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The Effects of Plyometric Training on Spinal and Supraspinal Motor Control

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Abstract

Purpose: The purpose of this study was to determine the effects of plyometric training on spinal and supraspinal motor control in healthy active females. *Methods:* A 2 (Group) x 2 (Session) x 2 (Stance) mixed model design was used for homosynaptic depression (HD) and recurrent inhibition (RI) data. A 2 (Group) x 2 (Session) mixed model design was used for H:M ratios and V-waves (V:M ratios) data. Thirty-one participants were recruited to participate in the study. Participants participated in either the training or control group for 6-weeks. All participants were measured pre- and post-intervention on H:M ratios, HD, RI, and V-waves. **Results:** There were no statistically significant interactions for any of the dependent variables ($p > 0.05$). There was a session main effect for RI ($p = 0.01$) as well as a group main effect ($p = 0.01$) and a stance main effect ($p < 0.01$) for HD. **Conclusions:** Performing plyometric training does not modulate the spinal or supraspinal motor control based on the findings of this study. Plyometric training is not the only component that should be used in Anterior Cruciate Ligament Injury Prevention Programs to alter motor control. A more challenging and higher impact plyometric exercises are suggested to allow more demand on the neural drive of the physically active participants.

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The Effects of Plyometric Training on Spinal and Supraspinal Motor Control in Anterior Cruciate Ligament Injury Prevention

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ABSTRACT

Purpose*:** The purpose of this study was to determine the effects of plyometric training on spinal and supraspinal motor control in healthy active females. ***Methods: Thirty-one participants were recruited to participate in the study. Participants participated in either the training or control group for 6-weeks. All participants were measured pre- and post-intervention on H:M ratios, HD, RI, and V-waves. A 2 (Group) x 2 (Session) x 2 (Stance) mixed model design was used for homosynaptic depression (HD) and recurrent inhibition (RI) data. A 2 (Group) x 2 (Session) mixed model design was used for H:M ratios and V-waves (V:M ratios) data. ***Results:*** There were no statistically significant interactions for any of the dependent variables ($p > 0.05$). There was a session main effect for RI ($p = 0.01$) as well as a group main effect ($p = 0.01$) and a stance main effect ($p < 0.01$) for HD. ***Conclusions:*** Performing plyometric training does not modulate the spinal or supraspinal motor control based on the findings of this study. Plyometric training is not the only component that should be used in anterior cruciate ligament injury prevention programs to alter motor control. Further investigation warrants research in a more challenging and higher impact plyometric exercises on the changes to neural drive of the physically active participants.

INTRODUCTION

Sport teams commonly use anterior cruciate ligament (ACL) injury prevention programs to reduce injury risk for non-contact ACL injuries and injury risk for noncontact ACL injuries.[1-6] Traditionally, ACL injury prevention programs have included multiple components: warm-up, stretching, strengthening, plyometrics, agility, and balance activities.[7] ACL prevention programs have multiple components used simultaneously making it difficult to determine the extent to which any one component contributes to the overall effectiveness of the program.

To gain a better understanding of the effectiveness of individual components in ACL prevention programs, two previous studies focused on adaptations of two components from a multiple component program.[8,9] One study by Myer et al compared plyometrics to a dynamic balance stabilization component and observed a reduction in ACL risk factors during a drop landing task in both groups.[8] In another study, Lephart et al compared plyometrics to a basic resistance-training program and tested participants during a jump landing task.[9] The results showed increased electromyography (EMG) activity, isokinetic strength, and a reduced risk of dangerous lower extremity joint motion following both training interventions.[9] The results from both studies supported the use of plyometric training to reduce potentially dangerous motions.[8,9] In addition to these two laboratory-based studies, plyometric training has also been identified as a primary contributing factor in programs that reduced the risk of ACL injuries in two separate meta-analyses.[7,10] The investigators who conducted both meta-analyses concluded that prevention programs including a plyometric component positively affected the reduction of injuries.[7,10] These results support the use of plyometrics in ACL prevention programs.[4,7,10]

