation of muscle can be modeled in two ways: on a continuous spectrum of potentiation; or on a spectrum from unpotentiation (or dispotentiation) to higher levels of potentiation (Brown & Loeb, 1998, Brown & Loeb, 1998). A synapse can go through a similar process as it can be depressed or potentiated (Junge, et al., 2004). Post-activation potentiation consists of both short-term potentiation of the neuron and potentiation of the muscle, as the proposed mechanisms affect the entire motor unit, leading to a rise in contractile strength (Brown & Loeb, 1998; Gullich & Schmidtbleicher, 1996; Hodgeson, Docherty, & Robbins, 2005; Tillin & Bishop, 2009; Wilson, et al., 2013; Xenofondos, Laparidis, Galazoulas, Bassa, & Kotzamanidis, 2010).

The four mechanisms of post-activation potentiation can be classified in two categories: muscular and neural. The muscular mechanisms consist of a change in pennation angle (Mahlfeld, Franke, & Awiszus, 2004) and phosphorylation of myosin regulatory light chains (Brown & Loeb, 1998). The neural changes that pertain to postactivation potentiation are as follows: increased recruitment of muscle fibers through increased firing rate of the nerve (Hodgeson, Docherty, & Robbins, 2005) and increased activity of higher order motor neurons (Gullich & Schmidtbleicher, 1996).

Mahlfeld et al. (2004) found that pennation angle changed 3-6 minutes after a maximal voluntary contraction but this change could only account for about one percent of the force production. However, Mahlfeld and colleagues (2004) stated that this change may be greater after the other effects of post-activation potentiation have dissipated. For this reason, pennation angle may not play a crucial role in augmenting force production until fatigue becomes a factor in performance.

Muscular potentiation has been mostly attributed to the phosphorylation of myosin regulatory light chains (Brown & Loeb, 1998). Phosphorylation of myosin regulatory light chains happens through a series of events starting with increased calcium  $(Ca^{2+})$  concentration in the myoplasm. The  $Ca^{2+}$  binds to calmodulin and the two bind to myosin light chain kinases, converting it to an active form. The activated kinase phosphorylates a specific serine residue in the amino-terminal of the regulatory myosin light chain causing an increase in myosin cross bridges in the force producing position. (Sweeney, Bowman, & Stull, 1993) Phosphorylation of myosin regulatory light chains may increase sensitivity to  $Ca^{2+}$  when levels are low in the myoplasm (Vandenboom, Grange, & Houston, 1993). This pertains to fatigue, as fatigue has been shown to decrease  $Ca^{2+}$  sensitivity (Debold, 2016).

Improvements in neural activity can also range from improved synchronization of motor units, decreased presynaptic inhibition, and increased central nervous input (Aagaard, 2003). It has been shown, in animal models, that electrical stimulation, twitch potentiation, can cause increased excitation potentials across synapses in the spinal cord (Gullich & Schmidtbleicher, 1996). This change in neural activity could be due to recruitment of higher order motor units (Gullich & Schmidtbleicher, 1996) and/or

decreased post synaptic failure, as transmittance failure is a regular occurrence (Tillin & Bishop, 2009).

Synaptic plasticity, such as potentiation and depression, can be both long- and short-term (Nadim & Manor, 2000). Nevertheless, post-activation potentiation most often pertains to short-term changes in strength, so for this literature review the focus will be on short-term synaptic plasticity. Short-term synaptic plasticity is a change in strength or excitability of the neuron for ten or less seconds (Nadim & Manor, 2000). These short-term neural changes come from repetitive synaptic activity within milliseconds, up to minutes (Junge, et al., 2004).

In the presynaptic terminal, Munc13-1 and upMunc13-2 bind to calmodulin in a  $Ca^{2+}$ -dependent manner to create short-term synaptic potentiation from the prior stimulus (Junge, et al., 2004). The reason Munc13s are important in synaptic strength is because they are important in exocytosis of neurotransmitters (Jahn & Fasshauer, 2012). Munc13s and Munc18s are integral parts of "zippering" Soluble N, Ethylmaleimide-Sensitive Fusion (NSF) Attachment Proteins (all together called SNAREs). An action potential propagating down the neuron changes the voltage of the membrane which opens voltage gated  $Ca^{2+}$  channels, causing an influx of  $Ca^{2+}$  (Jahn & Fasshauer, 2012). In rested levels of  $Ca^{2+}$  Munc13s are inactive, but in higher levels of  $Ca^{2+}$  the Munc13s become more active, increasing the neurons' strength (Junge, et al., 2004). This increase in activity can increase zippering of SNARE complexes and lead to increased exocytosis of neurotransmitters into the synaptic cleft (Jahn & Fasshauer, 2012; Junge, et al., 2004).

