

# Estimates of Stress Between the Hamstring Muscles

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## SUMMARY

**Background.** The cause of muscle damage and injury is often attributed to large strains. Studies have suggested that strain is the principal cause of muscle damage rather than force.

**Results.** In this paper we show that force is the principal cause of muscle damage whereas strain is a means of increasing force. The subtle difference has important implications, as many studies use strain as an indicator for injury risk. In addition, we show that the data better supports a theory of stress as the principal cause of injury, rather than force alone, and aligns with both the myofibril and observational data.

**Conclusions.** The implications of a stress-based model of injury is discussed within the paper.

## KEY WORDS

*Force; strain; stress; damage; injury; hamstring.*

## INTRODUCTION

Hamstring injuries can be considered to result from an indirect mechanism (1), though two specific mechanisms of hamstring injury appear to exist and are referred to as stretching and high-speed (forceful) (2). This classification arises from observations in dancers where hamstring strain injuries occur at low forces but long lengths (3) and injuries in sprinters that occur at short lengths but with high forces (4). Thus, these two posited mechanisms can be reduced to either excessive strain or excessive force. The mechanism also determines the muscle susceptible to injury. The semimembranosus (SM) is prone to injury under excessive strain (3) whereas the biceps femoris (BF) is at risk in forceful scenarios, such as sprinting (4). This suggests a unique quality exists dependent on the mechanism that renders each muscle susceptible to injury.

However, the notion of two separate mechanisms for strain injury is at odds with the experimental research in myofibrils, which suggests strain is the principal cause of injury (5,6). In accordance, simulation studies have found the biceps femoris is most lengthened whilst sprinting appearing to explain its susceptibility (7-10). Yet, if lengthening was the determiner of injury, then isometric contractions should not induce damage, but it is reported to do so (10). This is not the case. Likewise, although the biceps femoris is most lengthened during sprinting, its absolute length

is small. At larger absolute strains the semimembranosus is more commonly injured (3). Another variable must exist to explain this discrepancy.

## The role of myofiber strain in injury mechanics

The notion of strain causing injury rather than force arises from a seminal study in myofibrils that compared strain magnitudes of 12.5% to 25% with either high or low levels of force, achieved by manipulating the delay between onset and strain (5). Lieber and Friden found that regardless of force, damage was always greater in groups with larger strains, yet similar between muscles exposed to different forces. However, according to Hook's Law (Eq.1) the spring constant ( $-k$ ) means force ( $F$ ) is proportional to strain ( $s$ ). Thus, the high strain group intended to have low force could in fact have had greater force than the low strain group intended for high force. This would confound any attempt to attribute injury to strain or force.

It was reported that force was 40% greater in myofibrils in the high force group compared to the low force group but was only reported for myofibrils with a large strain magnitude. No results of force are given for the low strain group. Through Eq. 1, it is possible to estimate the forces in myofibrils of different strains and intended forces from Lieber and Friden. Therefore, the aim of this investigation was to

conduct a secondary analysis of their results to estimate the force in myofibrils of different strains and force (where the high force group correspond to the delayed onset group in Lieber and Friden). It is hypothesised that force in the high strain/low force group will be greater than the low strain/high force group.

## METHODS

Myofibril forces for the high strain groups were calculated using data provided by Lieber and Friden. For the high strain/low force group, absolute force was obtained using *WebPlotDigitizer* (11) and Figure 2A of Lieber and Friden, and then converted from grams to newtons using a ratio of 102:1. This showed myofibrils of the low force/high strain group produced 14 N of force. For the high force/low strain group, Lieber and Friden state force is 1.4 times greater than the low force group with 25% strain equalling 20 N of force. Myofibril forces for the low strain groups were calculated using Hook's Law (Eq. 1), which requires the absolute strain and the spring constant. The nominal length for each fibre is 55 mm so absolute strain can be calculated as 25% and 12.5% of this value. The large strain group was calculated to be 13.75 mm and the low strain group this equalled 6.875 mm. Spring constants for myofibrils were calculated using Hook's Law, by dividing force by the absolute strain in each of the 25% strain groups. As myofibrils were homogenous, the spring constants are expected to be the same for groups within high or low force regardless of the strain. For the low force group ( $k_{ES}$ ), the spring constant was 1.02 N/mm, and for the high force group the ( $k_{LS}$ ) was 1.45 N/mm. Thus, force in the low strain groups of high and low force were obtained by multiplying the absolute strain of 6.875 mm by their respective spring constant, as per table I. All methods were done in accordance with the ethical standards of the journal (12).

