LONGITUDINAL WORKLOAD MONITORING TO KEEP ATHLETES HEALTHY AND PERFORMING: CONCEPTUAL, METHODOLOGICAL, AND APPLIED CONSIDERATIONS IN SPORTS INJURY AETIOLOGY

by

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Abstract

Sports injury aetiology is a process in which internal and external risk factors contribute to an inciting event. The last decade has seen a rapid growth in research that identified how training and competition workloads relate to sports injury risk. My literature review highlighted that existing aetiology models do not describe how workloads contribute to injury. Furthermore, injury risk fluctuates on a time-scale in parallel to these workloads (e.g. daily), which creates several methodological and statistical challenges that have largely been ignored. If researchers are to understand how athletes’ workloads relate to injury risk, their conceptual aetiological models must be updated to incorporate training and competition workloads, and they should use appropriate statistical analyses.

After my literature review, I divide this dissertation into three parts. In Part 1 I discuss how workloads relate to injury. I present a novel workload—injury aetiology model, which expands on previous aetiological frameworks and details 3 ways that workloads contribute to injury: 1) exposing athletes to external risk factors and potential inciting events, 2) reducing injury risk through beneficial physiological changes, and 3) increasing injury risk through transient negative changes in athletes’ internal risk profiles. I then present mediation and moderation as potential causal approaches to understand how athlete risk factors interact with workload changes to alter injury risk.

In Part 2 I tackle the methodological challenges of analysing workload—injury data. I reviewed prospective cohort studies that reported intensive longitudinal data to analyse workload—injury data in team sports. I identified that few studies utilised statistical approaches that align with...
theoretical aetiology models or addressed the methodological challenges associated with longitudinal data. My analysis leads me to recommend mixed modeling as one advance, and I exemplify how it can be used by studying how player unavailability affects player outputs.

In Part 3 I integrate the conceptual and methodological considerations into two applied settings. First, I describe how a methodological/mathematical concern (mathematical coupling) may influence applied practice (multifaceted player load management) and research (explicitly reporting calculations). Finally, I use mixed modeling to examine pre-season workload and in-season injury risk, controlling for athletes’ weekly workloads.
Lay Summary

Participating in sport provides many health benefits. However, every minute a person participates in sport, they are exposed to potential injury risk.

The last two decades of sports injury research identified multiple ‘risk factors’ that contribute to sports injuries. These include athlete characteristics (e.g. strength, age, flexibility) and environmental characteristics (e.g. aggressive opponents, playing surface) that may protect or predispose individuals to injury. Until recently, how sport participation itself inherently changes an individuals’ injury risk in the next game or practice session has been largely absent.

In this dissertation I provide conceptual and methodological frameworks for how sport participation relates to an individual’s injury risk. Ultimately, understanding how sport participation contributes to injury and analysing these data more appropriately may inform future injury prevention efforts to keep more athletes healthy and performing.
Preface

I, Johann Dirk Windt, composed this dissertation in its entirety, with guidance and input from my supervisory team – Drs. Karim Khan, Tim Gabbett, Bruno Zumbo, and Ben Sporer. All major chapters within the dissertation body have been published in peer-reviewed journals, except chapter 5, which has been accepted and is in Press at BMJ Open. The details of each publication are included below.

A version of chapter 3 has been published in the British Journal of Sports Medicine. I conceived the idea for the manuscript and wrote the first draft, while Professor Gabbett contributed to the critical revision of the manuscript.


A version of chapter 4 has been published in the British Journal of Sports Medicine. I conceived the idea for this manuscript and wrote the first draft. All the other authors contributed to critical revision of the manuscript.

A version of chapter 5 is in Press at *BMJ Open* (Accepted September 4\(^{th}\), 2018). I conceived the idea for the paper through discussing longitudinal data analysis with Prof. Zumbo. I designed the search strategy with Dr. Clare Ardern and ran the search. Dr. Tim Gabbett and I reviewed all the identified systematic reviews and consensus statements to identify relevant papers. Dr. Clare Ardern and I identified original prospective cohort studies in the included systematic reviews/consensus statements that met our inclusion criteria. We also coded the articles according to the 50+ methodological criteria we established. I wrote the first draft of the manuscript. All authors were responsible for critical revision of the original manuscript and its revised version.


A version of chapter 6 has been published in *Journal of Science and Medicine in Sport*. I was primarily responsible for the study design (in collaboration with Drs. Ekstrand and McCall), data analysis (in collaboration with Dr. Zumbo) and writing the first draft of the manuscript. All authors contributed to critical revision of the manuscript.

A version of chapter 7 has been published in the *British Journal of Sports Medicine*. I simulated the data for this project and wrote the initial manuscript. Dr. Gabbett provided valuable input at the initial conception of the idea and throughout the manuscript revision process.


A version of chapter 8 has been published in the *British Journal of Sports Medicine*. Data collection for this chapter was completed by Daniel Ferris and Dr. Tim Gabbett with the Gold Coast Titans. The chapter started with a discussion between Prof. Tim Gabbett and me where we wanted to know whether players who completed more of the pre-season were better able to handle in-season demands. I performed the analysis and wrote the first draft of this manuscript, with input from all authors to refine the paper.


*Note my co-supervisor, Professor Karim Khan, is Editor in Chief of the *British Journal of Sports Medicine*. BJSM is the #1 ranked journal in the Thomson-Reuters journal category of Sports Science/Sports Medicine (of 81 journals, impact factor 7.867). He was not involved in handling of any of my papers at the British Journal of Sports Medicine.*
**Statement of Research Ethics Approval:**

No ethics approval was sought or needed for Chapters 3, 4, 5, or 7. Chapter 6 was approved by the UEFA research ethics board. Chapter 8 was approved by the Research Ethics Board of Australian Catholic University.