Plyometric training includes exercises that produce rapid and powerful movements resulting in an increase in neural activation.[11-13] Specifically in muscle groups of the lower extremity, plyometric training can increase muscle activation.[14-16] The neural mechanism responsible for the apparent benefit of plyometric training remains unknown. Spinal and supraspinal motor control measurements can be used to assess the connectivity and modulation of the sensory and motor systems which affect the involuntary muscle activations.[17]

The purpose of this study was to determine the effects of plyometric training on neuromuscular control in healthy active females. The plyometric component from an ACL prevention program was investigated using variables of spinal motor control via the ratio of H reflex to motor response (H:M ratios), homosynaptic depression (HD), recurrent inhibition (RI), and supraspinal motor control the ratio of volitional waves (V-waves) to motor response (V:M ratios).

METHODS

In order to determine changes to neuromuscular control, spinal and supraspinal motor control were measured 6 weeks apart in a plyometric training group and a control group. Thirty-one healthy college-aged females (16 plyometric training group and 15 control group) were recruited from Oregon State University. To participate in the study, participants were required to be physically active 3 times a week for approximately 30 minutes per day. A list of the exclusion criteria is presented in Table 1. All participants provided informed consent approved by an Oregon State University institutional review board for the protection of human participants that is regulated by US government standards on ethics approval.

Table 1.

Exclusion Criteria
Known neurological disorder
Injury to the lower extremity in the previous 6 months
Completed a season of basketball or volleyball within the last 12 months
Previous involvement in an ACL prevention program
Previous involvement in a 4-week or longer plyometric training program
Plan to change their personal workout during the course of the study

Participants were randomized into either a plyometric training group (n = 16) or a control group (n = 15) by blindly picking a group assignment out of a bag. Table 2 provides participant characteristics. All participants were tested before and after the 6-week intervention period. The dependent variables included measurements of spinal and supraspinal motor control. The same EMG and stimulation procedures were used for all spinal and supraspinal motor control variables in both data collection sessions. All participants were tested on their dominant leg as determined by their preferred kicking leg.

Table 2. Means and standard deviations for participant's age, height, and mass for each group

	Plyometric	Control
Age (yrs)	**22.5±3.2**	**22.7±2.3**
Height (cm)	**167.8±7.6**	**166.2±6.4**
Mass (kg)	**64.5±7.4**	**65.4±6.3**

Abbreviations: cm = centimeters; kg = kilograms; yrs = years

All EMG was recorded at 2000Hz using disposable, lubricated bipolar Ag/AgCl electrodes. Two recording electrodes were placed on the soleus, directly over the muscle belly between gastrocnemius and Achilles tendon. A reference electrode was placed on the lateral malleolus. Peak-to-peak waveform amplitudes were measured using AcqKnowledge software (v. 3.9.0; BIOPAC Systems, Inc., Goleta, CA).

A Grass Model S88 stimulator (Grass Instruments, Inc., Warwick, RI) was used to initiate the evoked potentials. For participant safety, a stimulus isolation unit and constant current unit (Grass Instruments, Inc., Warwick, RI) were connected between the stimulator and the participant. In the stimulating circuit, a $1cm^2$ stimulating electrode (12 mm unshielded electrode, BIOPAC Systems, Inc., Goleta, CA) was placed in the popliteal space of the knee over the tibial nerve, and a $3cm^2$ dispersal pad was positioned on the anterior portion of the knee, superior to the patella.

The spinal control measurements included H:M ratios, HD, and RI. For the HD and RI measurements, participants were tested in a double-legged stance followed by a single-legged stance. The two stances were observed to see if the change of balance support affected an individual's spinal control modulation. For the H:M ratios, participants were tested only in double-legged stance.

