Exploring the aetiology of high burden lower limb injuries in male professional rugby union players

PATRICK JOHN CAMERON CARDEN

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Abstract

The lower limb is the most frequently injured location in Rugby Union, resulting in significant injury burden to professional teams. In order to inform risk mitigation strategies, investigation of the aetiology of high burden lower limb injuries is required. However, sports injury aetiology is a complex problem that is dependent on a multitude of causal factors. The aim of the thesis was to advance knowledge of high burden knee ligament and hamstring injury aetiology in male professional rugby union players, by prospectively exploring the association between injury and selected intrinsic and extrinsic variables.

The first experimental study of the thesis (**Chapter Three**) illustrates that over a period of seven playing seasons, injuries to the lower limb placed the highest burden on the rugby team participating in the research project, compared to the upper limbs, trunk, head and neck. Of these injuries, the locations resulting in highest injury burden were to the knee ligaments and the hamstrings. Specifically, injuries to the anterior cruciate ligament (ACL) sustained during contact events were infrequent but resulted in the highest severity. Injuries to the medial collateral ligament (MCL) sustained during tackle events and rucking placed a high injury burden due to a high incidence rate combined with moderate magnitudes of injury severity. Finally, biceps femoris strain sustained during running was the most frequently occurring injury. The findings of the study provided a focus for the subsequent experimental chapters. **Chapter Five** demonstrates that isokinetic measures of hamstrings and quadriceps strength have poor predictive value in relation to hamstring strains (the highest AUC score being 0.57), despite being associated with an increased odds of sustaining semimembranosus and semitendinosus strains. Isokinetic strength variables were not associated with sustaining biceps femoris strains. Previous injury to the hamstrings was observed to be associated with an increased odds of subsequent hamstring injury when all muscles were pooled. Previous injury to either the MCL or lateral ankle ligaments was associated with an increased risk of biceps femoris injury and medial hamstring injury. This investigation also identified that chronic exposure of high magnitudes was associated

with an increased risk of hamstring injuries sustained during running, specifically exposure to high-speed running over 14 and 21 days prior to the week in which the injury was sustained. **Chapter 6** examined a variety of variables which were theoretically associated with contact ACL and MCL injury aetiology. The influence of previous injury history was examined in relation to contact MCL and ACL injury aetiology. Previous knee ligament injury not associated with an increased risk of sustaining a subsequent knee ligament injury during a contact event. However, previous hamstring and triceps surae muscle strains were associated with an increased risk of injury. Isokinetic assessment of both hamstring and quadriceps strength exhibited poor predictive ability in relation to contact knee ligament injury (highest AUC = 0.57). Chapter 6 also examined contact MCL and ACL injury aetiology in relation to lower limb biomechanics during a single-leg drop jump task. Both larger magnitudes of external knee abduction moment and hip adduction moment 50 ms post ground contact were associated with an increased risk of injury. The study highlighted the importance of modelling injury as a rare event in relation to analysis involving player workloads. A minority oversampling algorithm was used to mitigate the negative effects of class imbalance within the player-workloads data sets. Exposure to tackle events during a match was not related to sustaining an MCL or ACL injury from a tackle. When tackle and ruck exposure were combined, increased exposure (during 7 and 14 days preceding the injury) was associated with a decrease in the odds of sustaining a contact MCL or ACL injury where the inciting event involved a tackle or a ruck. Exposure to on-pitch physical activity (PlayerLoad^{TM}) in relation to contact MCL and ACL was also explored. Acute increases in PlayerLoad^{TM} (3-day EWMA and 7-day EWMA) were associated with an increased odds of sustaining MCL injury and ACL injury. Increased magnitudes of chronic PlayerLoad^{TM} exposure during the previous 7-days with a 3-day lag as well as the previous 14-days were both associated with an increased odds of MCL as well as pooled MCL and ACL injury.

In summary, the thesis explores the lower limb injuries which place a high burden on male professional rugby players in addition to a selection of variables that are associated with their aetiology. The injuries that placed the highest burden were knee ligament injuries (MCL and ACL)

sustained during contact as well as hamstring strains. The experimental chapters reinforce the importance of previous injury history in relation to injury aetiology, with previous injury to the proximal and distal tissues of the injured area increasing subsequent injury risk, suggesting that a more universal approach to rehabilitation may be required. The findings also demonstrate that isokinetic assessment of hamstring and quadriceps strength exhibits poor classification performance in relation to hamstring, MCL and ACL injury and should not be used to infer the subsequent risk of these injuries. In contrast, joint moments occurring at the early stages of ground contact during a single-leg drop-jump task are better at classifying knee ligament injury suggesting more dynamic tasks are required when investigating sports injury aetiology. Finally, the thesis explores the influence of player workloads in relation to injury aetiology, and highlights that the differences in this relationship depending on the injured tissue type as well as the data collection methodology. The studies within the thesis are some of the first to be conducted within a rugby union setting, with this in mind, the work within the thesis provides a conduit between epidemiological and mechanistic studies in addition to providing practical applications for men's' professional rugby teams.

TABLE OF CONTENTS

ABSTRACT 1

TABLE OF CONTENTS 4

LIST OF FIGURES 14

LIST OF TABLES 20

DECLARATION 23

ACKNOWLEDGEMENTS 24

CHAPTER 1: THESIS INTRODUCTION 25

CHAPTER 2: LITERATURE REVIEW PART 1 – CONCEPTS OF SPORTS INJURY RESEARCH 27

2.1 Overview 27

2.2 Key concepts in sports injury research 27

2.2.1 Injury definition 27

2.2.2 Injury prevalence, incidence rate and exposure 29

2.2.3 Injury severity 30

2.2.4 Injury burden 31

2.3 Theoretical models of injury prevention research and injury aetiology 34

2.3.1 Sequence of injury prevention model 35

2.3.2 The translating research into injury prevention practice framework 36

2.4 Theoretical models of injury aetiology 38

2.4.1 Multifactorial model of athletic injury aetiology 38

2.4.2 Cyclical operational model to investigate contact sports injuries 40

2.4.3 A dynamic recursive model of aetiology in sport injury 41

2.4.4 Workload-injury aetiology model 43

2.4.5 A Complex systems approach to sports injury 46

2.5 Literature review summary and rationale for first study 48

CHAPTER 3: STUDY.1. EPIDEMIOLOGY OF LOWER LIMB INJURIES IN MALE PROFESSIONAL RUGBY UNION PLAYERS: A 7-YEAR STUDY 51

3.1 Context 51

3.2 Introduction 51

3.3 Methods 53

3.3.1 Participants 53

3.3.2 Data collection 54

3.3.3 Data analysis 54

3.4 Results 55

3.4.1 Does lower limb injury present the highest injury burden compared to other gross locations within the cohort of players? 55

3.4.2 Which lower limb locations presented the largest injury burden during matches and training? 56

3.4.3 Which specific injuries present the greatest injury burden within each lower limb location? 57

3.4.4 What are the inciting events that result in high burden injuries? 59

3.4.4.1 ACL & MCL injury 59

3.4.4.2 Biceps femoris, Semimembranosus/tendinosis and hamstring muscle not identified strain 60

3.4.4.2 Ankle lateral ligament & inferior tibiofibular syndesmosis injury 62

3.5 Discussion 64

3.5.1 Does lower limb injury present the highest injury burden compared to other gross locations within the cohort of players? 65

3.5.2 Which lower limb locations presented the largest injury burden during matches and training? 65

3.5.3 Which specific injuries present the greatest injury burden within each lower limb location? & What are the inciting events that result in high burden injuries? 66

3.5.3.1 Knee injuries 66

3.5.3.2 Posterior thigh injuries 68

3.5.3.3 Ankle injuries 70

3.6 Conclusions 73

3.7 Next steps 74

CHAPTER 4: LITERATURE REVIEW PART 2: A REVIEW OF HAMSTRING AND KNEE LIGAMENT AETIOLOGICAL LITERATURE IN RUGBY UNION AND OTHER TEAM SPORTS 75

4.1 Overview 75

4.2 Aetiology of hamstring muscle strain injuries 76

4.2.1 Anatomy and function of the hamstrings 76

4.2.1.1 Biceps femoris muscle structure and function 76

4.2.1.2 Semimembranosus structure and function 78

4.2.1.3 Semitendinosus structure and function 78

4.2.2 Definition and pathophysiology of hamstring strain injury 79

4.2.3 Inciting events and mechanisms of hamstring strain injury 79

4.2.3.1. Sprint-type hamstring injury mechanism..... 80

4.2.3.2. Stretch-type hamstring injury mechanism..... 82

4.2.4 Variables associated with Hamstring strain injury aetiology..... 83

4.2.4.1 Non-modifiable intrinsic variables associated with hamstring strain injury aetiology 84

4.2.4.1.1 Previous injury 84

4.2.4.2 Modifiable intrinsic variables associated with hamstring strain injury aetiology 85

4.2.4.2.1 Isokinetic measures of hamstrings and quadriceps strength..... 85

4.2.4.3 Extrinsic variables associated with hamstring strain injury aetiology..... 86

4.2.4.3.1 Player Workload..... 86

4.2.4.3.2 High-speed running exposure..... 87

4.3 Aetiology of MCL and ACL injuries 88

4.3.1 Anatomy and function of the ACL and MCL ligaments 88

4.3.1.1 Anterior cruciate ligament structure and function..... 90

4.3.1.2 Medial collateral ligament structure and function 91

4.3.2 Definition and pathophysiology of knee ligament injury..... 92

4.3.3 Inciting events and mechanisms of knee ligament injury..... 93

4.3.3.1 ACL inciting events and injury mechanisms..... 94

4.3.3.1.1. Excessive anterior tibial translation – failure of the ACL's primary function..... 95

4.3.3.1.2 External knee abduction moment and external tibial internal rotation moment – failure of the ACL's secondary functions 96

4.3.3.1.3 The multiplanar combination mechanism 97

4.3.3.2 MCL injury inciting events and mechanisms 98

4.3.4 Variables associated with ACL and MCL injury aetiology 98

4.3.4.1 Non-modifiable intrinsic variables associated with ACL and MCL injury aetiology 99

4.3.4.1.1 Previous injury 99

4.3.4.2 Modifiable intrinsic variables associated with ACL and MCL injury aetiology 100

4.3.4.2.1 Isokinetic measures of hamstrings and quadriceps strength 100

4.3.4.2.2 Movement characteristics 102

4.3.4.2.2.1 Biomechanical analysis of bilateral vertical drop jumps 103

4.3.4.2.2.2 Biomechanical analysis of the inciting event in a laboratory setting 105

4.3.4.2.2.3 Biomechanical analysis of single-leg jumping and landing 106

4.3.4.3 Extrinsic variables associated with ACL and MCL injury aetiology 109

4.3.4.3.1 Exposure to contact events 109

4.3.4.3.2 Accelerometer derived measures of external workload 110

4.3.4.3.3 The use of PlayerLoad™ in professional rugby union and other contact sports 111

4.4 Methodological and statistical considerations for conducting aetiological research 113

4.4.1 Study design 113

4.4.2 Statistical analyses used to classify the association of a variable with injury risk and associated issues 114

4.4.3 Class imbalance: Accounting for injury as a rare event when analysing workload data 116

4.4.3.1 Data level approaches to class imbalance: Majority class reduction and minority class oversampling 117

4.5 Summary of aetiological literature review and rationale for the subsequent experimental studies 120

CHAPTER 5: STUDY.2. AN EXPLORATION OF FACTORS ASSOCIATED WITH HAMSTRING STRAIN INJURY IN MALE PROFESSIONAL RUGBY UNION PLAYERS 124

5.1 Context 124

5.2 Introduction 124

5.3 Methods 128

5.3.1 Participants 128

5.3.2 Data collection 132

5.3.2.1 Injury definition and surveillance 132

5.3.2.2 Knee joint isokinetic flexion strength 133

5.3.2.3 High-speed running exposure 134

5.3.3 External workload data analysis 134

5.3.4 Statistical analysis 135

5.4 Results 138

5.4.1 Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players? 138

5.4.2 Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players? 151

5.4.3 Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players? 163

5.5 Discussion 169

5.5.1 Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players? 169

5.5.2 Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players? 171

5.5.3 Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players? 174

5.6 Limitations 176

5.7 Conclusions 177

CHAPTER 6: STUDY.3. AN EXPLORATION OF FACTORS ASSOCIATED WITH KNEE LIGAMENT INJURY IN MALE PROFESSIONAL RUGBY UNION PLAYERS 178

6.1 Context 178

6.2 Introduction 179

6.3 Methods 186

6.3.1 Participants 186

6.3.2 Data collection 191

6.3.2.1 Injury definition and surveillance 191

6.3.2.2 Knee joint isokinetic flexion strength 191

6.3.2.3 Biomechanical data 191

6.3.2.3.1 Instrumentation 191

6.3.2.3.2 Biomechanical data collection 193

6.3.2.3.3 Biomechanical Data analysis 193

6.3.2.4 Exposure to on-pitch external workload and contact events 195

6.3.2.4.1 Instrumentation 195

6.3.2.4.2 Data analysis 196

6.3.3 Statistical analysis 197

6.4 Results 200

6.4.1 Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players? 200

6.4.2 Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players? 204

6.4.2.1 Is previous lower limb injury history associated with the risk of sustaining an MCL or ACL injury from a contact event? 204

6.4.2.2 Are previously identified isokinetic knee strength variables associated with the risk of sustaining MCL or ACL injury within the cohort of male professional rugby union players? 207

6.4.2.3 Are lower limb biomechanical variables during the early ground contact phase of a single-leg drop-jump task associated with the risk of sustaining MCL and ACL injury within the cohort of male professional rugby union players? 209

6.4.3 Is exposure to on-pitch physical activity and related to an increased risk of knee ligament injury for male professional rugby union players? 216

6.5 Discussion 224

6.5.1 Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players? 225

6.5.2 Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players? 229

6.5.2.1 Is previous lower limb injury history associated with the risk of sustaining an MCL or ACL injury from a contact event? 229

6.5.2.2 Are previously identified isokinetic knee strength variables associated with the risk of sustaining MCL or ACL injury within the cohort of male professional rugby union players? 230

6.5.2.3 Are lower limb biomechanical variables during the early ground contact phase of a single-leg drop-jump task associated with the risk of sustaining MCL and ACL injury within the cohort of male professional rugby union players? 233

6.5.3 Is exposure to on-pitch physical activity and related to an increased risk of knee ligament injury for male professional rugby union players? 236

6.6 Limitations of the study 240

6.7 Conclusions 241

CHAPTER 7: THESIS DISCUSSION 243

7.1 Introduction 243

7.2 Research questions addressed in the thesis and original contribution to knowledge 243

7.2.1 Which injuries present the highest burden in men's professional rugby union? 244

7.2.2 What factors are associated with an increased risk of hamstring injury in men's professional rugby union? 245

7.2.2.1 Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players? 245

7.2.2.2 Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players? 246

7.2.2.3 Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players? 246

7.2.3 What factors are associated with an increased risk of MCL and ACL injury sustained during contact events in men's professional rugby union? 247

7.2.3.1 Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players? 248

7.2.3.2 Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players? 248

7.2.3.3 Is exposure to on-pitch physical activity related to an increased risk of knee ligament injury for male professional rugby union players? 249

7.3 General discussion: Implications and impact of the work contained within the thesis 250

7.3.1 High level overview: Where does the work sit in relation to the wider literature in sports injury prevention? 250

7.3.2 Conducting aetiology research in sports teams – overcoming small sample sizes 255

7.3.2.1 Accounting for repeated entry into analysis - The use of GLMMs 256

7.3.2.2 Accounting for injury as a rare event when analysing workload data: Majority class reduction and minority class oversampling..... 257

7.3.3 Do previous injuries influence future injury risk? 259

7.3.4 Periodic health examinations to assess lower limb injury risk – are they worth it? 261

7.3.4.1 Isokinetic assessment of hamstring and quadriceps strength261

7.3.4.2 Biomechanical analysis of the single-leg drop-jump task 262

7.3.5 Player workload: do we directly analyse exposure to the inciting event or other variables? And how long for? 265

7.4 Impact of the thesis..... 267

7.4.1 Funding awarded by the participating professional rugby union team for a subsequent PhD project to examine academy player robustness combined with a change in policy to conduct PHE data collections on junior and senior academy players as well as objectively monitor player workloads..... 268

7.4.2 A change in policy to include biomechanical measures into the decision-making process when inferring lower limb injury risk and readiness to return to play. 268

7.4.3 Funding awarded by Innovate UK and the participating professional rugby union team through a Knowledge Transfer Partnership to further develop periodic health examinations and player monitoring during rehabilitation to examine lower limb injury risk specific to professional rugby union players. 269

7.4.4 Funding awarded by Red Bull UK to the professional rugby union team through the development of an on-site sports science laboratory at the stadium. 269

7.5 Limitations and directions for future research 270

7.5.1 Research design and analysis: Limitations and directions for future research..... 270

7.5.1.1 Multivariate approach 270

7.5.1.2 Linear vs non-linear analysis: embracing sports injury as a complex problem 271

7.5.2 Analysis of human movement: Limitations and directions for future research..... 271

7.5.2.1 Biomechanical analysis of external vs internal moments and forces..... 271

7.5.3 Player workloads: Limitations and directions for future research..... 272

7.5.3.1 Analysis of external vs internal player workloads..... 272

7.6 Practical recommendations 272

7.6.1 Aetiological factors associated with high burden lower limb injuries in professional rugby union players..... 273

7.6.2 Technical note 1 - What to include in a periodic health examination: Open-chain strength or closed-chain movement analysis?..... 276

7.6.2.1 Including biomechanical analysis in a PHE: Potential barriers to adoption and solutions. 276

7.6.3 Technical note 2 - Measuring and analysing external workloads in relation to lower limb injury risk: no silver bullets. 277

7.6.3.1 Incorporating exposure to PlayerLoad™ into the daily player workload monitoring process: Potential barriers to adoption and possible solutions. 278

7.7 Thesis Conclusion 279

CHAPTER 8: REFERENCE SECTION 280

CHAPTER 9: APPENDIX SECTION 318

9.1 Methodological study one: Baseline accuracy and reliability of the coda cx1 system 318

9.1.1 Introduction 318

9.1.2 Methods 318

9.1.3 Results & Discussion 319

9.2 Methodological study two: Within and between session reliability of lower limb motion and moments during running and jumping 321

9.2.1 Introduction 321

9.2.2 Methods 321

9.2.2.1 Participants 321

9.2.2.2 Biomechanical data collection and analysis 321

9.2.2.3 Statistical analysis 322

9.2.3 Results & Discussion 322

LIST OF FIGURES

N.b. Figures are numbered in relation to the chapter sections in the table of contents. For example,

the first figure in Chapter 2 is located in section 2.2.4.

CHAPTER 2

Figure 2.2.4: Example risk matrix plot to communicate injury burden (Bahr et al., 2018)

Figure 2.3.1: Four step model of injury prevention research by van Mechelen et al., (1987)

Figure 2.3.2: Finch's (2006) TRIPP model compared to the Sequence of injury prevention model by van Mechelen et al. (1987)

Figure 2.4.1: Multifactorial model of athletic injury aetiology (Meeuwisse, 1994)

Figure 2.4.2: Cyclical operational model of sports injury (Gissane et al., 2001)

Figure 2.4.3: Dynamic recursive model of aetiology in sport injury (Meeuwisse et al., 2007)

Figure 2.4.4: The workload-injury aetiology model (Windt and Gabbett, 2016)

Figure 2.4.4.2: Systems model representation of the impulse responses of fitness and fatigue (Calvert et al., 1976)

Figure 2.4.5: Example Complex systems web of determinants for ACL injury in (A) basketball athletes and (B) ballet dancers (Bittencourt et al., 2016)

CHAPTER 3

Figure 3.3.1: Flowchart of study participants over the duration of the study

Figure 3.4.4.1: ACL & MCL injury burden as a function of inciting events for match play and training. Circles – ACL injuries, Triangles – MCL injuries. All ACL and MCL injuries are presented in A, whereas an expanded view of the dashed square area in figure A is presented in B for clarity

Figure 3.4.4.2: Biceps femoris & Semimembranosus/tendinosis injury burden as a function of inciting events for match play and training. Circles – Biceps femoris, Squares - Semimembranosus/ tendinosis, Triangles – Hamstring strain - muscle not identified. All hamstring strain injuries are presented in figure A, whereas an expanded view of the dashed square area in figure A is presented in figure B for clarity

Figure 3.4.4.3: Ankle lateral ligament & inferior tibiofibular syndesmosis injury burden as a function of inciting events for match play and training. Circles – Lateral ligament sprain (ligament unidentified), Triangles – ATFL, Crosses – Multiple ankle ligaments sprain, Black Squares – AITFL, White Squares – Calcaneofibular ligament sprain. All ankle ligament injuries are presented in figure A, whereas an expanded view of the dashed square area in figure A is presented in figure B for clarity

CHAPTER 4

Figure 4.2.1.1: An illustration of the hamstrings muscle group, comprised of a) semitendinosus, b) semimembranosus, c & e) long head of biceps femoris and d) short head of biceps femoris depicted in 1) schematic illustration and 2) dissection photograph. Adapted from Kaeding and Borchers (2014)

Figure 4.2.3.1: Running gait cycle. A) early swing, B) mid swing, C) late swing, D) early stance or foot strike, E) mid stance and F) late stance or toe-off Adapted from Kenneally-Dabrowski et al. (2019)

Figure 4.2.3.2: Example of the jackal rucking position (The standing player wearing red), comprised of deep hip flexion combined with extended knees. Photo courtesy of Exeter Chiefs.

Figure 4.2.4.2.1: A representation of the approximation of hamstring and quadriceps strength using isokinetic dynamometry.

Figure 4.3.1a: Knee joint degrees of freedom

Figure 4.3.1b: Hierarchical structure of collagen in ligaments adapted from Nigg and Herzog (2007)

Figure 4.3.1c: The ligaments of the knee joint viewed in the frontal plane.

Figure 4.3.1.1: Medial view of the anterior collateral ligament (ACL) of the knee from the sagittal plane.

Figure 4.3.1.2: Medial view of the medial collateral ligament (MCL) of the knee from the sagittal plane.

Figure 4.3.2: Stress-strain curve disapplying the non-linear material behaviour of ligaments adapted from Blaha (2017).

Figure 4.3.4.2.2: An illustration of the challenges faced by researchers conducting biomechanical aetiological research.

Figure 4.3.4.2.2.1: Illustrated diagram of a bilateral vertical drop jump adapted from Hewett et al (2005).

Figure 4.3.4.2.2.3: Example single-leg drop jump sequence.

Figure 4.4.3.1a: An abstracted graphical example of the SMOTE algorithm on an imbalanced data set for minority observation x_1 in two-dimensional space. Where x_1 is the minority data point of interest, x_{1i} are the nearest neighbours (k = 3) and x_{ki} are the synthetically generated data (Majority class data = white circles, minority class data = black circles and synthetic minority class data = white circles with black centre).

Figure 4.4.3.1b: An abstracted graphical example of the ADASYN algorithm on an imbalanced data set, synthetic minority data points are clustered around high density areas of majority class data points. (Majority class data = white circles, minority class data = black circles and synthetic minority class data = grey squares).

Figure 4.5a: Theoretical workload-injury aetiology model for hamstring strain injuries sustained by male professional rugby union players which comprises the work undertaken in Chapter 5. Adapted from Windt and Gabbett (2016).

Figure 4.5b: Theoretical workload-injury aetiology model for contact MCL and ACL injuries sustained by male professional rugby union players which comprises the work undertaken in Chapter 5. Adapted from Windt and Gabbett (2016).

CHAPTER 5

Figure 5.3.1a: A worked example of the study exclusion criteria.

Figure 5.3.1b: A flowchart illustrating the movement of players throughout the study period.

Figure 5.4.1a: Distribution of A) previous hamstrings muscle group injury and B) previous MCL injury for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red).

Figure 5.4.1b: Distribution of A) previous ankle ligament injury and B) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red)

Figure 5.4.1c: Distribution of A) previous MCL injury & B) previous ankle ligament injury for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained whilst running (shaded in red).

Figure 5.4.1d: Distribution of previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained whilst running (shaded in red).

Figure 5.4.1e: Distribution of A) previous hamstrings muscle group injury only sustained during the previous year & B) previous MCL injury for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

Figure 5.4.1f: Distribution of A) previous MCL injury only sustained during the previous year, B) previous ankle ligament injury only sustained during the previous year & C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

Figure 5.4.1g: Distribution of A) previous MCL injury, B) previous ankle ligament injury and C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with biceps femoris strain injury regardless of inciting event (shaded in red).

Figure 5.4.1h: Distribution of A) previous MCL injury, B) previous ankle ligament injury and C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with biceps femoris strain injury sustained whilst running (shaded in red).

Figure 5.4.1i: Distribution of A) previous MCL injury, B) previous ankle ligament injury and C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with semimembranosis or semitendinosis strain injury regardless of inciting event (shaded in red).

Figure 5.4.2a: Distribution of Peak concentric knee flexion moment relative to body mass at A) 60°/s sec & B) 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red).
Figure 5.4.2b: Distribution of Peak concentric knee extension moment relative to body mass at 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red).

Figure 5.4.2c: Distribution of Peak concentric A) knee flexion moment relative to body mass at 60°/s sec & B) knee extension moment relative to body mass at 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained whilst running (shaded in red).

Figure 5.4.2d: Distribution of Peak A) absolute concentric knee flexion moment at 60°/s sec & B) concentric knee flexion moment relative to body mass at 60°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

Figure 5.4.2e: Distribution of Peak A) absolute concentric knee flexion moment at 180°/s sec & B) concentric knee extension moment relative to body mass at 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

Figure 5.4.2f: Distribution of peak concentric knee flexion moment relative to body mass at 60°/s sec for injury free players (shaded in grey) and players with semimembranosus and semitendinosus strain injury regardless of inciting event (shaded in red).

Figure 5.4.3a: Distribution of High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 3 Day EWMA, B) 7 Day EWMA and C) 7 Day Sum, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

Figure 5.4.3b: Distribution of Chronic High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 7 day EWMA with a 3 day lag, B) 7 Day Sum with a 7 day lag, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

Figure 5.4.3c: Distribution of Chronic High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 14 day EWMA, B) 14 day EWMA with a 3 day lag and C) 14 Day Sum with a 7 day lag, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

Figure 5.4.3d: Distribution of Chronic High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 21 day EWMA, B) 21 day EWMA with a 3 day lag and C) 21 Day Sum with a 7 day lag, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

CHAPTER 6

Figure 6.3.1a: A worked example of the study exclusion criteria.

Figure 6.3.1b: A flowchart illustrating the movement of players throughout the study period.

Figure 6.3.2.3.1: Biomechanical laboratory plan and capture volume, including CODA cx1 units (red circle and arrows), force platform (centre rectangle) and box (square to the left of force platform).

Figure 6.3.2.3.2: Single-leg drop vertical jump sequence.

Figure 6.3.2.3.3: Reporting convention of the biomechanical model used in the study, the image illustrates external and internal joint moments occurring during jump landing. Image adapted from Tait et al. (2022).

Figure 6.4.1a: Distribution of the 7-day EWMA total number of contact events for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated MCL and ACL injuries (shaded in green).

Figure 6.4.1b: Distribution of the 14-day EWMA total number of contact events for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated MCL and ACL injuries (shaded in green).

Figure 6.4.1c: Scatterplot and distribution of the total number of tackle and ruck events experienced by players vs game minutes played. Non-injured players are shaded grey, players sustaining MCL or ACL injury are shaded in red.

Figure 6.4.2.1a: Distribution of previous hamstrings muscle group injury for injury free players (shaded in grey) and players with MCL injuries sustained during contact events (shaded in red).

Figure 6.4.2.1b: Distribution of previous calf muscle group injury for injury free players (shaded in grey) and players with MCL injuries sustained during contact events (shaded in red).

Figure 6.4.2.1c: Distribution of previous hamstrings muscle group injury for injury free players (shaded in grey) and players with pooled MCL and ACL injuries sustained during contact events (shaded in red).

Figure 6.4.2.1d: Distribution of previous calf muscle group injury for injury free players (shaded in grey) and players with pooled MCL and ACL injuries sustained during contact events (shaded in red).

Figure 6.4.2.2a: Distribution of Absolute peak concentric knee extension torque at 180°/s for injury free players (shaded in grey) and players with pooled MCL and ACL injuries sustained during contact events (shaded in red).

Figure 6.4.2.3a: Distribution of A. External hip adduction moment and B. External knee abduction moment for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red).

Figure 6.4.2.3b: Mean and 95% CI External A) Hip Adduction and B) Knee abduction moment during the landing phase of a single-leg drop jump for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red).

Figure 6.4.2.3c: Distribution of A. External hip adduction moment and B. External knee adduction moment for injury free player-days (shaded in grey), and pooled MCL and ACL injuries sustained during contact events (shaded in red).

Figure 6.4.2.3d: Mean and 95% CI External A) Hip Adduction and B) Knee abduction moment during the landing phase of a single-leg drop jump for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red).

Figure 6.4.3a: Distribution of the A. 3-day and B. 7-day EWMA of Player LoadTM for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red) and ADASYN generated MCL injuries (shaded in green).

Figure 6.4.3b: Distribution of the 7-day EWMA of PlayerLoad™ for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated pooled MCL and ACL injuries (shaded in green).

Figure 6.4.3c: Distribution of the A. 7-day with 3-day lag, B. 14-day and C. 14-day with 3-day lag EWMA of Player LoadTM for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red) and ADASYN generated MCL injuries (shaded in green).

Figure 6.4.3d: Distribution of the A. 7-day with 3-day lag, B. 14-day and C. 14-day with 3-day lag EWMA of Player LoadTM for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated pooled MCL and ACL injuries (shaded in green).

CHAPTER 7

Figure 7.3.1a: Where the work contained within the thesis is situated in relation to both frameworks of sports injury prevention research conceived by Mechelen et al. (1987) and Finch (2006), in addition to the workload-injury aetiology model. Image adapted from both Finch (2006) & Windt and Gabbett (2016).

Figure 7.3.1b: An illustration of the number of mechanistic studies arising a small number of prospective studies in relation to non-contact ACL injury – adapted from Weir (2021).

Figure 7.6.1a: Practical recommendations related to hamstring strain injury aetiological variables examined in Chapter 5.

Figure 7.6.1b: Practical recommendations related to contact MCL and ACL aetiological variables examined in Chapter 6.

Figure 7.6.2.1: An anonymised example PHE testing schedule to illustrate the time demands and testing personnel required

CHAPTER 9

Figure 9.2.3: Bland-Altman plots displaying bias, upper and lower 95% limits of agreement (LoA) for the differences in joint moment between sessions (Y axis) plotted against the inter session mean of the joint moments (X axis) for: A) Hip Add/Abduction moment, B) Hip Ext/Flexion moment, C) Knee Abd/Adduction moment, D) Knee Ext/Flexion moment, E) Ankle Inv/Eversion moment and F) Ankle Plant/Dorsiflexion moment at 50ms + ground contact.

LIST OF TABLES

N.b. Tables are numbered in relation to the chapter sections in the table of contents.

CHAPTER 3

Table 3.4.1: Match and training injury Incidence, severity and burden by location.

Table 3.4.2: Match and training injury Incidence, severity and burden by lower limb location.

Table 3.4.3: Injury Incidence, severity and burden of as a function of high burden lower limb location and specific injuries.

CHAPTER 5

Table 5.4: Hamstring injury in relation to inciting event and data collection modality.

Table 5.4.1a: Univariate generalized linear mixed-models examining the association between previous injury history and hamstring strain injury regardless of muscle type and inciting event.

Table 5.4.1b: Univariate generalized linear mixed-models examining the association between previous injury history and hamstring strain injury regardless of muscle type sustained whilst running.

Table 5.4.1c: Univariate generalized linear mixed-models examining the association between previous injury history and hamstring strain injury regardless of muscle type sustained during contact events.

Table 5.4.1d: Univariate generalized linear mixed-models examining the association between previous injury history and biceps femoris strain injury regardless of inciting event.

Table 5.4.1e: Univariate generalized linear mixed-models examining the association between previous injury history and biceps femoris strain injury sustained whilst running.

Table 5.4.1f: Univariate generalized linear mixed-models examining the association between previous injury history and semimembranosis or semitendinosis strain injury regardless of inciting event.

Table 5.4.2a: Univariate generalized linear mixed-models examining the association between isokinetic variables and hamstring strain injury regardless of muscle type and inciting event.

Table 5.4.2b: Univariate generalized linear mixed-models examining the association between isokinetic risk factors and hamstring injury regardless of muscle type sustained during running.

Table 5.4.2c: Univariate generalized linear mixed-models examining the association between isokinetic variables and hamstring injury regardless of muscle type sustained during contact events.

Table 5.4.2d: Univariate generalized linear mixed-models examining the association between isokinetic variables and biceps femoris injury regardless of inciting event.

Table 5.4.2e: Univariate generalized linear mixed-models examining the association between isokinetic variables and semimembranosus and semitendinosus injury regardless of inciting events.

Table 5.4.3a: Univariate generalized linear mixed-models examining the association between acute exposure to high-speed running and hamstring injury regardless of muscle type sustained during running.

Table 5.4.3b: Univariate generalized linear mixed-models examining the association between chronic exposure to high-speed running and hamstring injury regardless of muscle type sustained during running.

CHAPTER 6

Table 6.3.2.3.1: Bilateral lower limb and pelvis marker set

Table 6.3.3: The effect of sampling process on class imbalance in the external workload data analysed in the study.

Table 6.4: MCL and ACL injury in relation to inciting event and data collection modality.

Table 6.4.1a: Univariate generalized linear mixed-models examining the association between acute and chronic exposure to tackle events and pooled MCL and ACL injury sustained during the tackle.

Table 6.4.1b: Univariate generalized linear mixed-models examining the association between acute and chronic exposure to tackle and ruck events and pooled MCL and ACL injury (Effects modelled as tertiles in italics).

Table 6.4.2.1a: Univariate generalized linear mixed-models examining the association between previous injury history and MCL injury.

Table 6.4.2.1b: Univariate generalized linear mixed-models examining the association between previous injury history and pooled MCL and ACL injury.

Table 6.4.2.2a: Univariate generalized linear mixed-models examining the association between isokinetic knee extension and flexion strength and MCL injury.

Table 6.4.2.2b: Univariate generalized linear mixed-models examining the association between isokinetic strength and pooled MCL and ACL injury.

Table 6.4.2.3a: Univariate generalized linear mixed-models examining the association between early ground contact limb sagittal plane internal joint moments and MCL injury.

Table 6.4.2.3b: Univariate generalized linear mixed-models examining the association between early ground contact limb sagittal plane internal joint moments and pooled MCL and ACL injury.

Table 6.4.2.3c: Univariate generalized linear mixed-models examining the association between early ground contact limb frontal plane joint external moments and MCL injury (Effects modelled as tertiles in italics).

Table 6.4.2.3d: Univariate generalized linear mixed-models examining the association between early ground contact limb frontal plane joint external moments and pooled MCL or ACL injury MCL injury (Effects modelled as tertiles in italics).

Table 6.4.3a: Univariate generalized linear mixed-models examining the association between acute exposure to on-pitch physical activity and MCL injury sustained during contact events.

Table 6.4.3b: Univariate generalized linear mixed-models examining the association between acute exposure to Rugby Activity and pooled MCL and ACL injury.

Table 6.4.3c: Univariate generalized linear mixed-models examining the association between chronic exposure to on-pitch physical activity (PlayerLoad™) and MCL injury.

Table 6.4.3d: Univariate generalized linear mixed-models examining the association between chronic exposure to Rugby Activity and pooled MCL and ACL injury.

CHAPTER 7

Table 7.3.4.2: Univariate generalized linear mixed-models examining the association between sagittal plane external joint moments and hamstring strain injury during ground contact of a SLDJ task.

CHAPTER 9

Table 9.1.3: Difference in the measured and known length of the calibration object, root mean square error and coefficient of variation for static and dynamic trials.

Table 9.2.3: Between session reliability of lower limb kinematic and kinetic data a single-leg drop jump task.

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Chapter 1: Thesis Introduction

The game of Rugby Union is physically demanding, and is predominantly composed of frequent bouts of high intensity activities such as sprinting, tackling and rucking interspersed with periods of lower intensity jogging and walking (Roberts et al., 2008). It is also one of the most played and watched sports in the world. Despite its popularity, the inherent risk of injury while participating in Rugby Union is sizeable with the incidences of match injury being amongst the highest reported in professional team sports (Williams et al., 2013; Williams et al 2021); although rates are comparable with other full-contact sports such as American Football (Lawrence et al., 2015), Rugby League (Booth et al., 2017) and Australian Rules Football (Saw et al., 2002). The lower limb is the most frequently injured location in Rugby Union (47 injuries per 1,000 player hours) (Williams et al., 2013), and lower limb injury burden has been examined by previous researchers at international level (Moore et al., 2015), and domestic professional competition within both the Northern (Brooks et al., 2005a, Brooks et al., 2005b; Brooks and Kemp, 2011; Kemp et al., 2018; Kemp et al., 2019), and Southern hemisphere (Quarrie and Hopkins, 2008). This evidence of lower limb injury burden is also observed at an amateur level within community Rugby Union (Roberts et al., 2013).

Of the myriad of lower limb injuries sustained by rugby union players, epidemiological studies have observed ligament injuries to the knee (anterior cruciate ligament and medial cruciate ligament) as well as hamstring muscle strains have consistently exhibited the highest injury burden compared to other reported lower limb injuries (Brooks et al., 2005a, 2005b; Brooks et al., 2006; Dallalana et al., 2007; Kemp et al., 2018; Kemp et al., 2019). Furthermore, the inciting events resulting in these injuries during rugby union matches and training sessions have been identified in previous epidemiological studies (Brooks et al., 2006; Dallalana et al., 2007; Kemp et al., 2018; Kemp et al., 2019).

Despite this, the exact mechanisms of those high burden injuries remain elusive (Quatman et al., 2010; McLean et al., 2015; Kenneally-Dabrowski et al., 2019), although they are thought to

be multifactorial with injury history, strength, movement characteristics and workload related variables commonly associated with increased risk in other team sports such as soccer, rugby league and Australian rules football (Green et al., 2020; Chia et al., 2020; Cronström et al., 2020; Hewett et al., 2005; Gabbett and Jenkins, 2011; Ruddy et al., 2018). As a result, periodic health examinations employing a combination of these variables have become commonplace in professional team sports to establish a player's physical characteristics in relation to injury risk and training status (van Dyk et al., 2016; van Dyk et al., 2017; King et al., 2018; King et al., 2019; King et al., 2021a; King et al., 2021b). However, the findings regarding risk factors for lower limb injury are often conflicting which is likely due to the differing populations and activity profiles of the participants (Bahr, 2016). Meaning that the aetiology may not be the same for each sport, and therefore specific investigation of the population of interest is necessary. At present, the majority of injury research in rugby union has focused on large scale injury epidemiology studies quantifying which injuries pose the highest burden (Brooks et al., 2005a, 2005b; Brooks et al., 2006; Dallalana et al., 2007; Sankey et al., 2008; Kemp et al., 2018; Kemp et al., 2019), or comparative research examining the mechanisms underpinning previously reported risk factors identified in other sporting populations (King et al., 2018; King et al., 2019; Brown et al., 2020). The limited number of existing prospective aetiological studies in rugby union have focussed on the all-encompassing risk of sustaining injury (e.g. any injury, any lower limb injury or any lower limb soft tissue injury) (Cross et al., 2015; Cross et al., 2016; Williams et al., 2017a; West et al., 2020a; West et al., 2020b; West et al., 2021a; West et al., 2021b). Although this has provided a great contribution to the understanding of general injury causation in rugby union, the mechanisms of injury and subsequent factors associated with different injuries are likely different, due to this, the examination of risk factors associated with each individual injury should be examined within rugby union players. Therefore, the purpose of the Thesis is to advance knowledge of high burden lower limb injury aetiology in male professional rugby union players via prospective investigation, with a specific focus on hamstring and knee ligament injuries.

Chapter 2.0: Literature review part 1 – Concepts of sports injury research

2.1 Overview

The purpose of this chapter is to summarise the literature underpinning basic epidemiological concepts relating to lower limb injury epidemiology in professional rugby union. By doing so, the literature review aims to provide a rationale for undertaking the current research, and provide context for findings of the subsequent experimental chapters.

2.2 Key concepts in sports injury research

To facilitate a comprehensive understanding of why sports injuries occur it is important to establish the types of injury sustained (van Mechelen et al., 1992, Fich, 2006, Bahr et al., 2020). In doing so, best practices can be developed to mitigate injury risk in addition to the development of primary care and rehabilitation of said injuries. However, in order to accomplish this, the quality of the initial epidemiological data is of prime importance (Bahr et al., 2020). This means there are a number of factors that must be taken into account when conducting research concerned with injury epidemiology and aetiology which include: the definition of injury, the manner in which the frequency of injuries are measured, the manner in which the severity of these injuries are recorded and the manner in which the total burden of said injuries is placed on the team(s) in question.

2.2.1 Injury definition

A fundamental issue when researching sports injury is to define what constitutes an injury and what does not, because the definition will influence the reported magnitudes of injury occurrence and severity. In a consensus statement, the International Olympic committee defined injury sustained during sport in the following manner: “Injury is tissue damage or other derangement of normal physical function due to participation in sports, resulting from rapid or repetitive transfer of kinetic energy” (Bahr et al., 2020). This definition was consistent with that used previously in rugby union research (Fuller et al., 2007a). This definition of injury accounts

for both sudden onset injuries resulting from specific identifiable events (e.g. a player being tackled to the side of the knee resulting in an MCL sprain); and injuries with a gradual onset, where a definable precipitating event was not possible to define (e.g. Achilles tendinopathy sustained after repetitive movement) (Bahr et al., 2020).

In epidemiological research, injuries may result in three distinct consequences: An injury may result in medical attention to a player, with the player subsequently being unable to fully participate in training or competition for at least one day is referred to as a 'time loss' injury (Fuller et al., 2007a). An injury may also result in medical attention received by a player, regardless of training and/or playing status being modified, termed a 'medical attention' injury (Fuller et al., 2007a). Finally, an injury that results in any pain or reduction in performance that resulted from sports participation irrespective of medical attention or time-loss is referred to as an 'any physical complaint' injury (Clarsen et al., 2013).

This classification of injury is intrinsically linked with both the ability to accurately and reliably record the onset and resolution of the injuries in question, and the mode of onset (sudden or gradual) (Brooks and Fuller, 2006; Bahr et al., 2020). In a review of large-scale sports injury surveillance projects conducted by Ekegren, Gabbe and Finch (2016), the authors observed the majority used a time loss classification of injury. However, research by Clarsen et al. (2013) demonstrated that the number of overuse injuries was underreported by a factor of ten when using a time-loss approach compared to repeated any physical complaint questionnaire-based measurement, which suggests that a time loss classification of injury may lead to a reporting bias of acute injuries compared to those that are more transient in nature (Walter et al., 1985). However, although the 'any physical complaint' classification of injuries may result in a more comprehensive representation of injuries sustained during sports participation, a larger number of resources are required to collate and act upon the larger amount of data compared to other injury classifications. Furthermore, although a greater number of gradual onset (overuse)

injuries are observed with the 'any physical complaint' classification, issues are associated with accurately determining the onset of the injury (Bahr et al., 2020).

A particular challenge for researchers has been to tailor the classification of injury to the injury profile of the sport in question, to ensure the data collected captures the majority of the burden experienced by the team or individuals whilst accounting for available resources (Cross et al., 2018). In rugby union, acute sudden onset injuries such as muscular strains and ligament sprains occur frequently (Brooks et al., 2005a; Brooks et 2005b; Kemp et al., 2018; Kemp et al., 2019). Because of this the IRB rugby injury consensus group has used a time loss injury classification as an operating definition for injury, defined as: "an injury that resulted in a player being unable to take a full part in future rugby training or match play for more than 24 hours from midnight at the end of the day the injury was sustained" (Fuller et al., 2007a).

2.2.2 Injury prevalence, incidence rate and exposure

In order to develop the best injury prevention strategies, the most frequently occurring injuries must be identified so resources can be allocated effectively. The prevalence and incidence rate of the injury of interest are fundamental concepts used to communicate injury frequency in sports injury research (Hodgson Phillips, 2000). Injury prevalence pertains to *how many* injuries were sustained compared to the population at risk during either a single time point (point prevalence) or a window of time (period prevalence) (e.g. 3% of rugby union players suffered from Achilles tendinopathy) (Equation 1) (Bahr et al., 2020).

$$Equation\ 1: injury\ prevalence = \frac{\sum^{n} injuries}{\sum^{n} population\ at\ risk}$$

A limitation of this concept is that it does not take the magnitude of exposure to risk during the allocated time period into account. Inter study comparison of prevalence values will therefore not yield like for like comparisons of injury risk outside of the population used to calculate the initial values of prevalence (van Mechelen et al., 1992, Brooks and Fuller, 2006). The injury

incidence rate pertains to *how often* new injuries occur per unit of exposure to risk (e.g. 5 hamstring injuries per 1000 player exposures) (Equation 2) (Hodgson Phillips, 2000). Exposure to risk can be measured in either the number of specific events (e.g. per 1000: tackles, jumps or meters of high-speed running) or the amount of exposure time (e.g. per 1000: player hours of match play or training) (Bahr et al., 2020).

$$Equation\ 2: incidence\ rate = \frac{\sum^{n} injuries}{\sum^{n} exposure}$$

The current best practice for reporting incidence rate in rugby union is per 1000 player hours of exposure (Equation 3) (Brooks et al., 2005a, 2005b; Brooks and Fuller, 2006; Kemp et al., 2018; Kemp et al., 2019), with match and training hours of exposure separated due to the differences in activity profile and subsequent injury risk (Brooks et al., 2005a; Brooks et al., 2005b; Brooks and Fuller; 2006; Fuller et al., 2007a). This approach allows for comparison of injuries within a standardised timeframe of exposure.

$$Equation\ 3: incidence\ rate\ per\ 1000\ player\ hours = \left(\frac{\sum^{n} injuries}{\sum^{n} exposure}\right) \times 1000$$

2.2.3 Injury severity

When conducting sports injury epidemiology research, the severity of an injury is another fundamental factor to be considered (van Mechelan et al., 1992). This is because the severity of injuries sustained by players within a team will impact players' availability for matches and training sessions thus potentially impacting team success (Williams et al., 2016). Furthermore, there are only a finite amount of time and resources available to players, clubs, practitioners and governing bodies responsible for developing and implementing injury prevention programmes in addition to post injury treatment. Therefore, those individuals strategically focus on mitigating the risk of the injuries that result in this worst-case scenario of low player availability (Williams et al., 2016; Fuller et al., 2018). There are numerous ways to quantify the severity of an injury, van Mechelen et al. (1992), stated injury severity could be classified using

the following criteria: nature of sports injury, duration and nature of treatment, sporting time lost, working time lost, permanent damage and financial cost. The international rugby board (IRB) injury consensus group combined four of those six previous criteria (all but permanent damage and financial cost) and have defined injury severity as: “The number of days that have elapsed from the date of injury to the date of the player’s return to full participation in team training and availability for match selection” (Fuller et al., 2007a). Furthermore, injury severity has been categorised into the following groups by Fuller and colleagues: slight (0–1 days), minimal (2–3 days), mild (4–7 days), moderate (8–28 days), severe (>28 days), ‘‘career-ending’’ and ‘‘non-fatal catastrophic injuries’’ (Fuller et al., 2007a). More recently, the categorisation of severity has been revised by the England professional rugby injury surveillance project group by combining the minimal and mild severity groups into a single group (2–7 days), furthermore the severe injury severity group was separated into two groups: 28–84 days and >84 days (Kemp et al., 2018; Kemp et al., 2019).

2.2.4 Injury burden

In sports injury epidemiology, burden is defined as the overall impact of a health problem in a specified population (Bahr et al., 2020). Therefore, the variable termed ‘injury burden’ is in fact the combined product of incidence *and* severity (Brooks and Fuller, 2006; Bahr et al., 2020). This can be calculated numerically or presented graphically in scatter plots termed risk matrices and/or risk contours (Figure 2.2.4) (Bahr et al., 2018). The concept of burden originated in the field of occupational health (Murray, 1994), and was initially applied to soccer injury epidemiology by Drawer and Fuller (2002). Since then, injury burden has subsequently presented in numerous rugby union injury epidemiology studies (Brookes et al., 2005a; Brookes et al., 2005b; Kemp et al., 2018; Kemp et al., 2019). Although it has previously been referred to as ‘risk’ in some of those studies (Brookes et al., 2005a; Brookes et al., 2005b; Brooks and Fuller, 2006), the variable is the product of incidence and severity rather than the conventional meaning of risk defined as ‘the average probability of injury per athlete’ (Knowels et al., 2006).

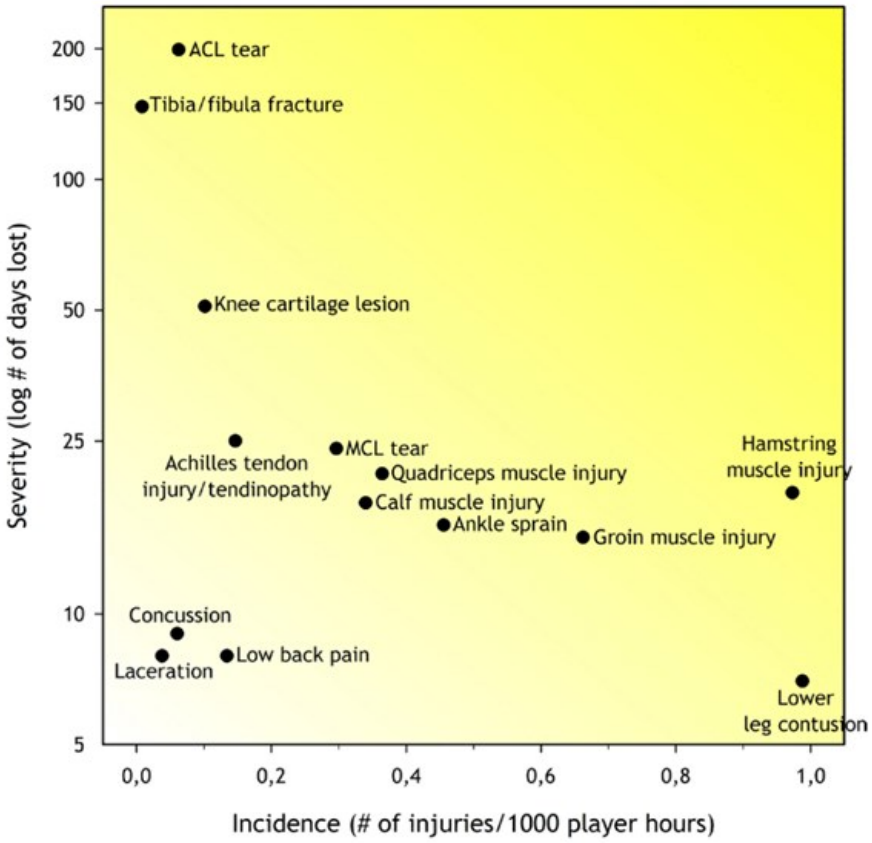


Figure 2.2.4 Example risk matrix plot to communicate injury burden (Bahr et al., 2018)

This construct is beneficial to sports injury epidemiology as it captures the extent of the injury problem and it can therefore be used to quantify the relative importance of the different injuries sustained within a team (e.g. do hamstring strains present a higher burden compared to thigh muscle haematomas?) and between different activities (e.g. between sports - football vs rugby; or mechanisms of injury within each sport - contact vs non-contact injuries) (Fuller and Drawer, 2002; Brooks and Fuller, 2006; Fuller et al., 2007a). As an example, an injury with a high incidence and low mean severity may pose an equivalent injury burden to another injury with a low incidence rate and a high mean severity and therefore both injuries should be allocated more resources to mitigate the risk of sustaining those injuries when compared to low burden injuries (low incidence/low mean severity) (Bahr et al., 2018).

At the time of writing there has been no mention of best practices for reporting injury burden in consensus statements regarding injury epidemiology within rugby union or any other sport,

indeed, this issue was highlighted by Bahr et al. (2020), in the most recent International Olympic Committee consensus statement. However, numerous publications examining injury epidemiology within rugby union including the most recent England professional rugby injury surveillance project reports have reported burden numerically (Brooks et al., 2005a; Brooks et al., 2005b; Kemp et al., 2018; Kemp et al., 2019), using the following equation (Equation 4):

$$Equation\ 4: injury\ burden\ per\ 1000\ player\ hours = \left(\frac{\sum^{n} days\ absense}{\sum^{n} exposure}\right) \times 1000$$

As a variable within the arsenal of injury epidemiology, injury burden portrays a gross view of the problem posed by the injury in question over a standardised period. However, a limitation associated with examining numerical values of injury burden in isolation (i.e. independent of the incidence rate and mean severity associated with an injury), is that it cannot be assumed that the burden values of individual injuries will impact a team in the same way (Fuller, 2018). Fuller (2018), used the hypothetical comparison of haematomas (high incidence & low severity) and anterior cruciate ligament injuries (low incidence & high severity), which have similar burden values due to their differing extremes of incidence and severity. The severity of each injury must be examined in conjunction with burden, injuries with higher severities (such as ACL injuries) may have a greater impact on the team because players will be unavailable for selection for a longer duration whereas, the majority of players who sustain haematomas will recover in time for the next competitive match (Fuller, 2018). Fuller (2018), also stated that the incidence rate of injuries must be considered in conjunction with burden and severity because the more frequently an injury occurs the greater the cumulative days absence will be. Furthermore, in the case of high incidence/low severity injuries (e.g. haematomas) the burden occurs immediately following the injury due to the short recovery period, whereas in the case of the low incidence/high severity injury (e.g. ACL injury), the overall impact of the injury is experienced for a longer period of time (Fuller, 2018). Fuller (2018), makes the argument that when considering the performance of the team (and player availability), although one may initially think the ACL injury would be the worst-case scenario due to the large amount of time loss and resources

associated with the injury. However, frequently occurring injuries may preclude a greater number of players from participating in training and matches placing the bulk of the injury burden within the playing season, whereas more severe injuries that occur less frequently may result in players rehabilitation periods encompassing the off season and/or the pre-season meaning less of the injury burden is placed within the competitive season (Fuller, 2018). The author must state that Fuller's (2018), last argument is flawed in considering the performance of the team at the exclusion of the welfare of the injured player. High severity injuries such as ACL injury have been shown to result in further health complications as players age such as osteoarthritis (Dare and Rodeo, 2014). Regardless, one should examine burden as the initial step to highlight injuries that have the largest impact on the team, once these injuries have been identified, the incidence rate and severity of the injury should then be examined to determine the manner in which each injury impact's player availability over the course of the duration of the injury recovery and rehabilitation process (Williams et al., 2016; Fuller et al., 2018).

2.3 Theoretical models of injury prevention research and injury aetiology

Injuries sustained by professional rugby union players (and all athletes) may have wide ranging consequences, not only for the injured player but also for all associated stakeholders (e.g. the team, coaches, backroom staff, management and governing bodies). As discussed in section 2.2.3 these consequences can include: a reduction in performance, permanent health problems in addition to a loss of earnings and incurred financial costs. In order for the research community to conduct sports injury research of a high standard that is of practical use to the key stakeholders the research process should be operationalised and a research framework should be employed (Finch, 2006). Therefore, an understanding of theoretical models of injury prevention and causation is necessary in order to successfully conduct sports injury research. The first part of this section presents an overview of the key models of injury prevention that have provided a framework for the majority of research undertaken in the area of sports injury. Following this, models of injury causation are explored.

2.3.1 Sequence of injury prevention model

In 1987 van Mechelen, Hlobil and Kemper proposed a four-step model for injury prevention termed the 'sequence of prevention' in order to operationalise the sports injury research process (Figure 2.3.1) (van Mechelen et al., 1987).

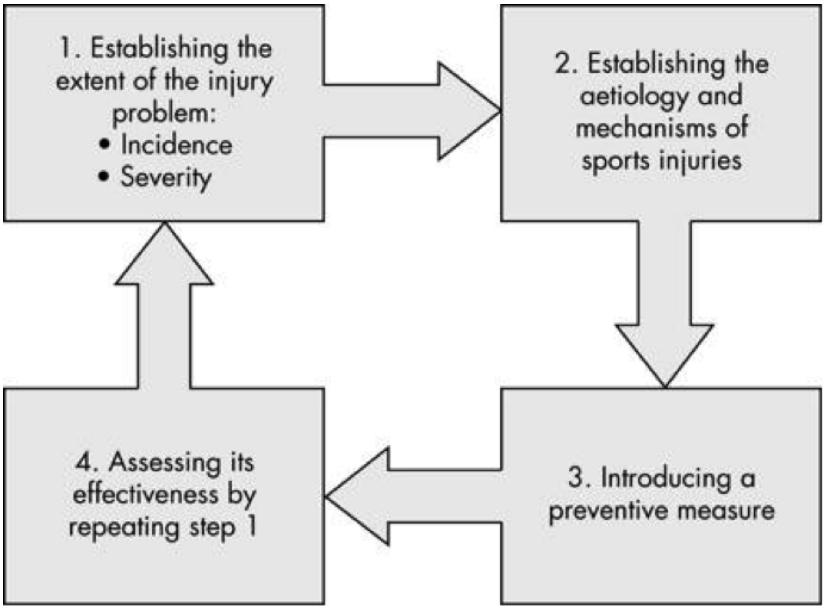


Figure 2.3.1 Four step model of injury prevention research by van Mechelen et al., (1987)

The first step in the sequence is to quantify the extent of the injury problem within the population of interest by conducting epidemiological studies (van Mechelen et al 1992). In doing so the incidence, severity and burden of the injuries in question can be established. Following this, the aetiology and mechanisms of injuries are to be established in the second step (van Mechelen et al 1992). Van Mechelen and colleagues stressed the need for a multifactorial multidisciplinary approach to examine the existence of risk factors associated with the injury of interest (van Mechelen et al., 1992). If risk factors and or injury mechanisms can be clearly identified the third step should be undertaken to introduce preventative measures to mitigate the risk and severity of sporting injuries. The sequence of prevention is an iterative process, as such the final step is to repeat the first step and compare epidemiological findings to determine whether the preventative measures have influenced the injury problem.

Although van Mechelen's model is a foundational piece of sports injury research some authors have criticized the sequence of prevention model for being too broad, specifically with regard to directions required for introducing preventative measures in the third step (Finch, 2006). Therefore, one should view it more as a theoretical framework to conduct sports injury research rather than a direct instruction.

2.3.2 The translating research into injury prevention practice framework

Finch developed van Mechelen and colleagues' work and created the translating research into injury prevention practice (TRIPP) framework of injury prevention research (Finch, 2006). The TRIPP model was developed to provide more context regarding the implementation of the preventative measures for key stakeholders including athletes, coaches, backroom staff and sports governing bodies (Finch, 2006).

The first and second steps of Finch's model are similar to van Mechelen and colleagues in establishing the incidence and severity of the injury in question, followed by examining the aetiology and mechanisms (Finch, 2006). However, stages three to five of the TRIPP model take a more granular approach compared to the third step of the sequence of prevention model.

Stage three of TRIPP focuses on examining potential solutions to the problem and the development of preventative measures (Finch, 2006). This stage is largely dependent on the risk factors and preventative factors identified by research in TRIPP stage two (Finch, 2006). Finch suggested that research conducted in stage three are mechanistic in nature being multidisciplinary laboratory-based or simulation studies of the potential protective effects of the measures in question (Finch, 2006).

Once preventative measures have been identified, TRIPP stage four corresponds to the assessment of the efficacy of the preventative measures identified in TRIPP stage three in 'ideal conditions' (Finch, 2006). For example, in this stage, the researchers (or those employed by the researchers) deliver the intervention linked to the research, and also incentives are provided to

the participants. As such, the research is often a proof of concept for larger scale studies. Therefore, Finch highlights the need to be cognizant that research conducted in this stage may lack real world efficacy when interpreted in isolation, for example, the hypothetical intervention may not be successful when the researchers are not present to provide the resources and time necessary to implement it to the letter (Finch, 2006).

The purpose of TRIPP stage five is to understand how the conclusions of research conducted under 'ideal conditions' from TRIPP stage four can be translated and implemented into a real-world context where behaviour is changed to adopt the injury prevention strategy (Finch, 2006). Research in stage five must take a cultural perspective to examine what motivators or barriers to the uptake of preventative measures are (Finch, 2006). Consultation with key stakeholders surrounding the sports injury problem (e.g. players, coaches, medical staff, conditioning staff, team management and sporting bodies) is needed at this stage before designing large-scale injury prevention measures to ensure successful implementation (Finch, 2006).

The final stage of TRIPP involves both the implementation of the intervention identified in stage 4 in a real-world context while taking into account the barriers to uptake identified in stage 5 (Finch, 2006). This is followed by a subsequent evaluation of the effectiveness of the intervention in an iterative process, similar to the four-step sequence of prevention model (Finch, 2006). Finch presents the differences between the TRIPP model and van Mechelen and colleagues in Figure 2.3.2. As previously mentioned, Finch updates van Mechelen's model with more detail paid to the development and application of preventative measures. Specifically encouraging the consideration of barriers and motivators to uptake of the injury prevention program from key stakeholders of the sport in question.

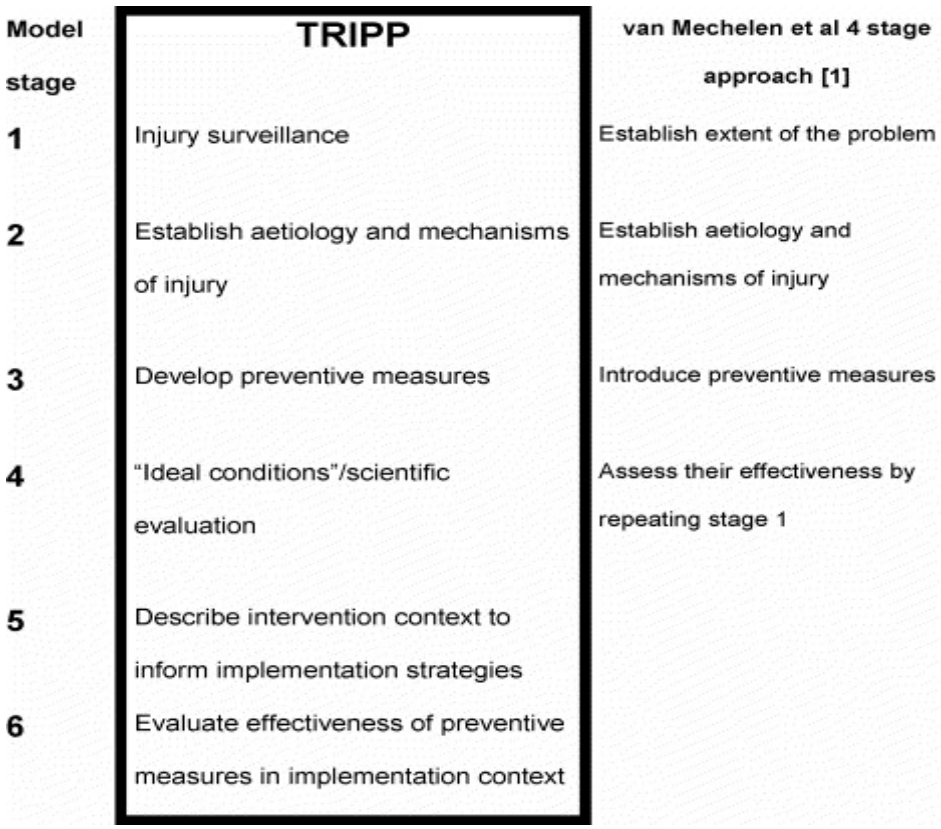


Figure 2.3.2 Finch's (2006) TRIPP model compared to the Sequence of injury prevention model by van Mechelen et al. (1987).

2.4 Theoretical models of injury aetiology

In addition to broad models of injury prevention research, several models of injury have specifically focused on the aetiology of sports injuries. In both van Mechelen et al. (1987) and Finch's (2006) frameworks of injury prevention, a vital step in the process was to establish the mechanisms and risk factors associated with the injury in question so interventions can be developed to prevent sports injury. As this is also one of the primary focuses of the PhD, an understanding of the models of injury aetiology is necessary.

2.4.1 Multifactorial model of athletic injury aetiology

Meeuwisse (1994), developed a multifactorial model of athletic injury aetiology (Figure 2.4.1) by adapting Hennekens and Buring's (1987) diagrammatic representation of how multiple factors produce disease. Meeuwisse (1994) proposed that unlike certain diseases where there

is a single cause, injury causation in sports was a multifactorial process where internal/intrinsic and external/extrinsic risk factors interact with each other to result in injury. Internal risk factors were defined as characteristics that are internal to the individual athlete (Meeuwisse, 1994), examples included: biomechanics, conditioning, maturational stage and somatotype. Intrinsic risk factors were theorised to predispose an athlete to injury, however, Meeuwisse (1994) stated that alone predisposing factors are rarely sufficient to cause injury unless external risk factors are encountered. External risk factors were defined as "having an impact on the athlete 'from without'" (Meeuwisse, 1994). Examples of external risk factors included rules, equipment as well as weather conditions and playing surfaces (Meeuwisse, 1994). In situations where internal and external factors are combined the athlete is termed susceptible to injury (Meeuwisse, 1994). However, a final link in the sequence of causation is required to result in injury, which is an inciting event or the mechanism of injury (Meeuwisse, 1994).

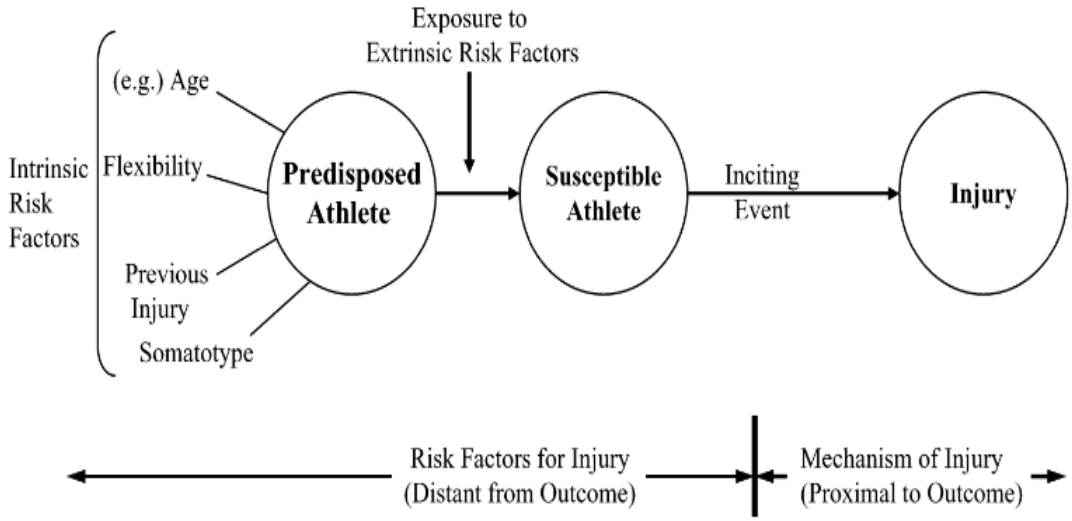


Figure 2.4.1 Multifactorial model of athletic injury aetiology (Meeuwisse, 1994)

Meeuwisse's model has made a valuable contribution to the work in suggesting that when sports medicine practitioners are examining injury prevention strategies, more focus should be paid to multiple internal and external risk factors in addition to the inciting event rather than just the inciting event in isolation. However, Gissane, White, Kerr and Jennings (2001) criticized the multifactorial model for conceptualising injury aetiology as a linear process with a defined start

and finish point. When in fact the likelihood is that risk factors for injury are likely to change with time, making it a cyclical process.

2.4.2 Cyclical operational model to investigate contact sports injuries

The cyclical operational model of injury developed by Gissane, Whie, Kerr and Jennings (2001) provided an extension to Meeuwisse's (1994) multifactorial model by changing the linear process of sports injury to a cyclical one where injury risk and the factors associated with it may change with repeated exposure (Figure 2.4.2).

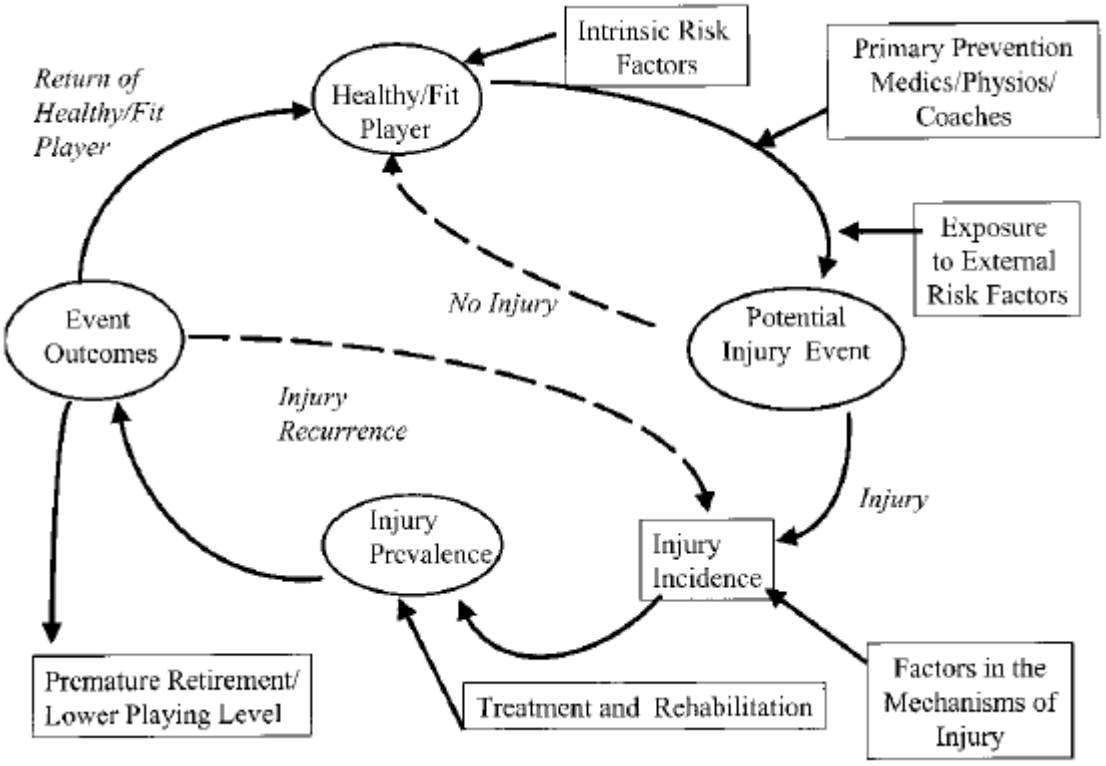


Figure 2.4.2 Cyclical operational model of sports injury (Gissane et al., 2001).

The model was specifically developed for contact team sports where numerous varied inciting events may be experienced within a competitive match as well as during training sessions. When exposed to an inciting event in the cyclical model the athlete may either go unscathed or become injured in the same manner as the multifactorial model. However, in the cyclical model, multiple outcomes may occur when an individual is injured:

1. Return to full sports participation after a period of rehabilitation.
2. After suffering an initial injury, a player may suffer a recurrence of the injury or another injury during the rehabilitation phase. In which case rehabilitation continues.
3. Premature retirement or re-enter sports participation at a significantly lower level (e.g. retire from professional sport but compete in a lower amateur league).

An advantage of the cyclical model is that a more realistic view of sports participation is portrayed, specifically in professional sport. Whereby injury is not an end point of the process, merely a part of the life cycle of an athlete in addition to rehabilitation and recovery. An additional conceptual advance of the cyclical model is that risk factors are temporal in nature. When an individual returns to sports participation from a period of rehabilitation their intrinsic risk factors will be different compared to pre-injury; furthermore, Gissane and colleagues (2001) proposed that the same was true of those colleagues that did not suffer injuries as well.

2.4.3 A dynamic recursive model of aetiology in sport injury

Following Gisane and colleagues' (2001) cyclical model, Meeuwisse, Tyreman, Hagel and Emery (2007) revisited the multifactorial model proposed by Meeuwisse (1994) in an attempt to account for the impact of repeated exposure to sporting activity following a potential injury event (Figure 2.4.3). Meeuwisse and colleagues conceded that the original multifactorial model was floored and followed an approach used in classical cohort studies of disease where individuals are followed in time to measure the outcome, which was often a finite end-point such as mortality (Meeuwisse et al., 2007), which in the overwhelming majority of cases a linear approach does not reflect the nature of sport, professional or otherwise.

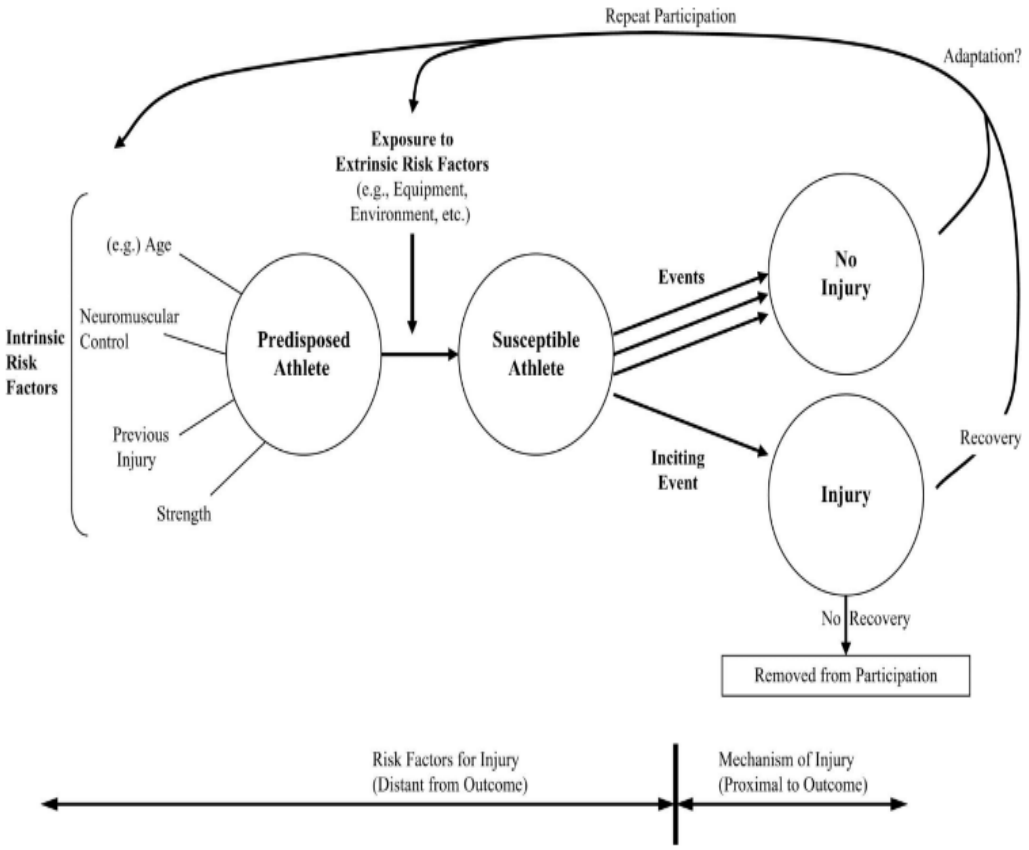


Figure 2.4.3 Dynamic recursive model of aetiology in sport injury (Meeuwisse et al., 2007).

Meeuwisse and colleagues' dynamic recursive model has accounted for the temporal nature of continued sports participation by including multiple states that an individual can transition into prior to, during and after an inciting event. The model proposed that internal risk factors such as lower limb strength can be altered via adaptation or as a result of the injury with continued participation of sport (Meeuwisse et al., 2007). Furthermore, how the athlete interacts external risk factors are also proposed to change over time with the dynamic recursive model (Meeuwisse et al., 2007). Meeuwisse and colleagues presented the example of a change in the athlete's behaviours to protective equipment or how they decelerate on a specific sport playing surface.

Although one could argue that the content of the dynamic recursive model is similar to the cyclical model, Meeuwisse and colleagues stress the importance of the temporal nature of injury risk. The model can be applied to athletes in a high temporal resolution (e.g. every contact event of a rugby union match) and a low temporal resolution (twice a year - when injury occurs).

Changes to the magnitude and interrelationships of internal and external risk factors occur in the presence or absence of injury (Meeuwisse et al., 2007). Because of this Meeuwisse and colleagues suggest to future researchers conducting prospective cohort studies record data at multiple timepoints during the timeframe of prospective studies to further determine the influence of the variables of interest on injury risk.

2.4.4 Workload-injury aetiology model

Advances in sports monitoring technology such as GPS and heart rate monitors have made quantification of athlete workloads (i.e. the data related to the volume and intensity of training and competition) easier and allow a daily frequency of data collection. Meeuwisse and colleagues suggested a relationship between athlete workload and injury risk may exist in the dynamic recursive model, however this relationship was not explored by the authors (Meeuwisse et al., 2007). Windt and Gabbett (2016) subsequently expanded the dynamic recursive model by providing explicit hypotheses regarding the influence of athlete workload on sports injury risk as a primary risk factor and a conduit to alter other risk factors. Indeed, in the workload-injury model athlete workload has multiple roles (Figure 2.4.4) (Windt and Gabbett, 2016; Windt and Gabbett, 2017):

1. Exposure - The first role of workload is the vehicle of exposure to external risk factors, which in turn leads to the potential for an increased number of injurious situations (inciting events).
2. Fitness - Workload is the primary pathway of positive adaptation of modifiable risk factors associated with training and competition (e.g. aerobic capacity).
3. Fatigue - Workload is the primary pathway of the negative consequences related to training and competition resulting in a decreased capacity of modifiable risk factors (e.g. neuromuscular control).