## RESULTS

**Table 2** shows myofibril forces are greater in in the high strain/low force group compared to the low strain/high force group (14 N and 10 N respectively). The mean differ-

ence in force between timings is ~4.5 N, whereas the mean difference in force between strains is ~8.5 N. The outcomes are displayed in **table II**.

## DISCUSSION

Friden and Lieber concluded strain is the principal cause of injury based on finding a significant effect of strain on force reductions whereas onset time (used to manipulate myofibril force) had no significant effect on force. However, the forces within these groups were not reported. The results of this analysis show that at high strains, the group intended to represent low force had 4 N greater force than the group intended for higher force but at low strains. The suggestion that strain is responsible for injury rather than force is incorrect, as strains leads to greater forces. The additional 4 N of force from greater strains is considerable given the peak tetanic tension in myofibrils is ~13N, and suggests that lengthening, on average, increased force by 31% of its peak force than when manipulating the force production through onset time.

As strain created greater forces, then it may seem that Lieber and Friden were correct in their conclusion that lengthening causes injury. Whilst we agree with Lieber and Friden that greater strains lead to greater muscle damage, stating it is not the result of high force is incorrect and not supported by the data. Instead, lengthening is a means of achieving greater forces. Under maximal conditions, and homogenous myofibrils, such as those used by Friden and Lieber, the larger strains will cause greater forces and thus injury. But this relationship does not hold between heterogeneous muscles because of differences in passive tension and active force production. Therefore, identifying force as the cause of injury is a subtle but important difference when comparing muscles.

For example, multiple studies have founded their work on Lieber and Friden and used peak strain to determine the hamstring muscle vulnerable to injury (7-10, 13-15). Yet many of these papers show active force differs between the muscles and therefore do not indicate the muscle most susceptible to injury (7-9). Without understanding the

**Table I.** Calculations to estimate stress.

Strain		Force	
Relative Strain (%)	Absolute strain (mm)	Low force (N)	High force (N)
25	$S_{25} = 55 \times 0.250$	A = 14	B = A × 1.40
12.5	$S_{12.5} = 55 \times 0.125$	C = $k_{ES} \times S_{12.5}$	D = $k_{LS} \times S_{12.5}$

**Table II.** Calculated strain and force values

Strain		Force (N)	
Relative Strain (%)	Absolute strain (mm)	Low force group	High force group
25	13.75	14	20
12.5	6.875	7	10

underpinning cause of injury it's possible the protocols put forward for investigation and practice are entrenched on a false understanding. For example, Guex claimed the optimal exercise for hamstring strengthening does not exist with limited strain being one of the reasons (16). This criticism has been echoed for the Nordic hamstring curl also (17). Yet, these conclusions may still be limited to myofibrils as the largest contributor to force when sprinting is the semi-membranosus, and not the commonly injured biceps femoris (7,9,18). Sprint simulations have also combined force and strain through Work to explain the biceps femoris' vulnerability, but this too has failed. Despite force seemingly causing injury in myofibrils, the current outcomes from sprint simulations do not identify the vulnerability of the biceps femoris in this common injury inducing movement. A new perspective is warranted to converge the observed data from simulations to that in myofibril experiments.

### Stress as an explanation for injury

It is well established in Newtonian mechanics that materials fracture under excessive tensile stress (19). Stress ( $\sigma$ ) is the measure of the internal force ( $F$ ) acting in a localised area ( $A$ ), and can be estimated by dividing the muscle force by its cross-sectional area (Eq. 2).

From the perspective that muscle is a biological material, the cause of fracture should not differ. Thus, stress is likely to be the principle cause of injury. In contrast to reported outcomes, this requires not only the forces to be considered but the area of the muscle too. In myofiber research the cross-sectional area (CSA) of homogenous myofibrils is expected to be similar and therefore differences in force would be proportional to stress, such that the force induced by strain would appear as the determining factor. As muscles differ in CSA, this does not hold true.