**Use of Published Material and Active Voice**

The UBC Faculty of Graduate and Postdoctoral Studies does not specify whether doctoral dissertations are to be written in the first or third person, in the active or passive voice, and examples of both can be seen on cIRcle – UBC’s online dissertation repository. Similarly, the Experimental Medicine Program does not specify a preferred style. As the leading medical journals in the field (e.g. JAMA, Lancet, The BMJ) recommend first person, active voice, my committee and I agreed that I would follow these journals’ author instructions for this dissertation.

All previously published material in this dissertation is reproduced with permission from the relevant journals (British Journal of Sports Medicine for Chapters 3, 4, 7 & 8, BMJ Open for Chapter 5, Journal of Science and Medicine in Sport for Chapter 6). Therefore, these papers (making up Chapters 3-8 of this dissertation) include plural language for the author team – ‘we’ instead of ‘I’. These pronouns were not changed for this dissertation. As a result, the reader will find what will, on the surface, appear to be ‘inconsistency’ in the voice between Chapters 1-2 and 9 (written specifically for the thesis), and Chapters 3-8 (published/accepted manuscripts).
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List of Abbreviations

ACL: Anterior Cruciate Ligament
ACWR: Acute:Chronic Workload Ratio
AFL: Australian Rules Football League
AIC: Aikeke Information Criteria
AMPK: 5' Adenosine Monophosphate-Activated Protein Kinase
ANOVA: Analysis of Variance
BIC: Bayesian Information Criteria
CI: Confidence Interval
DAG: Directed Acyclic Graph
DALDA: Daily Analysis of Life Demands
UEFA: Union of European Football Associations
GEE: Generalised Estimating Equation
GPS: Global Positioning Systems
HLM: Hierarchical Linear Modeling
HR: Heart Rate
ILD: Intensive Longitudinal Data
IOC: International Olympic Committee
MAR: Missing at Random
MBI: Magnitude Based Inferences
MCAR: Missing Completely at Random
mTOR: Mammalian Target of Rapamycin
NFL: National Football League
OR: Odds Ratio

OSTRC: Oslo Sports Trauma Research Centre

POMS: Profile of Mood States

PROM: Patient-Reported Outcome Measure

REST-Q: Recovery Stress Questionnaire

RR: Relative Risk

sRPE: session Rating of Perceived Exertion

TRIMP: Training Impulse

TRIPP: Translating Research into Injury Prevention Practice

TSB: Training Stress Balance

UCL: UEFA Champions League

UEFA: Union of European Football Association
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Dedication

Everly, my beloved daughter – I love you.

To my Lord, my wife, my family and friends.
Chapter 1: Introduction

While sport provides a means of physical activity and is thereby associated with a plethora of health benefits [1,2], participation comes with an inherent injury risk. While certain injuries occur more frequently in the presence of certain risk factors – e.g. higher anterior cruciate ligament (ACL) injury risk in female athletes [3] – all sports injuries occur during training or competition. Beyond simple ‘exposure’ to injury risk, the recursive nature of injury risk means that each training or competition event may alter individuals’ subsequent risk [4]. To understand the integral role of training and competition workloads in sports injury development, it is necessary to understand both 1) sports injury prevention and 2) workload monitoring. Once I have summarised these foundations, I will describe (i) how these two fields interact, (ii) the methodological and statistical challenges that have limited progress to date, and (iii) how both conceptual and methodological considerations may impact applied practice and research.

1.1 Sports injury research foundations

Sports injury research begins with a fundamental question: what constitutes a sports injury? After defining ‘sports injury’, I will describe the burden that sports injuries pose at the public health, organisation, and individual athlete levels. I will then outline the principles of injury prevention through the lens of Professor Willem van Mechelen’s ‘sequence of prevention’ model [5].
1.1.1 What is a ‘sports injury’?

Injuries occur when the transfer of physical forces exceed the body’s ability to handle them, and structural damage occurs [6]. While acknowledging that differentiating ‘disease’ and ‘injury’ presents several theoretical challenges [7], the above definition provides a basis to ground sport injury discussions. It also differentiates musculoskeletal sports injuries from other sports-related conditions, such as sports-related asthma or cardiac events. To operationalise the definition, I will outline the key characteristics of sports injury research – the definition, severity, mechanism, and classification of injuries.

1.1.1.1 Defining ‘cases’ in sports injury research

Injury definition is vital in the science of epidemiology [8]. Prior to 2005, few efforts were made to reach consensus on sports injury definitions [6]. A landmark sports injury definition was published in 2006 following the 1st World Congress on Sports Injury Prevention, where an expert panel defined a football injury as [9]:

“Any physical complaint sustained by a player that results from a football match or football training, irrespective of the need for medical attention or time loss from football activities. An injury that results in a player receiving medical attention is referred to as a ‘medical attention’ injury, and an injury that results in a player being unable to take part in future football training or match play as a ‘time-loss’ injury.” [9]

Other sports (e.g. cricket, rugby league, rugby union) have since adopted similar consensus definitions [10–15]. Within these frameworks, injuries may be divided into 3 separate categories.
1) **Any physical complaint** - sustained by a player that results from a football match or football training, irrespective of the need for medical attention.

2) **Medical attention injury** – any injury that requires medical attention

3) **Time loss injury** – any injury that causes a player to lose training or match time.