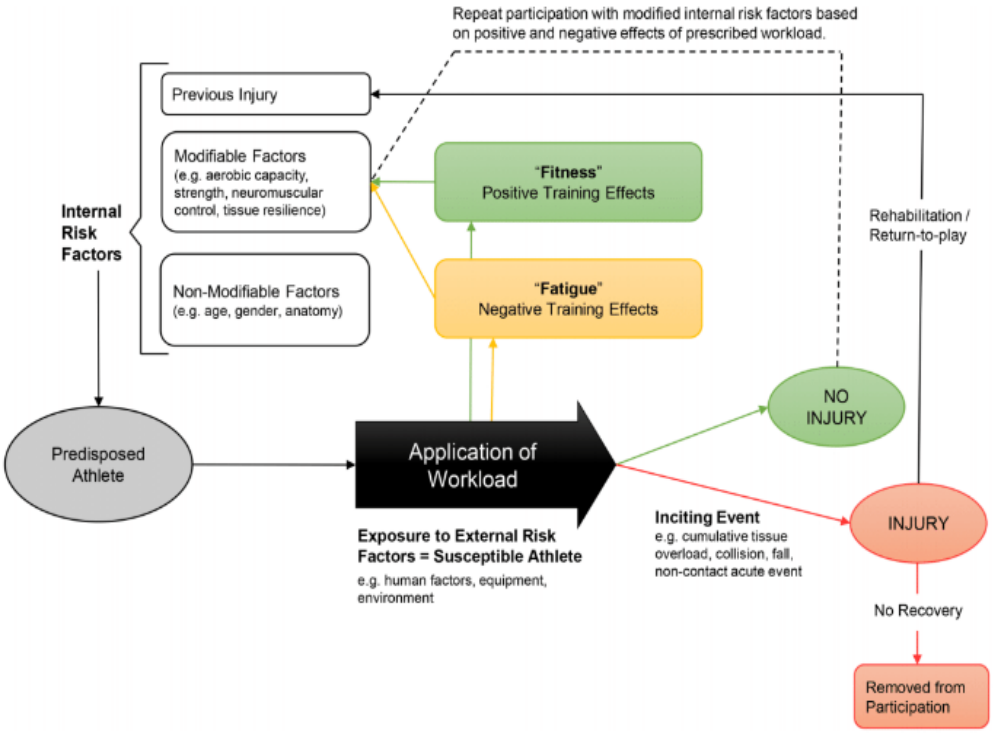


Figure 2.4.4 The workload-injury aetiology model (Windt and Gabbett, 2016).

In the workload-injury model, Windt and Gabbett (2016), reconceptualised the principles of Bannister et al. (1975) and Calvert et al. (1976) systems model, relating the positive and negative effects of training from physical performance to injury risk. In simplistic terms, the athlete is the system, the workload said athlete is exposed to as the input, and the physical performance the output (Calvert et al., 1976). The two broad outcomes of each training stimulus are fitness and fatigue, fitness, being the positive response to the dose of training load, occurs in the presence of sufficient recovery. Conversely fatigue is the negative response to the training load and may cause a reduction in performance capacity and/or an increase in injury risk (Windt and Gabbett, 2016; Windt and Gabbett, 2017). Each training dose causes a positive and negative impulse that both decay exponentially post session, with the fatigue component hypothesised to decay more rapidly (15 day decay time constant) compared to fitness (50 day decay time constant) (Figure 2.4.4.2) (Calvert et al., 1976).

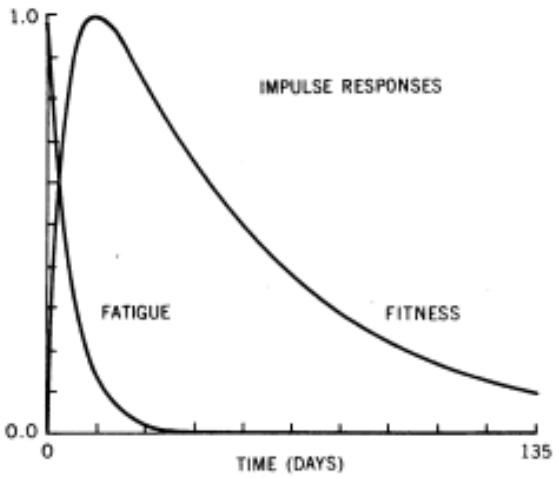


Figure 2.4.4.2 Systems model representation of the impulse responses of fitness and fatigue (Calvert et al., 1976).

In the model Windt and Gabbett (2016) equated fatigue with 'acute' workloads that athletes have just undertaken, and fitness with 'chronic' workloads which the athlete has experienced recently in the medium-term (e.g. 3 - 6 weeks). Comparison of the ratio of acute workloads by chronic workloads (termed the training-stress balance and more recently acute:chronic load ratio) is central to the workload-injury aetiology model and was hypothesised to provide an index of athlete preparedness to account for injury risk (Hulin et al., 2014, Blanch and Gabbett, 2015; Gabbett, 2016). Using the acute:chronic ratio, Windt and Gabbett (2016) proposed that the relationship with injury risk and athlete workloads is non-linear in that both high and low acute:chronic workload ratios may result in an increased injury risk. For example, a higher acute:chronic workload ratio would indicate an athlete experienced a substantially higher workload compared to their recent training history. Which Windt and Gabbett (2016) hypothesised would result in negative changes to an athlete's internal risk factors associated with fatigue which may increase the risk of injury. In contrast, a lower acute:chronic ratio was hypothesised would mean there is little or no variation in athlete workloads which may occur in the presence of sustained inactivity (where an individual lacks the protective factors associated with 'fitness') or sustained high workloads (where an individual has a higher exposure to injurious inciting events) (Gabbett, 2016; Windt and Gabbett, 2016).

The workload-injury model introduces a paradigm shift in the form of athlete workload as a vehicle for exposure to inciting events. Furthermore, although Meeuwisse et al. (2007) suggested that some risk factors may be time dependent, Windt and Gabbett (2016) hypothesised athlete workload has a cumulative influence on injury risk in that the factors associated with 'fatigue' and or 'fitness' will build up and decay over time. However, a limitation of the workload-injury model and the majority of research associated with it is that the manner in which workload influences the risk of specific injuries may not be uniform in nature (e.g. a hamstring strain during high-speed running vs an ankle lateral ligament injury during a rapid change of direction). Indeed, the overwhelming majority of research has been applied to assess the influence of athlete workload characteristics on the risk of sustaining non-contact soft tissue injuries with limited specific focus on individual muscles (Gabbett and Jenkins., 2011; Gabbett and Ullah., 2012; Cross et al., 2015; Williams et al., 2017; West et al., 2020). A further limitation of the model is that although Windt and Gabbett (2016) highlight that generally speaking the relationship between workload and injury risk is more than likely nonlinear, the complex interaction between work-load, the subsequent accumulation of fitness and fatigue influences interactions between risk factors (external and internal) and injury risk in general.

2.4.5 A Complex systems approach to sports injury

All of the previous models of sports injury aetiology have acknowledged that causes of injury are highly complex and likely dependant on many factors (Meeuwisse, 1994; Gissane et al., 2001; Meeuwisse et al., 2007; Windt and Gabbett, 2016). Indeed, the later models of Meeuwisse et al. (2007) and Windt and Gabbett (2016) accounted for the non-linear and recursive nature of sports injuries. However, Hulme and Finch (2015) and Bitencourt et al. (2016) asserted that the aforementioned models were not sufficient to delineate the complex interactions between factors due to the reductionist approach employed by researchers. Reductionism in this context is the simplification of the complex problem of sports injury aetiology into basic units, to enable the identification of isolated factors assumed to be the cause of injury rather than the relationships between multiple factors (Bitencourt et al., 2016). Hulme and Finch (2015) and

Bittencourt et al. (2016) proposed that it is necessary for sports injury researchers to move beyond reductionism and adopt a complex systems approach when examining injury aetiology.

A complex system is defined as a system composed of many components which may interact to varying degrees, these interactions may modify the components and also create new unpredictable components within the system iteratively (Bittencourt et al., 2016). There are a number of characteristics that complex systems exhibit:

1. Open system - A system that exchanges energy and matter with the environment without a loss of identity.
2. Inherent nonlinearity - The behaviour of the system is not proportional to the input. Abrupt changes in the configuration of the system (emergence) are the result of interaction *between* the components of the system rather *than* the actual units. This emergence could not be predicted based on the isolated behaviour of the components alone.
3. Recursive structure - The system output is repossessed as the new input.
4. Self-organisation - When certain values of system components reach a threshold, a spontaneous occurrence of order within the system is observed periodically.
5. Uncertainty - A lack of full predictability.

In the example complex systems model of aetiology represented in figure 2.4.4, Bittencourt et al. (2016) proposed that components (injury risk factors and protective factors) of the complex system (the injury in question) form nonlinear interactions, termed web of determinants by Philippe and Mansi (1998). Observation of the patterns exhibited by these interactions (regularities) results in a profile of injury risk, which can then be used as a whole to examine the association with the injury in question (the emerged pattern) (Bittencourt et al., 2016). In the example presented in figure 2.4.4 the emerging pattern of ACL injury is created by different regularities, with certain components having more of an influence than others.

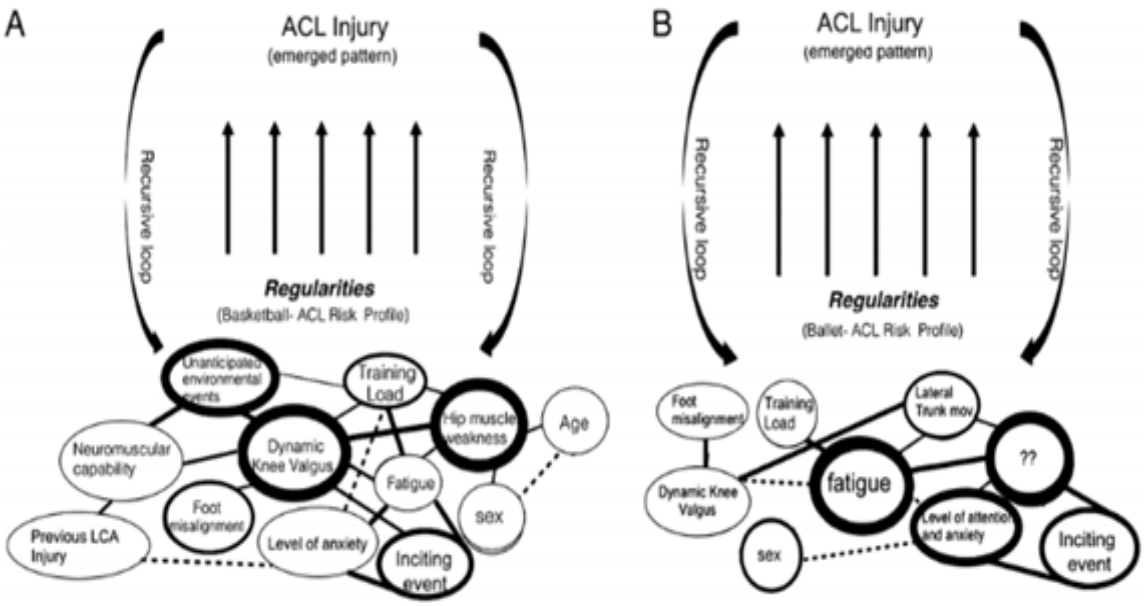


Figure 2.4.5 Example Complex systems web of determinants for ACL injury in (A) basketball athletes and (B) ballet dancers (Bittencourt et al., 2016).

The application of systems dynamics to sports injury aetiology is still part of an emerging field which is currently limited by the acceptance of analytical methods within sports science, with only a small number of studies that have embraced this paradigm (Hulme et al., 2017; Hulme et al., 2019). However, with technology allowing increasingly rich data sets to be collected by researchers and practitioners within sport the opportunity has arisen to explore the complex relationship between multiple variables and sports injury (Gabbett et al., 2019).

2.5 Literature review summary and rationale for first study

The overall aim of the thesis is to advance knowledge of workload and biomechanical risk factors for the development of high burden lower limb injuries for professional rugby union players.

Section 2 has highlighted fundamental concepts for the author to consider when undertaking the work. Although the previously mentioned frameworks and models have differences, a number of key themes emerged collectively upon examination. Both van Mechelen and colleagues (1992) and Finch (2006) stress the need for injury prevention research to follow a systematic approach directed by sound injury epidemiology and only then followed by a

subsequent examination of the aetiology of injuries. Furthermore, Finch (2006) states the importance of sports injury researchers working in conjunction with key stakeholders (rugby union players, coaches, backroom and medical staff) so the research will have a meaningful impact. All of the models state the importance of a multidisciplinary and multifactorial approach to examine injury aetiology and the associated mechanisms of the injuries in question (van Mechelen et al., 1992; Meeuwisse, 1994; Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2016). With this multifactorial approach, the delineation between intrinsic and extrinsic risk factors is also primary importance (van Mechelen et al., 1992; Meeuwisse, 1994; Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2016); so too is the understanding that certain risk factors are dynamic and may change with time, whereas some do not (Gissane et al., 2001; Meeuwisse et al., 2007; Windt and Gabbett., 2016). Indeed, the life cycle of a professional athlete such as a rugby union player is rarely linear (barring catastrophic injuries resulting in retirement); instead, it is cyclical. Players will be exposed to injurious events every time they train and compete which may result in injury and a subsequent period of rehabilitation or no injury at all (Gissane et al., 2001; Meeuwisse et al., 2007; Windt and Gabbett., 2016). One must also take into account that musculoskeletal injury has a far greater number of degrees of freedom compared with disease, as such conventional models of disease aetiology are not sufficient to explain the complex interactions between the large number of factors (risk and protective) associated with sports injuries (Meeuwisse 1994, Bittencourt et al., 2016).

In order to successfully explore the aetiology of high burden injuries in professional rugby union, the injuries that pose the highest injury burden must first be identified. The first experimental study will examine lower limb injury epidemiology within the cohort of professional rugby union players in conjunction with frameworks of injury prevention research (van Mechelen et al., 1992; Finch., 2006) as well as in accordance with the IRB consensus statement and the previous literature conducted (Brooks et al., 2005a; Brooks et al., 2005b; Brooks et al., 2006; Dallalana et

al., 2007; Sankey et al., 2008; Kemp et al., 2018; Kemp et al., 2019). Therefore, the following novel research questions that will be addressed in the first experimental study are:

the following research questions were explored:

1. Does lower limb injury present the highest injury burden compared to other gross locations within the cohort of professional male rugby union players?

   If the above research question is observed to be true in the cohort:

2. Which lower limb locations presented the largest injury burden during matches and training?

3. Which specific injuries present the greatest injury burden within each lower limb location?

   If specific high burden injuries are observed:

4. What are the inciting events that result in high burden injuries?

Chapter 3: Study.1. Epidemiology of lower limb injuries in male professional rugby union players: a 7-year study

3.1 Context

In the context of the PhD project, establishing the areas with the highest injury burden and the associated inciting events was paramount to ensure the subsequent research made a positive impact on the team co-funding the project and that it provided a meaningful contribution to the field of injury research in rugby union.

3.2 Introduction

Rugby union is a field-based team sport in which players undergo frequent bouts of high-intensity activity that are interspersed with periods of low-intensity movement (Roberts et al., 2008; Howe et al., 2020). Participation in rugby union is associated with a substantial risk of injury due to the exposure to the aforementioned high physical demands in combination with the repeated contacts and collisions with other players (Fuller et al., 2010). Because of this, rugby union has one of the highest reported injury incidence in professional team sports (Williams et al., 2021). Lower limb injury has previously been reported to present a particularly large injury burden in rugby union during matches and in training (Brooks et al., 2005a; Brooks et al., 2005b). Indeed, this was confirmed by meta-analyses, which concluded that for the overall injury burden in rugby union to be reduced, focus should be placed on lower limb injury prevention (Williams et al., 2013; Williams et al., 2021). In order to mitigate and reduce injury risk within rugby union, the epidemiology and aetiology of the lower limb injury problem must be understood. Not only is this a foundation for subsequent research but also represents the initial steps in developing a theoretical framework for sports injury prevention (presented in Chapter 2 section 2.4) (van Mechelen et al., 1987; van Mechelen et al., 1992; Finch, 2006). A number of studies have been conducted which have investigated injury occurrence in men's professional rugby union during match play (Brooks et al., 2005a; Fuller et al., 2010; Kemp et al.,

2014; Kemp et al., 2018; Kemp et al., 2019) and training (Brooks et al., 2005b; Kemp et al., 2014; Kemp et al., 2018; Kemp et al., 2019; West et al., 2021a). Later studies have also examined the epidemiology of injuries sustained to specific body locations including the knee (Dallalana et al., 2007; Montgomery et al., 2018), hamstring (Brooks et al., 2006), and ankle (Sankey et al., 2008). However, a limitation of these studies is that they have focussed on the aetiology only in a broad sense, with the majority only reporting the percentage prevalence of inciting events leading to injury (e.g. 43% of ACL injuries resulted from being tackled - Dallalana et al., 2007), without providing data regarding the incidence, severity or burden, thus limiting the understanding of the problem. Injury aetiology is complex in nature, with risk factors potentially being specific to certain injury types and inciting events, necessitating investigation of injury epidemiology at such a level (van Mechelen et al., 1992; Meeuwisse, 1994; Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2017; Bittencourt et al., 2016). Therefore, the purpose of the current exploratory study was to replicate the approach of previous epidemiological studies in rugby union, in accordance with existing theoretical models and frameworks for injury prevention, in order to provide a focus for the rest of the thesis. To facilitate this, the following research questions were explored:

1. Does lower limb injury present the highest injury burden compared to other gross locations within the cohort of professional male rugby union players?

   If the above research question is observed to be true in the cohort:

2. Which lower limb locations presented the largest injury burden during matches and training?
3. Which specific injuries present the greatest injury burden within each lower limb location?

   If specific high burden injuries are observed:

4. What are the inciting events that result in high burden injuries?

3.3 Methods

3.3.1 Participants

A total of 143 players from one club competing in the English premiership took part in the study over seven playing seasons (2012-13 Season to 2018-19 Season) (Figure 3.3.1). At the time of writing this was the highest level of national competition in professional rugby union in England. Players from the senior playing squad that sustained an injury during a competitive match or during training sessions were included in the study. The study was approved by the University of Exeter Sport and Health Sciences Ethics Committee.

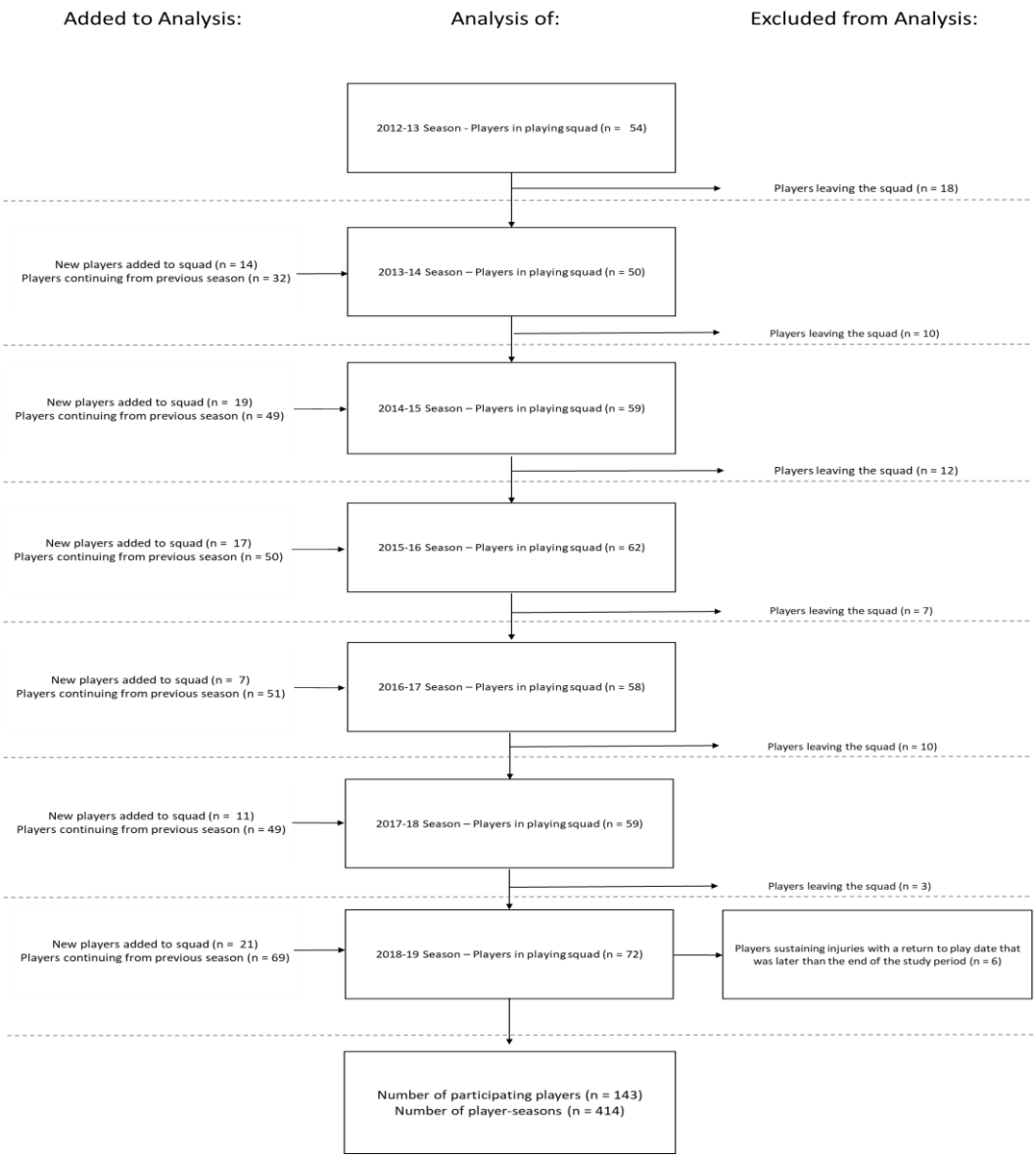


Figure 3.3.1 Flowchart of study participants over the duration of the study

3.3.2 Data collection

Injuries were reported using a modified Orchard Sports Injury Classification System (OSICS) (Orchard, 1995). In order to compare epidemiological data from the current study to previous studies investigating injury epidemiology in men's professional rugby union, a "time loss" injury definition was adopted in which: "any injury that prevents a player from taking part in all training or match activities typically planned for that day for a period greater than 24 hours from midnight at the end of the day the injury was sustained" (Fuller et al., 2007a). Time loss that resulted from illness and any non-rugby related medical conditions were excluded from the study. Injuries were further categorised by inciting event. Individual exposure data in hours were collected weekly for matches and training sessions for all players included in the study (Fuller et al., 2007a).

3.3.3 Data analysis

Injury severity was defined as the total number of days a player was absent from training and/or matches; the return from injury date was recorded when the player was available for full training and/or for match play (Fuller et al., 2007a). Continuous severity data was binned into the following categories previously used by Fuller et al. (2007a) combined with the most recent England professional rugby injury surveillance project report (PRISP) at the time of writing (Kemp et al., 2019): low severity injuries (2 - 7 days absence), moderate severity injuries (8 - 28 days absence), high severity injuries (29 - 84 days absence) and very high severity injuries (>84 days absence). Mean and median severity were calculated for all injuries. Injury burden was determined by dividing the sum of injury severity for a given injury category by the total exposure of the activity of interest (match or training) within the timeframe of interest (days absence/1000 player-hours) (Bahr et al., 2018; Bahr et al., 2020). Injury incidence was calculated as the total count of the injury of interest divided by the exposure of interest, which in the current study was per 1000 player-hours of matches and training sessions (Brooks et al., 2005a; Brooks et al., 2005b; Bahr et al., 2020). Injuries resulting in retirement were included for

incidence calculations but excluded for severity and burden calculations. Corresponding 95% confidence intervals (CI) of the mean were calculated for injury burden, incidence and severity to undertake a descriptive analysis. Statistical significance ($P \leq 0.05$) was assumed when the upper and lower 95% CI of the mean difference were larger than zero. Statistical analysis was conducted using R (version 3.6.3, R Foundation for Statistical Computing, Vienna, Austria).

3.4 Results

During the study 4851 hours of match play and 79082 hours of training were recorded. During this study period, a total of 384 injuries occurred during match play resulting in a total of 14103 days of player absence compared to 150 training injuries resulting in a total of 5982 days absence.

3.4.1 Question 1. Does lower limb injury present the highest injury burden compared to other gross locations within the cohort of players?

Lower limb injuries occurred more frequently and with greater burden than all other injuries to the head & neck, the trunk and upper limbs (Table 3.4.1). No differences in mean injury severity were present between injury locations (Table 3.4.1). Match injuries were observed to occur at a higher incidence rate and with greater injury burden when compared to injuries sustained during training activities; however, there was no difference in mean injury severity between match and training injuries (Table 3.4.1).

Table 3.4.1 Match and training injury Incidence, severity and burden by location.

		Total Number of Injuries	Incidence (per 1000 player hours & 95% CI)	Total Injury Severity (Days absent)	Mean Severity (Days absent & 95% CI)	Injury Burden (Days injured per 1000 player hours & 95% CI)
Head & Neck	Match	94	19.18 (14.78 - 23.58)†	1673	17.99 (1.88 - 34.09)	338.39 (238.11 - 438.67)†
	Training	11 ¶	0.14 (0.07 - 0.2) ¶	354 ¶	32.18 (2.85 - 61.52) ¶	5.08 (1.12 - 9.05) ¶
Upper Limb	Match	75	15.37 (11.12 - 19.62)†	3447	45.96 (14.91 - 77.01)	702.68 (420.69 - 984.67)†
	Training	13	0.16 (0.08 - 0.24)	554	42.62 (7.92 - 77.31)	7.74 (2.43 - 13.06)
Trunk	Match	16	3.36 (1.45 - 5.27)†	312	19.5 (1.00 - 41.31)	93.72 (27.5 - 159.95)†
	Training	13	0.17 (0.07 - 0.27)	499	38.38 (10.85 - 65.92)	7.6 (3.69 - 11.51)
Lower Limb	Match	199	41.16 (33.97 - 48.35)*†	8671	43.57 (1.00 - 88.57)	1787.41 (1374.32 - 2200.49)*†
	Training	113	1.41 (1.10 - 1.72)**	4575	40.49 (1.00 - 97.41)	56.04 (27.93 - 84.15)**

* Denotes a statistically significant increase of $P \leq 0.05$ between all other injury locations, ** Denotes a statistically significant increase of $P \leq 0.05$ between all other injury locations sustained during training, † Denotes a statistically significant increase of $P \leq 0.05$ between match and training injury, ¶ Denotes an additional injury resulting in retirement.

3.4.2 Question 2. Which lower limb locations presented the largest injury burden during matches and training?

Table 3.4.2 provides an overview of the incidence, severity and burden of injuries sustained during training and match play as a function of anatomical location. The incidence of match injuries occurred at significantly higher incidence rates and with larger magnitudes of injury burden when compared with training injuries for all lower limb locations ($P \leq 0.05$) (Table 3.4.2). However, with the exception of anterior thigh injuries, the mean severity of all other lower limb injuries was not significantly different between training and matches ($P > 0.05$).

Table 3.4.2 Match and training injury Incidence, severity and burden by lower limb location.

		Total Number of injuries	Incidence (per 1000 player hours & 95% CI)	Total injury severity (Days absent)	Mean severity (Days absent & 95% CI)	Injury burden (Days injured per 1000 player hours & 95% CI)
Ankle	Match	35	7.15 (5.59 - 8.71)^{AT,FT,HG}†	1210	34.57 (15.19 - 53.95)^{AT,HG,LL}	244.16 (159.3 - 329.02)^{AT,FT,HG} †
	Training	21	0.24 (0.10 - 0.39) ^{AT}	671	31.95 (9.04 - 54.86)	10.72 (4.39 - 17.06)$^{AT,\ FT}$
Anterior Thigh	Match	13	2.71 (1.27 - 4.15)†	122	9.38 (4.08 - 14.69)	29.82 (13.54 - 46.09)†
	Training	4	0.05 (0.01 - 0.08)	113	28.25 (14.63 - 41.87)††	2.57 (1.14 - 3.99)
Foot & Toe	Match	11	2.29 (0.25 - 4.33)†	518	47.09 (3.72 - 90.46)	180.16 (51.81 - 308.5)^{AT} †
	Training	8	0.11 (0.02 - 0.20)	124	15.5 (7.54 - 23.46)†	2.25 (1.41 - 3.09)
Hip & Groin	Match	19	3.92 (1.92 - 5.92)†	391	20.58 (6.15 - 35) ^{AT}	91.95 (45.56 - 138.35)^{AT}†
	Training	20	0.25 (0.15 - 0.34) ^{AT}	363	18.15 (7.45 - 28.85)	4.32 (1.68 - 6.95)
Knee	Match	59	12.2 (9.44 - 14.95)*†	4347	73.68 (14.27 - 133.08)$^{AT,\ HG,\ LL,\ Ank}$	893.81 (550.01 - 1237.6)*†
	Training	13	0.16 (0.08 - 0.24)	1738	133.69 (1.0 - 278.37) ^{FT}	24.52 (7.89 - 41.15)^{AT,FT,HG}
Lower Leg & Achilles tendon	Match	32	6.72 (4.43 – 9.00) ^{AT,FT}†	669	20.91 (1.83 - 39.99) ^{AT}	142.91 (62.17 - 223.64)^{AT}†
	Training	21	0.26 (0.16 - 0.37) ^{AT}	520	24.76 (5.93 - 43.59)	6.33 (2.03 - 10.62)
Posterior Thigh	Match	30	6.18 (4.10 - 8.25) ^{AT,FT}†	1414	47.13 (1.0 - 104.45) ^{AT}	299.21 (125 - 473.42)^{AT,HG} †
	Training	26	0.34 (0.16 - 0.51) ^{AT,FT}	1046	40.23 (14.32 - 66.14) ^{AT,FT,HG,Ank}	13.65 (5.29 – 22.00)^{AT,FT}

* Denotes a statistically significant increase compared to all other injury locations of $p \leq 0.05$. ^{AT} Denotes a statistically significant increase compared to anterior thigh injury of $p \leq 0.05$ within match or training groups. ^{FT} Denotes a statistically significant increase compared to foot & toe injury of $p \leq 0.05$ within match or training groups. ^{HG} Denotes a statistically significant increase compared to hip & groin injury of $p \leq 0.05$ within match or training groups. ^{LL} Denotes a statistically significant increase compared to lower leg & Achilles injury of $p \leq 0.05$ within match or training groups. ^{Ank} Denotes a statistically significant increase compared to lower leg & Achilles injury of $p \leq 0.05$ within match or training groups. † Denotes a statistically significant increase between match and training injury of $p \leq 0.05$ for specific injury location. †† Denotes a statistically significant increase between training and match injury of $p \leq 0.05$ for specific injury location.

Injuries to the knee during match play had the highest incidence and resulted in the highest burden over the course of the study, followed by injuries to the ankle, posterior thigh and the lower leg & Achilles tendon (Table 3.4.2). The mean severity of injuries for the majority of lower limb locations had overlapping confidence intervals so differentiation between all locations was not possible. The only exceptions were that injuries to the anterior thigh sustained during match

play resulted in the lowest magnitudes of severity, followed by injuries to the hip & groin and lower limb & Achilles tendon when compared to knee, ankle and posterior thigh injuries (Table 3.4.2).

3.4.3 Question 3. Which specific injuries present the greatest injury burden within each lower limb location?

Knee injuries sustained during match play were responsible for the highest injury burden over the course of the study, followed by ankle injuries, posterior thigh injuries and lower leg injuries & Achilles tendon (Table 3.4.2). Table 3.4.3 presents specific injuries sustained to the posterior thigh, knee, lower leg and ankle. For the majority of specific injuries presented, match injuries occurred at a higher incidence rate and with a higher burden than injuries sustained during training (Table 3.4.3). However, for the majority of specific injuries, mean severity between match and training injuries were not significantly different (Table 3.4.3).

Injuries to the knee ligaments resulted in the highest injury burden during the study. Specifically, injuries to the ACL exhibited the highest mean severity (with one exception - Table 3.4.3), whilst the MCL was one of the most frequently injured sites during the study whilst also exhibiting moderate to high magnitudes of mean severity. Muscular strain injuries to the hamstrings group also exhibited high magnitudes of injury burden - specifically biceps femoris muscle strain injuries sustained during match play which had one of the highest injury incidence rates and a moderate severity (Table 3.4.3). Injuries to ligaments around the ankle joint complex additionally resulted in high injury burden - specifically anterior talofibular ligament sprains and inferior tibiofibular syndesmosis injury, which had a high rate of occurrence and moderate severity (Table 3.4.3).

Table 3.4.3 Injury Incidence, severity and burden of as a function of high burden lower limb location and specific injuries.

	Total number of match injuries	Total number of training injuries	Match injury incidence (per 1000 player hours & 95% CI)	Training injury incidence (per 1000 player hours & 95% CI)	Total match injury severity (Days absent)	Total training injury severity (Days absent)	Mean match injury severity (Days absent & 95% CI)	Mean training injury severity (Days absent & 95% CI)	Match injury burden (Days injured per 1000 player hours & 95% CI)	Training injury burden (Days injured per 1000 player hours & 95% CI)
Posterior Thigh										
Biceps femoris muscle strain PT1	15	12	3.12 (1.67 - 4.58) PT3-5,K5,LL3-11,Ank3,4,6,7,8†	0.16 (0.04 - 0.27) PT5,K3,5,LL3,4,7,9,10,Ank4,5,7,8	631	446	42.07 (24.4 - 59.73) LL6,8,11,Ank7	37.17 (17.78 - 56.56)	154.12 (78.43 - 229.81) PT4,K4,LL2,4,6,8,11,Ank7†	8.7 (1.58 - 15.81) LL2,11,Ank4
Semimembranosus/ tendinosis muscle strain PT2	9	8	1.79 (0.13 - 3.44) LL3,Ank4	0.11 (0.04 - 0.17) PT5,K3,5,LL3,4,7,9,10,Ank4,5,7,8	245	354	27.22 (13.56 - 40.89) LL6,8	44.25 (8.69 - 79.81)	67.61 (25.67 - 109.54) LL6,8†	6.39 (2.89 - 9.89) K2,6,LL2,11,Ank4
Hamstring strain - muscle not identified PT3	2	3	0.43 (0.00 - 0.98)	0.03 (0.00 - 0.08)	459	202	19.00 - 440.00 LL8	67.33 (43.95 - 90.72) PT4,K2,6,LL1,4-6,Ank2,3	28.00 - 689.81 LL6,8†	8.2 (7.97 - 8.43) PT4,K2,6,LL1,2,4-6,8,11,Ank2-4
Hamstring spasm/cramps/trigger points with no clinical evidence of fibre disruption PT4	3	3	0.6 (0.04 - 1.17) LL5,Ank4	0.04 (0.00 - 0.09)	46	44	15.33 (2.67 - 28)	14.67 (2.85 - 26.48)	20.8 (4.47 - 37.13)†	1.85 (0.76 - 2.93) LL11,Ank4
Biceps femoris distal tendinopathy PT5	1	0	0.22 (0.00 - 0.66)†	0.00	33	0	33	0.00	51.5	0
Knee										
ACL Injury Kn1	7	3	1.44 (0.59 - 2.29) LL4,7,Ank4,6,8,9†	0.04 (0.00 - 0.08)	1711	1321	244.43 (197.64 - 291.21) PT1,2,4,K2-4,6,LL1,2,4,6,8,Ank2,3,5,7	440.33 (302.88 - 577.78) PT1-4,K2,4,6,LL1,4-6,8,Ank2,3,6,9	493.39 (321.49 - 665.29) PT1,2,4,K3,4,6,LL1,2,4,6,8,11,Ank2,5,7†	37.89 (30.04 - 45.74) PT1-4,K2,4,6,LL1-6,8,11,Ank2-4,6
MCL Injury K2	26	2	5.5 (2.49 - 8.50) PT3-5,K1,4,5,LL2-11,Ank3,4,6-9†	0.03 (0.00 - 0.06)	1410	39	54.23 (24.74 - 83.72) PT4,L2,L6,8,11,Ank7†	13.00 – 26.00	341.74 (228.6 - 454.88) PT1,2,4,K4,6,LL1,2,4,6,8,11,Ank2,5,7†	1.01 – 2.80 LL2,11,Ank4
PCL/Posterolateral corner injury Kn3	7	0	1.36 (0.23 - 2.50) LL5,Ank4†	0.00	558	0	79.71 (48.14 - 111.29) PT2,4,K4,6,LL1,2,4,6,8,Ank3,5,7	0.00	194.1 (135.53 - 252.68) PT2,4,K4,6,LL1,2,4,6,8,11,Ank5,7	0
Chondral/meniscal injury Kn4	5	5	0.98 (0.23 - 1.74) LL5,Ank4†	0.06 (0.01 - 0.10) PT5,K3,5,LL7,9,10,Ank5,7,8	138	332	27.6 (11.93 - 43.27) LL6,8	66.4 (20.03 - 112.77)	47.14 (26.29 - 68) LL6,8†	6.47 (1.35 - 11.6) LL2,11,Ank4
Patellofemoral/extensor mechanism Kn5	3	0	0.61 (0.00 - 1.46)†	0.00	304	0	101.33 (1.71 - 200.96)	0.00	221.25 (39.23 - 403.28) PT4,LL6,8	0
Other minor knee injury Kn6	11	3	2.31 (0.92 - 3.69) PT5,LL3,5,7-11,Ank4, 6,8,9†	0.04 (0.00 - 0.07)	226	46	20.55 (9.09 – 32.00) LL8	15.33 (7.48 - 23.18)	55.29 (26.17 - 84.41) LL6,8†	1.25 (0.68 - 1.82) Ank4
Lower Leg										
Medial gastrocnemius strain LL1	8	8	1.73 (0.5 - 2.96) LL5,Ank4†	0.1 (0.04 - 0.17) PT5,K3,5,LL3,4,7,9,10,Ank4,5,7,8	161	172	20.12 (9.42 - 30.83) LL6,8	21.5 (10.06 - 32.94)	47.99 (14.8 - 81.18) LL6,8†	3.04 (1.8 - 4.28) LL2,11,Ank4
Lateral gastrocnemius strain LL2	4	1	0.76 (0.00 - 1.73)	0.01 (0.00 - 0.03)	81	11	20.25 (11.75 - 28.75) LL6,8†	11.00	54.26 (43.04 - 65.49) PT4,LL6,8†	0.89 LL11,Ank4
Gastrocnemius strain - muscle subgroup not identified LL3	1	1	0.22 (0.00 - 0.66)	0.01 (0.00 - 0.03)	22	104	22.00	104.00	34.52 LL6,8†	8.38 PT4,K2,6,LL1,2,4-6,8,11,Ank2-4
Soleus muscle strain LL4	4	3	0.85 (0.26 - 1.44) LL5,Ank4†	0.04 (0.00 - 0.10)	84	61	21.00 (2.21 - 39.79)	20.33 (14.03 - 26.63)	31.28 (3.61 - 58.96)†	2.84 (2.31 - 3.36) K6,LL2,6,11,Ank4
Calf muscle strain - muscle not identified LL5	0	3	0.00	0.04 (0.00 - 0.10)	0	45	0.00	15 (7.93 - 22.07)	0	3.68⌂ K2,6,LL2,11,Ank4
Calf spasm/cramps/trigger points with no clinical evidence of fibre disruption LL6	2	2	0.45 (0.00 - 1.01)	0.02 (0.00 - 0.07)	14	24	5.00 - 9.00 LL8	1.00 - 23.00	7.84 - 14.05 LL8†	1.93⌂ K6,LL2,11,Ank4
Tibialis posterior strain LL7	1	0	0.19 (0.00 - 0.56)	0.00	28	0	28.00	0.00	36.95 LL6,8	0
Achilles tendon injury LL8	2	2	0.38 (0.00 - 0.85)	0.02 (0.00 - 0.05)	8	97	4.00 – 4.00 LL8	17.00 - 80.00	5.26 - 5.28	1.39 - 6.45 LL2,11,Ank4
Fractured midshaft fibula LL9	1	0	0.22 (0.00 - 0.66)	0.00	97	0	97.00	0.00	152.07 PT2,4,K4,6,LL1,2,4,6,8,11,Ank5,7	0
Fractured distal fibula LL10	1	0	0.22 (0.00 - 0.66)	0.00	111	0	111.00	0.00	173.24 PT2,4,K4,6,LL1,2,4,6,8,11,Ank5,7	0
Other lower leg injury LL11	9	1	1.88 (0.66 - 3.10) PT5,LL5,7,10,Ank4,6,8,9†	0.02 (0.00 - 0.05)	91	6	10.11 (2.48 - 17.74)	6.00	26.89 (9.52 - 44.26) LL8†	0.69 Ank4
Ankle										
Anterior talofibular ligament sprain Ank2	14	8	2.84 (1.38 - 4.31) PT3-5,LL3,5-10,Ank3,4,6,8,9†	0.09 (0.03 - 0.16) PT5,K3,5,LL2,3,7,9,10,Ank4,5,7,8	502	209	35.86 (19.47 - 52.25) LL6,8,11	26.12 (10.37 - 41.88)	118.3 (52.98 - 183.61) PT4,LL6,8,11,Ank7†	4.24 (2.46 - 6.02) K6,LL2,6,11,Ank4
Ankle lateral ligament sprain - ligament not identified Ank3	2	5	0.45 (0.00 - 1.32)	0.06 (0.00 - 0.12)	39	95	8.00 - 31.00	19 (8.91 - 29.09)	60.87 ⌂PT4,LL4,6,8,11,Ank7†	2.67 (1.17 - 4.17) LL2,11,Ank4
Calcaneofibular ligament sprain Ank4	0	1	0.00	0.01 (0.00 - 0.03)	0	1	0.00	1.00	0	0.08
Inferior tibiofibular syndesmosis injury Ank5	10	0	2.07 (0.87 - 3.27) PT5,LL3,5,7-10,Ank4,6,8,9†	0.00	370	0	37 (28.27 - 45.73) PT4,LL6,8,11,Ank7	0.00	106.53 (86.66 - 126.4) PT4,K4,6,LL1,2,4,6,8,11,Ank7	0
Ankle multiple ligaments sprain Ank6	1	5	0.19 (0.00 - 0.57)	0.06 (0.00 - 0.14)	80	255	80.00	51 (21.67 - 80.33)	108.3 PT4,K4,6,LL1,2,4,6,8,11,Ank7†	9.97 (1.97 - 17.97) K6,LL2,6,11,Ank4
Ankle deltoid ligament sprain Ank7	5	0	1.03 (0.03 - 2.04)†	0.00	56	0	11.2 (2.65 - 19.75)	0.00	27.63 (4.48 - 50.79)	0
Ankle Dislocation Ank8	1	0	0.19 (0.00 - 0.56) LL5,Ank4†	0.00	128	0	128.00	0.00	168.44 PT2,4,K4,6,LL1,2,4,6,8,11,Ank5,7	0
Other ankle/heel injury Ank9	1	2	0.19 (0.00 - 0.56)	0.02 (0.00 - 0.07)	7	111	7.00	11.00 – 100.00††	9.21 LL8	8.64⌂ PT3,4,K2,6,LL1-6,8,11,Ank2-4

* Denotes a statistically significant increase compared to all other injury locations of $p \leq 0.05$. † Denotes a statistically significant increase between match and training injury of $p \leq 0.05$ for specific injury location. †† Denotes a statistically significant increase between training and match injury of $p \leq 0.05$ for specific injury location. A combination of letters and numbers in superscript e.g. PT1 denotes a statistically significant increase compared to the corresponding injury (e.g. Biceps femoris muscle strain PT1) of $p \leq 0.05$ within match or training groups. ⌂ Denotes that all severity values used to calculate injury burden occurred within the same season and therefor are not presented with 95% CI. For severity and burden, when only 2 injuries occurred, both values are presented in place of mean (95% CI) e.g. Match Ank3. When only one injury occurred, a single figure is presented for severity and burden e.g. Match PT5.

3.4.4 Question 4. What are the inciting events that result in high burden injuries?

3.4.4.1 ACL & MCL injury

All injuries sustained to the ACL and MCL during match play were due to inciting events resulting from contact with another player rather than non-contact mechanisms (Figure 3.4.4.1). Injuries sustained during match play involving contact with another player resulted in larger magnitudes of injury burden ($P \leq 0.05$) and higher incidence rates ($P \leq 0.05$) for both ACL and MCL injuries compared to those sustained during training (Figure 3.4.4.1). No differences were observed between inciting events for ACL injury burden ($P > 0.05$) and incidence rate ($P > 0.05$) during match play, however being tackled resulted in greater magnitudes of mean severity compared to rucking during match play ($P \leq 0.05$). No differences were observed between contact and non-contact inciting events for ACL injury burden or incidence during training ($P > 0.05$) (Figure 3.4.4.1). Contact ACL injuries sustained during training appeared to have a higher injury burden and mean severity compared to non-contact ACL injuries, however statistical comparison was not possible due to the small sample of inciting events. For MCL injury, the inciting events that resulted in the highest magnitudes of MCL injury burden were being tackled during match play and rucks ($P \leq 0.05$), these inciting events also exhibited the highest incidence rates ($P \leq 0.05$). All injuries sustained to the MCL during training sessions were due to contact events (Figure 3.4.4.1). No differences were observed between inciting events for MCL injury during training ($P > 0.05$).

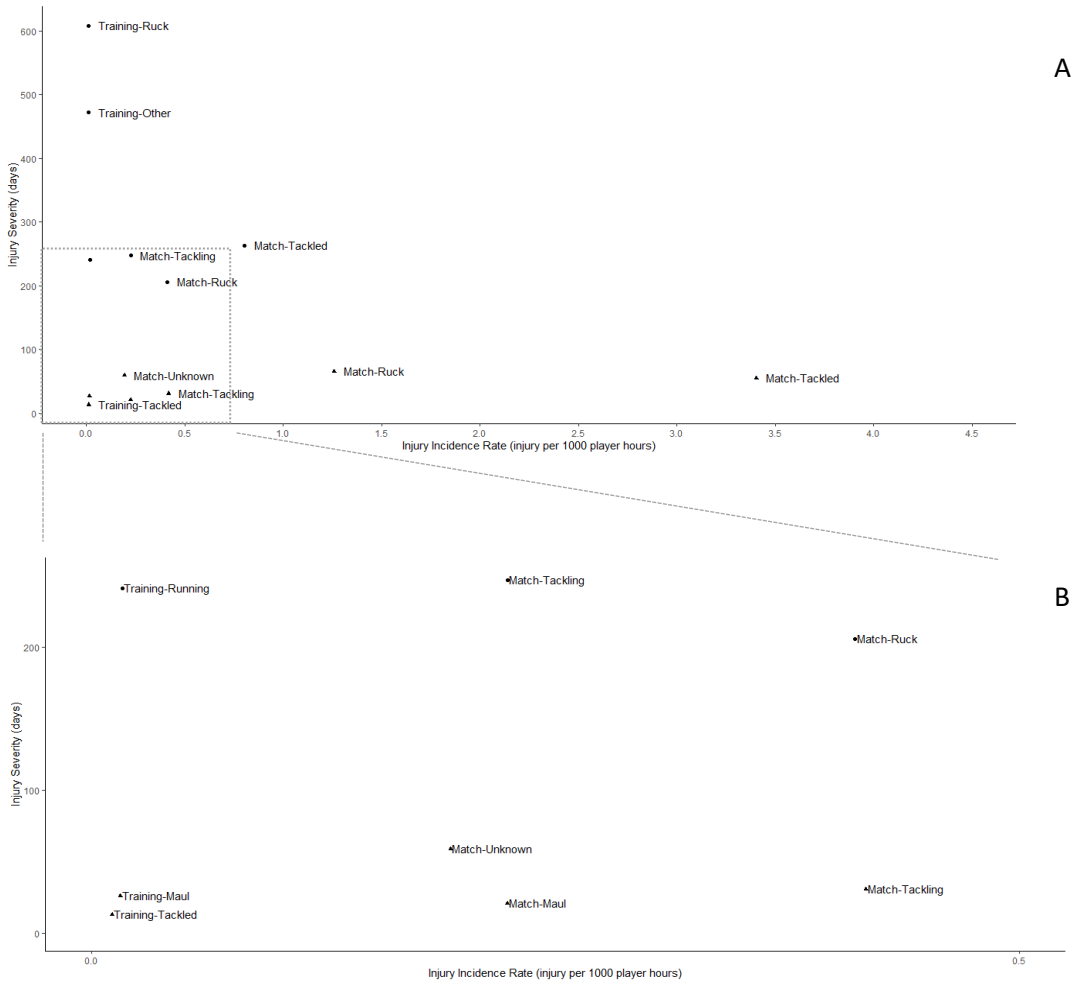


Figure 3.4.4.1 ACL & MCL injury burden as a function of inciting events for match play and training. Circles – ACL injuries, Triangles – MCL injuries. All ACL and MCL injuries are presented in A, whereas an expanded view of the dashed square area in figure A is presented in B for clarity.

3.4.4.2 Biceps femoris, Semimembranosus/tendinosis and hamstring muscle not identified strain

The biceps femoris was the most frequently injured muscle of the hamstrings muscle group (27 biceps femoris, 17 semimembranosus/tendinosis, 5 Unknown). Higher magnitudes of injury burden and incidence rates were observed for biceps femoris strains when running was the inciting event compared to tackling during match play ($P \leq 0.05$) (Figure 3.4.4.2); running also exhibited larger magnitudes of incidence rates compared to being tackled during match play (*P*

≤ 0.05), but not injury burden (*P* > 0.05) (Figure 3.4.4.2). No difference in burden or incidence rate was observed between running and rucking (*P* > 0.05). Furthermore, no differences in the mean severity of biceps femoris strain injuries were observed between inciting events during match play (*P* > 0.05). Running was the only inciting event resulting in biceps femoris strain injury (Figure 3.4.4.2).

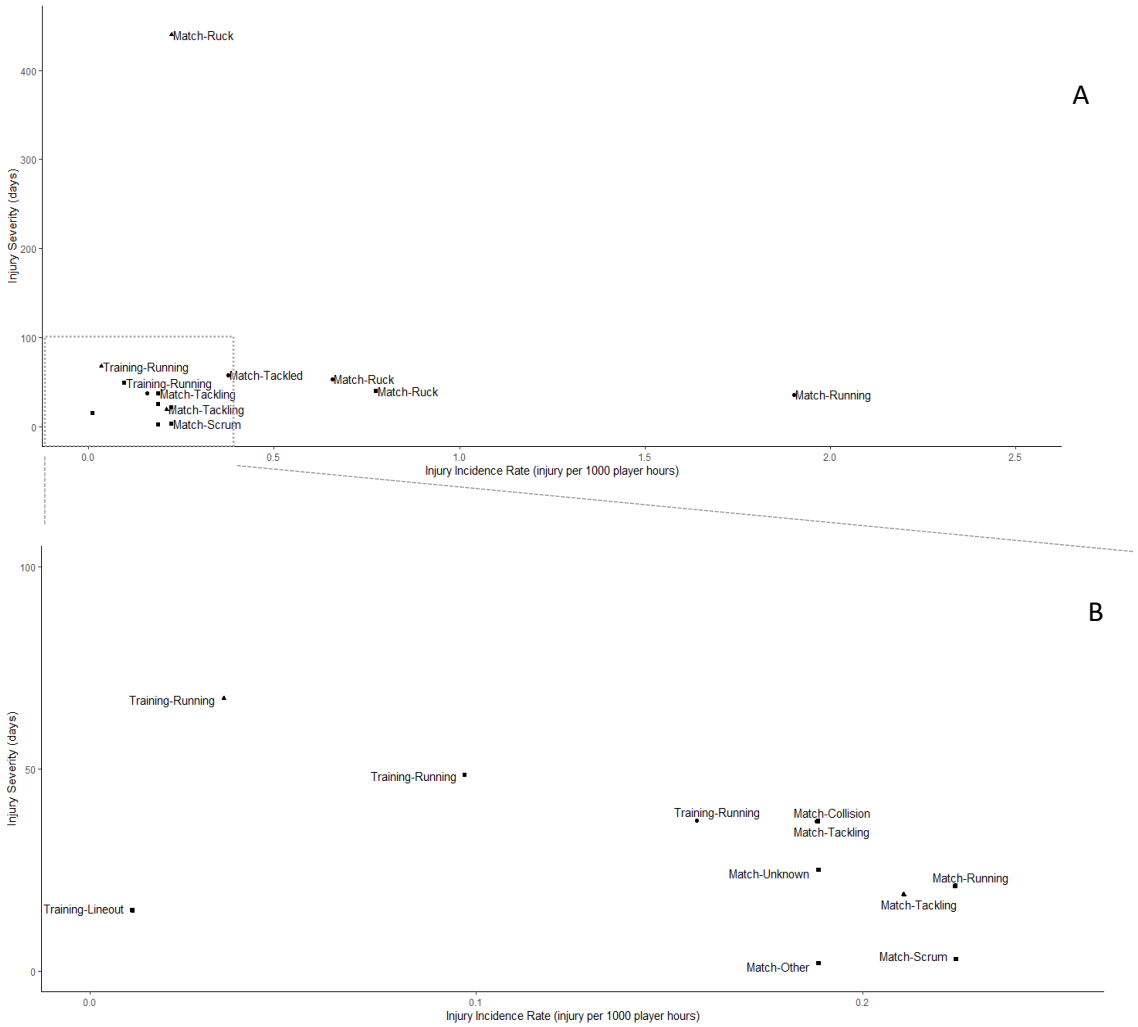


Figure 3.4.4.2 Biceps femoris & Semimembranosus/tendinosis injury burden as a function of inciting events for match play and training. Circles – Biceps femoris, Squares - Semimembranosus/ tendinosis, Triangles – Hamstring strain - muscle not identified. All hamstring strain injuries are presented in figure A, whereas an expanded view of the dashed square area in figure A is presented in figure B for clarity.

Semimembranosus/tendinosis strains sustained when rucking during match play resulted in the highest injury burden compared to all other match play Semimembranosus/tendinosis inciting events ($P \leq 0.05$) (Figure 3.4.4.2); injuries sustained from rucking also resulted in larger magnitudes of mean injury severity compared to all other inciting events with the exception of collisions ($P > 0.05$) (Figure 3.2). Furthermore, no differences in incidence rate were observed ($P > 0.05$). All semimembranosus/tendinosis strains sustained during training were non-contact in nature (Figure 3.4.4.2). Running was observed to exhibit larger magnitudes of semimembranosus/tendinosis strain injury burden, injury incidence rate and mean severity compared to landing from a lineout ($P \leq 0.05$) (Figure 3.2). A small number of hamstring muscle strains were sustained where the muscle subgroup was not identified (n = 5), of these rucking during match play resulted in the highest magnitudes of injury burden and mean severity when compared to tackling during match play and running during training ($P \leq 0.05$) (Figure 3.4.4.2).

3.4.4.3 Ankle lateral ligament & inferior tibiofibular syndesmosis injury

Anterior talofibular ligament sprains sustained from contact events during match play resulted in higher injury burden and larger magnitudes of mean severity compared to those sustained when running ($P \leq 0.05$) (Figure 3.4.4.3). Anterior talofibular ligament sprains sustained whilst being tackled during match play displayed higher incidence rates compared to running injury ($P \leq 0.05$) (Figure 3.4.4.3), there was no difference between all other inciting events ($P > 0.05$). No differences in injury burden, incidence or mean severity were observed between contact and non-contact inciting events resulting in anterior talofibular ligament sprains that occurred during training sessions ($P > 0.05$) (Figure 3.3). The only calcaneofibular ligament sprain occurred whilst rucking during training (Figure 3.4.4.3).

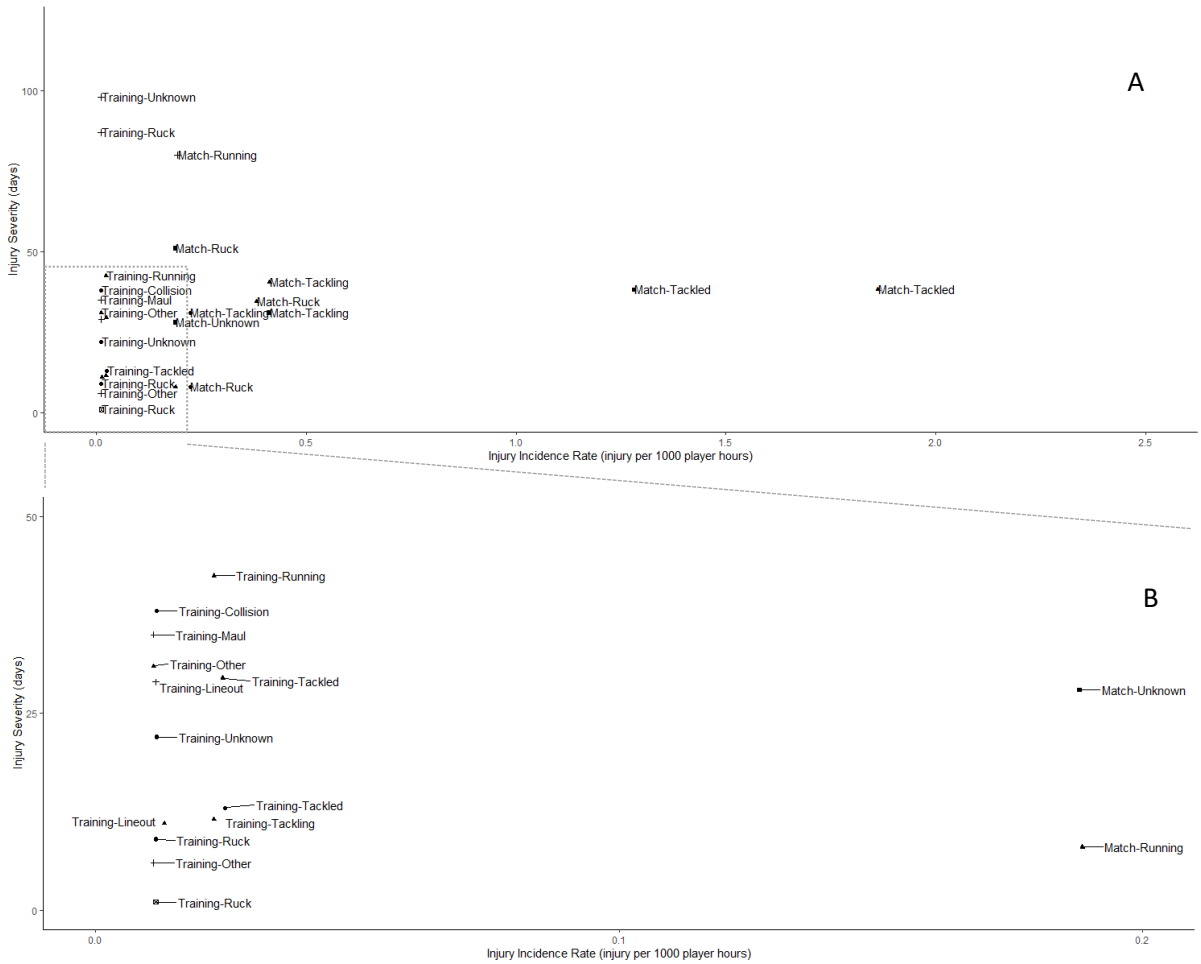


Figure 3.4.4.3 Ankle lateral ligament & inferior tibiofibular syndesmosis injury burden as a function of inciting events for match play and training. Circles – Lateral ligament sprain (ligament unidentified), Triangles – ATFL, Crosses – Multiple ankle ligaments sprain, Black Squares – AITFL, White Squares – Calcaneofibular ligament sprain. All ankle ligament injuries are presented in figure A, whereas an expanded view of the dashed square area in figure A is presented in figure B for clarity.

All lateral ankle sprains where the ligament was unidentified sustained during both match play and training sessions occurred during contact events, however comparison was not possible due to the small sample size of the subgroups (Figure 3.4.4.3).

Inferior tibiofibular syndesmosis injuries were only sustained during contact events whilst playing matches (Figure 3.4.4.3). Being tackled and rucking resulted in the largest magnitudes of injury burden compared to other inciting events ($P \leq 0.05$) (Figure 3.4.4.3). Being tackled

exhibited the largest magnitudes of injury incidence rates ($P \leq 0.05$) whereas rucking appeared to result in a higher mean severity (Figure 3.4.4.3).

3.5 Discussion

Establishing the extent of the injury problem is of primary importance when conducting sports injury research, as once areas of high burden and the subsequent injury mechanisms are identified, further prospective research can be conducted (van Mechelen et al., 1987; van Mechelen et al., 1992; Finch, 2006). This novel study has determined the incidence, severity and burden of lower limb injury with specific focus on the inciting events resulting in injury and anatomical areas affected. This represents an advance on previous literature which described injury in more general terms (Brooks et al., 2005a; Brooks et al., 2005b)

Injuries sustained during match play exhibited a higher injury burden and higher incidence rates compared with injuries sustained during training sessions regardless of injury location (Table 3.4.1). This finding has previously been observed in several prospective studies (Brooks et al., 2005a; Brooks et al., 2005b; Brooks et al., 2006; Fuller et al., 2010; Dallalana et al., 2007; Sankey et al., 2008; Kemp et al., 2018; Kemp et al., 2019), and confirmed in meta-analyses (Wiliams et al., 2013; Wiliams et al., 2021). A simple explanation for this finding is that the number of match exposure hours were much less than the hours of training exposure. As exposure is the denominator used for the calculation of both injury burden and injury incidence rate, smaller magnitudes of exposure will yield equivalent or larger burden or incidence rates despite smaller numerator values e.g. trunk injuries (Table 3.4.1). The increased burden and incidence of match injuries compared with training injuries observed in the current and other studies (Wiliams et al., 2013; Kemp et al., 2019) suggests that rugby matches may pose a different risk profile of injury compared to training due to the increased demands during contact events and greater running demands during a competitive match compared with training (Tee et al., 2016). Indeed, players are likely exposed to a higher frequency of more intense contact events during match play, whereas the contact events experienced on the training field such as tackles and rucks etc

will have procedures in place to mitigate any potential injury such as wearing padded tackle suits, using tackle shields or performing at submaximal intensities.

3.5.1 Question 1. Does lower limb injury present the highest injury burden compared to other gross locations within the cohort of players?

Injuries to the lower limb were observed to place the highest burden on the rugby team when compared to the upper limbs, trunk, and head & neck in the current study (Table 3.4.1). This higher magnitude of injury burden was due to the higher incidence of lower limb injuries compared to other locations rather than the mean severity of individual injuries (Table 3.4.1). This finding was consistent with previous studies examining injury epidemiology within men's professional rugby union (Williams et al 2013; Kemp et al., 2019).

3.5.2 Question 2. Which lower limb locations presented the largest injury burden during matches and training?

Over the course of the study, the lower limb location which sustained the largest number of injuries was the knee (13% of all injuries), followed by injuries to the posterior thigh (10%), ankle (10%) and the lower leg (10%) (Table 3.4.1 and 3.4.2). Although posterior thigh and ankle injuries have previously been observed to have high occurrence within injury surveillance projects in men's professional rugby union (Brooks et al., 2005a; Brooks et al., 2005b; Brooks et al., 2006; Fuller et al., 2010; Dallalana et al., 2007; Sankey et al., 2008; Kemp et al., 2018; Kemp et al., 2019), the occurrence of knee injuries was disproportionately high in the current study compared to earlier research, where anterior thigh injuries were most common during match play and training (Dallalana et al., 2007). However, more recent research examining injuries sustained by male professional rugby union players have reported knee injury incidence 95% confidence intervals of a similar range to those in the current study (Kemp et al., 2019). Injuries to the knee joint were also the most severe, resulting in 30% of the total time lost due to all injuries sustained within the study, with the posterior thigh (12%) and ankle (9%) being the

second and third most severe injuries respectively, whereas lower limb injuries resulted in only 6% of total time loss during the study period (Table 3.4.1 and 3.4.2).

3.5.3 Question 3. Which specific injuries present the greatest injury burden within each lower limb location? & Question 4. What are the inciting events that result in high burden injuries?

3.5.3.1 Knee injuries

ACL injuries sustained during matches and training and MCL injuries sustained during matches produced the highest injury burden in the current study (Table 3.4.3). However, the nature of the injury burden differed between ligaments: ACL injuries exhibited the highest average severities observed despite low numbers (n = 10), whereas MCL injuries were characterised by high rates of occurrence combined with high match injury severity (25 - 84 Days) and moderate to high training injury severity (13 - 26 Days). The difference in severity between ACL and MCL injuries observed in the current study as well as previous studies is due to the increased vascularisation of the MCL compared to the ACL (Jacobson, 2006; Kim et al, 2016). The mean severity for ACL and MCL injury observed in the current study were similar to previous research in men's professional rugby union conducted by Dallalana and colleagues (2007), and reported by the England professional rugby injury surveillance project steering group (Kemp et al., 2018). Although lower 95% confidence intervals of both ACL and MCL injury incidence rate overlapped with previously reported upper 95% confidence intervals (Dallalana et al., 2007; Kemp et al., 2019; Montgomery et al., 2018), the mean incidence was much higher than previously reported. Despite the larger magnitudes of exposure reported by Dallalana et al. (2007) compared to the current study for match play (by a factor of 3.46) and training (by a factor of 2.54) 10 ACL injuries and 28 MCL injuries were reported, suggesting a problem for this specific sample of players that will be explored in subsequent experimental chapters. All injuries to the ACL sustained during matches were due to inciting events where direct contact with another player was present (Figure 3.4.4.1). There were no differences in burden and incidence between events resulting in ACL injury during matches, whereas ACL injuries sustained during training sessions occurred via

both contact and non-contact mechanisms. Contact ACL injuries tended to be of higher severity and incidence compared to non-contact ACL injury, however there was only one non-contact injury so confidence intervals could not be calculated for proper comparisons to be made. A possible explanation for the lack of epidemiological differences observed between inciting events resulting in ACL injury was the low number of ACL cases (n = 10) compared to the number of inciting events (n = 6). Previous examination of ACL injury in men's professional rugby union is conflicting: for example, earlier research conducted by Dallalana and colleagues (2007), reported contact mechanisms of ACL injuries occurred at a higher proportion for matches (50%) compared to training (9%). In the same study, being tackled resulted in the highest proportion of ACL injury (43%) regardless of match or training, followed by tackling (29%) and changing direction (14%) (Dallalana et al., 2007). Later research examined video footage of ACL injuries sustained in men's professional rugby union matches. The authors reported the prevalence of contact ACL injuries during match play in men's professional rugby union were 14% higher than non-contact injuries. The two inciting events the authors identified as having the highest prevalence were the injured player being tackled and during deceleration change of direction movements during offensive running (Montgomery et al., 2018).

All MCL injuries during matches and training were sustained during contact events in the current study. Furthermore, the inciting events that resulted in the highest MCL injury burden during match play were being tackled (with the majority being tackled at the side) and around the ruck. The high injury burden observed from these inciting events was due to high incidence rates combined with moderate to high severities. These findings were supported by previous injury epidemiology research in men's professional rugby union (Dallalana et al., 2007) which also reported being tackled (38%) and rucking/mauling (25%) to have the highest proportion of MCL injury sustained during match play. MCL injuries sustained whilst playing other contact sports such as rugby league also indicates that being tackled is the most common inciting event for injury (Awwad et al., 2019). Conversely, no differences between specific inciting events were present during MCL injuries sustained whilst training (Figure 3.4.4.1). Although there was a lack

of difference in training MCL injury inciting events, the clear evidence from the match injuries inciting events, specifically being tackled from the side and during rucking, supports the proposed mechanism of MCL injury in the literature, i.e. an excessive tensile force acting on the MCL due to high magnitudes of valgus/abduction moment at the knee joint as a result of direct contact with the lower limb (Dallalana et al., 2007; Comfort and Abrahamson, 2010; Awwad et al., 2019).

3.5.3.2 Posterior thigh injuries

Hamstring muscle strain injuries also exhibited a high injury burden in the current study due to high incidence rates combined with moderate to high average injury severities (Table 3.4.2). Hamstring injuries have previously been reported to have one of the highest incidence rates in men's professional rugby union during matches and training (Brooks et al 2005a; Brooks et al 2005b; Brooks et al 2006). Indeed, the most recent PRISP report at the time of writing cited hamstring injuries as being consistently among the five most common match and training injuries, resulting in one of the highest magnitudes of injury burden (Kemp et al., 2019). The incidence rate for all hamstring muscle injuries from the present study was within the range reported by the most recent PRISP report at the time of writing during match play (4.68 hamstring injuries per 1000 player hours; 95% CI: 3.41 - 5.96) and training (0.36 hamstring injuries per 1000 player hours; 95%CI: 0.31 - 0.40) (Kemp et al., 2019). The injury burden values observed in the current study were also within the 95% confidence intervals reported by PRISP for match (167 days absence per 1000 player hours; 95% CI: 114 - 220) and training (10 days absence per 1000 player hours; 95% CI: 8 - 12) (Kemp et al., 2019). When individual hamstring muscles were analysed, no differences were observed between biceps femoris and semimembranosus/tendinosis strain injury burden, incidence and severity in the current study. This finding appeared to be in contrast with Brooks and colleagues (2006), who observed biceps femoris injuries had the higher prevalence whereas semimembranosus/tendinosis were reported to occur 43% less frequently. A limitation to Brooks and colleagues (2006) study was

that no incidence rates for specific hamstring injuries were presented, thus direct comparison is not possible. However, mean severities for individual hamstring muscles were reported by Brooks et al. (2006), which were smaller in magnitude than those reported in the current study. The reason for this disparity in mean severity between the current study and that of Brooks et al. (2006) was potentially due to the increased high-speed running demands for all players in the modern game. This is partially supported by later research presented by PRISP that showed similar magnitudes of mean injury severity for all hamstring injuries (Kemp et al., 2019). In the current study, biceps femoris injuries sustained in matches whilst running and rucking produced higher injury burden compared to tackling, due to higher incidence rates in the case of running (Figure 3.4.4.2). Contact based injuries to the semimembranosus/tendinosis during match play resulted in a higher injury burden compared to non-contact events, with injuries sustained whilst rucking being the most burdensome inciting event due to a higher incidence combined with a higher severity compared to other inciting events. During training sessions all biceps femoris and semimembranosus/tendinosis strains occurred via non-contact inciting events. Of these events, running presented the highest injury burden for both muscles. Comparison with previously reported inciting events resulting in specific hamstring injury was not possible because previous studies examining injury epidemiology in rugby union have only analysed epidemiology concerning inciting events leading to *all* hamstring injuries rather than specific muscles. In soccer, high speed running was reported to result in a higher frequency of biceps femoris injuries (Askling et al., 2013). Brooks et al. (2006) reported that for all hamstring muscle strains, running was the inciting event that resulted in the highest number of hamstring injuries and kicking was the event that resulted in the highest mean severity. Running was also the primary inciting event for all hamstring injuries in the latest PRISP report at the time of writing, with rucking and tackling the second and third most common inciting events. Rucking was also responsible for higher reported mean severities (64 days during the 2017-18 season) compared to running (32 days during the 2017-18 season) which was also observed in the current study (Kemp et al., 2019). Regardless of match or training, exposure to running appears to expose rugby union

players to hamstring injury risk; furthermore, although less frequently sustained, rucking may result in a hamstring injury of greater severity. In the presence of other risk factors, running and rucking events may subject the hamstring muscle fibres to injurious magnitudes of mechanical strains resulting in injury as a result of large external and internal forces (Hasselman et al., 1995; Järvinen et al., 2005; Kellis, 2018).

3.5.3.3 Ankle injuries

Injury to the lateral ligament complex of the ankle (anterior talofibular ligament, posterior talofibular ligament & calcaneofibular ligament) and the tibiofibular syndesmosis, presented a substantial injury burden in the current study (Table 3.4.2 & 3.4.3). This high injury burden was due to a combination of high incidence rates and moderate to high average severity, which were both within the 95% confidence intervals previously reported in rugby union by Sankey and colleagues (2008) for match and training injury. Furthermore, ankle injury burden was far greater for matches than training sessions, a finding that is in agreement with previous research and likely due to the increased exposure to contact situations associated with rugby matches compared to training sessions (Sankey et al., 2008). The ankle is complex and comprises multiple joints and ligaments; therefore, where possible analysis of individual ligaments may yield a better understanding of the injury problem. The anterior talofibular ligament (ATFL) presented a high injury burden within the current study; furthermore, it was one of the most frequently injured ankle ligaments (Table 3.4.3). The incidence rate and burden of ATFL injuries sustained during training were of similar magnitude to those previously reported in the most recent PRISP report at the time of writing (Incidence for 2017-18 season: 0.15 injuries per 1000 player hours; burden for 2017-18 season: 4.1 Days absence per 1000 player hours) (Kemp et al., 2019). Further comparison is limited because all ligaments of the lateral ankle ligament complex are frequently analysed together in rugby union epidemiological studies (Brooks et al., 2005a; Brooks et al., 2005b; Sankey et al., 2008). Injuries to the ankle lateral ligament complex in the current study were observed to present a large injury burden due to the high incidence rate and moderate to

high average severity of injuries (Table 3.4.3). The observed incidence rate was similar to the ankle lateral ligament injury incidence rate during matches (4.17 injuries per 1000 player hours; 95% CI: 3.30 - 5.27) and training (0.14 injuries per 1000 player hours; 95% CI: 0.10 - 0.20) previously reported by Sankey et al (2008); and those sustained during match play during the English rugby union premiership 2013-14 & 2015-16 seasons (2.0 - 2.9 injuries per 1000 player hours) (Kemp et al., 2019). The magnitudes of mean ankle lateral ligament injury severity were higher than previously reported (14 days absence; 95%CI: 11 - 17) by Sankey et al (2008). However, more recent research observed ankle lateral ligament injuries to be consistently among the three most common high severity injuries (28 - 84 days absence) sustained within training sessions by rugby players competing in the English premiership which is in agreement with the findings of the current study (Kemp et al., 2019). It is unknown why the disparity exists between the earlier findings of Sankey et al (2008) and the more recent findings of the present study and the most recent PRISP report at the time of writing (Kemp et al., 2019). Possible explanations may be either a more conservative approach to rehabilitation, a higher severity of the same injury sustained, or a combination of both. However, it is clear that further research is needed.

Contact injuries to both the ATFL and all lateral ankle ligaments during matches exhibited a substantially higher injury burden when compared to non-contact injuries due to a higher incidence and higher magnitudes of mean severity (Figure 3.4.4.3). Analysis of inciting events revealed the tackle presented the highest ATFL and all lateral ligament injury burden (Figure 3.4.4.3). There were no differences between contact and non-contact injuries or inciting events during training sessions, which was likely a result of the low rate of occurrence. This was consistent with Sankey et al. (2008) who observed a higher proportion of lateral ankle ligament injuries were sustained during contact situations, specifically when the injured player was being tackled. The findings of the current study, in combination with previous research, suggest that contact events, specifically the tackle, may expose rugby players to a higher risk of lateral ankle ligament sprain (Sankey et al., 2008). How this is the case still remains unclear although the

identified mechanisms for lateral ligament injury suggest that the ligaments are placed under extreme magnitudes of stress when the foot is rapidly inverted whilst in plantar flexion either in isolation or combined with internal rotation of the shank (Kristianslund et al., 2011). During contact events, in order to maintain possession of the ball and to gain as much territory as possible, players will often try to move or evade after the initial contact, sometimes resulting in the player being wrestled to the ground following a tackle - these rotational movements may promote the optimal conditions for lateral ankle sprains. Injuries to the anterior inferior tibiofibular syndesmosis (AITFL) presented a high injury burden. These injuries were only sustained during match play in contact situations (Table 3.4.3 & Figure 3.4.4.3). In a similar fashion, ankle ligament epidemiological data reported by Sankey and colleagues were smaller in magnitude (Mean severity 22 Days absent; 95% CI: 14 - 34; match incidence 1.13 injuries per 1000 player hours; 95% CI not reported) compared to the current study (Sankey et al., 2008). However, the findings of the present study are more consistent with those reported by PRISP, who reported 3.8 match injuries per 1000 player hours for the 2012-13 season, and similar values of match injury burden (123 days absence per 1000 player hours 95% CI: 105 - 142) (Kemp et al., 2014; Kemp et al., 2019). Furthermore, injuries to the AITFL have previously been reported to be 4th most common injury sustained whilst being tackled in English premiership rugby union (Kemp et al., 2019). Although a trend in being tackled was observed to be the most burdensome inciting event in the current study, this was not statistically significant (Figure 3.4.4.3).

The present study had several limitations and assumptions. In the greater context of epidemiological studies conducted within rugby union, the majority have analysed multiple teams competing within a league (Brooks et al., 2005a, 2005b; Brooks et al., 2006; Dallalana et al., 2007; Sankey et al., 2008; Kemp et al., 2018; Kemp et al., 2019). In comparison, the sample size (one team and ~59 players per year) within the current study was small. However, due to one professional rugby team co-funding the research project, this was unavoidable. As mentioned throughout the discussion, the findings of the current study are within the most recently reported 95% confidence intervals for epidemiological data presented by PRISP (Kemp

et al., 2019). An additional limitation to the study was that injury data were not analysed separately for forwards and backs. Although the seminal work of Brooks and colleagues (2005a & 2005b) and other previous studies (Brooks et al., 2006; Dallalana et al., 2007; Sankey et al., 2008) have examined the incidence and severity of injuries separately between forwards and backs, the purpose of the was to focus on the epidemiology of specific injuries that presented the highest burden and subsequently examine the inciting events responsible. Justification for this approach was provided by Williams et al. (2013), who found only a trivial difference in incidence and severity of injuries between forwards and backs when conducting a meta-analysis. Therefore, one should first examine the risk associated with inciting events of injury which would in turn influence the risk associated with each specialised position.