Stress as the cause of injury can explain why the myotendinous junction (MTJ) is a prevalent location for injury as the area lessens as the muscle tapers to the free tendon (20). This has been alluded to by Storey *et al.*, but an explicit investigation has not been performed. Earlier studies have investigated the aponeurosis size in relation to eccentric strength ( $r = 0.24$ ;  $p > 0.2$ ) but not regarding injury incidence (21).

The semimembranosus is the largest producer of force whilst sprinting, but a large CSA would reduce the stress. Conversely, a small CSA in biceps femoris would increase its stress and propensity for injury. The aim of this study was to identify whether peak stress is greatest in the biceps femoris by approximating the stress at each hamstring's MTJ during sprinting using previous simulations and morphology data. The hypothesis was that peak MTJ stress will be greatest in the biceps femoris.

## METHODS

According to Eq. 2, the peak stress at each hamstring's MTJ can be derived by their respective dividing peak force by the CSA of each MTJ. Estimates of each muscles MTJ CSA was obtained from the results of Storey (2016). A two-way 95% CI for each MTJ CSA was estimated from the data provided by Storey. The MTJ area data was obtained from different participants to those used in simulations which may reduce the validity of these estimates; however, aponeurosis area is not related to muscle size or area (Evangelidis *et al.* 2015) and so the average MTJ areas from Storey are a fair approximation of the participant populations used in the simulation studies.

The peak force for each hamstring muscle was obtained by combining the results of simulation research that used data from sprinting. Sprint data was used as this is a common and ubiquitous action during injury (22) and appears to produce the greatest hamstring activity (23). Sprint simulations were identified through prior knowledge and confirmed via a PubMed search using the terms *sprint\**, *Simulation*, and *hamstring* connected with the 'AND' Boolean operator. To be included, simulation studies had to identify the peak force for all 3 biarticular hamstrings (SM, semitendinosus [ST], and BF) whilst performing high-speed sprinting.

To account for the between study variability in peak force estimates, a random effects meta-analysis (24) was performed using R statistical software and peak force from each simulation to derive an estimate of the mean and its 95% confidence interval (CI) for peak force in each muscle. The point estimates for the mean and bounds of the 95% CIs for peak force and MTJ CSA were combined using Eq. 2 to obtain an estimate of the mean peak stress in each hamstring muscle and the bounds for the 95% CI.

## RESULTS

Three studies were retrieved for analysis of force (7-9) with a combined total of 38 participants (28 males and 10 females; Age: 24; stature: 177 cm; mass: 73 kg). For the morphology data, 5 male cadavers (10 limbs) were used (mean age: 75 years; Storey 2016).

**Table III** includes the random effects 95% CIs for peak force among the 3 simulation studies and the confidence interval for MTJ area for each muscle. On average, the biceps femoris experiences 4.24 N·cm<sup>2</sup> more stress than the SM and 7.36 N·cm<sup>2</sup> more than the ST. The variability in the biceps femoris stress estimate is also greater than the SM and ST. This is highlighted at the upper bounds of the stress estimate where the difference between BF and SM increased to 9.73 N·cm.

**Table III.** Estimates of 95% confidence intervals for peak force, MTJ area, and MTJ stress.

	Muscle	Lower bound	Mean	Upper bound
Peak force* (N)	BF	861	1408.4	1960.7
	SM	1470	2289	3108
	ST	340.2	450.1	559.3
MTJ Area (cm <sup>2</sup> )	BF	39.4	45.0	50.6
	SM	62.1	84.6	107.1
	ST	16.2	18.8	21.4
MTJ Stress (N·cm <sup>2</sup> )	BF	21.85	31.30	38.75
	SM	23.67	27.06	29.02
	ST	21.00	23.94	26.14

**Note:** \*Absolute peak force has been calculated for a 70 kg person.

## DISCUSSION

The aim of this investigation was to estimate whether stress could identify the biceps femoris vulnerability to injury during high-speed running. The findings presented here show the biceps femoris experiences the greatest MTJ stress. Prior to this study, strain was the only outcome to identify the biceps femoris vulnerability, but it is implausible for strain alone to be responsible for injury. Thus, stress is the first variable to align with both simulated and myofibril research. Chumanov's *et al.* (2011) simulation found the ST had greater peak force than the BF. However, the BF in this study had a comparatively small force contribution which seems to occur from the notably low BF excitation during simulation that does not align with electromyographic data (8). As a result, the BF stress in this study are likely underestimated and may be greater in reality.