An increase in the neurotransmitter acetylcholine leads to an increase in binding to unbound receptors on the plasma membrane, resulting in depolarization of the cell

(Burden, Sargent, & McMahan, 1979). This depolarization leads to release of Ca<sup>2+</sup> from the sarcoplasmic reticulum, which binds to calmodulin (Endo, 1977), leading to phosphorylation of myosin regulatory light chains and potentiation of the muscle. This increase in phosphorylation of myosin regulatory light chains leads to a greater number of myosin cross-bridges in the force producing position (Brown & Loeb, 1998).

The increases in synaptic and muscular strength from a conditioning contraction are thought to be the reasons for improved performance in post-activation potentiation research. However, in post-activation potentiation research, fatigue is often the reason stated for a lack of improvement in performance (Hodgeson, Docherty, & Robbins, 2005; McCann & Flanagan, 2010; Morana & Perrey, 2009; Sale, 2002; Tillin & Bishop, 2009; Wilson, et al., 2013; Xenofondos, Laparidis, Galazoulas, Bassa, & Kotzamanidis, 2010). Rassier and MacIntosh (2000) and Wilson and colleagues (2013) state that the mechanisms of fatigue and post-activation potentiation may coexist but it is only presented in these review articles and has remained relatively untested. However, in a muscle in a twitch potentiation study, Alway and colleagues (1987) found that the mechanisms of post-activation potentiation and fatigue do not effect potentiation.

Despite the previous findings, there is minimal and/or inconsistent research on the interaction of post-activation potentiation and fatigue (Always, Hughson, Green, Patla, & Frank, 1987; Andrews, Horodyski, MacLeod, Whitten, & Behm, 2016; Fletcher & Jones, 2004). The most likely reason that fatigue is implicated in post-activation potentiation research is for a lack of performance improvement because the two seem to affect many of the same mechanisms that affect performance.

Fatigue is known to decrease force output, among other things (Decorte, Lafaix, Miller, Wuyam, & Verges, 2012), while post-activation potentiation has been shown to increase force output (Brown & Loeb, 1998; Gullich & Schmidtbleicher, 1996; Hodgeson, Docherty, & Robbins, 2005; Tillin & Bishop, 2009; Vandenboom, Grange, & Houston, 1993). Fatigue has also been shown to decrease sensitivity to  $Ca^{2+}$  (Debold, 2016); conversely, post-activation potentiation increases phosphorylation of myosin regulatory light chains that can increase Ca<sup>2+</sup> sensitivity in a normal Ca<sup>2+</sup> environment (Szczesna, Zhao, Jones, Zhi, Stull, & Potter, 2002). In a low Ca<sup>2+</sup> environment, phosphorylation of myosin regulatory light chains has been shown to increase Ca<sup>2+</sup> sensitivity (Vandenboom, Grange, & Houston, 1993). Fatigue decreases neural activation and drive to the muscle (Amann, Sidhu, Weavil, Mangum, &Venturelli, 2015), while post-activation potentiation increases activation and drive to the muscle producing greater levels of force (Brown & Loeb, 1998; Gullich & Schmidtbleicher, 1996; Hodgeson, Docherty, & Robbins, 2005; Tillin & Bishop, 2009; Vandenboom, Grange, & Houston, 1993). For the reasons stated above the interaction of fatigue and post-activation potentiation need to be examined, to provide evidence as to the effect of fatigue on postactivation potentiation and vice versa.