3.6 Conclusions

This study is unique in comparison to previous rugby union injury epidemiology studies because particular focus has been paid to the inciting events that caused high burden injuries rather than just reporting proportions. The findings of the study are in agreement with previous research that lower limb injuries present the highest burden in men's professional rugby union, and indicate that injuries to the knee, posterior thigh and ankle joint pose the most injury burden. However, injury to the lateral ankle ligaments and AITFL a large variation in inciting events, combined with a low number of injured cases per inciting event, suggesting more epidemiological research is required before aetiological research can be conducted. In addition to highlighting the above areas of focus, specific injuries have also been highlighted for subsequent aetiological research, namely: ACL and MCL sprains, hamstring muscle strains. Of the previously mentioned high burden injuries, matches presented a larger injury burden compared to training sessions. Furthermore, contact with other players appeared to result in the clear majority of ligament sprains and subsequent time loss, whereas muscle strains were observed to occur more frequently during running.

Next steps

The key areas of injury burden have now been established for the rugby union team within the context of this thesis, with the findings being in broad agreement with previous rugby union injury epidemiology. With this initial work accomplished, it is possible to take a more focussed approach to examine the variables that are related to the injuries identified as posing the highest burden. In order to accomplish this, the following steps will be conducted:

1. A further literature review, that will examine existing research pertaining to ACL & MCL sprains and hamstring muscle group strains, where possible in the context of men's professional rugby union. The literature review will include:
    . Anatomy and physiology
    a. Pathophysiology
    b. Previously reported risk factors previously associated with rugby union and other team sports
    c. Methodological challenges associated with each injury
2. Synthesis of the review of literature to create research questions to conduct prospective study of risk factors associated with knee ligament and hamstring injury risk.

Chapter 4: Literature review part 2: A review of hamstring and knee ligament aetiological literature in rugby union and other team sports

4.1 Overview

The purpose of Chapter 4 was to summarise the literature underpinning the aetiology of the high burden lower limb injuries identified in Chapter 3. Specifically, it addressed literature related to the aetiology of hamstring muscle strains in addition to knee joint anterior cruciate ligament (ACL) and medial collateral ligament (MCL) sprains in professional rugby union. The aetiology of lower limb injuries is a complex problem highlighted in numerous theoretical models of aetiology discussed in Chapter 2 (Meeuwisse, 1994; Gissane et al., 2001; Meeuwisse et al., 2007; Windt and Gabbett, 2016; Bittencourt et al., 2016). In order to conduct aetiological research, a comprehensive understanding of the anatomy and function of the areas of interest is necessary, in conjunction with the pathophysiology of the injury of interest, the mechanism of injury and the inciting events that resulted in the injury. This need is more pertinent when planning aetiological research in an area that has previously received little focus (such as MCL injury), requiring the researcher to draw on first principles. Therefore, brief sections have been included to discuss the above areas. Methodological issues associated with conducting injury aetiology research will also be discussed. It is important to note that the scope of aetiological experimental chapters of the research project was constrained by the data collection equipment already available within the University of Exeter Sport and Health Sciences department and the Exeter Chiefs RFC. Due to this sections, 4.2.4 and 4.3.4 of this chapter will focus only on aetiological variables employing similar data collection methodologies. The literature review will provide a rationale for undertaking the prospective research in the subsequent experimental chapters.

4.2 Aetiology of hamstring muscle strain injuries

Hamstring strain injuries have been reported to present a high burden in professional rugby union epidemiology research (Brooks et al., 2006; Kemp et al., 2018; Kemp et al., 2019; Chapter 3 – Study 1). The purpose of the following section is: to provide a summary of the anatomy, pathophysiology and mechanisms of hamstring strain injury, highlight aetiological factors that have previously associated with injury risk and finally present some of the unique methodological challenges posed when conducting hamstring injury aetiological research in the context of men's professional rugby union.

4.2.1 Anatomy and function of the hamstrings

The term 'hamstrings' refers to three muscles which are located in the posterior compartment of the thigh: the biceps femoris (comprising a long head and short head) the semitendinosus and semimembranosus. The hamstrings are biarticular muscles, crossing both the knee and the hip joint, with the exception of the short head of biceps femoris, that only crosses the knee. Concentric contraction of the hamstrings muscle group results in flexion of the knee joint and extension of the hip (Marieb and Hoehn, 2007; Woodley and Mercer, 2005).

4.2.1.1 Biceps femoris muscle structure and function

Biceps femoris consists of a long and a short head (Figure 4.2.1.1). The long head is a bipennate biarticular muscle, which originates from the lateral quarter of the medial facet of the ischial tuberosity, it is also directly attached to the sacrotuberous ligament (Woodley and Mercer, 2005). The distal tendon blends with the short head of biceps femoris and inserts into the lateral and anterior aspects of fibular head and the tibial plateau and the lateral collateral ligament (Chelboun et al., 2001). The long head exhibits the second largest physiological cross-sectional area (PCSA) of the hamstring muscle group (Woodley and Mercer 2005).

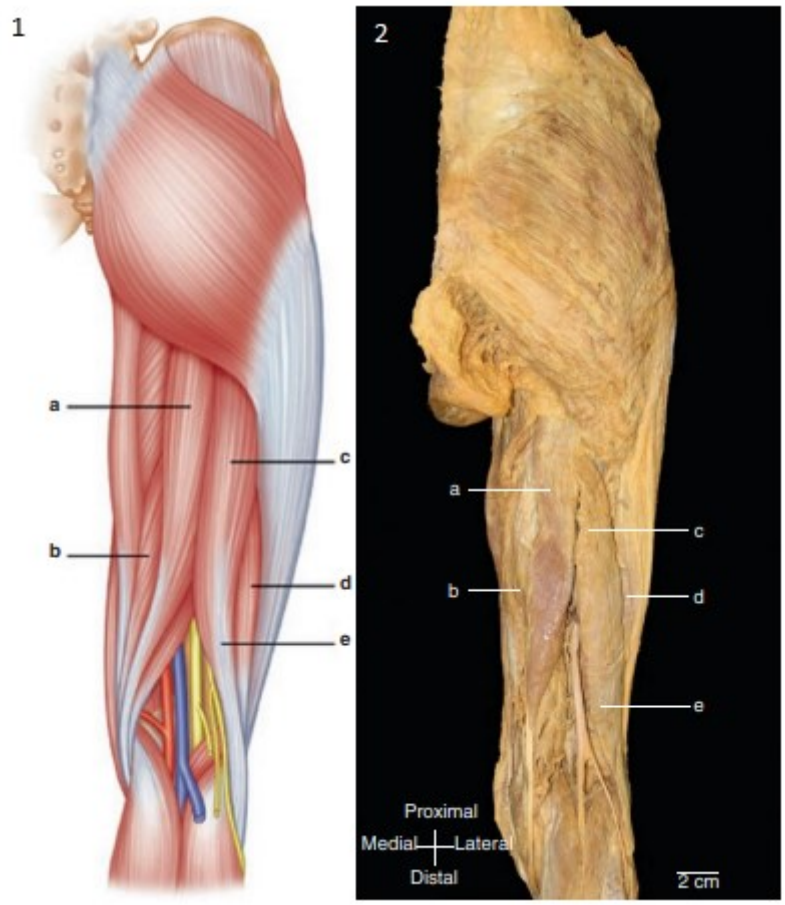


Figure 4.2.1.1 An illustration of the hamstrings muscle group, comprised of a) semitendinosus, b) semimembranosus, c & e) long head of biceps femoris and d) short head of biceps femoris depicted in 1) schematic illustration and 2) dissection photograph. Adapted from Kaeding and Borchers (2014).

The short head of biceps femoris is a monoarticular muscle which only crosses the knee joint with concentric contraction contributing to knee joint flexion. It does not have a proximal tendon, instead it arises from bony and fascial attachments from the length of the lateral lip of the linear aspera, the upper two thirds of the lateral supracondylar line and the lateral intermuscular septum (Kellis et al., 2010). The distal insertion of the short head of the biceps femoris is complex, the short head inserts into the long head of the biceps femoris, the posterolateral aspect of the lateral collateral ligament, insertion to the iliotibial band, the posterolateral aspect of the fibular head, the proximal and lateral tibia (Terry and LaPrade, 1996). The short head of biceps femoris has the smallest PCSA relative to all other muscles of the hamstrings group despite exhibiting the longest fascicle lengths compared to the other

hamstrings (Kellis, 2018), which when viewed in combination suggests that the muscle may only be capable of exerting small magnitudes of force (Kellis, 2018).

4.2.1.2 Semimembranosus structure and function

The semimembranosus is a biarticular muscle that comprises one of the 'medial' hamstrings (Figure 4.2.1.1). It has a unipennate arrangement of fibres in the proximal region, whereas the distal region is bipennate (Woodley and Mercer 2005). The proximal tendon of the semimembranosus originates at the lateral part of the ischial tuberosity. The distal fibres of the semimembranosus insert into a broad aponeurosis which has three main attachments: the posterior aspect of the medial tibial condyle, a portion blends with the popliteal fascia and the oblique popliteal ligament (Kellis, 2018). The semimembranosus exhibits the largest PCSA of the hamstrings group (Kellis, 2018).

4.2.1.3 Semitendinosus structure and function

The semitendinosus is the second of the 'medial' hamstring muscles (Figure 4.2.1.1). It is morphologically considered to be a single muscle biarticular muscle with a fusiform arrangement of fibres (Kellis, 2018). It is also characterised by a tendinous inscription dividing the muscle belly into superior and inferior portions (Woodley and Mercer 2005). The proximal semitendinosus fibres originate from multiple sites: the posteromedial facet of the ischial tuberosity via thick connective tissue, the medial border of the proximal biceps femoris long head tendon and an aponeurosis arising from the proximal biceps femoris long head tendon (Woodley and Mercer 2005; Kellis et al 2010). The distal fibres of semitendinosus insert into the medial surface of the tibia. Architecturally, the semitendinosus exhibits long and thin fibres, which are the longest of all the hamstring muscles, combined with the second smallest PCSA of the hamstring muscle group (Woodley and Mercer 2005).

4.2.2 Definition and pathophysiology of hamstring strain injury

In general terms muscular strain injuries occur in the presence of large mechanical stresses acting on the muscle that produce excessive mechanical strains above the magnitude the myofibrils are able to tolerate, which consequently results in partial to complete disruption of muscle fibres and is typically associated with the instantaneous onset of pain in the area of the injured (Garret et al., 1987; Lieber and Fridén, 1993; Järvinen et al, 2005). The severity of muscle strain injuries is dependent on the magnitude of mechanical stress applied in combination with the magnitude of strain experienced (Askling, Saartok and Thorstensson, 2006), the location of the injury (e.g. proximal aponeurosis, proximal bony origin, musculotendinous junction, muscle belly or the point of distal bony insertion) and finally, the physical integrity of the muscle at the time of injury (Agre, 1985).

4.2.3 Inciting events and mechanisms of hamstring strain injury

The following section will examine theoretical mechanisms of hamstrings muscle strain injury in conjunction with the inciting events resulting in injury associated with the mechanisms whilst participating in rugby union.

Hamstring strain injuries are subject to the same basic pathophysiology as other any other muscle group i.e. mechanical stress (force per unit area) is applied to the myofibrils which then results in large mechanical strains (relative deformation of the ligament) that exceed the capacity of said myofibrils causing rupture, this is particularly prevalent during eccentric contractions (Garret et al., 1987; Lieber and Fridén, 1993; Green et al., 2020). Previous studies have reported two distinct mechanisms of hamstring strain injury which place either large magnitudes of mechanical stress (Orchard, 2012; Liu et al., 2017), or large magnitudes of mechanical strain as the primary driver of injury (Askling et al., 2006; Askling et al., 2007a; Askling et al., 2007b). These mechanisms are commonly classified as either ‘sprint-type’ mechanisms due to their propensity to occur from an inciting event involving high-speed running

(large magnitudes of mechanical stress) (Orchard, 2012; Liu et al 2017); or 'stretch-type' mechanisms characterised by end range knee extension and hip flexion (large magnitudes of mechanical strain) (Askling et al., 2006; Askling et al., 2007a; Askling et al., 2007b; Askling et al., 2013).

4.2.3.1 Sprint-type hamstring injury mechanism

The sprint-type mechanism is characteristic of either high magnitudes of internal or external forces resulting in an eccentric overload of the injured muscle. Hamstring muscles have been observed to produce large magnitudes of internal force terminal swing phase of running the running gait cycle, in order to decelerate the tibia and fibula to control knee extension whilst also decelerating the femur to control hip flexion, prior to the foot striking the ground (Figure 4.2.3.1) (Hoskins and Pollard, 2005; Chumanov et al., 2011; Chumanov et al., 2012; Kenneally-Dabrowski et al., 2019). This mechanism is the more prevalent in biceps femoris injury (Askling et al., 2013). A partial explanation for this was provided by Thelen and colleagues (Thelen et al 2005a; Thelen et al 2005b) who observed that during sprinting, the long head of the biceps femoris is subjected to the largest magnitudes of muscle-tendon unit strain of the hamstrings group (110% of resting length); whereas the medial hamstrings (semimembranosus and semitendinosus) exhibit smaller magnitudes of muscle-tendon unit strain (107% and 108% of resting length). This is compounded by the long head of biceps femoris exhibiting a shorter moment arm around the knee compared with the medial hamstrings, resulting in a lower capacity to generate moments at longer MTU lengths (Thelen et al 2005a; Thelen et al 2005b; Thelen et al., 2006). During the terminal swing phase, fibres of long head of biceps femoris must produce larger magnitudes of muscle force and EMG activation than the medial hamstrings during the eccentric contraction characteristic of the terminal swing phase (Silder et al., 2010).

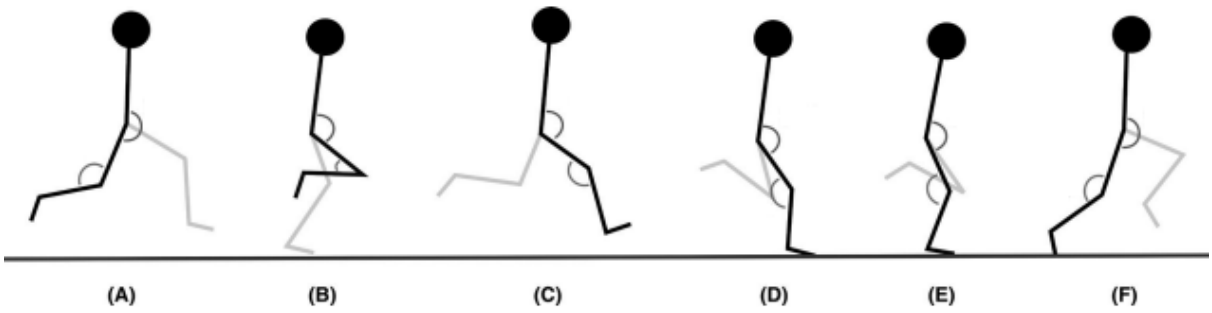


Figure 4.2.3.1 Running gait cycle. A) early swing, B) mid swing, C) late swing, D) early stance or foot strike, E) mid stance and F) late stance or toe-off Adapted from Kenneally-Dabrowski et al. (2019)

The other manner in which a sprint-type hamstring injury mechanism may occur is via the hamstring being subjected to large magnitudes of external force such as large magnitudes of ground reaction force resulting in large external extension moments at the knee joint and flexion moments at the hip when an individual's leg forcefully coming into contact with the ground during the initial contact portion of the gait cycle when running at high speed or indeed rapidly altering speed (Schache et al., 2011; Schache et al., 2012; Orchard, 2012; Kenneally-Dabrowski et al., 2019). To mitigate these external forces, the hamstring muscles assist in producing internal flexion moments at the knee joint and extension moments at the hip joint (Sun et al., 2015; Liu et al 2017). Indeed, a second peak in hamstring muscle activity was observed during early stance (the first peak terminal swing) (Yu et al., 2008; Chumanov et al., 2011), which is accompanied by maximum internal hip extension and knee flexion moment; and a secondary peak in the long head of biceps femoris muscle force (the primary again occurring in terminal swing) (Schache et al., 2012; Nagano et al., 2014).

Recent reports from the English professional rugby injury surveillance project (PRISP) highlighted that the most frequent mechanism of hamstring injury was running during matches resulting in an average 32 days severity (Kemp et al., 2017; Kemp et al., 2018). The PRISP reports did not specify which muscles of the hamstring group were injured, however, previous studies in other team sports such as soccer have observed the long head of the biceps femoris as the

most frequently injured muscle during high-speed running (Askling et al., 2013), providing indirect support.

4.2.3.2 Stretch-type hamstring injury mechanism

The stretch-type mechanism of injury occurs during movements where the hip joint is maximally flexed and knee is maximally extended, resulting in large magnitudes of mechanical strain (Askling et al 2006; Askling et al 2007a, 2007b; Askling et al., 2013). Magnetic resonance imaging studies have reported that this mechanism primarily affects the medial hamstrings, specifically the semimembranosus (Askling et al., 2006; Askling et al., 2007a). The semimembranosus displays greater local muscle stiffness compared to the other hamstring muscles, which is theorised to be a result of the relatively shorter fascicle lengths which may explain the susceptibility of muscle strains in positions of hip flexion couple with knee extension, compared to the more elastic long head of biceps femoris (Kellis, 2018).

A common inciting event in ruby union resulting from the stretch-type mechanism is during rucking, where players compete for possession of the ball following a tackle. The ball is in simplistic terms stationery and players will attempt to drive over the ball to gain possession. During these rucking and counter-rucking movements individuals are usually in a posture of large magnitudes of knee extensions and hip flexion with the torso inclined pushing against the ground and opponents. An extreme position during this contest is the jackaling position presented in figure 4.2.3.2 where a player tries to obtain possession of the ball and shield whilst in extreme hip flexion combined with knee extension. Injuries may arise when another player collides with the torso of the player in the jackal position, this external force is theorised to result in large external hip flexion and knee extension moments resulting in large magnitudes of mechanical strain exceeding the capacity of the injured muscle. Rucking and tackling have previously been highlighted as the second and third most common hamstring injury mechanisms occurring during match play (Kemp et al., 2018).

Figure 4.2.3.2 Example of the jackal rucking position (The standing player wearing red), comprised of deep hip flexion combined with extended knees. Photo courtesy of Exeter Chiefs RFC.

4.2.4 Variables associated with Hamstring strain injury aetiology

The following section will examine the existing literature concerning variables that have been previously reported as being associated with hamstring strain injury risk. Where possible, studies that with rugby union players as participants will be highlighted, however this is not always possible and in this case studies that have used participants from team sports will be used. The section was split into the following sections: non-modifiable intrinsic risk factors, modifiable intrinsic risk factors and external risk factors.

4.2.4.1 Non-modifiable intrinsic variables associated with hamstring strain injury aetiology

4.2.4.1.1 Previous injury

Numerous studies have observed that individuals are at a higher risk of sustaining a lower limb injury if they have previously sustained a lower limb injury (Gabbe et al., 2006; Hagglund et al., 2006; Orchard, 2001; Williams et al., 2017). Within the context of hamstring injury aetiology, a recent meta-analysis by Green and colleagues reported that previous hamstring injury was consistently associated with an increased risk of subsequent hamstring injury (Green et al., 2020). Within a male professional rugby union population, hamstring injuries were observed to have the third highest proportion of re-injury (Williams et al., 2017). Furthermore, there appears to be a higher risk of subsequent hamstring injury if the previous hamstring injury occurred more recently (i.e. within the same playing season) (Green et al., 2020). Said injury may result in structural and neurological maladaptation within the injured muscle including: reduced fascicle length (Timmins et al., 2016), atrophy (Sanfilippo et al., 2013), increased collagen formation (Silder et al., 2008) and reduced voluntary activation (Fyfe et al., 2013). If an injured individual does not follow an appropriate period of rehabilitation these maladaptations may reduce the hamstrings' ability to tolerate high magnitudes of stress and strain, contributing to an increased risk of re-injury (Williams et al., 2017).

Previous injury to other areas of the lower limb has also been observed to increase hamstring injury risk. Green and colleagues (2020) observed that individuals with a history of ACL injury had a 70% increase in hamstring injury risk. The mechanisms underpinning this increased risk are unclear, however, the reduced proprioception, strength deficits and altered gait that are associated with ACL injury may have an effect (Katayama et al., 2004; Abourezk et al., 2017; Tashman et al., 2004; Bourne et al., 2019). A previous triceps-surae strain was also found to increase risk of hamstring strain by 50% (Green et al., 2020). The authors suggested that a possible explanation for this was due to the reduction in exposure to high-speed running resulting in deconditioning of the hamstrings muscles (Duhig et al 2016).

4.2.4.2 Modifiable intrinsic variables associated with hamstring strain injury aetiology

4.2.4.2.1 Isokinetic measures of hamstrings and quadriceps strength

The force producing qualities of the hamstrings muscle group is one of the most commonly researched modifiable internal risk factors in the field of hamstring strain aetiology research (Green et al., 2020). Isokinetic assessment of knee flexion and extension has been the principal method to evaluate hamstring strength and has been extensively researched in relation to hamstring injury risk within sporting populations for the past ~25 years (Orchard et al., 1997; Green et al., 2020). Using this methodology, hamstring strength is approximated from the internal moment applied by the isokinetic dynamometer to ensure the chosen velocity of the dynamometer attachment remains constant in response to the sum external joint moment applied by the test participant (Figure 4.2.4.2.1) (Arampatzis et al., 2004; Baltzopoulos et al., 2012).

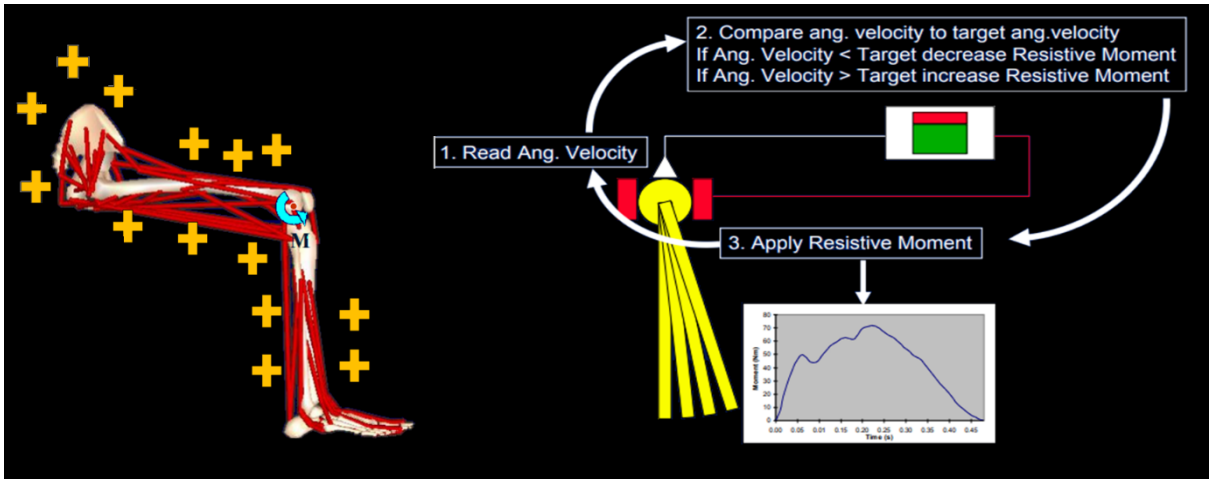


Figure 4.2.4.2.1 A representation of the approximation of hamstring and quadriceps strength using isokinetic dynamometry.

Recently, meta-analyses have reported that isokinetic variables have little relationship with the risk of sustaining a hamstring injury (Figure 4.2.4.2.1) (Green et al., 2017; Green et al., 2020). With prospective studies reporting either isokinetic knee flexion variables either having no effect (Bennell et al., 1998; Zvijac et al., 2013), or a small decrease in risk (van Dyk et al., 2016). The

only isokinetic variable identified to increase the risk of hamstring injury by Green and colleagues was increased peak knee extension moment (Green et al., 2017; Green et al., 2020). Despite strong evidence of limited effectiveness, lower limb isokinetic assessment is still a foundational part of the decision-making process regarding hamstring injury risk in professional team sports such as the participating professional rugby union team and is therefore routinely conducted during periodic health assessments.

4.2.4.3 Extrinsic variables associated with hamstring strain injury aetiology

4.2.4.3.1 Player Workload

A limited number of studies have investigated whether a relationship exists between player workloads and lower limb injury in professional rugby union with conflicting results and differing methodologies for workload quantification, however the majority of these studies did not specifically examine whether a relationship exists between workload and hamstring strain injury so will not be discussed further in this section (Lee, Garraway and Arneil, 2001; Brooks et al., 2008; Cross et al., 2016; West et al., 2020; West et al., 2021a). The only study to date to specifically examine whether a relationship exists between player workloads and the risk of hamstring strain injury was conducted by Brooks and colleagues (2006). An increased likelihood of sustaining a minor or moderate hamstring muscle injury (≤3 weeks' absence) during a match as the volume of training performed by the club in the preceding week increased (Brooks et al., 2006). Furthermore, when very high training volumes (of more than 12.5 hours per week) were undertaken in the preceding week, the likelihood of sustaining a major hamstring muscle injury (> 3 weeks severity) during a match also increased (Brooks et al., 2006).

Since the advent of affordable microtechnology, there has been an explosion of the integration of player workload into sports injury aetiology research. This has led to the adaption of sports injury aetiology models to include player workload not only as the primary vehicle for exposure to injurious events but also as a means of factoring in individual's other physical characteristics

associated with the both the positive and negative effects of training/physical activity termed by Windt and Gabbett (2016) as fitness and fatigue. The proliferation of microtechnology devices available has resulted in a plethora of different data collection modalities (e.g. GPS, Inertial measurement units, heart rate monitors) available to the sports medicine and conditioning staff within professional rugby union teams (West et al., 2019); in addition to other team sports and researchers within the field. Of these various options there are a multitude of different variables within each modality which may inform the end user of different aspects of workload, for example a GPS device may record both the total distance travelled regardless of velocity and also the total distance travelled within a specific speed zone for the same session. This wealth of information and options poses a challenge to applied practitioners working with sports teams and sports science researchers to know which modality and variables to select for use with field-based team contact sports such as professional rugby union (West et al., 2019).

4.2.4.3.2 High-speed running exposure

High-speed running is a frequently reported inciting event for hamstring strain injuries in pitch-based team sports such as rugby union (Brooks et al., 2006; Kemp et al., 2019; Chapter 3 – Study 1). As a result, researchers and practitioners have proposed that unaccustomed increases in the exposure of high-speed running is a key variable in the aetiology of non-contact hamstring strain injuries (Brooks et al., 2006; Green et al., 2020; Stokes et al., 2020). However, despite this proposed relationship, and the mass adoption of microtechnology based player workload monitoring systems in professional rugby union, no studies exist examining whether a relationship exists between data derived from player workload monitoring technology and hamstring strain injury in rugby union at the time of writing (2022). Nevertheless, studies have examined the existence of a relationship between high-speed running exposure and hamstring strain injury in relation to Australian rules football (Dulig et al., 2016; Ruddy et al., 2018). Acute exposure to high-speed running was observed to be associated with an increased risk of hamstring strain injury risk, when aggregated over a time period of 7 days prior to the injury in

an AFL cohort (Dulig et al., 2016; Ruddy et al., 2018). While these studies indicate a relationship exists between training load and injury risk, given the comparatively small amount of information specific to rugby union compared with other sports, the need for a more extensive study is warranted. Although the professional rugby union team participating in the project routinely examined fluctuations in acute and chronic exposure to high-speed running in relation to the decision-making process regarding hamstring injury risk and return to play. This was standardised to a timeframe of 7- and 21-days respectively, which provided further justification for a comprehensive analysis of the influence of timeframe of acute and chronic high-speed running exposure on the aetiology of hamstring injury.

4.3 Aetiology of MCL and ACL injuries

Injuries to the anterior cruciate and medial collateral ligaments of the knee have previously been observed to present a high burden in professional rugby union epidemiology research (Dallalana et al., 2007; Kemp et al., 2018; Kemp et al., 2019; Chapter 3 – Study 1 Injury epidemiology study). The purpose of the following section is: to provide a summary of the anatomy, pathophysiology and mechanisms of ACL and MCL injury, highlight factors that have previously associated with modulating the risk of ACL and MCL injury and finally present some of the unique methodological challenges posed when conducting ACL and MCL injury research in the context of men's professional rugby union.

4.3.1 Anatomy and function of the ACL and MCL ligaments

The knee joint complex is a stabilising hinge joint comprised of the femur, tibia, fibula. The primary movement is flexion and extension in the sagittal plane, it also has secondary motions in the frontal (adduction & abduction) and transverse plane (internal and external rotation of the tibia relative to the femur), all movements are accompanied by small magnitudes of linear translation (Figure 4.3.1a).

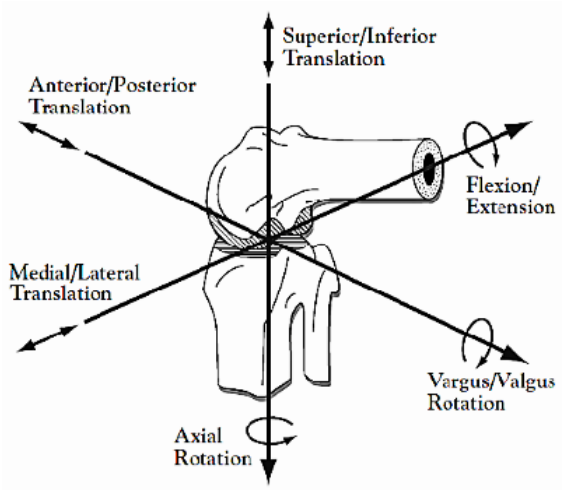


Figure 4.3.1a Knee joint degrees of freedom

The basic function of knee ligaments is to attach the articulating bones that make up the knee joint to one another, guide joint movement, maintain the congruency of the joint and possibly act as a strain sensor for the joint (Nigg and Herzog, 2007). Ligaments are primarily comprised of elastin and collagen fibres which has a hierarchical structure in nature with fibres organised in an undulating crimp (Figure 4.3.1b)

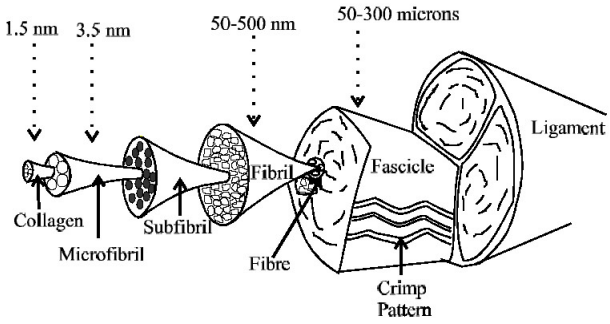


Figure 4.3.1b Hierarchical structure of collagen in ligaments adapted from Nigg and Herzog (2007).

The ligaments of the knee include: the anterior cruciate ligament (ACL) and the posterior cruciate ligament (PCL) both situated with the knee joint capsule, and the medial collateral ligament (MCL) and lateral collateral ligament (LCL), both located outside the joint capsule (Figure 4.3.1c).

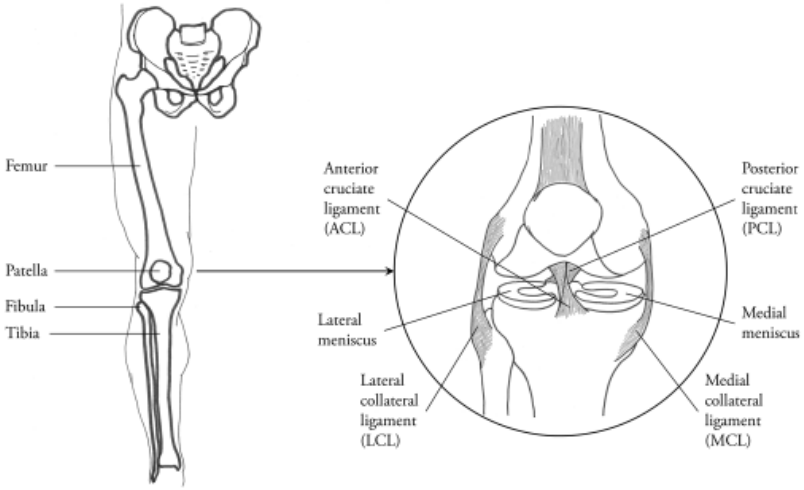


Figure 4.3.1c The ligaments of the knee joint viewed in the frontal plane.

4.3.1.1 Anterior cruciate ligament structure and function

The anterior cruciate ligament (ACL) attaches proximally to the lateral condyle of the femur, and is distally attached in front of the intercondyloid eminence of the tibia and is comprised of two distinct bundles (i. anteromedial bundle ii. posterolateral bundle), that twist in a lateral spiral from the femur to the tibia (i.e. anti-clockwise in the right knee and clockwise in the left) (Figure 4.3.1.1) (McLean et al., 2015). The mechanical load acting on the ACL alternates between each bundle throughout the normal range of motion. When the knee is in a position of full flexion, the anteromedial bundle becomes taut, whereas when the knee is in a position of full extension, the posterolateral bundle is taut (Moghaddam and Torkman, 2013).

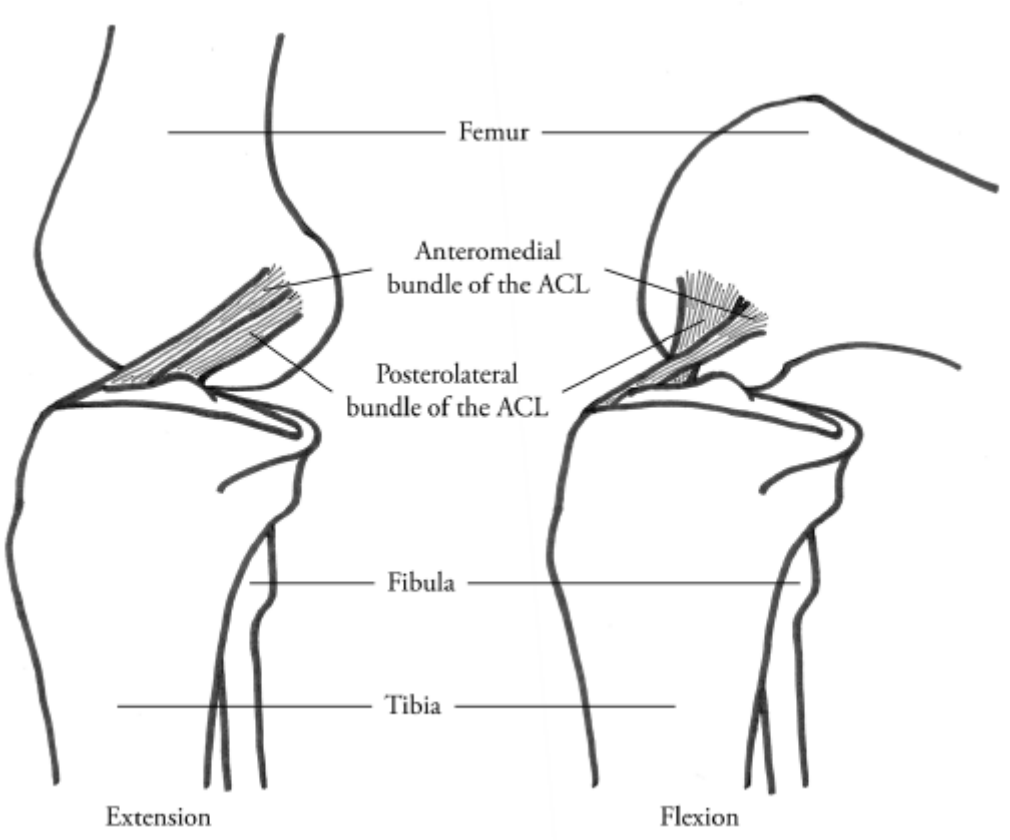


Figure 4.3.1.1Medial view of the anterior collateral ligament (ACL) of the knee from the sagittal plane.

The primary function of the ACL is to resist excessive anterior translation of the tibia relative to the femur due to the manner in which it attaches to the femur and tibia (Woo et al., 1999). Secondary functions of the ACL also include restraining tibial internal rotation relative to the femur (Grood et al., 1981; Nesbit et al., 2014; Bates et al., 2015), and restraining knee abduction whilst acting in conjunction with the MCL (Butler et al., 1980; Battaglia et al., 2009; Bates et al., 2017).

4.3.1.2 Medial collateral ligament structure and function

The medial collateral ligament (MCL) is broad and flat and located at the medial side of the knee joint. The MCL is proximally attached to the medial epicondyle of the femur below the adductor tubercle, and distally attached to the medial condyle of the tibia and medial surface of the body of the tibia (Figure 4.3.1.2).

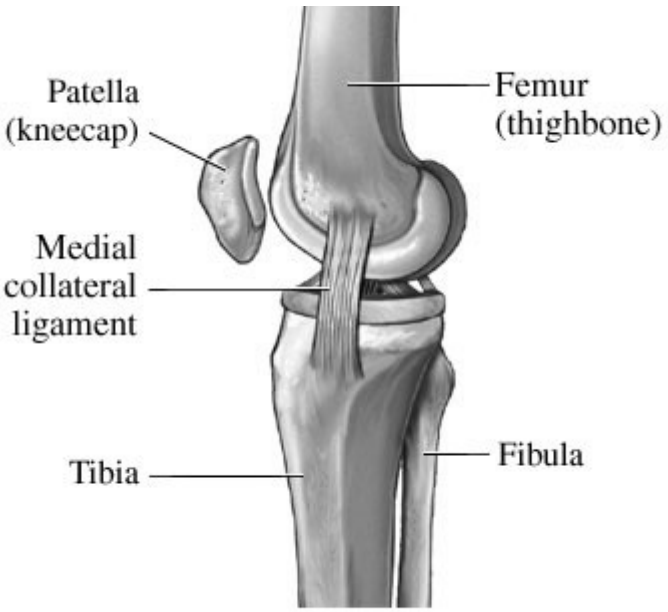


Figure 4.3.1.2 Medial view of the medial collateral ligament (MCL) of the knee from the sagittal plane.

The primary function of the MCL is to resist excessive valgus/abduction of the knee joint and external tibial rotation (Nigg and Herzog, 2007; Schein et al., 2012). The second function of the MCL is to restrain anterior tibial displacement in conjunction with the ACL (Nigg and Herzog, 2007).

4.3.2 Definition and pathophysiology of knee ligament injury

Ligaments are considered viscoelastic structures, and at a fundamental level ligament sprain injuries occur when a ligament undergoes high magnitudes of mechanical stress resulting in mechanical strain that is above the threshold of elastic linear ligament stress-strain behaviour (Figure 4.3.2) (Bach et al., 1997; Berns et al., 1992; Woo, 1982). When stresses exceed this threshold, the ligament behaves in a plastic manner, with larger magnitudes of strain resulting from smaller increases in stress, this in turn leads to the failure of n-number of ligament fibres resulting in a partial or complete ligament tear and in extreme combinations of stress and strain, total rupture of the ligament (Figure 4.3.2) (Nigg and Herzog, 2007; Bahr et al., 2020). The rate at which the ligament is strained has also observed to have an amplifying effect on ligament

failure, with higher strain rates resulting in a lower threshold of strain required for ligament failure (Pioletti et al., 1999; Withrow et al; 2006).

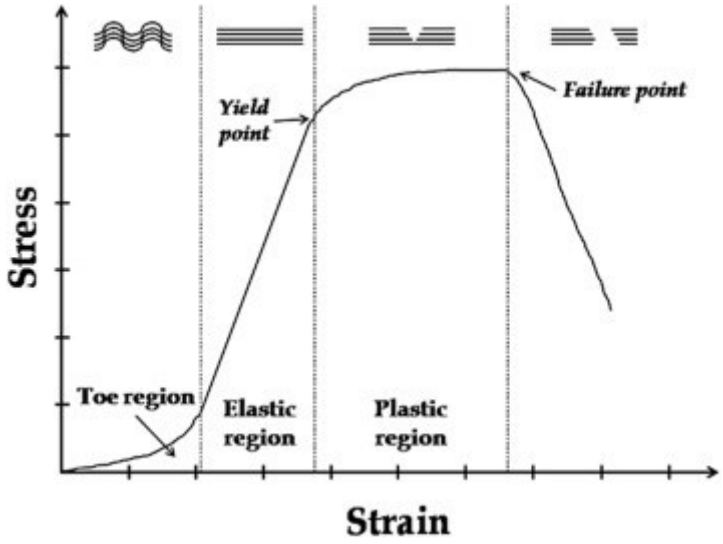


Figure 4.3.2 Stress-strain curve disapplying the non-linear material behaviour of ligaments adapted from Blaha (2017).

4.3.3 Inciting events and mechanisms of knee ligament injury

The following section will examine theoretical mechanisms of ACL and MCL injury in conjunction with the inciting events resulting in injury associated with each mechanism whilst participating in rugby union.

In sports injury research, ligament injuries are generally categorised as 'non-contact' or 'contact' injuries (Brooks et al., 2005a; Brooks et al., 2005b; Dallalana et al., 2007). Contact injuries are classified by the inciting injury event resulting from 'contact' with another player or object such as a rugby tackle whilst playing rugby union (Montgomery et al., 2018). This definition has been expanded to contact to any body part other than the injured leg as indirect contact, and contact to the injured leg as direct contact (Olsen et al., 2004). Whereas non-contact injuries are defined when the injury inciting event does not include bodily contact with another player or object excluding the feet on the playing surface such as a rapid deceleration or sharp change of direction (Montgomery et al., 2018).

Knee ligament injuries sustained whilst playing rugby union were observed to occur more frequently during contact events than non-contact (Brooks et al., 2005a; Brooks et al., 2005b; Dallalana et al., 2007). Dallalana and colleagues (2007) observed that professional male rugby union players sustained a higher proportion of contact knee ligament injuries (match 72%; training 48%) than non-contact knee injuries (match, 22%; training, 39%). Despite this, recent meta-analyses have highlighted that the majority of sports injury aetiology research concerning knee ligament injury has focused on the causes and risk factors of non-contact ligament injury such as non-contact ACL (Chia et al., 2020; Cronström et al., 2020). This is predominantly due to sampling bias, as the overwhelming majority of participants of those aetiological studies competed in non-contact sports such as soccer, netball and handball where direct contact with opposing players is illegal (Chia et al., 2020; Cronström et al., 2020). It should be noted that the mechanisms of contact-based ligament injury are theoretically similar to those of non-contact injury even if the inciting events are not. The only difference being that there are two inputs of external force: 1. External force in the form of ground reaction force from the injured player's foot (like in non-contact injuries); and 2. External force in the form of another player or object applied to the injured player e.g. the opposing player's shoulder contacting the injured player's anterior tibia.

4.3.3.1 ACL inciting events and injury mechanisms

The mechanism of ACL sprain injuries in rugby union and other field-based sports is complex, in that multiple inciting events have been observed to result in injury to the ACL (Dallalana et al., 2007; Montgomery et al., 2018). As a result, some uncertainty exists within the literature regarding the specific mechanisms of how the ACL is subjected to injurious mechanical loads. This is due to the complexity of mechanically testing the cadaveric ligament and knee joint complex (e.g. to establish ACL failure stress and strain) (McLean et al., 2015), the ethical issues surrounding in vivo research with regard to subjecting participants to injury, and capturing ACL

injuries that occur on the field with sufficient detail (i.e. Video footage at a high enough sampling frequency with enough camera angles to conduct three-dimensional analysis) (Montgomery et al., 2018; Della Villa et al., 2020; Johnson et al., 2018; Johnson et al., 2019). The proposed mechanisms of ACL injury are related to the primary and secondary functions mentioned in section 4.3.1.1.

4.3.3.1.1. Excessive anterior tibial translation – failure of the ACL's primary function

Excessive magnitudes of shear stress acting on the ACL via anterior tibial translation has been theorised to result in injury (Draganich and Vahey, 1990; Bates et al., 2018; Nasseri et al., 2020). This mechanism is due to the interaction between large magnitudes of axial compressive force acting on the tibiofemoral joint as a result of during sporting movements with large external forces and the geometry of the posterior-inferior-directed slope of both the medial and lateral tibial plateau (Meyer and Haut, 2008; McLean et al., 2011; Wall et al., 2012; Markolf et al., 2014; Markolf et al., 2018; Wang et al., 2019). This mechanism may be further exacerbated by large magnitudes of quadriceps contractions have previously been theorised to increase anterior tibial translation and therefore shear stress acting on the ACL due tensile forces acting on the tibial insertion via the patella tendon (Draganich and Vahey, 1990; De Morat et al., 2004; Wall et al., 2012; Bates et al., 2018; Nasseri et al., 2020). Finally, external forces applied either to the anterior of the femur or the posterior of the tibia may result in anterior tibial translation (relative to the femur) and thus place the ACL under injurious magnitudes of shear stress (Fleming et al., 2001; Meyer and Haut, 2008).

Retrospective examination of video footage of contact ACL injuries sustained during professional rugby union match play revealed that the anterior tibial translation mechanism was present when being tackled and tackling (Montgomery et al., 2018; Della Villa et al., 2021). Montgomery and colleagues reported that 78% of injuries where the inciting event was being tackled involved external loading from a tackling player in the sagittal plane (Montgomery et al., 2018). More detailed analysis of inciting events in professional rugby union match play revealed that 21% of

ACL injuries sustained during the tackle involved external force either applied to the posterior tibia or anteriorly resulting in tibiofemoral joint hyperextension, both of which the authors suggested likely resulted in anterior tibial translation (Della Villa et al., 2021).

4.3.3.1.2 External knee abduction moment and external tibial internal rotation moment – failure of the ACL's secondary functions

Excessive asymmetric tensile stress acting on the ACL via large magnitudes of either external knee abduction moment or external tibial internal rotation moment were also theorised as a mechanism for ACL injury (Quatman et al., 2010). Evidence of the ACL's secondary function of resisting knee abduction is contradictory, for example Mazzocca and colleagues suggested that the ACL could not be injured by excessive knee abduction moment in isolation without concomitant injury to the MCL which is the primary constraint (Mazzocca et al., 2003). However, numerous studies that have utilised robotic methods to examine the influence of external knee abduction moment on the mechanical properties of ACLs in cadaveric knee joint complexes and cadaveric lower limbs have reported significant increases in ACL strain (Kiapour et al., 2016; Kiapour et al., 2015; Shin et al., 2011; Withrow et al., 2006). The mechanism of large magnitudes of knee abduction moments on ACL injury is also supported by clinical imaging studies which observed bone bruises located at the posterolateral tibial plateau and the lateral femoral epicondyle in 80% of observed ACL injuries (Speer et al., 1992; Viskontas et al., 2008). A possible explanation for the observed increases in ACL strain following the application of an external knee abduction moment is related to the geometry of the articulating surfaces of the tibiofemoral joint. The motion of knee abduction is mechanically coupled with the motion of tibial internal rotation which results in injurious levels of tensile stress applied to the ACL during certain inciting events without MCL failure (Matsumoto et al., 2001; Ren et al., 2010; Fukuda et al., 2003; Gabriel et al., 2004; Hollis et al., 1991; Kanamori et al., 2002; Matsumoto et al., 2001; Ren et al., 2010).

The inciting events associated with external knee abduction moment mechanism have been reported by a limited number of studies which have examined recorded footage of ACL injuries sustained whilst playing professional rugby union (Montgomery et al., 2018; Della Villa et al., 2021) and soccer (Della Villa et al., 2020). External force applied to the lateral side of the injured leg when being tackled or rucking, either directly on, above or below the knee resulting in a large external knee abduction moment (Montgomery et al., 2018; Della Villa et al., 2020; Della Villa et al., 2021). However, these studies are limited by the low sampling frequencies of the video footage.

4.3.3.1.3 The multiplanar combination mechanism

The combination of the multiplanar stresses and strains acting on the ACL mentioned in the previous section is, at the time of writing the general consensus for the cause of ACL injury in team sports including rugby union (Levine et al., 2013; Kiapour et al., 2015; Kiapour et al., 2016; Bates et al., 2017; Bates et al., 2019). Indeed, cadaveric studies have reported combined loading of anterior tibia translation, external knee abduction moment and internal rotation moment have yielded larger magnitudes of ACL stress and strain compared to uniaxial loading (Berns et al., 1992; Markolf et al., 1995). However, it is important to understand that the combination of these multiplanar forces may vary depending on the inciting event that resulted in ACL injury to the individual. Examination of contact ACL injury footage during professional rugby union match play revealed that a varied number of inciting events and loading conditions resulted in injury: being tackled (from the front, from behind and from the side), tackling, rucking and scrummaging (Dallalana et al., 2007; Brooks et al., 2005a; Montgomery et al., 2018; Della Villa et al., 2021). The variation in inciting events has also been observed when examining ACL injury footage other team sports such as AFL (Cochrane et al., 2007), soccer (Della Villa et al., 2020) and basketball (Krosshaug et al., 2007). The combination of these findings suggests may provide an explanation for the varied inciting events observed in previous studies and the previous chapter.

4.3.3.2 MCL injury inciting events and mechanisms

The accepted mechanism for an MCL injury is the result of a large magnitude of external knee abduction moment acting on the knee joint, resulting in increased medial joint space between the femur and tibia which in turn causes excessive tensile stresses to act on the proximal and distal portions of the MCL accompanied by strains that exceed the capacity of the ligament resulting in failure (Matsumoto et al., 2001; Mazzocca et al., 2003; Andrews et al., 2017). The knee abduction moment may also be accompanied by with medial translation of the tibia and external rotation of the tibia (Marchant et al., 2011).

In rugby union the excessive knee abduction moment mechanism of MCL injury is most commonly the result of collision with another individual to either the proximal tibia or distal femur whilst the foot is fixed on the ground (Comfort and Abrahamson, 2010). With the most common inciting event observed to be when a player was tackled from the side (38% of 61 MCL injuries - Dallalana et al., 2007). Similar inciting events have also previously been observed in rugby league (Awwad et al., 2019) and soccer (Buckthorpe et al., 2021). Rucking and mauling activities were also observed to be inciting events for MCL injury in rugby union (25% of 61 MCL injuries - Dallalana et al., 2007), with changing direction being the only non-contact inciting event recorded (13% of 61 MCL injuries - Dallalana et al., 2007). The mechanism also explains non-contact MCL injuries where the inciting event is related to cutting or changing direction. In these scenarios the foot is fixed on the ground and the excessive abduction moment is caused by the position and acceleration of the other body segments such as the trunk.

4.3.4 Variables associated with ACL and MCL injury aetiology

The following section will examine the existing aetiological literature regarding variables that are associated with ACL and MCL injury risk. The research reviewed in the previous sections of this chapter has highlighted that the majority of knee ligament injuries occur during contact rather than non-contact situations in rugby union. Despite this, the majority of aetiological

literature associated with knee ligament injury sustained during team sports has focussed on establishing causal factors for non-contact ACL injuries (Chia et al., 2020; Cronström et al., 2020). Where possible, in the following section research from rugby union will be presented, however, although the injury burden of ACL and MCL injury is high in rugby union (Brooks et al., 2005a; Brooks et al., 2005b; Dallalana et al., 2007; Kemp et al., 2018; Kemp et al., 2019), there is a paucity of research exploring the factors associated with these injuries within a rugby union population. Furthermore, despite MCL injury presenting a significant injury burden to professional rugby union and soccer teams there are a distinct lack of aetiological studies examining the relationship between variables and MCL injury. Therefore, research examining selected variables associated with ACL and where possible MCL injury aetiology from similar field-based team sports will be reviewed as well in the following section. The section was split into the following sections: non-modifiable intrinsic risk factors, modifiable intrinsic risk factors and external risk factors.

4.3.4.1 Non-modifiable intrinsic variables associated with ACL and MCL injury aetiology

4.3.4.1.1 Previous injury

There is growing body of evidence that sustaining lower limb injury increases the risk of sustaining subsequent lower limb injury (Fulton et al., 2014; Toohey et al., 2017; Williams et al., 2017b; Pfeifer et al., 2019). A recent systematic review by Pfeifer et al. (2019), revealed that female team sports athletes who suffered a previous ACL injury or ankle sprain were at a higher risk of sustaining a subsequent injury on the same knee. This supported an earlier meta-analysis conducted by Toohey et al. (2017), that revealed injury to the knee joint (specifically the ACL), resulted in an increased risk of subsequent non-ACL knee injury in professional male and female soccer players. Previous ACL injury was also observed to be a risk factor for subsequent ACL injury in both the contralateral limb and previously injured limb in contact team sport (AFL) by Orchard et al. (2001).

Aetiological literature concerning previous MCL injury is limited, early studies report MCL re-injury to have a relatively low prevalence (7%–8%) (Derscheid and Garrick, 1981; Reider et al., 1994). The more recent findings of Lundblad and colleagues (2013), examined professional soccer players over 11 playing seasons of competition in the Union of European Football Associations Champions League, and observed that 11% of MCL injuries were MCL re-injuries. Within a male professional rugby union population, MCL and ACL injuries were observed to have a moderate proportion of re-injury (Williams et al 2017b). Several possible explanations have been presented for re-injury or subsequent injury to a different location, which may be present in isolation or combined together. These include: an insufficient access and or low levels to medical treatment and rehabilitation (Waldén et al., 2006; Nilstad et al., 2014); a premature return to play due to lack of player availability (Waldén et al., 2006), which may then either lead the individual to adopt altered movement strategies exposing the previously injured knee to risk of injury and leave the knee more vulnerable when undergoing potential inciting events (Waldén et al., 2006). The mechanical properties of a previously injured ligament have also been proposed to differ from non-injured ligaments, however this was only observed in high-severity ligament re-injury (Sevick et al., 2018).

4.3.4.2 Modifiable intrinsic variables associated with ACL and MCL injury aetiology

4.3.4.2.1 Isokinetic measures of hamstrings and quadriceps strength

The strength of the quadriceps and hamstrings assessed via isokinetic dynamometry has received a large research focus in relation to primary ACL injury risk as well as clearing injured individuals fit to return to play (Orchard et al., 1997; Burgi et al., 2019). As previously mentioned in section 4.2.4.2.1 of this chapter, isokinetic flexion and extension torque are used in the literature as a proxy from hamstring and quadriceps muscle strength (Baltzopoulos et al., 2012). The underlying mechanism associated with these variables is due to the observations of higher magnitudes of quadriceps strength in isolation or combined with lower magnitudes of hamstring strength resulting in increased anterior tibial translation placing injurious levels of mechanical

strain on the ACL, thus increasing the potential for injury (Draganich and Vahey, 1990; Bates et al., 2018 Nasseri et al., 2020; Section 4.3.3.1.1 of this chapter). Indeed, having stronger hamstring muscles have been theorised to decrease the magnitude of anterior translation of the tibia injury thus reducing the injurious amount of strain acting on the ACL (Li et al., 1999; DeMorat et al., 2004). However, despite these long-held theories, there are limited prospective studies examining ACL injury risk in relation to isokinetic strength of the knee joint (Myer et al., 2009; Söderman et al., 2001; Steffen et al., 2016; Uhorchak et al., 2003). These studies did not include a population that participated in contact sports and only examined non-contact ACL injuries sustained, thus limiting the generalisability to the current project (Myer et al., 2009; Soderman et al., 2001; Steffen et al., 2016). Furthermore, the findings of the prospective studies are also inconsistent, Myer et al (2009) observed female team sport athletes who sustained ACL injuries (n = 22) displayed smaller magnitudes of hamstrings strength when compared to male controls during isokinetic testing (300°/s), however, no differences in isokinetic quadriceps strength or the ratio of hamstrings strength to quadriceps strength (H/Q ratio) was observed. Whereas Soderman et al. (2001) observed that female athletes exhibited smaller magnitudes of H/Q ratio at slower isokinetic speeds (90°/s) compared to controls. In contrast to these positive findings, Uhorchak and colleagues (2003) reported that there was no difference in isokinetic hamstrings or quadriceps strength at 60°/s between injured and non-injured groups regardless of sex. Steffen et al. (2016) also reported no association between knee joint isokinetic strength variables and non-contact ACL injury in professional female team sport players. The lack of prospective studies examining previously examined isokinetic variables in a male professional rugby union population, combined with the extensive use within the participating rugby team warranted prospective investigation in relation to knee ligament injury risk.

4.3.4.2.2 Movement characteristics

Analysis of human movement and the accompanying forces *acting on (external)* and *produced by (internal)* the individual during dynamic tasks have long been theorised to influence knee ligament injury aetiology. However, although a large number of comparative studies are present within the biomechanical literature, recent meta-analyses highlighted a paucity of prospective aetiological studies that have examined existence of a relationship between biomechanical variables and knee ligament injury risk (Chia et al 2020; Cronström et al 2020). The reasons for this lack of research exclusive of ensuring adequate sample size and a sufficient number positive cases, are multifaceted and are summarised in figure 4.3.4.2.2.

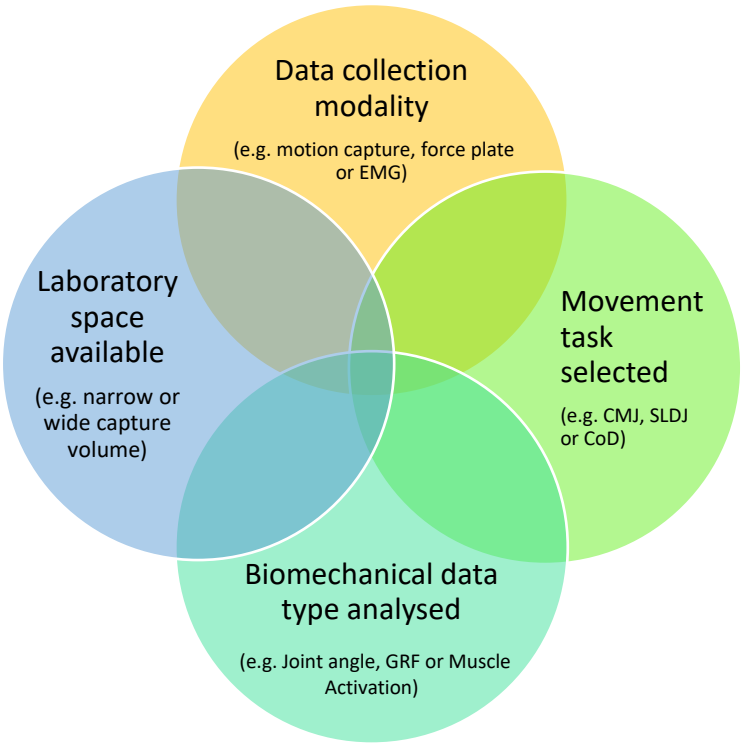


Figure 4.3.4.2.2 An illustration of the challenges faced by researchers conducting biomechanical aetiological research.

These issues relate to the challenge of what equipment, software and laboratory space are available to researchers. The interpretation of these biomechanical findings is further compounded by the task selected for analysis, for example the work contributions of the knee,

hip and ankle will vary if the task involved both legs (bilateral) or one leg (unilateral), if the goal of the task is to achieve a high vertical jump (e.g. vertical drop jump) or to rapidly change of direction (King et al., 2018; King et al., 2019). And due to these issues, the evidence-base for biomechanical variables for a given task in relation to knee ligament injury is smaller when compared to other forms of assessment such as isokinetic analysis of knee extension and knee flexion strength. The selected biomechanical studies in this section will be discussed in relation to the most commonly assessed movement tasks in relation to MCL and ACL aetiology, rather than specific variables.

4.3.4.2.2.1 Biomechanical analysis of bilateral vertical drop jumps

Excessive magnitudes of knee abduction angle and external knee abduction moment has received a large amount of research focus in relation to knee ligament injury risk, specifically the ACL. This is for the most part due to the work of Hewett and colleagues (2005), who presented one of the first studies examining whether a bilateral vertical drop jump task (Figure 4.3.4.2.2.1).

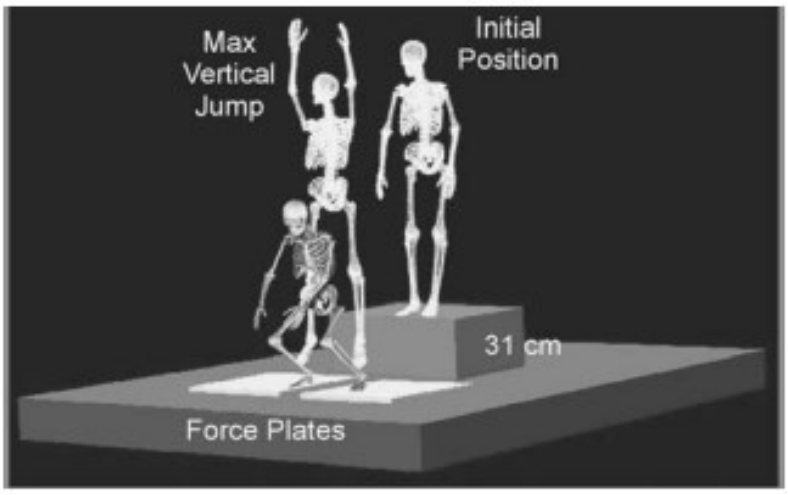


Figure 4.3.4.2.2.1 Illustrated diagram of a bilateral vertical drop jump adapted from Hewett et al. (2005).

In the study, greater magnitudes of knee abduction angle, external knee abduction moment and ground reaction force were associated with a higher risk of ACL injury in female high school team sport athletes (~16 years old) (Hewett et al., 2005). Although examining 'abnormal' knee

biomechanics during the vertical drop jump test have been advocated for use in injury risk screening (Ekegren et al., 2009; Myer et al., 2010; Nilstad et al., 2014), a limitation with Hewett and colleagues' initial study was the low number of cases suffering from ACL injury (n = 9) compared to the number of athletes that were injury free (n = 196) (Hewett et al., 2005). This dataset does not conform to the required ratio of injured to injury-free cases when conducting standard logistic regression (Conservative ratio of injured cases ≥ 0.20, lenient ratio of injured cases ≥ 0.10; Peduzzi et al., 1996) and is therefore should be classified as a rare-event making the associations to injury risk spurious. Indeed, the findings of Hewett and colleagues have not been replicated with success. Larger scale studies with more reported ACL injuries did not observe any relationship between knee abduction angle or moment and risk of ACL injury in a mixed high school-college cohort athletes (~18 years) (Smith et al., 2012) or female soccer and handball players competing nationally (Krosshaug et al., 2016). In a recent meta-analysis Cronström et al (2020) suggested that a possible explanation for these contradictory findings was that the external moments generated during the bilateral vertical drop jump may not pose a sufficient challenge to the neuromuscular system of mature well-trained athletes such as professional rugby union players to display movement characteristics and joint loading patterns associated with ACL risk (Smith et al., 2012; Krosshaug et al 2016). In comparison to a sidestep cutting task, although moderate correlations were observed between knee abduction angle (correlation coefficient = 0.706), the knee abduction moments were poorly correlated (correlation coefficient = 0.135), with sidestep moments being 6 times the magnitude of the vertical drop jump moments (Kristianslund and Krosshaug, 2013). This is also supported by more recent research by King and colleagues (King et al., 2018; King et al., 2019). These findings suggest more challenging tasks with an emphasis on unilateral loading such as rapid changes of direction or single-legged jumping tasks may be more appropriate to investigate knee ligament injury aetiology.

4.3.4.2.2.2 Biomechanical analysis of the inciting event in a laboratory setting

Theoretical models of injury aetiology have suggested that conducting a biomechanical analysis of the inciting events resulting in knee ligament injuries would be the most ecologically valid scenario to gain insight into the aetiology of said injuries (Donnelly et al., 2012; Edwards et al 2021a). However, this would require the players and the playing surface to undergo a level of instrumentation that would be both financially prohibitive and possibly impede the flow of training and match-play. Although estimating biomechanical loading from marker-less motion capture is an alternative to circumvent these issues, the techniques are reliant on camera multiple angles and is still an emerging data collection modality and therefore outside of the scope of the current project (Johnson et al., 2018; Johnson et al., 2019; Edwards et al., 2021a). Analysis of an approximation of the inciting event within a laboratory setting is more common approach within the literature (Weir, 2021; Edwards et al 2021a). As previously mentioned in section 4.3.3, aetiological studies examining biomechanical variables in relation to knee ligament injuries have exhibited a bias towards non-contact injuries. Indeed, in a recent review Weir (2021) highlighted that the majority of cross-sectional biomechanical studies examining inciting events of knee ligament injuries have focused on analysing change of direction tasks (Besier et al., 2003; Cerulli et al., 2003; Dempsey et al., 2007; Donnelly et al., 2012; Lee et al., 2013; Brown et al., 2014; Donnelly et al., 2017; Chinnasee et al., 2018; Maniar et al., 2018; Dos Santos et al., 2019; Smith et al., 2020; David et al., 2018a; David et al., 2018b). However, a limited number of biomechanical studies examining rugby union specific contact events exist, which include: tackling (Seminati et al., 2016; Seminati et al., 2017a; Seminati et al., 2017b; Tierney et al., 2018; Kerr et al., 2018; Tanabe et al., 2018; Kawasaki et al., 2018; Tierney and Simms, 2019), being tackled (Edwards 2021b), and scrummaging (Cazzola et al., 2014a; Cazzola et al., 2014b; Cazzola et al., 2015a; Cazzola et al., 2015b; Cazzola et al., 2015c). The majority of these studies were focussed on the aetiology of concussion, injury to the cervical vertebra, shoulder or upper limb rather than knee ligament injury and as a result the majority did not analyse lower limb

biomechanics, with the exception of three studies that only reported simple lower limb joint angles (Kerr et al, 2018; Kawasaki et al, 2018; Edwards 2021b). In addition, there is currently an absence of prospective studies examining biomechanical variables (such as knee abduction moment) recorded during performance of these inciting events (Weir 2021; Edwards et al 2021a). This is likely due to a combination of factors including the requirement for a large capture volume with a sufficient number of cameras (Edwards et al 2021a). This may be further compounded by the need for the markers used to track motion data to stay fixed to the participants, when colliding with a tackle bag or other person these may drop off which will require re-calibration of the model (Edwards et al 2021a). The combination would result in a lengthy and strenuous data collection session for the participants, which in a professional rugby union environment would be a challenge to ensure data is collected for a squad of 50 players within a training and competition schedule. This problem is also present in non-contact knee injury research with change of direction tasks requiring a sufficient number of cameras, large capture volumes and long data collection times (Weir, 2021). Single-leg jumping and landing tasks have been proposed as a proxy movement that mimics the knee joint loading synonymous with the knee ligament injury mechanisms described in section 4.3.3 of this chapter (Jones, et al, 2014; Cronström et al, 2020; Weir, 2021). These movements also have the added benefit of requiring smaller capture volumes and fewer cameras, resulting in a more viable addition to a mass injury screening PHE, whilst exposing athletes to similar mechanical loading conditions (Weir, 2021).

4.3.4.2.2.3 Biomechanical analysis of single-leg jumping and landing

Single-leg jumping tasks were observed to place an increased demand on the musculature of the lower limbs and trunk compared to bilateral jumping tasks, which suggest they may be more appropriate tasks in the context of knee ligament injury aetiology (Cronström et al., 2016). A variety of single-leg jumping tasks have been examined including: single-leg drop-jumps (King et al., 2018; King et al., 2019; King et al., 2021a), single-leg countermovement jumps (King et al.,

2018; King et al., 2021a), single-leg horizontal hops for distance (King et al., 2018; King et al., 2019; King et al., 2021a), and single-leg lateral hurdle hops (King et al., 2018). Due to the increased horizontal force component required to successfully complete the latter two tasks, one may conclude that they may theoretically place the individual under more ecologically valid loading conditions compared to the primarily vertical single-leg drop-jump and countermovement jump tasks (Sell et al, 2006). There is a paucity of literature concerning this, however, the findings of King and colleagues (King et al., 2018; King et al., 2019) suggests that this may not be the case, with a single-leg drop jump being able to discriminate between injured and uninjured limbs. Horizontal jumps also require additional methodological considerations when conducting biomechanical research to examine lower limb loading. For example, when examining landing biomechanics during a single-leg horizontal hops for distance, a sufficient number of force platforms are required (or one of a sufficient area) to ensure the athlete is not actively targeting landing on a specific location (Standard laboratory platforms are 400 x 600 mm). Due to the biomechanical resources available within the current PhD project, it was not possible to collect horizontal single-leg jumps, therefore the remainder of the section will focus on the single-leg drop jump task.

The single-leg drop jump task is a plyometric movement task that involves an individual dropping from a raised surface (commonly 20cm high), briefly landing on one leg, then rebounding into a vertical jump (Figure 4.3.4.2.2.3). Biomechanical variables recorded during the task were reported to exhibit medium to high reliability across a number of kinetic and kinematic variables (ICC 0.74 – 0.86) (Mohammadi et al., 2012).

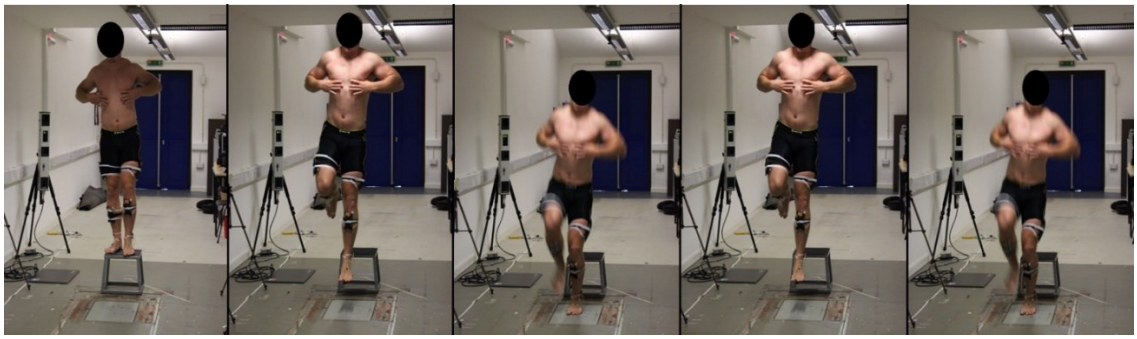


Figure 4.3.4.2.2.3 Example single-leg drop jump sequence.

Despite recommendations from researchers that the single-leg drop jump task should be used to examine knee ligament injury aetiology as an alternative to bilateral vertical drop jumps and change of direction tasks, previous research is limited to a small number of prospective studies examining the risk of primary and secondary ACL injury. Numata et al. (2018) reported female high school basketball and handball athletes who subsequently sustained ACL injuries exhibited larger magnitudes of medial knee displacement at initial contact with the ground compared to injury free controls, suggesting a link to the excessive knee abduction moment mechanism of ACL injury. King et al. (2018) conducted a retrospective analysis of male field-based team sports athletes who were 9-months post ACL injury. The authors reported that injured limbs exhibited smaller magnitudes of knee abduction moment compared to non-injured limbs (King et al., 2018). In subsequent cross-sectional study King and colleagues (2019), reported male field-based team sports athletes who were 9-months post ACL injury exhibited larger magnitudes of knee extension moment, and ankle plantarflexion moment when performing single-leg drop jumps with their non-injured limb compared to their injured limb. In a prospective study examining secondary ACL injury risk, King and colleagues (2021a) reported that when performing single-leg drop jumps male field-based team sports athletes who sustained a secondary ACL injury exhibited smaller magnitudes of ankle plantarflexion moment, combined with larger magnitudes of hip and knee extension moment and knee abduction moment. These findings suggest a combination mechanism discussed in section 4.3.3.1.3 of this chapter. The findings discussed in this section suggest further research is needed to determine if the single-

leg drop jump task can be used in conjunction with biomechanical analysis to examine the aetiology of knee ligament injuries, therefore it was included by the researcher as a novel measure in the battery of tests for the participating rugby team.

4.3.4.3 Extrinsic variables associated with ACL and MCL injury aetiology

4.3.4.3.1 Exposure to contact events

Since the inception of cyclical theoretical models of injury aetiology, the manner in which players are exposed to inciting events has long been theorised to influence injury risk (Gissane et al., 2001; Meeuwisse et al., 2007; Windt and Gabbett, 2017). In the context of rugby union, contact events such as the tackle and ruck have been frequently highlighted as high-risk inciting events which require players to have sufficient levels of physical conditioning and technical competency to mitigate these scenarios (Williams et al 2013; Stokes et al 2019; Stokes et al 2020; Edwards et al, 2021a; Chapter 3 – Study 1). Indeed, a high proportion of reported MCL and ACL injuries were sustained during contact events (Proportion) (Dallalana et al 2007; Montgomery et al 2018; Buckthorpe et al 2021; Chapter 3 – Study 1). Numerous descriptive studies examining contact events in rugby union match play (Deutsch et al., 1998;Duthie et al., 2005; Eaton et al. 2006; Fuller et al. 2007b; Quarrie et al. 2007; Fuller et al. 2008; Roberts et al. 2008; Quarrie et al. 2012; Hendricks et al.2013; Hendricks et al. 2014; Jones et al. 2014; Roberts et al. 2014; Bradley et al. 2017; Campbell et al. 2017; Tucker et al. 2017; Hendricks et al.2018; Tierney et al. 2020; Yamamoto et al.2020; Tierney et al. 2021), and, to a lesser extent during training (Vaz et al., 2012; Bradley et al., 2015; Campbell et al., 2017; Dubois et al., 2020). Following the work of Hulin et al. (2014), there has been increased speculation that unaccustomed increases in exposure to inciting events such as tackles and rucks may have the potential to increase the risk of injury (Tierney et al., 2020; Stokes et al., 2020; Edwards et al., 2021a). Despite these hypotheses, at the time of writing (2021) there has been no research examining whether fluctuations in contact events experienced by players over a series of matches are associated with MCL, ACL or any other lower limb injury in relation to injury risk despite contact events being acknowledged as

an area of increased injury risk by a number of epidemiological studies in rugby union (Brooks et al., 2005a; Brooks et al., 2005b; Fuller et al., 2007b; Dallalana et al., 2007; Roberts et al., 2008; Roberts et al., 2014; Kemp et al., 2018; Kemp et al., 2019). Therefore, there is a need to examine if fluctuations in exposure to contact events are associated with knee ligament injury in professional rugby union. Due to this contact events were included in the subsequent experimental study as a novel measure.

4.3.4.3.2 Accelerometer derived measures of external workload

The adoption of microtechnology based workload monitoring systems in professional rugby union and other sports teams has allowed an explosion of research focused on examining the existence of an aetiological relationship between player workloads and injury risk (West et al., 2019; Section 4.2.4.3 of this chapter). However, when exploring the aetiology of MCL and ACL injuries sustained during contact events, the use of distance and velocity-based variables derived from GPS data alone may not truly reflect on-pitch external workloads experienced by rugby union players (Howe et al., 2020). This is due to the diminished reliability and validity of GPS systems when analysing activities including rapid changes of direction and movements with little horizontal displacement that exhibit large magnitudes of changes in acceleration, which encompasses the majority of contact events in rugby union (e.g. the tackle, rucking and mauling) (Jennings et al., 2010; Couts et al., 2013; Boyd et al., 2013; Howe et al., 2020). The majority of mainstream microtechnology based workload monitoring systems available to professional rugby union teams also include inertial sensors such including triaxial accelerometers, triaxial gyroscopes and magnetometers sampling at 100 Hz (Catapult Sports, Melbourne, Victoria, Australia; STATSports, Newry, Northern Ireland; GPS Sports, Canberra, Australian Capital Territory, Australia). Indeed, accelerometer-based variables have been proposed as a solution to provide a more inclusive quantification of on-pitch external workload in contact team sports rather than variables derived from GPS (Gabbett et al., 2015; Granger et al., 2018). These variables have also exhibited corelations with selected internal workload variables including

session RPE (sRPE) (Casamichana et al., 2013), muscle oxygen saturation (Gómez -Carmona et al., 2019), and maximal oxygen uptake (Barrett et al., 2014).

Due to the increasing popularity commercially available workload monitoring systems in professional sports teams (West et al., 2019; Gómez-Carmona et al., 2020), a plethora of different accelerometer-derived workload variables have been developed for each monitoring system (Gómez-Carmona et al 2019). Of these variables the most common type are an accumulative vector summation of all three accelerometer axes or derivatives thereof over a period of interest, examples of these variables include: PlayerLoad™, developed by Catapult Sports (Melbourne, Victoria, Australia) (Boyd et al., 2011), Body Load developed by GPS Sports (Canberra, Australian Capital Territory, Australia) (Cunniffe et al., 2009) and Total Load developed by StatSports (Newry, Northern Ireland) (Bowen et al., 2017). It is important to note that although all of the variables use the first derivative of acceleration, the scaling factor the vector summation is different (PlayerLoad™ = divided by 100; Body Load = divided by players' body mass; Total Load = divided by 1000) (Weaving et al., 2014; Gómez-Carmona et al., 2019), therefore these variables are not interchangeable. Of these variables, PlayerLoad™ was reported to be the most frequently analysed of all accelerometer-based metrics employed in athlete workload monitoring research in a recent systematic review (Gómez-Carmona et al 2020). The professional rugby team which participated and co-funded the work contained within this thesis used a Catapult Sports monitoring system (Catapult Sports, Melbourne, Victoria, Australia) and as a result, the only available accelerometer-derived variable was PlayerLoad™. Due to this constraint, the following section will focus on studies that analysed PlayerLoad™.