Researchers citing the consensus document use the ‘time loss’ injury definition almost exclusively [16,17]. They argue that time loss, and even match-time loss definitions [18], are the most appropriate sport injury definitions because they 1) impact team performance and are therefore most relevant to coaching staff, and 2) can be collected more readily (which makes it feasible when resources are limited). However, time loss is the least sensitive measure of sports injury and it captures fewer injuries than the other two measures of injury [16]. Norway’s Professor Roald Bahr raised the argument that time loss measures underestimate the true burden of injuries in sports like volleyball, swimming, and high jumping, where athletes report pain and decreased function characteristic of ‘overuse’ injuries but may not miss training or competition time. In those sports, time-loss definitions provide low injury incidence, but the prevalence of pain is high and this can compromise player performance [16]. To capture this type of injury, Professor Bahr and colleagues, including Dr Ben Clarsen, developed the Oslo Sports Trauma Research Centre (OSTRC) Overuse Injury Questionnaire [19,20]. The key elements of this patient rated outcome measure (PROM) questionnaire included the weekly reporting of injuries, illnesses, or other health problems, and their effect on athletes’ training volume, performance, or symptoms [19,20].
1.1.1.2 Injury severity

Major consensus statements define 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” [9]. Grouping injury severity into categories like – slight (0 days) minimal (1 to 3 days), slight (4 to 7 days), moderate (8-28 days), severe (>28 days), and career ending is also common practice and described in the original football consensus statement [9].

1.1.1.3 Injury classification

Injuries can be classified according to their anatomical diagnosis, mechanism (overuse, acute), and whether they are new or recurrent.

1.1.1.3.1 Injury diagnosis

Classifying injuries according to their diagnosis includes identifying the general body region injured, the structures involved, and the specific injury nature. Traditional medical classification methods such as the International Classification of Diseases (ICD) coding system [21,22] and others [23] have major limitations when classifying sports injuries [24,25], so several sport specific coding systems have been developed. The Sports Medicine Diagnostic Coding System (SMDCS) [26], and the Orchard Sports Injury Classification, now in its 10th version [27–29], are both widely used in sports injury epidemiology settings [30–33].

1.1.1.3.2 Injury mechanism

Sports injuries may have an acute onset (i.e. usually to trauma) or overuse (i.e. gradual) onset. *Acute* injuries are those with a sudden, identifiable onset, often traumatic in nature [34]. Examples
of acute injuries include ACL ruptures, ankle ligament sprains, and concussions. Overuse injuries, as their name suggests, are generally described as accumulating from excessive strain over time. Their onset is progressive and the injury ‘event’ is difficult to identify [35]. Describing the inciting event provides greater understanding of the injury mechanism, such as whether it was contact or non-contact in nature [36].

1.1.1.3.3 Recurrent or new injury

Recurrent injuries are defined in consensus statements as injuries of the same type and at the same site as an original/index injury, occurring after an athlete returns to full participation [9]. Following these consensus statements, recurrent injuries were divided into ‘exacerbations’ if athletes had not returned to full participation, and ‘re-injuries’ if they had returned to full participation [37]. Although it is beyond the scope of this dissertation, accurately classifying recurrent injuries has received much attention in the last decade because previous injury is the most commonly cited risk factor for future injury, and understanding re-injury or injury exacerbation risks wholly depends on accurate classification [38–44].

Epidemiology relies on accurate case definition. Similarly, these considerations (injury definition, severity, and classification) are critical in sports injury research.

1.1.2 The burden of sports injuries

Sports injuries pose a notable public health burden, especially in youth populations [45]. The total economic burden of injuries in Canada is estimated at over $26 billion [46], and sports injuries make up 35% of injuries [47]. Youth (aged 12-19) are the most likely Canadian age group to
experience an injury, and sport causes 2 out of 3 injuries within this demographic [47]. These trends resemble those documented by international surveys [48,49]. Instead of surveys, medical records or insurance billing data may also be used to understand the public health burden of sport injuries. However, although sports injuries are a major cause of emergency room visits, many do not result in hospitalisation, so population-level data using these data are likely underreported [50].

Narrowing the scope from the population level to a team level, sports injuries carry performance and financial ramifications. Team sport success is compromised by high injury rates [51–53]. In professional sport, the financial costs associated with time loss injuries are substantial. For example, a single hamstring strain injury in Australian Rules Football (AFL) players was costed at over AU$ 40,000 (Australian dollars) because of loss of the player’s time from his employment [54]. In 2012, the average financial cost per AFL team exceeded AU$ 240,000 for hamstring strain injuries alone. These financial costs are even greater in sports where salaries are greater– e.g. a month of time-loss for a European soccer player is estimated at €500,000 [55], and hamstring strains cost US$350,000 per National Football League (NFL) team [54].

Narrowing the scope one step further, injuries burden individual athletes through performance decrements. Systematic review evidence demonstrates that injuries impair the chances of successful performance in athletes [56]. Elite track and field athletes were 7 times more likely to reach their predetermined performance goals if they were able to complete greater than 80% of their planned training [57]. Each missed week of training reduced the odds of success by 26% [57]. Depending on the injury, players may be out of competitive participation for weeks or months, and potentially encounter physical, financial, and psychological challenges associated with their
injuries. Once they return to sport, they may remain at increased risk for subsequent injury because previous injury is considered a risk factor for future sports injuries [58]. Sporting injuries like anterior cruciate ligament can have a long-term health impact on players, even after they discontinue their sporting participation [59–61]. Finally, sport injuries have also been linked to increased opioid prescription and misuse after athletes’ careers end [62].

