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# The Effects of Antagonist Prefatigue on Agonist Torque and Electromyography

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**ABSTRACT**

This study assessed the effects of hamstring prefatigue on peak torque, peak power, time to peak torque, knee angle of peak torque, and electromyography (EMG) activity of the hamstrings and quadriceps group during knee extensions at angular velocities of 60°, 180°, and 300°·s$^{-1}$. Twenty Division I wrestlers performed 5 maximal knee extensions in prefatigued and nonfatigued conditions of the hamstring group. This study demonstrated that when the hamstrings were prefatigued, the quadriceps produced significant decreases in peak torque of 1.7% ($p < 0.05$), peak power of 11% ($p < 0.05$), and rate to peak torque of 6.4% ($p < 0.01$) as compared with the nonfatigued state. When the hamstrings were prefatigued, they produced a 25% greater amount of EMG activity during knee extension ($p < 0.01$) than when not prefatigued. There was no significant difference in quadriceps EMG activity whether the hamstring group was prefatigued or not ($p > 0.05$). The decrease in quadriceps peak torque during the prefatigued condition was more pronounced ($p < 0.01$) at an angular velocity of 60°·s$^{-1}$ than at 180° or 300°·s$^{-1}$. In other words, prefatiguing the antagonist appears to be most detrimental to torque output of the quadriceps in the condition that most closely replicates the speed at which ''isotonic'' weight training occurs (60°·s$^{-1}$) and suggests a limitation to agonist-antagonist superset training.

***Key Words:*** angular velocity, peak power, peak torque, knee extension, coactivation, co-contraction, superset, compound set

***Reference Data:*** Maynard, J., and W.P. Ebben. The effects of antagonist prefatigue on agonist torque and electromyography. *J. Strength Cond. Res. 17(3):469–474. 2003.*

## Introduction

Most weight-training programs designed for increasing strength include multiple sets of 6 or less repetitions and 3–5 minutes of interset rest (14). Frequently, 1 exercise is performed for a specific number of sets before moving on to the next exercise. This type of training can be somewhat time-consuming due to the relatively long interset rest periods. Some body building publications have recommended ''antagonist training'' as a way to reduce the time spent in the weight room without compromising results of the training session. During antagonist training, agonist and antagonist muscles are trained ''back-to-back,'' without rest between sets, in what is also called a ''superset'' (1) or ''compound set'' (14). Unfortunately, the literature is inconsistent regarding the effect of this type of training.

Most functional movements involve agonist and antagonist co-contraction (2). Studies have shown antagonist muscles are active during movement caused primarily by the agonists (2, 17, 18). This phenomenon has been described as ''coactivation'' or ''co-contraction'' (8, 13, 24) and may affect agonist force production and velocity (23). Some evidence demonstrates that the antagonists may be particularly active while subjects perform dynamic movements against heavy loads (12, 22) and during limb deceleration (19). Questions remain regarding the potential effects of coactivation during superset training.

In an effort to evaluate the role of antagonist coactivation, researchers have attempted to ''prefatigue'' the antagonist and evaluate the effect on the agonist. Some evidence shows that prefatiguing may inhibit the antagonist and result in increased agonist force production (10, 11, 15, 16).

However, other evidence demonstrates that prefatiguing the antagonist may actually have an ergogenic effect on the antagonist that may attenuate agonist force potential. Bohannon et al. (3) found that 10 repetitions of knee flexion did not increase knee extension torque. Other studies indicate that isometric precontractions of the knee flexors actually increased flexor coactivation during leg extension, thus reducing extensor (agonist) force production (23, 24).

Ultimately, the effect of antagonist prefatigue may depend on agonist velocity. Burke et al. (9) suggested that agonist force output depends on the speed of agonist movement and amount of force necessary. More specifically, slow speed/high force agonist contractions preceded by slow speed/high force antagonist contraction resulted in a decreased force output from

the agonist. On the other hand, high speed/low force agonist contraction preceded by high speed/low force antagonist contraction resulted in an increase in force output from the agonist.