4.3.4.3.3 The use of PlayerLoad™ in professional rugby union and other contact sports

PlayerLoad™ value represents the on-pitch physical activity a player has experienced during a session or game, rather than an isolated count or magnitude of distance travelled or contact events (Gabbett, 2015; Barreira et al., 2017; Hulin et al., 2018). Although it was previously reported to be positively associated with manually coded contact events in rugby union (Roe et

al, 2016). PlayerLoad™ is defined as the "instantaneous rate of change of acceleration divided by a scaling factor" (Nicolella et al 2018). In physics, the instantaneous rate of change of acceleration is often termed "jerk" and is defined as:

$$\vec{J} = \frac{\Delta\, \vec{a}\,(t)}{\Delta \mathrm{t}}$$

where $\vec{a}$ is the vector quantity of acceleration and t is time (Eager et al., 2016). PlayerLoad™ was calculated as the vector quantity of each sensor of the triaxial accelerometer within each unit, defined by Catapult Innovations as:

$$Player\ LoadTM = \sum_{t=0}^{t=n} \frac{\sqrt{(fwd_{t=i+1} - fwd_{t=i})^2 + (side_{t=i+1} - side_{t=i})^2 + (up_{t=i+1} - up_{t=i})^2}}{100}$$

Where fwd is anterior-posterior acceleration, side is medio-lateral acceleration, up is vertical acceleration, t is time point and 100 is the scaling factor (Nicolella et al., 2018).

At the time of writing, no studies have utilised player load to examine the influence of on-pitch physical activity in relation to knee ligament injury in rugby union. Furthermore, PlayerLoad™ was not commonly used by male professional rugby union teams such as the one participating in the project (West et al., 2019). However, Cummins et al., (2019) used PlayerLoad™ and observed that acute increases in on-pitch physical activity were associated with sustaining any non-contact soft-tissue injury in male professional rugby league players. Hulin et al., (2020) reported similar findings in another cohort of professional rugby league players. As such, PlayerLoad™ may offer an insight into the existence of a relationship between external workload (including contact events) and knee ligament injury aetiology in men's professional rugby union, therefore it was included in the subsequent experimental study as a novel measure.

4.4 Methodological and statistical considerations for conducting aetiological research

The following section highlights key methodological issues that should be considered when conducting research to examine if a variable is related to the risk of developing the injury of interest. Some of these issues are particularly pertinent to aetiological studies conducted within professional sports teams over a number of playing seasons.

4.4.1 Study design

Selecting the appropriate study design is a fundamental step when conducting sports injury research. Indeed, a variety of study designs have previously been used for the investigation of risk factors in sports injury aetiology research. The following section highlights the strengths and weaknesses of each design in relation to conducting aetiological research with the focus of examining whether a variable is associated with developing the injury of interest.

During a case-control study, classification of injury status occurs first during recruitment. Following this the investigator will then collect information relating to previous exposure to the risk factors of interest (Fallowfield et al., 2005). Those sustaining an injury are matched with a control group who are injury free (Bahr and Holme, 2003). A strength of case-control studies is that they are economical to undertake and simple. However, the study design has potential for the exposure information to be biased due to retrospective recall required from the participants (Bahr and Holme, 2003).

In a cross-sectional study design, information regarding injury occurrence and risk factors is collected from a cohort at single time point (Fallowfield et al., 2005). A limitation of cross-sectional studies is the potential for recall bias, additionally the study design does not allow for cause-and-effect between exposure and injury to be established (Bahr and Holme, 2003).

During a prospective cohort study, participants are followed for a predetermined length of time. During this period, individuals will either sustain an injury, not sustain an injury, or may be

censored for alternative reasons. After the end of the study characteristics of the injured and non-injured groups are compared to determine if differences existed at baseline prior to onset of injury (Bahr and Holme, 2003). A strength of this design is that recall bias is reduced, due to information regarding the injury such as mechanism, type and location etc being collected in close proximity to the event (i.e. on the day or within the week) (Bonita et al., 2006). Furthermore, it is easier to establish the cause-and-effect nature of the risk factor variables examined in this manner due to all individuals being free from injury at the start of the study (Bonita et al., 2006).

Randomized controlled trials are often cited as providing the strongest level of evidence of experimental studies (excluding systematic reviews and meta-analyses). In the context of injury biomechanics, a number of individuals who have similar characteristics (e.g. age, mass, height and playing position) would be randomly assigned into two (or more) groups to test an intervention aimed at moderating the selected risk factor. The experimental group is provided with the intervention, whereas the other (the comparison or control group) either has an alternative intervention, a placebo or no intervention at all. The groups would then be followed up to see how effective the experimental intervention was at removing the risk factor. Outcomes are measured at specific times and any difference in injury between the groups is assessed. However, despite randomised control trials being one of the best forms of evidence, their use in sports injury research is challenging. This is due to both the lack of large-scale prospective cohort studies being conducted and a subsequent lack of consensus in which reported variables are risk factors. Additionally, it may not be feasible to randomly assign groups when working with small populations (i.e. professional sports teams) as the intervention may bleed over. In addition to the logistical reasons, in sports injury aetiology research there is the question of whether is it ethical to assign individuals into a group that will target reducing a risk factor and leave one group 'at risk'. Therefore, the subsequent experimental chapters will employ a prospective cohort study design.

4.4.2 Statistical analyses used to classify the association of a variable with injury risk and associated issues

Selecting the appropriate method of statistical analysis is paramount to sports injury aetiology research and will influence whether a variable is associated with the causation of injury of interest (Impellizerri et al., 2019). This section addresses statistical methods of analysing sport injury data and the issues relating to them.

Logistic regression has been commonly used in sports injury research to model effect of workload (Gabbett and Jenkins, 2011; Rogalski et al., 2013; Hulin et al., 2020), lower limb strength (Myer et al., 2010) and biomechanical variables (Hewett et al., 2005; Krosshaug et al., 2016) on the log odds of the dichotomous outcome variable of injury (injured: yes or no). In the context of the current project, a particular issue when conducting logistic regression in a multi-season prospective study is the assumption of independence of observations (i.e. cases are considered unique and are not repeated in the analysis). When not accounted for in conventional logistic regression or survival analysis failure of the assumption of independence of observations results in an increased type I error rate (Williamson et al., 1996; Diggle et al., 2013; Ruddy et al., 2019; Impellizzeri et al., 2020). Several studies have included the same individual in logistic regression models numerous times assuming that each subsequent season is not correlated within the individual ('Player-Season ID'). However, recurrent entries are unlikely to be statistically independent, and statistical methods are available that take correlation between events into account such as generalised linear mixed-models (Impellizzeri et al., 2020). The generalised linear mixed-model is an extension of conventional logistic regression (Generalised linear model) and overcomes the assumption of independence of observations by introducing a random effect to account for the unknown correlation between including individuals numerous times in the same analysis (Diggle et al., 2013).

4.4.3 Class imbalance: Accounting for injury as a rare event when analysing workload data

When conducting sports injury aetiology research that is focussed on examining whether a variable is associated with the risk of sustaining an injury, it is vital to ensure there is a sufficient number of injuries given the number of injury-free data points within the data set (Bahr, 2016). Indeed, a large proportion of researchers within the field of sports injury aetiology research employ statistical models such as logistic regression or GLMMs to estimate the odds of injury given the magnitude of a variable (Ruddy et al., 2019). However, these models require a minimum ratio of positive cases (i.e. injuries) to negative cases (i.e. injury free) to be observed without the minority class being considered a rare event (Conservative ratio >= 0.20, lenient ratio >= 0.10) (Peduzzi et al., 1996; Ruddy et al., 2019). The performance of these statistical models is reduced when the data used to train the models is predominantly comprised of one class (termed class imbalance); *the* model will always predict the more frequently occurring class leading to inflated type-2 error and erroneous model accuracy estimates biased (Krawczyk, 2016). However, class imbalance is inherently present many real-world applications such as the probability of individuals with debts defaulting on their payments (Krawczyk, 2016), detecting credit card fraud (He et al., 2008) and diabetes mellitus screening (Wang et al., 2019); or in the case of sports injury aetiology, the probability of injury given exposure to external workloads (i.e. the un-injured days vastly outnumber injured days). In these inherently unbalanced scenarios collecting more data will not result in class balance because more data will also be added to the majority class sample (e.g. high-speed running exposure during non-injured training days) as well as the minority class (e.g. hamstring injuries sustained during high-speed running) (Carey et al., 2018; Ruddy et al., 2019). A potential strategy for researchers to overcome class imbalance would be to wait for a sufficient number of injuries to occur, without the addition of other injury prevention measures. However, although this approach may work when collecting data once per year, when working with professional sports teams this may be in conflict with the medical staff's duty of care to the participating players. Furthermore, as previously mentioned this approach is simply not possible when examining the influence of

player workload on injury aetiology where data is collected multiple times per day, per week, per season for individuals resulting in thousands of data points. As a result, alternative statistical approaches are needed to reduce the negative impact of the observed class imbalance. The approaches to mitigate this issue broadly fall into three categories which are: A data level approach, where the data is modified in a manner that removes the class imbalance prior to analysis (Chawla et al., 2002; He et al., 2008; Stefanowski., 2016; Carey et al., 2018; Ruddy et al., 2019). A model or algorithm level approach, where the statistical model is modified to mitigate the bias toward the majority class (Krawczyk et al, 2015). Or a hybrid approach which combines the previous two categories (Woźniak et al., 2014). In the context of the thesis, an algorithm level approach was outside of the scope of the project, therefore the next section will focus on a data level approach.

4.4.3.1 Data level approaches to class imbalance: Majority class reduction and minority class oversampling

When employing a data-level approach, one can use majority sample reduction in order to reduce the number of data points (Krawczyk, 2016). As an example, when analysing player workload in relation to injury one could remove a subset of non-injured days from the data set (Carey et al., 2018).

An alternative option is to use a minority class oversampling technique to create synthetic samples are created based on pre-existing data from the minority class. The synthetic minority over-sampling technique (SMOTE) algorithm created by Chawla et al. (2002) was one of the first to be developed and is still commonly used in a variety of applications including injury aetiology research (Carey et al., 2018). SMOTE uses a k-nearest neighbours algorithm to add synthetic data points to the minority class based on the five nearest minority class data points (Figure 4.4.3.1) (Chawla et al., 2002).

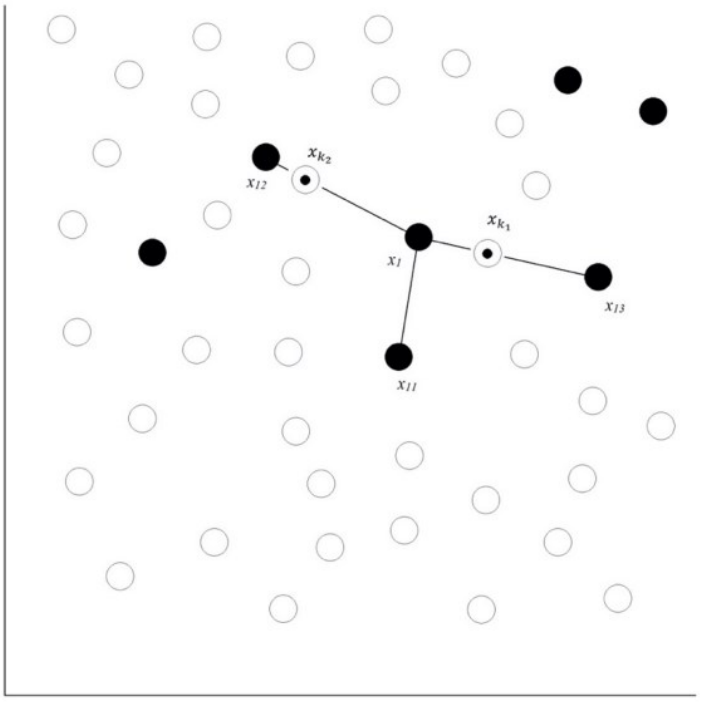


Figure 4.4.3.1a An abstracted graphical example of the SMOTE algorithm on an imbalanced data set for minority observation x_1 in two-dimensional space. Where x_1 is the minority data point of interest, x_{1i} are the nearest neighbours (k = 3) and x_{ki} are the synthetically generated data (Majority class data = white circles, minority class data = black circles and synthetic minority class data = white circles with black centre).

Despite the enduring popularity of the SMOTE algorithm, it is not without its limitations. The main limitation associated with SMOTE is the arbitrary generation of synthetic datapoints based on *all* of the minority class observations (He et al., 2008). This may result in the class-balanced dataset not reflecting the underlying distribution of the minority class in the original unbalanced data set, potentially impacting the any statistical models created using the data (He et al., 2008).

He et al. (2008) developed an alternative minority class oversampling algorithm termed the adaptive synthetic sampling approach (commonly referred to as ADASYN) to overcome the limitations of SMOTE. Rather than generating synthetic data points in a random distance between the minority data points of interest, the ADASYN algorithm generates synthetic data points based on the density of the majority class distribution (Figure 4.4.3.1b) (He et al., 2008). Therefore, within the of k-nearest neighbours regions where there is a higher density of majority class data points, more synthetic minority class data points are generated. Conversely, in k-nearest neighbours regions that have a sparse distribution of majority class data points, less

synthetic minority class data points are generated. Most importantly, in areas where no majority class data is present, no synthetic minority class data points are generated. He and colleagues' rational for the above logic was that, within an imbalanced data set the areas with a higher distribution of majority class data points would be harder to accurately classify compared with areas with fewer or no majority class data points (He et al., 2008).

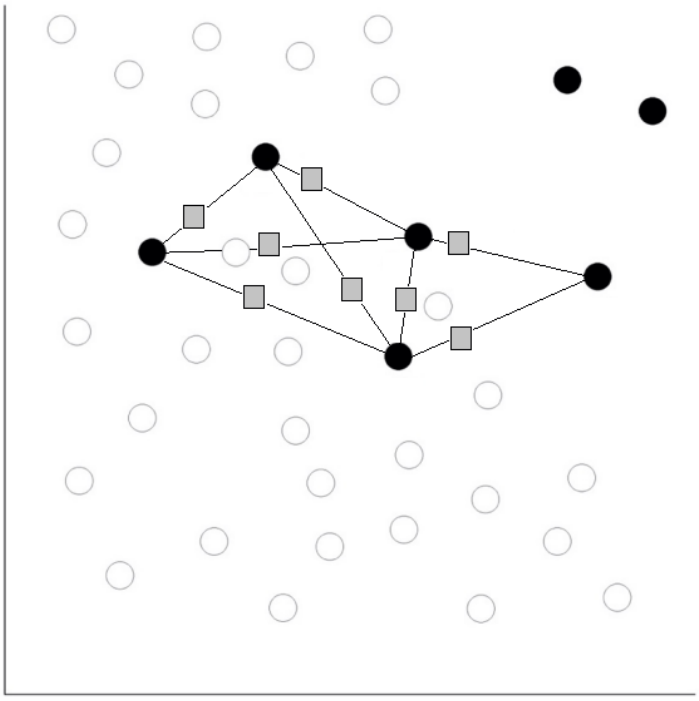


Figure 4.4.3.1b An abstracted graphical example of the ADASYN algorithm on an imbalanced data set, synthetic minority data points are clustered around high density areas of majority class data points. (Majority class data = white circles, minority class data = black circles and synthetic minority class data = grey squares).

When examining workload data in relation to injury hamstring and knee ligament injury aetiology within the subsequent experimental chapters of the thesis, the data will likely exhibit class imbalance. Therefore, the adoption of the processes outlined within this chapter to mitigate the influence of class imbalance is necessary. Specifically, selective majority class reduction and where appropriate minority class oversampling using ADASYN.

4.5 Summary of aetiological literature review and rationale for the subsequent experimental studies

The overall aim of the Thesis was to advance knowledge of high burden lower limb injury aetiology within men's professional rugby union. The epidemiological analysis conducted in chapter 3 highlighted that hamstring injuries, specifically those sustained to the biceps femoris whilst running and injuries to the MCL and ACL during contact events resulted in the highest magnitudes of injury burden.

Sports injury aetiology is a complex problem that is dependent on a multitude of factors including characteristics intrinsic to the at-risk athlete of interest, some of which are modifiable (e.g. lower limb strength) and some that are not (e.g. previous injury history) (Meeuwisse, 1994; Meeuwisse et al., 2007; Windt and Gabbett, 2016; Bittencourt et al., 2016). Other factors are external to the athlete, including workloads during matches and training which may compromise repeated exposure to injurious inciting events (e.g. sprinting or tackle events) (Gissane et al., 2001; Meeuwisse et al., 2007; Windt and Gabbett, 2016; Bittencourt et al., 2016).

The review of the literature in chapter 4 highlighted a common non-modifiable intrinsic variable associated with both an increased risk of hamstrings muscle injury and knee ligament injury was previous injury history. The effects of both previous injury to the tissue(s) of interest (e.g. the effect of previous hamstrings muscle strain injury on subsequent hamstring muscle strain injury), and previous injury to other tissues (e.g. the effect of previous ACL injury on subsequent hamstring muscle strain injury) were previously examined.

Previous literature surrounding modifiable variables that were intrinsic to an athlete were also explored in in chapter 4. The strength of the hamstrings and quadriceps muscle groups have been frequently explored in relation to both the aetiology of hamstring muscle strains and knee ligament injuries via isokinetic dynamometry. There was strong evidence of the lack of a causal relationship between isokinetic dynamometry derived measures of strength and hamstring muscle strain. Further to this, contrasting findings were observed between isokinetic variables and the odds of sustaining knee ligament injuries. However, further research was required due to the continued use of isokinetic assessment of the knee joint during injury screening PHEs in professional team sports such as rugby union including the team participating and co-funding the current work contained within the thesis.

The manner in which an athlete moves as well as the accompanying forces acting on and produced by said individual have long been considered modifiable internal aetiological variables. A number of sources examined in chapter 4 cited that several biomechanical variables occurring

during the early stages of ground contact were associated with knee ligament injury, however, the majority of the study designs are either retrospective in nature or cross-sectional research designs that have compared symptomatic individuals with injury-free controls. At the time of writing there was a paucity of studies prospectively investigating the influence of biomechanical variables during a single-leg drop jump task in relation to primary ACL and MCL risk in male team sport athletes. Due to this, prospective investigation of biomechanical variables in relation to knee ligament injury risk was warranted, and was therefore introduced as a novel part of the testing battery.

Extrinsic variables associated with the external workload athletes undertake during training sessions and match play have received a large research focus in recent years in relation to lower limb injury aetiology. The review of the literature in chapter 4 highlighted that despite a large number of prospective studies examining external workload variables in relation to general lower limb injury (e.g. any lower limb soft tissue injury), there was a lack of research focussed on the aetiology of specific injuries (e.g. hamstring strains sustained during sprinting; knee ligament injuries sustained during contact events). Limited evidence suggests that both chronic and acute exposure to high-speed running are related to hamstring injury, therefore further prospective research was required to validate the continued use by the participating team. In contrast, despite a large number of epidemiological studies identifying contact events such as the tackle are responsible for a high incidence of knee ligament injuries, no prospective research has examined the manner in which this exposure is related to knee ligament aetiology, and as a result was included as a novel variable.

Therefore, in order to further the research area of both hamstring strain and knee ligament injury aetiology in men's professional rugby union players, a comprehensive approach is needed in accordance with previously established models of sports injury aetiology research (Meeuwisse, 1994; Meeuwisse et al., 2007; Windt and Gabbett, 2016; Bittencourt et al., 2016). The following novel research questions that will be addressed in each experimental study as follows:

Chapter 5: Study 2 An exploration of factors associated with hamstring strain injury in male professional rugby union players

Question 1. What factors are associated with an increased risk of hamstring injury in men's professional rugby union?

Question 1.1. Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

Question 1.2. Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

Question 1.3. Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players?

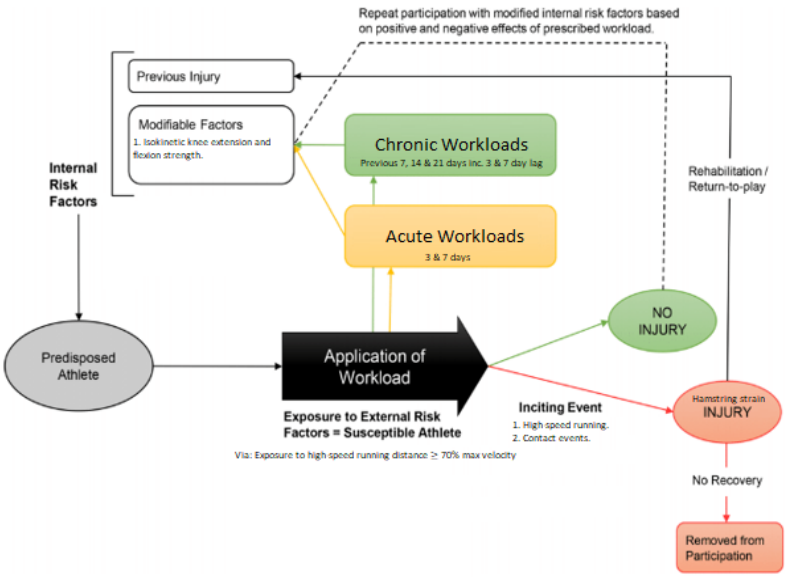


Figure 4.5a Theoretical workload-injury aetiology model for hamstring strain injuries sustained by male professional rugby union players which comprises the work undertaken in Chapter 5. Adapted from Windt and Gabbett (2016).

Chapter 6: Study 3 An exploration of factors associated with knee ligament injury in male professional rugby union players

Question 2. What factors are associated with an increased risk of MCL and ACL injury sustained during contact events in men's professional rugby union?

Question 2.1. Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players? i.e. Will individuals exposed to more tackles and rucks have an increased risk of sustaining MCL or ACL injury from a tackle or ruck event?

Question 2.2. Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players?

Question 2.2.1. Is previous lower limb injury history associated with the risk of sustaining an MCL or ACL injury from a contact event?

Question 2.2.2. Are previously identified isokinetic knee strength variables associated with the risk of sustaining MCL or ACL injury within the cohort of male professional rugby union players?

Question 2.2.3. Are lower limb biomechanical variables during the early ground contact phase of a single-leg drop-jump task associated with the risk of sustaining MCL and ACL injury within the cohort of male professional rugby union players?

Question 2.2.4. Are acute increases in exposure to on-pitch physical activity associated with an increased risk of MCL and ACL injury within male professional rugby union players?

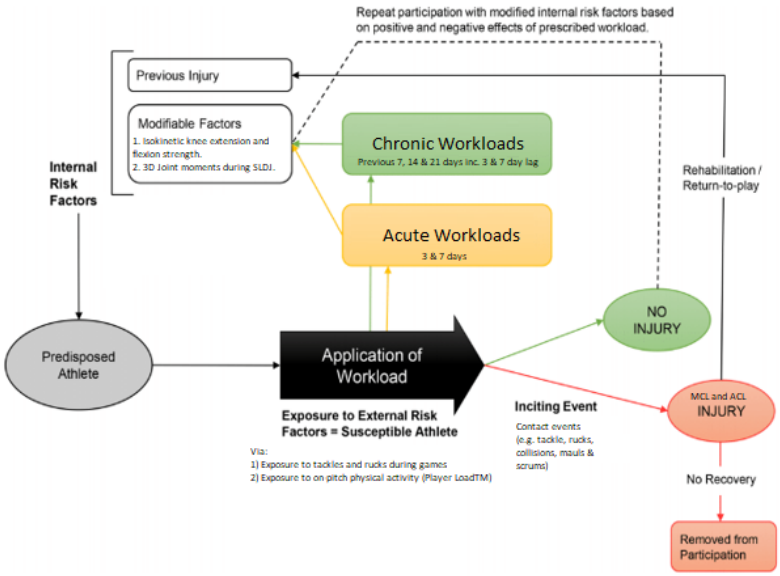


Figure 4.5b Theoretical workload-injury aetiology model for contact MCL and ACL injuries sustained by male professional rugby union players which comprises the work undertaken in Chapter 5. Adapted from Windt and Gabbett (2016).

Chapter 5: Study.2. An exploration of factors associated with hamstring strain injury in male professional rugby union players

5.1 Context

In the context of the PhD project, hamstring strains presented a high injury burden to the professional rugby team co-funding the project as well as the greater men's professional rugby union population. Establishing the efficacy of the current methods employed by the team to infer hamstring strain risk, and where possible proposing alternative solutions were important to ensure the research made a positive impact to the participating team and provided a meaningful contribution to the field of injury research in rugby union by informing the decision-making process in relation to player hamstring strain risk.

5.2 Introduction

Hamstring injury (HSI) poses a high injury burden in men's professional rugby union (Brooks et al., 2006; Kemp et al., 2019) and other field-based team sports (Askling et al., 2013; Ruddy et al., 2018), with the consequences impacting the performance and neuromuscular function of the injured player upon return to play. This may culminate in decreased team performance, impact individual player and team performance, as well as reduce the financial earnings of the individual and professional rugby team (Cross et al., 2016; Whiteley et al., 2021). Epidemiological studies examining time-loss sustained by male professional rugby union players have consistently reported that hamstring injuries exhibited amongst the highest incidence rates combined with moderate severities over the past 10 years (Kemp et al., 2019). These previous epidemiological findings were also consistent with that of the current work (Chapter 3 – Study 1 Injury epidemiology study). In accordance with previously established frameworks of injury prevention, once the epidemiological extent of an injury problem has been identified, examining the underpinning aetiology of hamstring injury is necessary in order to iteratively aid the

development of preventative measures, with the end goal being a reduction in hamstring injury incidence and severity (van Mechelen et al., 1987; Finch, 2006).

In a recent meta-analysis, Green et al. (2020) reported that numerous risk factors for hamstring injury have been proposed, including those that are non-modifiable such as age and a history of hamstring injury, which the authors observed to have the strongest evidence for a relationship with hamstring strain risk. In the context of male professional rugby union players, Williams et al. (2017) reported hamstring injuries had the third highest proportion of injury recurrence. Previous injuries to the knee ligaments, calf and ankle joint complex were also observed to increase subsequent hamstring injury risk (Green et al., 2020).

Green and colleagues (2020) additionally identified modifiable risk factors, which included musculoskeletal variables associated with hamstring strength, and external risk factors associated with exposure to high-speed running. Isokinetic assessment of hamstring strength has been the predominant method to establish hamstring strength and has been ubiquitously used as a proxy for hamstring injury risk within team sports for the past ~20 years (including the participating team) (Orchard et al., 1997; Green et al., 2020). However, recent meta-analyses have reported that isokinetic variables have little relationship with the risk of sustaining a hamstring injury (Green et al., 2017; Green et al., 2020). Despite this, lower limb isokinetic assessment is still routinely used in periodic health assessments and the decision-making process regarding hamstring injury risk in professional sports such as professional rugby union, including the professional rugby team co-funding this research project. A limitation with the majority of previous research within this area is that injuries to all hamstring muscles have been analysed rather than examining the individual muscles (biceps femoris, semimembranosus and semitendinosus) or in some cases the inciting event that resulted in injury. As these muscles have different architectural properties and functions (Kellis, 2018), the utility of isokinetic dynamometry may be overestimated with pooled ‘all hamstring injury’ data sets. Therefore, examination of individual muscle injury is needed to fully evaluate the efficacy of isokinetic

dynamometry in relation to hamstring injury risk. Furthermore, at the time of writing (2021) there has been no examination of whether a relationship exists between lower limb isokinetic variables and the risk of sustaining hamstring injury within a specific male professional rugby union player population.

High-speed running has been one of the most frequently cited inciting events for hamstring strains (Brooks et al., 2006; Kemp et al., 2019), accounting for the highest hamstring strain incidence rates in men's professional rugby union. Both researchers and practitioners have suggested that unaccustomed increases in high-speed running exposure would increase the risk of hamstring injury (Brooks et al., 2006; Green et al 2020; Stokes et al., 2020; Freeman et al., 2021). Despite this commonly held view, at the time of writing (2021) only a limited number of studies have specifically examined the relationship between high-speed running (or any external workload variables) and hamstring injury (Dulig et al., 2016; Ruddy et al., 2018). All studies observed that large weekly increases in high-speed running exposure were related to an increased risk of hamstring strain injury, supporting the previously mentioned hypothesis. These studies were conducted within Australian Rules football rather than rugby union which limits the generalisability for the current population due to the different activity profiles of each sport and moreover the diverse and specialised positions within rugby union. A number of studies have examined the relationship between workload and the risk of sustaining soft tissue injuries (pooled muscle and ligament including hamstring strain) in field-based team sports including rugby union (Colby et al., 2017; Malone et al., 2017; Colby et al., 2018; Malone et al., 2018; Cousins et al., 2019; Malone et al., 2019; Hulin et al., 2020), with the most popular independent variable being the acute:chronic workload ratio (ACWR) (Hulin et al., 2014). This is the ratio of the most recent exposure of workload ("acute workload") theorised by the authors to represent a component of fatigue and previous longer-term exposure to workload ("chronic workload") theorised to represent fitness (Gabbett, 2016; Windt and Gabbett, 2017). Using this logic researchers have also inferred that consistent chronic exposure to high-speed running may reduce the risk of hamstring strains (Edouard et al., 2019), with chronic exposure to moderate

magnitudes of high-speed running previously observed to reduce the risk of muscle strain injury in Gaelic football (Malone et al., 2017) and soccer (Malone et al., 2018). However, recent critical commentaries have questioned the appropriateness of the ACWR as a variable for inferring injury risk (Impellizzeri et al., 2020; Kalkhoven et al., 2021), with it being suggested that the use of ratio data such as the ACRW would potentially lead to spurious associations. Furthermore the approach of using a standardised ACWR (e.g. 7 day acute / 28 day chronic ACWR) adopted by many sports teams (including the participating professional rugby union team) to infer the risk of a plethora of injuries (e.g. soleus vs biceps femoris injury risk) and inciting events (e.g. injured whilst running vs injured during contact) may be overly simplistic (West et al., 2021c); and does not adhere to the principles presented in theoretical models of injury aetiology which suggests that injury risk is complex and multifactorial in nature, with internal and external factors associated with injuries varying between injury types, mechanisms and even inciting events to varying degrees for each individual (van Mechelen et al., 1992; Meeuwisse, 1994; Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2016; Bittencourt et al., 2016). Therefore, before the ACWR can be used to examine hamstring injury risk within men's professional rugby union one must first examine the manner and time course in which acute and chronic exposure to high-speed running influences hamstring injury risk, and in relation to other variables such as hamstring strength, age and previous hamstring injury history.

Therefore, the purpose of this study was twofold. Firstly, to determine if previously identified injury history and isokinetic hamstring strength variables were associated with specific hamstring strain injuries occurring during specific inciting events within male professional rugby union players. Secondly, to establish whether a relationship existed between high-speed running exposure and the risk of sustaining hamstring strains that occurred during high-speed running. To facilitate this, the following research questions were explored:

1. Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

H1: Sustaining a previous hamstring strain injury would increase the risk of sustaining a subsequent hamstring strain injury regardless of individual muscle and inciting event.

H2: Sustaining a previous lower-limb injury to another location would increase the risk of sustaining a hamstring strain injury regardless of individual muscle and inciting event.

2. Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

   H3: Players with larger magnitudes of isokinetic knee flexion strength would be less susceptible to hamstring strain regardless of muscle type or inciting event.

3. Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players?

   H4: Acute exposure to higher magnitudes of high-speed running would increase the risk of sustaining a hamstring strain whilst running.

   H5: Chronic exposure to moderate magnitudes of high-speed running would decrease the risk of sustaining a hamstring strain whilst running.

5.3 Methods

5.3.1 Participants

A total of 143 professional rugby union players from one club competing in the English premiership took part in the study spanning seven playing seasons (2012-13 Season to 2018-19 Season), which included repeated measurement of players equating to 414 player-seasons (Figure 5.3.1b). At the time of writing the team competed in the highest level of national competition in professional rugby union in England. At the start of each playing season during the second week of the preseason period, players underwent a periodic health examination (PHE) at the University of Exeter. As part of the PHE, assessment of knee joint isokinetic extension and flexion strength was performed.

Due to the longitudinal nature of the study, the majority of the exclusion criteria applied to the exclusion of players from the current playing season rather than the entire study. Players from the senior playing squad that sustained injuries to the hamstrings muscle group during a competitive match or during training sessions were included in the study. The following exclusion criteria were used for the study:

1. Once a player left the team, they were excluded from the analyses of subsequent playing seasons.
2. Players were excluded from the analysis of the playing season if they sustained a time loss injury prior to completing isokinetic testing within the same playing season.
    a. If the player returned from injury before the start of the next playing season, they were then re-entered into the analysis for the subsequent playing season.
    b. If the player did not return from injury before the start of the next playing season, they were then excluded from the analysis for the subsequent playing season.
3. Players were excluded from the analysis of the playing season if they were unable to undergo isokinetic testing within the same playing season.
    a. If the player was able to undergo isokinetic testing during the subsequent season they were re-entered into the analysis for the subsequent season.
    b. If the player was not able to undergo isokinetic testing during the subsequent season they were excluded from the analysis for the subsequent season.
4. Players who subsequently sustained lower limb time loss injury other than HSI after isokinetic testing within the same season were excluded from the analysis of the playing season.
    a. If the player returned from injury before the start of the next playing season, they were then re-entered into the analysis for the subsequent playing season.

b. If the player did not return from injury before the start of the next playing season, they were excluded from the analysis for the subsequent playing season.

5. Players who subsequently sustained a HSI but did not perform isokinetic testing within the same season were excluded from the analysis of the playing season.
    a. If the player returned from HSI before the start of the next playing season AND performed isokinetic during the subsequent PHE, they were then re-entered into the analysis for the subsequent playing season.
    b. If the player returned from HSI before the start of the next playing season AND they did not perform isokinetic testing during the subsequent PHE, they were excluded from the analysis for the subsequent playing season.

A worked example of the exclusion criteria is presented in figure 5.3.1a Furthermore, a flowchart illustrating the movement of players and repeated testing as well as the inclusion/exclusion criteria on the study cohort is presented in figure 5.3.1b. The study was approved by the University of Exeter Sport and Health Sciences ethics committee.

Figure 5.3.1a A worked example of the study exclusion criteria.

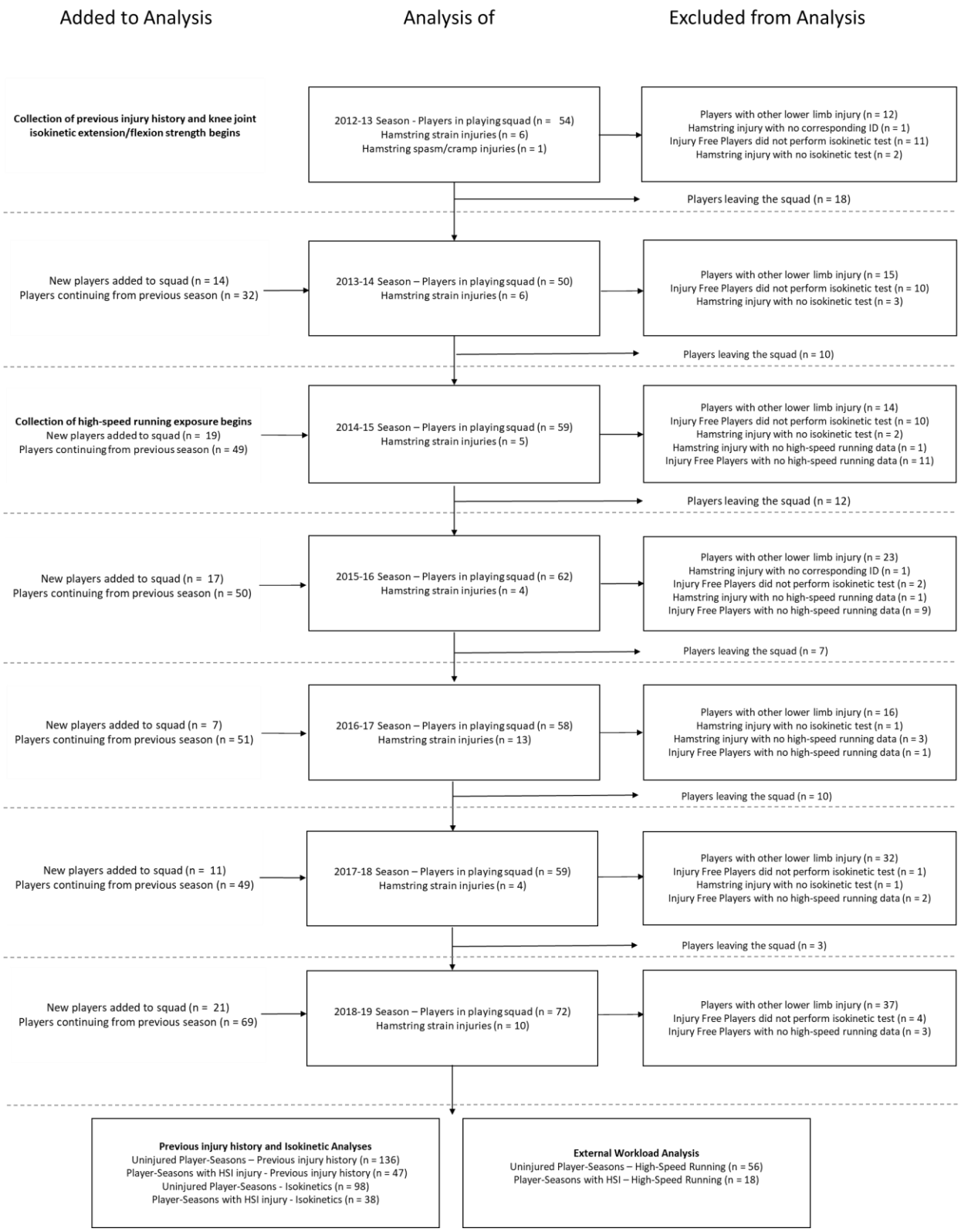


Figure 5.3.1b A flowchart illustrating the movement of players throughout the study period.

5.3.2 Data collection

5.3.2.1 Injury definition and surveillance

Hamstring injury was defined as acute pain located in the posterior thigh which occurred during either match play or training which was not due to laceration, abrasion or a haematoma (van Dyk et al., 2016). In order to compare hamstring injury data from the current study to previous studies investigating hamstring injury in men's professional rugby union only hamstring injuries that resulted in time loss were examined; a time loss hamstring injury was defined as "any injury to the hamstring group of muscles (excluding lacerations, abrasions, and hematomas) that prevented a player from taking full part in all training activities typically planned for that day and/or match play for more than 24 hours, from midnight at the end of the day the injury was sustained." (Brooks et al., 2006). Hamstring injuries were diagnosed based on clinical examination of the injured player by medical personnel at the rugby club, the diagnosis included pain on palpation, pain during isometric contraction and pain during muscle lengthening (van Dyk et al., 2016). The clinical diagnosis was supported with ultrasound and/or magnetic resonance imaging. Where evidence of muscle fibre disruption was present the hamstring injury was defined as a muscle strain (Brooks et al., 2006); where no fibre disruption was observed the injury was classified as "hamstring spasm/cramps/trigger points with no clinical evidence of fibre disruption" (Brooks et al., 2006). Hamstring injuries were further categorised by specific location (i.e. "biceps femoris", "medial hamstring - semitendinosus/semimembranosus" or as "muscle subgroup not identified") and inciting event in accordance with according with previous research (Fuller et al., 2007a; Brooks et al., 2006; Kemp et al., 2019). Injuries were reported using a modified Orchard Sports Injury Classification System (OSICS) (Orchard, 1995). Individual exposure data in hours were collected weekly for matches and training sessions for all players included in the study (Fuller et al., 2007a).

5.3.2.2 Knee joint isokinetic flexion strength

Isokinetic knee flexion and extension moment were assessed using an isokinetic dynamometer (Biodex Multi-Joint System 3; Biodex Medical Systems Inc, Shirley, NY, USA). Players performed a 10-minute warm-up consisting of cycling on a cycle ergometer (Monark 874E; Monark, Vansbro, Sweden) followed by personal stretching. Prior to each testing mode, players were provided with verbal instruction regarding the procedures of the specific tests and were allowed 5 practice repetitions with each test followed by a 2-minute rest period prior to the test.

All players were positioned on the isokinetic dynamometer with the hip joint flexed at 90°, with the dynamometer and seat adjusted to ensure that the knee joint centre of rotation was in line with the dynamometer centre of rotation (Arampatzis et al., 2004; Baltzopoulos et al., 2012). The knee joint was assessed over a 90° range of motion (End range extension = 5° from full knee extension, end range flexion = 95° from full knee extension). In order to minimise the error from secondary joint movement, straps were fixed across the trunk, waist and the thigh that was being tested, the non-testing lower limb was also secured and players were instructed to hold their arms across their chest (Otten et al., 2013). Verbal explanation was also given to the players to ensure secondary joint movement was at a minimum.

The isokinetic testing protocol was comprised of 4 different modes and isokinetic speeds investigated in previous research (Steffen et al., 2016; Bakken et al., 2018). Players performed 5 repetitions of concentric knee extension and flexion at 60 deg/s first. This was followed by 5 repetitions of concentric knee extension and flexion at 180 deg/s. The players then performed 5 repetitions of eccentric knee extension at 60 deg/sec. Finally, 5 repetitions of eccentric knee extension at 180 deg/sec were performed. A 2-minute rest period was provided between each testing mode. When all of the testing modes were completed on one side, the other limb was tested. During testing, all players were provided with verbal encouragement by those performing the testing (Hamilton et al., 2014; Tol et al., 2014).

5.3.2.3 High-speed running exposure

The external workload players were subjected to was quantified using a global positioning system (GPS), sampling at 10 Hz, which also housed a triaxial accelerometer, triaxial gyroscope and magnetometer, each sampling at 100 Hz (Optimeye X4; Catapult Innovations, Melbourne, Australia). During matches and training sessions, players wore a tight-fitting garment which housed the unit in the upper-back region, between the shoulder blades (Roe et al., 2016). Data from each unit was downloaded onto a laptop and processed using proprietary software (Catapult Openfield software; Catapult Innovations, Melbourne, Australia). Previous research employed absolute velocities when examining high-speed running exposure in relation to hamstring injury risk (Dulig et al., 2016; Ruddy et al., 2018). These studies were conducted in AFL and soccer populations where positions are more homogenous when compared with more specialist positions in Rugby Union (e.g. a 125 kg prop vs an 85 kg scrum half) which may encounter disparate physiological demand when running at the same absolute speed (Reardon et al., 2015), and for that reason relative speed zones are commonly used in professional rugby union (West et al., 2019). Relative velocity zones of ≥70%, ≥80% and ≥90% of max velocity (V_{max}) were used to quantify high-speed running exposure by the industry funder of the PhD project. High-speed exposure distances ≥70% V_{max} were selected for analysis over the other bands because some hamstring injuries were sustained without traveling any distance ≥80% or ≥90% V_{max}, furthermore, ≥70% V_{max} has been previously used to quantify high-speed running exposure in men's professional rugby union (Cousins et al., 2019).

5.3.3 External workload data analysis

High-speed running data were collected for every available player per on-field session per day. These were then aggregated to give a daily high-speed running distance per available player. In order to correctly calculate moving sum and moving average data on days where players did not play or train (e.g. within week non-training day & off season) zero values were imputed to the data-set for each player. Seven day summed high-speed running exposure was calculated for

the week of injury i.e. 0-7 days prior to injury (7-day sum) as well as the week prior to injury i.e. 8 – 14 days prior to injury (7-day sum 7-day lag) Exponentially-weighted moving averages (EWMA) were calculated over 3 and 7 days for acute exposure to high-speed running. Chronic exposure to high-speed running EWMA were calculated over 7-, 14- and 21-day timeframes. In addition, chronic variables were also calculated where high-speed running exposure was excluded within the 3 and 7 days prior to sustaining an injury (EWMA 3-day & EWMA 7-day lag). The exponentially-weighted moving average was calculated using the equation of Williams et al. (2017c):

$$EWMA_{today} = Workload_{today} \times \lambda a + ((1 - \lambda a) \times EWMA_{yesterday}$$

The term λa represents the decay factor and is a value between 0 and 1.

The formula for the decay factor is:

$$\lambda a = 2/(N + 1)$$

The term N is the time decay constant, equating to the previously mentioned timeframes (Williams et al., 2017c).

5.3.4 Statistical analysis

Statistical analysis was conducted using R open-source statistical software (version 4.0.3, R Foundation for Statistical Computing, Vienna, Austria). The independent variables' relationship with hamstring injury risk was analysed using generalised linear mixed-models (GLMM), which were selected for their ability to overcome the assumption of independence of observations, resulting in an inflated type-1 error rate when using conventional general linear models employed by the majority of previous studies examining player workload in relation to injury risk (Williamson et al., 1996; Diggle et al., 2013; Impellizzeri et al 2020). Models were fitted using the lme4 package, with sustaining a hamstring injury ("yes"/"no") as the dichotomous outcome variable.

The study included data from players over repeated days and seasons that were likely correlated (exploratory data analysis revealed this to be true for some variables); however, the magnitude of correlation was assumed to vary for each player. Therefore, a random effect was included in the model for player ID to account for the unknown correlation caused by including multiple player-days and player-seasons in the analyses.

Class imbalance was also identified as a common source of classification error when examining player workload in relation to injury risk i.e. the number of non-injured days outnumber the number of injured days (resulting in inflated type-2 error) (Kuhn and Johnson, 2013; Carey et al., 2018; Ruddy et al., 2019). The unfiltered dataset exhibited class imbalance with 18 injuries and 5408 injury free player-days resulting in a ratio of 0.003. For all analyses involving high-speed running exposure, a majority sample reduction approach was chosen to overcome the issue of class imbalance (Krawczyk et al., 2016; Carey et al., 2018). Following the calculation of EWMA variables the following majority sample reduction approach process was employed:

1. Data was sampled from the high-speed running dataset which comprised of daily training and match play data for 143 players over 5-years.

2. The only high-speed running exposure data points selected from the 143 players that passed the inclusion criteria and either occurred:

a. On the dates hamstring strain injuries occurred where running was the inciting event.

And b. The 28 days prior to the dates where hamstring injury occurred.

The resulting data set (post majority sample reduction) contained continuous 29 days high-speed running exposure data for each hamstring injury of interest for all participants (18 injuries, 1851 injury free player-days with a ratio of 0.01).

All variables were modelled as tertile categorical groups to allow for interpretation for the rugby team participating in the study. The tertile categorical groups were created based on frequency into low magnitude, medium magnitude and high magnitude groups, with the reference group being the medium group. For multivariate analysis the moderating variate was entered as a categorical variable ("yes"/"no") depending on the outcome of the univariate analysis. Statistical significance for each model was tested using the likelihood ratio test to determine overall model significance and profiled 95% confidence intervals of the odds ratio to determine if each category was significant. Within sample model fit was assessed by calculating the area under the receiver operating characteristic (ROC) curve also referred to as AUC score. The ability of the model to classify novel data was internally validated using leave-one-out cross validation (LOOCV) with an adjusted mean AUC score and 95% confidence intervals of the AUC scores calculated from all LOOCV iteration. Classification performance was defined by the following rule of thumb by Hosmer et al. (2013): ≤ 0.50 = no better than random guess; ≥ 0.5 - < 0.7 = poor classification performance; ≥ 0.7 - < 0.8 = acceptable classification performance; ≥ 0.8 - < 0.9 = excellent classification performance; ≥ 0.9 = outstanding classification performance.

5.4 Results

During the 7-year study period, a total of 49 hamstring muscle strain/tear injuries occurred resulting in a total of 2337 days of player absence. Table 5.1 provides an overview of the number of hamstring muscle strain/tear injuries sustained during the study period in relation to inciting event and data collection modality.

Table 5.4 Hamstring injury in relation to inciting event and data collection modality.

	Total Participants	Total Player-Seasons	All Hamstring Strain Injury	Hamstring subgroup not identified	Biceps-femoris	Semimem/Semiten
2012 -13 to 2018-19 sample n - Previous Injury History	117	183	47	5	25	17
Running inciting event			32	3	21	8
Contact-based inciting events*			15	2	6	7
Miscelanious inciting event**			2	0	0	2
2012 -13 to 2018-19 sample n - Isokinetics	84	136	38	5	18	15
Running inciting event			23	3	14	6
Contact-based inciting events*			13	2	4	7
Miscelanious inciting event**			2	0	0	2
2014 -15 to 2018-19 sample n - High-Speed Running Exposure						
Running inciting event	51	71	18	2	11	5

* denotes inciting events involving contact with at least one other player, categorised as: rucking, tackling, being tackled, scrummaging, collision, or other contact. ** denotes miscelanious inciting events categorised as: lineout landing or unknown inciting event.

5.4.1 Question 1. Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

Table 5.2 presents results from univariate analyses that examined the influence of previous injury history variables and sustaining hamstring strain injury regardless of muscle type and inciting event. Sustaining a previous hamstring strain, MCL injury or lateral ankle ligament injury were associated with an increased odds of sustaining hamstring strain injury ($P \leq 0.05$, OR 95% CI > 1) (Table 5.4.1a; Figure 5.4.1a, Figure 5.4.1b). However, sustaining a previous hamstring strain injury within a year, sustaining a previous ACL injury and sustaining a previous calf muscle strain were not associated with sustaining a subsequent hamstring strain injury ($P > 0.05$) (Table 5.4.1a).

Table 5.4.1a Univariate generalized linear mixed-models examining the association between previous injury history and hamstring strain injury regardless of muscle type and inciting event.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previous Hamstring Injury History (per injury)	0.03*	2.21 (1.08 - 4.52)	0.71	0.60 (0.50 - 0.70)
Previous Years Hamstring Injury History (per injury)	0.07		0.70	
Previous ACL Injury History (per injury)	0.13		0.69	
Previous MCL Injury History (per injury)	0.01*	4.06 (1.4 - 11.79)	0.70	0.56 (0.44 - 0.68)
Previous Years MCL Injury History (per injury)	0.06		0.68	
Previous Calf Injury History (per injury)	0.12		0.69	
Previous Years Calf Injury History (per injury)	0.11		0.70	
Previous Ankle Ligament Injury History (per injury)	0.0001*	8.85 (2.55 - 30.69)	0.75	0.67 (0.56 - 0.77)
Previous Years Ankle Ligament Injury History (per injury)	0.0001*	8.64 (1.71 - 43.61)	0.74	0.62 (0.52 - 0.73)

*** denotes statistical significance (P ≤ 0.05)**

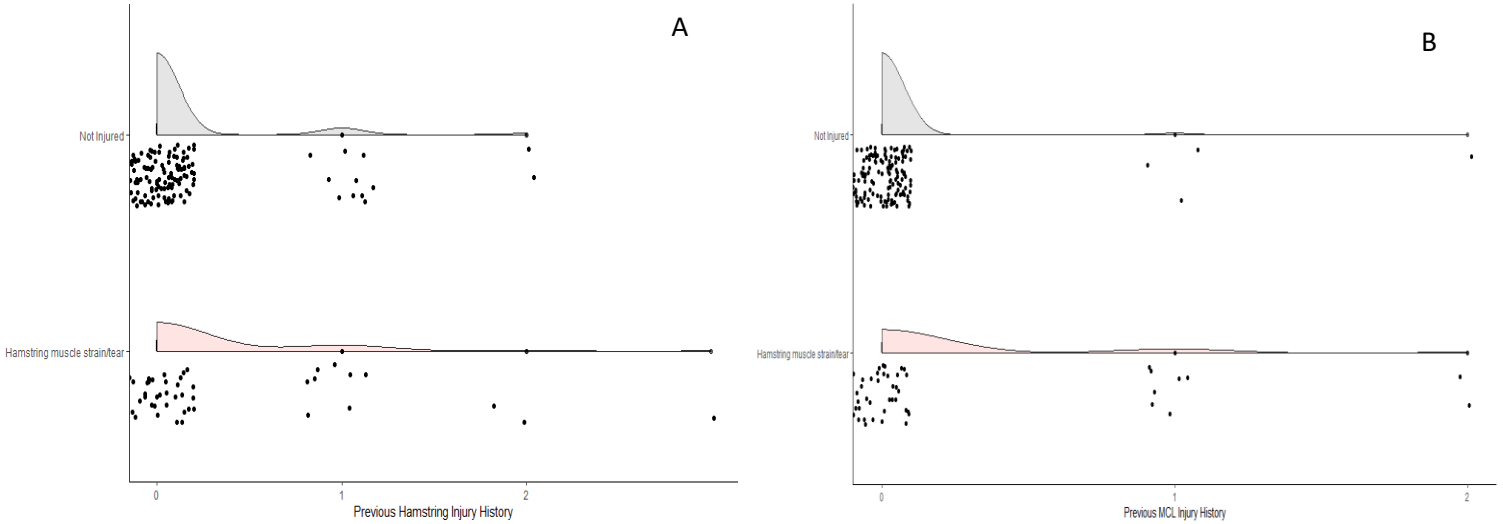


Figure 5.4.1a Distribution of A) previous hamstrings muscle group injury and B) previous MCL injury for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red).

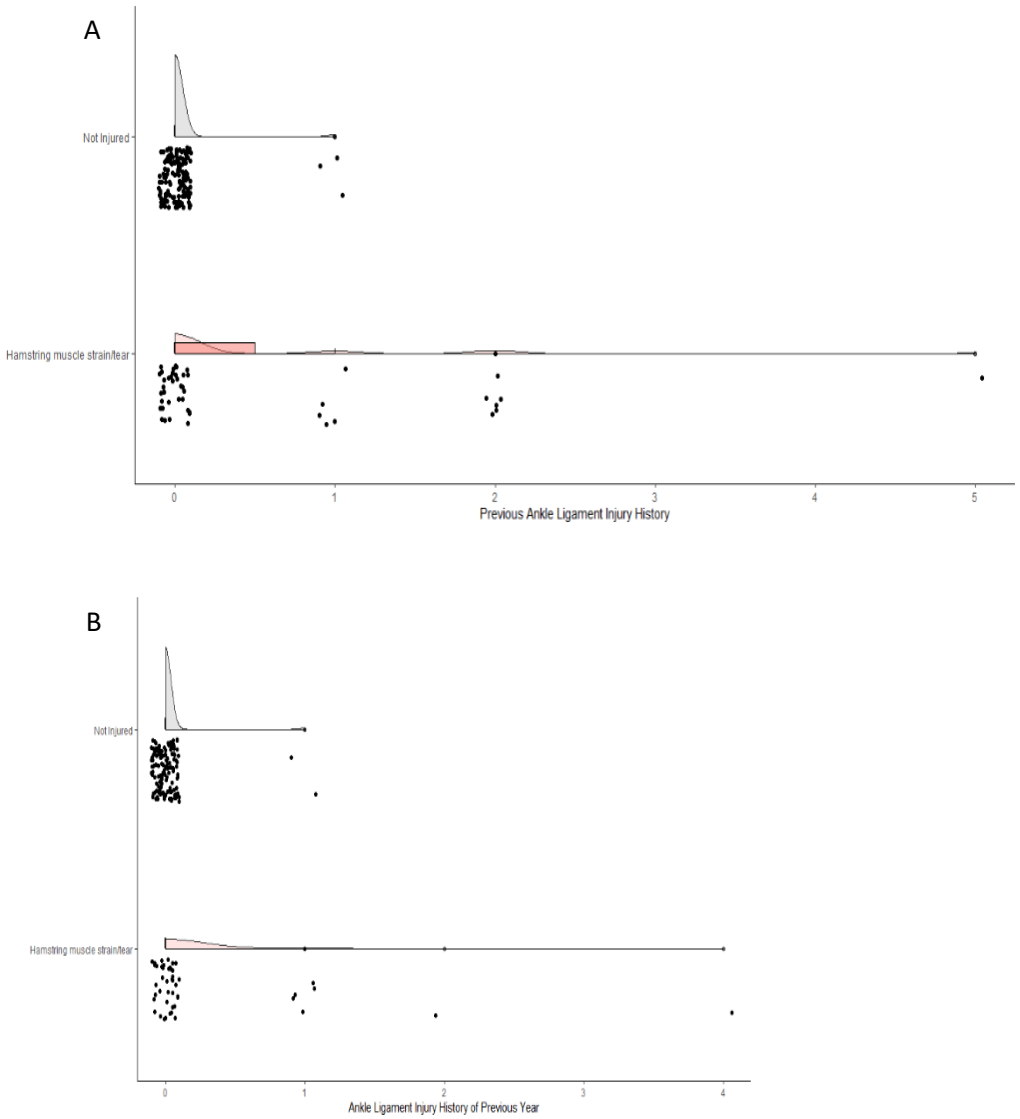


Figure 5.4.1b Distribution of A) previous ankle ligament injury and B) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red)

Table 5.4.1b presents results from univariate analyses that examined the risk of sustaining hamstring strain injury whilst running (regardless of muscle type) in relation to previous injury history variables. Sustaining a previous MCL injury and lateral ankle ligament injury were both associated with an increased odds of sustaining hamstring strain injury ($P \leq 0.05$, OR 95% CI > 1) (Table 5.4.1b; Figure 5.4.1c & Figure 5.4.1d). However, a previous hamstring strain injury, ACL injury and a calf muscle strain were not associated with sustaining a subsequent hamstring strain injury ($P > 0.05$) (Table 5.4.1b).

Table 5.4.1b Univariate generalized linear mixed-models examining the association between previous injury history and hamstring strain injury regardless of muscle type sustained whilst running.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previous Hamstring Injury History (per injury)	0.06		0.71	
Previous Years Hamstring Injury History (per injury)	0.65		0.68	
Previous ACL Injury History (per injury)	0.21		0.69	
Previous MCL Injury History (per injury)	0.05*	3.69 (1.02 - 13.39)	0.69	0.37 (0.25 - 0.5)
Previous Years MCL Injury History (per injury)	0.11		0.68	
Previous Calf Injury History (per injury)	0.18		0.66	
Previous Years Calf Injury History (per injury)	0.25		0.69	
Previous Ankle Ligament Injury History (per injury)	0.0001*	7.47 (2.16 - 25.91)	0.75	0.55 (0.42 - 0.67)
Previous Years Ankle Ligament Injury History (per injury)	0.0001*	6.61 (1.26 - 34.57)	0.74	0.57 (0.44 - 0.69)

*** denotes statistical significance (P ≤ 0.05)**

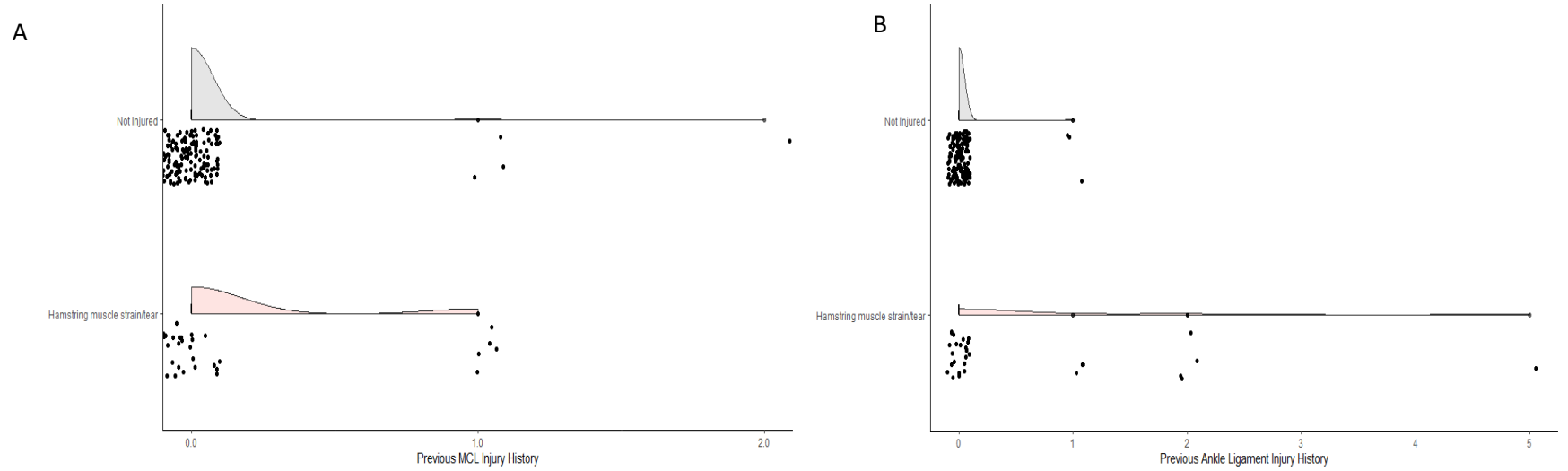


Figure 5.4.1c Distribution of A) previous MCL injury & B) previous ankle ligament injury for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained whilst running (shaded in red).

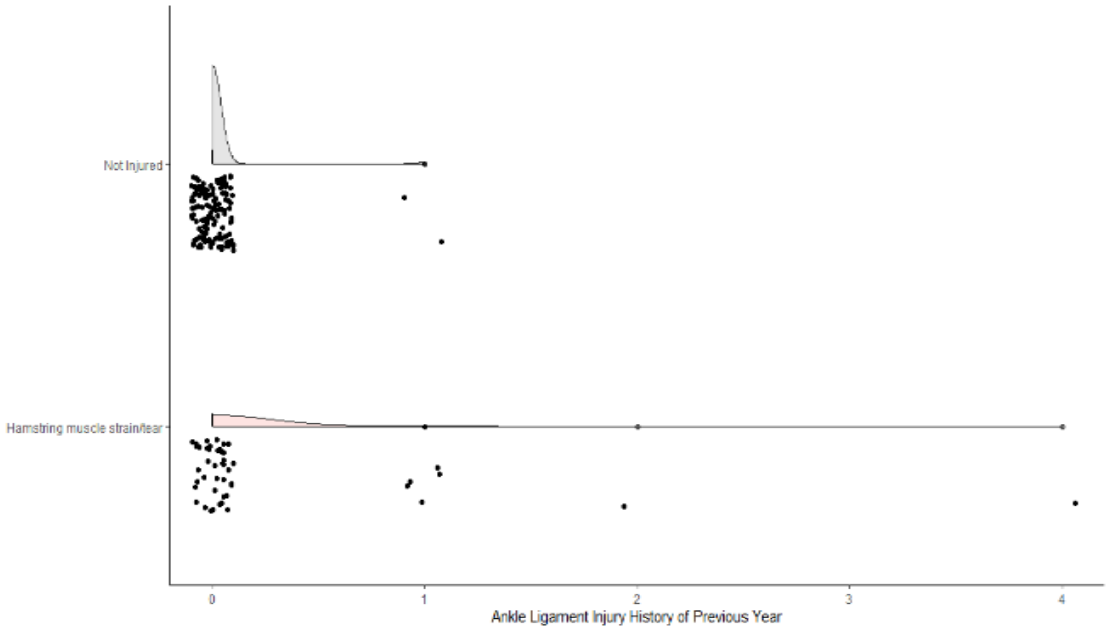


Figure 5.4.1d Distribution of previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained whilst running (shaded in red).

Table 5.4.1c presents results from univariate analyses that examined the risk of sustaining hamstring strain injury during contact events (regardless of muscle type). Sustaining a previous hamstring strain within a previous year, MCL injury or lateral ankle ligament injury were associated with an increased odds of sustaining hamstring strain injury ($P \leq 0.05$, OR 95% CI > 1) (Table 5.4.1c; Figure 5.4.1e & Figure 5.4.1f). However, sustaining a previous ACL injury and sustaining a previous calf muscle strain were not associated with sustaining a subsequent hamstring strain injury ($P > 0.05$) (Table 5.4.1c).

Table 5.4.1c Univariate generalized linear mixed-models examining the association between previous injury history and hamstring strain injury regardless of muscle type sustained during contact events.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previous Hamstring Injury History (per injury)	0.14		0.56	
Previous Years Hamstring Injury History (per injury)	0.02*	4.61 (1.35 - 15.81)	0.74	0.21 (0 - 0.43)
Previous ACL Injury History (per injury)	0.37		0.72	
Previous MCL Injury History (per injury)	0.01*	4.7 (1.55 - 14.2)	0.59	0.21 (0 - 0.43)
Previous Years MCL Injury History (per injury)	0.02*	6.35 (1.26 - 31.94)	0.56	0.14 (0 - 0.33)
Previous Calf Injury History (per injury)	0.60		0.68	
Previous Years Calf Injury History (per injury)	0.68		0.70	
Previous Ankle Ligament Injury History (per injury)	0.02*	6.35 (1.26 - 31.94)	0.74	0.14 (0 - 0.33)
Previous Years Ankle Ligament Injury History (per injury)	0.03*	11.17 (1.44 - 86.49)	0.56	0.14 (0 - 0.33)

*** denotes statistical significance (P ≤ 0.05)**

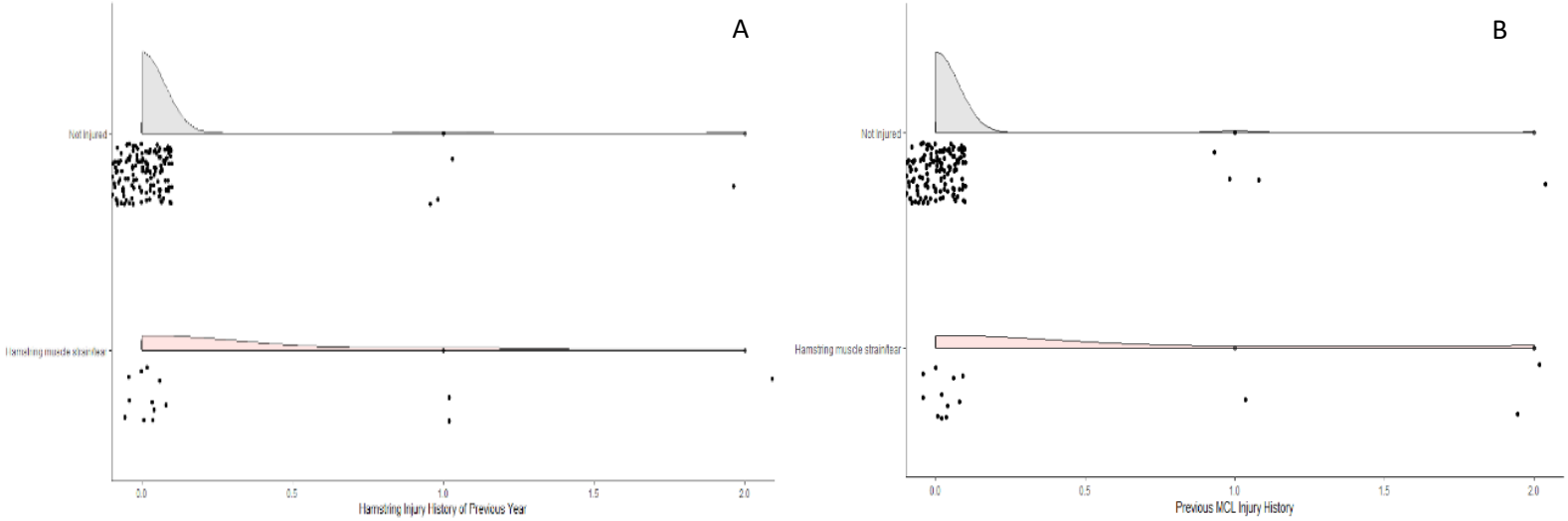


Figure 5.4.1e Distribution of A) previous hamstrings muscle group injury only sustained during the previous year & B) previous MCL injury for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

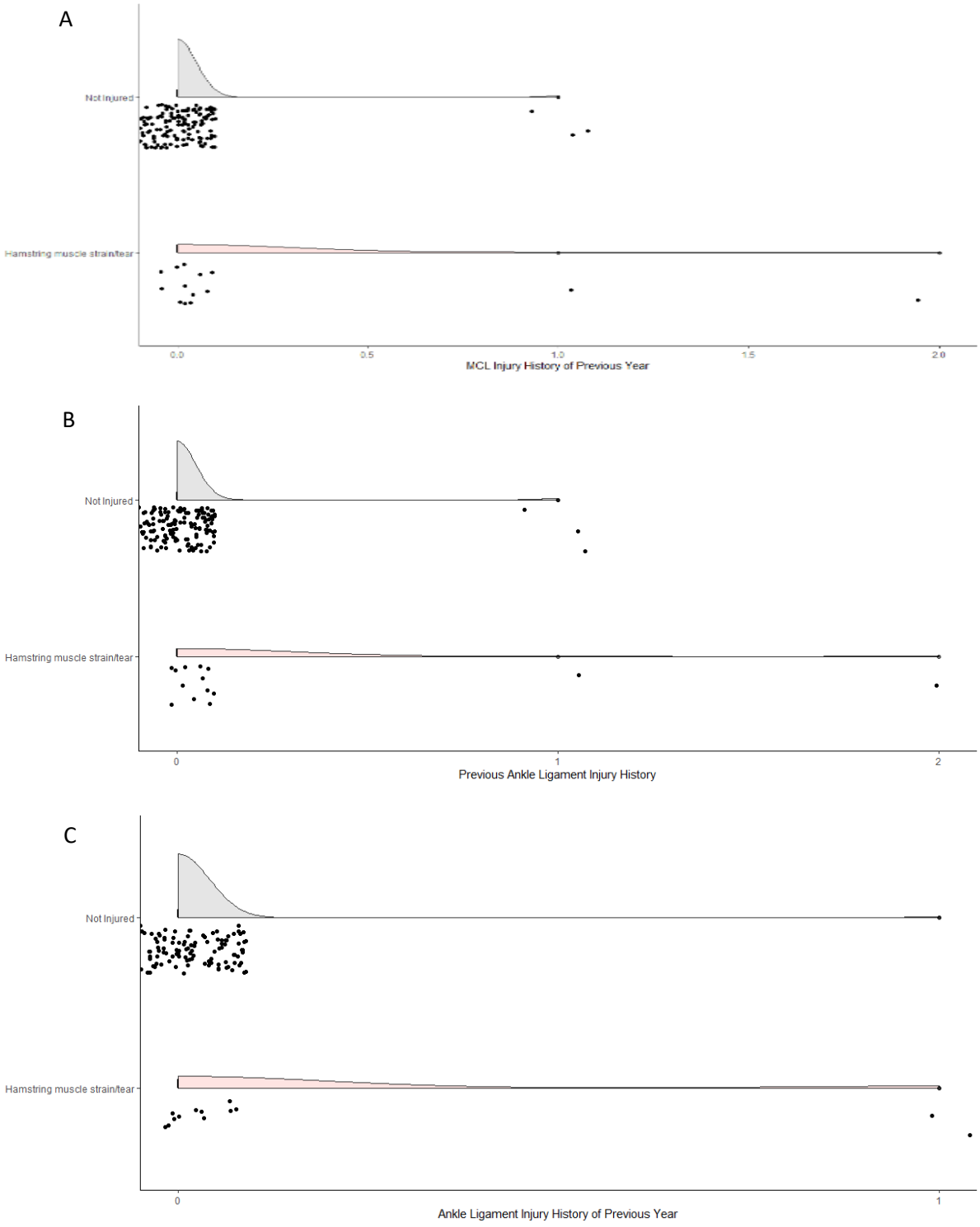


Figure 5.4.1f Distribution of A) previous MCL injury only sustained during the previous year, B) previous ankle ligament injury only sustained during the previous year & C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

Table 5.4.1d presents results from univariate analyses that examined the risk of sustaining biceps femoris strain injury regardless of inciting event. Sustaining a previous MCL injury or lateral ankle ligament injury were associated with an increased odds of sustaining biceps femoris strain injury ($P \leq 0.05$, OR 95% CI > 1) (Table 5.4.1d; Figure 5.4.1g). However, sustaining any previous hamstring muscle strain, a previous ACL injury and a previous calf muscle strain were not associated with sustaining a subsequent biceps femoris strain injury regardless of inciting event ($P > 0.05$) (Table 5.4.1d). Similar findings were also observed for biceps femoris strain injury sustained whilst running (Table 5.4.1e; Figure 5.4.1h) and semimembranosus or semitendinosus strain injury regardless of inciting event (Table 5.4.1f; Figure 5.4.1i). Previous injury to the MCL or lateral ankles were significantly associated with an increased odds of injury ($P \leq 0.05$, OR 95% CI > 1).

Table 5.4.1d Univariate generalized linear mixed-models examining the association between previous injury history and biceps femoris strain injury regardless of inciting event.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previous Hamstring Injury History (per injury)	0.21		0.73	
Previous Years Hamstring Injury History (per injury)	0.46		0.74	
Previous BF Injury History (per injury)	0.22		0.72	
Previous Years BF Injury History (per injury)	0.57		0.71	
Previous SM/ST Injury History (per injury)	0.29		0.75	
Previous Years SM/ST Injury History (per injury)	0.38		0.75	
Previous ACL Injury History (per injury)	0.25		0.72	
Previous MCL Injury History (per injury)	0.02*	4.48 (1.26 - 15.87)	0.73	0.54 (0.4 - 0.68)
Previous Years MCL Injury History (per injury)	0.07		0.71	
Previous Calf Injury History (per injury)	0.33		0.72	
Previous Years Calf Injury History (per injury)	0.47		0.73	
Previous Ankle Ligament Injury History (per injury)	0.0002*	8.47 (1.89 - 37.84)	0.79	0.65 (0.53 - 0.76)
Previous Years Ankle Ligament Injury History (per injury)	0.01*	5.89 (1.06 - 32.82)	0.77	0.62 (0.5 - 0.74)

*** denotes statistical significance (P ≤ 0.05)**

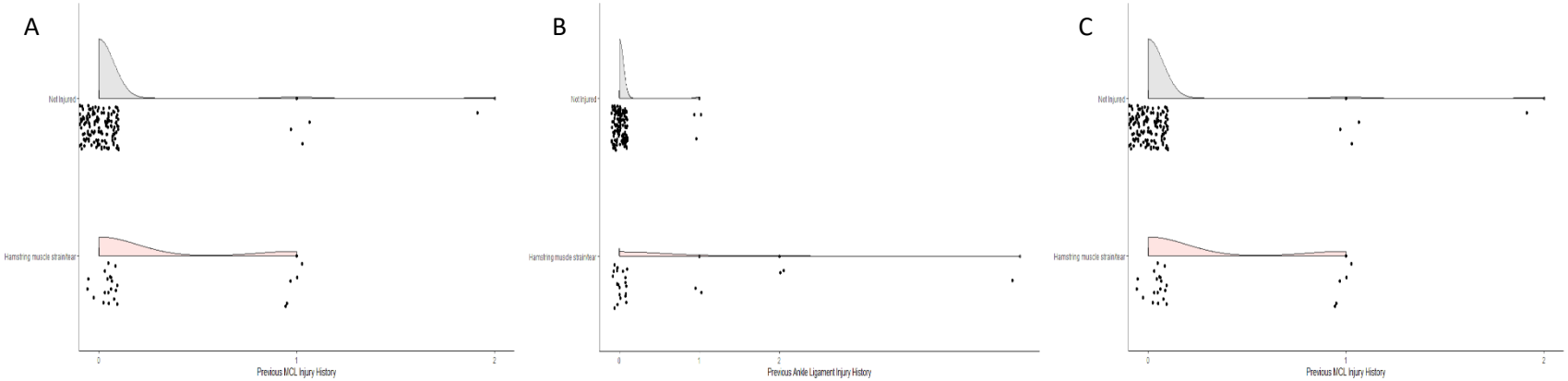


Figure 5.4.1g Distribution of A) previous MCL injury, B) previous ankle ligament injury and C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with biceps femoris strain injury regardless of inciting event (shaded in red).

Table 5.4.1e Univariate generalized linear mixed-models examining the association between previous injury history and biceps femoris strain injury sustained whilst running.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previous Hamstring Injury History (per injury)	0.25		0.75	
Previous Years Hamstring Injury History (per injury)	0.92		0.75	
Previous BF Injury History (per injury)	0.13		0.76	
Previous Years BF Injury History (per injury)	0.60		0.75	
Previous SM/ST Injury History (per injury)	0.51		0.76	
Previous Years SM/ST Injury History (per injury)	0.81		0.75	
Previous ACL Injury History (per injury)	0.31		0.75	
Previous MCL Injury History (per injury)	0.04*	4.45 (1.17 - 16.86)	0.77	0.2 (0.02 - 0.39)
Previous Years MCL Injury History (per injury)	0.19		0.75	
Previous Calf Injury History (per injury)	0.41		0.75	
Previous Years Calf Injury History (per injury)	0.88		0.75	
Previous Ankle Ligament Injury History (per injury)	0.0003*	8.2 (1.86 - 36.26)	0.81	0.65 (0.52 - 0.78)
Previous Years Ankle Ligament Injury History (per injury)	0.01*	4.8 (0.93 - 24.88)	0.79	0.11 (0 - 0.25)

*** denotes statistical significance (P ≤ 0.05)**

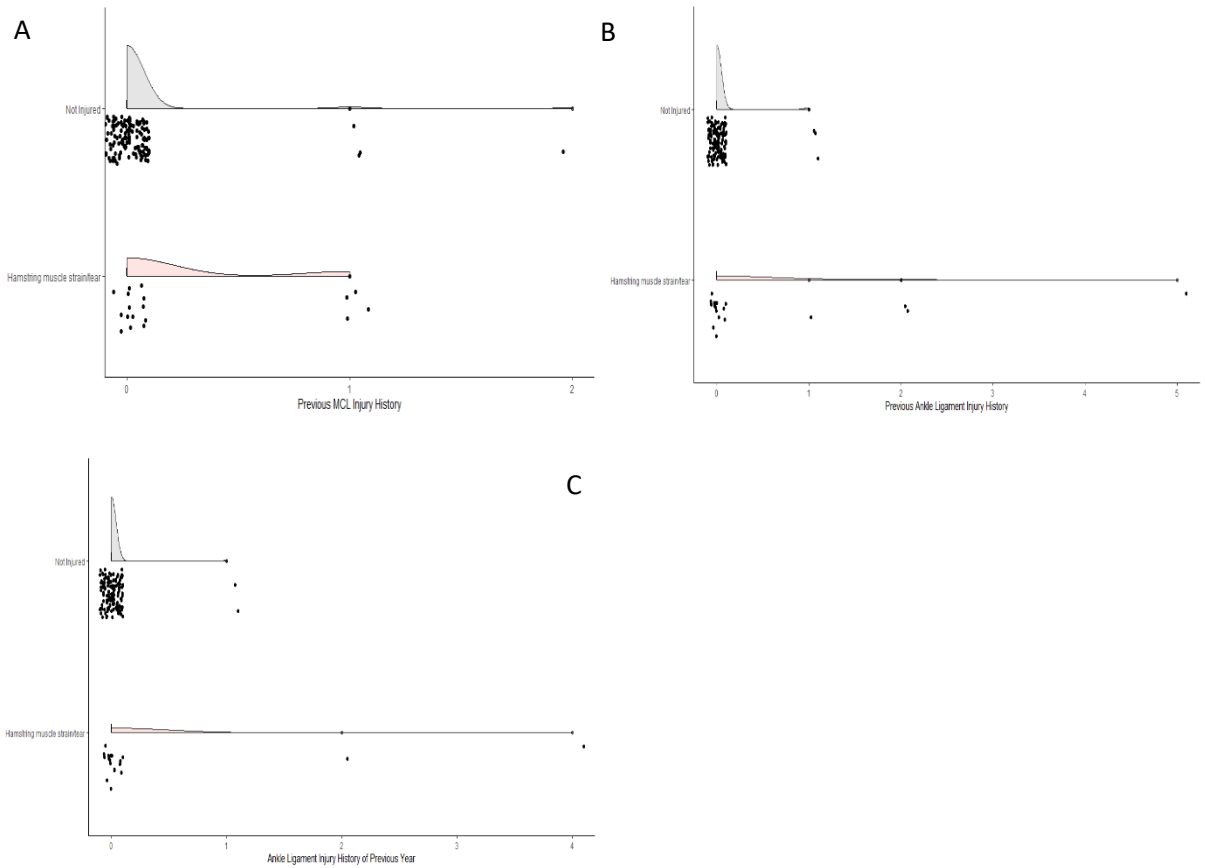


Figure 5.4.1h Distribution of A) previous MCL injury, B) previous ankle ligament injury and C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with biceps femoris strain injury sustained whilst running (shaded in red).