1.1.3 Foundations of sports injury prevention
Professor van Mechelen’s 1992 landmark ‘sequence of sports injury prevention research’ model outlined a 4-step process: 1) injury surveillance to establish the extent of the injury problem, 2) aetiological research to identify relevant risk factors, 3) preventative strategies to correct modifiable risk factors, and 4) re-evaluating the injury problem to assess a preventative measure’s effectiveness [5]. Over a decade later, Prof. Caroline Finch identified that van Mechelen’s model was limited by its reliance on the scientific environment; it overlooked the need to implement preventative measures in the real world. She introduced the Translating Research into Injury Prevention Practice (TRIPP) Framework [63]. Keeping the first 3 steps from van Mechelen’s model, Prof. Finch changed the 4th stage to scientific evaluation of preventive measures in ‘ideal conditions’, introduced a 5th stage – describing intervention context to inform implementation strategies – and a 6th stage – evaluating effectiveness of preventive measures in the implementation context. My dissertation is primarily on sports injury aetiology, so I will outline the first two steps in van Mechelen’s model below before proceeding to understanding athlete workloads.
1.1.3.1 Injury surveillance – understanding the extent of the problem

Sports injury surveillance is systematically tracking injury occurrences within a sporting population across a given time. Surveillance is not an end in itself, but it is a necessary step to establish the incidence of injury across sporting contexts, and at different times within a sporting environment [63]. While injury incidence (injury cases/1000 participation hours) is the most common outcome of injury surveillance studies, it is not the only one. For example, prevalence proportion (usually point prevalence) equals the number of injured athletes at a given time divided by the total number of athletes [64]. In sports where overuse/chronic injuries are common and athletes may play through pain, some have argued that prevalence measures may be more appropriate than incidence measures [16,65]. While injury incidence is usually expressed as an injury incidence rate (most common – e.g. injuries/1000 hours), incidence proportions are also possible – total # of injured athletes over a given time (e.g. season) divided by the total number of athletes [64]. Accurate surveillance should also include injury severity – the number of days lost due to an injury. Finally, injury burden may be calculated by multiplying injury incidence by injury severity, which may identify the injuries with the most substantial impact [66,67].

Injury surveillance also provides the evidence to evaluate injury risk across and within various sports settings [63]. Different physiological demands, technical demands, and playing characteristics create disparate injury risk across sports. These differences also extend to different competitive levels (i.e. recreational vs. professional), and sporting activity (i.e. training vs. competition).
1.1.3.2 Injury aetiology – understanding why sports injuries occur

Aetiology is the study of why events occur. Building on research from the field of infectious diseases [68], Canada’s Professor Winne Meeuwisse first described sports injuries as multifactorial in 1994 [69]. His model described how internal (e.g. age, sex, flexibility) and external risk factors (e.g. playing surface, opponent behaviour, equipment) contribute to risk and may eventually coincide in an inciting event [69]. Meeuwisse subsequently extended this model and clarified that these risk factors may interact or confound the relationship between other risk factors and injury risk [70].

Two subsequent iterations of the multifactorial injury aetiology model expanded on methodological considerations [71] and defined the inciting event details [36]. In 2007, Meeuwisse and colleagues described the recursive nature of sports injury risk [4].

Accurate injury surveillance that tracks injury events and measures multiple risk factors can identify subgroups that are at higher or lower injury risk. Risk factors may be modifiable or non-modifiable. Using ACL injuries as an example – women’s football (soccer) players may be at a fourfold higher risk compared to their male counterparts [3]. Among female athletes, those with higher knee abduction moments and knee abduction angles during landing are at an increased risk of ACL tears [72]. Sex exemplifies an essentially non-modifiable risk factor while neuromuscular control during landing exemplifies a modifiable factor [73].

Training and competition workload – the intensity and duration of sporting activity – has been increasingly linked to injury risk [74–76]. While workload may be considered a risk factor for
sports injury, it has not been included in previous aetiological models, and whether it should be classified as an internal or external risk factor remains unknown.

1.2 Foundations of workload monitoring

As sport has prehistoric origins, so too does training for sports and hence ‘workload’ – the volume and intensity of training and competition in sport. Workload measurement forms one substantial pillar of my PhD research. Research interest in workload has exploded in the last decade because of inventions and advances in technologies such as Global Positioning Systems (GPS), accelerometers, heart rate monitors, video tracking systems, and others, all of which allow athletes and coaches to quantify workload in much more detail than they could even 20 years ago. Training load monitoring to measure athletes’ training, recovery, performance, and stress has become common practice [77]. Statistical advances and the computational power to manage vast datasets have further created what we might consider a niche in the applied sports sciences. The remainder of part 1.2 is my concise summary of the key literature in this field that has informed my dissertation.

Training load is a combination of training intensity, duration and frequency” (Smith, 2003, pg. 1111). Training load monitoring was rooted in observing athletes during workouts – particularly endurance athletes’ heart rates during steady state or interval training or performing index/standard workouts [79].
1.2.1 Physiological basis for athlete monitoring

General adaptation syndrome (also widely known as the “stress response”) was described in 1936 by Canadian scientist Hans Selye, who described three phases after a stressor was applied: 1) alarm, 2) resistance, and 3) exhaustion [80,81]. Although challenged in the literature [82,83], the exercise sciences rely heavily on Selye’s initial work in describing the physiological adaptations to training and subsequent ‘supercompensation’ [84,85]. A training ‘stress’ induces a period of alarm (acute fatigue). This is followed by a period of resistance wherein the body adapts to the stress so that it is better equipped to handle the stressor in the future (i.e. supercompensation). In the sport and exercise sciences, Selye’s work has been adopted an informed modern-day periodisation theory [84,86].

Periodisation theory dictates that subsequent stressors should be applied after enough time has elapsed for supercompensation. Waiting too long between stressors means the athlete loses adaptations and returns to a baseline level (detraining), while too little time between stressors may interrupt the adaptation process. If high volume and intensity training is continually repeated without adequate rest, maladaptation and overtraining may result [87–89], and this is akin to Selye’s exhaustion.