Research is inconclusive regarding the effects of antagonist prefatigue and its effect on agonist performance. Furthermore, previous studies have not evaluated how a set of concentric contractions of the antagonist, performed prior to a set of concentric contractions of the agonist, influences the agonist. This type of research design is necessary to more closely evaluate superset training. The purpose of this study was to investigate the agonist peak torque, peak power, time to peak torque, peak torque knee angles, and hamstring and quadricep electromyography (EMG) at various speeds of isokinetic knee extension following antagonist prefatigue.

## Methods

### *Experimental Approach to the Problem*

Independent variables included prefatigue or nonprefatigued antagonist conditions. Dependent variables included average peak torque (N·m), time to average peak torque (milliseconds), knee angle of peak torque (degrees per second), average peak power (watts), and agonist and antagonist EMG. Subjects in this study prefatigued their antagonist (hamstring) muscle group by performing 5 repetitions of maximal isokinetic leg flexion. Dependent variables were then assessed during 5 repetitions of subsequent maximal leg extensions.

### *Subjects*

Subjects included 20 male Division I wrestlers (age = 20.3 ± 0.7 years; weight = 70.0 ± 8.6 kg). All subjects participated in a resistance training and conditioning program, were allowed 48 hours recovery prior to data collection, and were without lower-extremity pathology. All subjects volunteered and signed an informed consent prior to participating. The Marquette University Institutional Review Board approved this study.

### *Instrumentation: Isokinetic Dynamometer*

Measurements of knee extensor torque and power were assessed using an isokinetic dynamometer (Biodex Medical, Inc., Model 900-220, Shirley, NY) and recorded and stored using the Biodex Advantage Software Program (version 3.2). Isokinetic testing was used since it offers readily quantifiable torque at several contraction velocities. This type of data would be difficult to obtain isotonically. The dynamometer was set at sensitivity position ''C,'' programmed for concentric/concentric knee extension/flexion, and reset and calibrated based on limb weight, limb length, age, body weight, and test protocol for each subject. Knee extension and knee flexion were defined as 0° and 90°, respectively. The dynamometer shaft was set to limit the range of motion for flexion of the knee to 105°. The axis of rotation of the dynamometer was placed in line with the lateral epicondyles of the knee. The dynamometer software automatically accounted and adjusted for limb weight and gravity of the subject's leg.

### *Instrumentation: Electromyography*

EMG data were collected using surface electrodes, a signal amplifier, and an analog to digital converter (BIOPAC Systems, Inc., Model MP100, Goleta, CA; 5). Previous research has identified a direct relationship between EMG and muscle force production for both static and dynamic muscle actions (3, 7). Data were recorded from the vastus medialis, vastus lateralis, and the biceps femoris of the right leg of each subject and stored in a laptop computer using Acqknowledge 3.6. Research suggests that EMG is the most common method used to evaluate agonist/antagonist motor unit activity (25).

The EMG was calibrated by adjusting to achieve a zero balance by having subjects relax their right leg while sitting in the dynamometer. EMG data were sampled at 500 Hz for 30 seconds. An electrical stimulation machine was used to identify the motor points in the muscle bellies of the vastus lateralis, vastus medialis, and the biceps femoris to determine EMG electrode placement. At these motor points, hair was shaved and the skin was mildly abraded with fine sandpaper and cleansed with alcohol. Self-adhesive electrodes were placed 2.5 cm apart on each of the muscle bellies, and ground electrodes were placed on the medial and lateral epicondyles of the femur.

### *Warm-Up*

The warm-up consisted of 5 minutes on a bicycle ergometer, brief static stretching of leg and hip muscles for 10–15 seconds per muscle group, and 5 repetitions of knee flexion/extension at 50% intensity at angular velocities of 300°, 180°, and 60°·s$^{-1}$. Subjects were allowed 5 minutes to rest before the testing.