Table 5.4.1f Univariate generalized linear mixed-models examining the association between previous injury history and semimembranosus or semitendinosus strain injury regardless of inciting event.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previous Hamstring Injury History (per injury)	0.12		0.76	
Previous Years Hamstring Injury History (per injury)	0.48		0.75	
Previous BF Injury History (per injury)	0.13		0.75	
Previous Years BF Injury History (per injury)	0.61		0.75	
Previous SM/ST Injury History (per injury)	0.25		0.76	
Previous Years SM/ST Injury History (per injury)	0.40		0.75	
Previous ACL Injury History (per injury)	0.37		0.75	
Previous MCL Injury History (per injury)	0.01*	4.88 (1.51 - 15.8)	0.79	0.23 (0.03 - 0.44)
Previous Years MCL Injury History (per injury)	0.12		0.74	
Previous Calf Injury History (per injury)	0.06		0.75	
Previous Years Calf Injury History (per injury)	0.07		0.74	
Previous Ankle Ligament Injury History (per injury)	0.0001*	12.22 (2.99 - 49.92)	0.82	0.35 (0.12 - 0.58)
Previous Years Ankle Ligament Injury History (per injury)	0.02*	12.16 (1.65 - 89.7)	0.81	0.17 (0 - 0.36)

*** denotes statistical significance (P ≤ 0.05)**

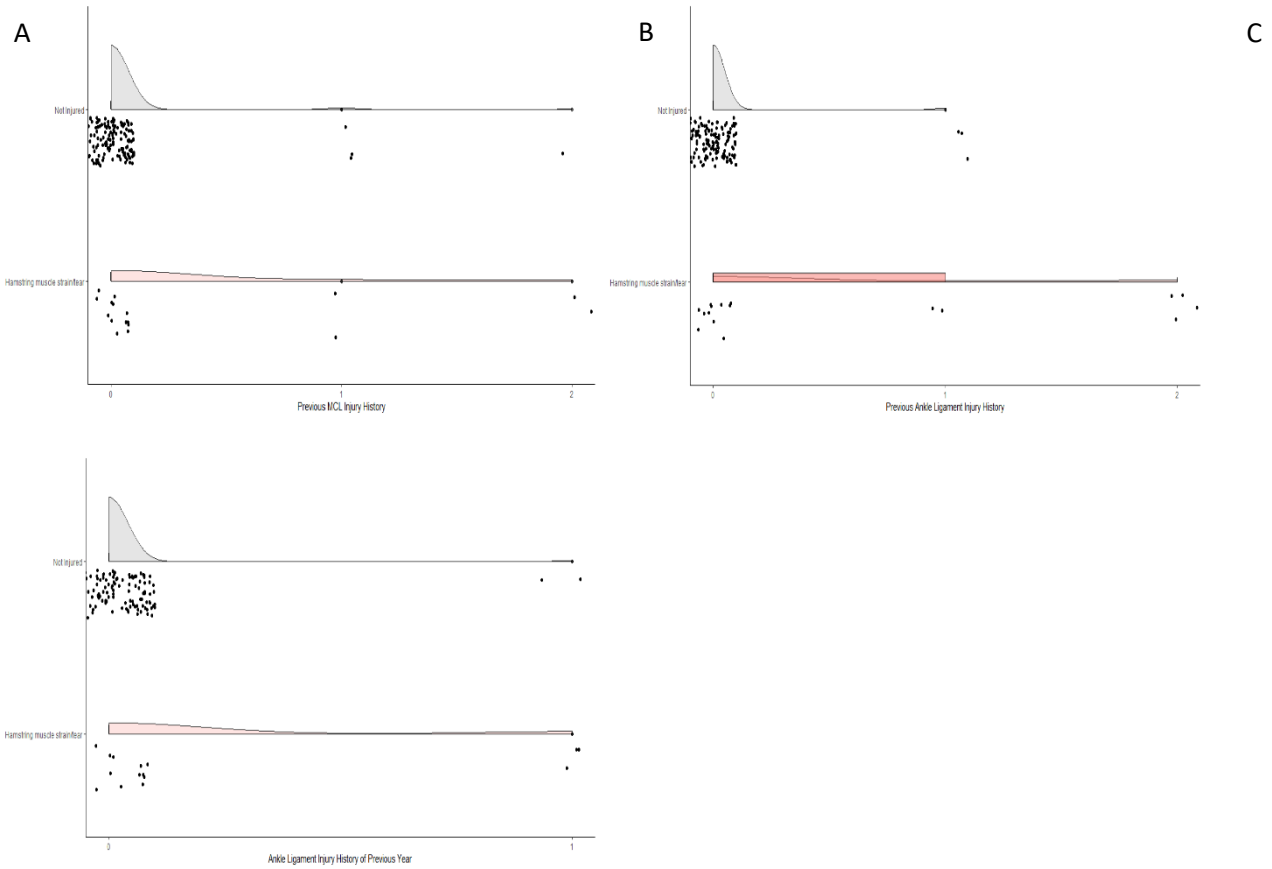


Figure 5.4.1i Distribution of A) previous MCL injury, B) previous ankle ligament injury and C) previous ankle ligament injury only sustained during the previous year for injury free players (shaded in grey) and players with semimembranosus or semitendinosus strain injury regardless of inciting event (shaded in red).

5.4.2 Question 2. Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

Table 5.4.2a presents results from univariate analyses that examined the influence of isokinetic variables in relation to hamstring strain injury regardless of muscle type and inciting event. Peak concentric knee flexion moment relative to body mass at both 60 and 180°/s sec were significantly associated with hamstring strains when all muscles and inciting events were pooled (Table 5.4.2a; Figure 5.4.2a). Moderate and lower magnitudes were associated with a decreased odds of injury (OR 95% CI < 1) whereas higher magnitudes displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), therefore the effect was inconclusive. Peak concentric knee extension moment relative to body mass at 180°/s sec was also significantly associated with hamstring strains when all muscles and inciting events were pooled (Table 5.4.2a; Figure 5.4.2b). Moderate magnitudes were associated with a decreased odds of injury (OR 95% CI < 1), whereas higher magnitudes resulted in increased odds of injury (OR 95% CI > 1).

Table 5.4.2a Univariate generalized linear mixed-models examining the association between isokinetic variables and hamstring strain injury regardless of muscle type and inciting event.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Isokinetic Knee Flexion Moment Variables:				
Peak concentric knee flexion moment at 60°/s - Absolute	0.17		0.96	
Peak concentric knee flexion moment at 60°/s - Relative to body mass (per tertile group):	0.02*		0.97	0.47 (0.36 - 0.57)
Low: 0.87 - 1.32 N·m/kg		*0.21 (0.03 - 0.89)*		
Reference - Medium: 1.33 - 1.57 N·m/kg		*0.3 (0.08 - 0.75)*		
High: 1.58 - 2.09 N·m/kg		*1.58 (0.45 - 6.28)*		
Peak concentric knee flexion moment at 180°/s - Absolute	0.07		0.96	
Peak concentric knee flexion moment at 180°/s - Relative to body mass (per tertile group):	0.04*		0.96	0.45 (0.34 - 0.56)
Low: 0.34 - 0.98 N·m/kg		*0.36 (0.06 - 1.41)*		
Reference - Medium: 0.99 - 1.17 N·m/kg		*0.24 (0.06 - 0.63)*		
High: 1.18 - 1.53 N·m/kg		*2.15 (0.59 - 9.91)*		
Isokinetic Knee Extension Moment Variables:				
Peak concentric knee extension moment at 180°/s - Absolute	0.46		0.96	
Peak concentric knee extension moment at 180°/s - Relative to body mass (per tertile group):	0.004*		0.98	0.51 (0.4 - 0.62)
Low: 0.94 - 1.73 N·m/kg		*0.4 (0.05 - 1.77)*		
Reference - Medium: 1.74 - 1.95 N·m/kg		*0.17 (0.03 - 0.49)*		
High: 1.96 - 2.42 N·m/kg		*4.07 (1.12 - 24.32)*		

*** denotes statistical significance (P ≤ 0.05)**

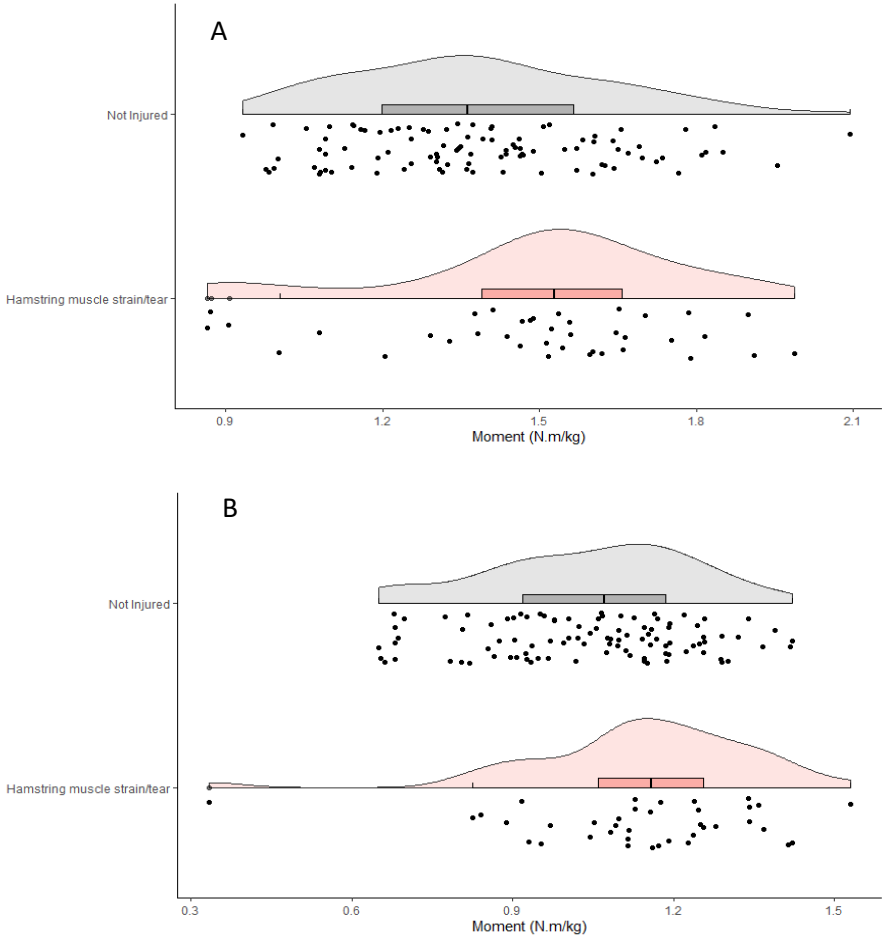


Figure 5.4.2a Distribution of Peak concentric knee flexion moment relative to body mass at A) 60°/s sec & B) 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red).

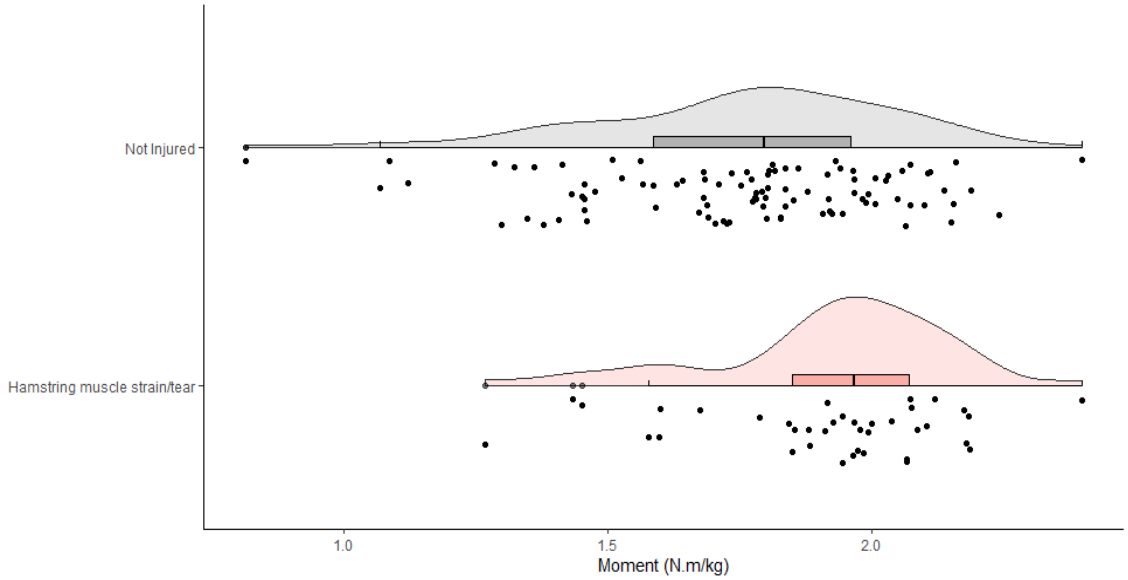


Figure 5.4.2b Distribution of Peak concentric knee extension moment relative to body mass at 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type and inciting event (shaded in red).

Table 5.4.2b presents results from univariate analyses that examined the influence of isokinetic variables in relation to hamstring strain injury regardless of muscle type sustained whilst running. Peak concentric knee flexion moment relative to body mass at both 60 was significantly associated with hamstring strains sustained whilst running when all muscles were pooled (Table 5.4.2b; Figure 5.4.2b). Moderate and lower magnitudes were associated with a decreased odds of injury (OR 95% CI < 1) whereas both higher and lower magnitudes displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), and were therefore inconclusive. Peak concentric knee extension moment relative to body mass at 180°/s sec was significantly associated with hamstring strains sustained whilst running when all muscles were pooled (Table 5.4.2c; Figure 5.4.2b). Moderate magnitudes were associated with a decreased odds of injury (OR 95% CI < 1), whereas higher magnitudes resulted in increased odds of injury (OR 95% CI > 1).

Table 5.4.2b Univariate generalized linear mixed-models examining the association between isokinetic risk factors and hamstring injury regardless of muscle type sustained during running.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Isokinetic Knee Flexion Moment Variables:				
Peak concentric knee flexion moment at 60°/s - Absolute	0.15		0.99	
Peak concentric knee flexion moment at 60°/s - Relative to body mass (per tertile group):	0.03*		0.98	0.45 (0.32 - 0.58)
Low: 0.87 - 1.30 N·m/kg		*0.12 (0 - 3.09)*		
Reference - Medium: 1.31 - 1.51 N·m/kg		*0.04 (0 - 0.59)*		
High: 1.52 - 2.09 N·m/kg		*3.53 (0.39 - 32.12)*		
Peak concentric knee flexion moment at 180°/s - Absolute	0.56		0.98	
Peak concentric knee flexion moment at 180°/s - Relative to body mass	0.08		0.98	
Isokinetic Knee Extension Moment Variables:				
Peak concentric knee extension moment at 180°/s - Absolute	0.66		0.98	
Peak concentric knee extension moment at 180°/s - Relative to body mass (per tertile group):	0.001*		0.99	0.57 (0.44 - 0.7)
Low: 0.81 - 1.73 N·m/kg		*1.5 (0.18 - 12.25)*		
Reference - Medium: 1.74 - 1.92 N·m/kg		*0.02 (0 - 0.28)*		
High: 1.93 - 2.40 N·m/kg		*17.24 (1.75 - 170.2)*		

*** denotes statistical significance (P ≤ 0.05)**

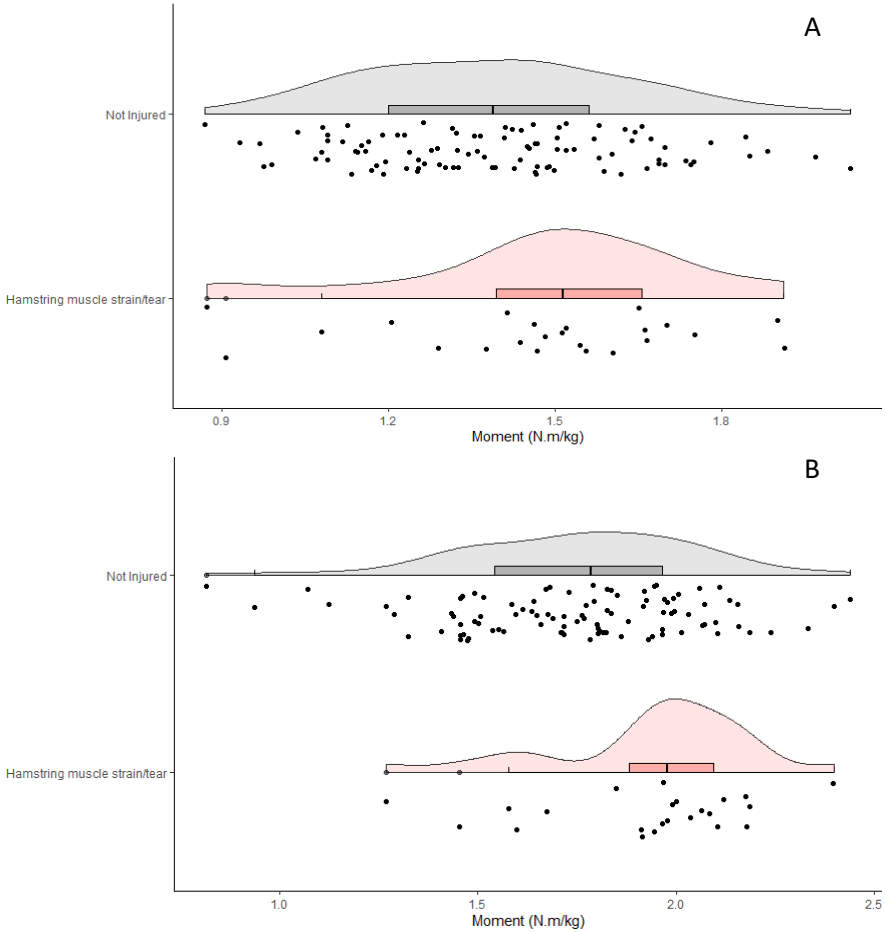


Figure 5.4.2c Distribution of Peak concentric A) knee flexion moment relative to body mass at 60°/s sec & B) knee extension moment relative to body mass at 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained whilst running (shaded in red).

Table 5.4.2c presents results from univariate analyses that examined the influence of isokinetic variables in relation to hamstring strain injury regardless of muscle type sustained during contact events. Peak concentric flexion moment at 60°/s was significantly related to injury regardless of absolute or relative scaling (Table 5.4.2c; Figure 5.4.2d). For both variables moderate magnitudes were associated with a decreased odds of injury (OR 95% CI < 1). Higher magnitudes displayed a trend towards an increased odds of injury (Table 5.4.2c), however, odds ratios for both higher and lower magnitudes were inconclusive (lower 95% CI < 1 & upper 95% CI > 1). Peak absolute concentric flexion moment at 180°/s was also significantly related to contact-based hamstring strains (Table 5.4.2c; Figure 5.4.2e). Moderate magnitudes were associated with decreased odds (OR 95% CI < 1), whereas higher magnitudes were associated with increased odds of sustaining hamstring injury (OR 95% CI > 1). Lower magnitudes were observed to be inconclusive (lower 95% CI < 1 & upper 95% CI > 1). Peak relative concentric extension moment at 180°/s was significantly related to contact-based hamstring strains. Moderate magnitudes were associated with decreased odds (OR 95% CI < 1), whereas both higher and lower magnitudes were observed to be inconclusive (lower 95% CI < 1 & upper 95% CI > 1).

Table 5.4.2c Univariate generalized linear mixed-models examining the association between isokinetic variables and hamstring injury regardless of muscle type sustained during contact events.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Isokinetic Knee Flexion Moment Variables:				
Peak concentric knee flexion moment at 60°/s - Absolute (per tertile group):	0.02*		0.97	0.57 (0.41 - 0.72)
Low: 74.3 - 130 N·m		*0.29 (0.03 - 3.28)*		
Reference - Medium: 130 - 154.8 N·m		*0.08 (0.02 - 0.38)*		
High: 154.9 - 237 N·m		*3.34 (0.74 - 15)*		
Peak concentric knee flexion moment at 60°/s - Relative to body mass (per tertile group):	0.02*		0.90	0.57 (0.41 - 0.72)
Low: 0.87 - 1.25 N·m/kg		*0.32 (0.03 - 3.28)*		
Reference - Medium: 1.26 - 1.51 N·m/kg		*0.08 (0.01 - 0.51)*		
High: 1.52 - 2.09 N·m/kg		*3.38 (0.71 - 16.04)*		
Peak concentric knee flexion moment at 180°/s - Absolute (per tertile group):	0.02*		0.97	0.57 (0.41 - 0.72)
Low: 38.3 - 98.2 N·m		*0.31 (0 - 2.86)*		
Reference - Medium: 98.3 - 115.3 N·m		*0.05 (0 - 0.19)*		
High: 115.4 -154.8 N·m		*4.82 (1.03 - 18.34)*		
Peak concentric knee flexion moment at 180°/s - Relative to body mass	0.23		0.99	
Isokinetic Knee Extension Moment Variables:				
Peak concentric knee extension moment at 180°/s - Absolute	0.12		0.92	
Peak concentric knee extension moment at 180°/s - Relative to body mass (per tertile group):	0.04*		0.97	0.48 (0.27 - 0.68)
Low: 0.66 - 0.95 N·m/kg		*1.08 (0.11 - 10.23)*		
Reference - Medium: 0.96 - 1.15 N·m/kg		*0.03 (0 - 0.41)*		
High: 1.16 - 1.42 N·m/kg		*7.06 (0.79 - 63.42)*		

*** denotes statistical significance (P ≤ 0.05)**

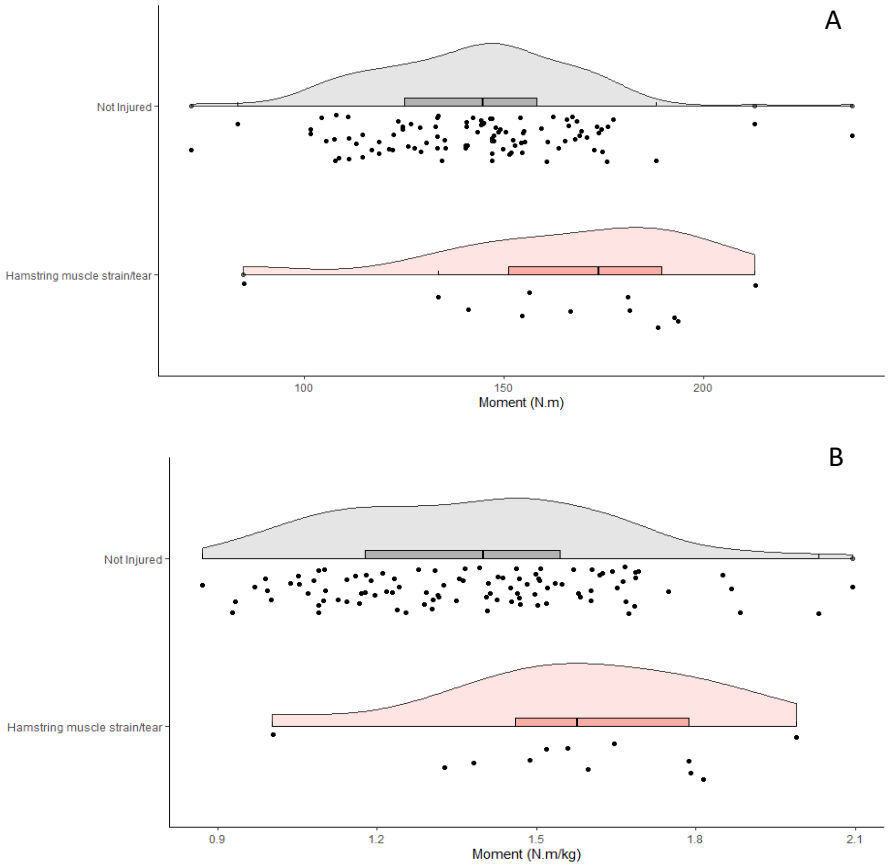


Figure 5.4.2d Distribution of Peak A) absolute concentric knee flexion moment at 60°/s sec & B) concentric knee flexion moment relative to body mass at 60°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

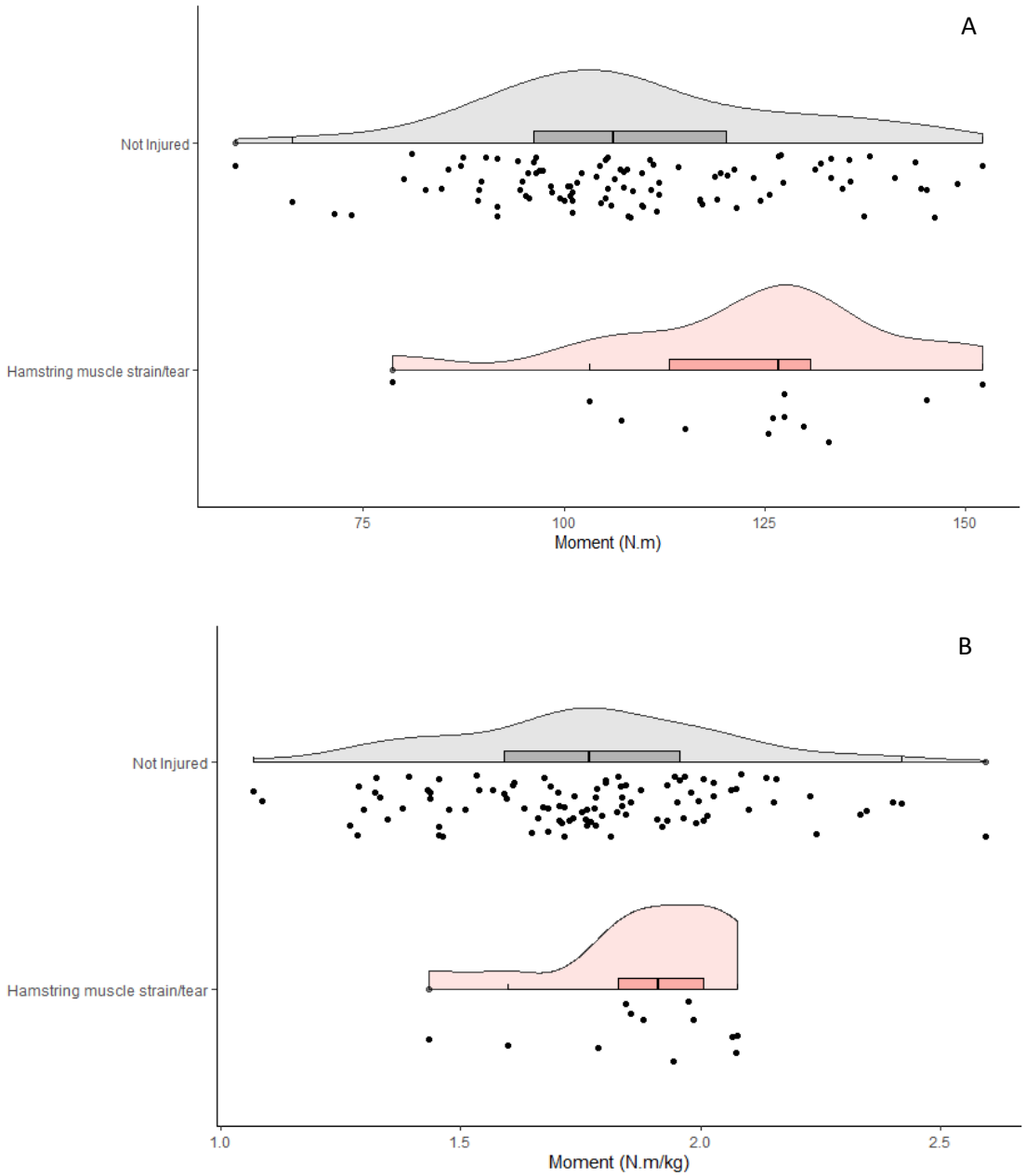


Figure 5.4.2e Distribution of Peak A) absolute concentric knee flexion moment at 180°/s sec & B) concentric knee extension moment relative to body mass at 180°/s sec for injury free players (shaded in grey) and players with hamstring strain injury regardless of muscle type sustained during contact events (shaded in red).

Table 5.4.2d presents results from univariate analyses which examined the risk of sustaining biceps femoris strain in relation to isokinetic variables. No Isokinetic variable was associated with biceps femoris strain risk when all inciting events were pooled or when running inciting events were analysed in isolation (Table 5.4.2d).

Table 5.4.2d Univariate generalized linear mixed-models examining the association between isokinetic variables and biceps femoris injury regardless of inciting event.

Independent Variable	P value	OR (95% CI)	Apparent AUC
Biceps femoris strains sustained during all inciting events:			
Isokinetic Knee Flexion Moment Variables:			
Peak concentric knee flexion moment at 60°/s - Absolute	0.20		0.90
Peak concentric knee flexion moment at 60°/s - Relative to body mass	0.24		0.76
Peak concentric knee flexion moment at 180°/s - Absolute	0.77		0.89
Peak concentric knee flexion moment at 180°/s - Relative to body mass	0.52		0.81
Isokinetic Knee Extension Moment Variables:			
Peak concentric knee extension moment at 180°/s - Absolute	0.52		0.81
Peak concentric knee extension moment at 180°/s - Relative to body mass	0.09		0.63
Biceps femoris strains sustained during running:			
Isokinetic Knee Flexion Moment Variables:			
Peak concentric knee flexion moment at 60°/s - Absolute	0.46		0.99
Peak concentric knee flexion moment at 60°/s - Relative to body mass	0.28		0.98
Peak concentric knee flexion moment at 180°/s - Absolute	0.47		0.99
Peak concentric knee flexion moment at 180°/s - Relative to body mass	0.13		0.99
Isokinetic Knee Extension Moment Variables:			
Peak concentric knee extension moment at 180°/s - Absolute	0.74		0.99
Peak concentric knee extension moment at 180°/s - Relative to body mass	0.18		0.97

*** denotes statistical significance (P ≤ 0.05)**

Isokinetic strength in relation to semimembranosus and semitendinosus strain risk

Table 5.4.2e presents results from univariate analyses which examined the risk of sustaining semimembranosus and semitendinosus strains in relation to isokinetic variables. Peak concentric flexion moment at 60°/s was significantly related to hamstring strains sustained during contact events regardless of absolute or relative scaling (Table 5.4.2e; Figure 5.4.2f). Moderate magnitudes were associated with a decreased odds of injury (OR 95% CI < 1), whereas higher magnitudes were associated with increased odds of sustaining hamstring injury (OR 95% CI > 1). Furthermore, lower magnitudes were observed to be inconclusive (lower 95% CI < 1 & upper 95% CI > 1) (Table 5.4.2e).

Table 5.4.2e Univariate generalized linear mixed-models examining the association between isokinetic variables and semimembranosus and semitendinosus injury regardless of inciting events.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Isokinetic Knee Flexion Moment Variables:				
Peak concentric knee flexion moment at 60°/s - Absolute	0.08		0.99	
Peak concentric knee flexion moment at 60°/s - Relative to body mass (per tertile group):	0.05*		0.99	0.47 (0.31 - 0.63)
Low: 0.87 - 1.29 N·m/kg		*1 (0.17 - 5.73)*		
Reference - Medium: 1.30 - 1.51 N·m/kg		*0.09 (0.02 - 0.24)*		
High: 1.52 - 2.03 N·m/kg		*3.75 (1.01 - 18.11)*		
Peak concentric knee flexion moment at 180°/s - Absolute	0.77		0.99	
Peak concentric knee flexion moment at 180°/s - Relative to body mass	0.19		0.99	
Isokinetic Knee Extension Moment Variables:				
Peak concentric knee extension moment at 180°/s - Absolute	0.46		0.99	
Peak concentric knee extension moment at 180°/s - Relative to body mass	0.19		0.99	

*** denotes statistical significance (P ≤ 0.05)**

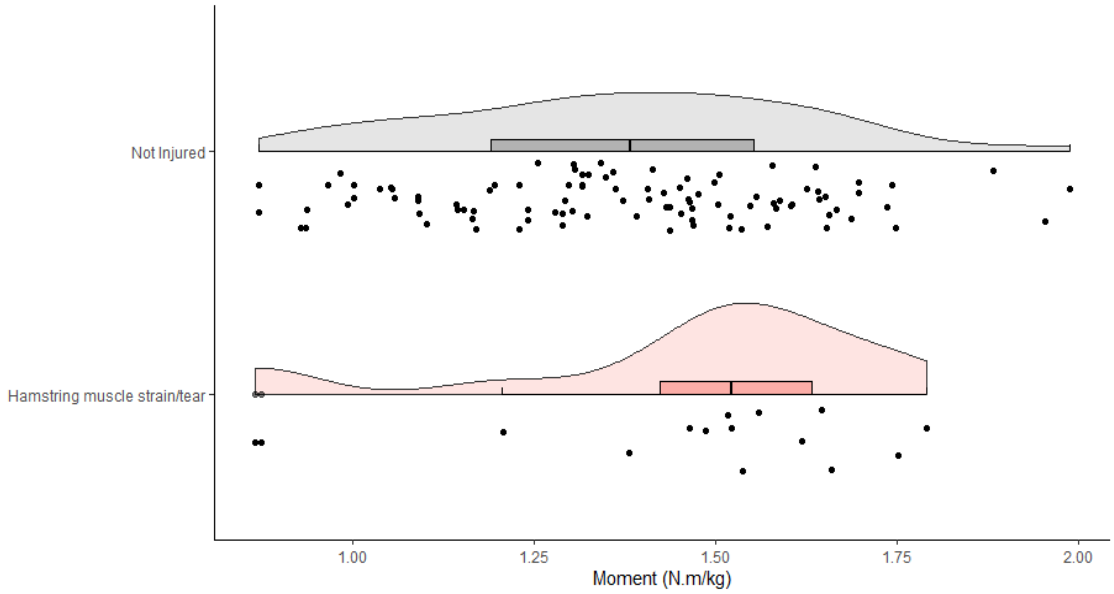


Table 5.4.2f Distribution of peak concentric knee flexion moment relative to body mass at 60°/s sec for injury free players (shaded in grey) and players with semimembranosus and semitendinosus strain injury regardless of inciting event (shaded in red).

5.4.3 Question 3. Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players?

Acute exposure to high-speed running in relation to hamstring strain risk – only sustained while running, regardless of muscle type

Table 5.4.3a presents univariate analyses on the effect of acute high-speed running exposure had in relation to hamstring strain risk where the inciting event was running.

Table 5.4.3a Univariate generalized linear mixed-models examining the association between acute exposure to high-speed running and hamstring injury regardless of muscle type sustained during running.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Acute High Speed Running Variables:				
High-Speed Running Distance ≥70% Max Velocity - 3 Day EWMA (per tertile group):	0.02*		0.94	0.54 (0.44 - 0.65)
Low: 0 - 18.3 m		*0.19 (0 - 0.98)*		
Reference - Medium: 18.4 - 58 m		*0 (0 - 0.01)*		
High: 58.1 - 408.2 m		*2.13 (0.75 - 7.99)*		
High-Speed Running Distance ≥70% Max Velocity - 7 Day EWMA (per tertile group):	0.04*		0.94	0.53 (0.42 - 0.63)
Low: 0 - 17.2		*0.15 (0 - 0.71)*		
Reference - Medium: 17.3 - 50.1 m		*0 (0 - 0.01)*		
High: 50.2 - 278.1		*1.53 (0.5 - 4.96)*		
High-Speed Running Distance ≥70% Max Velocity - 7 Day Sum (per tertile group):	0.002*		0.95	0.55 (0.46 - 0.63)
Low: 0 - 102 m		*0 (0 - 0)*		
Reference - Medium: 102.1 - 315.9 m		*0 (0 - 0.01)*		
High: 316 - 1714 m		*1.3 (0.44 - 4.64)*		

*** denotes statistical significance (P ≤ 0.05)**

All variables were significantly associated with hamstring strains (P ≤ 0.05). Lower and moderate magnitudes of acute exposure to high-speed running were associated with a decreased odds of injury for all variables a decreased odds of injury (OR 95% CI < 1). High exposure to high-speed running exhibited a trend towards increased hamstring strain risk, however all variables displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), therefore the true effect was inconclusive (Table 5.4.3a; Figure 5.4.3a).

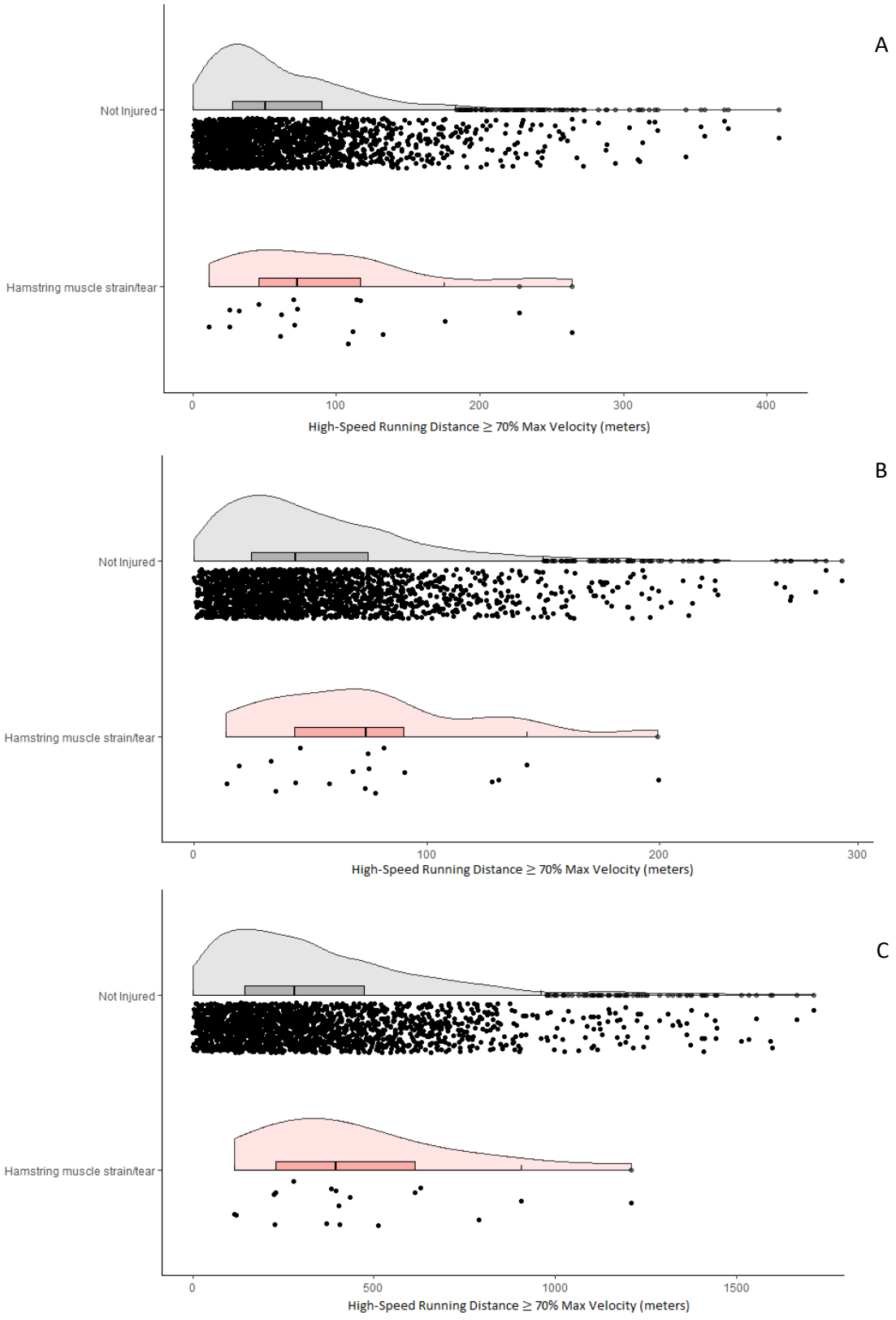


Figure 5.4.3a Distribution of Acute High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 3 Day EWMA, B) 7 Day EWMA and C) 7 Day Sum, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

Chronic exposure to high-speed running in relation to hamstring strain risk – only sustained while running, regardless of muscle type

Univariate models of chronic exposure to high-speed running in relation to the risk of sustaining a hamstring strain whilst running are displayed in table 5.4.3b.

Table 5.4.3b Univariate generalized linear mixed-models examining the association between chronic exposure to high-speed running and hamstring injury regardless of muscle type sustained during running.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Chronic High Speed Running Variables:				
High-Speed Running Distance ≥70% Max Velocity - 7 Day EWMA - 3 Day Lag	0.06		0.93	
High-Speed Running Distance ≥70% Max Velocity - 7 Day EWMA - 7 Day Lag (per tertile group):	0.02*		0.94	0.65 (0.52 - 0.78)
Low: 0 - 8.94 m		*0.19 (0.02 - 1.74)*		
Reference - Medium: 8.95 - 36.3 m		*0 (0 - 0)*		
High: 36.4 - 278.09 m		*2.26 (0.71 - 7.17)*		
High-Speed Running Distance ≥70% Max Velocity - 7 Day Sum - 7 Day Lag (per tertile group):	0.02*		0.95	0.54 (0.44 - 0.65)
Low: 0 - 60 m		*0.19 (0 - 1.01)*		
Reference - Medium: 61 - 246 m		*0 (0 - 0.01)*		
High: 247 - 1600 m		*2.22 (0.88 - 9.01)*		
High-Speed Running Distance ≥70% Max Velocity - 14 Day EWMA (per tertile group):	0.04*		0.95	0.53 (0.42 - 0.63)
Low: 0 - 15.7 m		*0.15 (0.01 - 0.96)*		
Reference - Medium: 15.8 - 44.9 m		*0 (0 - 0.01)*		
High: 45 - 219.4 m		*1.49 (0.47 - 4.98)*		
High-Speed Running Distance ≥70% Max Velocity - 14 Day EWMA - 3 Day Lag (per tertile group):	0.02*		0.95	0.55 (0.44 - 0.65)
Low: 0 - 11.6 m		*0.18 (0 - 1)*		
Reference - Medium: 11.7 - 38.2 m		*0 (0 - 0.01)*		
High: 38.3 - 216 m		*2.18 (0.79 - 9.36)*		
High-Speed Running Distance ≥70% Max Velocity - 14 Day EWMA - 7 Day Lag (per tertile group):	0.01*		0.94	0.65 (0.5 - 0.79)
Low: 0 - 11.1 m		*0.24 (0.01 - 1.77)*		
Reference - Medium: 11.2 - 36.3 m		*0 (0 - 0.01)*		
High: 36.4 - 209.5 m		*3.4 (1.03 - 14.87)*		
High-Speed Running Distance ≥70% Max Velocity - 21 Day EWMA (per tertile group):	0.02*		0.95	0.55 (0.45 - 0.65)
Low: 0 - 15.1 m		*0.17 (0.01 - 1.2)*		
Reference - Medium: 15.2 - 43.0 m		*0 (0 - 0.01)*		
High: 43.1 - 204.7 m		*2.08 (0.63 - 7.56)*		
High-Speed Running Distance ≥70% Max Velocity - 21 Day EWMA - 3 Day Lag (per tertile group):	0.02*		0.95	0.55 (0.44 - 0.65)
Low: 0 - 12.4 m		*0.18 (0.02 - 1.64)*		
Reference - Medium: 12.5 - 37.8 m		*0 (0 - 0.01)*		
High: 37.9 - 204.7 m		*2.14 (0.64 - 7.14)*		
High-Speed Running Distance ≥70% Max Velocity - 21 Day EWMA - 7 Day Lag (per tertile group):	0.01*		0.95	0.65 (0.5 - 0.79)
Low: 0 - 11.6 m		*0.23 (0.01 - 1.72)*		
Reference - Medium: 11.7 - 36.2 m		*0 (0 - 0.01)*		
High: 36.3 - 204.7 m		*3.31 (1.14 - 16.8)*		

*** denotes statistical significance (P ≤ 0.05)**

All variables with the exception of 7-day EWMA with a 3-day lag (P = 0.06), were significantly associated with hamstring strain (Table 5.4.3b; Figures 5.4.3b-d). Although low magnitudes of chronic high-speed running exposure variables displayed a trend toward lower odds of injury, the only variable that presented a decreased odds of injury were high-speed running exposure 14-day EWMA (OR 95% CI < 1). In a similar manner, high magnitudes of chronic exposure to high-speed running displayed a trend towards an increased odds of hamstring strain, however only

high-speed running exposure 14-day and 21-day EMWA variables when calculated with a 7-day lag displayed increased odds of hamstring strain (OR 95% CI > 1).

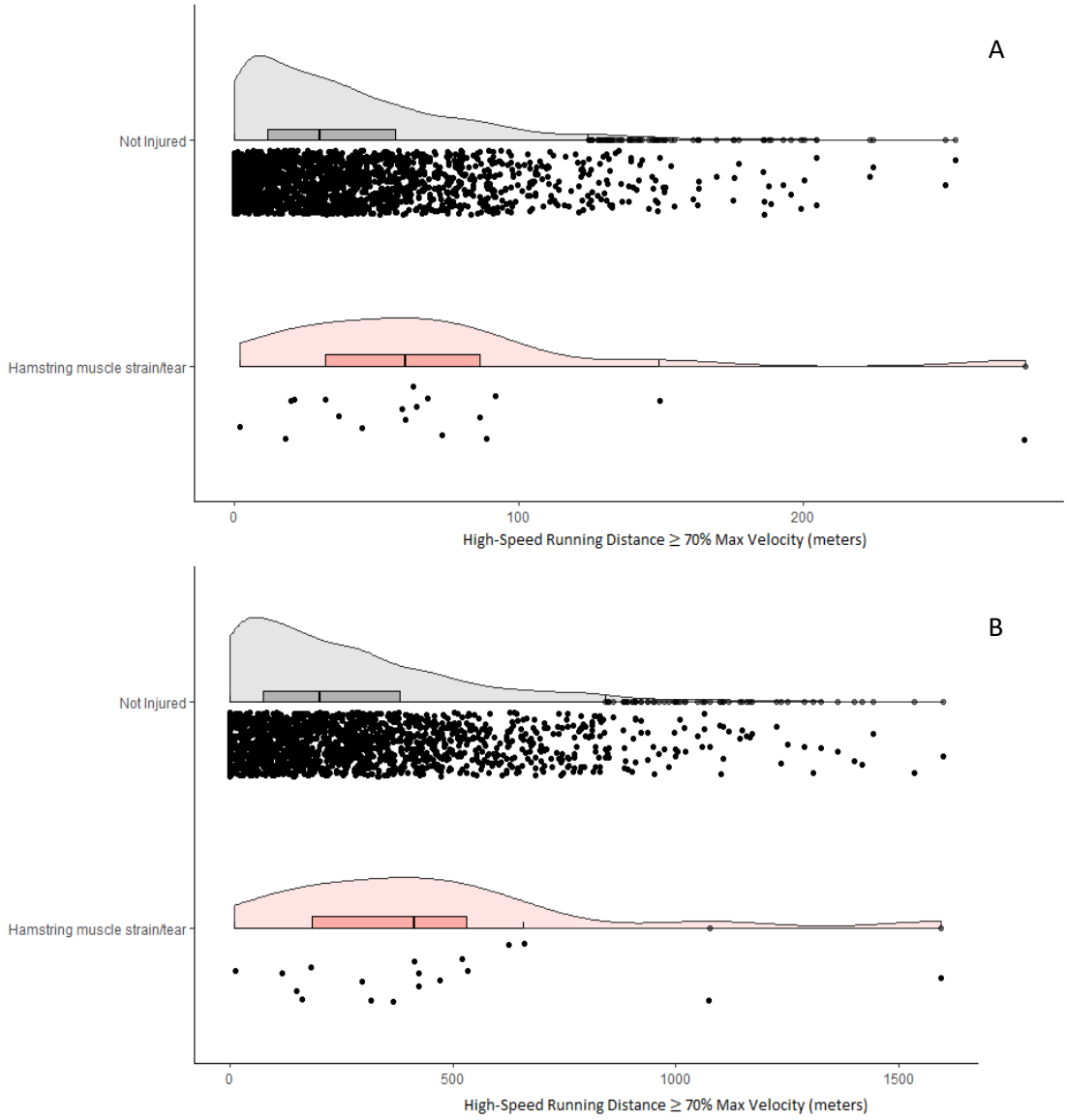


Figure 5.4.3b Distribution of Chronic High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 7 day EWMA with a 3 day lag, B) 7 Day Sum with a 7 day lag, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

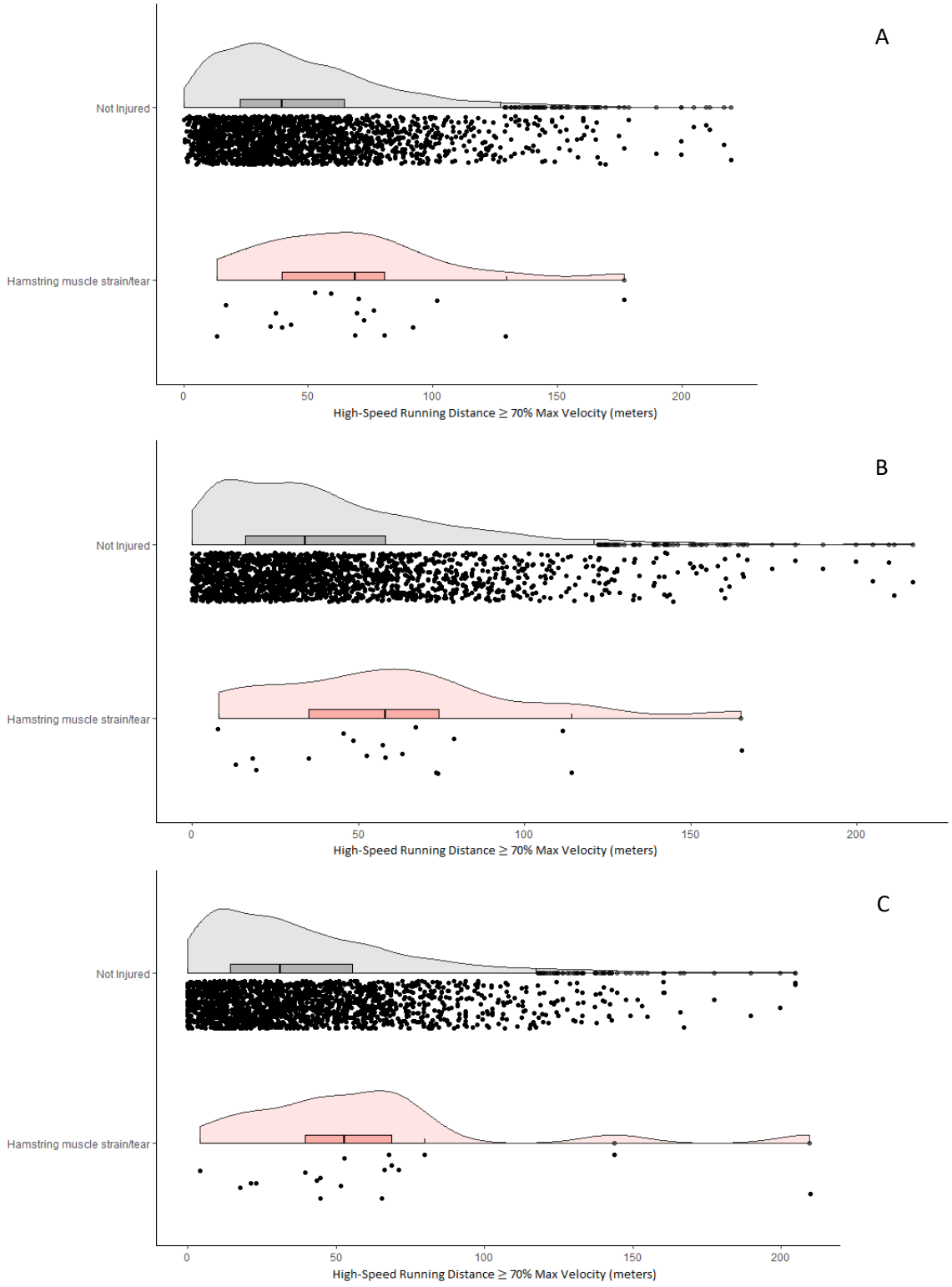


Figure 5.4.3c Distribution of Chronic High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 14 day EWMA, B) 14 day EWMA with a 3 day lag and C) 14 Day Sum with a 7 day lag, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

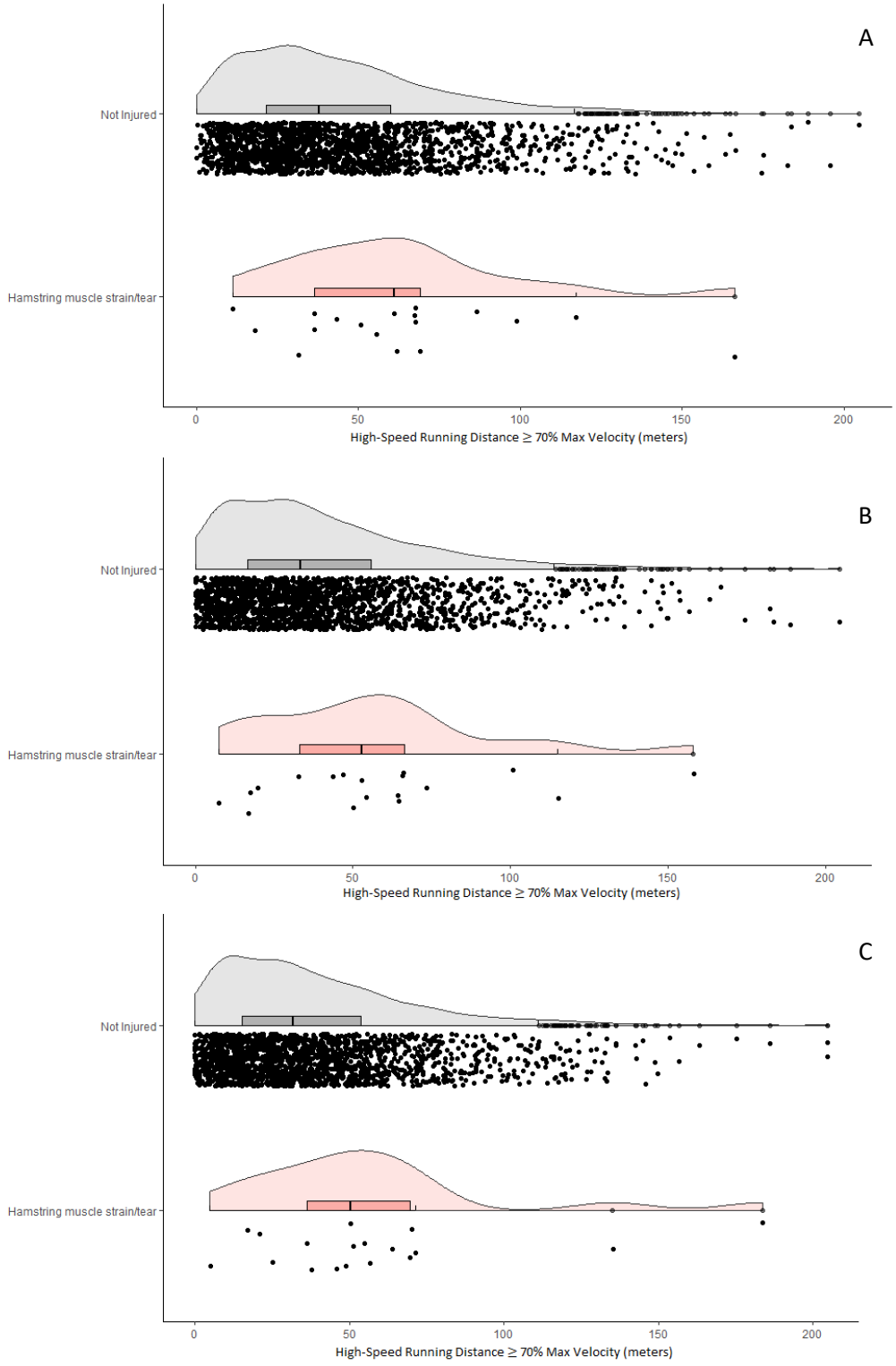


Figure 5.4.3d Distribution of Chronic High-Speed Running Distance ≥ 70% Max Velocity calculated as: A) 21 day EWMA, B) 21 day EWMA with a 3 day lag and C) 21 Day Sum with a 7 day lag, for injury free player-days (shaded in grey) and hamstring strain injuries regardless of muscle type sustained when running (Shaded in red).

5.5 Discussion

Hamstring strain injury present a high burden to male professional rugby union teams (Brooks et al., 2005a; Brooks et al., 2005b; Brooks et al., 2006; Kemp et al., 2018; Kemp et al., 2019). The purpose of this chapter was to examine the aetiology of hamstring strain injury in a male professional rugby union player population. Previously identified variables associated with hamstring strain were selected for were examined for analysis in the following research questions. The first research question examined the existence of a relationship between previous injury history and the odds of sustaining hamstring strains. When all hamstring muscles were pooled, previous injury to the hamstrings were observed increase the odds of subsequent hamstring strain. Previous injury to the MCL and lateral ankle ligaments were both associated with an increase in the odds of sustaining a subsequent biceps femoris injury or medial hamstring strain. The second research question examined the existence of a relationship between isokinetic hamstring and quadriceps strength variables and hamstring strains. Increased magnitudes of isokinetic strength variables were associated with increased odds of both pooled hamstring strains regardless of inciting events as well as medial hamstring muscle strains. Whereas biceps femoris strains were not associated with any isokinetic strength variables. The final research question examined the effect of acute and chronic exposure to high-speed running on the odds of sustaining hamstring strains during running. The influence of acute exposure to high-speed running was inconclusive, whereas larger magnitudes of chronic high-speed running exposure was associated with an increased odds of hamstring injury.

5.5.1 Question 1. Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

The first research question examined whether previous lower limb injuries were associated with hamstring strains. It was hypothesised that sustaining a previous hamstring strain injury would increase the risk of sustaining a subsequent hamstring strain. The findings of a recent meta-analysis by Green et al. (2020) formed the rationale for this hypothesis. In the current study,

sustaining previous hamstring injuries were observed to increase the odds of subsequent hamstring injury when all hamstring injuries were pooled (Table 5.4.1a). However, the hypothesis was not supported in relation to the risk of sustaining subsequent strains to specific hamstring muscles (Tables 5.4.1d - 5.4.1f), or when the inciting event was running (Table 5.4.1b). Green and colleagues also highlighted that hamstrings were at a higher risk of re-injury within a year following the original injury (Green et al., 2020), this was only observed for pooled hamstring injuries sustained during contact events (Table 5.4.1c). The manner in which hamstring injuries are rehabilitated by the medical staff working for the rugby team participating in the project offers a contributing factor for these divergent findings compared to Green et al. (2020). Previous injury history has long been considered a risk factor in team sports (Orchard et al., 1997; Bennell et al., 1998; Gabbe et al., 2006; Hägglund et al., 2006; Henderson et al., 2010), and although there was a paucity of aetiological research within rugby union prior to the start of the study, best practice recommendations for rehabilitation highlight the high rate of hamstring strain re-injury (Heiderscheit et al., 2010). However, this may also help to explain the increased risk sustaining a recent previous hamstring strain had on subsequent hamstring injury sustained during a contact event. The previously cited research in the early 2000s focussed on soccer and Australian rules football; the former being non-contact and the latter being classified as full-contact but not involving contact events where the hip is in a flexed position and the knee is in an extended position such as rucking in rugby union. Early rehabilitation strategies had a 'return to running' focus, preparing athletes for the physical demands of multidirectional sprinting and decelerating of team sports (Heiderscheit et al., 2010). However, a return to rugby approach is also needed to ensure the individual can withstand the rigours of rugby union specific movements following a period of cessation (Stokes et al., 2020).

It was also hypothesised that sustaining a previous injury to another lower limb location would be associated with an increased risk of hamstring injury. Again, the rationale behind this hypothesis was due to the findings of Green et al. (2020). However, in contrast to Green and colleagues' findings sustaining a previous ACL injury or calf injury were not associated with an

increased risk of any form of hamstring strain (Tables 5.4.1d - 5.4.1f). In contrast, previous injuries to the MCL and lateral ankle ligaments were associated with an increased odds of all forms of hamstring injury analysed within the study (Tables 5.4.1d - 5.4.1f). These findings were in agreement with Malliaropoulos et al. (2018), who reported that national level track and field athletes with a preceding ankle ligament injury had a higher risk of subsequent hamstring injury. The authors hypothesised that in some cases athletes may alter their movement strategy during locomotion and other sporting tasks following an ankle injury (Malliaropoulos et al., 2018). This may then be exacerbated by an inadequate rehabilitation process (Malliaropoulos et al., 2018). Previous research has observed alterations in lower limb and pelvis biomechanics following lateral ankle ligament injuries, including alterations in joint kinetics to favour knee and hip loading (Doherty et al., 2015a; Doherty et al., 2015b; Doherty et al., 2015c). With the end result potentially subjecting the hamstrings to higher magnitudes of mechanical and stresses and strains.

Previous injury variables demonstrated a mixed ability to classify out of sample hamstring injuries. The variable with the highest classification performance score was previous lateral ankle ligament injury which demonstrated LOOCV AUC scores of 0.65 – 0.67 for pooled hamstring strains regardless of inciting events as well as biceps femoris strains. However, caution is needed when interpreting these results as AUC scores smaller than 0.7 are considered poor (Hosmer et al 2013).

5.5.2 Question 2. Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

It was hypothesised that players who displayed larger magnitudes of knee flexion strength would have a reduced risk of sustaining hamstring strain. However, this hypothesis was not accepted, with the highest magnitudes of knee flexion strength associated with an increased odds of sustaining hamstring strains (Tables 5.4.2a –5.4.2e; figures 5.4.2a – 5.4.2f). This finding was contrary to the majority of previous studies that have examined isokinetic knee flexion

strength variables in relation to hamstring strain risk (Green et al., 2017; Green et al., 2020), which have observed either no effect (Bennell et al., 1998; Zvijac et al., 2013), or a small decrease in risk (van Dyk et al., 2016). Whiteley et al. (2017), reported athletes who subsequently injured their hamstrings exhibited larger magnitudes of isokinetic hamstring and quadriceps strength, however the authors did not propose any explanation for the mechanisms underpinning their findings. This was at the time of writing (2021) the first study to comprehensively examine isokinetic knee strength in relation to hamstring strain risk within a male professional rugby union population. Given the diverse physical demands of rugby union (Duthie et al., 2003; Stokes et al., 2020; West et al., 2020), and the unique anthropometric characteristics of rugby union players (Zemski et al., 2015), generalising the findings of previous research that has predominantly used soccer and AFL populations may not be appropriate for the study cohort (Green et al., 2017). A possible explanation for this finding may be due to the underlying architecture of the hamstring muscles. Previous studies have observed individuals following a concentric contraction focussed hamstring training program have smaller muscle fascicle lengths when compared to matched individuals following an eccentric focussed training program (Timmins et al., 2016). Shorter hamstring fascicle lengths have also been observed to increase hamstring strain risk (Timmins et al., 2016). Measurement of fascicle length was not possible during the current project; however future research should examine if there was a mechanism.

Although the increased magnitudes of isokinetic strength were associated with increased odds of all hamstring strains (Table 5.4.2a), those sustained when running (Table 5.4.2b), and sustained during a contact event (Table 5.4.2c) and medial hamstring muscle strains (Table 5.4.2e), the ability of these categorical isokinetic variables to categorise new out of sample data (i.e. to subsequently predict whether a player will sustain a hamstring strain after an isokinetic test) was observed to be poor, with the highest LOOCV AUC score observed being 0.57. When used in isolation findings have limited clinical value, equating to 7% more than random chance (Hosmer et al., 2013). This finding supports the previous findings of van Dyk and colleagues (van

Dyk et al., 2016; van Dyk et al., 2017) who reported that isokinetic assessment of knee strength had no clinical value in predicting hamstring injury risk in soccer. It is therefore recommended that for the wider professional rugby union population the findings should be replicated. However, for the study population and the professional rugby team co-funding the PhD project it is recommended that isokinetic assessment of knee strength in its current form is not used in the clinical decision-making process to infer hamstring strain risk or for returning to play post rehab.

Isokinetic strength variables were also investigated in relation to specific hamstring muscle injury risk (i.e. the odds of sustaining a biceps femoris strain; the odds of sustaining semimembranosus/ semitendinosus strain). No isokinetic variables were associated with biceps femoris injury (Table 5.4.2d), whereas concentric hamstring strength at the slower speed condition (60°/s) was associated with an increased odds of medial hamstring injury (Table 5.4.2e). This is a novel finding, as at the time of writing (2021) this is the first study to examine specific hamstring muscle strain risk in relation to isokinetic strength variables which questions the efficacy of the conventional method of isokinetic testing of the knee joint (i.e. During 90° hip flexion). A number of studies have examined hamstring muscle activation in relation to resistance exercise, employing surface electromyography or functional MRI (Bourne et al., 2018). The majority of these studies reported that the long head of the biceps femoris and semimembranosus were preferentially activated more during hip extension compared to knee flexion, whereas the semitendinosus displayed higher levels of activation during knee flexion exercises (Bourne et al., 2018). A limitation of the current study was that the medial hamstrings were grouped together which may have potentially confounded the findings in relation to the isokinetic strength measurements. Regardless, the isokinetic assessment in the current study and the overwhelming majority of previous studies examining isokinetic strength in relation to hamstring strength (Green et al., 2017), are not suitable to comprehensively examine the strength of the biceps femoris and possibly the semimembranosus. It is recommended that as an alternative isokinetic assessment of hip extension and or assessment of the force produced

during Nordic hamstring exercise should be employed if insight into specific hamstring muscle risk is required (Green et al., 2017; Green et al., 2020).

5.5.3 Question 3. Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players?

The second purpose of the study was to examine high-speed running exposure in relation to hamstring strain risk. It was hypothesised that higher magnitudes of acute exposure to high-speed running would increase the risk of sustaining a hamstring strain whilst running. Although a trend was observed with higher magnitudes of acute high-speed running exposure appearing to increase the odds of hamstring strain (especially 3-day EWMA), this was inconclusive due to the wide distribution of the hamstring injury group (Table 5.4.3a; Figure 5.4.3a). The finding suggests that, other variables may modify the influence of acute exposure to high-speed running in relation to hamstring strain risk. Previous examination of acute exposure to high-speed running in relation to hamstring strain risk observed a significant association with increased injury risk over a time period of 7 days in an AFL cohort (Dulig et al., 2016; Ruddy et al., 2018). Interestingly, in a recent study examining the relationship between player workloads and any soft tissue injuries in male professional rugby union players, West et al. (2020b) reported that acute 3-day workloads were not associated with injury. Regardless of the different injury of interest (any soft tissue injury vs hamstring injuries sustained during running) and data collection modality (Self-reported session rating of perceived exertion vs GPS derived high-speed running exposure) between the studies, the agreement is of interest and suggests the manner in which workload is managed differs from other sports such and requires further investigation.

It was also hypothesised that chronic exposure to moderate magnitudes of high-speed running would decrease the risk of sustaining a hamstring strain. This was supported in the current study, and was previously observed in other field-based team sport studies examining the influence of chronic exposure in relation to all lower limb injuries (Malone et al., 2016; Malone et al., 2018; Malone et al., 2019). Furthermore, chronic exposure to high magnitudes resulted in an increased

risk of injury in the current study (5.4.3b), specifically chronic exposure to high-speed running over 14 and 21 days prior to the week where the injury was sustained, were the only certain high-speed running variable associated with an increased risk of hamstring strain. Similar findings were reported by Ruddy and colleagues (2018), who observed large magnitudes of high-speed running exposure over 3 and 4-week were significantly associated with increased hamstring strain risk. The timeframes were also in agreement with West et al. (2020b), who observed larger magnitudes of 14-day chronic workloads (measured via session rating of perceived exertion) were associated with an increased injury risk (all soft tissue) in male professional rugby union players.

The basic pathophysiology of hamstring (and other skeletal muscle) strains is considered be acute, resulting from high tensile forces acting instantaneously on the myofibrils which results in mechanical strains that exceed capacity causing rupture. The mechanism of this pathophysiology in the hamstrings muscle group is either thought to be via an event with a large external force such as the foot striking the ground during the early stance phase within the running gait cycle; or an event with a large internal force (trying to decelerate the shank from extending in the late swing phase when sprinting) as the driver for the process of muscle strain. It is plausible that chronic exposure to high magnitudes of high-speed running may result in the accumulation of eccentrically induced muscle damage (Morgan, 1990). Which may make the hamstrings more susceptible to strain injury (Opar et al., 2012). Although this hypothesis has not yet been tested, the time frame of eccentrically induced muscle damage (Morgan, 1990), accompanied with the subsequent increased risk of macroscopic trauma (Timmins et al., 2016), is suggestive that prior running exposure may influence susceptibility of hamstring strain injury for an extended period of time.

The ability of high-speed running variables to classify novel data was mixed (Table 5.4.3a – 5.4.3a). The highest LOOCV AUC scores were for univariate 21 Day EWMA - 7 Day Lag (LOOCV AUC = 0.65). The LOOCV AUC scores for high-speed running exposure observed in the current

study were within a similar range to those reported by Ruddy and colleagues (2018) in a larger cohort of hamstring injuries in Australian football (AUC = 0.5 - 0.63). It is recommended that for the wider professional rugby union population the findings should be replicated with a larger sample size using multiple teams. For the study population and the professional rugby team co-funding the PhD project it is recommended that high-speed running exposure should be examined using a EWMA over a four-week window of time, with particular emphasis paid to the magnitude of exposure in the previous three weeks exclusive of the past seven days. For conservative hamstring strain risk management acute exposure to high-speed running should also be monitored for large magnitudes. Additionally, the relationship between exposure to high-speed running and hamstring strain should be further examined. The manner in which an individual moves when running at high-speed has been highlighted as an area for future prospective research (Green et al., 2020). This was not possible in the current study because the laboratory space and capture volume were too small to replicate the running velocities observed during match play and training.

5.6 Limitations

The present study had several limitations and assumptions. The sample size (one team and ~35 players per year) within the current study was small. However, due to one professional rugby team co-funding the research project, this was unavoidable. However, the incidence of hamstring injuries observed was within the most recently reported by the England professional rugby injury surveillance project (Kemp et al., 2019). For the isokinetic analysis, when comparing the sample size and number of injuries sustained to previous prospective studies (Green et al., 2017), the current study had the fifth largest sample size (5/12) and third largest number of injured cases (3/12). For the analysis of exposure to high-speed running there was an issue of class imbalance within the dataset (i.e. un-injured days outnumbered injured days). Due to the constrained timeframe and the single team co-funding the project collecting more data was not an option. Furthermore, due to the observed incidence rates of hamstring injury, an additional

season of data collection would have resulted in a negligible increase in the injured case number relative to the un-injured cases. Steps were taken to mitigate the influence of class imbalance, a conservative sample reduction was employed (Carey et al., 2018), combined with the apparent AUC scores of all models undergoing leave-one-out cross-validation to evaluate the model's ability to classify novel data (Carey et al., 2018). As previously mentioned in the discussion, the LOOCV AUC scores for high-speed running exposure observed in the current study were within a similar range to those reported in a larger cohort of hamstring injuries in AFL (AUC = 0.5 - 0.63) (Ruddy et al., 2018).

5.7 Conclusions

This study was unique in comparison with previous prospective studies examining hamstring injury risk in that it was the first at the time of writing to examine both isokinetic knee strength and high-speed running exposure within a male rugby union population.

The findings of the study were in agreement with previous prospective studies that although there is an association between isokinetic knee strength and hamstring strain the ability of isokinetic variables to classify hamstring strains is poor and therefore should be used with caution. Previous injury to the hamstrings were associated with an increased odds of subsequent hamstring injury when all muscles were pooled. A novel finding was that previous MCL and lateral ankle ligaments injuries were associated with an increased risk of biceps femoris injury and medial hamstring injury. The current findings relating to high-speed running exposure suggest rather than acute unaccustomed increases the high-speed running being the metaphorical 'straw that broke the camel's back', high magnitudes of exposure in the three-weeks preceding injury are actually the 'accumulation of straw that exceeded the camel's capacity'. As such, particular focus should be paid to building players' tolerance to high-speed running gradually and to ensure that chronic exposure to high-speed running is considered when planning training sessions.

Chapter 6: Study 3 An exploration of factors associated with knee ligament injury in male professional rugby union players

6.1 Context

In the context of the PhD project, injury to the MCL and ACL knee ligaments resulting from contact events presented a high injury burden to the professional rugby team co-funding the project in addition to the greater men's professional rugby union population. Establishing the efficacy of the current methods employed by the team to infer MCL and ACL injury risk, and where possible proposing alternative solutions were paramount to ensure the research made a positive impact to the team co-funding the research project and provided a meaningful contribution to the field of injury research in rugby union by informing the decision-making process in relation to player MCL and ACL strain risk. This study posed a different challenge to that of chapter 5, there is a paucity of research concerning the aetiology of knee ligament injury sustained during contact events, despite the epidemiological evidence highlighting it as an area of high injury burden. In the absence of previous research, the author had to design the study from the first principles proposed in injury prevention frameworks and models of injury aetiology. Biomechanical analysis of a single-leg drop jump task and exposure to both contact events and accelerometer derived on pitch physical activity were added as novel measures to the rugby team's pre-existing risk stratification process of using heuristics based on the results of isokinetic dynamometry of the knee and previous injury history. Following the work completed in chapter 5, alternative options were investigated to mitigate the issue of class imbalance inherent within the area of player workload analysis. From this research, a minority over sampling approach was employed in conjunction to the majority under sampling approach used in the previous chapter to mitigate the influence of class imbalance. Due to the novel research focus, the analysis was conducted in relation to odds of injury per unit increases of the independent variables of interest rather than odds of injury per tertile categorical groups in the previous chapter.

6.2 Introduction

Knee ligament injury poses a high burden in men's professional rugby union (Dallalana et al., 2007; Kemp et al., 2018; Kemp et al., 2019; West et al., 2021b; Williams et al., 2021; Chapter 3) in addition to other field-based team sports (Awwad et al., 2019; Della Villa et al., 2020; Buckthorpe et al 2021). In the short to medium term, the consequences of such injuries may negatively impact neuromuscular function of the injured player upon return to play. This may impact the individual and team through decreased performance as well as financial earnings of the individual and professional rugby team (Williams et al., 2016). Moreover, the long-term effects of injury to the knee ligaments may result in an increased risk of developing osteoarthritis, subsequently resulting in further medical costs, then later a decrease in quality of life (Simon et al., 2015; Davies et al., 2017).

Epidemiological studies examining time-loss injuries sustained by male professional rugby union players have consistently reported that knee ligament injuries, specifically injuries to the medio-collateral ligament (MCL) and the anterior-cruciate ligament (ACL) resulted in some of the largest magnitudes of injury burden compared to other injuries sustained (Brooks et al., 2005a; Brooks et al., 2005b; Dallalana et al., 2007; Kemp et al., 2018; Kemp et al., 2019; West et al., 2021b; Chapter 3). However, the manner of injury burden differs between ligaments, MCL injuries are reported to have a high rate of incidence combined with moderate and high injury severities (Kemp et al., 2019). Whereas ACL injuries occur less frequently, but exhibit some of the highest magnitudes of time loss (Kemp et al., 2019). Of these knee ligament injuries, the overwhelming majority reported were sustained during an inciting event involving direct contact between multiple players (e.g. being tackled, tackling, rucking, mauling and scrummaging) rather than non-contact inciting events such as rapidly changing direction (Dallalana et al., 2007; Chapter 3). During these 'contact' knee ligament injuries, direct contact was made between another player and either with the knee, or to the lower limb above or below the injured knee (Montgomery et al., 2018; Buckthorpe et al., 2021). These contacts are typically either on the lateral side of the injured limb (MCL & ACL injury) resulting in large magnitudes of knee joint

abduction moment combined with medial translation of the tibia (Montgomery et al., 2018; Buckthorpe et al., 2021), or contact to the anterior side of the femur or the posterior side of the tibia (ACL injury) resulting in excessive anterior tibial translation (Buckthorpe et al., 2021). The findings from the previous epidemiological studies were also consistent with that of the current work (Chapter 3 – Study 1 Injury epidemiology study).

