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Contributors to Fatigue Resistance of the Hamstrings and Quadriceps in Cerebral Palsy

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Abstract

Background—The purpose of this study was to elucidate relationships between quadriceps and hamstrings voluntary muscle fatigue and upper motor lesion impairments in cerebral palsy in order to gain a better understanding of their contribution to the observed fatigue resistance.

Methods—Seventeen ambulatory subjects with cerebral palsy (mean age: 17.0, SD = 4.8 years) were recruited. Quantitative measures of strength, spasticity, cocontraction, and stiffness for both muscle groups were collected on an isokinetic dynamometer and entered in a factor analysis. The resulting factors were used as independent variables in a multiple regression analysis with quadriceps and hamstrings fatigue as dependent variables.

Findings—Five independent factors explained 90% of the variance. In order of loadings, higher hamstring cocontraction and spasticity and lower hamstring strength were associated with lower levels of hamstring fatigue. Higher quadriceps cocontraction and lower quadriceps strength were the most predictive of lower levels of quadriceps fatigue.

Interpretation—Greater motor impairments of the agonist muscle, particularly cocontraction, spasticity, and weakness, were associated with lower rates of muscle fatigue of the same muscle during performance of a voluntary fatigue protocol for the hamstrings and quadriceps. Muscles are highly adaptable; therefore, the results of this study suggest that the observed fatigue resistance may be due to the effect of the primary neural insult on motor unit recruitment and rate modulation or the result of secondary adaptations to spasticity, weakness, or excessive cocontraction.

Keywords

Muscle Fatigue; Muscle Strength; Rehabilitation; Muscle Spasticity; Cocontraction; Weakness

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Introduction

Muscle fatigue is defined as a reduction in force output that occurs during sustained activity (Bigland-Ritchie et al., 1983). Debate over the mechanisms responsible for lower levels of muscle fatigue, known as fatigue resistance, in certain populations has been a topic of discussion for many years. For example, greater fatigue resistance has been observed in children (Ratel et al., 2003) and healthy older adults compared to young adults (Lanza et al., 2004) and in females compared to males (Hunter and Enoka, 2001). Despite the inconsistency of these findings, several mechanisms have been proposed to explain the fatigue resistance, primarily group differences in the following: muscle mass, absolute torque levels, muscle morphology, voluntary activation, patterns of motor unit recruitment and rate modulation, and energy metabolism or substrate utilization (Enoka and Stuart, 1992; Hunter and Enoka, 2001; Lanza et al., 2004; Pincivero et al., 2003; Ratel et al., 2003).

We have previously shown that the hamstrings and quadriceps in children and young adults with cerebral palsy (CP) are more fatigue resistant than those of typically developing peers during performance of a voluntary fatigue protocol (Moreau et al., 2008b). This finding has also been corroborated by Stackhouse et al., (2005) during an electrically elicited peripheral fatigue test of the quadriceps. In addition to the aforementioned mechanisms, additional factors specific to neurological injury such as weakness, cocontraction, spasticity, and stiffness may play a significant role in the fatigue resistance observed. CP is a multifaceted disorder and as such, complex interrelationships may exist among upper motor neuron lesion impairments. The relationships between muscle fatigue and these upper motor neuron lesion impairments are poorly understood and may prove important in the understanding of fatigue, as these may contribute directly or indirectly to the level of muscle fatigue.

Marked strength deficits have been well documented in children with CP (Wiley and Damiano, 1998). The etiology of weakness in CP is believed to be multifactorial and may include decreased central drive to the agonist due to the lesion itself, spasticity, antagonist cocontraction, secondary changes in the properties of the muscles fibers, decreased muscle size, or some combination of the above (Damiano et al., 2001; Malaiya et al., 2007). Differences in strength have been postulated to explain the differences in muscle fatigue observed between older and younger adults, children and young adults, and females and males (Allman and Rice, 2002; Hunter and Enoka, 2001; Ratel et al., 2003). These studies suggest that stronger individuals have a higher susceptibility to fatigue due to the ability to generate higher absolute torque levels or due to greater muscle mass. Therefore, it is reasonable to assume that weaker muscles, such as in CP, would be inherently less fatigable.

Other muscle abnormalities which may play a role in the development of fatigue resistance in individuals with CP include excessive collagen accumulation (Booth et al., 2001) and increased stiffness at both the cellular (Friden and Lieber, 2003) and whole muscle level (Hufschmidt and Mauritz, 1985). Stiffness is defined as a length-dependent resistance to movement. It has been suggested that increased stiffness may be a compensation for weakness, thus allowing better utilization of elastic energy during functional activities, such as gait (Lamontagne et al., 2000; Svantesson and Sunnerhagen, 1997).