Stress as the cause of injury has fundamental implications towards our understanding of injury mechanics. Previously, lengthening was believed to be the fundamental cause of injury (5,8), but as demonstrated, lengthening likely causes injury because it results in greater forces. The notion of stress rather than strain as the muscle damaging factor aligns with earlier findings that showed muscle damage after shortening muscle actions (25). A phenomenon not possible according to the lengthening model. Nonetheless, strain may have an additive effect to injury risk separate from the increased passive tension. The volume of the MTJ does not change during lengthening therefore an increase would cause a decrease in MTJ area and a subsequent increase in stress (19). Although not the principal cause, the greater strain in the BF whilst sprinting may compound its vulnerability to injury here. Estimates of maxima hamstring muscle CSA show the SM undergoes the smallest reduction (2.3%) compared to the BF which reduces the most (8.6%) (26).

Therefore, stress at peak lengths for the biceps femoris are expected to be considerably larger than estimated here, as MTJ measurement used in this analysis were measured *ex vivo* and not lengthened.

More critical implications exist for our understanding of injury prevention. The maximum force in a local area that a material can withstand could be the principal factor in preventing injury. Understanding the factors that determine this threshold would be of great value for optimising injury prevention programmes and screening. For example, the protective role of structures binding actin to the extracellular matrix has been shown in mice studies. Mice over-expressing the  $\alpha$ 7BX2 integrin (a common isoform of a muscular integrin) display reduced membrane damage after downhill running ( $p < 0.05$ ) suggesting the increase in integrin and actin to laminin connections increase structural soundness (27). Adding micro dystrophin to mdx mice also reduces muscle damage (28). Likewise, the ACTN3 R577X polymorphism (alpha actinin 3 which binds to z discs) is associated with greater shear modulus in the hamstrings, suggesting larger stress and thus risk (29). Whilst the polymorphism was not associated to injury, the stress value for the modulus was calculated at the muscle belly and not the MTJ (29).

Reduced ability to produce eccentric force is associated with hamstring injury (30,31) and accordingly strength training has been shown to reduce hamstring injury incidence (32). Yet, if stress is the principal factor causing injury, then increasing strength should increase the risk of injury. One explanation is that strength training increases collagen in the MTJ, likely to manage to the new capacity to produce force (33). In addition, eccentric strengthening may be important for reducing fascicle strain (24) and thus reducing the passive force component from strain.

There are inevitable limitations with the approach used in this investigation. Using the peak force data from simulations for comparison assumes that the force distribution is equivalent within each muscle. This is unlikely considering fascicle strain appears non-linear (34), but lengthening is concentrated towards the MTJ adding validity to use of peak force in MTJ stress estimates.

From a practitioner perspective, it may be more effective to utilise supramaximal eccentric exercise at short muscle lengths to expose muscles to high stress without reducing MTJ CSA and causing excessive stresses. This progression would seemingly minimise muscle damage (and the possible acute injury risk) and improve adherence to prevention programmes, where longer length training could impair adherence due to soreness (35). Future research should investigate whether muscle damage can be limited by grad-

ually progressing eccentric exercise to longer lengths whilst still inducing adaptations.

## CONCLUSIONS

Until now, simulation studies have failed to identify a cause of injury that aligns with the understanding of injury in myofibril research. Using the data of simulations, this study has shown that stress is greatest for the biceps femoris whilst sprinting and may explain its susceptibility to injury, particularly at the MTJ. Future simulation studies should include stress as an outcome to calculate more valid estimates for each hamstring muscle to confirm these findings.

## CONFLICT OF INTERESTS

The authors declare that they have no conflict of interests.

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