TITLE

Knee flexor strength and bicep femoris electromyographical activity is lower in previously strained hamstrings.

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INTRODUCTION

Hamstring strain injuries, characterised by acute pain in the posterior thigh and disruption of hamstring muscle fibres, are the primary injury sustained in a number of sports [Orchard & Seward, 2010; Woods et al., 2004; Drezner et al., 2005] and re-injury rates are also high [Orchard & Seward, 2010]. The high rate of injury and re-injury, combined with the fact that a previous hamstring strain injury is the most significant risk factor for future injury [Arnason et al., 2004], suggests that our understanding of the neuromuscular maladaptations that occur following hamstring strain requires further attention.

Previous hamstring strain injury has been associated with between-limb differences in eccentric strength that is typically greater than concentric strength deficits [Croisier et al., 2002; Lee et al., 2009]. Furthermore these deficits in eccentric strength are still present despite athletes returning to full training and competition [Croisier et al., 2002; Lee et al., 2009]. Whilst the retrospective nature of these findings cannot be taken to suggest that hamstring injury has resulted in these deficits, it is agreed that hamstring strain injury does lead to maladaptation [Opar et al., 2012]. Importantly, prospective studies in both sprinters and soccer players have identified eccentric knee flexor strength deficits as elevating hamstring strain injury risk [Croisier et al., 2008; Sugiura et al., 2008]. These findings suggest the importance of eccentric strength for the prevention of hamstring strain injury and that eccentric weakness should be corrected following injury to reduce the risk of a recurrence. However a clear understanding of the mechanisms underpinning the decline in eccentric strength following hamstring strain injury is required in order to develop more appropriate exercise interventions. Whilst evidence does exist of persistent atrophy of biceps femoris long head (BF) up to 23 months following grade I and II hamstring strain injuries [Silder et al., 2008] this muscular maladaptation does not explain why the decline in hamstring strength appears to be greater in eccentric actions [Croisier et al., 2002; Lee et al., 2009].

Surprisingly the impact of strain injuries on the neural function of the involved musculature has been largely overlooked. Hamstring strain injury has been reported to result in acute [Verrall et al., 2001] and chronic pain [Croisier et al., 2002; Jönhagen et al., 1994]. This muscular pain also has the potential to alter central nervous function at both the spinal and supraspinal level [Mense, 2003], and might therefore be expected to result in a restriction of electromyographical activity and the median power frequency of this activity during contraction. Furthermore this restriction may be specifically confined to the muscle and contraction mode responsible for the noxious stimulus. Therefore the purpose of this study was to assess concentric and eccentric hamstring torque, surface EMG (sEMG) activity and the median power frequency of the sEMG signal of recreational athletes with and without a history of unilateral hamstring strain injury. It was hypothesised that the previously injured hamstrings would display strength, sEMG activity and median power frequency deficits during fast and slow eccentric contractions, but not concentric contractions, compared to the contralateral limb. Furthermore, we hypothesised that lower levels of sEMG activity and median power frequency would be confined specifically to the previously injured hamstring muscle (i.e. BF or medial hamstrings (MH)). It was also hypothesised that the control group would display no differences in any of the aforementioned variables between dominant and non-dominant limbs. As a confirmatory secondary analysis, it was also hypothesised that the between limb differences in eccentric hamstring torque, sEMG and median power frequency would be greater in previously injured athletes compared to the control group.

MATERIALS AND METHODS

Participants

Twenty-eight recreationally active males participated in the study, with most competing in Australian football, rugby, soccer or sprinting. Thirteen athletes (26.2 ± 5.8 years; 1.80 ± 0.04m; 83.0 ± 14.8kg) had at least one unilateral hamstring strain injury (INJ) within the last 18 months and all had suffered a grade II injury previously. Another 15 athletes (26.7 ± 5.8 years; 1.8 ± 0.05m; 83.5 ± 7.9 kg) had no history of hamstring strain injury (UI). All participants were free of any other injury to the lower limbs and were fully active in their chosen sport at the time of testing. All testing procedures were approved by the University Human Research Ethics Committee. Participants gave informed written consent prior to testing after having all procedures explained to them.

Injury questionnaire

Following recruitment, participants completed an injury questionnaire with their chosen practitioner (i.e. physiotherapist) who had previously diagnosed and treated all the athletes hamstring strain injury. As per previous investigations [Sole et al., 2011], the notes taken from clinical examination were used to detail the date of injury and return to pre-injured levels of training and competition, severity (grade I, II or III) [Blankenbaker & Tuite, 2010], location (dominant or non-dominant limb; BF or MH head; proximal or distal) and rehabilitation details of all previous hamstring strain injuries. Limb dominance was determined as the preferred kicking limb. Athletes were considered to be successfully rehabilitated when they returned to pre-injured levels of training and were available for competition [Fuller et al., 2006]. Athletes who were unable to obtain data on all prior hamstring strains from their practitioner were excluded from the study.

EMG recording

Bipolar pre-gelled Ag/AgCl sEMG electrodes (10mm diameter, 25mm inter-electrode distance) were used to record electromyographical activity from the MH and BF. After preparation of the skin via shaving, light abrasion and sterilisation, electrodes were placed on the posterior thigh half way between the ischial tuberosity and tibial epicondyles with electrodes oriented parallel to the line between these two land marks, as per SENIAM guidelines [Hermens et al., 2000]. The reference electrode was placed on the ipsilateral head of the fibula. Muscle bellies were identified via palpation during forceful isometric knee flexion and correct placement was confirmed by observing sEMG activity during active internal and external rotation of the flexed knee to assess cross talk between MH and BF.