It is well-recognized that many children with CP have spasticity, or a velocity-dependent increased resistance to movement due to hyperexcitable stretch reflexes (Lance, 1980) as well as excessive contraction. Cocontraction in persons with upper motor neuron lesions is thought to be caused by reciprocal facilitation/excitation of the agonist and antagonist (Myklebust et al., 1982) or decreased disynaptic or presynaptic reciprocal inhibition of the antagonist muscle during agonist activation (Morita et al., 2001). Cocontraction has been suggested to generate opposing torque throughout the range of motion (Baratta et al., 1988). In addition, full

activation of the agonist may be impaired due to reciprocal inhibition (Milner et al., 1995; Tyler and Hutton, 1986). Similarly, spasticity of the antagonist muscle may also impair the maximum force output of the agonist, secondary to the hyperexcitable stretch reflex. Therefore, increased antagonistic cocontraction and spasticity could theoretically reduce the efficiency of force output, leading to fatigue, although this theory has not been tested experimentally.

We chose the quadriceps muscles because of their important role in maintaining upright posture in standing and walking, as well as in other functional activities such as squatting and stair climbing. Appropriate timing and reciprocation of the hamstring and quadriceps muscles influence excursion of the knee joint during gait, which is an important determinant of stride length, gait velocity, and toe clearance. It is especially important to measure agonist/antagonist muscle pairs in those with CP where spasticity and cocontraction are commonly reported (Damiano, 1993; Damiano et al., 2001). Although we have previously shown that the quadriceps and hamstrings in people with CP are less fatigable than typically developing peers (Moreau et al., 2008b), possible contributors to the fatigue resistance specific to upper motor neuron lesions have yet to be investigated. Therefore, the purpose of this study was to quantify spasticity, stiffness, cocontraction, and strength of the quadriceps and hamstrings in this same group of subjects with mild to moderate CP in order to determine which variables were associated with fatigue of these muscle groups. Because individuals with upper motor neuron lesions have been shown to have both central and peripheral contributions to fatigue (de Haan A. et al., 2000; Lenman et al., 1989; Toffola et al., 2001), a voluntary muscle fatigue protocol was chosen to test the combined influence of both aspects of fatigue. We hypothesized that greater weakness and stiffness of the agonist would be associated with lower levels of agonist muscle fatigue. Conversely, greater antagonist cocontraction and spasticity would be associated with increased muscle fatigue of the agonist muscle.

Methods

Participants

Seventeen subjects with CP (mean age: 17.0; SD = 4.8 years; range = 10 to 23) were recruited for the study. All subjects were ambulatory either with or without assistive devices and were within Gross Motor Function Classification System Levels I (n = 9), II (n = 5), and III (n = 3). Subjects were excluded if they underwent orthopedic surgery within 12 months prior to the testing, received Botulinum toxin injections to the quadriceps or hamstrings within 6 months prior to the testing, or suffered from knee pain. The study was approved by the Institutional Review Board at our institution. Written consent was obtained from each participant over 18 years of age or parental consent obtained for participants under 18.

Procedures

Setup—An isokinetic dynamometer (Biodex Medical Systems Incorporated, Shirley, NY, USA) was used to collect angular displacement, angular velocity, and torque data for both the passive and active trials. Subjects were positioned in the Biodex chair as described previously in more detail (Moreau et al., 2008a). The more involved lower extremity was tested for subjects with bilateral involvement. The passive range of motion designated as “comfortable” by the patient was determined and used to set the limits of motion for the rest of testing sessions. Subjects were instructed to keep their arms folded across their chest for all trials.

Surface electromyography (EMG) of the lateral hamstrings and quadriceps was collected with the MA-300 EMG system (Motion Lab Systems, Baton Rouge, LA, USA). The skin was cleansed and abraded with alcohol prep pads before placement of the electrodes. The Ag-AgCl bipolar electrode pair was positioned one-third of the distance between the ischial tuberosities and the popliteal crease on the muscle belly of the biceps femoris and on the muscle belly of

the rectus femoris, one-third of the distance between the anterior iliac superior spine and the patella proximally. A ground electrode was placed on the anterolateral surface of the participant's upper thigh. Proper electrode placement was verified through use of the WinDaq Acquisition software (Dataq Instruments, Dayton, OH, USA) in conjunction with manual muscle testing. The bandpass width used for collection was 0 to 500 Hz, and the signal was sampled at 1000 Hz per channel with an amplification of up to 20,000. The common mode rejection ratio (CMRR) was 100 dB.