In the process of collecting the H:M ratio, a complete Hoffmann reflex (H-reflex) recruitment curve was collected for each participant.[18] The H-reflex measurement was recorded by increasing the stimulus intensity in small increments from motor threshold to maximal muscle response (Mmax). The maximum sensory fiber reaction to an electric stimulus (Hmax) and Mmax values were used to form a H:M ratio. After the H:M ratio was recorded, the testing stimulus intensity was set to a level that elicited a H-reflex amplitude at approximately 10% of Mmax while in a double-legged stance. The stimulus intensity remained constant during the HD and RI measurements for each trial during both double-legged and single-legged stances.

Homosynaptic depression was measured using a pair of stimuli set to an intensity that produced an H reflex amplitude at approximately 10% of Mmax. Eight paired reflexes were collected with 100ms separating the stimuli in each pair. The depression was measured for each pair of reflexes and then averaged. The averaged depressions were calculated by dividing the amplitude of the second H-reflex, the conditioned reflex, by the amplitude of the first unconditioned H-reflex amplitude. The percentage of HD was represented by [(1- (amount of depression)) X 100].

Recurrent inhibition was assessed using an unconditioned stimuli at an intensity of 10% of Mmax followed by a conditioning stimuli (supramaximal) applied for 10ms.[19] Eight conditioned reflexes and eight unconditioned reflexes were collected. The conditioned and the unconditioned reflexes were alternated every two trials during a measurement set. The reflexes were found for each stimulus then were averaged as set. The amount of RI was calculated by dividing the average of the conditioned reflex trials by the average of the unconditioned reflex trials. The percentage of RI was represented by [(1- (amount of inhibition)) X 100].

Supraspinal motor control was assessed through the use of V-waves. V-waves are an electrophysiological variant of the H-reflex that reflects the magnitude of alpha motor neuron output during voluntary muscle contraction.[20,21] V-wave measurements were performed by delivery of a supramaximal stimulus to the tibial nerve while the participant performed a maximal plantar flexion isometric contraction. To perform the isometric contractions, participants were seated in the Biodex System 3 (Biodex Systems, Inc.; Shirley, NY) in a semi-reclined position. The foot of the participant's dominant leg was placed in the footplate and secured with straps. The knee was positioned at 60° of knee flexion and the ankle was positioned at 0° plantar flexion. The plantar flexion muscle group was tested because we were most interested in the neural pathway modulation, and this muscle group was used through the entire training protocol.

Before recording V-wave measurements, three plantar flexion maximal voluntary isometric contractions (MVIC) were collected. A light stimulus was positioned in front of the participant to notify them when to begin the maximal contraction. The digital trigger threshold for the supramaximal stimulus was set at 90% of the average MVIC across the three trials. When participants reached 90% of MVIC, a supramaximal stimulus was delivered to the tested leg. Three trials were collected with one-minute rest between each trial. The peak-to-peak amplitude of Mmax and V-wave were recorded for each trial. The V-wave was normalized to Mmax to form the V:M ratio. The average of the three V:M ratios were used for the analysis.

Following baseline measurements, the training group participated in a 6-week plyometric training program and the control group was asked not to change their daily physical activities for the duration of the study. The plyometric training program chosen aligns with the plyometric section of the program described by Hewett et al.[1] Some of the exercises in the plyometric program section included wall jumps, tuck jumps, broad jumps stick landing, squat jumps, double leg cone jumps, 180° jumps, bounding in place, scissor jumps, mattress jumps, and hop to stick landing.[1] All of the exercises were less than 30 seconds in duration. The exercises were designed to be lower impact and work predominantly on form while going through tasks that required a subjects to leave one's feet.[1] Participants in the training group performed the exercises three times a week on alternating days, for 30-minutes sessions. If a participant missed more than four training session (less than 78% attendance), they were excluded from the post-intervention testing session. In the current study, the participants reported to the Neuromechanics Research Laboratory to perform the training session under the direct supervision of a research study team member. The investigators educated participants and provided feedback on exercises including: 1) correct posture and body alignment; 2) jumping straight up with no excessive movement; 3) soft landings with bent knees; and 4) instant reloading for preparation of the next jump. All exercises were demonstrated and the participants were thoroughly instructed on proper techniques.