Isokinetic dynamometry

Assessment of concentric and eccentric knee flexor strength was performed on a Biodex Systems 3 Dynamometer (Biodex Medical Systems, Shirley, NY). Participants were seated on a custom pad, placed on top of the original seat, which contained two holes at the level of the posterior mid thigh to minimise movement artefact from sEMG electrodes on the dynamometer seat. The hips were flexed at 85˚ from neutral with the lateral epicondyle of the femur carefully aligned with fulcrum of the dynamometer. The tested leg was attached to the lever of the dynamometer via a Velcro strap and padded restraints were fastened across the trunk, hips and mid thigh of the tested leg to isolate movement to the knee joint. The range of motion was set at 5˚-90˚ of knee flexion (0˚=full knee extension) and correction for limb weight was performed.

Three sets of four submaximal contractions of the knee extensors and flexors were performed at $+240^{0}.s^{-1}$ as a warm-up to prepare the participant for maximal effort in the following sets. Concentric testing for both legs consisted of three sets of three consecutive maximum voluntary contractions (MVC) of the knee extensors and flexors at velocities of $+60^0.s^{-1}$ and $+180^0.s^{-1}$ with 30 seconds rest between sets. Athletes were motivated verbally by the investigators to encourage maximal effort throughout the range of motion. Eccentric testing ($-60^0.s^{-1}$ and $-180^0.s^{-1}$) was identical except that only eccentric contraction of the knee flexors was performed by the participant (whereby the knee joint was extended despite active contraction of the knee flexors) and at the completion of each contraction the investigators returned the lever to the starting position. The leg and velocity testing orders were randomised but concentric contractions were always performed before eccentric contractions. All participants were required to attend at least one familiarisation session to ensure consistency of MVCs and one testing session with ≥ seven days between sessions.

Data analysis

Dynamometer torque and lever position data were transferred to computer at 1 kHz and stored for later analysis. Average peak torque was defined as the mean maximal torque of the six highest torque contractions at each velocity. Surface EMG was sampled simultaneously with dynamometer data at 1kHz through a 16-bit PowerLab26T AD recording unit (ADInstruments, New South Wales, Australia) (amplification = 1000 between 10Hz-1kHz; common mode rejection ratio = 110dB) and stored for later analysis where it was fourth order Butterworth filtered between 20-500Hz (24dB roll off) using MATLAB (MathWorks, Natick, Massachusetts) and then full wave rectified using the root-mean-square method across a 100ms window. At each velocity, sEMG data were averaged across a knee joint ROM between 15^o-35^o as this is where deficits in sEMG have been noted previously [Sole et al., 2011]. Data at all velocities was then normalised to the maximal averaged sEMG amplitude recorded during MVCs at $+180^0.s^{-1}$ [Aagaard et al., 2002; Seger et al., 1994; Westing et al., 1991]. For this process the data was separated in tertiles throughout the ROM (15^o-35^o, 35^o -

60°, 60° -80°) and the tertile exhibiting the highest amplitude of sEMG was used for normalisation. Median power frequency was determined from the non-rectified sEMG signal via Fast Fourier transform with Hann window function applied [Aagaard et al., 2000] across the entire ROM using LabCart 7.3 (ADInstruments, New South Wales, Australia) with 1Hz frequency resolution. This resulted in 1.08 and 0.36 second time epochs for analysis of contractions at ± 60 and $180^0.s^{-1}$ respectively. Median power frequency was analysed over a larger ROM (15-80°) than sEMG activity to allow for a valid estimation of frequency. Median power frequency was defined as the frequency at which 50% of total power was reached for each time epoch.

Statistical analysis

Data were analysed using JMP version 10.0 Pro Statistical Discovery Software (SAS Inc). In the primary analysis, comparisons were made between the injured and uninjured limbs in the INJ group and between dominant and non-dominant limbs in the UI group. Dependent variables were compared using one tailed paired t tests for both groups to allow an equal likelihood for finding significant differences between limbs [Lee et al., 2009]. Data are presented as means and standard deviation. Bonferroni corrections were performed to account for four comparisons made for each dependent variable across the velocities used, with significance set at $p < 0.0125$. In the confirmatory secondary analysis independent t tests for unequal variance were used to compare the between limb differences of the dependent variables in the INJ (uninjured limb minus injured limb) and UI groups (dominant limb minus non-dominant limb) as assumptions for equal variance between groups was not met. For the secondary analysis significance was set at $p < 0.05$ and data are presented as mean differences and 95% confidence intervals. To assess the magnitudes of the differences for the primary and secondary analyses Cohen's d was calculated to report effect size (ES).

RESULTS

Participants

There was no significant difference between the UI and INJ groups with respect to age, height or body mass. The details of injury histories of all athletes from the INJ group can be found in Table 1. All athletes from the INJ group reported largely standard rehabilitation progression (i.e. [Heiderscheit et al., 2010]) guided by their physiotherapist.

Average peak torque

There were significant differences in average peak torque between limbs in the INJ group, with the previously injured limb weaker at all contraction modes and velocities (Figure 1a & Table 2). No differences in average peak torque were noted between limbs in the UI group (Figure 1b & Table 2). Between limb differences in torque were significantly greater in the INJ group compared to the UI group at all contraction modes and velocities, except for concentric contractions at $180^{0}.s^{-1}$ (Table 5).

sEMG activity

Biceps femoris long head electromyographical activity was significantly lower in the previously injured limb compared to the contralateral uninjured limb in the INJ group during eccentric contractions but not concentric contractions (Figure 2a & Table 3). There were no differences between limbs in the INJ group for MH electromyographical activity at any contraction mode or velocity (Figure 3a & Table 3). In the UI group there were no differences in activation between limbs for BF (Figure 2b & Table 3) or MH (Figure 3b & Table 3) at any contraction mode or velocity. Between limb differences in electromyographical activity were greater in the INJ group compared to the UI group only for

BF at $-180^0.s^{-1}$ (Table 5). All other between limb differences in electromyographical activity were similar between INJ and UI groups, although a trend existed at $-60^0.s^{-1}$ (Table 5).