Isokinetic Passive Testing—The passive testing consisted of repeated extension and flexion of the knee within the preset range of motion with a 1 second pause during the reversal of motion. The subjects were instructed to relax their muscles. Three passive repetitions were performed at 5, 10, 30, 60, 90, and 120 degrees/second. A 30 second rest period was provided between each velocity. Surface EMG of the quadriceps and hamstrings were monitored during the test to provide verification that the muscles were not voluntarily activated. Angular displacement, angular velocity, and torque data from the dynamometer were collected with the EMG data and internally synchronized using the WinDaq Acquisition software.

Isokinetic Fatigue Testing—The subjects performed 5 to 10 submaximal concentric, reciprocal knee flexion and extension repetitions to familiarize themselves with the procedure. Five minutes of rest was given prior to the muscle fatigue protocol. The fatigue protocol consisted of reciprocal, maximal concentric knee extension and flexion at 60 degrees/second for 35 repetitions. This protocol was shown to be feasible in a group of subjects with mild to moderate CP (Moreau et al., 2008a). Verbal encouragement and visual feedback of the torque value presented on the monitor was used to encourage maximum effort.

Data Analysis

Only data in the constant velocity portion of all trials were analyzed, thereby negating the effects of inertia. Prior to data analysis, calculation of the limb weight was taken between 25 and 45 degrees of knee flexion in order to remove the gravitational effects of the limb and attachment from each trial. The algorithm provided by the Biodex Advantage Software Operations Manual (Version 3.29/3.30) was utilized for the gravity correction.

Spasticity—Spasticity was measured as the peak resistive torque (RT) during the isokinetic portion of the passive range at 60 and 120 degrees/second, with EMG verification of a stretch response (Damiano et al., 2001). Spasticity of the hamstrings was measured as the peak RT during passive knee extension, and spasticity of the quadriceps was measured as the peak RT during passive knee flexion.

Stiffness—Passive elastic stiffness was calculated as the slope of the resistance torque by angle curve during the constant velocity portion of the 5 degree per second passive trial. The slow speed was used in order to minimize the effect of velocity and reflexive activity (Lee et al., 2002) so that the resistance provided by the passive mechanical properties of the tissues was measured. Stiffness measured during knee extension was referred to as hamstring stiffness, and stiffness measured during knee flexion was called quadriceps stiffness. To provide a measure of velocity-dependency, stiffness was also calculated at 30, 60 and 90 degrees/second. Both reflexive and elastic stiffness play a role in those with spastic responses at these higher velocities.

Cocontraction—The recorded EMG data from the strength assessment were full-wave rectified and smoothed with a low pass filter at 6 Hz using a fourth-order zero lag Butterworth filter. The filtered EMG was separated into a flexion and extension phase during the isokinetic portion and the mean absolute value was calculated. Hamstring cocontraction during the

maximal knee extension contraction was calculated as the ratio of the mean absolute value of the biceps femoris EMG activity during the extension phase to the EMG activity during the flexion phase, multiplied by 100. Quadriceps cocontraction during the maximal knee flexion contraction was calculated as the ratio of the mean absolute value of the rectus femoris EMG activity during the flexion phase to the EMG activity during the extension phase, multiplied by 100 (Baratta et al., 1988; Weir et al., 1998).

Fatigue and Strength—The measure of fatigue was the rate of decline in peak torque (PT) represented by the slope of the linear regression, beginning with the first value of the highest 5 consecutive repetitions and ending with the 35th repetition (Pincivero et al., 2001). This fatigue calculation has been previously shown to be reliable in this population (Moreau et al., 2008a). The slope was calculated separately for knee extension and flexion. The maximum PT over the 35 repetitions of the fatigue protocol was also recorded separately for the quadriceps and hamstrings as the measure of maximal strength.

Statistical Analyses—Bivariate correlations were calculated in order to determine the simple linear relationships among variables and the directionality of those relationships. In order to reduce the number of intercorrelated variables into a smaller set of independent factors, all variables were entered into a factor analysis except for the fatigue variables, which would later serve as dependent variables. Only factors with eigenvalues greater than 1 were retained and varimax rotation was used to maximize the variance among variables within factors. Variables within each factor received a loading. Factor loadings measure the degree of involvement or the weight of each variable within a factor. The extracted factors were then used as independent variables in a multiple regression analysis where the dependent variables were the fatigue variables for the quadriceps and hamstrings.