Once the epidemiological extent of an injury problem has been identified, in accordance with existing frameworks of injury prevention it is necessary to examine the underpinning aetiology of the injury in order to iteratively aid the development of preventative measures, with the end goal being a reduction observed in the incidence of MCL and ACL injury arising from contact events and associated severity (van Mechelen et al., 1987; Finch, 2006). Previously been proposed for knee ligament injury have primarily focussed on the ACL, with the strongest evidence observed between non-modifiable variables including such as previous injury history, to the knee ligaments in addition to the proximal and distal tissues of the lower limb (Fulton et al., 2014; Smith et al., 2012; Waldén et al., 2006; Williams et al., 2017). Modifiable risk factors have also been proposed including: increased exposure to the inciting event where the injury was sustained i.e. more exposure to rugby tackles or ruck events (Dallalana et al., 2007; Hendricks et al., 2017; Stokes et al., 2020; Williams et al., 2021; Edwards et al 2021a) or unaccustomed increases in external workload (Windt and Gabbett, 2016; Windt and Gabbett, 2017). Other previously identified modifiable risk factors include musculoskeletal variables associated with hamstrings and quadriceps strength (Burgi et al., 2019), and frontal plane biomechanical variables of the lower limb such as large magnitudes of external knee abduction moment (the rotational force originating outside of the body acting to force the knee into abduction) (Hewett et al., 2005). However, the majority of these risk factors pertain to knee ligament injuries sustained during inciting events where no contact with an opposing player or object has occurred (non-contact). Despite this, these variables are routinely used to infer an individual's risk of knee injury regardless of the inciting event and to quantify whether rugby

players are safe to return to play following injury within the club participating in this project as well as the greater professional sport community. The mechanisms of contact-based ligament injury are theoretically similar to those of non-contact injury, even when the inciting events are not. The only difference being two inputs of external force: 1. External force in the form of ground reaction force from the injured player's foot (like in non-contact injuries); and 2. External force in the form of another player or object applied to the injured player e.g. the opposing player's shoulder contacting the injured player's anterior tibia. Therefore, there is a need to examine the efficacy of the use of these variables in relation to the risk of sustaining an MCL or ACL injury during a contact event.

Isokinetic assessment of quadriceps and hamstrings strength has been ubiquitously used as a proxy for knee ligament injury risk within team sports for the past 20 years (Orchard et al., 1997; Burgi et al., 2019), as well as the professional Rugby Union club participating in the research project. The mechanism associated with these isokinetic assessments is that injury to the ACL occurs via the 'stronger' quadriceps exerting large tensile force on the tibia, and/or 'weaker' hamstrings not being able to exert an equal and opposite tensile force on the tibia resulting in excessive anterior tibial translation subjecting the ACL to excessive magnitudes of stress and strain (Draganich and Vahey, 1990; Bates et al., 2018; Nasseri et al., 2020). However, there are limited prospective studies which observed varying relationships between knee ligament injury risk in relation to isokinetic strength of the knee joint (Myer et al., 2010; Söderman et al., 2001; Steffen et al., 2016; Uhorchak et al., 2003; Bakken et al., 2018). Of these studies the majority have only examined non-contact ACL injuries sustained by female team sport athletes (Myer et al., 2010; Söderman et al., 2001; Steffen et al., 2016), making translation of the findings to male professional rugby players ambiguous. Furthermore, at the time of writing (2021) there has been no examination of whether a relationship exists between lower limb isokinetic variables and the risk of sustaining an MCL injury despite isokinetic testing being recommended by researchers (Kim et al., 2016) and being a return to play criteria at the rugby union club participating in the research project.

Abnormal lower limb biomechanics during sporting movements involving large magnitudes of force generated in a short amount of time have long been theorised to be associated with knee ligament injury risk (Hewett et al., 2005). With larger external knee abduction moments during these tasks placing both the MCL and ACL come under excessive magnitudes of mechanical stress and strain resulting in immediate failure (Matsumoto et al., 2001; Mazzocca et al., 2003; Hewett et al., 2005; Kiapour et al., 2015; Kiapour et al., 2016; Andrews et al., 2017; Bates et al., 2017; Buckthorpe et al., 2021; Weir, 2021). In addition, knee ligament injuries typically occur within 50 milliseconds of impact (Koga et al., 2010; Hewett et al., 2016; Stuelcken et al., 2016). However, due to the resources required for data collection and the associated time cost have been no prospective studies examining biomechanical variables during contact events at the time of writing (2021). Instead, prospective studies have favoured the use of jumping and landing tasks due to the reported similarity in joint kinetics (Jones, et al, 2014; Cronström et al, 2020; Weir, 2021). However, these studies have focussed on ACL injury aetiology despite both the burden posed by MCL injuries and the function of the MCL being to restrain excessive external knee abduction moments (Nigg and Herzog, 2007; Schein et al., 2012). The existence of a relationship between external knee abduction moment and ACL injury was observed by early research (Hewett et al 2005). However, subsequent studies reported conflicting findings (Krosshaug et al., 2016), with a recent meta-analysis reporting that knee abduction moment was not related to ACL injury risk (Cronström et al., 2020). Cronström and colleges observed that the difficulty of movement task selected for the study population may have explained the discrepancies in the findings (Cronström et al., 2020). As an example, a high magnitude of knee abduction moment during a bilateral drop-jump task resulted in an increased odds of sustaining ACL injuries in female high school team sport athletes (Hewett et al., 2005), whereas no relationship was observed during the same task in female professional team sport athletes (Krosshaug et al., 2016; Smeets et al., 2019) or military recruits (Goerger et al., 2015). Due to this, selection of single-leg jumping and landing tasks have been recommended for assessing knee ligament injury risk due to the increased stabilisation demands of the lower limbs and trunk

(Cronström et al., 2020). The single-leg drop jump has recently been recommended for biomechanical analyses concerned with knee ligament injury aetiology (King et al., 2018; King et al., 2019; King et al., 2021a). When examining lower limb biomechanics in relation to knee ligament injury risk, neuromuscular control of the hip joint (specifically hip abduction) was also previously cited to influence injury risk by limiting modifying knee abduction moments (Khayambashi et al., 2016; Maniar et al., 2018). However, in a recent meta-analysis, Chia and colleagues (2020) observed that the influence of hip abduction moments as a potential risk factor for knee ligament injury was conflicting, with the authors concluding there is limited support. The overwhelming majority of studies included in the meta-analyses by both Cronström et al. (2020) and Chia et al. (2020) have only examined non-contact ACL injuries sustained by team sport athletes competing in non-contact sports, making interpretation of the findings to male professional rugby players unclear. Therefore, examination of the existence of a relationship between lower limb biomechanical variables during a single-leg drop-jump task and the risk of sustaining an MCL injury and pooled MCL and ACL injury data is necessary to substantiate recommendations for risk assessment and return to play criteria by researchers (Kim et al., 2016). and therefore should be included as a novel measure in the testing battery.

Unaccustomed increases in exposure to potentially injurious magnitudes of on pitch-physical activity involving contact events like the tackle and rucking have been speculated to increase the risk of injury in contact team sports like Rugby Union (Tierney et al., 2020; Edwards et al., 2021a). However, despite this commonly held hypothesis, at the time of writing (2021) there has been no research examining the fluctuations in contact events experienced by players in relation to injury risk despite contact events being acknowledged as an area of increased injury risk. As a result, monitoring exposure to contact events has not been widely adopted within male professional rugby union teams, including the participating team (West et al., 2019). A popular variable when examining if a relationship is present between workload and the risk of sustaining injuries in field-based team sports including rugby union is the acute:chronic workload ratio (ACWR) (Hulin et al., 2014; Cousins et al., 2019). The ACWR comprises the ratio between the

most recent exposure of workload ("acute workload") theorised by the authors to represent a component of fatigue and previous exposure to workload examined over a longer duration ("chronic workload") theorised to represent a protective fitness (Gabbett, 2016). However, a Recent critique of ACWR questioned the appropriateness of it as a variable for inferring injury risk (Impellizzeri et al., 2020; Kalkhoven et al., 2021), with the authors of the critique suggesting that the use of ratio data such as the ACRW may result in spurious associations. A further limitation of some of the studies to date is that the use of standardised ACWR (e.g. 7 day acute / 28 day chronic ACWR) may be inappropriate within rugby union (West et al., 2021c). Furthermore, using set ACWR timeframes to infer the risk of a plethora of injuries (e.g. soleus vs biceps femoris injury risk) and inciting events (e.g. injured whilst running vs injured during contact) fails to adhere to the principles presented in theoretical models of injury aetiology. These models suggest that injury risk is complex and multifactorial in nature, with internal and external factors associated with injuries varying between injury types, mechanisms and even inciting events to varying degrees for each individual (van Mechelen et al., 1992; Meeuwisse, 1994; Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2017; Bittencourt et al., 2016). Therefore, before any ACWR can be applied to examine MCL and ACL injury risk within a population (i.e. men's professional rugby union) one must first examine the manner in which acute and chronic exposure to contact events influences knee ligament injury risk.

Therefore, the purpose of this study was threefold. The first purpose was to determine if exposure to the inciting events where contact injury occurred were associated with knee ligament injury within male professional rugby union players. The second purpose was to establish whether a relationship existed between previously identified injury history, isokinetic knee joint strength and biomechanical variables and contact knee ligament injury within male professional rugby union players. The third purpose was to establish whether a relationship existed between exposure to on-pitch physical activity and knee ligament injury within male

professional rugby union players. To facilitate this, the following research questions were explored:

1. Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players? i.e. Will individuals exposed to more tackles and rucks have an increased risk of sustaining MCL or ACL injury from a tackle or ruck event?

   H1: Acute exposure to higher magnitudes of tackle and ruck events exposure would increase the risk of sustaining an MCL or ACL injury from a contact event.

   H2: Chronic exposure to higher magnitudes of tackle and ruck events would decrease the risk of sustaining an MCL or ACL injury during a contact event.

2. Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players?

4. Is previous lower limb injury history associated with the risk of sustaining an MCL or ACL injury from a contact event?

   H3: Sustaining a previous MCL and/or ACL injury would increase the risk of sustaining an MCL or ACL injury from a contact event.

   H4: Sustaining a previous injury to the hamstrings muscle group would increase the risk of sustaining an MCL or ACL injury from a contact event.

   H5: Sustaining a previous injury to the triceps surae muscle group would increase the risk of sustaining an MCL or ACL injury from a contact event.

5. Are previously identified isokinetic knee strength variables associated with the risk of sustaining MCL or ACL injury within the cohort of male professional rugby union players?

   H6: Isokinetic knee flexion strength variables would not influence the odds of players sustaining an MCL or ACL injury from a contact event.

H7: Isokinetic knee extension strength variables would not influence the odds of players sustaining an MCL or ACL injury from a contact event.

6. Are lower limb biomechanical variables during the early ground contact phase of a single-leg drop-jump task associated with the risk of sustaining MCL and ACL injury within the cohort of male professional rugby union players?

   H8: Players with larger magnitudes of external knee abduction moment would be more susceptible to sustaining an MCL or ACL injury from a contact event.

   H9: Players with larger magnitudes of external hip adduction moment would be more susceptible sustaining an MCL or ACL injury from a contact event.

3. Are acute increases in exposure to on-pitch physical activity associated with an increased risk of MCL and ACL injury within male professional rugby union players?

   H10: Acute exposure to higher magnitudes of on-pitch physical activity exposure would increase the risk of sustaining an MCL or ACL injury from a contact event.

   H11: Chronic exposure to higher magnitudes of on-pitch physical activity would decrease the risk of sustaining an MCL or ACL injury from a contact event.

6.3 Methods

6.3.1 Participants

A total of 143 professional rugby union players from one club competing in the English premiership took part in the study spanning seven playing seasons (2012-13 Season to 2018-19 Season). Throughout the study period repeated measurements were collected from the cohort of players, equating to 414 player-seasons (Figure 6.1 & 6.2). At the time of writing the team competed in the highest level of national competition in professional rugby union in England. At the start of each playing season during the second week of the preseason period, players underwent a periodic health examination (PHE) at the University of Exeter. As part of the PHE assessment of knee joint isokinetic extension and flexion strength was performed. Three-

dimensional biomechanical analysis of the lower limbs and pelvis during a single-leg drop jump to stabilisation was also performed. The study was approved by the University of Exeter Sport and Health Sciences ethics committee.

Because of the longitudinal nature of the study, the majority of the exclusion criteria applied to the exclusion of players from the current playing season rather than the entire study. Therefore, players from the senior playing squad who sustained injuries to the MCL and/or ACL during a competitive match or during training sessions were included in the study (Brooks et al., 2005a; Brooks et al., 2005b; Dallalana et al., 2007). The exclusion criteria from the previous study (Chapter 5 - Study 2) was adapted for the current study:

6. Once a player left the team, they were excluded from the analyses of subsequent playing seasons.
7. Players were excluded from the analysis of the playing season if they sustained a time loss injury prior to completing a PHE within the same playing season.
    a. If the player returned from injury before the start of the next playing season, they were then re-entered into the analysis for the subsequent playing season.
    b. If the player did not return from injury before the start of the next playing season, they were then excluded from the analysis for the subsequent playing season.
8. Players were excluded from the analysis of the playing season if they were unable to undergo a PHE within the same playing season.
    a. If the player was able to undergo a PHE during the subsequent season they were re-entered into the analysis for the subsequent season.
    b. If the player was not able to undergo PHE testing during the subsequent season they were excluded from the analysis for the subsequent season.

9. Players who subsequently sustained lower limb time loss injury other than an MCL or ACL after a PHE within the same season were excluded from the analysis of the playing season.
    a. If the player returned from injury before the start of the next playing season, they were then re-entered into the analysis for the subsequent playing season.
    b. If the player did not return from injury before the start of the next playing season, they were excluded from the analysis for the subsequent playing season.
10. Players who subsequently sustained a MCL or ACL but did not perform a PHE within the same season were excluded from the analysis of the playing season.
    a. If the player returned from MCL or ACL injury before the start of the next playing season AND performed isokinetic during the subsequent PHE, they were then re-entered into the analysis for the subsequent playing season.
    b. If the player returned from an MCL or ACL injury before the start of the next playing season AND they did not perform the subsequent PHE, they were excluded from the analysis for the subsequent playing season.
11. If on-pitch physical activity data and/or contact event data were not recorded for a player over the 28-day period prior to an MCL or ACL injury the player would be excluded from the external workload analyses regardless of sustaining an injury or not.

A worked example of the exclusion criteria in the present study is presented in figure 6.1. Furthermore, a flowchart illustrating the movement of players and repeated testing as well as the inclusion/exclusion criteria on the study cohort is presented in figure 6.2.

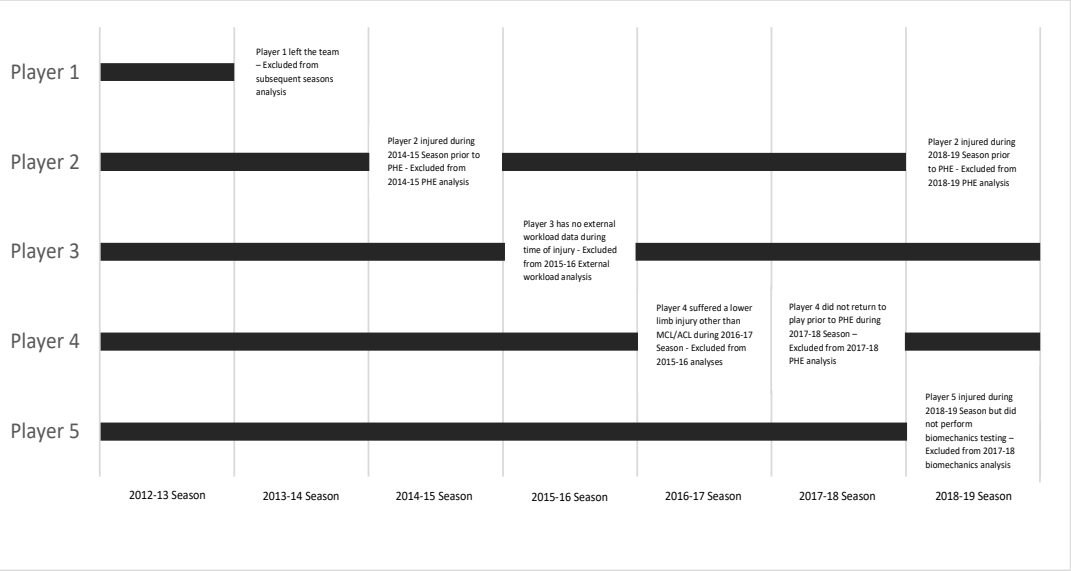


Figure 6.3.1a A worked example of the study exclusion criteria.

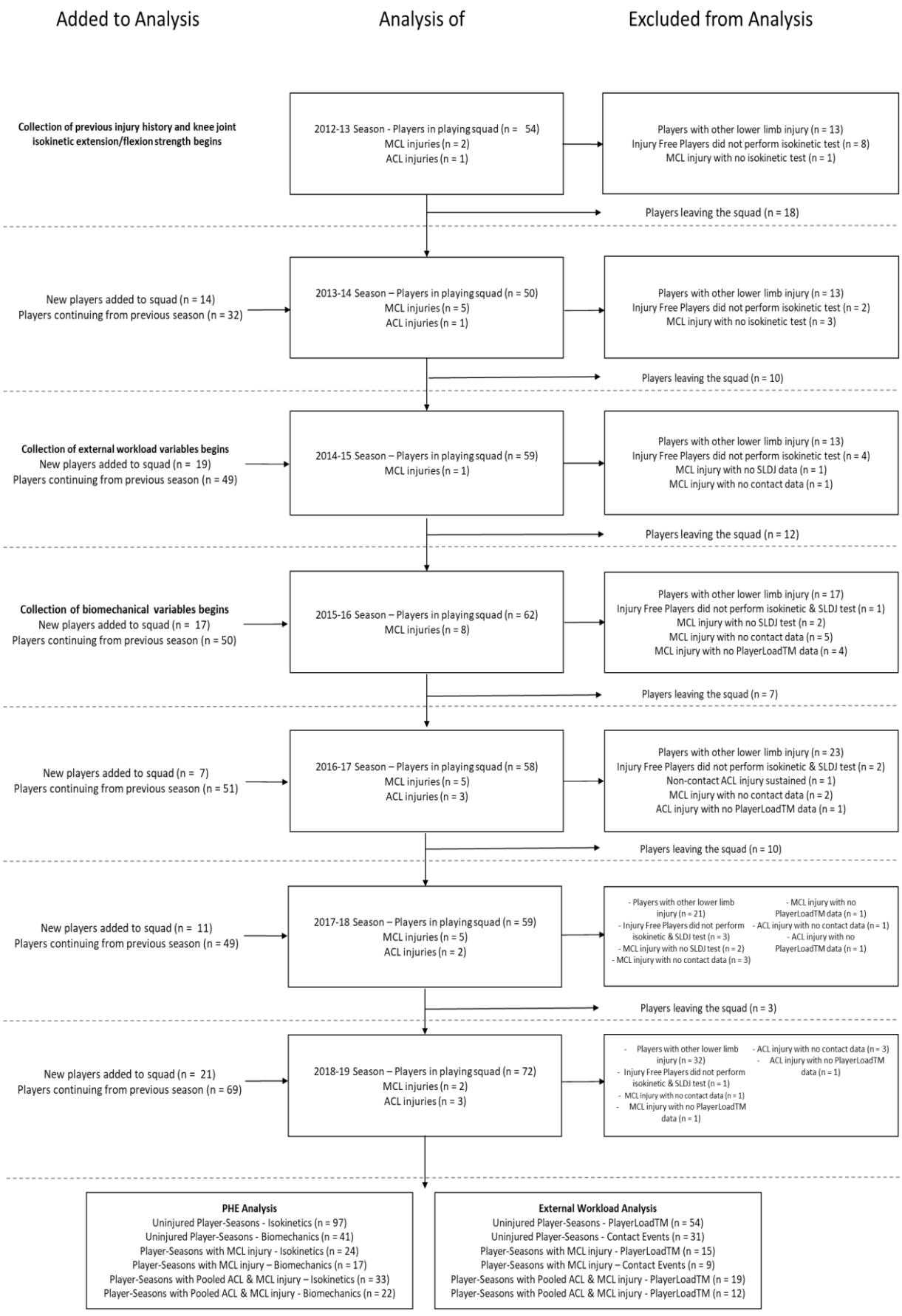


Figure 6.3.1b A flowchart illustrating the movement of players throughout the study period.

6.3.2 Data collection

6.3.2.1 Injury definition and surveillance

Knee ligament sprain was defined as a tear (including partial or complete tears) of the anterior cruciate ligament (ACL) or medial collateral ligament (MCL) (Bahr et al., 2020). A time loss definition of injury was used in the study, and was defined as "any injury to the MCL or ACL that prevented a player from taking full part in all training activities typically planned for that day and/or match play for more than 24 hours, from midnight at the end of the day the injury was sustained." (Brooks et al 2005a; Brooks et al 2005b; Dallalana et al., 2007). Knee ligament injuries were diagnosed using magnetic resonance imaging, following an initial clinical examination by medical personnel at the rugby club (Hewett et al., 2005; Krosshaug et al., 2016; Hwang et al., 2018). Injuries were reported using a modified Orchard Sports Injury Classification System (OSICS) (Orchard, 1995).

6.3.2.2 Knee joint isokinetic flexion strength

Isokinetic knee flexion and extension moment were assessed using an isokinetic dynamometer (Biodex Multi-Joint System 3; Biodex Medical Systems Inc, Shirley, NY, USA), using the same data collection and analysis procedures in Chapter 5 Section 5.3.2.2.

6.3.2.3 Biomechanical Data

6.3.2.3.1 Instrumentation

Kinematic data were collected using four CODA cx1 units sampling at 200 Hz (Charnwood Dynamics, Rothley, Leicestershire, UK). Active LED markers were secured to the lower limbs, pelvis and trunk to create a 6 degree of freedom biomechanical model of the lower limb and pelvis in accordance with the International Society of Biomechanics (ISB) recommendations (Wu et al., 2002; Derrick et al., 2020) (Table 6.3.2.3.1). Force data were synchronously collected with

kinematic data using a 0.6 m x 0.4 m force platform sampling at 1000 Hz (BP400600HF, AMTI, Massachusetts, USA) (Figure 6.3.2.3.1).

Table 6.3.2.3.1 Bilateral lower limb and pelvis marker set

Rigid Foot with Hallux	Shank	Thigh	Pelvis
Anatomical Markers	**Anatomical Markers**	**Anatomical Markers**	**Anatomical & Tracking Markers**
Head of 5th Metatarsal	Tip of the lateral malleolus	Lateral femoral epicondyle	Left anterior superior iliac spine
Head of 1st Metatarsal	Tip of the medial malleolus	Medial femoral epicondyle	Right anterior superior iliac spine
Calcaneus	Lateral femoral epicondyle	Greater trochanter or femur	Left posterior superior iliac spine
Hallux	Medial femoral epicondyle		Right posterior superior iliac spine
Tracking Markers	**Tracking Markers**	**Tracking Markers**	**Tracking Markers**
Head of 5th Metatarsal	A rigid cluster of three markers	A rigid cluster of three markers	Left iliac crest
Base of the 3rd Metatarsal			Right iliac crest
Lateral Calcaneus			Left posterior superior iliac spine
Hallux			Right posterior superior iliac spine

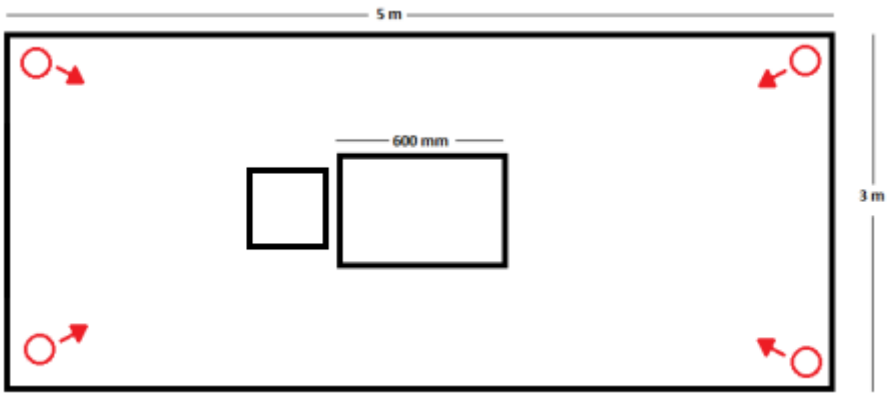


Figure 6.3.2.3.1 Biomechanical laboratory plan and capture volume, including CODA cx1 units (red circle and arrows), force platform (centre rectangle) and box (square to the left of force platform).

6.3.2.3.2 Biomechanical data collection

All participants attended a familiarisation session prior to data collection to ensure they were acquainted with the single-leg drop jump task and testing procedures. A static standing trial was collected for all participants prior to the single-leg drop jump task to ensure the 3-dimensional position of the anatomical markers could be recreated in relation to the tracking markers (Table 6.3.2.3.1). The anatomical markers were then removed for the single-leg drop jump task. Participants were instructed to drop off a 0.2 m box and contact the centre of the force plate with one leg and jump as high as possible (First Contact), participants were encouraged to have a short ground contact time (Figure 6.3.2.3.2). Upon landing, participants were instructed to balance and stabilise on the jumping leg for a minimum of 3 s (Second Contact) (Akins et al., 2013; King et al., 2018). During the movement participants were instructed to keep their hands on their hips and to keep the non-jumping leg as still as possible. Five successful trials were collected per leg per participant after familiarisation. Trials were deemed unsuccessful if participants used arm-swing, or used the non-jumping leg for propulsion or stabilisation.

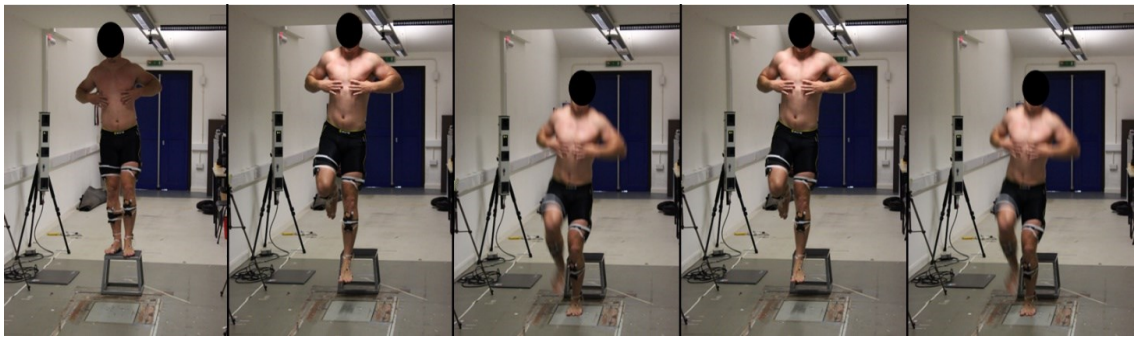


Figure 6.3.2.3.2 Single-leg drop vertical jump sequence.

6.3.2.3.3 Biomechanical Data analysis

Due to the maximal nature of the task, the trial with combination of the shortest contact time and the highest jump height was selected for analysis (Richter et al., 2019), rather than averaging multiple trials to avoid the creation of artificial artefacts in the magnitude and temporal distribution of the moment time series data (Dames et al., 2017). Raw kinematic and GRF data

were interpolated and low-pass filtered at 12 Hz using a fourth order zero-lag Butterworth filter (Kristianslund et al., 2012). The cut-off frequency was determined via residual analysis of marker coordinate data (Winter, 2009). Three-dimensional Euler angles were calculated for the hip, knee and ankle, joint moments of the knee and ankle were calculated using Newton-Euler inverse dynamics where external moments are assumed to be equal and opposite to internal moments (Figure 6.3). Joint moments were modelled and presented in accordance with ISB recommendations (Wu et al., 2002; Derrick et al., 2020), sagittal plane joint moments were modelled as internal moments (e.g. an internal knee extension moment originates within the body and is present during ground contact to prevent the knee from flexing), frontal plane moments were modelled as external moments (e.g. an external knee abduction moment acts on the knee and originates outside the body, it is present during ground contact and is the rotational force trying to abduct the knee) (Derrick et al., 2020). All data were analysed during the first ground contact phase during the single-leg drop-jump. Ground contact occurred when the vertical ground reaction force signal (GRF - Fz) exceeded 20 N and toe-off occurred when Fz decreased below 20 N. Joint angle, GRF and joint moment data were processed using Codamotion ODIN 01.06 (Charnwood Dynamics Ltd., Rothley, Leicestershire, UK), with subsequent analysis extracting sagittal and frontal plane joint moments generated at the hip, knee and ankle performed using customised Python scripts (Python 2.7).

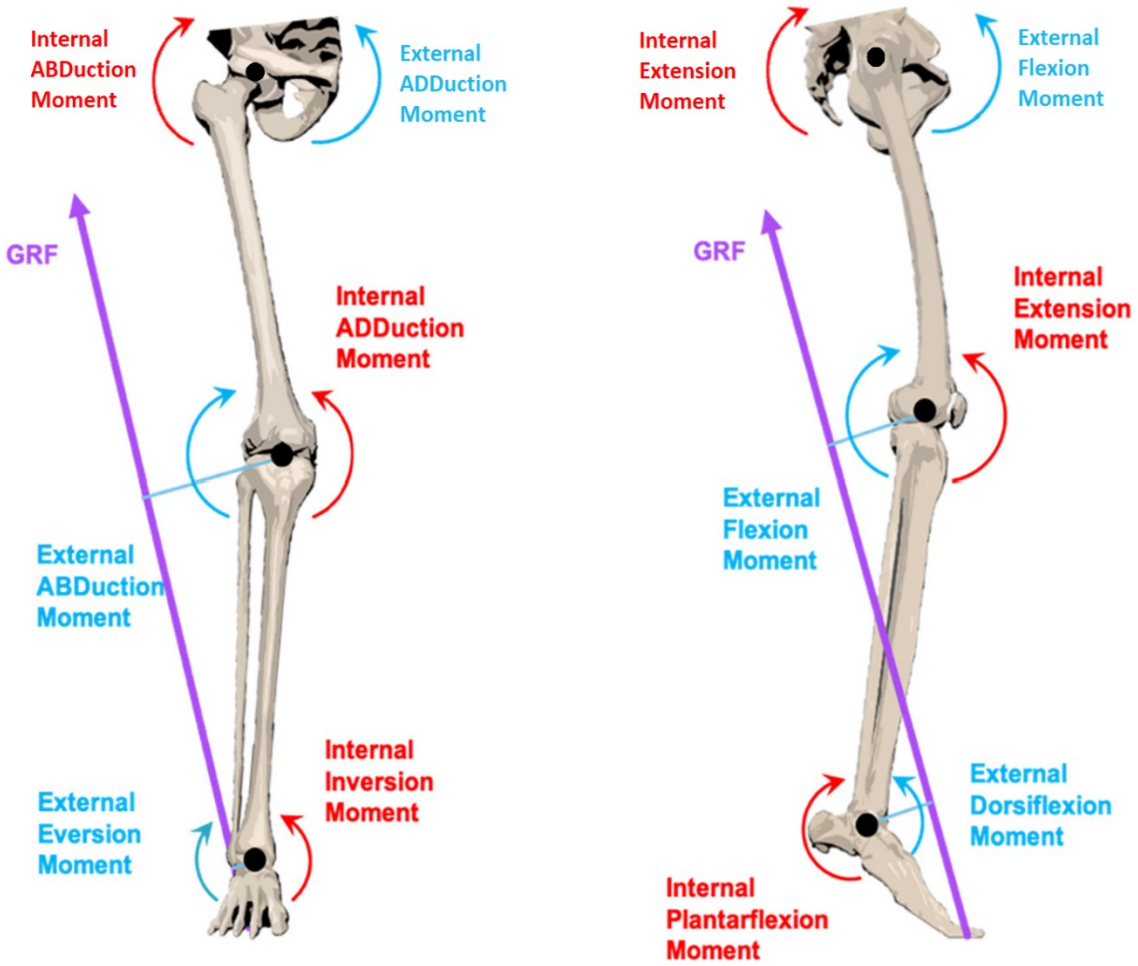


Figure 6.3.2.3.3 Reporting convention of the biomechanical model used in the study, the image illustrates external and internal joint moments occurring during jump landing. Image adapted from Tait et al. (2022).

6.3.2.4 Exposure to on-pitch external workload and contact events

6.3.2.4.1 Instrumentation

On-pitch physical activity for each player was collected using triaxial accelerometers sampling at 100 Hz contained within a global positioning system (GPS) unit (sampling at 10 Hz) (Optimeye X4; Catapult Innovations, Melbourne, Australia). The units also housed a triaxial gyroscope and magnetometer, each sampling at 100 Hz. During matches and on-pitch training sessions, players wore a tight-fitting garment which housed the unit in the upper-back region, between the shoulder blades (Roe et al., 2016). Contact events (tackles, being tackled, defensive & offensive rucking, defensive & offensive mauling, scrummaging and general collisions) were recorded during every first team and second team match by the rugby club's

notational analysts using Sports Code Elite notational analysis software (Sportec, NSW, Australia). Due contact events were not recorded for training sessions.

6.3.2.4.2 Data analysis

After each match or on-pitch training session, data from each player tracking unit was downloaded onto a laptop and processed using proprietary software (Catapult Openfield software; Catapult Innovations, Melbourne, Australia). PlayerLoad™ is a proprietary variable within Catapult Openfield software and was used to quantify player on-pitch physical activity within the study. It was previously defined as an "instantaneous rate of change of acceleration divided by a scaling factor" (Nicolella et al., 2018). In physics, the instantaneous rate of change of acceleration is often termed "jerk" and is defined as:

$$\vec{J} = \frac{\Delta\, \vec{a}\,(t)}{\Delta \mathrm{t}}$$

where $\vec{a}$ is the vector quantity of acceleration and t is time (Eager et al., 2016). PlayerLoad™ was calculated as the vector quantity of each sensor of the triaxial accelerometer within each unit, defined by Catapult Innovations as:

$$Player\ LoadTM = \sum_{t=0}^{t=n} \frac{\sqrt{(fwd_{t=i+1} - fwd_{t=i})^2 + (side_{t=i+1} - side_{t=i})^2 + (up_{t=i+1} - up_{t=i})^2}}{100}$$

Where fwd is anterior-posterior acceleration, side is medio-lateral acceleration, up is vertical acceleration and 100 is the scaling factor (Nicolella et al 2018).

PlayerLoad™ data were collected for every available player per on-field session per day. Which were then aggregated to give a daily value per available player. In order to correctly calculate moving sum and moving average data on days where players did not play or train (e.g. within week non-training day & off season) zero values were imputed to the data-set for each player. Exponentially-weighted moving averages (EWMA) were calculated over 3 and 7 days for acute exposure to on-pitch physical activity. Chronic exposure to on-pitch physical activity EWMA

were calculated over 7-, 14- and 21-day timeframes. In addition, chronic variables were also calculated where on-pitch physical activity was excluded within the 3 and 7 days prior to sustaining an injury (EWMA 3-day & EWMA 7-day lag). The EWMA was calculated using the equation of Williams et al. (2017c):

$$EWMA_{today} = Workload_{today} \times \lambda a + ((1 - \lambda a) \times EWMA_{yesterday}$$

The term λa represents the decay factor and is a value between 0 and 1.

The formula for the decay factor is:

$$\lambda a = 2/(N + 1)$$

The term N is the time decay constant, equating to the previously mentioned timeframes (Williams et al., 2017c). The same approach was used to calculate moving the EWMA for total contact events. However, because contact events were only recorded for first and second team matches, zero values were imputed to the data-set for each player on the non-match days meeting the above requirements. Zero values imputation was selected over removing non-training days from the analysis to preserve the temporal structure of the data set for the EWMA. Only 7-day acute workload variables and 7-, 14- and 21-day chronic variables with and without a 7-day lag were calculated for total contact events.

6.3.3 Statistical analysis

Statistical analysis was conducted using R open-source statistical software (version 4.0.3, R Foundation for Statistical Computing, Vienna, Austria). The independent variables' relationship with both MCL injury risk and pooled MCL and ACL injury risk was analysed using generalised linear mixed-models (GLMM) using the lme4 package, which were selected for their ability to overcome the assumption of independence of observations, resulting in an inflated type-1 error rate when using conventional general linear models employed by the majority of previous studies examining player workload in relation to injury risk (Williamson et al., 1996; Diggle et al., 2013; Impellizzeri et al., 2020). Models were fitted using the lme4 package, with sustaining

either: 1. MCL injury (“yes”/”no”); or 2. MCL or ACL injury (“yes”/”no”) as the dichotomous outcome variables. In a similar manner to the previous chapter (Chapter 5 - Study 2), the present study included data from players over repeated days and seasons that were likely correlated (exploratory data analysis revealed this to be true for some variables); however, the magnitude of correlation was assumed to vary for each player. Therefore, a random effect was included in the model for player ID to account for the unknown correlation caused by including multiple player-days and player-seasons in the analyses.

Class imbalance has been previously identified as a common source of classification error when examining player workload in relation to injury risk i.e. when the number of non-injured days outnumber the number of injured days, the model will always predict the more frequently occurring class leading to inflated type-2 error and erroneous model accuracy estimates (e.g. an AUC > 0.9 with a true positive rate of 0 and a true negative rate of 1) (Kuhn and Johnson, 2013; Carey et al., 2018; Ruddy et al., 2018). For all analyses involving external workload (i.e. exposure to on-pitch physical activity or contact events), a two-step approach was employed to overcome class imbalance following the calculation of EWMA variables. The first step was to reduce the majority class within the sample data set (Krawczyk et al., 2016; Carey et al., 2018). The study used the same majority class reduction approach as the previous chapter (i.e. the post majority sample reduction contact event and PlayerLoad^{TM} data sets both contained continuous 29 days high-speed running exposure data for each MCL and ACL injury of interest for all participants (Table 6.3.3). However, although this reduced the number of injury free days, the sample data set still exhibited a substantial class imbalance when analysing both MCL injury and pooled MCL and ACL injury (Table 6.3.3), therefore a second step was taken to overcome class imbalance. An adaptive synthetic minority oversampling algorithm (ADASYN) was employed using the smotefamily package, to create synthetic minority class data points (i.e. injuries within the contact event and PlayerLoad^{TM} data sets) to ensure the ratio of injuries to injury free data points was 0.20 (Peduzzi et al., 1996). The ADASYN algorithm generated synthetic injury data based on

the density distribution of the injury free data points per 5-nearest neighbour regions of the minority class (i.e. injury data) (Table 6.3.3) (He et al., 2008; Krawczyk et al., 2016; Chapter 4 section 4.4.3.1).

Table 6.3.3 The effect of sampling process on class imbalance in the external workload data analysed in the study.

Dependent Variable	Independent Variable	Sampling Process	Injury Count	Injury Free Player-days	Ratio (Injuries/Injury Free)
Pooled MCL and ACL Injury	Contact Events	Original Dataset	14	4429	0.003
		Majority Undersampled Dataset	14	266	0.05
		ADASYN Dataset	53	266	0.20
MCL Injury	PlayerLoad™	Original Dataset	15	5462	0.003
		Majority Undersampled Dataset	15	2482	0.006
		ADASYN Dataset	496	2482	0.20
Pooled MCL and ACL Injury	PlayerLoad™	Original Dataset	19	5462	0.003
		Majority Undersampled Dataset	19	2482	0.008
		ADASYN Dataset	496	2482	0.20

All variables were modelled and reported as continuous (i.e. the odds of injury per unit increase of the independent variable) with those reaching statistical significance subsequently modelled as tertile categorical groups to allow for interpretation for the rugby team participating in the study. The tertile categorical groups were created in the same manner as the previous chapter, i.e. based on frequency (low, medium and high), with the reference group being the middle group.

Statistical significance for each model were tested using the likelihood ratio test to determine overall model significance and profiled 95% confidence intervals of the odds ratio to determine if each category was significant. Within sample model fit was assessed by calculating the area under the receiver operating characteristic (ROC) curve also referred to as AUC score. The ability of the model to classify novel data was internally validated using leave-one-out cross validation (LOOCV) with an adjusted mean AUC score and 95% confidence intervals of the AUC scores calculated from all LOOCV iteration. Classification performance was defined by the following rule of thumb by Hosmer et al. (2013): ≤ 0.50 = no better than random guess; ≥ 0.5 - < 0.7 = poor

classification performance; ≥ 0.7 - < 0.8 = acceptable classification performance; ≥ 0.8 - < 0.9 = excellent classification performance; ≥ 0.9 = outstanding classification performance.

6.4 Results

During the 7-year study period, a total of 28 MCL and 9 ACL injuries occurred during contact events. These injuries resulted in a total of 1449 (MCL) and 2971 days (ACL) of player absence respectively. Table 6.4 provides an overview of the number of knee ligament injuries sustained during the study period that met the study criteria in relation to data collection modality.

Table 6.4 MCL and ACL injury in relation to inciting event and data collection modality.

	Total Participants	Total Player-Seasons	Pooled MCL and ACL Injury	MCL Injury	ACL Injury
2012 -13 to 2018-19 sample n					
Previous Injury History	111	173	37	28	9
Isokinetics	83	131	33	24	9
2014 -15 to 2018-19 sample n					
Contact Events	35	45	14	10	4
PlayerLoadTM	52	73	19	15	4
2015 -16 to 2018-19 sample n					
Biomechanics	49	63	22	17	5

6.4.1 Question 1. Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players?

Acute and chronic exposure to contact events in relation to pooled MCL and ACL injury sustained during contact events

Table 6.4.1a presents univariate analyses examining the effect of both acute and chronic exposure to tackle events in relation to the odds of sustaining an MCL or ACL injury where the inciting event was being tackled or tackling. Acute and chronic tackle exposure variables were not statistically associated with sustaining MCL and ACL injury ($P > 0.05$).

Table 6.4.1a Univariate generalized linear mixed-models examining the association between acute and chronic exposure to tackle events and pooled MCL and ACL injury sustained during the tackle.

Independent Variable	P value	Apparent AUC
Acute Number of Tackle Events Variables:		
7-Day EWMA (per + 1 Unit Increase)	0.69	0.95
Chronic Number of Tackle Events Variables:		
7-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.58	0.94
14-Day EWMA (per + 1 Unit Increase)	0.73	0.95
14-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.49	0.95
21-Day EWMA (per + 1 Unit Increase)	0.79	0.94
21-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.58	0.95
Acute Number of Tackle Events Relative to Game Minutes Played Variables:		
7-Day EWMA (per + 1 Unit Increase)	0.37	0.94
Chronic Number of Tackle Events Relative to Game Minutes Played Variables:		
7-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.32	0.94
14-Day EWMA (per + 1 Unit Increase)	0.71	0.95
14-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.26	0.95
21-Day EWMA (per + 1 Unit Increase)	0.93	0.94
21-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.30	0.95

Table 6.4.1b presents univariate analyses examining the effect of both acute and chronic exposure to tackle and ruck events in relation to the odds of sustaining an MCL or ACL injury where the inciting event was the tackle or the ruck. Both the 7-day and 14-day EWMA of the total number of tackle and ruck events were significantly associated with a decreased odds of sustaining an MCL or ACL injury during contact when modelled as a continuous variable and when and when not accounting for injury as a rare event ($P \leq 0.05$, OR 95% CI < 1 & ADASYN OR 95% CI < 1) (Table 6.4.1b; Figures 6.4.1a – 6.4.1b). However, when modelled as tertile groups there was no significant relationship with knee ligament injury for both variables ($P \geq 0.05$) (Table 6.4.1b).

Table 6.4.1b Univariate generalized linear mixed-models examining the association between acute and chronic exposure to tackle and ruck events and pooled MCL and ACL injury (Effects modelled as tertiles in italics).

Independent Variable	P value	OR (95% CI)	ADASYN OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)	ADASYN LOOCV AUC (95% CI)
Acute Number of Tackle and Ruck Events Variables:						
7-Day EWMA (per + 1 Unit Increase)	0.02*	0.80 (0.65 - 0.96)	0.83 (0.72 - 0.97)	0.96	0.61 (0.46 - 0.77)	0.67 (0.62 - 0.72)
7-Day EWMA (per tertile group):	*0.10*			*0.96*		
Chronic Number of Tackle and Ruck Events Variables:						
7-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.65			0.95		
14-Day EWMA (per + 1 Unit Increase)	0.04*	0.73 (0.46 - 0.97)	0.70 (0.54 - 0.91)	0.96	0.59 (0.44 - 0.75)	0.69 (0.64 - 0.74)
14-Day EWMA (per tertile group):	*0.23*			*0.96*		
14-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.70			0.95		
21-Day EWMA (per + 1 Unit Increase)	0.07			0.96		
21-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.78			0.94		
Acute Number of Tackle and Ruck Events Relative to Game Minutes Played Variables:						
7-Day EWMA (per + 1 Unit Increase)	0.61			0.95		
Chronic Number of Tackle and Ruck Events Relative to Game Minutes Played Variables:						
7-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.34			0.95		
14-Day EWMA (per + 1 Unit Increase)	0.38			0.94		
14-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.35			0.95		
21-Day EWMA (per + 1 Unit Increase)	0.33			0.95		
21-Day EWMA with a 7 - Day Lag (per + 1 Unit Increase)	0.39			0.95		

*** denotes statistical significance (P ≤ 0.05)**

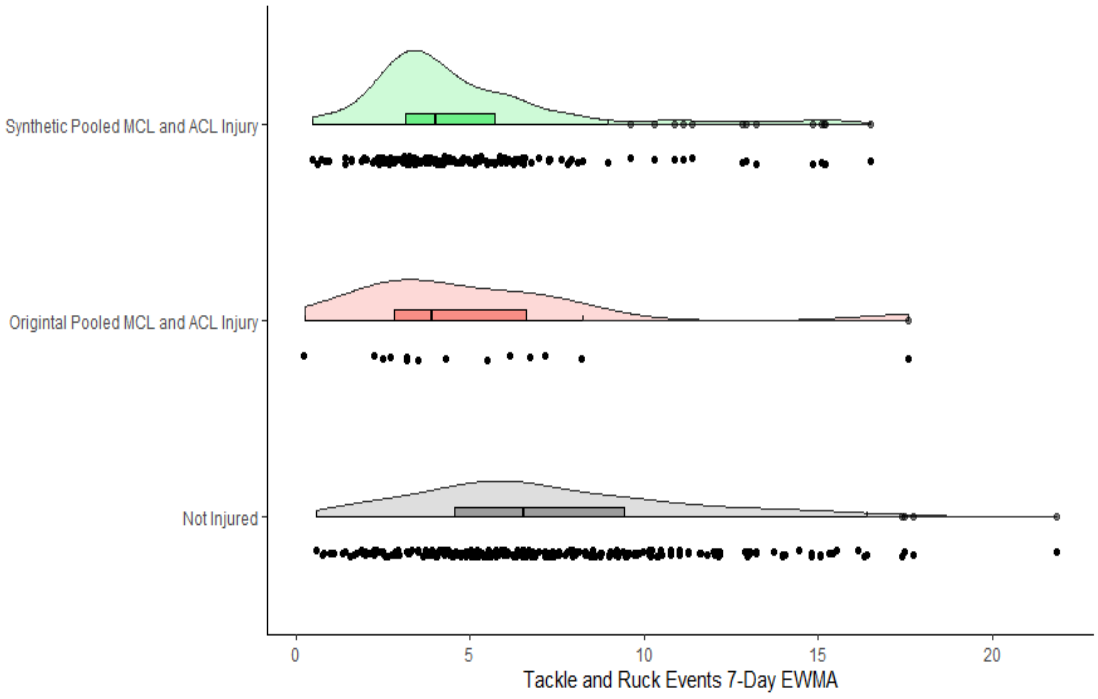


Figure 6.4.1a Distribution of the 7-day EWMA total number of contact events for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated MCL and ACL injuries (shaded in green).

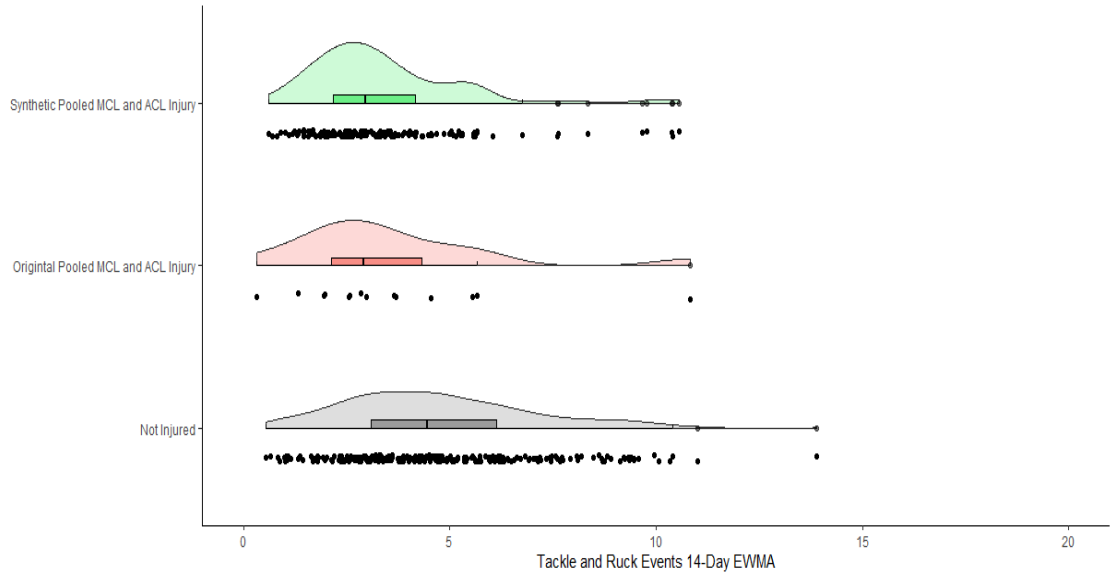


Figure 6.4.1b Distribution of the 14-day EWMA total number of contact events for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated MCL and ACL injuries (shaded in green).

Acute or chronic exposure to tackle and ruck events relative to game minutes played were not statistically associated with sustaining MCL and ACL injury (Table 6.4.1b). When examining tackle and ruck events in relation to game minutes played (Figure Table 6.4.1c), injuries are sustained throughout the duration of match-play when players are exposed to a small number of contact events as well as a large number of contact events.

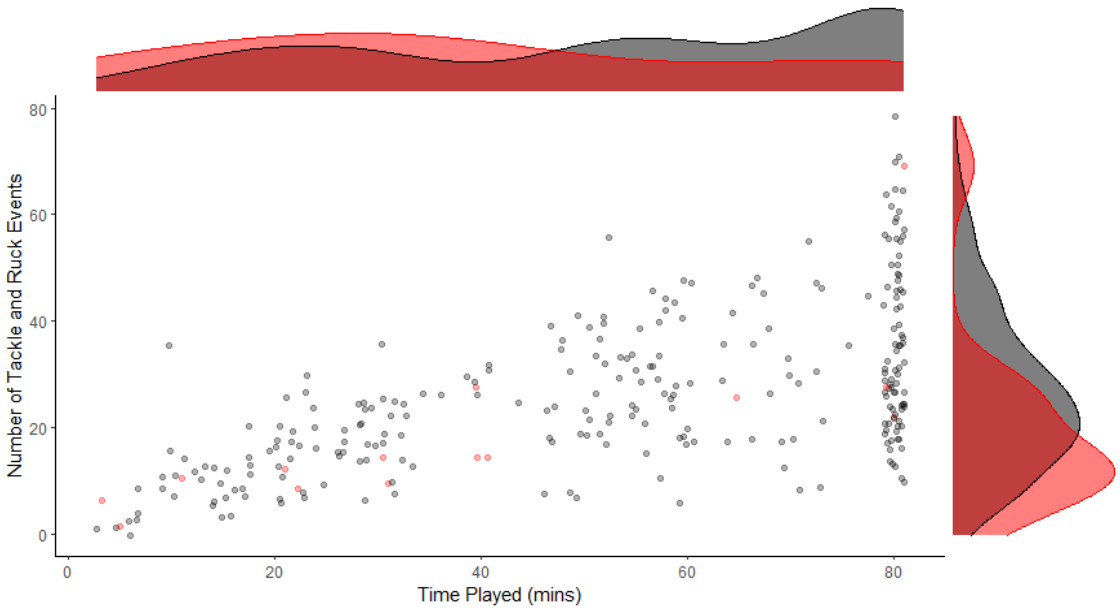


Figure 6.4.1c Scatterplot and distribution of the total number of tackle and ruck events experienced by players vs game minutes played. Non-injured players are shaded grey, players sustaining MCL or ACL injury are shaded in red.

6.4.2 Question 2. Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players?

6.4.2.1 Question 2.i. Is previous lower limb injury history associated with the risk of sustaining an MCL or ACL injury from a contact event?

Table 6.4.2.1a presents results from univariate analyses that examined the risk of sustaining MCL injury in relation to previous injury history variables. Sustaining a previous hamstring injury or a previous calf injury were both associated with an increased odds of sustaining MCL injury ($P \leq 0.05$, OR 95% CI > 1) (Table 6.4.2.1a; Figure 6.4.2.1a – 6.4.2.1b). However, a previous ACL or MCL injury was not statistically associated with sustaining MCL injury ($P > 0.05$).

Table 6.4.2.1a Univariate generalized linear mixed-models examining the association between previous injury history and MCL injury.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previoius Hamstring Injury History (per injury)	0.001*	4.25 (1.95 - 9.25)	0.98	0.68 (0.53 - 0.83)
Previoius ACL Injury History (per injury)	0.12		0.99	
Previoius MCL Injury History (per injury)	0.83		0.98	
Previoius Calf Injury History (per injury)	0.03*	3.24 (1.21 - 8.65)	0.99	0.71 (0.56 - 0.85)

*** denotes statistical significance (P ≤ 0.05).**

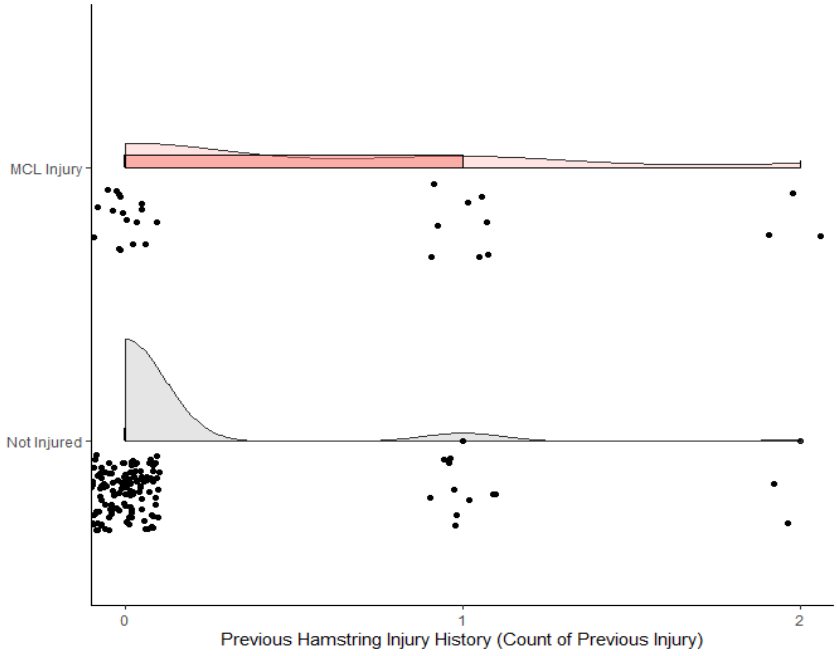


Figure 6.4.2.1a Distribution of previous hamstrings muscle group injury for injury free players (shaded in grey) and players with MCL injuries sustained during contact events (shaded in red).

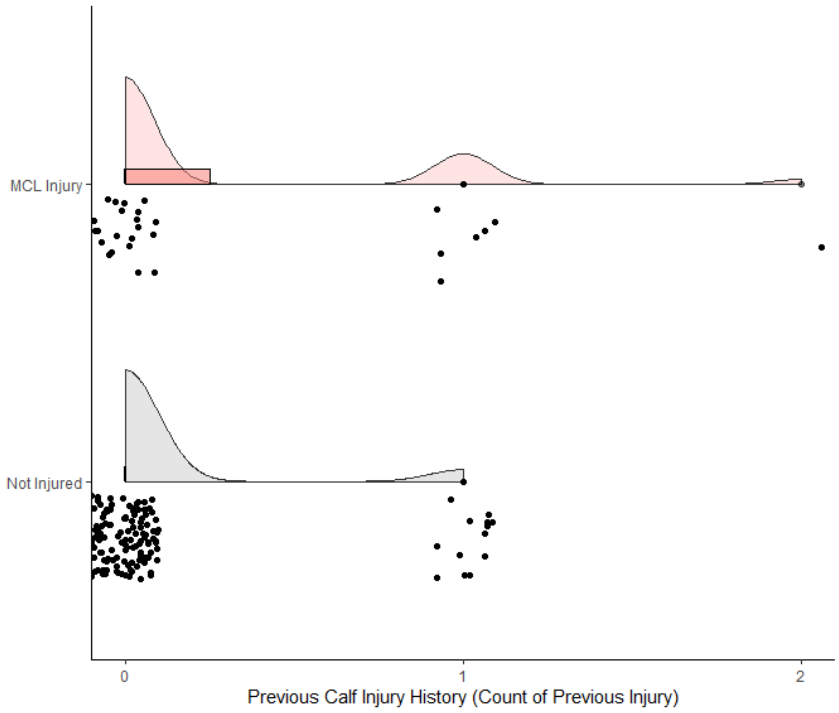


Figure 6.4.2.1b Distribution of previous calf muscle group injury for injury free players (shaded in grey) and players with MCL injuries sustained during contact events (shaded in red).

Table 6.4.2.1b presents results from univariate analyses that examined the risk of sustaining pooled MCL and ACL injury in relation to previous injury history. Sustaining a previous hamstring injury or calf injury were both associated with an increased odds of sustaining pooled knee ligament injury ($P \leq 0.05$, OR 95% CI > 1) (Table 6.4.2.1b; Figure 6.4.2.1c – 6.4.2.1d); whereas a previous ACL or MCL injury was not statistically associated with sustaining pooled knee ligament injury ($P > 0.05$).

Table 6.4.2.1b Univariate generalized linear mixed-models examining the association between previous injury history and pooled MCL and ACL injury.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Previoius Hamstring Injury History (per injury)	0.001*	4.13 (1.96 - 8.71)	0.97	0.35 (0.20 - 0.50)
Previoius ACL Injury History (per injury)	0.26		0.98	
Previoius MCL Injury History (per injury)	0.08		0.96	
Previoius Calf Injury History (per injury)	0.02*	2.98 (1.17 - 7.59)	0.98	0.70 (0.57 - 0.82)

*** denotes statistical significance (P ≤ 0.05).**

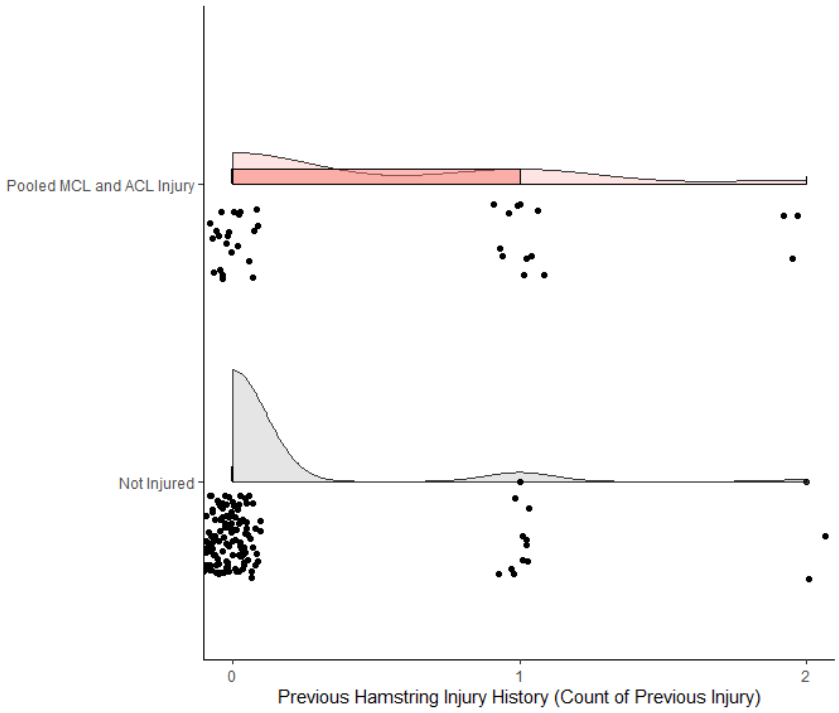


Figure 6.4.2.1c Distribution of previous hamstrings muscle group injury for injury free players (shaded in grey) and players with pooled MCL and ACL injuries sustained during contact events (shaded in red).

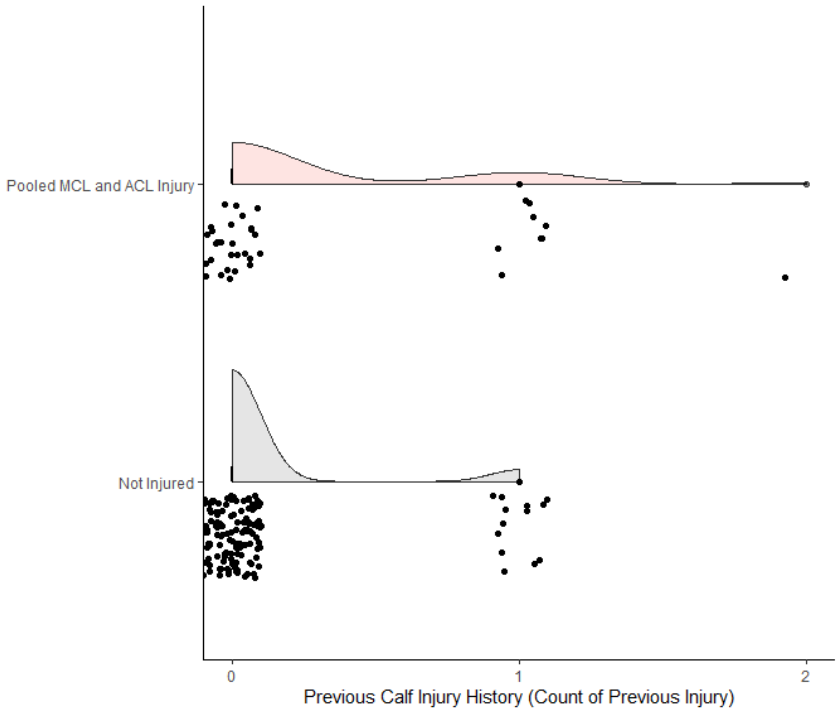


Figure 6.4.2.1d. Distribution of previous calf muscle group injury for injury free players (shaded in grey) and players with pooled MCL and ACL injuries sustained during contact events (shaded in red).

6.4.2.2 Question 2.ii. Are previously identified isokinetic knee strength variables associated with the risk of sustaining MCL or ACL injury within the cohort of male professional rugby union players?

Table 6.4.2.2a presents results from univariate analyses that examined the risk of sustaining MCL injury in relation to isokinetic strength variables. No Isokinetic variable was associated with MCL injury sustained during contact events ($P > 0.05$).

Table 6.4.2.2a Univariate generalized linear mixed-models examining the association between isokinetic knee extension and flexion strength and MCL injury.

Independent Variable	P value	OR (95% CI)	Apparent AUC
Isokinetic Knee Extension Moment Variables:			
Absolute peak concentric knee extension torque at 60°/s (per + 1 N.m)	0.69		0.53
Peak concentric knee extension torque relative to body mass at 60°/s (per + 1 N.m/kg)	0.50		0.56
Absolute peak concentric knee extension torque at 180°/s (per + 1 N.m)	0.10		0.62
Peak concentric knee extension torque relative to body mass at 180°/s (per + 1 N.m/kg)	0.17		0.55
Isokinetic Knee Flexion Moment Variables:			
Absolute peak concentric knee flexion torque at 60°/s (per + 1 N.m)	0.72		0.59
Peak concentric knee flexion torque relative to body mass at 60°/s (per + 1 N.m/kg)	0.85		0.48
Absolute peak concentric knee flexion torque at 180°/s (per + 1 N.m)	0.14		0.63
Peak concentric knee flexion torque relative to body mass at 180°/s (per + 1 N.m/kg)	0.31		0.57

Table 6.4.2.2b presents results from univariate analyses the association between sustaining pooled MCL and ACL injury and isokinetic strength variables. Absolute peak concentric knee extension torque at 180°/s was significantly associated with sustaining ACL and MCL injury when modelled as a continuous variable ($P \leq 0.05$) (Figure 6.4.2.2a), and groups as tertiles. When modelled as a tertile ($P \leq 0.05$), moderate magnitudes were associated with a reduced odds of injury (OR 95% CI < 1), whereas higher magnitudes were associated with an increased odds of injury (OR 95% CI > 1). Lower magnitudes of Absolute peak concentric knee extension torque at 180°/s were observed to be inconclusive (lower OR 95% CI < 1 & upper OR 95% CI > 1) (Table 6.4.2.2b).

Table 6.4.2.2b Univariate generalized linear mixed-models examining the association between isokinetic strength and pooled MCL and ACL injury.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Isokinetic Knee Extension Moment Variables:				
Absolute peak concentric knee extension torque at 60°/s (per + 1 N.m)	0.91		0.66	
Peak concentric knee extension torque relative to body mass at 60°/s (per + 1 N.m/kg)	0.75		0.66	
Absolute peak concentric knee extension torque at 180°/s (per + 1 N.m)	0.03*	1.01 (1.00 - 1.03)	0.62	0.57 (0.45 - 0.70)
Absolute peak concentric knee extension torque at 180°/s (per tertile group):	0.01*		0.63	0.51 (0.39 - 0.63)
Low: 95 - 167 N.m		*1.4 (0.29 - 6.81)*		
Reference - Medium: 167.1 - 199 N.m		*0.07 (0.02 - 0.3)*		
High: 199.1 - 268 N.m		*7.68 (1.56 - 37.73)*		
Peak concentric knee extension torque relative to body mass at 180°/s (per + 1 N.m/kg)	0.08		0.58	
Isokinetic Knee Flexion Moment Variables:				
Absolute peak concentric knee flexion torque at 60°/s (per + 1 N.m)	0.54		0.63	
Peak concentric knee flexion torque relative to body mass at 60°/s (per + 1 N.m/kg)	0.93		0.67	
Absolute peak concentric knee flexion torque at 180°/s (per + 1 N.m)	0.08		0.63	
Peak concentric knee flexion torque relative to body mass at 180°/s (per + 1 N.m/kg)	0.20		0.60	

*** denotes statistical significance (P ≤ 0.05)**

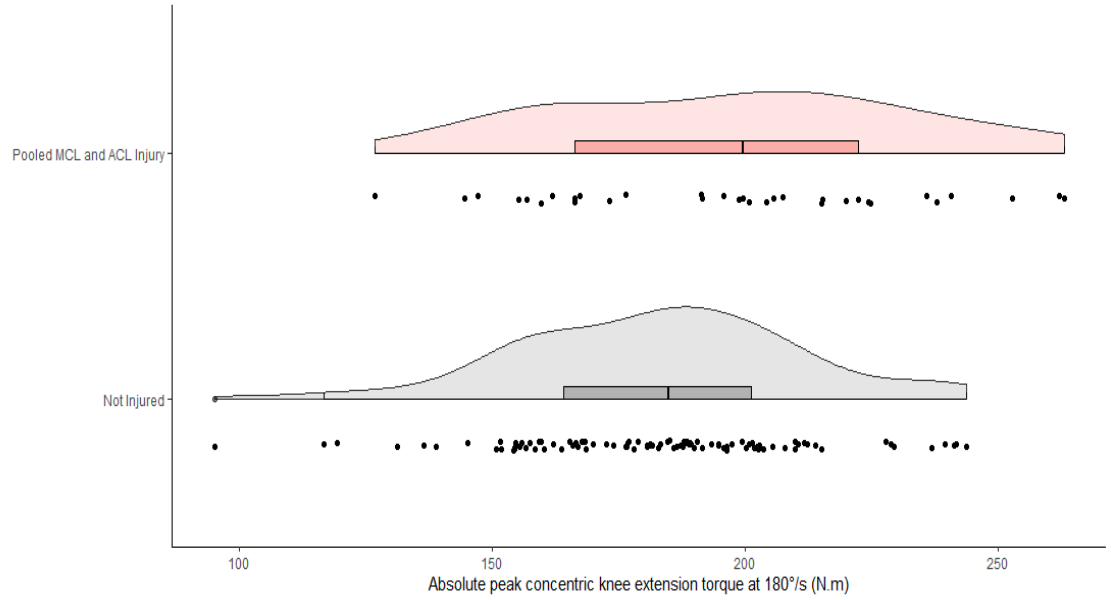


Figure 6.4.2.2a Distribution of Absolute peak concentric knee extension torque at 180°/s for injury free players (shaded in grey) and players with pooled MCL and ACL injuries sustained during contact events (shaded in red).

6.4.2.3 Question 2.iii. Are lower limb biomechanical variables during the early ground contact phase of a single-leg drop-jump task associated with the risk of sustaining MCL and ACL injury within the cohort of male professional rugby union players?

Table 6.4.2.3a presents results from univariate analyses which examined the association between lower limb sagittal plane internal joint moments generated during the early stage of single leg ground contact and the odds of sustaining MCL injury. No sagittal plane variables were statistically associated with MCL injury sustained during contact events when modelled as continuous variables ($P > 0.05$).

Table 6.4.2.3a Univariate generalized linear mixed-models examining the association between early ground contact limb sagittal plane internal joint moments and MCL injury.

Independent Variable	P value	OR (95% CI)	Apparent AUC
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.74		0.55
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.44		0.51
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.76		0.53
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.72		0.54
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.17		0.61
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.15		0.63
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.45		0.59
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.43		0.44
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.80		0.53
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.61		0.56
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.65		0.53
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.90		0.53

Table 6.4.2.3b presents results from univariate analyses which examined the association between the odds of sustaining an ACL or MCL injury during contact events and lower limb sagittal plane internal joint moments generated during the early stage of single leg ground contact. No sagittal plane variables were associated with ACL or MCL injury ($P > 0.05$).

Table 6.4.2.3b Univariate generalized linear mixed-models examining the association between early ground contact limb sagittal plane internal joint moments and pooled MCL and ACL injury.

Independent Variable	P value	OR (95% CI)	Apparent AUC
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.54		0.56
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.98		0.54
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.89		0.53
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.99		0.52
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.55		0.57
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.38		0.58
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.69		0.50
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.92		0.49
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.99		0.51
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.59		0.57
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.51		0.55
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.71		0.51

Table 6.4.2.3c presents results from univariate analyses that examined the association between the odds of sustaining MCL injury and lower limb frontal plane joint moments generated during the early stage of single leg ground contact. When modelled as continuous variables, increased magnitudes of frontal plane joint moments generated at the hip and knee were significantly associated with increased odds of sustaining an MCL injury during contact events ($P \leq 0.05$, OR 95% CI > 1) (Figure 6.4.2.3a). When modelled as tertile variables, moderate magnitudes of frontal plane hip moment were associated with a decreased odds of MCL injury (OR 95% CI < 1), whereas higher magnitudes were associated with increased odds of injury (OR 95% CI > 1). Lower magnitudes of frontal plane hip moment displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), therefore the effect was inconclusive. Frontal plane knee joint moment modelled as tertile groups were not significantly associated with MCL injury ($P \geq 0.05$).

Table 6.4.2.3c Univariate generalized linear mixed-models examining the association between early ground contact limb frontal plane external joint moments and MCL injury (Effects modelled as tertiles in italics).

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.005*	5.62 (1.5 - 21.09)	0.99	0.65 (0.48 - 0.83)
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 50 ms (per tertile group):	*0.003**		0.99	*0.62 (0.45 - 0.79)*
Low: 0.10 - 1.15 N.m/kg		*0.66 (0.13 - 3.45)*	0.99	
Reference - Medium: 1.151 - 1.62 N.m/kg		*0.27 (0.09 - 0.80)*	0.99	
High: 1.621 - 2.00 N.m/kg		*4.17 (1.00 - 17.31)*	0.99	
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.87		0.99	
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.69		0.99	
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.84		0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.001*	3.58 (1.18 - 12.11)	0.99	0.71 (0.57 - 0.85)
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 50 ms (per tertile group):	*0.23*		0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.08		0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.10		0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.53		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.16		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.38		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.26		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.39		0.99	

*** denotes statistical significance (P ≤ 0.05)**

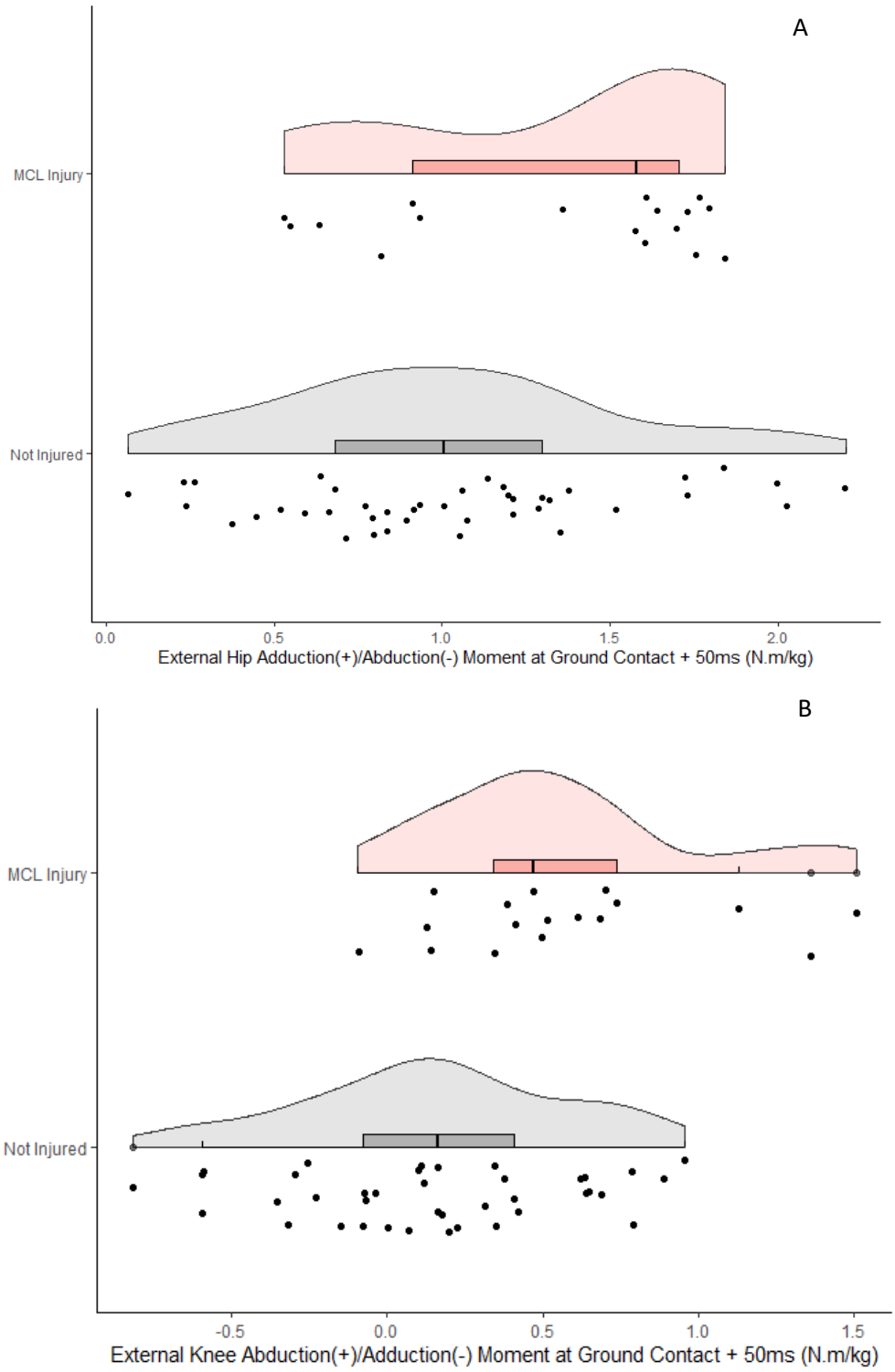


Figure 6.4.2.3a Distribution of A. External hip adduction moment and B. External knee abduction moment for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red).