### *Testing Procedure*

Subjects were secured in the dynamometer with thigh, pelvic, and torso straps to reduce the amount of extraneous body movements. The start position was set at 105° of flexion. Each subject was tested at angular velocities of 60°, 180°, and 300°·s$^{-1}$ in the prefatigued and nonfatigued hamstring conditions with each condition separated by a 10-minute break and test order randomly assigned.

Prefatigue was accomplished by performing 5 maximal repetitions of leg flexion, with passive extension, at angular velocities of 60°, 180°, and 300°·s$^{-1}$. Immediately following each prefatiguing set, each subject performed a set of 5 maximal repetitions of leg extension, with passive flexion, at the same angular velocity of the prefatiguing set. EMG data were collected for

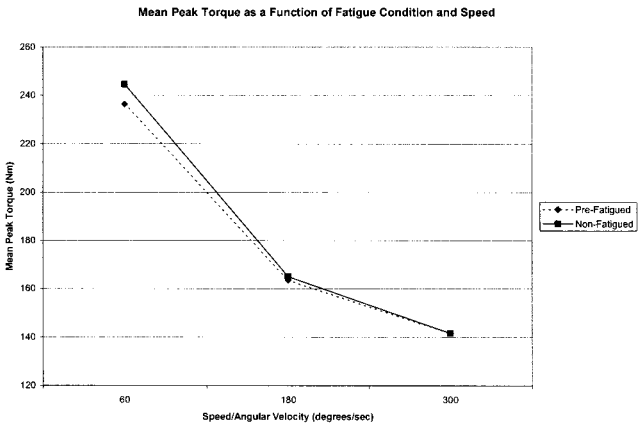


**Figure 1.** Mean peak torque of the agonist was decreased when the antagonist was prefatigued ($p < 0.05$). As angular velocity increased, mean peak torque decreased ($p < 0.01$). When angular velocity was slower and the antagonists were prefatigued, mean peak torque of the agonist was decreased ($p < 0.01$).

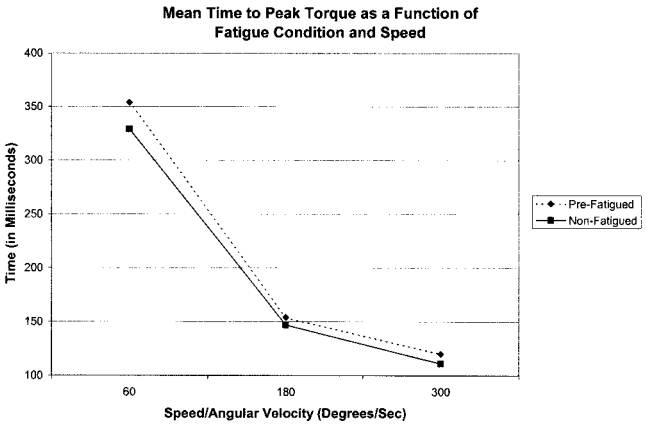


**Figure 2.** Agonist mean peak torque was reached at a faster rate when the antagonist was prefatigued ($p < 0.01$). As angular velocity increased, peak torque was reached at a faster rate ($p < 0.01$). When the antagonist is prefatigued and angular velocity is slow, it takes longer for the agonist to achieve mean peak torque ($p < 0.01$).

all repetitions for each speed and condition, and average values were used.

### *Statistical Analyses*

This study used a within-subjects design with repeated measures. A 2 × 3 (fatigue state × speed of movement) ANOVA was performed using Statistical Program for the Social Sciences (SPSS Inc., Chicago, IL) software. The Biodex software program recorded instantaneous torque, angular velocity, instantaneous power, and joint position at 0.01-second intervals. For the Biodex data, the averages of the 5 repetitions of knee extensions for peak torque, peak power, time to peak torque, and knee joint angle at peak torque were used to determine levels of significance. For the EMG data, Acqknowledge software was used to transform the data to absolute values; the data was then smoothed, and the mean integrated EMG (IEMG) data were gathered and used to determine significance levels. Statistical significance was accepted at $p \leq 0.05$.

