THE EFFECT OF FATIGUE INDUCED FROM A SIMULATED HOCKEY MATCH ON BIOMECHANICAL ACL INJURY RISK FACTORS IN ELITE FEMALE FIELD HOCKEY PLAYERS

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This study investigated the effect of fatigue induced from team sport on the biomechanical factors of non-contact anterior cruciate ligament (ACL) injury risk. Peak knee moments, full body kinematics and lower limb muscle activation of 14 elite female athletes were measured during unplanned sidestepping prior to, and following, a 15 minute team sport simulation circuit. There were no changes in peak knee moments. Reductions in knee flexion angle and total muscle activation (TMA) of muscles crossing the knee (n=9) were observed ($p<0.05$). This was explained by reductions in hamstring TMA with a reduction in gluteal (n=2) TMA also found ($p<0.05$). Future research is needed to understand neurophysiological mechanisms associated with the unique and unintuitive muscle activation strategies adopted by team sport athletes when fatigued.

KEY WORDS: Sidestepping, neuromuscular, kinematics, moments, valgus.

INTRODUCTION: Non-contact anterior cruciate ligament (ACL) injuries are common in team sports which require athletes to perform sidestepping, landing and sudden stopping manoeuvres (Cochrane, Lloyd, Buttfield, Seward, & McGivern, 2007). These tasks are also commonly performed under significant time constraints in order to respond to the multiple external stimuli that are present during game play. This places significant demand on the central nervous system (CNS) to appropriately control posture in order to mitigate (i.e. through appropriate kinematic solutions) or support against (i.e. via muscle activation strategies) the external forces applied to the body. Significant research has been dedicated toward optimising modifiable biomechanical risk factors associated with an ACL injury event. In-vivo, in-vitro and in-silico research have identified that elevated combined peak knee extension, valgus and internal rotation moments, while the knee is in an extended posture (i.e. $<20^{o}$ flexion), elevates ACL strain and injury risk. Research has shown females to be 4-6 times more likely to sustain an ACL injury in sport relative to their male counterparts (Hewett et al., 2005). Additionally, it has been found that lower extremity injuries occur more during competition versus practise, where a greater proportion of game play injuries occur in the second half (Hawkins, Hulse, Wilkinson, Hodson, & Gibson, 2001; Nagle et al., 2017). This begs the question as to whether fatigue characteristics of normal game play have a role in ACL injury risk via the inability of the peripheral and/or the central nervous systems to effectively or efficiently coordinate movement and support the knee joint when external knee loading is high.

This study aimed to determine the effect of match simulated fatigue on biomechanical (i.e. peak knee moments and kinematics) and neuromuscular (i.e. total muscle activation) ACL injury risk factors among a group of elite female field hockey players. We hypothesised that following 15 minutes of a team sport game simulation circuit, athletes would display physiological measures of fatigue (i.e. heart rate and blood lactate). As a result of this fatigue, we further hypothesised athletes would have elevated peak knee moments, display

'*high-risk*' kinematic strategies and elevated muscle activation relative to pre-fatigue measures.

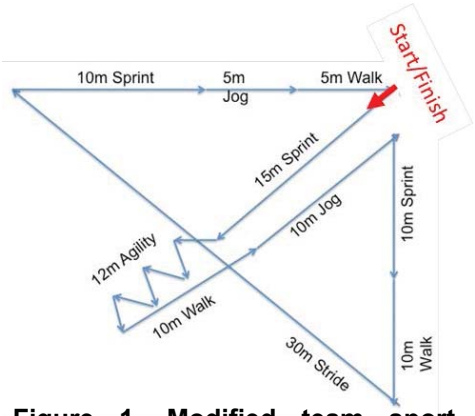

Figure 1. Modified team sport simulation circuit

METHODS: Fourteen elite female field hockey players (25.4±3.3 yrs, 1.7±0.1 m, 66.1±9.3 kg) completed three sets of a five minute modified team sport simulation circuit, with 2 minutes rest between sets (Figure 1) (Bishop, Spencer, Duffield, & Lawrence, 2001). Following each set, maximum, minimum and mean heart rate (HR), blood lactate (BLa mMol/L), set duration (minutes) and metres per minute ($m.min^{-1}$) were recorded. At completion of the simulation circuit, session rating of perceived exertion (sRPE) and rating of perceived fatigue (sRPF) were recorded.