Fatigue is defined as a decreased ability of the muscle to produce force (Davis & Walsh, 2009; Decorte, Lafaix, Miller, Wuyam, & Verges, 2012). The effect of fatigue results in a loss of exercise capacity, increased perception of effort, and decreased power production (Davis & Walsh, 2009). Neural fatigue can be caused by central and peripheral mechanisms (Christian, Bishop, Billaut, & Girard, 2014); and the nervous system plays a role in sensing changes that cause fatigue (Amann, Sidhu, Weavil,

Mangum, &Venturelli, 2015). Central fatigue is a direct effect of decreased performance of the cerebral cortex, spinal cord, or a lack of motivation (Davis & Walsh, 2009). Cerebral fatigue (a part of central fatigue) may come from reduced drive of the descending tract of the spinal cord or decreased motivation (Taylor, Todd, & Gandevia, 2005). When the spinal cord becomes fatigued there is decreased alpha motor neuron excitation and recruitment rate (Taylor, Todd, & Gandevia, 2005). The decreased excitation and recruitment will decrease generation of force from the muscle. Measurement with electromyography (EMG) amplitude to task failure (EMG amplitude increases as fatigue degrades performance); will display a decrease in excitation and recruitment of these neurons (Taylor, Todd, & Gandevia, 2005).

Central and peripheral fatigue can be affected by group III and IV afferent fibers that sense blood flow, oxygen transport, and metabolite concentration (i.e. lactic acid) (Amann, Sidhu, Weavil, Mangum, &Venturelli, 2015). These group III/IV fibers feedback on the central and peripheral nervous systems to decrease drive and activation of muscles (Amann, Sidhu, Weavil, Mangum, &Venturelli, 2015). This feedback shows a strong interaction of the central and peripheral nervous system and the muscle's activity that may lead to fatigue. As post-activation potentiation also effects the central and peripheral nervous systems, there is evidence that a conditioning contraction may stimulate group III/IV fibers to feedback on and cause a synaptic depression of peripheral and central motor neurons (Amann, Sidhu, Weavil, Mangum, &Venturelli, 2015)(Andrews, Horodyski, MacLeod, Whitten& Behm, 2016).

A warm up activity is one of the few activities that acutely affects fatigue (Bishop, 2003a), although research is limited on how this occurs with post-activation potentiation. Impairments in excitation-contraction coupling from low-frequency fatigue may be overcome when using post-activation potentiation as part of the warm up (Rassier & MacIntosh, 2000). This shows that the fatigue can be overcome by a prior contractile stimulus that causes post-activation potentiation, during low frequency fatiguing activities. Agreeing with Rassier and MacIntosh (2000), Andrews et al. (2016) found that, in unilateral jumping, post-activation potentiation may overcome the effects of fatigue in a leg that was stimulated by a conditioning contraction.

Andrews and colleagues (2016) assessed the interaction of post-activation potentiation and fatigue and the effect of a conditioning contraction on a non-conditioned leg. The structure of the Andrews et al. (2016) study utilized a Bulgarian split squat to simulate the effect of post-activation potentiation on single leg countermovement and drop jumps. After the jumps they tested the conditioned and non-conditioned legs on three separate occasions. The conditioned leg was only tested on one day, which was meant to be the post-activation potentiation test day. The non-conditioned leg was tested on two occasions: a post-activation potentiation test day and a control day. Single leg countermovement and drop jumps were assessed on 3 occasions on each testing day: one minute, five minutes and ten minutes after the conditioning contractions. Andrews and coworkers (2016) found that on the day that the conditioned leg was tested jump performance improved to a greater extent than when the same protocol was performed, but the non-conditioned leg was tested. The conditioned leg decreased performance less when tested (from fatigue), which shows that the effect of post-activation potentiation may overcome the effect of fatigue. The issue is that there was no assessment of the

interaction of the mechanisms of these two phenomena. For this reason future research needs to assess performance changes and mechanistic changes from the interaction.

A warm up has been shown to play a role in increasing neural activity (Bishop, 2003a), which is likely due to a change in potentiation (Junge, et al., 2004). Brown and Loeb (1998) had previously shown that, as unpotentiation can be reached through as little as 10 minutes rest, muscle can become potentiated by a few cycles of locomotion. Post-activation potentiation and a warm up have both been shown to improve jumping and sprinting performance (Fletcher & Jones, 2004). This evidence adds to the idea that a conditioning contraction may be used as a warm up protocol.