## Results

Figure 1 shows a main effect by fatigue condition and speed of movement as well as a 2-way interaction effect by fatigue condition and speed of movement for average peak torque of the quadriceps group. These results demonstrate that when the antagonist muscle group was prefatigued, the peak torque of the agonist was diminished ($p < 0.05$). Additionally, Figure 1 shows that greater angular velocity decreases peak torque ($p < 0.01$). The 2-way interaction effect demonstrates that when the antagonist is prefatigued, the average peak torque is decreased, especially when the angular velocity is slower ($p < 0.01$). Average peak torques were 3.5% or 8.3 N·m greater at $60°\cdot s^{-1}$ and

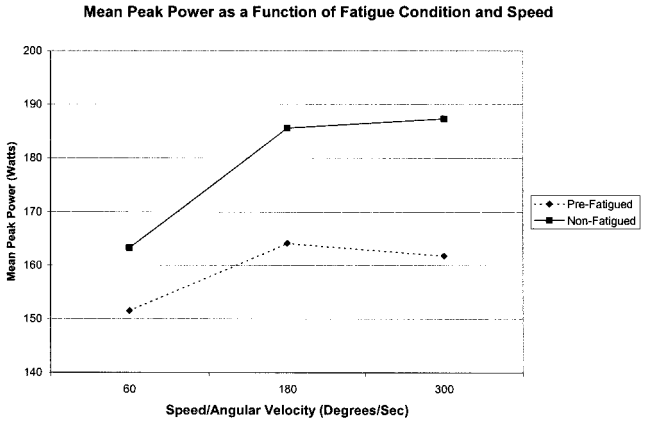


**Figure 3.** Mean peak power of the agonist is decreased when the antagonist is prefatigued ($p < 0.05$). As angular velocity increased, mean peak power of the agonist increased ($p < 0.05$).

0.88% or 1.4 N·m greater at $180°\cdot s^{-1}$ than they were at $300°\cdot s^{-1}$.

Figure 2 shows a main effect by fatigue condition and speed of movement as well as an interaction effect by fatigue condition and speed of movement for average time to reach peak torque in the quadriceps muscle group. The main effect by fatigue condition is evidenced by the quadriceps reaching peak torque faster when the antagonist muscle group was not prefatigued ($p < 0.01$). The main effect by speed of movement establishes that at greater angular velocity, peak torque is reached earlier ($p < 0.01$). The interaction effect by speed of movement and fatigue condition demonstrates that when the antagonist muscle is prefatigued, it will take longer for the agonist to achieve peak torque ($p < 0.01$).

Figure 3 shows a main effect by fatigue condition and speed of movement for average peak power in the

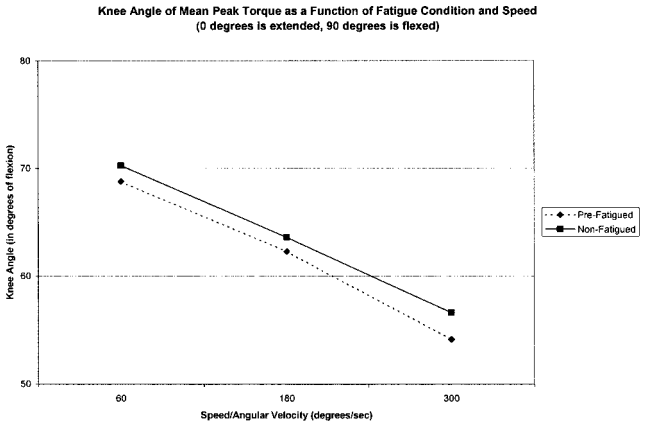


**Figure 4.** Mean peak torque of the agonist occurs later in the range of motion during leg extension as angular velocity increased ($p < 0.01$).