Three-dimensional (3D) motion capture of each participant undertaking a previously published sidestepping protocol (Besier, Lloyd, Ackland, & Cochrane, 2001) were collected prior to and immediately following the match simulation protocol. Peak knee moments and full body kinematics were recorded using a 22-camera Vicon MX/T40 system at 240Hz (Oxford Metrics, Oxford, UK) and force plate data at 2,400 Hz (AMTI, Watertown, MA). Muscle activation of 11 lower limb muscles were recorded using a wireless surface electromyography (sEMG) system (Myon, Motion Lab Systems, USA) sampling at 2,400 Hz.

Customised software in MatLab 7.8 (The Math Works, Massachusetts, USA) was used to process sEMG data, as per Donnelly et al (2014). Maximum functional excitation of each muscle (n=11; gluteus maximus, gluteus medius, biceps femoris, semitendinosus, vastus medialis, vastus lateralis, tensor fasciae latae, rectus femoris, adductor magnus, medial gastrocnemius, lateral gastrocnemius) recorded during the pre-fatigued dynamic trials were used to amplitude normalise (%MVC) each muscle's sEMG signal. Mean total muscle activation (TMA) of all lower limb muscles crossing the knee joint and individual muscle groups (gluteal, quadriceps, hamstrings and gastrocnemii) were calculated. All biomechanical data were analysed during the weight acceptance phase (WA) of unplanned sidestepping. Physiology and biomechanical data were compared prior to, and following, the game simulation circuit using paired t-tests ($\alpha = 0.05$) in SPSS 17.0.1 (IBM, Chicago, Illinois).

RESULTS AND DISCUSSION: The modified team sport simulation circuit covered 2,400 m at 165 $m.min^{-1}$. In a study of distance covered in women's field hockey, Gabbett (2010) found that a typical midfielder covers distance of 1,732.7 m per quarter at 99 $m.min^{-1}$. The team sport simulation circuit sRPE scores were '*somewhat hard*' to '*hard*' and sRPF scores were high. Mean ($p=0.008$) and maximum HR ($p=0.002$) increased following set 3 in comparison with set 1 (Table 1). Blood lactate increased following each set of the simulated game circuit ($p<0.001$). Previous research has shown senior athletes to have BLa levels of 5.6mM/L immediately following a match (Ghosh, Goswami, Mazumdar, & Mathur, 1991). These physiological and perceived exertion findings suggest athletes were in a fatigued state following the sport simulation circuit. As such, results will refer to athletes as non-fatigued (baseline) and fatigued (post-simulation circuit).

Table 1. Mean (SD) set duration, mean HR, maximum HR and post set BLa following each set for all athletes (n=14).

	Time (min)	Mean HR	Maximum HR	BLa (mM/L)
Baseline				1.3 (0.6)
Set 1	4.8 (0.2)	155 (12)	177 (6)	6.2 (2.9)^
Set 2	4.9 (0.3)	176 (9)	186 (10)	7.2 (4.0)^
Set 3	4.9 (0.3)	179 (10)*	188 (9)*	8.3 (4.0)^*

^Significantly different from baseline (p<0.05) | *significantly different from set 1 (p<0.05)

There were no significant differences in peak knee moments between non-fatigued and fatigued states (Table 2). This is in contrast to findings from Chappell et al (2005) who found increases in peak knee valgus moments and peak proximal tibial anterior shear forces during jump landing following a generalised neuromuscular fatigue protocol. Similarly to Chappell et al (2005) and Borotikar et al (2008), knee flexion at foot strike reduced following the simulation circuit (p=0.030). This finding is congruent with ACL injury events that have been documented when the knee is in an extended posture (Krosshaug, Slauterbeck, Engebretsen, & Bahr, 2007). Cadaveric research has also shown that peak ACL strain occurs at knee flexion angles less than 20 degrees (Hashemi et al., 2011).