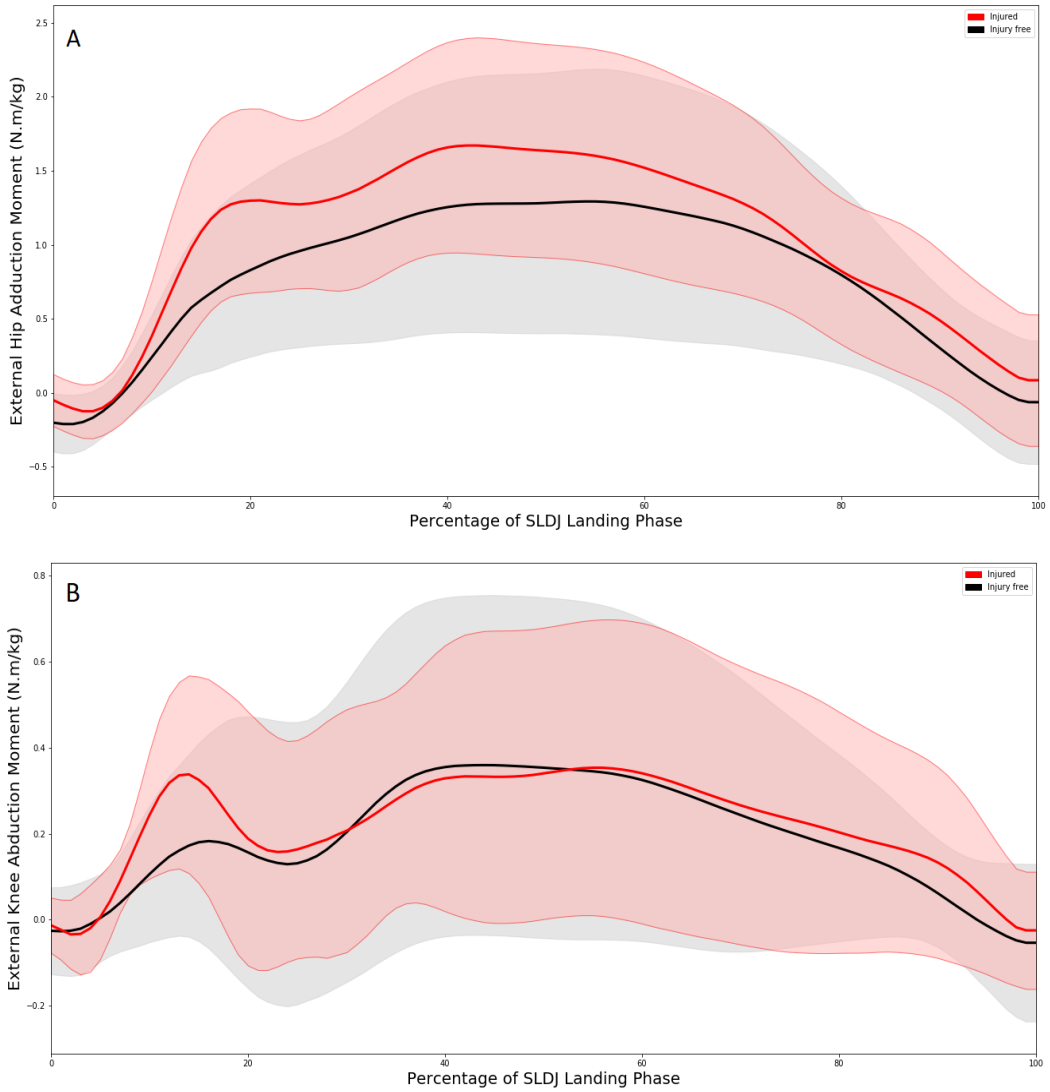


Figure 6.4.2.3b Mean and 95% CI External A) Hip Adduction and B) Knee abduction moment during the landing phase of a single-leg drop jump for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red).

Table 6.4.2.3d presents results from univariate analyses which examined the association between the odds of sustaining MCL or ACL injury and lower limb frontal plane joint moments generated during the early stage of single leg ground contact. When modelled as continuous variables, increased magnitudes of frontal plane joint moments generated at the hip and knee were significantly associated with increased odds of MCL or ACL injury sustained during contact events ($P \leq 0.05$, OR 95% CI > 1) (Figure 6.4.2.3c). When modelled as tertile variables, both higher magnitudes of frontal plane hip and knee moment were associated with increased odds of injury (OR 95% CI > 1). Moderate magnitudes of frontal plane hip moment were associated with a decreased odds of sustaining a MCL or ACL injury (OR 95% CI < 1), whereas moderate magnitudes

of frontal plane knee moment displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), therefore the effect was inconclusive. Lower magnitudes of frontal plane hip moment displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), so were inconclusive in relation to injury association. Lower magnitudes of frontal plane knee moment were associated with decreased odds of MCL or ACL injury (OR 95% CI < 1).

Table 6.4.2.3d Univariate generalized linear mixed-models examining the association between early ground contact limb frontal plane external joint moments and with pooled MCL and ACL injuries (Effects modelled as tertiles in italics).

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.006*	4.66 (1.42 - 15.27)	0.99	0.63 (0.48 - 0.79)
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 50 ms (per tertile group):	*0.003**		0.99	*0.56 (0.38 - 0.73)*
Low: 0.10 - 1.12		*0.66 (0.13 - 3.45)*	0.99	
Reference - Medium: 1.121 - 1.52		*0.27 (0.09 - 0.73)*	0.99	
High: 1.521 - 2.83		*4.17 (1.00 - 17.31)*	0.99	
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.91		0.99	
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.51		0.99	
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.82		0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.02*	4.56 (1.29 - 16.14)	0.99	0.64 (0.50 - 0.79)
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 50 ms (per tertile group):	*0.005**		0.99	*0.42 (0.27 - 0.57)*
Low: 0.082 - 0.119 N.m/kg		*0.13 (0.02 - 0.68)*	0.99	
Reference - Medium: 0.12 - 0.50 N.m/kg		*0.83 (0.36 - 1.93)*	0.99	
High: 0.51 - 1.51 N.m/kg		*1.20 (0.36 - 4.04)*	0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.68		0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.72		0.99	
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.91		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.19		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.38		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.38		0.99	
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.79		0.99	

*** denotes statistical significance (P ≤ 0.05)**

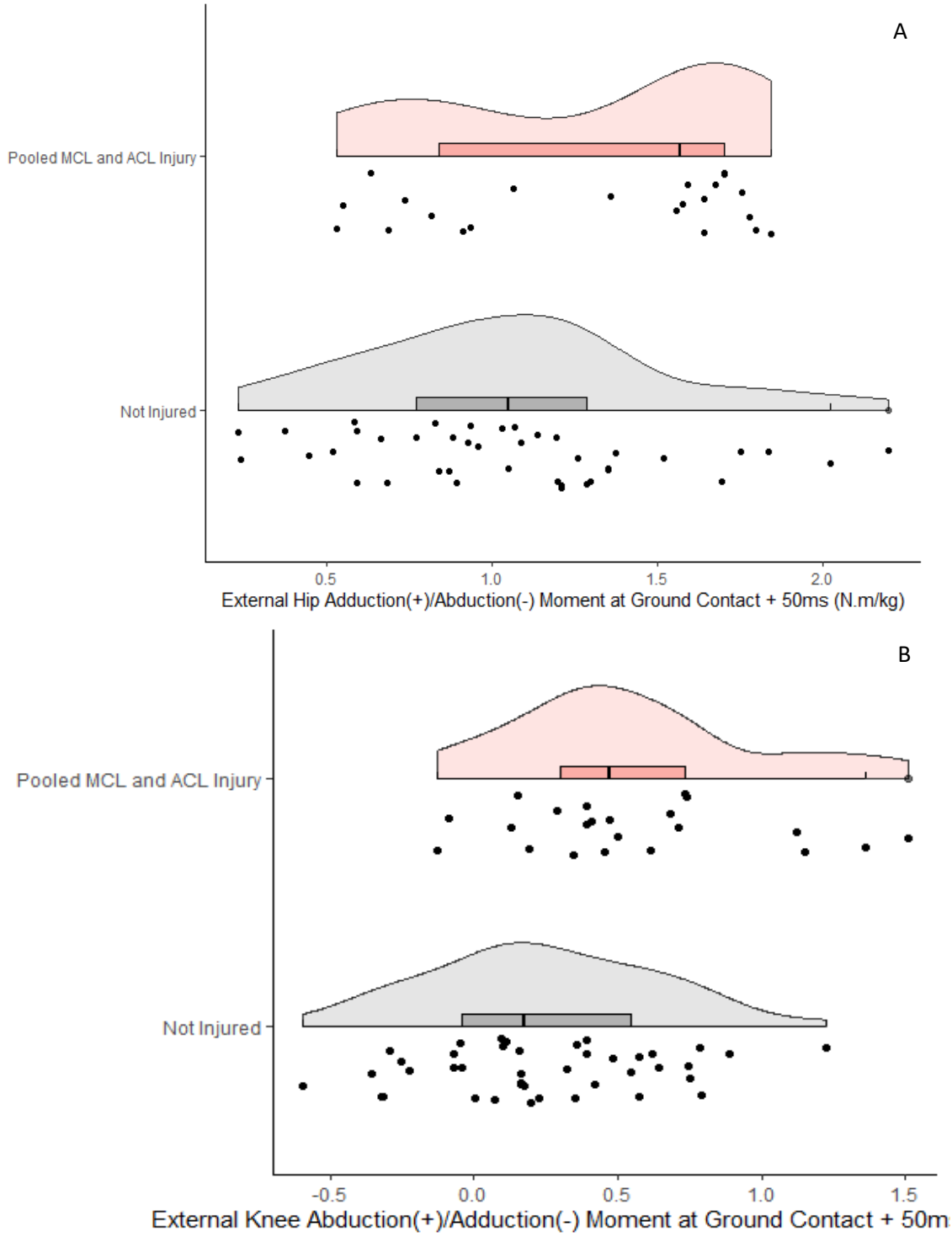


Figure 6.4.2.3c Distribution of A. External hip adduction moment and B. External knee adduction moment for injury free player-days (shaded in grey), and pooled MCL and ACL injuries sustained during contact events (shaded in red).

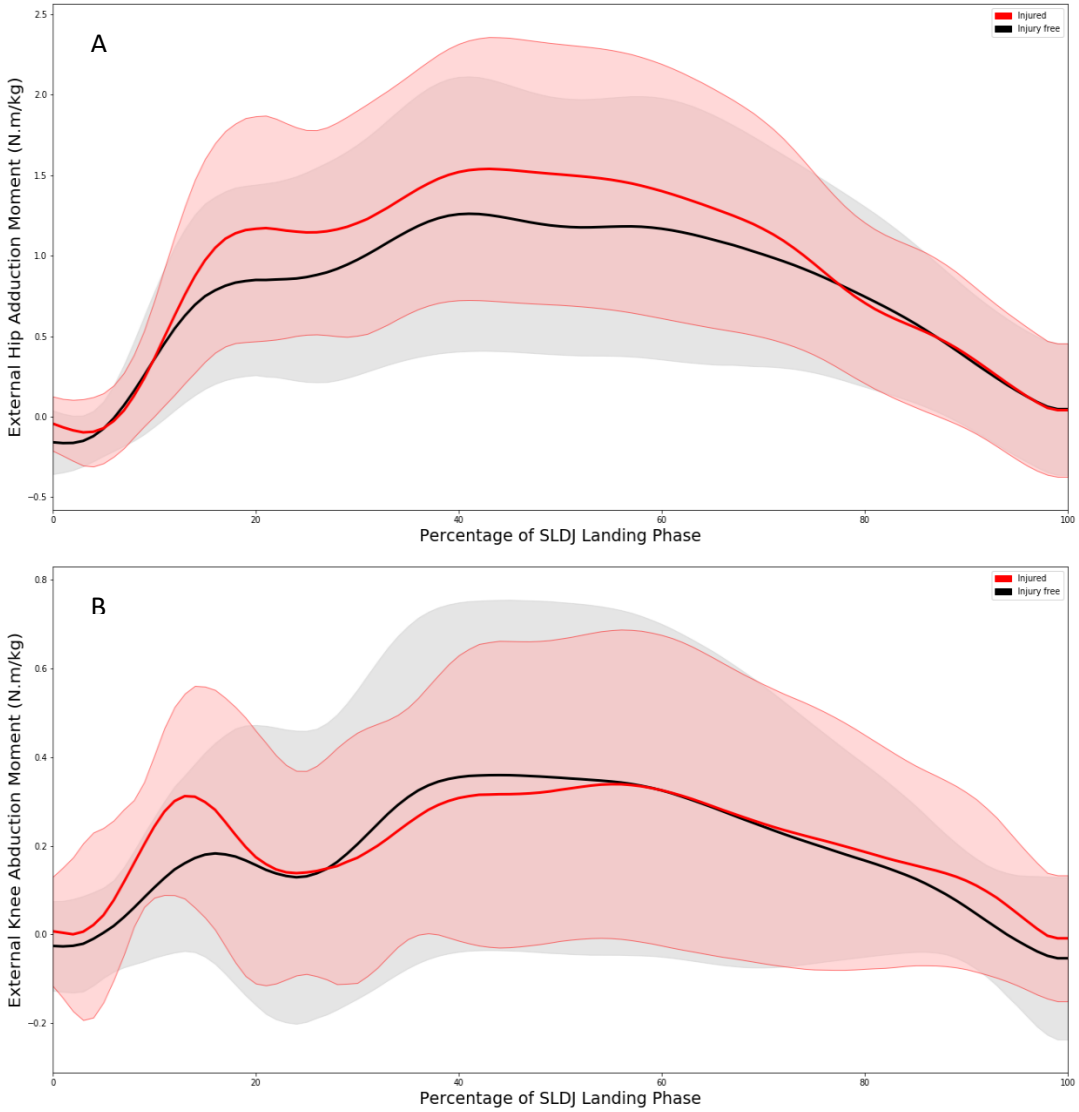


Figure 6.4.2.3d Mean and 95% CI External A) Hip Adduction and B) Knee abduction moment during the landing phase of a single-leg drop jump for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red).

6.4.3 Question 3. Is exposure to on-pitch physical activity and related to an increased risk of knee ligament injury for male professional rugby union players?

Acute exposure to on-pitch physical activity in relation to MCL injury and pooled MCL and ACL injury sustained during contact events

Table 6.4.3a presents univariate analyses examining the relationship between sustaining an MCL injury and of acute exposure to on-pitch physical activity (PlayerLoad^{TM}). Larger magnitudes of

all acute PlayerLoad™ variables were associated with small increases in the odds of sustaining MCL injury when modelled as continuous variables and when and when not accounting for injury as a rare event ($P \leq 0.05$, OR 95% CI > 1 & ADASYN OR 95% CI > 1) (Figure 6.4.3a). However, when modelled as tertile groups, despite both 3- and 7-Day EWMA variables being statistically associated with MCL injury ($P \leq 0.05$), only lower magnitudes of 7-Day EWMA were associated with decreased odds of sustaining MCL injury when not accounting for the injury class as a rare event (OR 95% CI < 1 & ADASYN OR 95% CI < 1). All other tertile groups displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), so were inconclusive in relation to injury association.

Table 6.4.3a Univariate generalized linear mixed-models examining the association between acute exposure to on-pitch physical activity and MCL injury sustained during contact events.

Independent Variable	P value	OR (95% CI)	ADASYN OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)	ADASYN LOOCV AUC (95% CI)
Acute PlayerLoadTM Variables:						
3-Day EWMA (per + 1 AU)	0.03*	1.01 (1.00 - 1.01)	1.01 (1.00 - 1.01)	0.96	0.56 (0.39 - 0.73)	0.68 (0.65 - 0.70)
3-Day EWMA (per tertile group):	*0.05**			*0.97*	*0.32 (0.27 - 0.38)*	*0.64 (0.61 - 0.67)*
Low: 12 - 210 AU		*0.14 (0.02 - 1.18)*	*0.13 (0.04 - 1.24)*			
Reference - Medium: 210.1 - 303 AU		*0 (0 - 0.02)*	*0 (0 - 0.02)*			
High: 303.1 - 875 AU		*1.04 (0.32 - 3.42)*	*0.54 (0.21 - 1.57)*			
7-Day EWMA (per + 1 AU)	0.02*	1.01 (1.00 - 1.02)	1.01 (1.00 - 1.02)	0.97	0.59 (0.45 - 0.72)	0.66 (0.64 - 0.69)
7-Day EWMA (per tertile group):	0.02*			0.97	0.49 (0.33 - 0.64)	0.63 (0.60 - 0.66)
Low: 27 - 187 AU		0.15 (0 - 0.77)	0.10 (0.06 - 0.92)			
Reference - Medium: 187.1 - 246 AU		0 (0 - 0.02)	0 (0 - 0.02)			
High: 246.1 - 520 AU		1.72 (0.53 - 5.99)	1.28 (0.55 - 3.00)			

*** denotes statistical significance (P ≤ 0.05)**

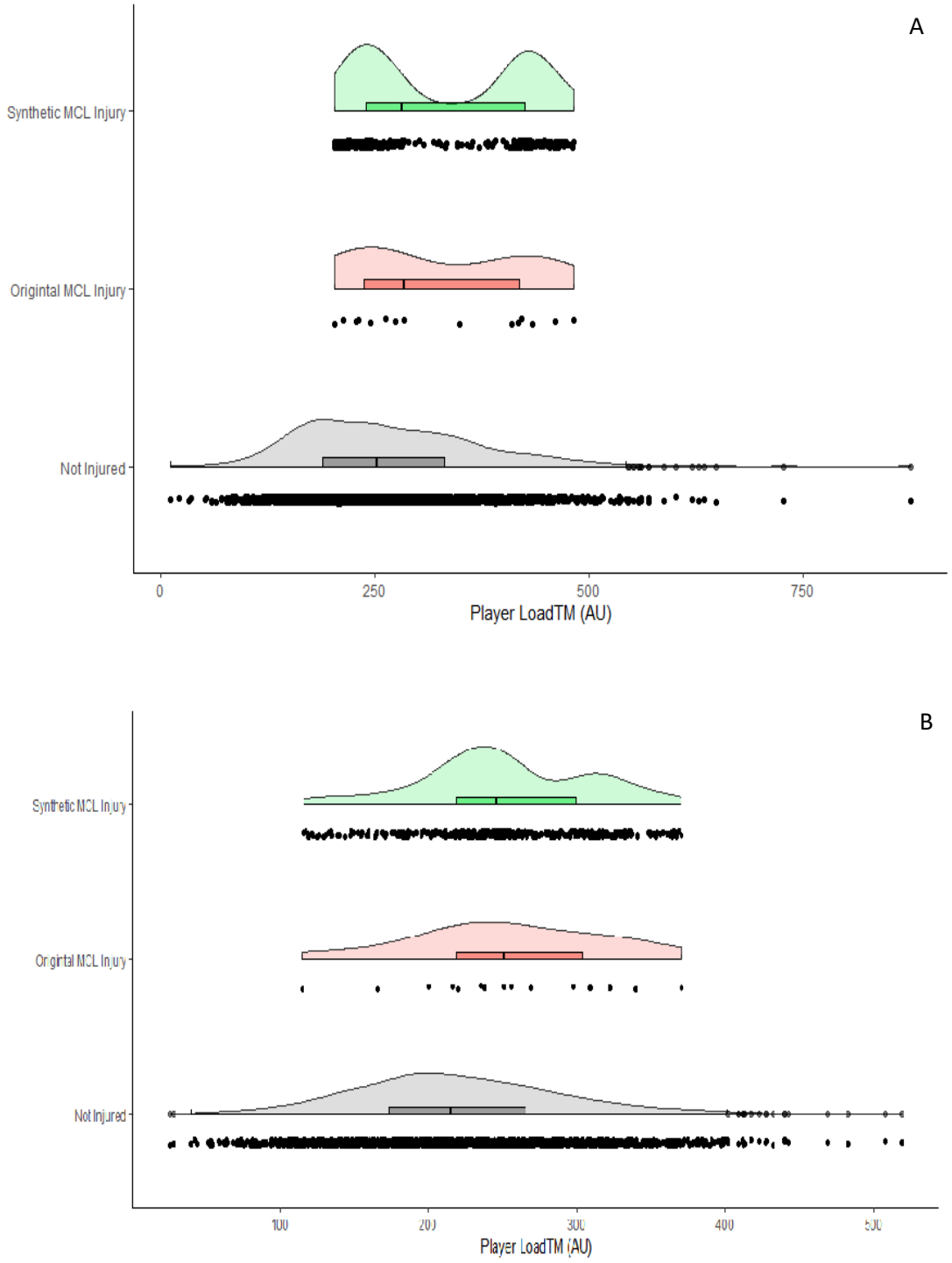


Figure 6.4.3a Distribution of the A. 3-day and B. 7-day EWMA of Player LoadTM for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red) and ADASYN generated MCL injuries (shaded in green).

Table 6.4.3b presents univariate analyses examining the effect of acute exposure to Rugby Activity in relation to pooled MCL and ACL injury, 7-Day EWMA were statistically associated with

sustaining MCL or ACL injury ($P \leq 0.05$). When modelled as a continuous variable, larger magnitudes of 7-Day EWMA PlayerLoad™ were associated with small increases in the odds of sustaining MCL or ACL injury when and when not accounting for the injury as a rare event ($P \leq 0.05$, OR 95% CI ≥ 1 & ADASYN OR 95% CI ≥ 1) (Figure 6.4.3b). When modelled as tertile groups, 7-Day EWMA PlayerLoad™ was not statistically associated with sustaining an MCL or ACL injury ($P \leq 0.05$).

Table 6.4.3b Univariate generalized linear mixed-models examining the association between acute exposure to Rugby Activity and pooled MCL and ACL injury.

Independent Variable	P value	OR (95% CI)	ADASYN OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)	ADASYN LOOCV AUC (95% CI)
Acute PlayerLoadTM Variables:						
3-Day EWMA (per +1 AU)	0.13			0.95		
7-Day EWMA (per +1 AU)	0.05*	1.01 (1.00 - 1.01)	1.01 (1.00 - 1.02)	0.96	0.61 (0.49 - 0.73)	0.60 (0.58 - 0.62)
7-Day EWMA (per tertile group):	*0.13*			*0.96*		

*** denotes statistical significance (P ≤ 0.05)**

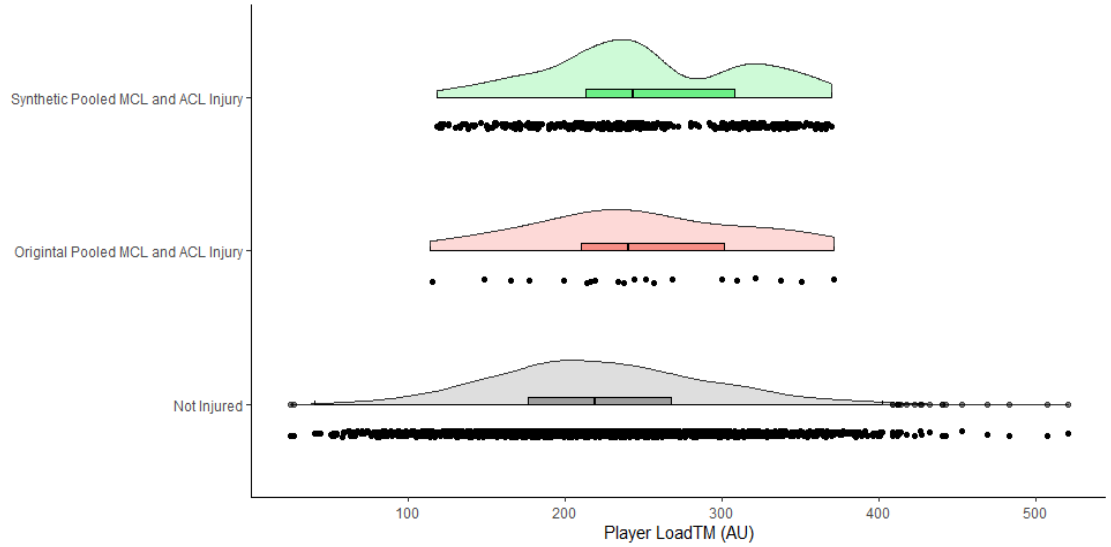


Figure 6.4.3b Distribution of the 7-day EWMA of PlayerLoad™ for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated pooled MCL and ACL injuries (shaded in green).

Chronic exposure to on-pitch physical activity in relation to MCL injury and pooled MCL and ACL injury sustained during contact events

Table 6.4.3c presents univariate analyses examining the relationship between sustaining an MCL injury and of chronic exposure to on-pitch physical activity (PlayerLoad™). Higher magnitudes of PlayerLoad™ experienced within both 3-10 days (7-day EMWA with 3 day lag), 14 days (14-day EMWA) and 3-17 days (14-day EMWA with 3 day lag) prior to injury were associated with small increases in the odds of sustaining MCL injury when modelled as continuous variables and when and when not accounting for injury as a rare event ($P \leq 0.05$, OR 95% CI ≥ 1 & ADASYN OR 95% CI ≥ 1) (Figure 6.4.3c).

Table 6.4.3c Univariate generalized linear mixed-models examining the association between chronic exposure to on-pitch physical activity (PlayerLoad™) and MCL injury.

Independent Variable	P value	OR (95% CI)	ADASYN OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)	ADASYN LOOCV AUC (95% CI)
Chronic PlayerLoadTM Variables:						
7-Day EWMA with a 3-Day Lag (per + 1AU)	0.01*	1.01 (1.00 - 1.02)	1.01 (1.00 - 1.02)	0.96	0.62 (0.47 - 0.78)	0.67 (0.64 - 0.70)
7-Day EWMA with a 3 Day Lag (per tertile group):	*0.002**			*0.97*	*0.55 (0.43 - 0.67)*	0.64 (0.61 - 0.67)
Low: 0 - 145 AU		*0.24 (0.01 - 1.94)*	*0.23 (0.14 - 1.18)*			
Reference - Medium: 145.1 - 221 AU		*0 (0 - 0.01)*	*0 (0 - 0.01)*			
High: 221.1 - 427 AU		*4.99 (1.17 - 21.16)*	*5.01 (1.46 - 7.82)*			
7-Day EWMA with a 7-Day Lag (per + 1AU)	0.67			0.95		
14-Day EWMA (per + 1AU)	0.03*	1.01 (1.00 - 1.03)	1.02 (1.01 - 1.03)	0.97	0.44 (0.27 - 0.62)	0.60 (0.57 - 0.62)
14-Day EWMA (per tertile group):	*0.04**			*0.97*	*0.43 (0.27 - 0.59)*	*0.58 (0.56 - 0.61)*
Low: 21 - 175 AU		*0.29 (0.04 - 1.67)*	*0.12 (0.04 - 0.40)*			
Reference - Medium: 175.1 - 224 AU		*0 (0 - 0.01)*	*0 (0 - 0.01)*			
High: 224.1 - 421 AU		*2.72 (0.7 - 12.61)*	*0.97 (0.30 - 3.11)*			
14-Day EWMA with a 3-Day Lag (per + 1AU)	0.02*	1.01 (1.00 - 1.03)	1.02 (1.01 - 1.03)	0.96	0.50 (0.35 - 0.65)	0.59 (0.56 - 0.62)
14-Day EWMA with a 3-Day Lag (per tertile group):	*0.05**			*0.97*	*0.31 (0.23 - 0.39)*	*0.61 (0.58 - 0.64)*
Low: 0 - 156 AU		*0.22 (0 - 1.04)*	*0.15 (0.05 - 0.47)*			
Reference - Medium: 156.1 - 213 AU		*0 (0 - 0.01)*	*0 (0 - 0.01)*			
High: 213.1 - 365 AU		*1.96 (0.57 - 7.79)*	*1.76 (0.51 - 3.18)*			
14-Day EWMA with a 7-Day Lag (per + 1AU)	0.76			0.95		
21-Day EWMA (per + 1AU)	0.08	1.01 (1.00 - 1.03)		0.97		
21-Day EWMA with a 3-Day Lag (per + 1AU)	0.08			0.97		
21-Day EWMA with a 7-Day Lag (per + 1AU)	0.94			0.95		

*** denotes statistical significance (P ≤ 0.05)**

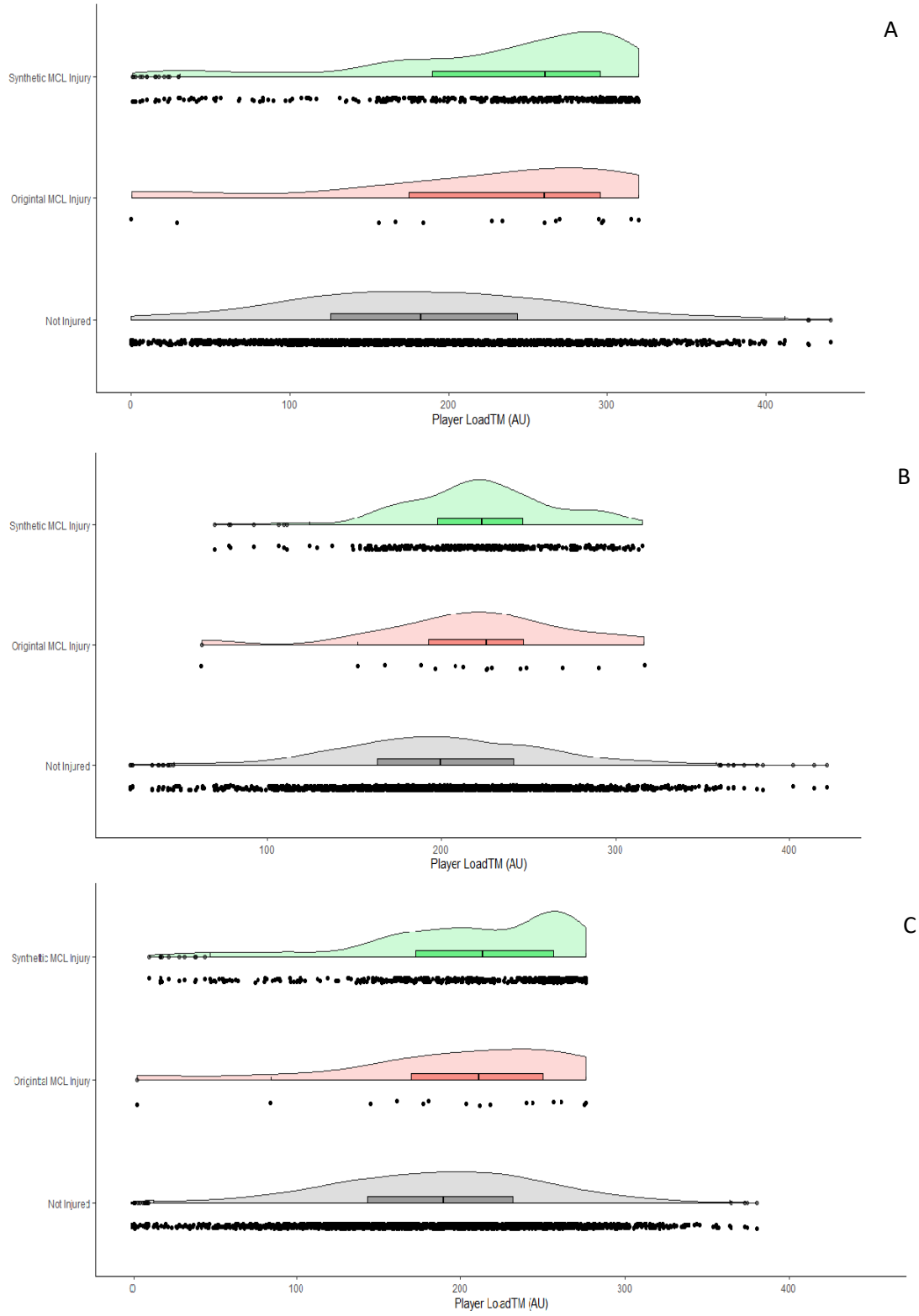


Figure 6.4.3c Distribution of the A. 7-day with 3-day lag, B. 14-day and C. 14-day with 3-day lag EWMA of Player Load™ for injury free player-days (shaded in grey), MCL injuries sustained during contact events (shaded in red) and ADASYN generated MCL injuries (shaded in green).

These variables (7-day EMWA with 3 day lag, 14-day EMWA & 14-day EMWA with 3 day lag) were also significantly associated with MCL injury when modelled as tertile groups ($P \leq 0.05$) (Table 6.4.3c). Higher magnitudes of 7-day EMWA PlayerLoad™ with a 3-day lag was associated with an increased odds of sustaining an MCL injury (OR 95% CI ≥ 1 & ADASYN OR 95% CI ≥ 1). However, higher magnitudes of 14-day EMWA PlayerLoad™ and 14-day EMWA PlayerLoad™ with a 3-day lag displayed non-unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), so were inconclusive in relation to the effect of injury association. Lower magnitudes of both 14-day and 14-day EMWA PlayerLoad™ with a 3-day were associated with a decreased odds of sustaining an MCL injury (OR 95% CI ≥ 1 & ADASYN OR 95% CI ≥ 1).

Table 6.4.3d presents univariate analyses examining the relationship between sustaining an MCL or ACL injury and of chronic exposure to on-pitch physical activity (PlayerLoad™). Larger magnitudes of PlayerLoad™ within both 3-10 days (7-day EMWA with 3-day lag) and 3-17 days (14-day EMWA with 3-day lag) prior to injury were associated with small increases in the odds of sustaining MCL or ACL injury when modelled as continuous variables and when and when not accounting for injury as a rare event ($P \leq 0.05$, OR 95% CI ≥ 1 & ADASYN OR 95% CI ≥ 1) (Figure 6.4.3d).

Table 6.4.3d Univariate generalized linear mixed-models examining the association between chronic exposure to Rugby Activity and pooled MCL and ACL injury.

Independent Variable	P value	OR (95% CI)	ADASYN OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)	ADASYN LOOCV AUC (95% CI)
Chronic PlayerLoadTM Variables:						
7-Day EWMA with a 3-Day Lag (per + 1AU)	0.0003*	1.01 (1.01 - 1.02)	1.01 (1.01 - 1.02)	0.96	0.69 (0.59 - 0.79)	0.72 (0.69 - 0.74)
7-Day EWMA with a 3 Day Lag (per tertile group):	*0.00004**			*0.96*	*0.66 (0.53 - 0.79)*	*0.77 (0.75 - 0.80)*
Low: 0 - 151 AU		*0.15 (0.00 - 0.85)*	*0.30 (0.19 - 1.34)*			
Reference - Medium: 151.1 - 225 AU		*0 (0 - 0.01)*	*0 (0 - 0.01)*			
High: 225.1 - 441 AU		*5.42 (2.13 - 16.53)*	*3.75 (1.23 - 11.45)*			
7-Day EWMA with a 7-Day Lag (per + 1AU)	0.85			0.95		
14-Day EWMA (per + 1AU)	0.06			0.96		
14-Day EWMA with a 3-Day Lag (per + 1AU)	0.01*	1.01 (1.01 - 1.02)	1.02 (1.01 - 1.03)	0.96	0.6 (0.48 - 0.72)	0.63 (0.60 - 0.65)
14-Day EWMA with a 3-Day Lag (per tertile group):	*0.02**			*0.97*	*0.57 (0.45 - 0.69)*	*0.66 (0.63 - 0.68)*
Low: 0 - 165 AU		*0.24 (0.00 - 0.71)*	*0.11 (0.07 - 0.97)*			
Reference - Medium: 165.1 - 223 AU		*0.00 (0.00 - 0.01)*	*0.00 (0.00 - 0.01)*			
High: 223.1 - 381 AU		*2.04 (0.82 - 5.43)*	*1.66 (0.56 - 4.89)*			
14-Day EWMA with a 7-Day Lag (per + 1AU)	0.85			0.95		
21-Day EWMA (per + 1AU)	0.15			0.96		
21-Day EWMA with a 3-Day Lag (per + 1AU)	0.06			0.96		
21-Day EWMA with a 7-Day Lag (per + 1AU)	0.66			0.95		

*** denotes statistical significance (P ≤ 0.05)**

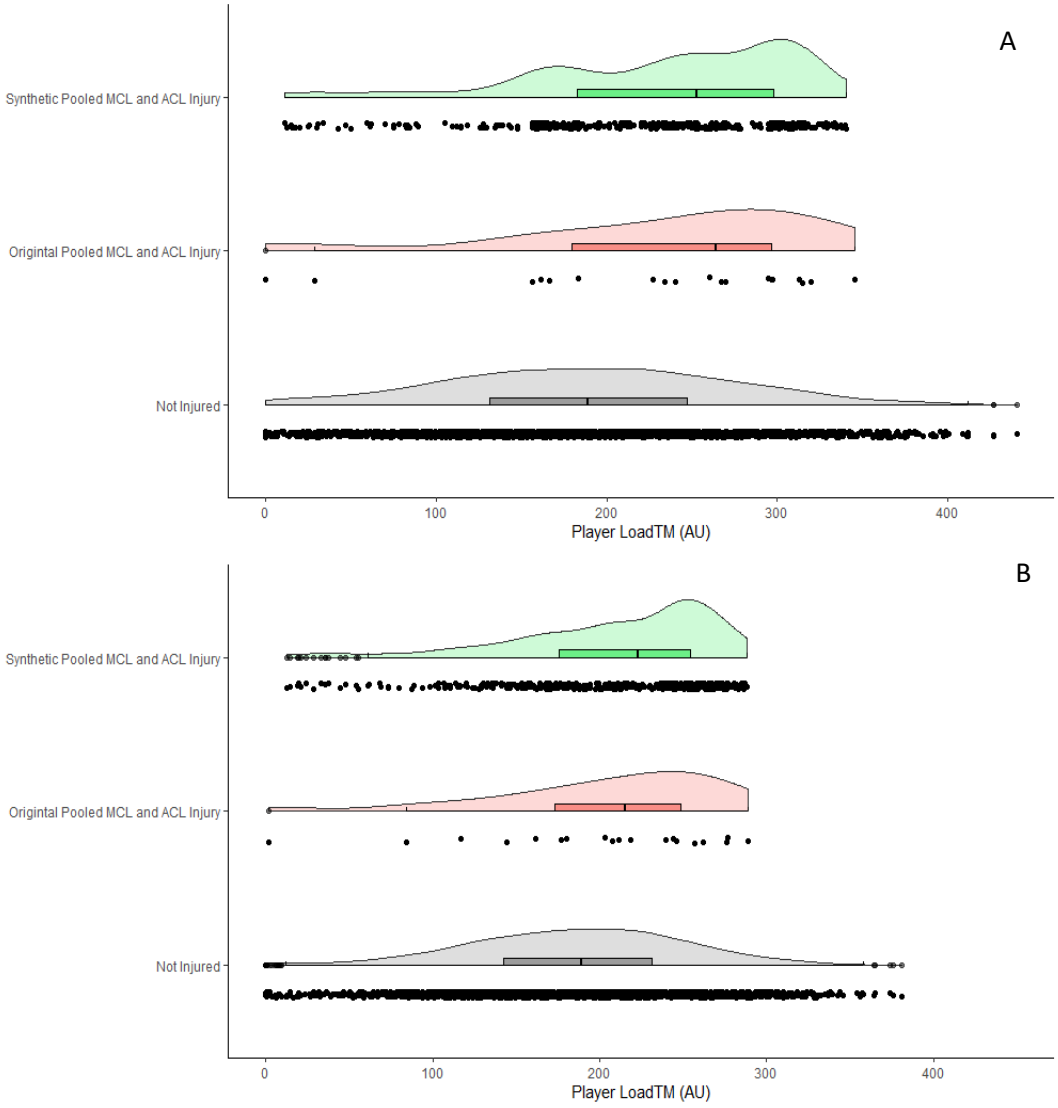


Figure 6.4.3d Distribution of the A. 7-day with 3-day lag, B. 14-day and C. 14-day with 3-day lag EWMA of Player Load™ for injury free player-days (shaded in grey), pooled MCL and ACL injuries sustained during contact events (shaded in red) and ADASYN generated pooled MCL and ACL injuries (shaded in green).

When modelled as tertile groups, 7-day EMWA with 3-day lag and 14-day EMWA with 3-day lag were significantly associated with sustaining an MCL or ACL injury ($P \leq 0.05$) (Table 6.4.3d). Higher magnitudes of 7-day EMWA PlayerLoad™ with a 3-day lag was associated with an increased odds of sustaining an MCL or ACL injury (OR 95% CI ≥ 1 & ADASYN OR 95% CI ≥ 1). Whereas, higher magnitudes of 14-day EMWA PlayerLoad™ with a 3-day lag displayed non-

unified OR 95% CI (lower 95% CI < 1 & upper 95% CI > 1), so were inconclusive in relation to the effect of injury association. Lower magnitudes of 14-day EMWA PlayerLoad^{TM} with a 3-day were associated with a decreased odds of sustaining an MCL injury (OR 95% CI ≥ 1 & ADASYN OR 95% CI ≥ 1), whereas lower magnitudes of 7-day EMWA PlayerLoad^{TM} with a 3-day lag had an inconclusive effect (OR lower 95% CI < 1 & upper 95% CI > 1).

6.5 Discussion

Medial collateral ligament and anterior cruciate ligament injury have presented a high burden to male professional rugby union teams throughout the past decade (Brooks et al., 2005a; Brooks et al., 2005b; Dallalana et al., 2007; Kemp et al., 2019). These injuries are more commonly sustained during events involving contact with other players (Dallalana et al., 2007; Montgomery et al., 2018). The overall purpose of this study was to examine the aetiology of MCL and ACL injury sustained during contact events in a male professional rugby union player population. Components associated with contact knee ligament aetiology were examined individually via three research questions. The first research question examined the existence of a relationship between a player's exposure to inciting contact events and the odds of sustaining knee ligament injury in contact. It was found that both increases in acute and chronic exposure to contact events decreased the odds of sustaining a contact knee ligament injury. The second research question examined the existence of a relationship between previously identified risk factors of knee ligament injury which included: Previous injury history, knee isokinetic strength and lower limb joint kinetics during the early stage of a single-leg drop-jump. Previous injury to the hamstring and calf muscle groups, larger magnitudes of isokinetic knee extension strength, larger magnitudes of hip adduction and knee abduction moment during at 50 ms of ground contact during a single-leg drop jump task were all associated with an increased odds of sustaining a knee ligament injury during contact. The third research question examined the existence of a relationship between acute and chronic exposure to on-pitch physical activity and the odds of sustaining a knee ligament injury during contact. Increased magnitudes of both acute and chronic exposure to on-pitch physical activity were associated with an increased odds of

sustaining a knee ligament injury in contact, whereas moderate magnitudes of chronic exposure were associated with a decreased odds of sustaining injury. A detailed discussion of the study findings is presented in the subsequent sections.

6.5.1 Question 1. Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players?

The purpose of the first research question was to examine whether exposure to larger magnitudes of tackle and ruck events during match play were inherently related to MCL or ACL injury sustained during a tackle or ruck events for male professional rugby union players. It was hypothesised that acute exposure to higher magnitudes of tackle and/or ruck event exposure would increase the odds of sustaining an MCL or ACL injury from a contact event during match play. However, this hypothesis was not supported, when examining the tackle event in isolation, no relationship was observed between tackle exposure and pooled knee ligament injury sustained during tackle events (Table 6.4.1a). Additionally, the hypothesis was also observed to be incorrect when examining exposure to both tackle and ruck events in relation to the odds of sustaining an MCL or ACL injury during a tackle or ruck event (Table 6.4.1b). Contrary to the hypothesised direction of the effect, larger magnitudes of acute exposure to tackle and ruck incidents were instead associated with a significant decrease in the odds of sustaining an MCL or ACL injury (Figure 6.4.1a – 1b). Despite there being a number of epidemiological studies reporting injury incidence associated with contact based inciting events when training for and playing rugby union (Brooks et al., 2005a; Brooks et al., 2005b; Fuller et al., 2007b; Dallalana et al., 2007; Kemp et al., 2018; Kemp et al., 2019), there is a dearth of studies examining exposure to contact events as a risk factor in comparison to the relationship between external and internal workload and other types of injury (Tierney et al., 2020; Edwards et al., 2021a). Due to this, comparison of the present findings to previous literature was extremely limited, with previous aetiological research focussed on concussion. Chéradame and colleagues (2021) observed that larger magnitudes of exposure to tackles over a season were associated with an increased risk

of concussion when the inciting event was a tackle in male professional rugby union players. Contact events in rugby union were previously observed to have high rates of injury incidence combined with moderate to high severity when examined in relation to all injuries and specifically knee ligament injuries (Brooks et al., 2005a; Brooks et al., 2005b; Fuller et al., 2007b; Dallalana et al., 2007). In a study of training related injuries, West and colleagues (2021a), reported that injuries sustained during full contact sessions were the most frequent.

One may interpret the findings relating to research question one as confirmation that tackle and ruck events are not inherently risky and players would be at less risk of sustaining a contact knee ligament injury when exposed to acute increases in the number tackles, tackling and rucks performed. However, this interpretation of the findings is not advised. When examining Figure 6.4.1c, injuries were sustained throughout the duration of match-play when players are exposed to a small number of contact events as well as a large number of contact events, with a slight trend towards injuries occurring earlier in the match. Furthermore, the contact exposure relative to game minutes played was not significantly associated with knee ligament injury. One may these findings with the common statement used by coaches and medical staff within professional contact sports, that 'contact injuries are unpredictable' (West et al 2021a). However, the author does not advocate this interpretation. Rather, these findings suggest the negative relationship between contact exposure and knee ligament injury may be influenced by the non-injured players being exposed to slightly larger magnitudes of contact events due to the longer time course of exposure (i.e. playing the whole game). This, in turn provides indirect support for the complex multivariate nature of sports injury aetiology models proposed by numerous authors that contact knee injury is dependent on several specific conditions. And as such, other variables internal and external to the injured player influence the risk of knee ligament injury sustained during a contact event rather than the absolute count of exposure (van Mechelen et al., 1992; Meeuwisse, 1994; Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2017; Bittencourt et al., 2016). For example, reduced neuromuscular control, the accumulation of fatigue of the injured individual, the technique of

individuals involved in the contact events (Hollander et al., 2021; Burger et al., 2016; Burger et al., 2017; Cross et al., 2019; Hendricks et al., 2015; Tucker et al., 2017), and the velocity of the other player involved in the contact event (Cross et al., 2019). Although not recorded in the current study, the magnitude and impact location may also influence the risk of knee ligament injury making the conditions. Indeed, previous studies examining video footage of contact injuries have reported that common situations are associated with these injuries. For example, ACL injuries occurred most frequently during the tackle and involved direct impact to the front of the knee resulting in anterior tibial translation and/or direct impact to the lateral side of the injured limb resulting in large knee abduction moments acting on the ACL (Montgomery et al., 2018; Della Villa et al., 2020). Video analysis of MCL contact injury in men's professional soccer revealed injury occurred when the lateral side of the injured limb was impacted directly on the joint, above or below resulting in large knee abduction moments acting on the MCL (Buckthorpe et al., 2021). This is supported in part by the previous epidemiological findings of Study 1, the overwhelming majority of MCL injuries were sustained where the individual was impacted from the side.

It was also hypothesised that chronic exposure to higher magnitudes of tackle and ruck events would decrease the odds of sustaining an MCL or ACL injury during a contact event. This was not supported when examining exposure to tackle events and tackle related knee ligament injury in isolation (Table 6.4.1a). However, when examining exposure to ruck and tackle events in relation to knee ligament injury, larger magnitudes of chronic exposure were associated with a decreased odds of injury over a 14-day period (Table 6.4.1b). This finding may provide support for the adapted application of Banister's model for athletic performance (1975) popularised by the work of Hulin et al. (2014), Windt and Gabbett (2016), in which the workload an individual undertakes over an extended period of time (chronic workload) is considered a proxy for fitness (Windt and Gabbett, 2017). Higher chronic workloads were previously reported to decrease the risk of injury in male professional rugby league players (Hulin et al., 2016). As previously stated, the concept of 'fitness' has been associated with both higher levels of aerobic and anaerobic

performance (Malone et al., 2016). In the current study chronic exposure to higher numbers of contact events may not only help to prepare the individual for the physiological demands of rugby union (Gabbett, 2016) but additionally influence the development of technical components associated with the contact events (e.g. body position during when being tackled) (Hendricks et al., 2016; Hendricks et al., 2018). However, an alternative interpretation of the findings is that the chronic 14-day EWMA value may also influenced by the non-injured players being exposed to slightly larger magnitudes of contact events within the acute 7-day time frame which was included in the calculation of the 14-day EWMA. It is important to note that none of the chronic variables using a lag (i.e. the contact events sustained on the day of injury and 6-days preceding were not included in the EWMA calculations) were significantly associated with knee ligament injury further highlighting this interpretation. Therefore, simply using counts of exposure to contact events when inferring knee ligament injury risk should be viewed with caution.

The ability of contact variables alone to classify out of sample injury cases in relation to pooled MCL and ACL injury data was poor (LOOCV AUC < 0.7) (Hosmer et al 2013) (Table 6.4.1a – 6.4.1b). The highest LOOCV AUC score was for the univariate 14-day EWMA modelled as a continuous variable when accounting for rare events (ADASYN LOOCV AUC = 0.69), followed by the 7-Day EWMA modelled in the same manner (ADASYN LOOCV AUC = 0.67). The out of sample classification performance of all variables were reduced when not accounting for pooled knee injury as a rare event using ADASYN (LOOCV AUC lower 95% CI < 0.5 & upper 95% CI > 0.5). It is therefore recommended that for application to the wider professional rugby union population the findings should first be replicated and combined with video analysis of contact MCL and ACL injury in order to confirm the existence of common scenarios of each injury with particular focus placed on the location and magnitude of the injurious impact. For the study population and the professional rugby team participating in the PhD project it is recommended that exposure to contact events should be included in clinical decision-making process to infer MCL and ACL risk

or for returning to play post rehabilitation. The findings of research question one provided further justification for examining the subsequent research questions of the study.

6.5.2 Question 2. Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players?

6.5.2.1 Question 2.i. Is previous lower limb injury history associated with the risk of sustaining an MCL or ACL injury from a contact event?

The first part of the second research question examined whether previous lower limb injury history was associated with the risk of sustaining an MCL or ACL injury from a contact event. It was hypothesised that sustaining a previous MCL or ACL injury would increase the risk of sustaining a subsequent knee ligament injury from a contact. The rationale for the hypothesis was that the previously injured ligaments would be less resistant to mechanical fatigue following the plastic deformation to the ligament causing the previous injury (Nigg and Herzog, 2007). However, the hypothesis was not supported. In the current study, previous knee ligament injury was not associated with either subsequent isolated MCL injury or pooled MCL and ACL injury risk (Table 6.4.1a – Table 6.4.1b). These results were contrary to previous research findings that reported previous ACL injury history increased the risk of subsequent ligament injury (Fulton et al., 2014; Smith et al., 2011; Waldén et al., 2006). A possible explanation for the contrasting findings in comparison to the present study was the majority of knee ligament injuries in injury history analysis were sustained to the MCL (28 MCL injuries, 9 ACL injuries). Due to the extra-articular location and increased vascularisation of the MCL compared to the ACL, the MCL has greater potential to heal after injury and this often leads to full recovery of the mechanical characteristics of the ligament (Jacobson, 2006). It was also hypothesised that sustaining a previous injury to either the hamstrings or triceps surae muscle groups would increase the risk of MCL and pooled MCL and ACL injury, and this was supported in the current study (Table 6.4.1a – Table 6.4.1b). The rationale behind these hypotheses were that as supporting structures crossing the knee joint, both muscle groups were responsible for reducing excessive mechanical

stresses and strains acting on the knee ligaments via resisting excessive anterior translation and abduction of the tibia (Li et al., 1999; Maniar et al., 2018; More et al., 1993; Nasseri et al., 2020; Morgan et al., 2014). Impaired neuromuscular function often occurs after muscle strain injury including: reductions in eccentric knee flexor strength (Lee et al., 2009); reductions in voluntary myoelectrical activity when performing maximal knee flexor eccentric contractions (Opar et al., 2013a; Sole et al., 2015); and reductions in voluntary myoelectrical activity during the initial portion of eccentric contraction (Opar et al 2013b). Therefore, the previously injured muscles may not be able to sufficiently reduce the loads acting on the MCL and ACL.

The ability of previous injury variables to classify out of sample knee ligament injuries were mixed. For both MCL injuries in isolation and when pooled with ACL injuries, previous injury to the triceps-surae muscle group exhibited moderate performance (LOOCV AUC > 0.7 & < 0.8) (Hosmer et al 2013) (Table 6.4.1a – Table 6.4.1b). Whereas the classification performance of previous injury to the hamstrings muscle group was poor (LOOCV AUC < 0.7) (Hosmer et al 2013) (Table 6.4.1a – Table 6.4.1b). In light of these findings, it is recommended that previous injury history to the triceps-surae muscle group should be taken into account when designing preventative conditioning programmes for male professional rugby players. Furthermore, players sustaining said injuries should be monitored to establish if any neuromuscular maladaptation is present following muscle strain injury.

6.5.2.2 Question 2.ii. Are previously identified isokinetic knee strength variables associated with the risk of sustaining MCL or ACL injury within the cohort of male professional rugby union players?

The efficacy of using isokinetic measures of knee extension and flexion strength to infer knee ligament risk within the cohort of male professional rugby union players was also examined in the study. The rationale for examining this question was primarily due to the ubiquitous use of this test by the men's professional rugby union team participating in the research project, to inform future knee ligament risk and clear injured players to return to play. Furthermore,

isokinetic assessment of the knee joint has long been part of periodic health examination in relation to knee ligament injury risk assessment of the past ~25 years. Therefore, examination of this testing modality was appropriate. It was hypothesised that isokinetic knee flexion strength variables would not influence the risk of sustaining either an MCL injury or pooled knee ligament injury (MCL and ACL), which was supported (Table 6.4.2.2a - Table 6.4.2.2b). This finding was in agreement with previous studies that have examined isokinetic knee strength variables in relation to knee ligament injury risk within a male professional soccer population (Bakken et al., 2018), female professional soccer and handball players (Steffen et al., 2016) and male and female military cadets (Uhorchak et al., 2003). A possible explanation for the absence of a relationship between knee ligament injury and isokinetic knee flexion strength was that although the medial hamstring muscles assist in reducing the external abduction moment associated with MCL and ACL injury mechanisms (Kim et al., 2016), by contributing to an internal knee adduction moment (Manair et al., 2018), the peak knee flexion moment recorded during isokinetic dynamometry represents a combined product of all internal moments and external moments acting on the dynamometer attachment (Baltzopoulos et al., 2012). As such, the individual contribution of the medial hamstring muscles were not possible to establish during this form of assessment.

Impaired neuromuscular function of the hamstrings group may indeed be associated an increased risk of knee ligament injury, as highlighted by the finding that previous injury to the hamstring muscles increased the odds of sustaining an MCL and pooled MCL and ACL injury. However, assessing maximal isokinetic strength comprises only one facet of neuromuscular function. In the case of knee ligament injury, the consensus is that the ligament is exposed to supramaximal stresses and strains at the early stages of ground contact (Koga et al., 2010; Hewett et al., 2016; Stuelcken et al., 2016); so, the ability of the surrounding musculature to attenuate the injurious forces at ground contact is likely to be more important compared to the maximal strength of a muscle group. Internal hip joint extension and ankle joint plantarflexion moments were examined at 50 ms post ground contact during a single-leg drop-jump task as a

proxy for this. However, no relationship was observed between any of the sagittal plan joint moments and knee ligament injury risk (Table 6.4.2.3a - Table 6.4.2.3b).

It was also hypothesised that isokinetic knee extension strength would not be associated with an increased odds of sustaining isolated MCL or pooled MCL and ACL injury. This was supported for isolated MCL injury (Table 6.4.2.2a). However, when both MCL and ACL injuries were pooled, larger magnitudes of knee extension strength at the higher angular velocity condition (180 deg.sec) were indeed associated with an increased odds of injury (Table 6.4.2.2b; Figure 6.4.2.2a). This finding was in agreement with previous prospective studies which also reported that individuals exhibiting larger magnitudes of isokinetic knee extension strength had a higher odds of sustaining knee ligament injury (DeMorat et al., 2004). However, despite increased magnitudes of isokinetic knee extension strength at a high angular velocity being associated with increased odds of pooled MCL and ACL injury sustained during contact events (Table 6.4.2.2b) the ability of this isokinetic variable to categorise out of sample data (i.e. to subsequently predict whether a player will sustain an MCL or ACL after an isokinetic test) was observed to be poor. The highest LOOCV AUC score observed being 0.57. When used in isolation, findings have limited clinical value, equating to 7% more than random chance when modelled as a continuous variable and 1% more than random change when modelled as a categorical variable. This finding supports the previous findings of Bakken and colleagues (2018) who reported that isokinetic assessment of knee strength had no clinical value in predicting knee ligament injury risk in soccer. Therefore, for the study population and the professional rugby team participating in the PhD project it is recommended that isokinetic assessment of knee strength in its current form is not used to such an extent in the clinical decision-making process to infer MCL and ACL risk or for returning to play post rehabilitation.

6.5.2.3 Question 2.iii. Are lower limb biomechanical variables during the early ground contact phase of a single-leg drop-jump task associated with the risk of sustaining MCL and ACL injury within the cohort of male professional rugby union players?

The relationship between lower limb biomechanics during a single-leg drop-jump were also investigated in relation to MCL and pooled MCL and ACL injury risk. It was hypothesised that players exhibiting larger magnitudes of external knee abduction moment during early ground contact would be more susceptible to both MCL injuries as well as pooled MCL and ACL injuries sustained during a contact event. These hypotheses were observed to be correct, with increased magnitudes of external knee joint abduction moment being associated with an increased odds injury (Table 6.4.2.3c – Table 6.4.2.3d; Figure 6.4.2.3a - Figure 6.4.2.3d). Comparison to previous research is limited to prospective studies examining the risk of sustaining a first ACL injury and the risk of ACL re-injury because at the time of writing (2022), no prospective studies examining the influence of lower limb landing biomechanics in relation to MCL injury risk were in existence. Previous research examining the relationship between knee ligament (ACL) injury (and re-injury) and lower limb biomechanics during a single-leg drop-jump landing were not in agreement with the findings observed in the current study. For example, in two recent prospective studies from King and colleagues which examined risk factors associated with ACL re-injury in male field-based team sport athletes, the authors reported that during a single-leg drop-jump knee abduction moment had either no relationship with injury (King et al., 2021a), or a small negative relationship (King et al., 2021b). A possible explanation for the difference in findings was that in the present study, the injuries were sustained during contact events, rather than non-contact inciting events such as rapidly changing direction. Numerous mechanisms of ACL injuries have been proposed e.g. via excessive anterior tibial translation, via excessive knee abduction or via excessive knee abduction coupled with tibial external rotation (McLean et al., 2015; Bates et al., 2019), and it is possible that the mechanism of ACL injuries sustained during contact events is different compared to ACL injuries sustained during non-contact events (Montgomery et al., 2018).

In the absence of previous studies examining MCL injury risk in relation to lower limb biomechanics one must draw on first principles relating to the structure and function of the MCL in relation to the anatomy of the lower limb and pelvis as well as the mechanism of MCL injury to provide a mechanistic explanation for the biomechanical findings within the current study. The function of the MCL is to support the medial side of the knee by restraining excessive abduction moments (Nigg and Herzog, 2007). Previous studies that have examined the mechanisms of contact MCL injury reported that all injurious events (direct contact with the lateral knee or lateral lower limb) resulted in a substantial abduction moment subjecting the MCL to magnitudes of stress and strain resulting in failure of n-fibres of the ligament (Buckthorpe et al., 2021; Chapter 3). Therefore, in the case of MCL injuries sustained during a contact event, a relationship between larger magnitudes of external knee abduction moment and injury appears to be mechanistically possible. It is possible that for those exhibiting larger magnitudes of external knee abduction moment, the MCL is held more taut (i.e. already under larger magnitudes of mechanical strain) in the players natural stance, meaning a smaller perturbation of external force is needed at the medial knee to reach injurious strains in the ligament.

A possible explanation of the observed relationship between increased knee abduction moments and ACL injury during contact events is supported by some recent cadaveric studies which report an association between increased knee abduction moment and ACL failure (Schilaty et al., 2019; Ueno et al., 2020). In a recent systematic review of bone bruising occurring as a result of ACL injury, a high number (approx. 70%) of bone bruises were located on the lateral side, which is suggestive of the presence of high knee abduction moments (Zhang et al., 2019).

The aetiology of knee ligament injury is a result of multiple factors, including knee abduction moment and hip adduction moment. The hypotheses of the existence of a relationship between increased magnitudes of external hip joint adduction moment and an increased odds of both injury MCL and pooled knee ligament injury (MCL and ACL) were observed to be correct (Table

6.4.2.3c – 6.4.2.3d; Figure 6.4.2.3a - Figure 6.4.2.3d). Again, comparison to the existing literature was limited to prospective studies examining the risk of sustaining ACL injury and re-injury due to the novel focus of MCL injury. The findings of the study were supported by Khayambashi and colleagues (2016), who observed an association between decreased magnitudes of isometric hip abduction strength and an increased risk of non-contact ACL in amateur male and female team sport athletes. A possible explanation for these findings was provided by simulation research conducted by Maniar and colleagues (2018), who observed the hip abductors (gluteus medius, gluteus maximus and piriformis) had the greatest potential to oppose the external knee abduction moment. Therefore, it is possible that the injured players in the study were unable to use the musculature of the hip to create an internal knee abduction moment to reduce the large magnitude external knee abduction moments. The current study employed an inverse dynamics approach to investigate joint moments, therefore internal moments were modelled as equal and opposite to the external joint moments and are therefore not reported in the study as separate variables. The internal hip abduction moments of the injured players in the study were therefore higher in magnitude in than the non-injured players. This is counterintuitive, as one of the key concepts in neuromuscular training as a preventative measure for knee ligament injury is conditioning the hip musculature to abduct the femur to assist in preventing excessive knee abduction moments (Kim et al., 2016). The larger magnitudes of internal hip abduction moment (external hip adduction moment) in fact indicates the individuals are having to work more to resist the external hip adduction moment and knee abduction moment (Winter, 2009), due to the pelvis and femur segments comprising the hip joint are in a greater amount of adduction compared to the individuals exhibiting lower magnitudes of hip abduction moment.

Establishing the predictive performance of the biomechanical measures is a key step in the injury prevention framework. The ability of the biomechanical variables to categorise out of sample data for MCL injury was observed to be moderate with the knee abduction moment at 50 ms having a LOOCV AUC score of 0.71 (Hosmer et al., 2013) (Table 6.4.2.3c). Hip abduction moment at 50 ms displayed lower predictive performance when categorising out of sample MCL injury

data with the LOOCV AUC score observed being 0.65 (Table 6.4.2.3c). Predictive performance of the biomechanical variables was poor (AUC < 0.7) when MCL and ACL injuries were pooled (Table 6.4.2.3c – 6.4.2.3d), with LOOCV AUC scores for knee abduction moment at 50 ms being 0.64 and for hip abduction moment 0.63. When compared to the predictive performance of the isokinetic variables, the knee and hip abduction moment were observed to be of more clinical value. Specifically, performance of knee abduction moment in relation to sustaining MCL injury via contact equated to 21% more than random chance when modelled as a continuous variable. This finding supports the previous findings of Bakken and colleagues (2018) who reported that isokinetic assessment of knee strength had no clinical value in predicting knee ligament injury risk in soccer. Which raises questions over the continuation of the current practice of isokinetic assessment of knee extension and flexion strength as a means to assess knee ligament injury risk in professional rugby union. For the study population and the professional rugby team participating in the PhD project it is recommended that lower limb biomechanical assessment of a single-leg drop-jump should replace isokinetic evaluation of knee joint strength and included in the clinical decision-making process to infer MCL and ACL risk or for returning to play post rehabilitation.

6.5.3 Question 3. Is exposure to on-pitch physical activity and related to an increased risk of knee ligament injury for male professional rugby union players?

The third aim of the study was to examine the existence of a relationship between acute increases in the exposure to external workload and the risk of sustaining MCL and ACL injury during contact events within male professional rugby union players. Accelerometer derived measures of external workload are comprised of the frequency and magnitude of both contact events as well as locomotion and other activities during the period of interest such as passing a rugby ball (Gabbett, 2015; Barreira et al., 2017; Hulin et al., 2018). The resultant PlayerLoad™ value presented a broad picture of physical activities a player has experienced during a session or game (Barreira et al., 2017), rather than an isolated count and magnitude of contact events

or distance travelled and was therefore termed ‘on-pitch physical activity’ in the current study for clarity. It was hypothesised that acute exposure to higher magnitudes of on-pitch physical activity would increase the risk of sustaining both an MCL injury and pooled knee injury (MCL and ACL injury) during a contact event. The rationale behind these hypotheses were from the findings of previous epidemiological literature associated with the injury suggesting that a lack of adequate conditioning may be associated with an increased knee ligament injury risk (Dallalana et al., 2007). The hypothesis was observed to be correct, with acute increases over 3-days and 7-days associated with an increased odds of sustaining MCL injury (Table 6.4.3a; Figure 6.4.3a), whereas pooled MCL and ACL injury were only associated with acute increases over 7-days (Table 6.4.3c; Figure 6.4.3c). Due to the novel research focus of the current study previous examination of exposure to accelerometery derived on-pitch physical activity in relation to specific knee ligament injury risk was not possible, thus comparison was limited to studies examining the risk of sustaining broader injury such as ‘all soft tissue injury’. Cummins et al. (2019) observed that acute increases in on-pitch physical activity were associated with sustaining any soft-tissue injury in male professional rugby league players when analysing the same PlayerLoad^{TM} metric used in the current study. This was also observed by Hulin et al. (2020) in another cohort of professional rugby league players. However, it should be noted that in both studies, the injuries analysed were non-contact in nature (Cummins et al., 2019; Hulin et al., 2020). It was also hypothesised that moderate magnitudes of chronic on-pitch physical activity exposure would decrease the risk of sustaining both an MCL injury and pooled knee injury (MCL and ACL injury) during a contact event. The standard chronic timeframe of 21-days was not associated with knee ligament injury risk. However, increased magnitudes of on-pitch physical activity exposure in the medium term (previous 7- and 14-days) were associated with an increased odds of MCL and pooled knee ligament injury (Table 6.4.3c - Table 6.4.3d; Figure 6.4.3c - Figure 6.4.3d), which was in agreement with the recent findings of West and colleagues (2021c), despite the differences in workload measure and injury type examined. Furthermore, moderate magnitudes of previous 7-day and 14-day chronic on-pitch physical activity were

associated with a decreased odds of sustaining knee injury (Table 6.4.3c - Table 6.4.3d). These findings were contrary to Cummins and colleagues (2019), who observed no association between soft tissue injury risk and accelerometer derived loads summed over the previous 14- and 21-days in male professional rugby league players. In addition, the authors reported higher magnitudes of PlayerLoad™ summed over the previous 28-day were associated with a decreased risk of injury. Hulin and colleagues (2020) also reported that higher magnitudes of PlayerLoad™ EWMA over the previous 28-days were associated with a decreased risk of non-contact lower limb injury in male professional rugby league players. The contrasting findings may be explained by the activities undertaken between each rugby code. Although there is a large amount of tackling and wrestling-type activity during rugby league, previous research has reported that increases in PlayerLoad™ is more related with locomotion and rapid changes of direction (Hulin et al., 2018). In contrast, in addition to tackling, locomotion and changing direction, rugby union involves a more varied number of contact events (e.g. rucking, mauling & scrummaging), which researchers have observed is associated with increases in PlayerLoad™ (Roe et al., 2016).

The basic pathophysiology of ligament sprain is considered be acute, resulting from high mechanical tensile stresses acting instantaneously on the collagen bundles within the ligament. This causes mechanical strains that exceed the threshold of normal elastic deformation capacity causing rupture (Nigg and Herzog, 2007; Bates et al., 2019). The mechanism of the pathophysiology for contact MCL and ACL injuries were previously observed to be via an event with at least two large external force inputs. This could be the foot striking the ground combined with an opposing player tackling either to the lateral side of the injured knee as the driver for the process for MCL (Buckthorpe et al., 2021; Dallalana et al., 2007), and in some cases the ACL (Dallalana et al., 2007; Montgomery et al 2018); or to the front of the knee or back of the calf for the ACL (Dallalana et al., 2007; Montgomery et al., 2018). It is plausible that the increases in external workload, either undertaken during an acute period or over an extended period of time (or possibly a combination thereof) would result in the individual to be in an increased state of

peripheral fatigue, resulting in reduced neuromuscular function (Cé et al., 2020). The influence of rugby union and rugby league activity on measures of fatigue (Pointon et al., 2012; Webb et al., 2013; McLean et al., 2010; Twist et al., 2012; Twist and Highton, 2013; McLellan and Lovell, 2012), muscle damage (McLellan et al., 2011a; McLean et al 2010; Twist et al., 2012; Takarada, 2003), and inflammation (McLellan et al., 2011a; McLellan et al., 2011b; Smart et al, 2008) has been well documented. As previously mentioned, the knee joint is reliant on the supporting musculature of the triceps-surae and hamstrings muscle groups in addition to the muscles which contribute to abduction of the hip (gluteus medius, gluteus maximus and piriformis). Which, when unimpaired by previous injury may reduce both excessive external knee abduction and anterior tibial translation and the associated stresses and strains acting on the MCL and ACL during activity involving large internal and external forces applied over short durations (Manair et al., 2018), such as rugby union contact events. Therefore, a possible mechanism underpinning the association between larger magnitudes of both acute and chronic workloads being associated with knee ligament injury observed is a reduction in the ability of those muscles to attenuate the injurious forces acting on the knee ligaments.

The ability of on-pitch physical activity variables to classify out of sample data was mixed (Table 6.4.3c - Table 6.4.3d). The highest LOOCV AUC score for isolated MCL injury was for the univariate 3-day EWMA modelled as a continuous variable when accounting for rare events (ADASYN LOOCV AUC = 0.68), followed by the 7-Day EWMA with a 3-Day Lag modelled in the same manner (ADASYN LOOCV AUC = 0.67). In contrast the out of sample classification performance of all variables were reduced when not accounting for MCL injury as a rare event (LOOCV AUC lower 95% CI < 0.5 & upper 95% CI > 0.5). The on-pitch physical activity variable exhibiting the highest out of sample classification performance in relation to pooled MCL and ACL injury was the 7-day EWMA with a 3-day lag modelled as a tertile group when accounting for rare events (ADASYN LOOCV AUC = 0.77), followed by the same 7-day EWMA with a 3-day lag when modelled as a continuous variable accounting for rare events (ADASYN LOOCV AUC = 0.72). Out of sample classification performance was reduced when not accounting for pooled

MCL and ACL injury as a rare event, however the reduction in performance was not as substantial as the analysis focusing solely on MCL injury.

Based on the findings, it is recommended that for future research concerning the wider professional rugby union playing population, PlayerLoad^{TM} and other accelerometer derived on-pitch physical activity variables should be further examined to determine the underpinning mechanism resulting in the increased risk of contact knee ligament injury. Furthermore, external workload should be analysed in relation to specific injuries (e.g. MCL vs Lateral ankle ligaments) rather than the broad injury criteria previously utilised in studies (e.g. lower limb soft tissue injury) while accounting for injury as a rare event (Carey et al., 2018).

For the professional rugby team participating in the PhD project, it is recommended that PlayerLoad^{TM} is incorporated into decision making process when monitoring player external workloads in relation to knee ligament injury risk. Specifically, the magnitude of acute exposure to on-pitch physical activity should be examined using an EWMA of PlayerLoad^{TM} over a 3-day window of time for contact MCL injury and a 7-day window of time for pooled MCL and ACL injury. Examining multiple acute timeframes may not be prudent for the medical and conditioning staff. Therefore, an acute 7-day timeframe is suggested for both injury groups as a compromise due to the negligible change in classification performance between 3-day and 7-day EWMA for MCL injury (Table 6.4.3c - Table 6.4.3d). The magnitude of exposure in the previous ten days exclusive of the past three days (7-day EWMA with a 3-day lag) should also be monitored in relation to knee ligament injury risk.

6.6 Limitations of the study

All experimental work is subject to limitations, in a similar manner to the previous experimental study (Chapter 5) the sample size of the current study was constrained to one professional rugby team (n = 143). However, due to the participating professional rugby team co-funding the research project combined with the rigorous exclusion criteria, this was unavoidable. To mitigate

this, the method of statistical analysis was selected to allow multiple seasons to be analysed per player. Furthermore, the incidence of MCL and ACL injuries observed was within that which was most recently reported by the England professional rugby injury surveillance project (Kemp et al., 2019). Finally, as mentioned in section 6.3.2.4.1, contact events were only recorded during competitive matches, this was unavoidable during the project due to the increased requirement for personnel and recording equipment which was outside of the scope of the project. This was mitigated somewhat by the training practices of the team, who pursued a policy of limited bone on bone full body contact during the training week. Therefore, caution is needed when interpreting the findings relating to question 1 to a larger population.

6.7 Conclusions

This study was unique in comparison with previous prospective studies examining contact knee ligament injury risk in that it was the first at the time of writing to examine risk factors in relation to sustaining a knee ligament injury from a contact event, where the previous research has focussed on non-contact injury events. Another novel aspect of the study was that it was the first prospective study to explicitly examine factors associated with MCL injury. The findings of this study suggest that contact aetiology injury is complex, with sole exposure to contact events actually decreasing the risk of injury. The author proposes that rather than viewing contact injuries are unpredictable, contact knee injury is dependent on several variables associated with contact injury risk (albeit in varying degrees). Previous injury to the triceps-surae and hamstring muscle groups were observed to increase the odds of sustaining both MCL injury and pooled MCL & ACL injuries via a hypothesised decrease in neuromuscular function diminished the ability of the muscles to reduce the loading of the knee ligaments (Li et al., 1999; Maniar et al., 2018; More et al., 1993; Nasseri et al., 2020; Morgan et al., 2014). Whereas previous injury to the knee ligaments was not observed to have a relationship with subsequent knee ligament injuries. Isokinetic knee flexion strength was not associated with sustaining contact knee ligament injuries. Furthermore, despite larger magnitudes of isokinetic knee extension strength at 180 degrees/sec being associated with an increased odds of sustaining MCL and ACL injury, the test

displayed poor classification ability. These findings suggest that the constraints associated with isokinetic assessment of knee joint strength reduce the value of the test for inferring knee ligament injury risk. In contrast when examining lower limb biomechanics during a single-leg drop-jump task, higher magnitudes of external hip adduction and knee abduction moments during early ground contact during landing were both associated with both an increased odds of sustaining MCL and pooled MCL and ACL injury sustained in contact. Which suggests that injured players were unable to use the musculature of the hip to reduce the large magnitude external knee abduction moments associated with injurious stresses and strains acting on the MCL and ACL. Larger magnitudes of acute and chronic increases in on-pitch physical activity were also observed to be associated with an increased odds of sustaining MCL and ACL injury during contact. The proposed mechanism for these findings was an increase in peripheral fatigue resulting in the diminished ability of the supporting muscles of the knee joint to attenuate the injurious forces acting on the knee ligaments. Although it was outside of the scope of the current study to conduct multivariate analysis of contact knee ligament injury due to the sample size, the study has however identified and internally validated several variables to be included in said the possible future development of such a model, specifically including: The 14-Day EWMA of the number of tackles and rucks experienced, previous triceps-surae injury history, knee external abduction moment at 50 ms and the 7-Day EWMA with a 3-Day Lag of on-pitch physical activity.

Chapter 7: Thesis discussion

7.1 Introduction

The aim of the thesis was to advance knowledge of high burden lower limb injury aetiology in male professional rugby union players, with a specific focus on hamstring and knee ligament injuries. Several novel research questions were presented in Chapters 2 and 4 which were addressed in the subsequent experimental chapters of this thesis (Chapters: 3, 5 and 6). The purpose of this chapter is to provide the reader with a summary of the main findings within the thesis in order to 1) appraise the degree to which the proposed research questions have been addressed, as well as 2) determine the extent of an original and significant contribution to existing knowledge the work within the thesis has provided. In addition, a general discussion of the key themes arising from the thesis as well as an examination of the benefits and limitations of the methodological approach used during this body of work is presented, alongside practical implications and proposed areas of future research arising from the work.

7.2 Research questions addressed in the thesis and original contribution to knowledge

The inception of this PhD research project was due to the interest of Exeter Chiefs RFC in reducing the injury burden caused by lower limb injury sustained during the course of the pre-season and playing season. Since 2012 the team has participated in periodic health examinations at the University of Exeter and as a result, the coaching, medical and conditioning staff were interested in utilising the data to identify any variables that were associated lower limb injury aetiology to inform and enhance the pre-existing injury prevention strategies. To facilitate the project research focus, a review of key theoretical models of sports injury prevention and causation was conducted in Chapter 2. A common theme within the frameworks of sports injury prevention research was that in order to successfully explore the aetiology of high burden injuries in a sport, the injuries that posed the highest injury burden must first be identified (van Mechelen et al 1992; Finch 2006). Men's professional rugby union has a wealth of

epidemiological literature (Brooks et al., 2005a; Brooks et al., 2005b; Brooks et al., 2006; Dallalana et al., 2007; Sankey et al., 2008; Kemp et al., 2018; Kemp et al., 2019), in addition to a consensus statement for injury epidemiological research proposed by the governing body of the sport, the International Rugby Board (Fuller et al 2007a). Chapter 2 and the previous injury epidemiology research conducted in rugby union provided a basis for the first experimental study (Chapter 3), the purpose of which was to explore the epidemiology of lower limb injuries in male professional rugby union players. This resulted in the formulation of the first research question:

7.2.1 Which injuries present the highest burden in men's professional rugby union?

Chapter 3: Study 1 Epidemiology of lower limb injuries in male professional rugby union players: a 7-year study

The main finding of this study was that injuries to the lower limb placed the highest burden on the rugby team when compared to the upper limbs, trunk, and head & neck. Specifically, injuries to the ACL sustained during contact events resulted in the highest injury burden, followed by injuries to the MCL sustained during the tackle and rucking, biceps femoris strain sustained during running and anterior talofibular ligament sprains sustained during contact events.

- **Original contribution to knowledge:** A descriptive investigation of high burden lower limb injuries in a male professional rugby union playing population with particular focus on reporting the injury burden and associated incidence rates and severities in relation to the inciting events of specific injuries.