Future research will need to verify the interaction of post-activation potentiation and fatigue through the mechanisms that they have in common and through their effect on performance. Research in this area should assess changes in phosphorylation of myosin regulatory light chains, peripheral and central neural activity, and pennation angle during fatiguing activities. Mechanistic research should investigate decrements in performance (force production, rate of force development, etc....) to examine relationships with the changes in physiological mechanisms during performance. This research could lead to a better understanding of the effect of prior contractile history on muscular and neural performance in sports and exercise.

### **Chapter III: Conclusions and Recommendations**

Post-activation potentiation and fatigue are thought to interact through their common mechanisms, but this has not been thoroughly tested. Andrews and colleagues (2016) found that a conditioning contraction that stimulates post-activation potentiation can overcome the effect of fatigue. However, the effects of fatigue have been shown to decrease post-activation potentiation related effects or have been shown to not interact with post-activation potentiation. This leads to the question, do post-activation potentiation and fatigue interact?

The authors of the present study attempted to assess this interaction through a protocol that could stimulate the effects of post-activation potentiation and then to stimulate fatigue. The present study consisted of familiarization/baseline testing and experimental and control testing days. On the experimental day the participants performed conditioning contractions and on the control day the participants rested. The participants then performed a fatiguing rebound jump protocol and were assessed for performance during the fatigue protocols.

The present study was not able to fully verify the existence of an interaction of post-activation potentiation and fatigue. The most likely reasons for this lack of evidence are due to individual variation, the small number of participants, a need for a better maximal performance assessment at the beginning of the fatigue protocol (such as a maximal countermovement jump), and a population that may not have been suited for this type of activity.

Future research should make use of a larger sample size, assess for variability between responders and non-responders, and have a better maximal performance measure

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(such as a maximal countermovement jump) at the beginning of the fatigue protocol. The present study had limitations, but did show promise in some areas, shown in the tendency to have: improved lactate clearance or the "mobilization" phenomena, increased accumulated impulse and a less rapid decrease in mean frequency.

Future studies on the interaction of post-activation potentiation and fatigue should increase sample size to allow for the assessment of responders and non-responders to a conditioning contraction. These studies should allow for individualized rest intervals to account for the variability in the length of time needed to show potentiation, as found by Comyns et al. (2006). Future researchers should also incorporate a maximal performance test, such as a counter movement jump at the beginning of the fatigue protocol, to allow for an assessment of improvements in maximal performance from post-activation potentiation.

The above recommendations will allow for an assessment of the interaction of fatigue and post-activation potentiation, in respect to performance, but the mechanisms also need to be assessed. If possible it would be beneficial to measure phosphorylation of myosin regulatory light chains (muscle biopsy) before and after the conditioning contraction, and after the fatigue protocol. Mechanistic research would benefit from measurement of peripheral and central neural fatigue through the entire protocol. Central and peripheral neural fatigue can be measured with EMG and transcranial magnetic stimulation. Pennation angle could be assessed with functional magnetic resonance imaging; however, this would need to be done during the fatigue protocol, which would prove to be difficult, if not impossible with the current methodology.

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# **Appendix A: Informed Consent**

# NORTHERN MICHIGAN UNIVERSITY DEPARTMENT OF HEALTH AND HUMAN PERFORMANCE

# CONSENT TO ACT AS A HUMAN SUBJECT

Subject Name (print):\_\_\_\_\_ Date \_\_\_\_\_

1. I hereby volunteer to participate as a subject in exercise testing. I understand that this testing is part of a study entitled: "Post-Activation Potentiation Decay or Fatigue Delay". The purpose of the present study is to assess the effect of post-activation potentiation on the onset of fatigue and degradation of jumping performance.

I hereby authorize Ryan L. Meidinger, Randall L. Jensen, Sarah Clarke, Lanae Joubert and/or assistants as may be selected by them to perform on me the following procedures:

(a) I understand that I will perform 2 fatiguing rebound jump protocols on a sledge, after doing 4 maximal voluntary isometric (held position) quarter squat contraction in the experimental trial and one maximal voluntary isometric quarter squat contraction in the control trial. I will also not intake caffeine the day that or alcohol 48 hours prior to trial testing and I will eat a full meal and hydrate 2 to 4 hours prior to testing. The following is a depiction of the sledge I will be performing the fatigue protocol on.