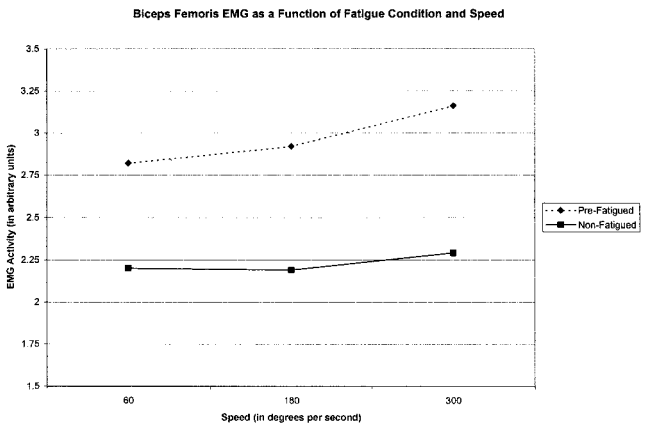


**Figure 5.** Antagonist integrated EMG (IEMG) coactivation was greater during leg extension when the antagonists were prefatigued ($p < 0.01$). Antagonist IEMG did not change as a result of changing angular velocity.

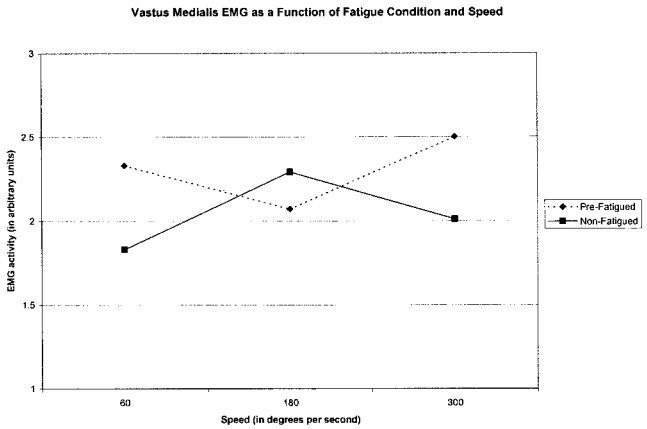


**Figure 6.** There were no significant differences in integrated EMG (IEMG) activity of the vastus medialis between conditions.

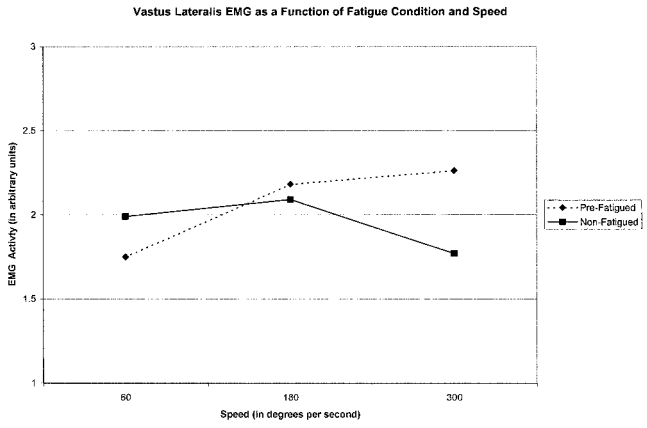


**Figure 7.** There were no significant differences in integrated EMG (IEMG) activity of the vastus lateralis between conditions.

quadriceps muscle group. The main effect by fatigue condition reveals that when the antagonist is prefatigued, the average peak power is diminished ($p < 0.05$). The main effect by speed of movement substantiates that the greater the angular velocity during leg extension, the greater the average peak power of the quadriceps muscle group ($p < 0.05$).