I would respectfully contend that, as written, general adaptation syndrome may oversimplify the training load—response process in two ways. First, adaptation occurs at the cellular/molecular level [90–92]. At the cellular level, exercise disrupts homeostasis and activates several signals, including mechanical stretch, calcium concentrations, hypoxia, and muscle energy status. Based on the strength and combination of these signals, signalling cascades lead to transcription and
translation of proteins, which will facilitate improved capacity to handle similar stressors in the future [92,93]. Two primary examples include 1) resistance training activating the mTOR pathway, which leads to muscle hypertrophy through satellite cell proliferation, and 2) endurance training activating the enzyme best known as AMPK (5' AMP-activated protein kinase), which facilitates mitochondrial biogenesis and consequently improves cellular aerobic capacity [92]. Therefore, contextualising the ‘stress’ and ‘adaptation’ to the specific training goal extends a ‘general’ view of adaptation.

The second oversimplification from general adaptation syndrome is assuming a consistent, solely biologically mediated stress—response relationship. This assumption is challenged by the individual variability to standard training programs [94]. As the science into the elements of stress has advanced [95], Dr. John Kiely has challenged traditional periodisation theory [82,83]. Contrary to periodisation plans that assume a purely biologically mediated, consistent dose-response relationship, Kiely contends that a given workload will produce varying adaptations, depending on an athlete’s genotype, phenotype, stress history, prior training history, and current stress status (e.g. psycho-emotional and cognitive state) [83]. Acknowledging these oversimplifications, general adaptation syndrome underpins modern training principles. Therefore, coaches, athletes, and of course researchers, are motivated to quantify athletes’ training and the way they are (or are not) responding. In short, ‘workload’ is the main modifiable variable that affects performance, but the relationship is complex. Coaches seek to measure the various dimensions of workload (see below) accurately and titrate workload to try to optimise the athlete’s performance.
1.2.2 Defining and quantifying workloads

‘Load’, according to a recent International Olympic Committee (IOC) consensus statement [65], is the “sport and non-sport burden (single or multiple physiological, psychological, or mechanical stressors) as a stimulus applied to a human biological system” (pg. 1031). In this dissertation, I will use this definition for ‘load’ and ‘workload’ interchangeably. Using this definition, workloads can also be differentiated into external and internal training loads. External workloads refer to the work performed by the athlete, while the internal workload describes athlete’s physiological response to a training bout [96]. A given external load will elicit different internal responses in each individual, based on external load characteristics (quality and quantity/organisation), as well as the athlete’s characteristics (genetics, fitness level, training background, etc.) (Figure 1.1) [97]. Careful consideration should be given to what internal or external measures of load are most appropriate, valid, and reliable given the sporting/athlete context, and the goals in collecting these data [77].
**Figure 1.1:** Proposed model of the relationship between external and internal training loads.

With permission, from [97].

### 1.2.2.1 External Load

External load refers to what an athlete ‘does’, measured using objective criteria. The simplest examples include total distance run (e.g. meters) or weight lifted (e.g. kg) but today, external load can include power output (Watts), speed (e.g. m/s), acceleration/deceleration (m/s²), and neuromuscular function (e.g. sprint performance (s), countermovement jump test (rate of force development in Newtons/second, jump height in cm). The widespread adoption of wearable microsensors, which use global position system (GPS) and accelerometers to collect load data have become the most common means of collecting external load in many sporting contexts [98,99].
1.2.2.2 Internal Load

Internal load refers to an athlete’s physiological or perceptual response to a given external load. An athlete group could be given a standard external load – e.g. a 2km run in 10 minutes, but their internal loads – e.g. heart rate, lactate accumulation, rating of perceived exertion (RPE) would all differ. There is some debate about whether psychological variables (e.g. Profile of Mood States), which capture an athlete’s current psychological state, should also be considered internal load measures [77,96]. I respectfully contend, with other applied workload— injury researchers, that psychological variables should be analysed separately within the ‘workload-monitoring cycle’, because they represent an athlete’s subjective wellness that culminates through training and non-training stressors [100].

1.2.3 Purposes of athlete monitoring

Athlete monitoring can serve at least 4 purposes; 1) Planning, periodising, and implementing training plans, 2) predicting performance, 3) monitoring adaptation, and 4) mitigating injury risk.

1.2.3.1 Planning, periodising and implementing training

Periodised training programs attempt to apply training loads with specific proportions and magnitudes to maximise the benefits of adaptation [101]. These programs may err in two ways: 1) training loads that are too low fail to promote adaptation, or 2) training loads that are too high increase the potential for maladaptation, illness, and overtraining, as well as increased injury risk [101]. I appreciate that this ‘Goldilocks principle’ for training is easy to articulate and difficult to
execute – my thesis aims to contribute to execution, particularly through mitigating injury risk as one potentially negative outcome.

Monitoring training loads facilitates the implementation of periodised plans in at least three ways. First, coaches’ planned training loads and actual athlete training loads may not match, so measuring player workloads allows coaches to understand actual loads. [102]. Second, load monitoring can be used to track how athletes are performing and responding to training over time. By understanding external loads, internal loads, athlete wellness and readiness, practitioners can make on-the-fly modifications to account for individual differences in training responses [82,100]. Finally, elite athletes are differentiated by extremely small margins [103]. Tapering is commonly used to minimise fatigue and maximise performance at key events [104]. However, to perform an effective taper and achieve the performance benefits, loads need to be accurately and reliably tracked, so that they can be precisely altered leading into competition. There is room for more science in this field.

1.2.3.2 Predicting performance

In the mid-1970s, Professor Eric Banister and colleagues proposed a systems model for predicting performance following training, which built on the foundation of the positive and negative training effects [105]. Their model included a positive function of training effect (fitness), and a negative function for fatigue based on an athlete’s heart-rate based training impulse (TRIMP). An athlete’s modeled performance was the difference between fitness and fatigue. Subsequent performance prediction models [106,107] mathematically model training loads and expected responses. While criticisms have been leveled against Banister’s model since it oversimplifies the training—
response relationship [108], these models all analysed longitudinal load data to predict future performance and provided coaches/scientists a framework to understand athlete training responses [77].