Median power frequency

One participant from the INJ group was a clear outlier (median power frequency was more than 3 standard deviations above the mean for eccentric contractions) and was removed from analysis. There were no differences in median power frequency at any velocity between legs in the INJ group for BF or MH (Table 4). A similar lack of differences was noted at all velocities for the UI group for BF or MH median power frequency (Table 4). The between limb differences in median power frequency did not differ between the INJ and UI groups at any contraction mode or velocity (Table 5).

DISCUSSION

It is accepted that a prior hamstring strain injury results in maladaptation of the previously injured tissue [Opar et al., 2012]. Whilst a number of muscular maladaptations have been reported previously [Brockett et al., 2004; Croisier et al., 2002; Lee et al., 2009; Silder et al 2008; Silder et al., 2010; Worrell et al., 1991], the impact of a prior hamstring strain injury on neural function has been scarcely examined [Sole et al., 2011]. The current study used between limb comparisons of normalised sEMG activity and median power frequency to determine differences in neural hamstring function between injured and uninjured limbs. This method eliminates a number of confounding factors by ensuring that muscle lengths and electrode locations are identical between trials within and between limbs and has been used extensively to assess relative muscle activation in maximal concentric and eccentric contraction [Aagaard et al., 2002; Seger et al., 1994; Westing et al., 1991].

From the INJ group in the current study, the novel findings were that the previously injured limb, when compared to the contralateral uninjured limb displayed 1) a lower level of sEMG activity specifically in the previously injured muscle (BF) during slow and fast eccentric contractions (Figure 2a & Table 3); and; 2) there was no difference in the median power frequency in either the previously injured BF or uninjured MH (Table 4). Furthermore, lower levels of strength were observed across all contraction modes and velocities in the injured limb compared to the uninjured limb in the INJ group (Figure 1a). In contrast the control group showed no differences between dominant and non-dominant limbs in any of the tested variables indicating there is no influence of limb dominance (Figure 1b, 2b, 3b; Table 2, 3, 4). These findings were mostly supported by confirmatory analysis which indicated that the between limb differences in knee flexor torque at all contraction modes and velocities, except for the fastest concentric contractions, and BF sEMG during fast eccentric contraction was greater in INJ group compared to the UI group (Table 5).

This study is, to our knowledge, the first to identify lower levels of sEMG activity specifically in the previously injured BF muscle compared to a contralateral uninjured BF. Recent evidence examining a similar phenomenon did not find a muscle specific, between limb differences in sEMG activity following a hamstring strain injury [Sole et al., 2011]. The discrepancies between the findings from the current study and the previous study by Sole and colleagues (2011) work may be attributed to the inclusion of athletes with bilateral injury histories which may have contributed to the lack of difference in sEMG activity between the injured leg and the contralateral control limb in earlier work [Sole et al., 2011]. However our finding that, when comparing BF sEMG across the two groups, only during eccentric contractions at $-180^{0}.s^{-1}$ was the between limb difference significantly greater in the INJ compared to the UI group, somewhat confirms a previous similar finding by Sole et al.

(2011). Whilst there was no significant between limb difference in BF sEMG during eccentric contractions at $-60^{0}.s^{-1}$ when comparing the two groups in the current study, the large ES (d=0.74) indicates that a significant difference may have existed with an increased sample size.

Reductions in muscle activation during eccentric contractions is due to reduced motor unit recruitment and/or firing rates [Webber & Kriellaars 1997] which impact upon maximal torque generation capabilities. Following hamstring strain injury it has been suggested that the purpose of reduced hamstring activation would be to protect the damaged tissue from high force contraction [Opar et al., 2012]. Hamstring strain injuries themselves are characterised by acute pain in the posterior thigh [Verrall et al., 2001] with reports of chronic pain not uncommon [Croisier et al., 2002; Jönhagen et al., 1994] and this has the potential to result in long-term re-organisation of the nervous system at the spinal and supraspinal levels [Mense, 2003]. The current study confirms that, even in athletes who have been successfully rehabilitated and have returned to competition, sEMG activity of the BF remains suppressed. This would indicate that, for the current cohort, contemporary rehabilitation practices were unsuccessful at addressing deficits in the activation of BF. This is of concern from the perspective of HSI recurrence given submaximal stimulation of *in-situ* animal muscle reduces the amount of stress that muscle can withstand before the occurrence of stretch induced failure [Garrett et al., 1987]. This may indicate that the previously injured BF is unable to withstand the same amount of stress before failure compared to an uninjured muscle, thus increasing the likelihood of re-injury. The observation of no between limb differences in median power frequency in the INJ group suggests that prior hamstring strain injury may not impact upon average muscle fibre conduction velocity [Linnamo et al., 2000]. It should also be acknowledged that a number of other factors also influence the median power frequency of the electromyographical signal and further investigation examining these factors discretely is warranted.