For HD and RI, a 2 (group) x 2 (session) x 2 (stance) mixed model ANOVA was applied to the data. A 2 (group) x 2 (session) mixed model ANOVA was applied because the H:M ratios were only collected in a double-legged stance and the V:M ratios were collected on a Biodex chair. An alpha level of $p < 0.05$ was used for all analyses. All data were explored for extreme outliers that

were greater than three standard deviations from the mean. All statistical analyses were performed using SPSS software, version 19 (SPSS, Inc. Chicago, IL).

RESULTS

The plyometric training participants attended 92% of the training sessions and no participants missed more than four training sessions. All means and standard deviations of the dependent variables are presented in Table 3. Exploration of the data revealed two participants were extreme outliers from HD analysis, one participant from RI analyses, and six participants from the V:M ratios analyses because of data collection error.

A 2 x 2 x 2 mixed model ANOVA for HD revealed a group main effect (Plyometric: 70.12±27.42; Control: 43.40±37.37; $p < 0.01$) and stance main effect (Double-legged: 76.24±21.47; Single-legged: 39.19±48.46; $p < 0.001$). There was a session main effect for RI (Pre: 52.37±36.43; Post: 72.91±19.19; $p < 0.01$). There was no significant difference for any other variable ($p > 0.05$).

Table 3. Dependent variables H:M ratios, HD DL, HD SL, RI DL, RI SL, and V:M ratios means and standard deviations are presented below.

	Plyometric		Control	
Dependent Variables	**PRE**	**POST**	**PRE**	**POST**
H:M ratios	**0.62±0.19**	**0.58±0.19**	**0.58±0.21**	**0.64±0.16**
HD DL	**78.99±13.24**	**86.42±19.43**	**73.55±21.11**	**64.01±27.49**
HD SL	**50.93±42.64**	**64.16±34.38**	**21.78±43.18**	**14.25±57.69**
RI DL	**50.05±28.76**	**69.83±21.46**	**55.57±35.78**	**67.31±19.88**
RI SL	**54.91±35.41**	**79.77±14.04**	**48.93±45.75**	**74.73±21.37**
V:M ratios	**0.24±0.16**	**0.25±0.20**	**0.23±0.17**	**0.19±0.18**

Abbreviations: DL, Double-legged stance; H:M ratios, the ratio between maximal Hoffmann reflex and the maximal Muscle response; HD, Homosynaptic depression; Post, Post intervention; Pre, Pre intervention; RI, Recurrent inhibition;; SL, Single-legged stance; V:M ratios, the ratio between V-wave and maximal Muscle response. There was no significance in data ($p > 0.05$).

DISCUSSION

Plyometrics are a primary component in ACL injury prevention programs.[7,10] When athletes perform these programs, feedback is given to make sure the exercises are being performed properly. There is a general understanding that plyometrics increase neural drive to improve speed and strength for sport specific movements.[11-13] However, the mechanism responsible for the neural changes had not previously been investigated. The objective of this study was to determine the effects of plyometric training on spinal and supraspinal motor control in healthy active females.

Theoretically, inhibition of a neuron occurs in the monosynaptic reflex loop when a previous stimulus has already met the axon's threshold.[22, 23] HD is a measurement of inhibition in a reflex loop that occurs because of the neuron being recently activated by a previous stimulus in the presynaptic axon.[22] No significant interactions were observed in the HD analysis; however, we did observe significant group and stance main effects. The expectation in our study was that the plyometric training group would show similar results to the power-trained athletes in the study by Earles et al.[24] The power trained athletes in that study had a lower HD when compared to the non-plyometric control group. Decreased HD modulation suggests greater action potentials reached the targeted muscle when performing high-force movements.[24] Because plyometric exercises use the stretch shortening cycle to achieve explosive movements, HD should theoretically result in a decreased reflex modulation.[24-26] In our study, the group main effect was unexpected and the plyometric training group had means that were higher both pre- and post-intervention compared to the control group. There were no training effects on HD modulation.