1.2.3.3 Monitoring adaptation to training loads

By monitoring training loads with health and performance measures, researchers can assess an athlete’s fatigue levels and his/her potential readiness to train. Improved competition performances or maximal exercise tests may indicate training adaptation, but also cause substantial fatigue on the athlete and are therefore used sparingly. Therefore, less fatiguing methods are regularly utilised. Individual deviation from ‘normal’ loads during similar load sessions, or discrepancy between internal and external load measures, could indicate a state of fatigue [96]. These training load measures can be used in conjunction with other markers, such as heart rate variability, hormone levels (testosterone, cortisol, catecholamines), mood state and recovery status (REST-Q, POMS), and others [109]. Submaximal ‘index’ tests have also been designed for similar purposes [110].

In addition to ‘objective’ measures of performance, athlete self-report (e.g. wellness surveys) can provide a simple measure of athlete response to training. Dr. Anna Saw and colleagues concluded in a 2016 systematic review that these were superior to the objective tests of athlete responses. [111]. As there is no single ‘best’ marker of training response, current gold standard among professional teams is to collect several markers of training load and athlete responses.
1.2.4 Sport workloads and injury risk

In the last 15 years, a growing body of research has investigated the links between training workloads and sports injuries. Systematic reviews [74–76] and a 2016 International Olympic Committee (IOC) Consensus Statement [65] have found that rapid workload increases are associated with increased injury risk, while findings regarding high absolute workloads have differed – some showing them as a risk factor, while others identifying them as protective. Australia’s Dr. Tim Gabbett’s ‘training load injury prevention paradox’ paper [112] described how high chronic workloads may be protective against injury, especially when athletes get to those loads safely [113,114].

Surprisingly, training and match workloads had been overlooked in the aetiological models [4,36,69,71] that existed when I started this thesis. Workloads are unlikely to contribute to all injuries, since each injury stems from a unique collection of risk factors. For example, workload likely plays a lesser role in an ice hockey player’s concussion sustained from an illegal cross-check to the head than a soccer player’s hamstring strain while sprinting for the ball. Therefore, a number of workload— injury investigations focused on non-contact soft-tissue injuries as the primary outcome, since these are considered ‘preventable’ and may be more sensitive to load changes [115]. To summarise this section, I note that there has been no synthesis of traditional risk factors along with workload in previous aetiology models. (Please see Chapter 3)
1.3 Measuring and analysing athlete workload – a key for research and applied sports science

To date this literature review has focused on workload as a concept. In practice, what is the best measure of workload? Professional teams take various factors into account including cost, technological capabilities, cultural concerns (will athletes agree to wear heart rate monitors), and personnel. I contend that the measurement properties of any workload metric must also be investigated very carefully.

This should greatly influence the choice of workload measurement tool, but I fear it has been rather neglected in both the research and the applied settings. Here I review the state of the art with a view to identifying research that is needed in this domain of psychometrics – the intersection of sports science/medicine and biostatistics/epidemiology. I begin with the theory before I discuss how that can be relevant in sports science – specifically in injury—workload research.

1.3.1 Traditional measurement property views

Traditional sport science/medicine research referenced the clinimetric literature to describe measures’ reliability, validity, and responsiveness [116]. Reliability quantifies a measure’s ‘repeatability’ or stability. Validity broadly asks whether something measures what it is supposed to measure but may also be divided into ‘types of validity’. Krosshaug and Verhagen [117] describe ‘face validity’ (whether a test appears to measure something), ‘content validity’ (determined by a team of experts who decide whether a measures captures what it intends to measure), ‘construct validity’ (whether a measure corresponds to a theoretical underpinning), and ‘criterion validity’ (correlation of a measure with a ‘gold standard’) [117].
Criterion validity can be further subdivided into concurrent validity – correlation with a gold standard at the same time – and predictive validity – correlation with a gold standard at a future point in time [117]. Responsiveness describes whether a given measure captures changes in what it is trying to measure over time [118]. For example, a fatigue measure should change following a heavy training or competition workload [119,120].

1.3.2 Unified validity theory

Unified validity theory greatly influenced psychometrics in the last half century, but to my knowledge has not been introduced into sport medicine and science. Grounded in the work of Meehl, Cronbach, and others, US psychologist Samuel Messick primarily drove a unified view of validity theory, where construct validity takes centre stage [121–123]. Replacing ‘types of validity’, this unified validity theory proposed by Messick is defined in the Standards of Psychological Testing as follows:

“Validity is an integrated evaluative judgment of the degree to which empirical evidence and theoretical rationales support the adequacy and appropriateness of interpretations and actions based on test scores or other modes of assessment.” [122].

Unpacking this definition reveals the primary considerations, which differ from other, traditional validity views.
1.3.2.1 Validity is about inferences, not about measures

Theoretically, we could measure the circumference around someone’s head just above the ears. This circumference provides information. We could use this information to infer what someone’s hat size may be, or we could make a conclusion about someone’s intelligence [118]. The inference in the first case is much more appropriate – more valid – than the latter. In this way, validity is less about the measurement itself, and more about the inference one intends to make from the measurement.

1.3.2.2 There are multiple sources of validity evidence

If validity is primarily concerned about whether a measure is appropriate, meaningful, and useful, it follows that people may ask to what degree this is the case. We might ask how the measure relates to other variables that are related, or unrelated, to our primary variable of interest. Do subject matter experts believe that our measure of interest is assessing what we think we are measuring? Does our measure differ between groups where we expect there to be differences? Is the internal structure of the measure related to the central idea we are hoping to assess? Other validity frameworks refer to each of these things as ‘types’ of validity or reliability, while unified validity theory articulates that each of these inquiries provide a different source of evidence to ascertain whether certain inferences are appropriate and meaningful given a certain measurement [124].