It has been proposed previously that the suppression of hamstring muscle activation following hamstring strain injury has the potential to limit adaptation during the rehabilitation process [Opar et al., 2012]. This model suggests early to middle stage rehabilitation for hamstring strain injury typically involves avoidance of excessive stretching of the involved tissue and submaximal exercise performed through limited range of motion in an attempt to prevent proliferation of scar tissue [Heiderscheit et al., 2010]. Such an approach might be expected to result in a reduction of in-series sarcomeres [Williams & Goldspink, 1978] and induce atrophy [Silder et al., 2008] potentially reducing the optimal length of the hamstrings [Brockett et al., 2004] which would be unfavourable given the need for the hamstrings to generate high eccentric forces at relatively long muscle lengths in running [Thelen et al., 2005]. Late stage rehabilitation involving more forceful eccentric contractions at long muscle lengths might be expected to overcome these maladaptations [Lynn & Morgan, 1994], however, suppression of hamstring activation, as reported in the current study, would reduce the stimulus the previously injured muscle is exposed to, thus potentially compromising the adaptive response to rehabilitation. The present study suggests that chronic lowering of hamstring activation following strain injury could sabotage the rehabilitation process. Still, the full impact of prior hamstring strain injury on neurological control of the involved muscle/s and impact on adaptation requires further attention.

The current study found strength at all velocitiess and contraction modes was lower in the previously injured limb compared to the uninjured limb. Previous work has found eccentric but not concentric declines in strength [Lee et al., 2009] or greater eccentric deficits (22-24%) compared to concentric deficits (10-11%) following hamstring strain injury [Croisier et al., 2002]. As muscle shortening velocity is known to influence maximal tension generating capacity [Fenn & Marsh, 1935] the different concentric velocities used in previous work may explain the inconsistent findings for this contraction mode. In line with this, the percentage difference in strength between previously injured and uninjured limbs tested at a comparable velocities ($+60^0.s^{-1}$) is similar in the current study (10.9%) and previous work (11%) [Croisier et al., 2002]. The much larger decline in eccentric strength reported elsewhere [Croisier et al., 2002] is less likely to be due to differences in eccentric testing velocities as eccentric strength is largely unaffected by lengthening velocity. It may be, however, explained by differences in rehabilitation practices of the respective cohorts given the greater appreciation for eccentric conditioning in hamstring strain injury prevention in recent times [Petersen et al., 2011]. Perhaps not surprisingly, more recent studies have reported smaller eccentric strength differences in the order of 13% [Lee et al., 2009], which is comparable to the 10.9-12.5% differences reported in the current study.

Uniformly lower concentric and eccentric strength, as observed in the current study, would be expected if strength was determined solely from muscle cross sectional area and volume, given the noted atrophy of BF following hamstring strain injury [Silder et al., 2008]. Interestingly, sEMG activity was lower only during eccentric contractions, despite lower strength across contraction modes and velocities. This suggests that reductions in BF activity contribute to prolonged eccentric, but not concentric, weakness following hamstring strain injury. It might therefore be expected that the decline in eccentric strength following hamstring strain injury would be of a greater relative magnitude than concentric strength, but this is not supported by the current data. It may be that other muscles which contribute to knee flexion, that were not examined in the current study, such as the short head of biceps femoris, gastrocnemius and sartorius, increase their involvement during maximal eccentric contraction in a previously injured leg to help overcome the limitation in sEMG activity of BF. Indeed, compensatory hypertrophy of the short head of biceps femoris has been reported previously [Silder et al., 2008], suggesting hamstring strain injury may lead to increased use of uninjured musculature, however further examination of this area is warranted.

There are some limitations in the present study's methodology. The retrospective nature of the study does not allow for the determination of whether the reduction in sEMG activity of BF is the cause of or the result of injury. Prospective studies are required to determine if low levels of BF activity elevates the risk of sustaining a future hamstring strain injury. It should be noted, however, that whilst prospective studies have determined that a between limb eccentric strength difference of approximately 4.5% is associated with future hamstring strain injury [Suguiura et al., 2008], post-injury eccentric weakness is reported to be between 13-24% [Croisier et al., 2002; Lee et al, 2009], suggesting hamstring injury enhances eccentric knee flexor weakness, most probably via neuromuscular maladaptation. Also using the maximal activation data from the fastest concentric movement velocity ($+180^{o}.s^{-1}$) to normalise the sEMG data as per previous investigations [Aagaard et al., 2000] has the potential to mask any between-limb differences in sEMG activity at this velocity, however given the important nature of eccentric strength in hamstring strain injury aetiology, sEMG activity during eccentric contraction was of most interest. Finally, the power of the current study may have been too small to detect between limb differences in variables not determined to be significantly different in current study. We have reported ES for all comparisons (Table 2-4) to further illustrate the strength of the between limb differences. The ES data suggests that, in particular, the study may have been underpowered to detect differences in the electromyographical activity of the MH and the median power frequency between injured and uninjured limbs. A larger sample size should be a consideration for future work, notwithstanding the difficulty in recruiting athletes for the INJ group.

In conclusion, this study is the first to report that athletes with a history of unilateral hamstring strain injury display reductions in the sEMG activity of a previously injured BF during eccentric contractions and no difference in the median power frequency of either hamstring head during concentric or eccentric contractions. Furthermore strength was suppressed during both contraction modes in the injured limb compared to the uninjured limb. Previous hamstring strain injury may result in between limb alterations in neuromuscular function and rehabilitation practices need to consider the recovery of strength and activation during eccentric contractions as markers of successful rehabilitation as this may assist in reducing the incidence of hamstring strain injury recurrence.

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CONFLICT OF INTEREST

NA

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TABLES

Table 1. Hamstring strain injury information for most recent injury for athletes recruited to the injured group.