Results

Factor Analysis

Table 1 lists the mean and standard error of the mean (SEM) of all variables used in the factor analyses for the quadriceps and hamstrings. Appendix 1 illustrates the bivariate correlations among the variables. Stiffness of the quadriceps at 90 degrees/second was removed from the factor analysis due to missing data values from three subjects in which we were unable to obtain a measure at this velocity.

The factor analysis revealed five independent factors, which accounted for 90% of the variance. Table 2 lists only variables with factor loadings greater than .40. Factor 1 was composed of very high loadings (>.90) of hamstring stiffness variables, while Factor 2 was associated with quadriceps stiffness. Factor 3 consisted of high loadings related to the hamstring variables, particularly hamstring cocontraction and spasticity. Factor 4 consisted of high loadings related to the quadriceps, particularly spasticity. Factor 5 was dominated by quadriceps cocontraction. Hamstring and quadriceps strength loaded together in Factors 4 and 5, as well as hamstrings strength in Factor 3. Examination of the loadings within these factors collectively illustrates the inverse relationship of strength with cocontraction and spasticity and the direct relationship of hamstring cocontraction and spasticity.

The results of the multiple regression analysis, where the extracted factors were used as independent variables, revealed that only Factor 3 significantly predicted hamstring fatigue ($P = .05$). Loadings within Factor 3, in order of weight, indicated that higher hamstring cocontraction and spasticity and lower hamstring strength were associated with lower levels of hamstring fatigue. Factor 5 was the only significant predictor of quadriceps fatigue ($P = .04$). Loadings within Factor 5, in order of weight, indicated that higher quadriceps

cocontraction and lower quadriceps and hamstring strength were predictive of lower levels of quadriceps fatigue.

Discussion

The primary result of the regression analysis was that motor impairments of the agonist muscle load together and collectively influence fatigue of that same muscle. Specifically, greater hamstring cocontraction, spasticity, and weakness, in order of loadings, were the strongest predictors of the lower rate of hamstring fatigue, while greater quadriceps cocontraction and strength were the strongest predictors of greater quadriceps fatigue. Greater weakness was associated with lower levels of muscle fatigue, or fatigue resistance, of the agonist muscle as hypothesized. However, greater cocontraction and spasticity of the agonist muscle, rather than the antagonist, was associated with lower rather than higher levels of muscle fatigue. These results suggest that the greater the motor impairments of a particular muscle, the lower the rate of muscle fatigue. This hypothesis is supported partly by previous work which shows that participants with CP with greater gross motor involvement (i.e. higher GMFCS levels) are less fatigable than those that are higher functioning (Moreau et al., 2008b).

These findings could be the direct result of the primary injury to the central nervous system, impairing neural output. Normally, the force of a muscular contraction is determined by both firing rate or rate modulation and the recruitment of additional motor units (De Luca and Erim, 1994). According to orderly recruitment, smaller motor units are recruited first, followed by larger, more fatigable motor units. Motor unit recruitment and firing rates have been reported to be decreased in CP (Rose and McGill, 2005; Stackhouse et al., 2005) and in patients with hemiparesis as a result of stroke (Gemperline et al., 1995; Jakobsson et al., 1992). People with CP who have greater hamstring and quadriceps impairments (i.e., cocontraction, spasticity, weakness) would likely have even greater impairments in motor unit recruitment and firing rates. The inability to recruit higher threshold motor units due to impaired motor pathways or to increase firing rates sufficiently would result in the primary recruitment of lower threshold (slow) motor units. This would mean that a larger percentage of force is generated from Type I (slow, oxidative) fibers rather than the larger Type II (fast, fatigable) fibers, responsible for high force production, thus leading to increased fatigue resistance and weakness. The relationship between weaker muscles and fatigue resistance has also been suggested in the literature by others investigating weakness in stroke (Toffola et al., 2001) as well as gender and age differences in muscle fatigue (Allman and Rice, 2002; Hunter and Enoka, 2001; Ratel et al., 2003).