Based on a previous study by Sefton et al, H-reflex measurements should display greater inhibition in the single-legged stance or during locomotion compared to the double-legged stance.[27] In our findings, we observed decreased inhibition of HD during the single-legged stance for both the plyometric training and control groups. Sefton et al also reported decreased HD in single-legged stance in their healthy participants. In that study, the researchers compared a chronic ankle instability group to a healthy control group on segmental spinal reflex. HD was measured to observe if there were modulation differences when going from a double-legged to a single-legged stance between groups. The healthy participants had approximately a 15% decrease in HD going from double-legged to single-legged stances. The chronic ankle instability group was unable to modulate the same way as the healthy group. This finding suggests that healthy participants were able to modulate to an unstable surface better than participants with chronic ankle instability.[27] The effort to maintain single-legged stance causes multiple action potentials to be sent through the reflex

loop, which increases inhibition in the spinal cord.[27] Plyometric training did not cause a significantly lower HD when the postural demand increased.

Recurrent inhibition (RI) is a gain regulator in the postsynaptic axon that shows the amount motor output in the monosynaptic reflex loop.[28] Participants who performed plyometric training were expected to have greater RI because plyometric exercises impacts the activation of the entire motor neuron pool during high explosive activities. In Earles et al, power-trained athletes had higher RI compared to endurance-trained athletes and non-trained participants.[24] Plyometric training consistently causes overload to the motor neurons with action potentials that activate the recurrent collaterals in order to modulate the postsynaptic axon.[24] Plyometric training was expected to cause an adaptation to the neuron threshold after several weeks.[14]

In addition, a decreased RI was expected in the single-legged stance following plyometric training because of the requirement to increase the stability in the lower extremity during training. In this study, both groups increased RI during the post-intervention testing session and there was no significant difference between groups or stances. This suggests that both groups had increased gain regulation after the six weeks of exercise or training.

The V-wave was used as a measurement for the change of magnitude of alpha motor neuron output during voluntary muscle contraction.[20] We did not observe any significant interactions or main effects. The effects of resistance training on V:M ratios have been previously reported.[20,29,30] The increases in V:M ratios were observed following a 14-week lower extremity resistance strength training program, a 5-week plantar flexion resistance training program, and a 4-week plantar flexion isometric contraction training program.[20,29,30] Following training, there was greater descending neural drive that occurred in the efferent neurons that cleared the pathway for action potentials to travel through the reflex loop. This occurred as a result of enhanced neural drive from the descending pathway, which was caused by training effects. The increased descending control stems from adaptation to resistance training.[20] There were no training effects or adaptation that occurred to neural descending control.

Resistance training induced an increase in V:M ratios.[20,29,30] Plyometric training, a higher-level exercise intensity training, did not show the same impact as resistance training did on V:M ratios. The same increase in V:M ratios was expected following plyometric training, but was not observed in our study. The plyometric training in this study might not have been at a high enough level of intensity relative to the participants' workouts prior to the study. This program includes body weight exercises without external resistance. The plyometric training component is a part of an ACL injury prevention program, which is usually used to focus on correcting proper techniques during functional movements.

Limitations

One limitation in this study was not monitoring what physical activities the control group was performing during the intervention. We relied on the subjects' self-reporting of compliance with the study's exclusion criteria. Another possible limitation in this study was the variability of the H-reflex measurements when assessed in different stances. H-reflex measurements were closely monitor to reduce as much variability as possible. In addition, a healthy control group was utilized during this experiment; there might be a different response in an ACL post-surgical population. A final possible limitation of this study was that the spinal and supraspinal variables used may not truly measure the changes that may occur from plyometric exercises.

CONCLUSION

The results of our study indicated that there were no significant neuromuscular control differences following plyometric training in healthy active females. In addition, these results suggest there were no training effects on any of the neural control variables. Future investigation would be suggested to determine the neural control variables plyometric training may directly effect and to determine the appropriate overload to achieve modification of the variables.

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