Subject	Time since HSI (months)	Rehabilitation duration (weeks)	Location	Total HSIs sustained
1	2	4	Dominant, Proximal BF	1
2	3	4	Non dominant, Proximal BF	3
3	8	4	Non dominant, Distal BF	1
4	7	2	Non dominant, Proximal BF	2
5	3	4	Dominant, Proximal BF	4
6	5	2	Non dominant, Distal BF	2
7	18	4	Non dominant, Distal BF	1
8	4	4	Non dominant, Proximal BF	2
9	2	5	Non dominant, Proximal BF	2
10	5	3	Non dominant, Proximal BF	4
11	2	2	Dominant, Proximal BF	2
12	3	6	Non dominant, Distal BF	4

13	7	3	Non dominant, Proximal BF	3

HSI, hamstring strain injury; BF, biceps femoris. All prior injuries were confined to the same leg and muscle as most recent injury however location on muscle (proximal or distal) differed in some instances.

Table 2. Knee flexor torque of athletes with and without a history of unilateral hamstring strain injury during concentric and eccentric contraction.

Movement velocity ($^{o}.s^{-1}$)	Injured Group			
	Injured limb	Uninjured limb	p	ES
+180	109.29 (± 13.14)	118.64 (± 12.47)	0.0036*	0.78
+60	132.00 (± 21.28)	146.01 (± 15.49)	0.0013*	0.70
-60	166.76 (± 30.19)	185.02 (± 25.22)	0.0007*	0.57
-180	163.82 (± 30.43)	184.37 (± 22.33)	0.0007*	0.74
		Uninjured group		
	Dominant limb	Non-dominant limb	p	ES
+180	127.13 (± 22.12)	122.73 (± 21.24)	0.0608	0.20
+60	154.93 (± 24.27)	151.59 (± 25.10)	0.1558	0.14
-60	199.71 (± 31.46)	198.68 (± 33.30)	0.4341	0.03
-180	194.84 (± 25.97)	194.60 (± 28.84)	0.4828	0.01

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean (± standard deviation). *Significance was set at $p < 0.0125$. Cohen's d was used to calculate effect size.

Table 3. Normalised electromyographical activity of the biceps femoris long head and medial hamstrings of athletes with and without a history of unilateral hamstring strain injury during concentric and eccentric contraction.

Movement velocity ($^{o}.s^{-1}$)	Injured group							
	Biceps femoris				Medial hamstrings			
	Injured limb	Uninjured limb	P	ES	Injured limb	Uninjured limb	P	ES
+180	0.96 (±0.06)	0.99 (± 0.02)	0.0894	[a]	0.95 (± 0.07)	0.98 (± 0.06)	0.0622	[a]
+60	0.89 (± 0.20)	0.93 (± 0.12)	0.2255	0.18	0.91 (± 0.23)	0.96 (± 0.13)	0.2412	0.09
-60	0.58 (± 0.17)	0.71 (± 0.17)	0.0025*	0.47	0.58 (± 0.21)	0.64 (± 0.12)	0.1296	0.06
-180	0.53 (± 0.20)	0.66 (± 0.18)	0.0003*	0.58	0.52 (± 0.22)	0.61 (± 0.15)	0.0770	0.26
	Uninjured group							
	Biceps femoris				Medial hamstrings			
	Dominant limb	Non-dominant limb	P	ES	Dominant limb	Non-dominant limb	P	ES
+180	0.97 (± 0.06)	0.99 (± 0.02)	0.1602	[a]	0.94 (± 0.11)	0.94 (± 0.12)	0.4444	[a]
+60	0.95 (± 0.16)	0.97 (± 0.18)	0.2703	-0.12	0.93 (± 0.26)	0.97 (± 0.23)	0.2890	-0.16
-60	0.70 (± 0.21)	0.69 (± 0.17)	0.4275	0.05	0.64 (± 0.25)	0.67 (± 0.16)	0.3077	-0.14
-180	0.60 (± 0.26)	0.61 (± 0.14)	0.4052	-0.05	0.56 (± 0.23)	0.59 (± 0.15)	0.2538	-0.15

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean (± standard deviation). *Significance was set at $p < 0.0125$. Cohen's d was used to calculate effect size (ES). [a] ES for electromyographical activity could not be calculated given the use of this data in the normalisation process.

Table 4. Median power frequency of the biceps femoris long head and medial hamstrings of athletes with and without a history of unilateral hamstring strain injury during concentric and eccentric contraction.

Movement velocity ($^{o}.s^{-1}$)	Injured group							
	Biceps femoris				Medial hamstrings			
	Injured limb	Uninjured limb	P	ES	Injured limb	Uninjured limb	P	ES
+180	61.70 (± 5.82)	64.70 (± 9.00)	0.1005	0.40	67.75 (± 6.25)	71.15 (± 8.34)	0.1680	0.47
+60	60.30 (± 6.64)	62.11 (± 7.80)	0.2220	0.25	58.70 (± 7.48)	62.78 (± 9.57)	0.1655	0.48
-60	64.78 (± 7.83)	66.92 (± 9.35)	0.2530	0.24	62.85 (± 9.63)	66.03 (± 15.53)	0.2950	0.25
-180	63.04 (± 6.38)	68.03 (± 13.73)	0.1030	0.50	64.68 (± 9.42)	70.43 (± 18.49)	0.2140	0.41
	Uninjured group							
	Biceps femoris				Medial hamstrings			
	Dominant limb	Non-dominant limb	P	ES	Dominant limb	Non-dominant limb	P	ES
+180	63.57 (± 11.35)	62.82 (± 7.41)	0.3580	0.08	74.84 (± 13.24)	72.04 (± 7.71)	0.2460	0.26
+60	62.71 (± 7.60)	62.84 (± 7.51)	0.4670	-0.02	69.44 (± 10.44)	66.28 (± 6.28)	0.1025	0.37
-60	63.25 (± 9.37)	63.38 (± 6.89)	0.4620	-0.02	70.24 (± 15.52)	66.42 (± 13.50)	0.2075	0.26
-180	64.22 (± 12.62)	66.05 (± 8.26)	0.2400	-0.17	70.21 (± 18.21)	71.05 (± 13.62)	0.4275	-0.05

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean (± standard deviation). Significance was set at $p < 0.0125$. Cohen’s d was used to calculate effect size (ES).