Table 2. Mean (SD) kinetics and kinematics at initial foot contact (IC) during the WA phase of unplanned sidestepping prior to (non-fatigued) and following the modified team sport simulation circuit (fatigued).

	Non-fatigued	Fatigued	*p*	d
Peak knee extension moment ($Nm.kg^{-1}.m^{-1}$)	2.22 (0.36)	2.25 (0.33)	0.642	0.076
Peak knee valgus moment ($Nm.kg^{-1}.m^{-1}$)	0.79 (0.40)	0.78 (0.30)	0.906	-0.018
Peak knee internal rotation moment ($Nm.kg^{-1}.m^{-1}$)	0.31 (0.12)	0.34 (0.13)	0.316	0.268
Knee flexion angle (IC) (^{0})	***18.5 (5.6)***	***15.2 (5.9)****	***0.030***	***-0.565***
Mean knee flexion angle (^{0})	39.2 (3.9)	41.0 (5.47)	0.374	0.378
Trunk flexion (IC) (^{0})	5.4 (3.2)	8.5 (4.1)	0.436	0.847
Trunk flexion RoM (^{0})	11.1 (4.9)	10.9 (4.2)	0.492	-0.037
Trunk lateral flexion (IC) (^{0})	-6.0 (2.8)	-6.4 (2.5)	0.348	0.231
Trunk rotation (IC) (^{0})	-10.8 (5.3)	-10.8 (8.4)	0.358	0.004

*Significantly different from non-fatigued (p < 0.05)

Opposing our hypothesis, TMA of muscles crossing the knee joint decreased by 13% following the game simulation circuit (p=0.020), which were primarily explained by significant reductions in hamstring muscle activation (p=0.001). In addition, reductions in gluteal (p=0.012) activation were also observed (Figure 2). These observed reductions in TMA were surprising and are somewhat contradictory to related research which have found increases or no change in activation following fatigued scenarios (Dimitrova & Dimitrov, 2003). The reduction in TMA observed in this study may be the result of a variety of neurophysiological factors; 1) inability of the central nervous system to evoke excitation; 2) delayed onset of muscle activation and/or; 3) a modification to the lower limb kinematics (i.e. reduced knee flexion) to generate more force from the tendinous component of muscle (Garland & Gossen, 2002). Finally, the majority of literature associated with fatigue and sEMG has focussed upon isometric muscle contractions over long time periods, where the present study investigates muscle excitation with muscles under concentric and eccentric loading over short time intervals (<0.12s). It is therefore possible that neuromuscular markers of fatigue like activation amplitude may differ during high velocity sport environments. Frequency analyses are currently being conducted on these data to map changes in median power frequency over time, another neuromuscular marker of fatigue.

A notable limitation to this study was that an Olympic female level athletic population was used limiting the generalisability of these findings to male athletes and/or athletes at lower levels of competition.

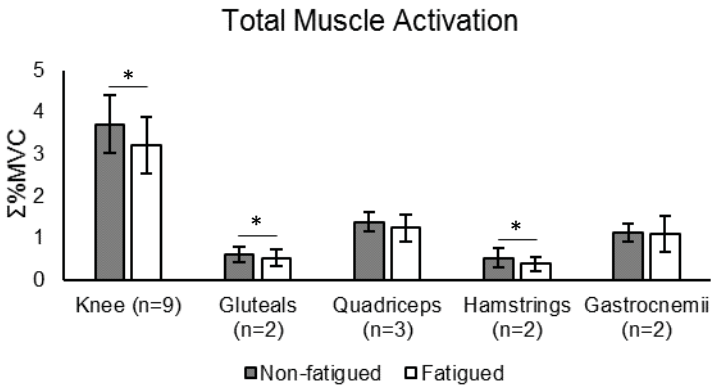

Figure 2. Total muscle activation during WA of unplanned sidestepping prior to (non-fatigued) and following (fatigued) the game simulation circuit. *p<0.05