1.3.2.3 Validity is an integrative evaluative judgement

An important concept, often overlooked by undergraduate students is that a measure cannot be ‘valid’ or ‘invalid’ [121,124,125]. Rather, whether a specific measure can be used for a specific
purpose is likened to a continuum that ranges from completely useless to extremely appropriate. To evaluate where an inference from a given measure sits, researchers must evaluate the strength and breadth of evidence surrounding a given measure [125]. How does the measure relate to other variables? Do changes on the measurement reflect changes in the gold standard measurement? Do experts agree the measure reflects the underlying construct it is designed to measure? What is the measurement really measuring, or what are participants thinking when they complete the measure? A unified approach identifies that multiple sources of evidence should be integrated when evaluating the reliability of certain measurement-informed inferences.

1.3.2.4 How unified validity theory may contribute to workload— injury research

Unified validity theory has the potential to inform validation work across sport sciences, including workload— injury research. In published manuscripts and in conference presentations, it is common to hear that a ‘validated measure’ was used. These statements often refer to a few papers where one or two sources of validity evidence was examined. By incorporating unified validity theory into sport sciences, researchers may consider the degree of confidence they have in their specific inferences, instead of determining a binary (valid/invalid) outcome for their measurements. When choosing a workload measure (internal or external), unified validity theory provides a lens for researchers to consider all the relevant sources of evidence and decide how appropriate workload measures and derived variables are for their specific sporting environment.
1.4 Methodological and statistical limitations in athlete monitoring and injury prevention research

Prospective cohort studies are the most frequent study design within the workload—-injury literature [74,76]. Teams with the resources and budget to track workload longitudinally are often in elite sport environments, where performance is paramount and randomised controlled trials are not feasible. Therefore, most datasets capable of measuring workload and injury occurrences consist of longitudinal, observational data collected on several athletes on a regular (daily/weekly) basis. Considering the recursive nature of injury risk, varying across each session, it is important the data are collected frequent enough to capture these fluctuations, and statistical approaches are employed that can effectively analyse these changes. Professor Linda Collins described this principle in her threefold model of longitudinal data – where the theoretical model (aetiology), data collection frequency (temporal design), and statistical model should be as closely aligned as possible [126].

Longitudinal data present several challenges that researchers must account for when choosing an analytical approach. They must differentiate between- and within-subject effects [127], correlated errors of measurement (i.e. dependency caused by repeated measures), missing data, and potentially uneven time intervals [128]. Although there have been great advances in the area of longitudinal data analysis [128–130], the challenges above mean that certain statistical approaches are more appropriate. Although authors have systematically reviewed the workload—-injury research and quantitatively synthesised the evidence, to date none have evaluated the workload--sports injury literature to assess the quality of statistical analyses.
1.5 Integrated athlete monitoring and injury research and practice

Athletes’ training and competition workloads are strongly associated with injury risk [65,74,76,77]. How this fact impacts applied research and practice depends on (1) aetiological, (2) methodological, and (3) statistical considerations, and aligning these three areas.

Conceptually, existing aetiological models have not explicitly explained how workloads relate to injury risk, nor how they interact with other identified risk factors to culminate in sport injury occurrence. As aetiological understanding is among the first steps of injury prevention, researchers and practitioners should understand the mechanisms by which workloads contribute to sports injury.

Methodologically, applied researchers should consider what measures to collect in their specific sport settings. This decision requires an integrated, evaluative judgement of which measure is most appropriate for their purposes (in this case injury risk), considering all the available evidence. If they consider that daily workloads influence subsequent injury risk, researchers must also collect data frequently enough to capture these workloads and fluctuating risk.

Statistically, sports medicine researchers may only explore the workload—injury association adequately when they employ statistical analyses that address the challenges inherent with intensive longitudinal data.

Workload monitoring has taken center-stage in sports science/medicine research practice. A growing body of literature indicates that workloads are strongly associated with injury. However,
as technological advances allow more workload data to be collected than ever before, there is a
need for aetiological and methodological frameworks to guide future workload—injury
investigations.
Chapter 2: Aims of the Dissertation

My objective in this dissertation was to investigate a conceptual and methodological framework that explains how training and competition workloads are associated with sports injury risk.

My specific aims were,

1) To critically interrogate current sport injury aetiological models and ask why workload was not included in any of them. This led to my suggestion to update the workload—injury aetiology model to include training loads (Chapter 3).

2) To describe how workload data and risk factors (internal and external) may mediate or moderate the workload—injury association (Chapter 4).

3) To methodologically review the statistical techniques that authors have used to examine workloads and sports injuries using longitudinal data using Linda Collins’ three-part framework. (Chapter 5). This chapter includes a framework of best practice for longitudinal data analysis broadly and for scientists in the workload—injury field specifically.

4) Using the framework outlined in Chapter 5 and my subsequent recommendations for future investigations, to use multilevel modeling of intensive longitudinal data to examine the effect of player unavailability on players’ physical match workloads (Chapter 6).

5) To demonstrate how a statistical concern (mathematical coupling in the acute:chronic workload ratio) may inform workload—injury research and applied practice (Chapter 7).

6) To examine pre-season participation and in-season injury risk, accounting for in-season workloads using multi-level modelling (Chapter 8).
These 6 aims can be considered as 3 complementary parts, each addressing one component of my thesis title:

Part 1 (Chapters 3-4). **Conceptual considerations** of the workload— injury association,

Part 2 (Chapters 5-6). **Methodological considerations** of the workload— injury association,

Part 3 (Chapters 7-8). **Applied considerations** of the workload— injury association.
Chapter 3: How do training and competition workloads relate to injury? The workload—injury aetiology model

3.1 Preamble

Workloads had increasingly been associated with injury risk in the literature over the last decade, as described in Chapter 1. However, aetiological models to date had not included workloads explicitly in their frameworks, and could not adequately explain the workload—injury association. This discrepancy led to the focus of this chapter – an extended sports injury aetiology framework: the workload—injury aetiology model.