Table 5. Comparison of between limb differences in knee flexor torque and normalised electromyographical activity and median power frequency of the biceps femoris long head and medial hamstrings in athletes with and without a history of hamstring strain injury, during concentric and eccentric contraction.

Movement velocity ($^{o}.s^{-1}$)	Knee flexor torque							
	Injured group	Uninjured group	P	ES				
+180	9.34 (3.03 to 15.66)	4.40 (-1.33 to 10.13)	0.2208	0.48				
+60	14.01 (5.98 to 22.02)	3.34 (-3.48 to 10.16)	0.0379*	0.83				
-60	18.26 (8.68 to 27.84)	1.03 (-12.10 to 14.17)	0.0312*	0.85				
-180	20.55 (9.72 to 31.37)	0.24 (-11.56 to 12.04)	0.0110*	1.03				
	Normalised electromyographical activity							
	Biceps femoris				Medial hamstrings			
	Injured group	Uninjured group	P	ES	Injured group	Uninjured group	P	ES
+180	0.03 (-0.01 to 0.07)	-0.01 (-0.05 to 0.02)	0.0919	[a]	0.03 (-0.01 to 0.06)	0.00 (-0.08 to 0.07)	0.4070	[a]
+60	0.04 (-0.07 to 0.15)	-0.03 (-0.11 to 0.06)	0.3271	0.41	0.05 (-0.10 to 0.21)	-0.04 (-0.17 to0.10)	0.3661	0.36
-60	0.13 (0.05 to 0.22)	0.01 (-0.09 to 0.11)	0.0542	0.74	0.07 (-0.06 to 0.20)	-0.03 (-0.15 to 0.09)	0.2395	0.46
-180	0.13 (0.07 to 0.19)	-0.02 (-0.15 to 0.12)	0.0473*	0.82	0.09 (-0.04 to 0.21)	-0.03 (-0.13 to 0.07)	0.1210	0.61
	Median power frequency							

	Biceps femoris				Medial hamstrings			
	Injured group	Uninjured group	P	ES	Injured group	Uninjured group	P	ES
+180	3.00 (-1.86 to 7.85)	0.74 (-3.55 to 5.04)	0.4570	0.29	3.40 (-4.04 to 10.84)	2.80 (-5.71 to 11.30)	0.9078	0.04
+60	1.81 (-3.21 to 6.84)	-0.12 (-3.24 to 3.00)	0.4835	0.37	4.08 (-4.75 to 12.90)	3.16 (-1.94 to 8.26)	0.8462	0.08
-60	2.15 (-4.72 to 9.01)	-0.13 (-3.02 to 2.75)	0.5122	0.27	3.18 (-9.44 to 15.80)	3.82 (-5.93 to 13.57)	0.9315	-0.03
-180	4.99 (-3.18 to 13.15)	-1.83 (-7.26 to 3.59)	0.1442	0.60	5.76 (-9.63 to 21.14)	-0.84 (-10.52 to 8.85)	0.4377	0.31

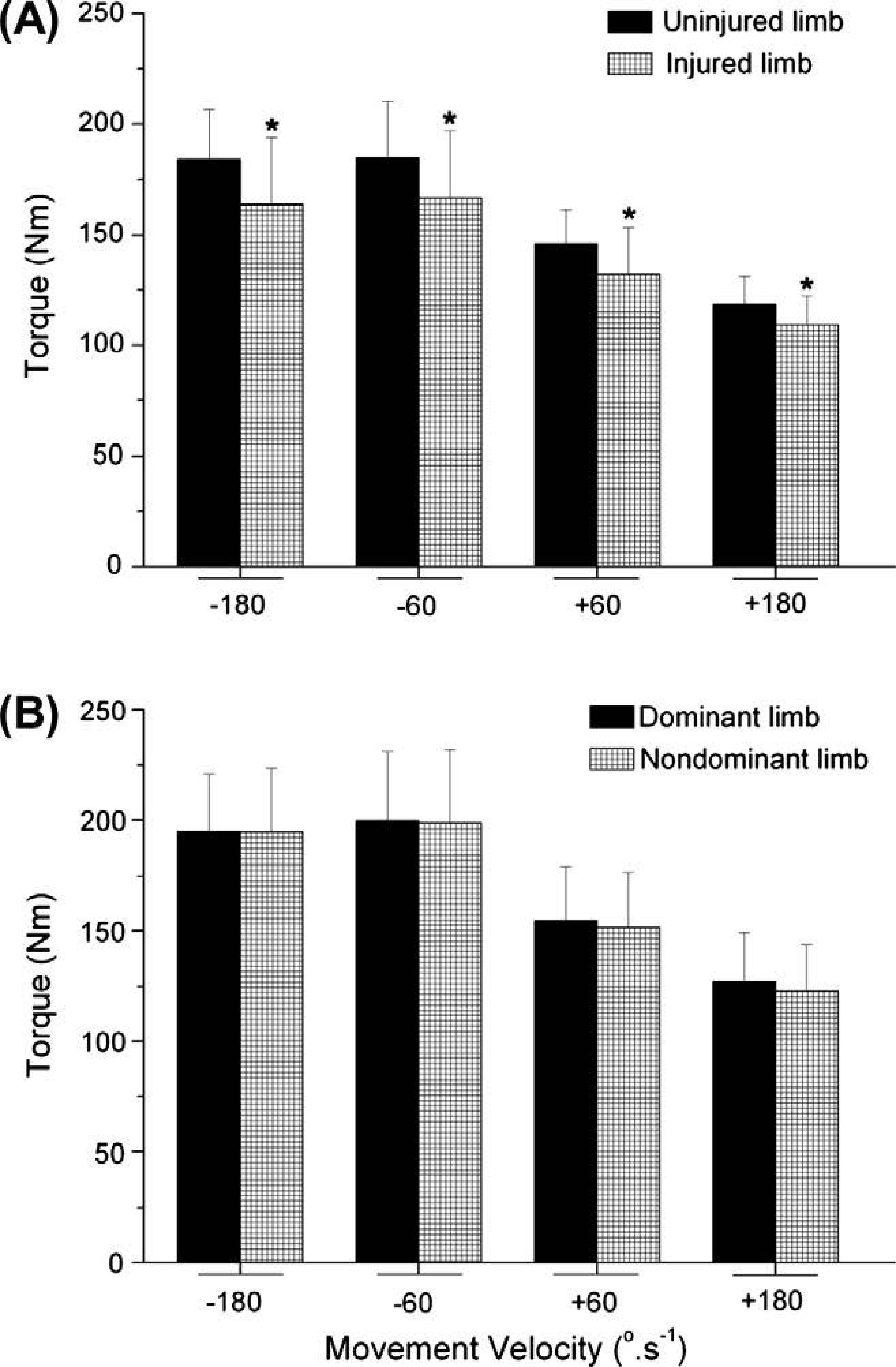
Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean differences (95% confidence intervals). *Significance was set at $p < 0.05$. Cohen's d was used to calculate effect size (ES). [a] ES for electromyographical activity could not be calculated given the use of this data in the normalisation process.