Morphological muscle adaptations occurring over time secondary to the abnormal neural input, such as increased type I fiber predominance (Castle et al., 1979; Marbini et al., 2002; Rose et al., 1994) and type II fiber atrophy (Castle et al., 1979; Ito et al. (1996) could further contribute to the observed fatigue resistance. Rose and McGill (2005) and Gemperline (1995) have suggested that the inability to increase firing rates as described in the previous paragraph may alter the precise match between the properties of the motoneuron and the mechanical properties of the muscle fibers, thus leading to muscle structural changes and weakness. Specific to the vastus lateralis in CP, Castle et al. (1979) reported increased percentages of type I fibers and specific atrophy of type IIb (fast, fatigable) fibers. Ito et al. (1996) also reported decreased numbers of type II fibers and even type IIb fiber deficiency in 40% of gastrocnemius specimens from patients with CP. Therefore, the neural contributions to fatigue resistance may be further perpetuated by altered muscle properties that occur secondary to weakness, spasticity, and cocontraction.

It would be reasonable to expect that both cocontraction and spasticity of the antagonist muscle would increase the rate of muscle fatigue of the agonist muscle during a voluntary performance

due to mechanical inefficiency and involuntary resistance to movement. For example, hamstring cocontraction and spasticity during knee extension would have a negative effect on net quadriceps torque production (Ikeda et al., 1998). Surprisingly, our results show that increased cocontraction and spasticity of the agonist, rather than the antagonist, are related to lower muscle fatigue of the agonist, which is opposite to what we had hypothesized. Co-contraction is a general description for the simultaneous activation of muscles across a joint, and often coexists with spasticity which is characteristic of upper motor neuron lesions (Damiano, 1993; Knutsson et al., 1997) and other involuntary mechanisms such as dystonia (Bertolasi et al, 2003). Reciprocal facilitation and/or lack of reciprocal inhibition are thought to be the pathophysiological mechanisms underlying cocontraction, which may entrain hyperactive stretch reflexes over time, thus contributing to the development of spasticity (Crone et al., 2003). The results of our factor analysis revealed that hamstring cocontraction and spasticity were directly related and had the highest loadings within the predictive factors, indicating the strongest inverse relationship with hamstring muscle fatigue. An alternative explanation is that functioning on a daily basis with excessive amounts of muscle cocontraction and spasticity about the knee and thus, overcoming resistance to movement from reciprocal facilitation, could have an endurance training effect on the muscle.

The relationship of muscle fatigue with a quantitative measure of spasticity has not been previously investigated in those with upper motor neuron lesions. Other studies related to fatigue in upper motor neuron disorders refer to muscles under investigation as spastic or paretic but do not provide a quantitative measure of the amount or presence of spasticity (de Haan A. et al., 2000; Schwid et al., 1999; Toffola et al., 2001). This is noteworthy because spasticity may affect certain muscles to varying degrees within and between individuals, while not affecting others. Therefore, it is important to document the magnitude of spasticity for the muscles under investigation.

Clinically, it is of importance to emphasize that although the muscles were observed to be more enduring, they are also inherently weaker, which means that the level of force that is able to be sustained may not be of an appropriate magnitude to be functional. These results even suggest that the greater the spasticity, weakness, and cocontraction, the more enduring the muscle. Whether the fatigue-resistance is the direct result of the neural impairments or a secondary adaptation is unknown.

Caution should be used in extrapolating the results of this study to muscle groups other than the hamstrings and quadriceps, as muscle characteristics such as size, fiber type distribution, fiber arrangement, recruitment, and rate coding strategies differ across muscle groups. Furthermore, only motor impairments specific to upper motor neuron lesions were explored in the study. Therefore, other neural, peripheral, or metabolic factors that could potentially contribute to fatigue beyond the scope of this study cannot be ruled out.

Conclusion

In conclusion, abnormal muscle activation of the agonist muscle, particularly cocontraction, spasticity, and weakness, were related to lower rates of muscle fatigue during performance of a voluntary fatigue protocol for the hamstrings and quadriceps. The results of this study provide a novel contribution to the literature in that the effect of muscle spasticity on muscle fatigue has not been evaluated before in any neurological population. Muscles are highly adaptable; therefore, the results of this study suggest that the observed fatigue resistance may be due to the effect of the primary neural insult on motor unit recruitment and rate modulation or the result of secondary adaptations to spasticity, weakness, or excessive cocontraction.