(b) I understand that I will have one electrode attached to my right quadriceps (rectus femoris) halfway between the iliac crest and the superior portion of the patella. The electrode will be used to assess muscle activity, via electromyography, while performing the fatigue protocol.

2. The procedures outlined in paragraph 1 [above] have been explained to me.

I understand that the procedures described in paragraph 1 (above) involve the following risks and discomforts: musculoskeletal injuries including but not limited to; muscle strains, ligament sprains, joint dislocations, and abrasions. There may be minor skin irritation and redness from the placement and skin preparation for the electromyography electrodes and from removal. In order to

prevent any of the above-mentioned risks, I understand that the examiners shall adopt the necessary measures to prevent them such as: having strict inclusion criteria and monitoring of fatigue during the fatigue protocol. However, I understand that I can terminate any testing at any time at my discretion. I should stop any test if I experience any abnormalities such as dizziness, light-headedness, or pain, etc.

- 3. I understand that I may gain important information about my jumping performance and a profile of my fatigue when performing rebound jumps. I may also benefit from this study by learning if I will have an effect from post-activation potentiation and how it affects my fatigue.
- 4. I understand that Ryan L Meidinger, Randall L. Jensen, Sarah Clarke, Lanae Joubert and/or appropriate assistants, as may be selected by them, will answer any inquiries that I may have at any time concerning these procedures and/or investigations.
- 5. I understand that all data, concerning myself will be kept confidential and available only upon my written request. I further understand that in the event of publication, no association will be made between the reported data and myself.
- 6. I understand that there is no financial compensation for my participation in this study.
- 7. I understand that in the event of physical injury directly resulting from participation, compensation cannot be provided. However if injury occurs, emergency first aid will be provided and the EMS system activated.
- 8. I understand that I may terminate participation in this study at any time without prejudice to future care or any possible reimbursement of expenses, compensation, or employment status.
- 9. I understand that if I have any further questions regarding my rights as a participant in a research project I may contact Robert L. Winn (906-227-2700)<u>rwinn@nmu.edu</u>, Assistant Provost of Graduate Education/Research of Northern Michigan University Any questions I have regarding the nature of this research project will be answered by Dr. Randall Jensen (906-227-1184)<u>rajensen@nmu.edu</u> or Ryan L. Meidinger <u>rmeiding@nmu.edu</u>.

Subject's Signature:	

Witness: \_\_\_\_\_ Date: \_\_\_\_\_

# **Appendix B: PAR-Q**

Physical Activity Readiness Questionnaire - PAR-Q (revised 2002)

# PAR-Q & YOU

#### (A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	1.	Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor?				
		2.	Do you feel pain in your chest when you do physical activity?				
		3.	In the past month, have you had chest pain when you were not doing physical activity?				
		4.	Do you lose your balance because of dizziness or do you ever lose consciousness?				
		5.	Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?				
		6.	ls your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart con- dition?				
		7.	Do you know of <u>any other reason</u> why you should not do physical activity?				
lf you answe	ered		<ul> <li>YES to one or more questions</li> <li>Talk with your doctor by phone or in person BEFORE you start becoming your doctor about the PAR-Q and which questions you answered YES.</li> <li>You may be able to do any activity you want — as long as you start sl those which are safe for you. Talk with your doctor about the kinds of a</li> <li>Find out which community programs are safe and helpful for you.</li> </ul>	owly and build up gradually. Or, you may need to restrict your activities to			
If you ans start b safest : take pa that yo have yo	wered NG ecoming i and easie art in a fit u can pla our blood	) hone much i st way ness a n the l press	Uestions estly to <u>all</u> PAR-Q questions, you can be reasonably sure that you can: more physically active — begin slowly and build up gradually. This is the to go. appraisal — this is an excellent way to determine your basic fitness so best way for you to live actively. It is also highly recommended that you ure evaluated. If your reading is over 144/94, talk with your doctor ming much more physically active.	<ul> <li>DELAY BECOMING MUCH MORE ACTIVE:         <ul> <li>if you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or</li> <li>if you are or may be pregnant – talk to your doctor before you start becoming more active.</li> </ul> </li> <li>PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.</li> </ul>			
			he Canadian Society for Exercise Physiology, Health Canada, and their agents assume Ir doctor prior to physical activity.	no liability for persons who undertake physical activity, and if in doubt after completin			