FIGURE LEGENDS

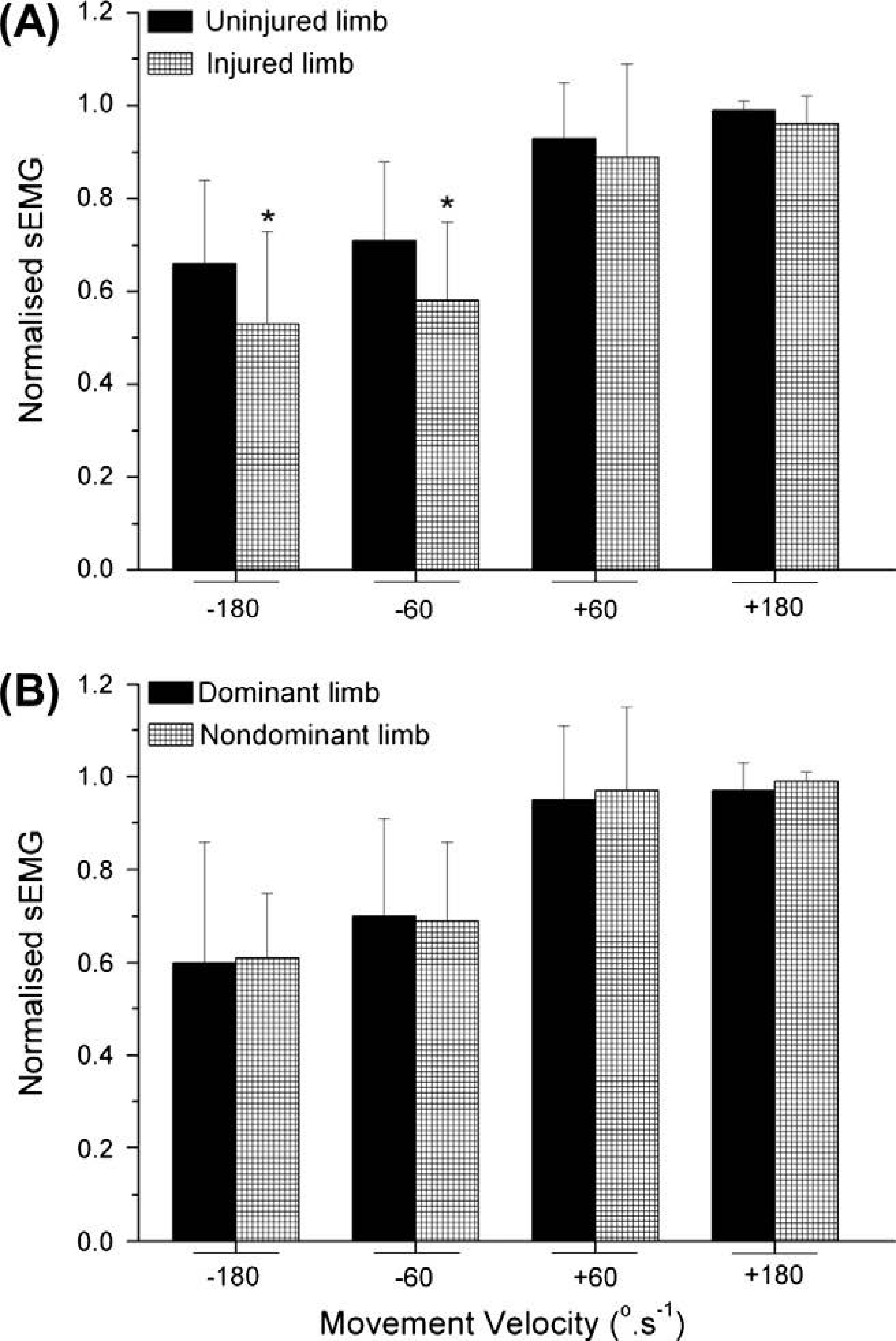
Figure 1: Knee flexor average peak torque at four different isokinetic velocities from the A) injured athletes and B) uninjured athletes. Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Error bars display standard deviation. * $p < 0.0125$ injured vs uninjured limbs.