The findings from Chapter 3 provided a focus for the subsequent experimental studies within the thesis. Specifically, these were injury to the biceps femoris and other muscles of the hamstrings muscle group and injury to both the ACL and MCL during contact events, with specific focus on the tackle. Although anterior talofibular ligament sprains (and all ankle ligament injuries) presented a large injury burden in Chapter 3, the varied number of inciting events

resulting in ankle ligament injury resulted in a challenge to select an appropriate movement task to include in the pre-season data collection sessions. This was also compounded by the type of motion capture system available for the project, the CODA motion capture system was an 'active' marker system which obtained 3-D marker coordinate trajectory data from powered LED markers. The system had a limit of 28 markers to be used whilst recording at the required sampling frequency (200 Hz). To fully examine ankle joint loading associated with the aetiology of ankle ligament injuries a multi segment biomechanical model of the foot would be required (Pothrat et al., 2015). These models require 12 - 14 markers per foot (Carson et al., 2001; Leardini et al., 2007), therefore employing this methodology was not possible in conjunction with the marker set required to examine knee joint aetiology. As a result, examining variables associated with ankle ligament injury aetiology was outside of the timeframe of the project. Therefore, the focus of the rest of the thesis was to explore the aetiology of hamstring strains in addition to MCL and ACL injuries sustained during contact events. Following a review of the literature which focussed on injury aetiology (Chapter 4), the following research question was examined in Chapter 5:

7.2.2 What factors are associated with an increased risk of hamstring injury in men's professional rugby union?

Chapter 5: Study 2 An exploration of factors associated with hamstring strain injury in male professional rugby union players

In Chapter 5, the research question was separated into the following sub-questions for clarity:

7.2.2.1 Is previous lower limb injury history associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

Previous injury to the hamstrings were associated with an increase in the odds of sustaining a subsequent hamstring strain, however this was only observed when all muscles were pooled. A

novel finding was that previous injury to the lateral ankle ligaments and MCL were both associated with an increased risk of biceps femoris injury and medial hamstring injury.

7.2.2.2 Are isokinetic knee strength variables associated with the risk of sustaining a hamstring strain within the cohort of male professional rugby union players?

Higher magnitudes of knee flexion strength were associated with an increased odds of sustaining hamstring strains regardless of mechanism. However, when specific muscles were analysed, isokinetic strength variables were not associated with biceps femoris strain, but were associated with the risk of sustaining semimembranosus and semitendinosus strains. Regardless of these findings, all isokinetic variables exhibited poor out of sample injury classification performance, with the highest LOOCV AUC score observed being 0.57.

7.2.2.3 Is high-speed running exposure associated with an increased risk of sustaining a hamstring strain within male professional rugby union players?

Acute high-speed running exposure appeared to increase the odds of hamstring strain (especially 3-day EWMA), although this was inconclusive due to the wide distribution of the hamstring injury group. Chronic exposure to moderate magnitudes of high-speed running was observed to decrease the risk of sustaining a hamstring strain. In contrast, chronic exposure to high magnitudes resulted in an increased risk of hamstring injury, specifically exposure to high-speed running over 14 and 21 days prior to the week in which the injury was sustained. These findings were facilitated by the addition of examining multiple acute and chronic timeframes of high-speed running exposure on the aetiology of hamstring injury, to standard timeframes used by the participating team.

- **Original contribution to knowledge:** Prospective investigation into the existence of an association between hamstring muscle strain, previous injury and isokinetic strength variables in a male professional rugby union playing population
    - In relation to the hamstring muscle group
    - In relation to the individual muscles of the hamstring muscle group
    - In relation to the inciting event

- **Original contribution to knowledge:** Prospective investigation into the existence of an association between hamstring muscle group strain sustained whilst running and GPS derived exposure to high-speed running in a male professional rugby union playing population
    - In relation to different time frames of acute exposure
    - In relation to different time frames of chronic exposure

Whereas there was a wealth of previous hamstring injury research to guide the formulation of the research questions in Chapter 5, as well as a large number of hamstring injuries sustained by the professional rugby team participating in the project, the aetiology of contact injury to the ACL and especially the MCL has received little to no research focus in men's professional rugby union and in professional team sport as a whole. Regardless of this, the epidemiological findings from Chapter 3 and key epidemiological literature in professional rugby union highlighted that injury to the MCL and ACL presented a substantial injury burden to teams and should therefore be further investigated (Dallalana et al., 2007). In order to explore the aetiology of the above contact knee ligament injuries in the thesis, it was important to start with the first principles proposed in injury prevention frameworks and models of injury aetiology, namely: identifying the inciting event, understanding the mechanism of injury and if possible, identifying associated variables. This led to the formulation of the following primary research question and sub questions:

7.2.3 What factors are associated with an increased risk of MCL and ACL injury sustained during contact events in men's professional rugby union?

Chapter 6: Study 3 An exploration of factors associated with knee ligament injury in male professional rugby union players

In Chapter 6, due to the novel research focus the research question was separated into the following sub-questions:

7.2.3.1 Is exposure to tackle and ruck events inherently related to an increased risk of knee ligament injury for male professional rugby union players?

Contact event exposure was introduced as a novel workload variable in the study, as this was not in the participating team's pre-existing risk stratification process. Exposure to the number of tackle events during a match was not related to sustaining an MCL or ACL injury where the inciting event involved a tackle. When tackle and ruck events were pooled, increased exposure to the number of events during 7 and 14 days preceding the injury was associated with a decreased risk of sustaining an MCL or ACL injury where the inciting event involved a tackle or a ruck. This finding supported previous suggestions that the aetiology of contact knee ligament injury involved more factors than exposure to the inciting events (Gisane et al., 2001; Windt and Gabbett, 2016), which justified the formulation of the subsequent research questions in Chapter 5.

- **Original contribution to knowledge:** Prospective investigation into the existence of an association between MCL and pooled MCL and ACL sprain injury sustained during contact events and exposure to the number of inciting contact events in a male professional rugby union playing population.
    - In relation to different time frames of acute exposure
    - In relation to different time frames of chronic exposure
    - In relation to specific inciting events
    - Accounting for injury as a rare event

7.2.3.2 Are previously identified variables of knee ligament injury risk associated with contact knee ligament injury within male professional rugby union players?

Previous injury to the either the MCL or ACL was not associated with an increased risk of sustaining a subsequent knee ligament injury during a contact event. In contrast, previous injury to either the hamstring or triceps surae muscle groups were associated with an increased risk of contact MCL injury and pooled contact MCL and ACL injury. No relationship was observed between isokinetic knee strength variables and contact MCL injury risk. However, when both MCL and ACL injuries were pooled, larger magnitudes of knee extension strength were

associated with an increased odds of injury, regardless the classification performance of all isokinetic variables was poor. A biomechanical analysis of player lower limb joint kinetics during a single-leg drop-jump task was added to the testing battery as a novel measure during the project. Individuals exhibiting larger magnitudes of external knee abduction moment and hip adduction moments during the early stages of ground contact during a single-leg drop-jump were both associated with an increased risk of MCL or ACL injury during contact.

- **Original contribution to knowledge:** Prospective investigation into the existence of an association between MCL and pooled MCL and ACL sprain injury sustained during contact events and isokinetic strength variables in a male professional rugby union playing population.
- Prospective investigation into the existence of an association between MCL and pooled MCL and ACL sprain injury sustained during contact events and lower limb biomechanical variables during a single-leg drop-jump task in a male professional rugby union playing population.

7.2.3.3 Is exposure to on-pitch physical activity related to an increased risk of knee ligament injury for male professional rugby union players?

Player on-pitch physical activity exposure was introduced as a novel workload variable during the study, as it was not adopted by the participating team prior to the PhD project. Acute increases in player on-pitch physical activity data over 3-days and 7-days were both associated with an increased odds of sustaining MCL injury, whereas pooled MCL and ACL injury were only associated with acute increases over 7-days. Increased magnitudes of on-pitch physical activity exposure during the previous 7-days with a 3-day lag as well as the previous 14-days were both associated with an increased odds of MCL and pooled MCL and ACL injury.

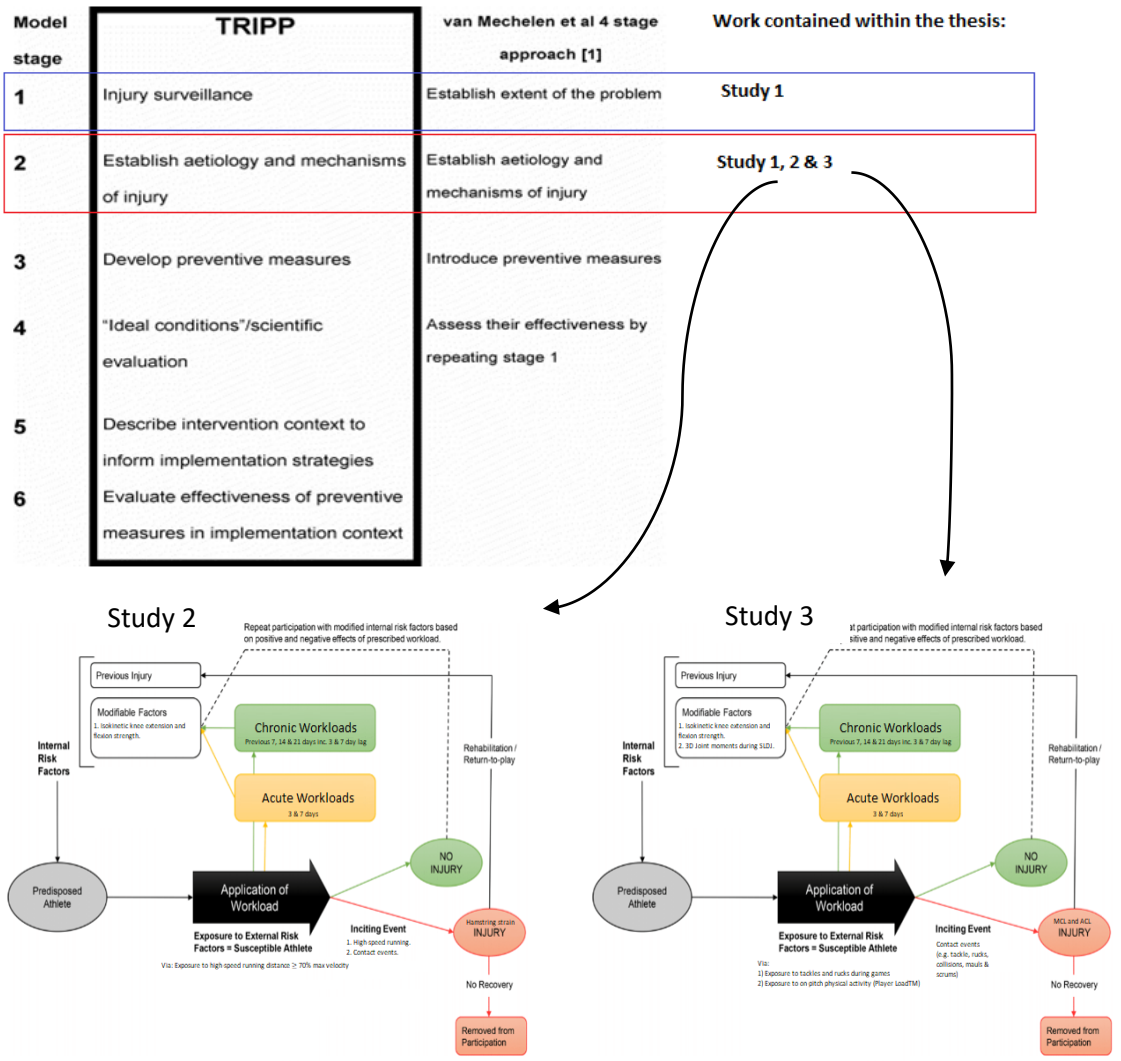
- **Original contribution to knowledge:** Prospective investigation into the existence of an association between MCL and pooled MCL and ACL sprain injury sustained during contact events and accelerometer derived exposure to on-pitch physical activity in a male professional rugby union playing population
    - In relation to different time frames of acute exposure
    - In relation to different time frames of chronic exposure
    - Accounting for injury as a rare event in relation to daily analysis of workload

7.3 General discussion: Implications and impact of the work contained within the thesis

The overall purpose of the thesis was to examine the aetiology of high burden injury in professional rugby union. This work was undertaken by exploring different physical characteristics of the rugby players taking part in the research project as well as the external workloads imposed upon them in accordance with key models of injury aetiology (van Mechelen et al., 1992; Meeuwisse, 1994; Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2017; Bittencourt et al., 2016). The following section will discuss key themes raised by the thesis in relation to the research field and where possible highlight practical implications and impact arising from the thesis.

7.3.1 High level overview: Where does the work sit in relation to the wider literature in sports injury prevention?

In accordance with both frameworks of injury prevention research created by van Mechelen and colleagues (1987; 1992) and Finch (2006), the work contained within the thesis focussed on identifying lower limb injuries resulting in the highest burden and the initial steps necessary to examine the aetiology of said injuries (Figure 7.3.1a).



7.3.1a Where the work contained within the thesis is situated in relation to both frameworks of sports injury prevention research conceived by van Mechelen et al. (1987) and Finch (2006), in addition to the workload-injury aetiology model. Image adapted from both Finch (2006) & Windt and Gabbett (2016).

When reviewing the literature surrounding aetiological variables related to the injuries examined within the thesis in Chapter 4, prospective research studies examining injury aetiology within rugby union were few in number in comparison to the considerable amount of sports injury research which has focused on establishing the epidemiology of injuries, or comparative mechanistic research conducted in-vitro or in-silico which were based on the findings of the limited number of prospective aetiological studies. All forms of research mentioned are key to informing the iterative process of sports injury prevention research, however in order to develop successful preventative measures to reduce the risk of high burden injuries the aetiology of an

injury must be thoroughly understood (Finch, 2006). This not only requires the aetiology to be researched in a rigorous manner by *one* key study; instead, what is more important is that a sufficient number of studies are conducted with the end goal of conducting a meta-analysis to establish whether an overall effect exists and if present, the direction of its influence (Maughan et al., 2018). There are numerous methodological challenges associated with conducting prospective research which may also explain the relative lack of such studies (See Discussion section 7.3.2), which is likely compounded by the simple fact that the opportunity to work with a professional sports population for an extended period of time rarely presents itself. A real-world example of this issue was presented by Bahr (2016) who illustrated the ramifications of the absence of sufficient prospective aetiological research in the field of non-contact ACL injury prevention research. A substantial amount of non-contact ACL injury prevention research has relied on the findings of one early prospective study by Hewett and colleagues (2005), which reported an association between higher magnitudes of knee external knee abduction moments during drop-jump landings and ACL injury. However, subsequent studies and a recent meta-analysis reported that larger magnitudes of external knee abduction moment was not related to non-contact ACL injury risk (Cronström et al 2020). Regardless of these later studies, extensive time and resources continue to be invested into researching the mechanism of how higher external knee abduction moments are generated and methods to reduce them. The figure below adapted from Weir (2021) illustrates the lack of prospective aetiological studies in comparison to the larger proportion of mechanistic studies conducted based on the prospective findings (Figure 7.3.1b).

Figure 7.3.1b An illustration of the number of mechanistic studies arising a small number of prospective studies in relation to non-contact ACL injury – adapted from Weir (2021).

There is no denying that the above studies still make an important contribution to the literature and this section does not seek to criticise the work undertaken by the authors, but rather highlight the amount of research that has used the findings of Hewett et al (2005) as a justification to examine the mechanisms of ACL injury. A larger evidence base of prospective aetiological research is needed to confirm and highlight new aetiological variables. With this in mind, the work contained within this thesis was intended to be a conduit between epidemiological studies and the mechanistic studies; to prospectively examine variables associated with the aetiology of the high-burden injuries highlighted by the epidemiological studies and identify areas for further detailed analysis.

Sports injury aetiology is a complex problem and it is likely that the cause is indeed multifactorial, with the example of the variables examined within the thesis (e.g. exposure to external workloads, previous injury history, biomechanics and lower limb strength) all interacting with each other to influence the risk of sustaining a given injury. This assertion is far from novel, with Meeuwisse highlighting the need for a multivariate approach in his model of sports injury aetiology in 1994, and it has been a prominent part of all other subsequent models of injury aetiology (Gissane et al., 2001; Finch, 2006; Meeuwisse et al., 2007; Windt and Gabbett, 2016; Bittencourt et al., 2016). More recently, the non-linear manner in which variables interact with each other in relation to injury risk and each other was the subject of a call for a paradigm

shift in sports injury aetiology research by Bittencourt and colleagues (2016). This approach may provide further advances in the understanding of sports injury aetiology, and highlights that an interacting profile of aetiological variables for a given injury is context specific (e.g. dependant on the mechanism of injury and the inciting event) and may change over time for an individual depending on the circumstances (Bittencourt et al., 2016). As a hypothetical example of this complexity, a professional rugby union player may have an increased genetic predisposition to musculotendinous or ligament injuries due to a polymorphism (e.g. COL1A1) (Brazier et al., 2019), and has sustained a hamstring injury early in their career at a semi-professional club followed by an ineffective period of rehabilitation. When viewing this aetiological variable in isolation in conjunction with the findings of the thesis, one may expect that the individual would be susceptible to an increased risk of a subsequent hamstring injury due to the exhibit reduced fascicle length (Timmins et al., 2016), atrophy (Sanfilippo et al., 2013), increased collagen formation (Silder et al., 2008) and reduced voluntary activation (Fyfe et al., 2013). However, these mechanistic variables associated with the previous hamstring injury also result in lower magnitudes of lower limb strength coupled with poor movement quality and an inability to attenuate large magnitudes of external moments during the early stages of ground contact which may now also increase the risk of sustaining a knee ligament injury during a contact scenario. Having subsequently moved to a professional club, the player is exposed to an unaccustomed increase in exposure to on-pitch physical activity over the week prior to their first competitive match due to the preparation methods of the new team, this build-up of fatigue in turn further exacerbates the player's poor movement quality and inability to quickly attenuate external moments which results in the player suffering an MCL injury when being tackled during the game. The individual then undergoes an intense period of rehabilitation which results in an increased ability to attenuate moments during early stance due to increases in lower limb strength. During this phase, the player is exposed to graduated exposure to contact and deceleration activities as well as a high chronic high-speed running load due to the strength and conditioning department's interpretation of the A:C ratio for soft tissue injury prevention. In

addition to the positive adaptions of the rehabilitation phase resulting in a decreased risk of knee ligament injury, the adaptions have also resulted in a further reduction fascicle length of the hamstrings muscle group which coupled with the large magnitudes of chronic high-speed running results in the individual sustaining a hamstring strain whilst sprinting to score a try on their first game back from injury. The aforementioned example is a scenario that is not uncommon within professional rugby union teams, and adopting a complex approach could result in a richer understanding of injury aetiology as well as ecologically valid conclusions drawn. As stated in the previous paragraph, larger evidence base of prospective aetiological research is needed, but what should also be required is to embrace the complexity of this problem rather than adopting the conventional reductionist approach.

Professional team sports industries such as rugby union do not stand still, indeed practitioners within medical and conditioning departments continue to use a plethora of tests and variables to infer injury risk and readiness to return to play - in some cases regardless of a sufficient base of evidence (West et al. 2019), this is often driven by the need for *any* perceived increase in performance (marginal or substantial). Therefore, there is need for sports injury researchers to continue to examine the efficacy of these practices in relation to injury aetiology and where possible, ensuring future analyses capture the inherent complexity and multivariate nature of the problem.

7.3.2 Conducting aetiology research in sports teams – overcoming small sample sizes

The research project was co-funded by a professional rugby team playing in the top flight of domestic league in England at the time of writing (2022). This meant that a substantial amount of data could be collected on the participating players over an extended period of time (4 – 12 years), however this did raise some methodological challenges which have been experienced by other researchers and professional sports teams attempting to gain insight into the relationship between player variables and sustaining injury.

Ensuring sufficient sample size is fundamental for reducing a risk of type I and II error within an experimental study (Ruddy et al, 2019). In chapter 3 the sample size was relatively small in relation to previous studies that have examined injury epidemiological in professional rugby union which have analysed multiple teams competing within a league, often over multiple seasons (Brooks et al., 2005a, 2005b; Brooks et al., 2006; Dallalana et al., 2007; Sankey et al., 2008; Kemp et al., 2018; Kemp et al., 2019). Increasing the sample size by including other teams competing at the same level of competition was however not possible due to constraints of the one professional rugby team co-funding the research project. Despite the smaller sample size, the findings of chapter 3 were however within the most recently reported 95% confidence intervals for epidemiological data presented by the England professional rugby injury surveillance project which suggests that the sample is indeed representative of the greater population (Kemp et al., 2019).

7.3.2.1 Accounting for repeated entry into analysis - The use of GLMMs

To overcome the impact of lower sample sizes in Chapters 5 and 6, generalized linear mixed-models (GLMMs) were employed. As is common place in team sport injury research and applied practice, the data analysed was often longitudinal in nature and contained repeated inclusions of players (e.g. multiple player-seasons in Chapter 5 and 6). This correlated data structure meant that conventional statistical analyses such as logistical regression when conducting classification analyses are not appropriate due to the assumption of independence of observations. Previous studies that have ignored this assumption such as Gabbett and Jenkins (2011), Rogalski et al. (2013), Hulin et al. (2020), Myer et al. (2010), Hewett et al. (2005), and Krosshaug et al. (2016). These were likely subject to inflated Type I error rates as a result of smaller standard errors when random effects have not controlled for (Williamson et al., 1996 Diggle et al., 2013; Ruddy et al., 2018).

7.3.2.2 Accounting for injury as a rare event when analysing workload data: Majority class reduction and minority class oversampling

In injury aetiology research, ensuring that the number of un-injured cases does not substantially outweigh the number of injured cases (referred to as 'class imbalance') is paramount, in addition to adequate sample size (Peduzzi et al., 1996; Krawczyk, 2016; Ruddy et al., 2019). Class imbalance can result in classification error by erroneously inflating the accuracy estimates of the model by always predicting the majority class (i.e. the non-injured players) (Kuhn and Johnson 2013; Krawczyk, 2016; Carey et al 2017; Ruddy et al 2018). As a rule of thumb, the ratio of injured cases to non-injured cases has been proposed as 0.10, with one predictor variable per 10 injured cases (Peduzzi et al 1996). In Chapter 5, when comparing the class balance of the isokinetic analyses to previous prospective studies (Green et al 2018), the current study third largest number of injured cases (3/12), despite having had the fifth largest sample size (5/12). This was also the case in chapter 6, which demonstrated the second largest number of injured cases in relation to other prospective studies examining the influence of external knee abduction moment during a jump landing task in relation to sustaining knee ligament injury risk (2/4) selected by Cronström and colleagues (2020) in a recent meta-analysis. Despite, the current study having the third lowest total sample size when also compared to the same selected studies (3/4) (Cronström et al 2020). This was also observed when comparing the sample size and injured cases to previous quality prospective studies examining the influence of external hip adduction moment during jump landing presented in a recent meta-analysis (Chia et al 2020), the current study had the second largest number of injured cases (2/4).

In the analysis of exposure to external workloads in chapters 5 and 6 the issue of class imbalance within the dataset was present (i.e. un-injured days outnumbered the number of injuries). This was not the case for the isokinetic and biomechanical data due to the limited frequency of data collections i.e. conducted during each pre-season compared to the daily collection of player workload data, however these data would have ideally been collected multiple times per season

per player and therefore may have also resulted in class imbalance. Due to the combination of a single team participating and co-funding the project and a constrained timeframe in which to collect the data, collecting more data was not an option. Furthermore, in light of the observed incidence rates of the injuries in question, an additional season of data collection would have resulted in a negligible increase in the injured case number relative to the un-injured cases. Steps were taken to mitigate the influence of class imbalance in the thesis. In chapter 5, a conservative sample reduction was employed to reduce the number of non-injured exposures (Krawczyk, 2016; Carey et al., 2018). When combined with the apparent AUC scores of all models undergoing leave-one-out cross-validation to evaluate the model's ability to classify novel data (Colby et al., 2017; Carey et al., 2018; Colby et al., 2018), the LOOCV AUC scores for high-speed running exposure observed in the current study were within a similar range to those reported in a larger cohort of hamstring injuries in AFL (AUC = 0.5 - 0.63) (Ruddy et al., 2018). Building on this, in chapter 6 a conservative sample reduction was employed to reduce the number of injury free days, combined with a minority oversampling technique (He et al., 2008; Krawczyk., 2016; Carey et al., 2018). The use of minority oversampling techniques is novel to the fields of sports science and sports medicine and some may argue that using such techniques should be avoided as may lead to an over-fitted model. Responses to this potential criticism on ethical grounds include: 1. To ensure a sufficient ratio between the number of injury free training days recorded and the number of injuries observed without the minority class being considered a rare event (Conservative ratio >= 0.20, lenient ratio >= 0.10) (Peduzzi et al 1996; Ruddy et al., 2019), one would have to wait for a sufficient number of injuries to occur without the addition of other injury prevention measures. However, this appears to be an issue even with one of the largest studies to date conducted by West et al. (2021a), who examined the majority of a professional rugby playing population in a longitudinal multi-team design. West and colleagues (2021a) reported a range of 258 – 438 time-loss injuries sustained during training sessions and a range of 103200 – 159398 hours of training exposure over an 11-season period. When using the 95% CI of the mean length of training days (95% CI 6 h 30 min to 7 h 6 mins) this results in a ratio of

injured cases to injury-free training days between 0.01 and 0.02, qualifying the injuries in this study as rare events. This simple example highlights that class imbalance is inherent even within studies with the largest sample size in the field of player workload. Therefore, accounting for injuries as a rare event when examining injury-workload aetiology research is necessary, with suggestions of 'more multicentre studies must be undertaken' or 'a larger sample size is needed' may not be sufficient. 2. Not accounting for injury as rare event and avoiding the 'made up data' of such algorithms as ADASYN resulted in more optimistic injury classification scores in the current study compared to those tempered by the combined use of ADASYN and LOOCV. As such, relying on these naïve models may lead to incorrect conclusions during the injury risk decision making process and time wasted in future research. Not accounting for injury as a rare event in relation to external workload may therefore go against the old adage *primum non nocere*.

7.3.3 Do previous injuries influence future injury risk?

A history of previous injury has long been established as an influential factor in the aetiology of lower limb injury (Orchard et al., 1997; Bennell et al., 1998; Gabbe et al., 2006; Hägglund et al., 2006; Waldén et al., 2006; Henderson et al., 2010; Smith et al., 2011; Fulton et al., 2014; Green et al., 2020). As a variable, previous injury is conventionally categorised as non-modifiable i.e., once an individual has sustained an injury, they cannot un-injure themselves. However, in reality this non-modifiable variable represents a cluster of other mechanistic variables associated with injured tissue. For example, muscle strains may exhibit reduced fascicle length (Timmins et al., 2016), atrophy (Sanfilippo et al., 2013), increased collagen formation (Silder et al., 2008) and reduced voluntary activation (Fyfe et al., 2013). In some cases, these variables can be subsequently modified depending on the time course of tissue healing, the success of the period of rehabilitation and the post-rehabilitation management of the injured individual (Timmins et al., 2016). This cluster of mechanistic variables may explain the historical association between previous injury and subsequent injury risk of the same site. However, in both Chapter 5 and 6

for the overwhelming majority of analyses, previous injury did not increase the odds of subsequent injury to the same site (the one exception being previous hamstring injury increased the odds of subsequent hamstring injury when all muscle groups and inciting events were pooled). It is important to note the influence of playing level when investigating subsequent injury risk to the same sight, or more importantly, the supporting infrastructure that is associated with playing level. The rugby players participating in the project were full time professionals who had access to on-sight physiotherapists and sports medicine doctors as part of their contracts. When injured they went through a full-time rehabilitation and return to play process. In many cases, these resources are not available to semi-professional or amateur which may explain the previous positive findings regarding the associations between previous and subsequent injury of the same site (Sevick et al., 2018).

The majority of previous research has focused on the influence of previous injury on subsequent injury risk of the same site (Fulton et al., 2014; Green et al 2020), for example the influence of previous hamstring injury on subsequent hamstring injury. Chapters 5 and 6 also explored the influence of previous injuries which occurred proximally and distally to the injured site (e.g. the influence of previous hamstring injury on subsequent MCL injury risk). A common theme in both studies was that previous injury to the surrounding tissues increased injury risk rather than a previous injury to the site of interest. This does in part highlight the complexity of previous injury history in the pathway of injury aetiology and provide justification for future examination of the underpinning physiological, biomechanical and psychological variables associated with previous injury history. However, when the findings are viewed in conjunction with the wider field it does suggest that for the team participating in the study, the rehabilitation strategy should be reviewed. The demands of rehabilitating the injured muscle or ligament to ensure a player is available to play in as timely a manner as possible should be tempered with a more universal approach focussing on the surrounding tissues.

7.3.4 Periodic health examinations to assess lower limb injury risk – are they worth it?

This PhD project was in part a result of the professional rugby union team participating in and co-funding the project undergoing historic periodic health examinations at the University of Exeter. This mainly involved isokinetic assessment, with the addition of biomechanical assessment introduced as a novel measure by the researcher during the PhD project from the 2015-16 pre-season onwards. The following section will discuss the findings from the isokinetic and biomechanical analyses conducted in chapter 5 and 6.

7.3.4.1 Isokinetic assessment of hamstring and quadriceps strength

Isokinetic strength assessment has been a mainstay of periodic health assessments and sports injury prevention research for ~25 years due to the relative ease of data collection and the speed of processing the results. The findings of the thesis supported previous meta-analyses which reported non-existent or weak relationships between isokinetic assessment of knee strength and both hamstring injuries and knee injuries. The ability of a test to successfully classify the outcome of interest (in this case, injury to the ACL, MCL or hamstring muscles) was another fundamental consideration of inclusion to a PHE. The injury classification performance of isokinetic dynamometry within the thesis was poor and equated to a similar accuracy one would achieve from flipping a coin (Chapter 5 and Chapter 6). These findings have also been observed by other researchers (Steffen et al., 2016; Green et al., 2017; Green et al., 2020; King et al., 2021a), and suggest that the use of isokinetic dynamometry for inference of injury risk in professional rugby union should not be used or at the very least should viewed with caution. One exception may be during the early phases of lower limb injury rehabilitation when individuals are primarily prescribed open chain exercise prior to regaining the capacity to perform dynamic movement tasks such as jumping or running (Thorborg et al., 2020; King et al., 2021a).

7.3.4.2 Biomechanical analysis of the single-leg drop-jump task

Analysis of human movement and the accompanying forces *acting on (external)* and *produced by (internal)* the individual have been a common feature of PHEs with the aim of inferring injury risk. However, there is a discrepancy between the method of data collection and type of data analysed (i.e. joint angle only, external joint moments via inverse dynamics, EMG or estimated muscle forces via a forward dynamics solution) and the task selected for the analysis (walking, running or jumping etc) in research, applied practice and commercial ventures. Due to these issues, the evidence base for biomechanical assessments in relation to lower limb injury is smaller than that of isokinetic analysis of knee joint extension and flexion strength. In the thesis a single-leg drop-jump task was selected because it was considered to provide a challenge to the neuromuscular systems of the deconditioned rugby players in the first week of pre-season, not only in the sagittal plane but also in the frontal and transverse planes which differentiated the task from the isokinetic assessments conducted in the thesis. In addition, it was also one of the primary return to play tests used by the medical staff for knee ligament injury and had the additional benefit of being able to be performed in a short timeframe within the small capture volume of the University of Exeter biomechanics laboratory. Studies that have been published recently provided confirmation in the choice of the single-leg drop jump over bilateral jumps and horizontal hops due to the higher frontal plane moments (King et al 2018; King et al 2019) associated with knee MCL and ACL injury aetiology. The ability of the SLDJ task biomechanical analysis to classify knee ligament injury was mixed. When examining the classification performance for MCL injuries sustained during contact, external knee joint abduction moment during the early ground contact phase displayed the joint highest classification performance in Chapter 6. When MCL and ACL injuries were pooled, the classification performance of all biomechanical variables decreased, however, the biomechanical analyses still outperformed the isokinetic variables.

The biomechanical task selected for analysis was tailored to examine the existence of aetiological factors associated with contact knee ligament injury rather than hamstring injury. The primary reason for this choice was that at the time of planning the prospective research studies that would later become Chapters 5 and 6 (October 2014 - February 2015) there was an absence of prospective biomechanical research examining hamstring strain injury, with the majority of aetiological research focussed on strength variables measured via isokinetic dynamometry. After conducting an initial epidemiological analysis on the hamstring injury data (Chapter 3) and examining the inciting events from pre-existing rugby union injury epidemiological studies (Brooks et al., 2005a; Brooks et al., 2005b; Brooks et al., 2006; Williams et al., 2013; Kemp et al 2018; Kemp et al 2019; Williams et al 2021), a biomechanical analysis of a high-speed running task was initially planned to be included in the yearly PHE data collections. However, this was vetoed by the medical staff of the professional rugby team participating in the project because the team had sustained a number of hamstring injuries during the pre-season 30-meter sprint speed testing in the two years previous. When combined with small capture volume of the University of Exeter biomechanics laboratory and the limited timeframe allowed for conducting the PHEs by the participating team, the author decided to change the focus of chapter 4 to examine if exposure to high-speed running was indeed associated with hamstring injury to provide a body of evidence for future research to explore high-speed running biomechanics. Subsequently, the biomechanical assessment task was targeted at exploring knee ligament injury aetiology.

The discussion points raised in chapter 3 combined with the findings of recent meta-analyses (Green et al, 2017; Green et al, 2020) suggested that due to the biarticular nature of the long head of biceps femoris, semitendinosus and semimembranosus, examination of moments acting on an individual's hip may exhibit a stronger relationship with hamstring strain injury compared to the reported isokinetic strength variables. A subsequent analysis was conducted in order to examine the existence of a relationship between external moments experienced in the sagittal plane during the SLDJ task and hamstring strain injury using the same methodology used in

Chapter 6. The findings presented in table 6.4.4.2.1 provided support to the postulation that higher magnitudes of external moment acting on the hip are associated with the aetiology of hamstring strain injury in professional rugby union rather than isokinetic knee flexion strength. Furthermore, the biomechanical analysis revealed that higher magnitudes of external hip flexion moments at 100 ms post ground contact demonstrated a marked improvement in classification performance (AUC of 0.68) compared to the isokinetic variables (highest AUC of 0.57).

Table 7.3.4.2 Univariate generalized linear mixed-models examining the association between sagittal plane external joint moments and hamstring strain injury during ground contact of a SLDJ task.

Independent Variable	P value	OR (95% CI)	Apparent AUC	LOOCV AUC (95% CI)
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.02*	2.11 (1.10 - 4.05)	0.73	0.58 (0.40 - 0.75)
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.003*	2.73 (1.29 - 5.80)	0.78	0.68 (0.52 - 0.84)
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.09		0.71	
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.63		0.71	
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.20		0.65	
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.77		0.70	
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.81		0.70	
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.56		0.70	
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 50 ms (per + 1 N.m/kg)	0.20		0.68	
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 100 ms (per + 1 N.m/kg)	0.30		0.69	
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 150 ms (per + 1 N.m/kg)	0.18		0.63	
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 200 ms (per + 1 N.m/kg)	0.97		0.69	

* denotes statistical significance ($P \leq 0.05$)

When viewed in combination, the findings of the thesis suggest that when conducting a PHE with the aim of gaining an insight into an individual's susceptibility of sustaining a contact MCL or ACL injury or a hamstring strain injury, researchers and sports medicine staff should examine external moments acting on an individual during a dynamic task rather than an isokinetic analysis of the knee joint. The caveat being that the selected task for biomechanical analysis should exposure players to a similar profile of joint kinetics to that of the inciting event. Therefore, to comprehensively analyse the risk of hamstring strain a different movement task may be required such as high-speed running.

7.3.5 Player workload: do we directly analyse exposure to the inciting event or other variables? And how long for?

The workload variables analysed in within the thesis represented components of external workload i.e. the workload imposed on the players due to the training routine and match play rather than internal workload i.e. the physiological response of the individual player to the external workload (Impellizzeri et al., 2019). The majority of these external workload variables related to exposure to the inciting event, with magnitude of high-speed running exposure analysed in relation to the odds of sustaining hamstring strain injury whilst running and the magnitude of tackle and ruck events sustained in relation to pooled MCL and ACL injuries sustained during a tackle or ruck event. These inciting events were analysed to test the hypotheses that a) unaccustomed increases in magnitudes of acute exposure to the inciting event in question were associated with an increased odds of injury and b) high magnitudes of chronic exposure to inciting events would decrease the odds of injury (Hulin et al., 2014; Windt and Gabbett, 2016; Malone et al., 2017; Malone et al., 2018; Malone et al., 2019). However, in Chapters 5 and 6 this was not observed to be the case for exposure to high-speed running and contact events, with larger magnitudes of acute exposure having no association to the odds of sustaining a hamstring strain and being associated with a decrease in the odds of sustaining pooled MCL and ACL injuries. Again, in contrast to the hypotheses of Windt and Gabbett (2016), chronic exposure to inciting events was observed to increase the odds of sustaining injury. These findings were supported by the recent work of West et al. (2020b), who reported a similar trend whilst examining the relationship between self-reported player workloads and soft tissue injuries in male professional rugby union players. Specifically, acute workloads were not associated with an increased odds of injury, whereas larger magnitudes of chronic workloads appeared to increase the odds (West et al., 2020b). These differences are possibly the result of the manner in which player workloads are managed in professional rugby union in comparison to other field sports and requires further investigation. However, it should also be noted that the for the majority of the study period the medical and conditioning staff were actively

employing workload management strategies inspired by the acute:chronic workload ratio (Hulin et al., 2014; Windt and Gabbett, 2016; Malone et al., 2017; Malone et al., 2018; Malone et al., 2019; Chapter 2, Section 2.4.4). As such, player workloads relating to high-speed running exposure and contacts were monitored for acute 'spikes', with workloads programmed to be high over a 28-day period. Due to the lack of previous research focus on the knee ligament injuries in relation to workloads, an additional external workload variable was also analysed derived from accelerometery termed PlayerLoad^{TM}. Rather than a count of the volume of exposure to the inciting events, this variable was an approximation of the total number of impacts or jolts an individual experienced (e.g. via contact with the ground or another player) during the timeframe of interest (Eager et al., 2016). This is dependent on both the tasks performed during the session, and how the individual moves during said tasks (Barreira et al., 2017; Hulin et al., 2020), which represented a construct of the on-pitch physical activity undertaken by the individual. In contrast to tackle and ruck exposure in Chapter 6, larger magnitudes of PlayerLoad^{TM} were related to an increased odds of knee ligament injury when aggregated over both acute and chronic timeframes. Simplistically, this suggests that for knee ligament injuries sustained during contact, a frequency count of exposure to the inciting event alone is not sufficient to increase the risk of injury and quantification of the location and magnitudes of force sustained for each contact may be more appropriate. Of note is the fact that PlayerLoad^{TM} was not part of the workload monitoring practice of the participating rugby team and as a result, an individual's workload was not modified when acute increases occurred. These findings suggest that when following the training philosophy of the acute:chronic workload ratio without sufficient periods of de-loading, the interaction between workload and injury aetiology may merely be shifted rather than nullified.

Another question raised by the field of workload – injury research is what constitutes 'acute' and 'chronic' workload? The injuries analysed within the thesis were classified as acute in nature with seemingly instantaneous onset resulting from excessive mechanical loads resulting in the failure of muscle fibres or ligaments (Bahr et al., 2020). The majority of research in the area of

player workload in relation to injury used a standard timeframe for acute and chronic workloads (7 and 28 days, respectively) (Gabbett, 2016). This approach was recently questioned (Carey et al 2017; West et al 2021c); however, the researchers did not examine the influence of the site of injury or the tissue type. In chapter four higher magnitudes of acute exposure to high-speed running was associated with a larger odds of sustaining hamstring injury when using a 3-day timeframe to calculate acute workload when compared to a 7-day time frame. However, negligible differences were observed between 3- and 7-day acute timeframes in chapter five when examining exposure to on-pitch physical activity in relation to contact MCL risk. Furthermore, in chapter five when contact MCL and ACL injuries were pooled, only a 7-day timeframe of exposure to on-pitch physical activity was significantly associated with injury. This was further demonstrated when looking at the timeframes for calculating chronic workloads. Longer timeframes of high-speed running exposure (14-day with a 7-day lag & 21-day with a 7-day lag) were observed to be more associated with hamstring injury risk in Chapter 5. The inverse was observed for knee ligament injury in Chapter 6, with the shortest timeframe of chronic exposure to on-pitch physical activity (7-day with a 3-day lag) exhibiting the strongest relationship to an increased risk of both contact MCL injury and pooled contact MCL and ACL injury. When viewed in combination, these findings provide support to the hypotheses proposed throughout the thesis that the influence workload has in relation in lower limb injury aetiology is both complex and specific to the site and tissue type of the injury of interest (Vanrenterghem et al., 2017; Impellizzeri et al., 2019a; Verheul et al., 2020; Kalkhoven et al., 2021). As such, a universal approach to determining the timeframes used to analyse the influence of acute and chronic workloads in relation to injury risk may be inappropriate and should be avoided where possible.

7.4 Impact of the thesis

A benefit of this industry part-funded PhD project was the close working relationship with the professional rugby team where the author was embedded and was therefore able to directly communicate the findings over the duration of the project and to witness their impact. These

impacts included engagement and knowledge transfer with the key stakeholder groups (e.g. sports medicine practitioners, strength and conditioning practitioners, rugby coaches and of course the participating players) as well as changes in practice regarding player welfare. The following section will discuss the impact arising from the thesis in relation to the professional rugby team co-funding the PhD.

7.4.1 Funding awarded by the participating professional rugby union team for a subsequent PhD project to examine academy player robustness combined with a change in policy to conduct PHE data collections on junior and senior academy players as well as objectively monitor player workloads.

Previous injury history was observed to be both associated with sustaining the high burden injuries examined within the thesis as well as displaying one of the highest injury classification performance values. Often injuries sustained by players earlier in their career were observed to leave them more susceptible to future injury. When these findings were presented to the key stakeholder groups, the resulting discussion highlighted a gap in the sports science support of the team and from the research project. The current focus was on collecting data from players in the senior playing squad rather than academy players, which in some cases meant at the midpoint or end point of their professional career. In the case of the participating team, individuals would start playing for the academy from the age of 13 – 16 years and potentially sustain injuries during this time. Therefore, the participating team provided further funding to gain an insight into the epidemiology and aetiology of injuries sustained by academy rugby players aged 13 – 16, 16 – 18 and 18 – 23 years old.

7.4.2 A change in policy to include biomechanical measures into the decision-making process when inferring lower limb injury risk and readiness to return to play.

Following presentation and discussion of the findings of Chapters 5 and 6 to medical and conditioning staff of the participating team, the decision was made to integrate biomechanical

variables into the decision-making process when inferring MCL, ACL and Hamstring strain injury risk. As previously stated, these biomechanical variables displayed a greater classification performance compared to the poorly performing isokinetic variables which should therefore enhance the risk mitigation process, improving player welfare.

7.4.3 Funding awarded by Innovate UK and the participating professional rugby union team through a Knowledge Transfer Partnership to further develop periodic health examinations and player monitoring during rehabilitation to examine lower limb injury risk specific to professional rugby union players.

The adoption of biomechanical variables into the team's injury risk decision-making policy combined with the findings of the thesis related to player workloads resulted in further collaboration with the participating professional rugby team. For the PhD, PHE data were only included from preseason data collections and were focussed on examining the existence of aetiological variables associated with the identified high-burned injuries. The purpose of the Knowledge Transfer Partnership was to further develop the current PHE and perform multiple collections throughout the season and with the integration of internal and external analysis of player workloads. An additional purpose was to collect biomechanical data throughout the time course of lower limb injury rehabilitation period.

7.4.4 Funding awarded by Red Bull UK to the professional rugby union team through the development of an on-site sports science laboratory at the stadium.

The adoption of sports science practices exhibited by the participating rugby union team during this PhD project was one of the key factors that resulted in the formulation of a partnership between the team and Red Bull UK, which led to the development of an on-site sports science laboratory at the team's stadium.

7.5 Limitations and directions for future research

In the thesis, the majority of research questions posed were addressed for the first time in the context of high burden lower limb injury in professional rugby union. As is common when conducting both novel research and working in an applied setting with professional sports teams, some limitations were unavoidable. This section therefore highlights the limitations from the author's reflections on the work and proposed potential areas of future research in relation to the following themes: 1. Research design and analysis, 2. human movement and 3. player workloads. Note that section 7.3.2 of this chapter has already discussed issues related to sample size and class imbalance.

7.5.1 Research design and analysis: Limitations and directions for future research

7.5.1.1 Multivariate approach

For the analyses conducted within the thesis a univariate approach was employed (Chapters 3, 5 & 6). Recent commentaries and work in other fields suggest that this does not account for the inherent complexity associated with classification of injury during participation in professional team sport (Bahr and Holme, 2003; Quatman et al., 2009; Mendiguchia et al., 2012; Nielsen et al., 2016). The reason for adopting a univariate analysis was that the majority of analyses were novel, being either the first conducted in a professional rugby union population at the time of writing (2022) in the case of chapter 5, or the first conducted in the research space in the case of examining aetiological variables associated with contact MCL injury in chapter 6. Due to this, it was the work in this thesis sought to lay the foundations for subsequent analysis. Furthermore, recent models of injury aetiology research have highlighted the need for a paradigm shift from conventional multivariate statistics, where the interpretation relates to the influence of an increase of the independent variable of interest when all other covariates are held constant to examining the states brought about by the interactions between the multiple independent variables (Bittencourt et al., 2016; Hulme et al., 2017; Hulme et al., 2019). However, this is still an emerging concept in the field and was outside of the scope of the current project.

7.5.1.2 Linear vs non-linear analysis: embracing sports injury as a complex problem

The analyses used to examine the association between the hypothesised aetiological variables and high burden injuries assumed that a mainly linear relationship existed between the logarithm of the odds of the probability of being injured and one or more independent variables (Hosmer et al., 2013). However, a assuming a linear relationship exists between injury and independent variables may be naïve (Impellizzeri et al., 2020; Kalkhoven et al., 2021). In chapters 5 independent variables were analysed as dichotomous groups, employing a similar approach as previous workload-injury aetiology research which has reported a quadratic relationship between exposure to workload and injury in a multitude of settings (Gabbett, 2016). This approach is considered quasi-non-linear as the work of the thesis and the majority of previous studies used a logistic regression rather than a truly non-linear approach such as a polynomial logistic regression (Ruddy et al., 2019). However, employing true non-linear analysis is complex and was out of the scope of the current project.

7.5.2 Analysis of human movement: Limitations and directions for future research

7.5.2.1 Biomechanical analysis of external vs internal moments and forces

The biomechanical analyses conducted in chapters 6 and the subsequent analyses conducted in section 7.3.4.2 of the discussion employed an inverse dynamics approach, where joint moments were modelled as the net rotational force acting on a joint as a result of force external to the body such as ground reaction force. When conducting inverse dynamic analyses, internal moments are modelled as equal and opposite in magnitude to the external joint moments; however, numerous musculoskeletal modelling studies that have employed a forward dynamics approach have observed this approach to be incorrect with discrepancies between internal and external joint moments reported. This may have explained the reported lack of association between sagittal plane external ankle dorsiflexion moment and knee ligament injuries in chapter 6 in comparison to the relationships observed by Maniar et al. (2020) who employed a forward

dynamics approach. Although the use of a forward dynamics approach was the preferred choice for chapter 6, the EMG system at the University was wired rather than wireless and was therefore not compatible with the batch method of participant preparation during the PHE data collection sessions (See Discussion section 7.3.4.2). Furthermore, participant preparation would have increased player PHE data collection time to an unacceptable level.

7.5.3 Player workloads: Limitations and directions for future research

7.5.3.1 Analysis of external vs internal player workloads

All player workload variables analysed within chapters 4 and 5 of the thesis were exposure to external workloads, which can be defined as physical work undertaken during match play and/or prescribed during training (Vanrenterghem et al., 2017; Impellizzeri et al., 2019a; Verheul et al., 2020; Kalkhoven et al., 2021). However, the findings within the thesis chapters suggest that the accumulation of excessive magnitudes of external workloads over a 4-week time and 10-day time course for hamstring injury and knee ligament injury respectively. This suggests that individuals may be in a state of peripheral fatigue when injured, therefore examining the body's psychophysiological responses to cope with the requirements of the external workloads applied, termed internal workload (Vanrenterghem et al., 2017; Impellizzeri et al., 2019a; Verheul et al., 2020; Kalkhoven et al., 2021) may have been a more prudent and resulted in a consistent data collection modality. However, the purpose of the thesis was primarily to explore the initial stages of aetiological relationships between workload and injury for specific injuries and as such examining internal workloads was outside the scope of the thesis. It is, however, recommended that future research examine the relationship between internal workloads, hamstring injury and contact knee ligament injury.

7.6 Practical recommendations

The following section aims to distil the findings contained within the thesis and provide some practical recommendations to the key stakeholders of the male professional rugby union team

participating in the PhD project in addition to sports medicine and strength and conditioning practitioners in the wider professional rugby union community involved in the day-to-day decision-making processes of player physical preparation and injury risk mitigation.

7.6.1 Aetiological factors associated with high burden lower limb injuries in professional rugby union players

In chapter 3 an epidemiological review of the injuries sustained by the participating team was conducted. The review found that lower limb injuries placed the highest burden compared to the upper limbs, trunk, and head & neck over the course of the project. ACL injury sustained during contact events resulted in the highest injury burden, followed by injuries to the MCL sustained during the tackle and rucking and biceps femoris strain sustained during running. Variables hypothesised to be associated with the aetiology of these high burden injuries were examined in chapters 5 and 6. The findings of these studies and the arising recommendations are contained within the infographics below (Figures 7.6.1a and 7.6.1b) to aid the medical and conditioning staff based at professional rugby union teams with the decision-making process of inferring injury risk.

The majority of the data analysed within chapter 5 was collected over a longer period of time when compared with chapter 6, and as such there were a larger number of hamstring injury cases to analyse. In chapter 5, all variables bar previous injury were analysed by being separated into dichotomous groups (e.g. the odds of sustaining injury in relation to being within low hamstring strength group vs being in the medium hamstring strength group) where the medium group was the reference. This decision was due to the larger number of hamstring injuries sustained during the project, the larger body of scientific literature to draw from in comparison to chapter 6, and due to the request of the co-funding professional rugby union team. In contrast, there was less scientific evidence to draw from for the injuries analysed in chapter 6. Because of this, the variables were analysed in a continuous manner (e.g. the odds of sustaining injury in relation a + 1 N.m/kg unit increase in knee abduction moment).

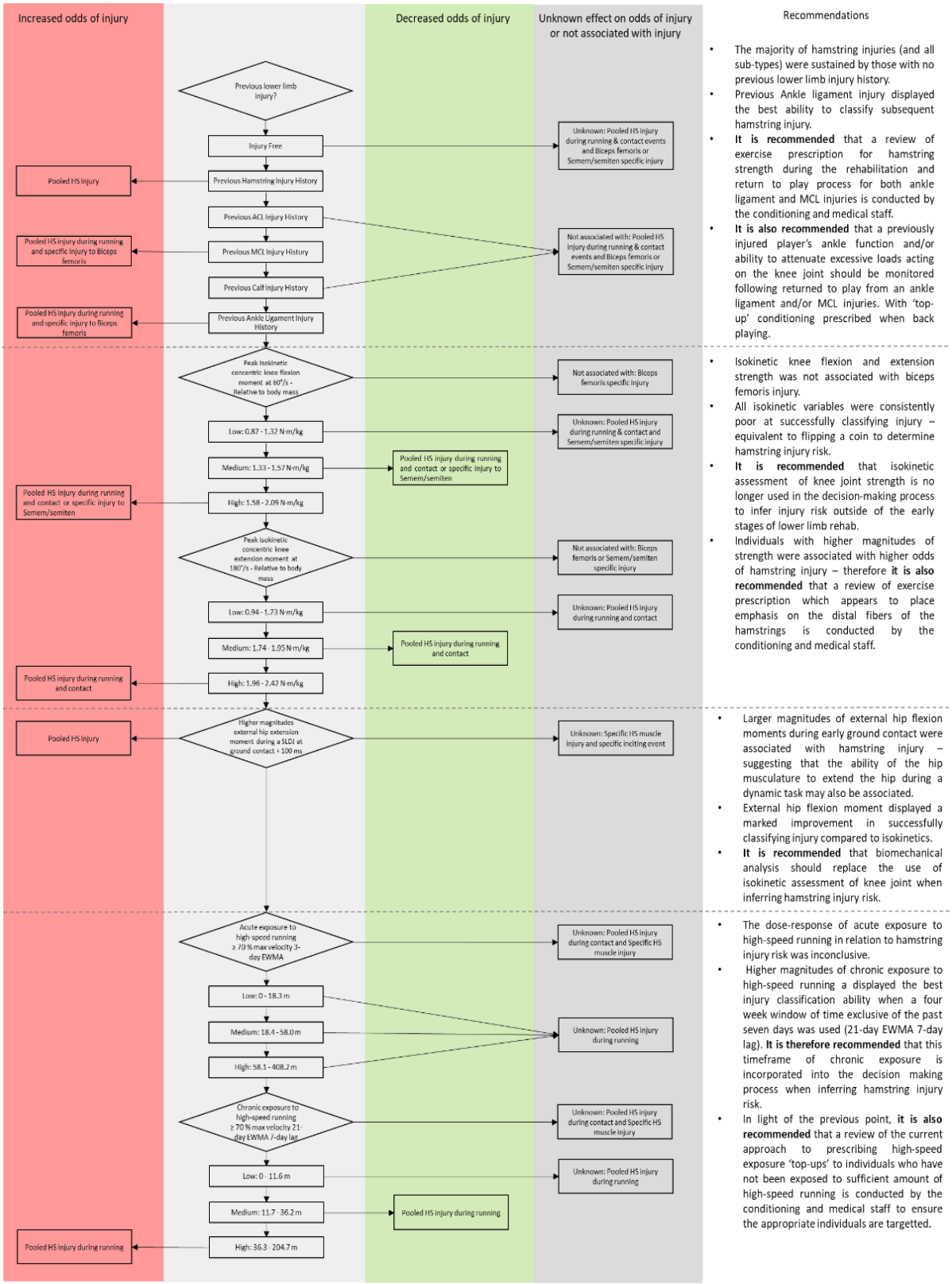


Figure 7.6.1a Practical recommendations related to hamstring strain injury aetiological variables examined in Chapter 5.

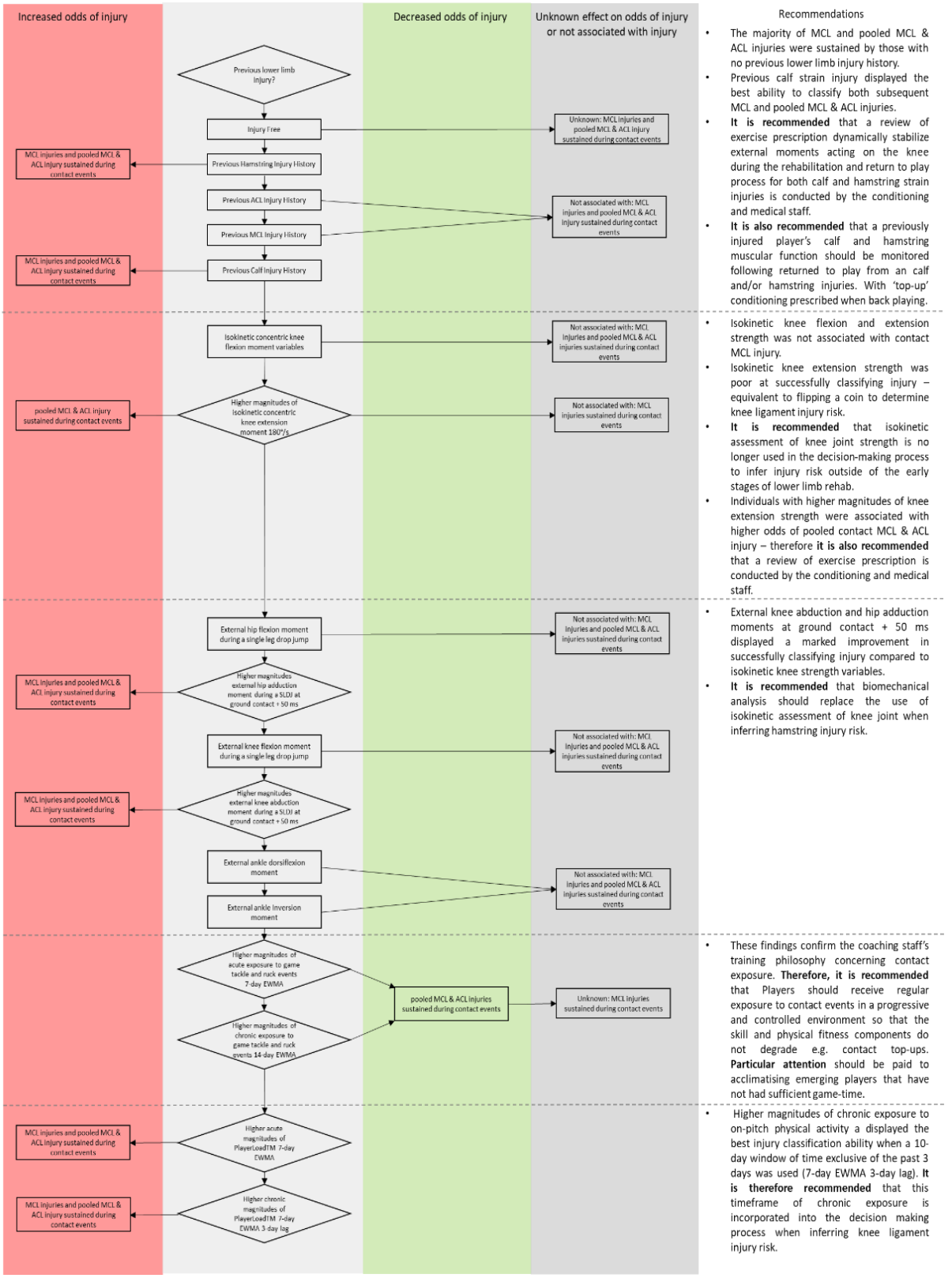


Figure 7.6.1b Practical recommendations related to contact MCL and ACL aetiological variables examined in Chapter 6.

7.6.2 Technical note 1 - What to include in a periodic health examination: Open-chain strength or closed-chain movement analysis?

The findings of chapters 5 and 6 suggest that, in its current form, isokinetic assessment of knee extension and flexion strength is poor at classifying individuals with either hamstring strain injury, contact MCL or ACL injury. These findings are in agreement with previous larger scale studies examining hamstring strain injury (Green et al., 2017; Green et al., 2020; Bennell et al., 1998; Zvijac et al., 2013; van Dyk et al., 2016). Therefore, relying on these variables may actually do more harm than good, detracting from the baseline knowledge and intuition of the experienced medical and conditioning staff working closely with the players. As a result of these findings, it is recommended that isokinetic assessment should be limited to the early stages of lower limb rehabilitation when restoring open chain strength knee extension and flexion strength is the goal for both knee ligament and hamstring injuries. Outside of the early stages of rehabilitation, analysis of the external moments during a dynamic task should be used, such as a single-leg drop jump.

7.6.2.1 Including biomechanical analysis in a PHE: Potential barriers to adoption and solutions.

1. The cost of a motion capture system has commonly been cited as barrier to the adoption of biomechanical analysis into the injury prevention decision-making processes in professional team sport. However, due to the advances in motion capture technology systems the price when combined with a force platform is comparable to that of an isokinetic dynamometer.
2. A second cited barrier to adoption is the space required to cite the force platform and perform the biomechanical analysis. This was overcome by the team participating in the research project by partnering with a local university and in the later stages of the project, embedding a force platform within the pushing sled track of the gym. Resisted sled pushing exercise is now a foundational part of conditioning in professional rugby union (Bourne et al., 2018; Thorborg et al., 2020), and as such all of the professional rugby union competing in the English Premiership have these facilities.
3. A third cited common barrier to adoption is the time required to collect and process motion capture data compared to isokinetics and more recently force platforms and devices measuring force during Nordic hamstring exercise. However, the data collected in this thesis should dispel this notion. The author was allotted two days per pre-season PHE to collect data on ~45 players. During these days each player had a 1 hour 45 min to 2-hour timeslot to undergo an isokinetic assessment of knee joint flexion and extension strength and a biomechanical assessment of a single-leg drop-jump task in addition to other components of the PHE. The figure below is an anonymised schedule

from one of the test days to illustrate the timing and organisation required to achieve this (Figure 7.6.2.1). The main failure point of the mass PHE days was ensuring enough trained personnel were present to cover all of the testing stations. In addition to the sports science support duties at the co-funding professional rugby union team the author taught on undergraduate and post graduate modules so could recruit interested individuals who wanted to get hands on experience of working in with professional athletes. These individuals were trained by the author over a two-month period to ensure they collected data accurately in a professional manner and were able to troubleshoot any problems that arose during all within the timeframes set by the co-funding professional rugby union team.

Forwards Testing - Tuesday 03rd of July 2018

Time	Tests	Room	Players	Player Notes	Testers
8.00	Knee Isokinetics Shoulder Isokinetics Cogsport & SCAT Gait Analysis & Drop to Stabilise	BC 23 RB 23 RB 23 RB 35		 Shoulder, Cogsport & SCAT Only No shoulder Isokinetics	
10.00	Knee Isokinetics Shoulder Isokinetics Cogsport & SCAT Gait Analysis & Drop to Stabilise	BC 23 RB 23 RB 23 RB 35			
12.00	Knee Isokinetics Shoulder Isokinetics Cogsport & SCAT Gait Analysis & Drop to Stabilise	BC 23 RB 23 RB 23 RB 35			
14.00	Knee Isokinetics Shoulder Isokinetics Cogsport Gait Analysis & Drop to Stabilise	BC 23 RB 23 RB 23 RB 35			
16.00	Knee Isokinetics Shoulder Isokinetics Cogsport Gait Analysis & Drop to Stabilise	BC 23 RB 23 RB 23 RB 35			
18.00	Finish				

All Players bring gym shorts, vest and skins shorts. Reserved parking at University of Exeter - St Lukes Campus Magdelan Rd carpark EX1 2LU. All players cycle through all tests unless specified

Figure 7.6.2.1 An anonymised example PHE testing schedule to illustrate the time demands and testing personnel required

7.6.3 Technical note 2 - Measuring and analysing external workloads in relation to lower limb injury risk: no silver bullets.

The findings of both chapters 5 and 6 suggest that not only does the relationship vary between the dose of external workload and response of the risk of injury for different injuries; but also, different modalities of external workloads may be more or less related do different tissue types of injury (i.e. GPS high-speed running exposure and hamstring injury vs PlayerLoad^{TM} exposure

to on-pitch physical activity vs MCL and ACL injury). As such it appears that relying on one modality of external workload such as GPS may not be adequate for inferring injury risk for all lower limb injury scenarios. If using high-speed running in isolation to infer the odds of sustaining hamstring injuries chronic exposure should be modelled over either 1) a 14 Day EWMA with a 7 day lag; or 2) a 21 day EWMA with a 7 day lag. It is also recommended that PlayerLoad™ is incorporated into decision making process when monitoring player external workloads in relation to contact MCL and pooled MCL& ACL ligament injury risk, using either a 3 or 7 day EWMA for to model acute workloads and a 7 day EWMA with a 3 day lag to model chronic workloads.

7.6.3.1 Incorporating exposure to PlayerLoad™ into the daily player workload monitoring process: Potential barriers to adoption and possible solutions.

A barrier to the adoption of accelerometer derived variables into the injury prevention decision-making processes is how to interpret what the arbitrary units of PlayerLoad™ mean in comparison to a more straightforward metric such as 'distance travelled in a given relative speed zone' obtained via GPS (Reardon et al., 2015; West et al., 2019). The author advices medical and conditioning staff to interpret PlayerLoad™ as an approximation of the total number of jolts or impacts (jerk = change in acceleration) an individual experiences during the timeframe of interest (Eager et al., 2016) which is dependent on both the tasks performed during the session, and how the individual moves during said tasks (Barreira et al., 2017; Hulin et al., 2018). In simple terms, this means that a player could flag as having high PlayerLoad™ values from the following pitch-based sessions: a big contact session for a back rower, a session where a second-row performs a large number of lineouts and/or a set of small-sided games where a winger stays on for the entire duration. This is supported by previous evidence which suggests that PlayerLoad™ provides a more holistic picture of on-pitch physical activity rather than relying solely on GPS derived variables which are unable to accurately quantify rapid changes in direction and impacts from contact events (Gabbett et al., 2015; Roe et al., 2016; Granger et al., 2018).

With the above in mind, it is recommended that PlayerLoad™ is incorporated in the decision-making process to deload players' on-pitch activity (e.g., higher magnitudes outside of player's normal range = less on-pitch training) to prevent non-functional overreaching (i.e. 'a pull'). This is in contrast to high-speed running exposure that is commonly used to: 1. deload players who have been exposed to higher magnitudes of high-speed running than what their normal range is (i.e. 'a pull'); and 2. 'Top-up' a player's exposure to high-speed running if they have not been exposed for a sustained period of time (i.e. 'a push') (Stokes et al., 2020). It should be noted that it would not be appropriate to use PlayerLoad™ as a means to 'push' players due to the all-

encompassing nature of what it represents (i.e. any work done when the player tracking system is recording). However, when combined with exposure to high-speed running and exposure to contacts, the addition of PlayerLoad^{TM} would comprise a comprehensive approach to player external workload monitoring for professional rugby union players.

7.7 Thesis Conclusion

The purpose of the thesis was to identify high burden lower limb injuries in men's professional rugby union and where possible identify variables associated with the aetiology of these injuries. These injuries were: hamstring muscle strains, particularly biceps femoris strains sustained while running, and MCL and ACL sprains sustained during tackle and ruck events. The aetiology of these injuries is complex and the work within the thesis suggests that each injury may have a specific constellation of associated variables. Being embedded within a professional rugby union team competing at the top flight of English competition enabled a substantial amount of rich data to be collected longitudinally which was unique in comparison to other studies. The findings of the prospective studies suggest that isokinetic assessment of knee extension and flexion strength in its current format has limited value in relation to injury risk classification and where possible a biomechanical analysis of a more complex dynamic task may provide a better tool. Furthermore, the work within the thesis suggests that a one-size-fits-all approach to analysing player workload may not be appropriate with the mechanisms associated with muscle strain and ligament sprains differing in acute and chronic time scales and the influence of exposure to the inciting event.

Chapter 8

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Chapter 9 Appendix: 9.1 Methodological study one: Baseline accuracy and reliability of the coda cx1 system

9.1.1 Introduction

Three-dimensional motion capture systems are used in biomechanical research and clinical practice. Evaluating the accuracy and reliability of a three dimensional motion capture system is vital to ensure that the variations observed in data are due to real changes rather than error (Maynard *et al.*, 2003). The accuracy and reliability of the Coda cx1 active marker system (Charnwood Dynamics Ltd, Leicestershire) has received little attention, with only one previous study having investigated the reliability of lower limb joint angles using the cx1 system (Kiernan *et al.*, 2014). Although favourable inter-rater and intra-rater reliability was observed in lower limb joint angles, the baseline accuracy and reliability of the cx1 system was not examined. Therefore, the aim of this study was to quantify the accuracy and reliability of marker trajectory data obtained from the Coda cx1 system.

9.1.2 Methods

Four active markers were attached to each spherical end of a section of Peak Motus calibration frame (Peak Performance, Englewood, USA) used as a calibration object in the investigation which had a known length of 0.520631927 m.

Marker trajectory data were collected at 200 Hz with four synchronised Coda cx1 units (Charnwood Dynamics Ltd, Leicestershire). Static and dynamic measurements of the calibration object were collected. Static measurement consisted of five 5-second trials collected with the calibration object positioned in the centre of the testing area. Dynamic measurement consisted of five 5-second dynamic trials with the calibration object moved primarily in the following directions: medio-lateral (X), anterior-posterior (Y) and vertical (Z). This process was repeated seven days after the initial session. Marker trajectory data was filtered using a zero-lag fourth

order Butterworth low-pass filter with a cut-off frequency of 12 Hz in MATLAB R2012a (The MathWorks Inc, USA). Virtual centroids were calculated from the filtered marker data to estimate the centre of each of the spherical ends of the calibration object, the distance between the two centroids was calculated to provide a resultant segment length for the longitudinal axis. The values were compared to the known length of the calibration object to determine the accuracy, between and within trial reliability. The accuracy of the system was assessed by calculating the route mean square error (RMSE) and the percentage coefficient of variation (CV %) was also calculated to determine the within and between session reliability.

9.1.3 Results & Discussion

All conditions displayed relatively low RMSE values, with the largest observed during movement in the X and Y axes (Table 9.1). Additionally all conditions displayed low CV values both within and between sessions. The results of the study suggest that the Coda cx1 system has acceptable accuracy and reliability for three dimensional maker trajectory data. This supports the previous findings of Kiernan *et al.* (2014), giving confidence in the accuracy and reliability of marker trajectory data collected using the Coda cx1 system.

Table 9.1.3 Difference in the measured and known length of the calibration object, root mean square error and coefficient of variation for static and dynamic trials.

	Static Difference (mm)	Dynamic X Axis Difference (mm)	Dynamic Y Axis Difference (mm)	Dynamic Z Axis Difference (mm)
Actual	520.631927			
Session 1				
Trial 1	0.05	-0.62	0.36	0.62
Trial 2	0.03	1.26	1.17	-0.24
Trial 3	0.03	0.28	-0.52	-0.55
Trial 4	1.04	-0.45	-1.52	0.42
Trial 5	0.01	-0.34	0.24	0.35
Mean	520.86	520.66	520.58	520.75
SD	0.45	0.77	1.02	0.49
CV%	0.001	0.001	0.002	0.001
Session 2				
Trial 1	0.03	0.56	0.02	-0.36
Trial 2	-0.01	-1.44	0.87	0.47
Trial 3	-0.98	-0.34	1.01	-0.94
Trial 4	-0.01	0.89	0.59	-0.02
Trial 5	0	1.17	0	0.53
Mean	520.44	520.8	521.13	520.57
SD	0.44	1.06	0.47	0.61
CV%	0.001	0.002	0.001	0.001
RMSE	0.45	0.84	0.79	0.51
Pooled Mean	520.65	520.73	520.85	520.66
Pooled SD	0.48	0.88	0.8	0.53
Inter Session CV %	0.001	0.002	0.002	0.001

9.2 Methodological study two: Between session reliability of lower limb moments during a single-leg drop jump task

9.2.1 Introduction

Altered joint kinetic variables have previously been associated with knee ligament injury aetiology. However, when conducting prospective research over multiple years, establishing the reliability and accuracy of equipment is essential (Maynard et al., 2003). The baseline accuracy and reliability of the Coda cx1 active marker system (Charnwood Dynamics Ltd, Leicestershire) previously been established to be within an acceptable range (See section 9.1 of this chapter). The aim of this study was to quantify the between session reliability of lower limb joint moments collected using the Coda cx1 system synchronised with an AMTI BP400600HF force plate.

9.2.2 Methods

9.2.2.1 Participants

Eighteen injury free male professional English Premiership Rugby Union players participated in the study. The study was part of a larger study that was approved by the University of Exeter Sport and Health Sciences ethics committee.

9.2.2.2 Biomechanical data collection and analysis

Kinematic data were collected using four CODA cx1 units sampling at 200 Hz (Charnwood Dynamics, Rothley, Leicestershire, UK) with force data collected synchronously using a 0.6 m x 0.4 m force platform sampling at 1000 Hz (BP400600HF, AMTI, Massachusetts, USA). Biomechanical analysis of lower limb joint moments during a single-leg drop jump task was conducted using the same data collection and analysis procedures in Chapter 6 Section 6.3.2.3. Following the first data collection the participants attended a second data collection session conducted in the same conditions.

9.2.2.3 Statistical analysis

A 95% limits of agreement analysis was selected to measure the absolute reliability of joint moment data between data collection sessions, as well as providing estimates of both systematic bias and random error (Altman and Bland, 1983; Bland and Altman, 1996a; Bland and Altman, 1996b; Atkinson and Nevill, 1998).

9.2.3 Results and Discussion

A primary aim of the Thesis was to explore whether altered lower limb biomechanics were associated with an increased odds of sustaining knee ligament injury over. This study was conducted over multiple seasons and as a result, acceptable between session reliability of the three-dimensional joint angles and moments derived from the CODA cx1 system and AMTI force plates were key.

The findings of the current study confirmed that sagittal and frontal plane three-dimensional joint kinetics collected during the early ground contact phase of a single-leg drop jump task exhibited negligible magnitudes of systematic bias between data collection sessions (Table 9.2.3, Figure 9.2.3), in relation to the magnitudes of joint moments observed in Chapter 6.

Table 9.2.3 Between session reliability of lower limb kinematic and kinetic data a single-leg drop jump task.

Independent Variable	Systematic Bias (N.m/kg)	95% LOA (N.m/kg)	Random Error (N.m/kg)
Hip Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms	-0.11	-0.55 - 0.33	± 0.44
Hip Joint Adduction(+)/Abduction(-) Moment at Ground Contact + 50 ms	0.04	-0.44 - 0.52	± 0.48
Knee Joint Extension(+)/Flexion(-) Moment at Ground Contact + 50 ms	0.04	-0.38 - 0.45	± 0.42
Knee Joint Abduction(+)/Adduction(-) Moment at Ground Contact + 50 ms	-0.04	-0.29 - 0.22	± 0.25
Ankle Joint Plantarflexion(+)/Dorsiflexion(-) Moment at Ground Contact + 50 ms	-0.07	-0.73 - 0.6	± 0.67
Ankle Joint Inverion(+)/Eversion(-) Moment at Ground Contact + 50 ms	0.01	-0.18 - 0.2	± 0.19

When compared to previous research examining the between session reliability of single-leg drop jump task, similar magnitudes were reported for between session random error of hip extension, hip adduction, knee abduction and ankle inversion moments (Myer et al., 2015).

However, in the present study larger magnitudes of random error were observed for knee extension and ankle plantar flexion moments compared to that of Myer and colleagues (2015). This finding may possibly be due to the difference in participant population (Female high-school volleyball players) and the associated difference in muscle mass distribution of the thigh and calf segments where the tracker markers were placed. When examining the magnitudes of random error in relation to the magnitudes of joint moments observed in Chapter 6, the magnitude of random error associated with hip adduction appeared to be larger to the mean difference between the injured and non-injured groups, whereas the magnitude of random error associated with knee abduction moment was smaller. These findings may additionally explain why the classification ability of hip adduction moments were less than knee abduction moments.

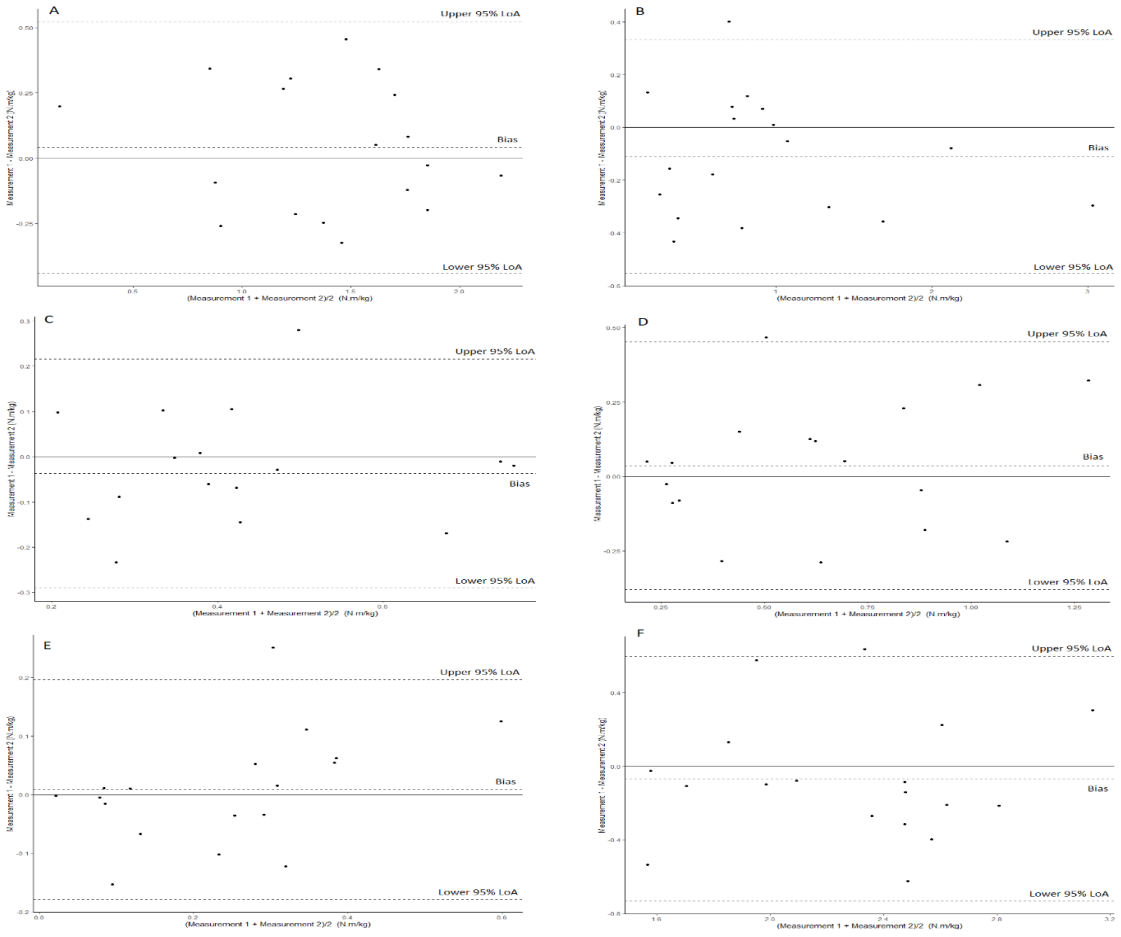

Figure 9.2.3 Bland-Altman plots displaying bias, upper and lower 95% limits of agreement (LoA) for the differences in joint moment between sessions (Y axis) plotted against the inter session mean of the joint moments (X axis) for: A) Hip Add/Abduction moment, B) Hip Ext/Flexion moment, C) Knee Abd/Adduction moment, D) Knee Ext/Flexion moment, E) Ankle Inv/Eversion moment and F) Ankle Plant/Dorsiflexion moment at 50ms + ground contact.