CONCLUSION: Following a simulated quarter of hockey, elite female hockey athletes displayed physiological markers of fatigue, however this did not elicit deleterious effects in peak knee moments associated with ACL injury risk. The observed reductions in hamstring and gluteal TMA following the simulated team circuit course may be a unique neuromuscular, musculo-tendon strategy to increase muscle efficiency when fatigued. Future research is needed to understand the neurophysiological mechanisms associated with the unique and unintuitive muscle activation strategies adopted in this study by female team sport athletes when fatigued.

REFERENCES:

Besier, T. F., Lloyd, D. G., Ackland, T. R., & Cochrane, J. L. (2001). Anticipatory effects on knee joint loading during running and cutting maneuvers. *Medicine and Science in Sports and Exercise, 33*(7), 1176-1181.

Bishop, D., Spencer, M., Duffield, R., & Lawrence, S. (2001). The validity of a repeated sprint ability test. *Journal of Science and Medicine in Sport, 4*(1), 19-29.

Cochrane, J. L., Lloyd, D. G., Buttfield, A., Seward, H., & McGivern, J. (2007). Characteristics of anterior cruciate ligament injuries in Australian football. *Journal of Science and Medicine in Sport, 10*(2), 96-104.

Dimitrova, N., & Dimitrov, G. (2003). Interpretation of EMG changes with fatigue: facts, pitfalls, and fallacies. *Journal of Electromyography and Kinesiology, 13*(1), 13-36.

Donnelly, C. J., Elliott, B. C., Doyle, T. L. A., Finch, C. F., Dempsey, A. R., & Lloyd, D. G. (2015). Changes in muscle activation following balance and technique training and a season of Australian football. Journal of Science and Medicine in Sport, 18(3), 348-352.

Garland, S. J., & Gossen, E. R. (2002). The muscular wisdom hypothesis in human muscle fatigue. *Exercise and sport sciences reviews, 30*(1), 45-49.

Ghosh, A., Goswami, A., Mazumdar, P., & Mathur, D. (1991). Heart rate & blood lactate response in field hockey players. *The Indian journal of medical research, 94*, 351-356.

Hashemi, J., Breighner, R., Chandrashekar, N., Hardy, D. M., Chaudhari, A. M., Shultz, S. J., . . . Beynnon, B. D. (2011). Hip extension, knee flexion paradox: A new mechanism for non-contact ACL injury. *Journal of Biomechanics, 44*(4), 577-585.

Hawkins, R. D., Hulse, M., Wilkinson, C., Hodson, A., & Gibson, M. (2001). The association football medical research programme: an audit of injuries in professional football. *British Journal of Sports Medicine, 35*(1), 43-47.

Hewett, T. E., Myer, G. D., Ford, K. R., Heidt, R. S., Colosimo, A. J., McLean, S. G., Succop, P. (2005). Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes. *American Journal of Sports Medicine, 33*(4), 492-501.

Krosshaug, T., Slauterbeck, J. R., Engebretsen, L., & Bahr, R. (2007). Biomechanical analysis of anterior cruciate ligament injury mechanisms: three-dimensional motion reconstruction from video sequences. *Scandinavian Journal of Medicine & Science in Sports, 17*(5), 508-519

Nagle, K., Johnson, B., Brou, L., Landman, T., Sochanska, A., & Comstock, R. D. (2017). Timing of Lower Extremity Injuries in Competition and Practice in High School Sports. *Sports health*.

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