Figure 4 shows a main effect by speed of movement for the average peak torque knee angle during leg extensions. The main effect by speed of movement demonstrates that the greater the angular velocity of the leg extension, the later the peak torque occurs in the range of motion of the limb ($p < 0.01$). When the antagonist was prefatigued, peak torque occurred an average of 1.76° later in the range of motion than when the antagonist was not prefatigued.

Figure 5 shows a main effect by fatigue condition of the biceps femoris IEMG activity. The main effect exhibits that when the antagonist muscle group was prefatigued, the biceps femoris produced a greater amount of IEMG activity during the knee extension ($p < 0.01$). There was no significant difference in the IEMG of the biceps femoris when the angular velocity increased.

Figures 6 and 7, respectively, evidence no significant difference in the vastus medialis or vastus lateralis IEMG activity as a function of fatigue condition or speed of movement.

## Discussion

Results demonstrated diminished agonist torque and power, as well as increased time to peak torque when the antagonist muscle group was prefatigued. This finding may be explained, in part, by the increased cocontraction of the antagonist muscle as evidenced by increased hamstring (antagonist) IEMG during the knee extension. In this study, attempts to prefatigue the antagonist actually appear to have had a facilitative or ergogenic effect on the antagonist, thus inhibiting the agonist.

Unlike the present study, previous studies have

demonstrated that antagonist prefatigue has no effect (6) or increases agonist force production (10, 11, 15, 16). These differences may be partially explained by the fact that subjects in these studies used maximum voluntary isometric muscle action or alternated antagonist and agonist muscle actions for each repetition. In the present study, subjects performed an entire set of antagonist concentric contractions prior to the test set of agonist concentric contractions in order to more closely approximate superset training. On the other hand, the present study is consistent with previous research that suggests that antagonist prefatigue has a facilitative, as opposed to an inhibitory, effect on the antagonist muscle groups, resulting in decreased agonist performance (23, 24).

The results are also consistent with the findings of Burke et al. (9) who reported decreased force output of agonist contractions following slow-speed antagonist contractions. The diminished agonist force production associated with antagonist prefatigue appears dictated, in part, by agonist velocity. Previous research has indicated that peak torque is greater at lower angular velocities (4, 20). Since torque is greater at lower movement velocities, the potential for torque to be impaired by antagonist co-activation may also be greater. In the present study, antagonist prefatigue had little to no effect on the subsequent torque production of the agonist at angular velocities greater than $180°\cdot s^{-1}$. However, at slower movement speeds ($60°\cdot s^{-1}$), antagonist prefatigue had a significant effect on torque production of the agonist.

Results of this study support the idea that there may be an ergogenic warm-up effect associated with weight training. Ultimately, the acute ergogenic or fatiguing role of weight training exercise may depend on neurogenic factors such as inhibition or excitation or bioenergetic factors such as substrate depletion and may be dependent on acute training volume (21).

Finally, research in this area has been conducted using isokinetic dynamometers, presumably in order to prescribe quantifiable exercise stimuli and to collect precise measurements of torque at a variety of velocities. Unfortunately, limitations exist in the degree to which findings associated with isokinetic testing can be generalized to isotonic training.

## Practical Applications

This study demonstrates that antagonist/agonist superset training may not be the most effective system of weight training as evidenced by a reduction of torque, time to peak torque, and power during subsequent agonist sets. Traditional weight training systems may be more advantageous. Agonist performance is especially attenuated at slower isokinetic velocities ($60°\cdot s^{-1}$), which may be most similar to the velocities associated with high-load isotonic exercises. Alternatively, antagonist prefatigue had little effect on agonist performance at relatively high isokinetic speeds (greater than $180°\cdot s^{-1}$), raising the question about whether agonist force is impaired when conducting explosive movement such as when using maximum power training or plyometrics in a superset with weight training. Future research should investigate the effect of antagonist/agonist training in isotonic exercise.

*Note:* Jeremy Maynard is with the Engineer Brigade, 38th ID (Mechanized), Swartz Creek, MI 48473.

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## *Acknowledgments*

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