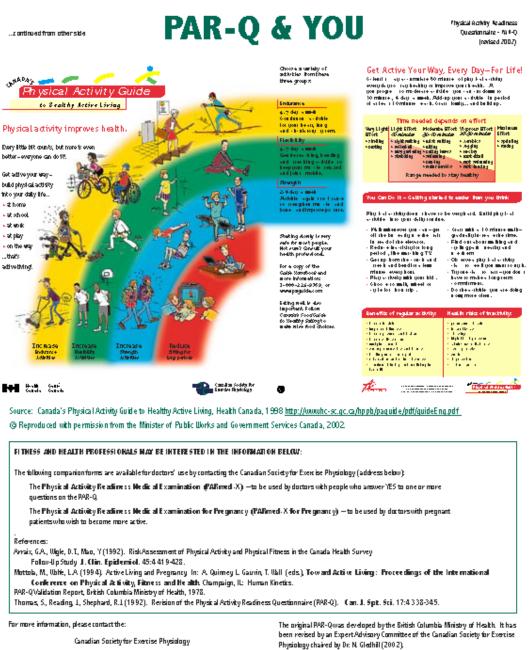
No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME								
SIGNATURE						DATE		
		r the age of majority)				WITNESS		
		This physical activity clea comes invalid if your condi						
	© Canadian	Society for Exercise Physiology	Supported by:	Н	lealth Canada	Santé Canada	continued on oth	her side

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Canadian Society for Exercise Physiology 202-185 Somerset Steet West Ottawa, ON K2P 0.2 Tel. 1-877-651-3755 • FAX (613) 234-3565 Online: www.csep.ca

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Supported by Health Santé Canada Canada

-Q-AAP (revisé 2002)».

Disponible enfrançais sous le titre «Questionnaire sur l'aptitude à l'activité physique

## **APENDIX C: IRB APPROVAL**



#### NORTHERN MICHIGAN UNIVERSITY Memorandum

OFFICE OF GRADUATE EDUCATION AND RESEARCH 1401 Presque Isle Avenue Marquette, MI 49855-5301 906-227-2300 | Fax: 906-227-2315 www.nmu.edu/graduatestudies

TO:	Ryan Meidinger School of Health and Human Performance
CC:	Randall Jensen School of Health and Human Performance
DATE:	March 31, 2017 Robert Winn, Ph.D.
FROM:	Robert Winn, Ph.D. Interim Dean of Arts and Sciences/IRB Administrator
SUBJECT:	IRB Proposal HS17-843 IRB Approval Dates: 3/31/2017 - 3/31/2018 Proposed Project Dates: 3/28/2017 - 5/5/2017 "Post-Activation Potentiation Decay or Fatigue Delay"

The Institutional Review Board (IRB) has reviewed your proposal and has given it final approval. To maintain permission from the Federal government to use human subjects in research, certain reporting processes are required.

- A. You must include the statement "Approved by IRB: Project # HS17-843" on all research materials you distribute, as well as on any correspondence concerning this project.
- B. If a subject suffers an injury during research, or if there is an incident of non-compliance with IRB policies and procedures, you must take immediate action to assist the subject and notify the IRB chair (dereande@nmu.edu) and NMU's IRB administrator (rwinn@nmu.edu) within 48 hours. Additionally, you must complete an Unanticipated Problem or Adverse Event Form for Research Involving Human Subjects
- C. Please remember that informed consent is a process beginning with a description of the project and insurance of participant understanding. Informed consent must continue throughout the project via a dialogue between the researcher and research participant.
- D. If you find that modifications of methods or procedures are necessary, you must submit a Project Modification Form for Research Involving Human Subjects before collecting data.
- E. If you complete your project within 12 months from the date of your approval notification, you must submit a Project Completion Form for Research Involving Human Subjects. If you do not complete your project within 12 months from the date of your approval notification, you must submit a Project Renewal Form for Research Involving Human Subjects. You may apply for a one-year project renewal up to four times.

NOTE: Failure to submit a Project Completion Form or Project Renewal Form within 12 months from the date of your approval notification will result in a suspension of Human Subjects Research privileges for all investigators listed on the application until the form is submitted and approved.

All forms can be found at the NMU Grants and Research website: http://www.nmu.edu/grantsandresearch/node/102