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Table 1

Means and standard error of the mean (SEM) for all variables

	Fatigue Slope (N·m·rep ⁻¹)	PT 60°/s (N·m)	RT 60°/s (N·m)	RT 120°/s (N·m)	Stiff 5°/s (N·m)	Stiff 30°/s (N·m)	Stiff 60°/s (N·m)	Stiff 90°/s (N·m)	Cocon- traction (%)
Quads	.645 (.108)	55.029 (5.117)	2.139 (.116)	2.830 (.245)	.055 (.011)	.077 (.015)	.078 (.014)	.070 (.014)	15.376 (4.001)
Hams	.344 (.059)	23.976 (2.821)	8.393 (.803)	9.870 (1.112)	.274 (.027)	.312 (.028)	.335 (.033)	.366 (.032)	82.557 (13.866)

Hams = hamstrings; Quads = quadriceps; RT = resistance torque; PT = peak torque; Stiff = stiffness

Table 2
Factor analysis results. Variables are shown in loading order, where loadings are > .40

Factors	Load
Factor 1: Hamstring stiffness	
Hams stiffness 60°/s	.98
Hams stiffness 30°/s	.95
Hams stiffness 90°/s	.94
Hams stiffness 5°/s	.93
Hams RT 60°/s	.61
Hams RT 120°/s	.57
Factor 2: Quadriceps stiffness	
Quads stiffness 30°/s	.95
Quads stiffness 60°/s	.93
Quads stiffness 5°/s	.82
Factor 3: Hamstring cocontraction and spasticity	
Hams cocontraction	.85
Hams RT 120°/s	.78
Hams RT 60°/s	.70
Hams PT 60°/s	-.59
Factor 4: Quadriceps spasticity	
Quads RT 60°/s	.81
Quads RT 120°/s	.71
Quads PT 60°/s	-.63
Hams PT 60°/s	-.59
Factor 5: Quadriceps cocontraction	
Quads cocontraction	.92
Quads PT 60°/s	-.60
Hams PT 60°/s	-.46

+/- loadings indicate that the relationships within the factor are inverse to one another. Direction of relationships is determined from correlation coefficients (Appendix 1). Hams = hamstrings; Quads = quadriceps; RT = resistance torque; PT = peak torque

APPENDIX 1

Pearson (r) correlations

	Q Slope	H Slope	OPT 60°/s	HPT 60°/s	QStiff 5°/s	QStiff 30°/s	QStiff 60°/s	QStiff 90°/s	HSdiff 5°/s	HSdiff 30°/s	HSdiff 60°/s	HSdiff 90°/s	QRT 60°/s	QRT 120°/s	HRT 60°/s	HRT 120°/s	Quad Cocon	Hams Cocon	
Q Slope		.69*	.69*	.74*	-.49*	-.43	-.36	-.64*	.31	.20	.16	.15	-.17	-.13	.20	-.06	-.54*	-.57*	
H Slope			.54*	.78*	-.49*	-.24	-.22	-.72*	.27	.06	.15	.13	-.30	-.15	.10	-.11	-.42	-.52*	
QPT 60°/s				.71*	-.40	-.41	-.37	-.45	.41	.30	.23	.25	-.38	-.39	.52*	.28	-.61*	-.16	
HPT 60°/s					-.33	-.17	-.12	-.51	.33	.20	.22	.22	-.26	-.21	.10	-.13	-.46	-.49	
QStiff 5°/s						.85*	.72*	.79*	-.11	.24	.10	-.07	.53*	.32	-.14	-.07	.41	.21	
QStiff 30°/s							.93*	.86*	-.13	.07	-.04	-.14	.43	.13	-.18	-.12	.42	.32	
QStiff 60°/s								.90*	-.13	.07	-.08	-.15	.31	.09	-.19	-.09	.41	.35	
QStiff 90°/s									-.16	.10	-.01	-.07	.17	-.13	.02	.14	.69*	.75*	
HSdiff 5°/s										.83*	.92*	.90*	.14	.43	.70*	.57*	-.41	-.15	
HSdiff 30°/s											.89*	.72*	.13	.39	.53*	.41	-.05	-.16	
HSdiff 60°/s												.92*	.22	.51*	.64*	.55*	-.22	-.20	
HSdiff 90°/s													.20	.45	.72*	.69*	-.25	-.07	
QRT 60°/s														.55*	-.11	-.14	-.05	-.04	
QRT 120°/s															.00	.04	-.05	-.22	
HRT 60°/s																.93*	-.29	.31	
HRT 120°/s																	-.05	.43	
Quad Cocon																			.26
Hams Cocon																			