Figure 2: Biceps femoris long head normalised surface electromyography (sEMG) at four different isokinetic velocities from the A) injured athletes and B) uninjured athletes. Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Error bars display standard deviation.* $p < 0.0125$ injured vs uninjured limbs.

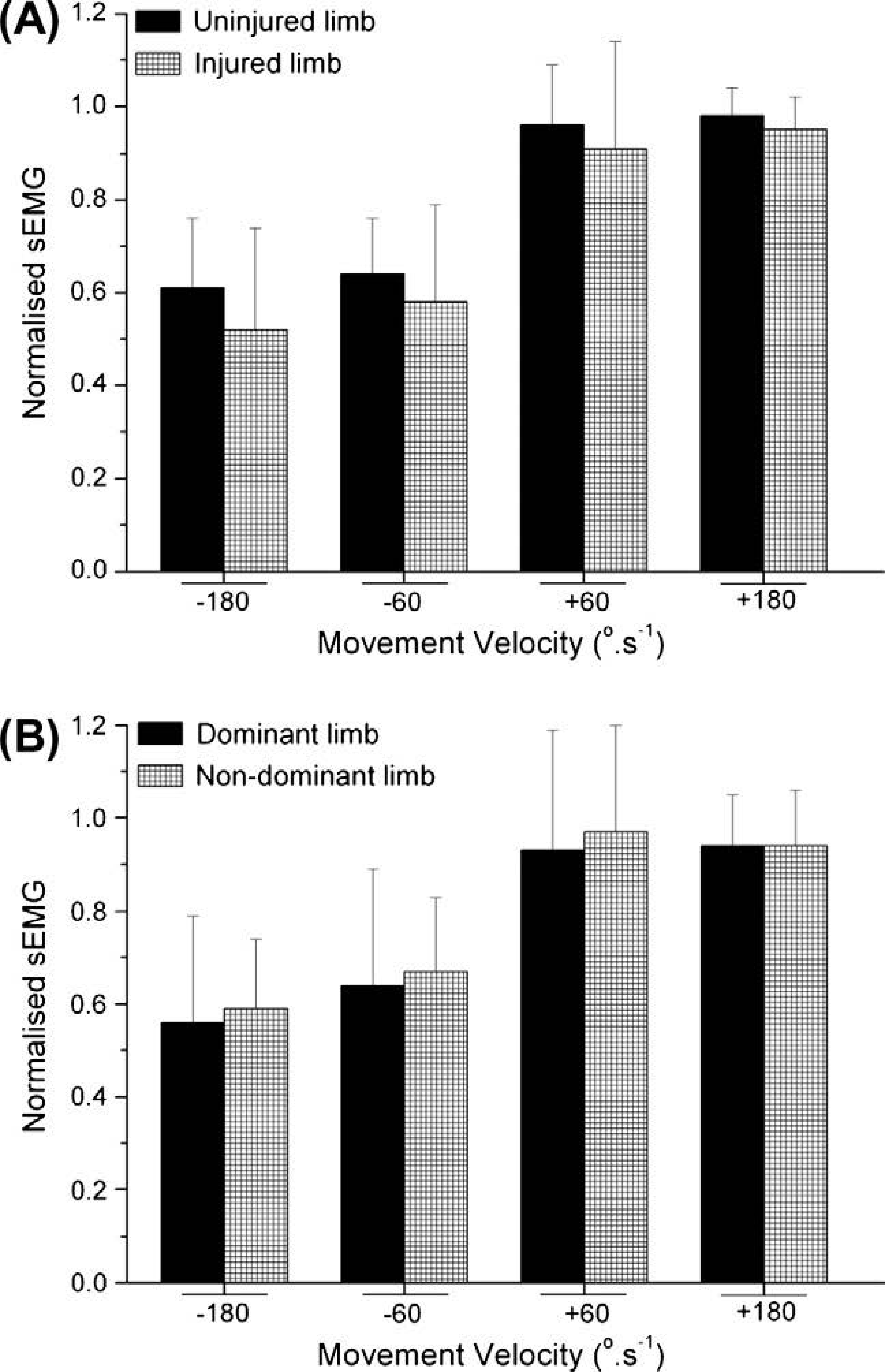
Figure 3: Medial hamstring normalised surface electromyography (sEMG) at four different isokinetic velocities from the A) injured athletes and B) uninjured athletes. Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Error bars display standard deviation.



(A)
Uninjured limb
Injured limb
Torque (Nm)
0
50
100
150
200
250
*
*
*
*
-180
-60
+60
+180
(B)
Dominant limb
Nondominant limb
Torque (Nm)
0
50
100
150
200
250
-180
-60
+60
+180
Movement Velocity (°.s⁻¹)



(A)
Uninjured limb
Injured limb
Normalised sEMG
0.0
0.2
0.4
0.6
0.8
1.0
1.2
*
*
-180
-60
+60
+180
(B)
Dominant limb
Nondominant limb
Normalised sEMG
0.0
0.2
0.4
0.6
0.8
1.0
1.2
-180
-60
+60
+180
Movement Velocity (°.s^{-1})



(A)
Uninjured limb
Injured limb
Normalised sEMG
0.0
0.2
0.4
0.6
0.8
1.0
1.2
-180
-60
+60
+180
Movement Velocity (°.s-1)
(B)
Dominant limb
Non-dominant limb
Normalised sEMG
0.0
0.2
0.4
0.6
0.8
1.0
1.2
-180
-60
+60
+180
Movement Velocity (°.s-1)