The rest of this chapter is a reproduction of text and figures from the following article [131]:


3.2 Introduction

Athletic injuries are common in a variety of sports [132,133], compromising performance [52,53,134], posing a financial burden to organisations, [54] and potentially causing long-term health consequences [135]. The causes for these injuries are numerous, highlighted by a number of multifactorial injury etiology models [4,36,69]. However, regardless of the interplay of risk factors or inciting biomechanical event, every athletic injury is sustained while athletes are exposed to the demands of the sport, either through competition, or through practice workloads.
that are applied to athletes with the goal of inducing positive physiological changes and maximising performance.

While adequate workloads are necessary for fitness and performance improvements through adaptation and skill acquisition, high workloads and especially ‘spikes’ in loads are strongly associated with injuries [52,113,115,136,137]. Although this association has been demonstrated in soccer [75], Australian Football [138–140], basketball [141], rugby union [52], rugby league [113,142,143] and cricket players [136,144,145], few have explained the underlying mechanisms through which workloads relate to injuries. Moreover, existing injury etiology models cannot account for the relationship between workloads and injury risk. The purpose of this paper is to present an updated etiology model that accounts for the effects of workloads on athletic injuries. After reviewing previous injury etiology models, we discuss the known association of workloads and injuries, and present an updated model that explains a threefold contribution of workloads to injury risk. Finally, we discuss the research and practice implications of the updated model.

3.3 Why do athletes get hurt? Injury etiology models

The multifactorial nature of athletic injuries is well known [4,69,71]. Single risk factors fail to adequately predict injuries, and the limitations of univariate analyses of risk factors have been discussed [69]. Instead, multivariate techniques are recommended, which enable researchers to examine multiple risk factors which come together to cause an injury [69,71]. The first of these multifactorial models was proposed by Meeuwisse in 1994 [69]. Meeuwisse built upon chronic disease models to propose an epidemiologic, multifactorial approach to understanding injury causation and investigating athletic injuries (Figure 3.1). Within this multifactorial model, athletes
have intrinsic (internal) risk factors that inherently predispose them to injury. Some of these factors (e.g. age) are non-modifiable, while others (e.g. flexibility) are modifiable. These predisposed athletes are then exposed to extrinsic (external) risk factors, such as the playing surface, protective equipment, or opponent behaviour, which make them susceptible to an injury. Finally, a certain inciting event occurs wherein the biomechanical stress of the event exceeds the tolerance of the athlete’s tissues, and injury ensues. Lastly, Meeuwisse also acknowledged that risk factors are interrelated, and in a subsequent article addressed the importance of interpreting both interaction and confounding effects in injury etiology research [70].
Figure 3.1: Meeuwisse’s first multifactorial injury etiology model proposed in 1994.
With permission, from [69]
In two subsequent papers published in the *British Journal of Sports Medicine* [36,71], Bahr and colleagues built upon this first multifactorial model, highlighting and focusing on certain aspects (Figure 3.2). First, they expanded the description of both internal and external risk factors and the subsequent methodological implications [71]. Second, they proposed a more thorough description of the biomechanical factors contributing to the inciting event [71], based on the comprehensive biomechanical injury model of McIntosh [146].
Figure 3.2 Bahr & Krosshaug’s 2005 comprehensive etiology model.
BMD = bone mineral density, ROM = range of motion. With permission, from [36].
Most recently, Meeuwisse and colleagues proposed a modification to these etiology models in the form of a “dynamic, recursive injury etiology model” (Figure 3.3) [4]. This dynamic model of risk and causation acknowledges that previous linear models do not account for the dynamic and non-linear nature of athletes’ injury risk. Moreover, sport participation may or may not result in an injury occurrence. In cases where participation doesn’t result in injury, it actually modifies certain risk factors through physiological adaptation to the training stress. On the other hand, if an athlete does experience an injury, there is also acknowledgement that an athlete may either recover and return to play with a modified injury risk, or never recover, resulting in complete removal from participation.
Figure 3.3: Meeuwisse and colleagues’ 2007 dynamic, recursive sports injury etiology model. With permission, from [4].
This continued evolution in understanding injury etiology helps to inform injury prevention efforts. It provides a framework in which investigation scope can be defined, indicates appropriate statistical analyses, highlights important variables to consider, and in the case of the dynamic model, reminds researchers that participation not only predisposes to injury, but modifies subsequent risk. Further, the consistent modification of the model also highlights the need for further research, and willingness to amend the model as more knowledge is accumulated [147].

Notably, workloads are not included as an internal risk factor nor an external risk factor in existing etiology models. In fact, although workloads are strongly associated with injury [52,113,115,136], their role in the etiology of athletic injuries is uncertain, given that they are not explicitly included anywhere within the previous models.

### 3.4 Workloads and injuries – what do we know?

#### 3.4.1 Defining and quantifying workloads

Workloads, as defined in Gabbett and colleagues’ systematic review on workloads, performance and injury, are “the cumulative amount of stress placed on an individual from multiple training sessions and games over a period of time” [75]. As Smith explains, they are “a combination of … [training and competition] intensity, duration and frequency” [78]. Essentially, workloads are a quantification of the demands imposed on an athlete during one or more matches or training bouts. Ideally, training is prescribed in such a way that the athletes’ homeostasis is disrupted, and optimal adaptation occurs during recovery. This fine balance seeks to avoid, on the one hand – insufficient workloads that fail to induce adaptation or result in detraining, and on the other hand – excessive loads that induce maladaptation or overtraining [78,96,109]. However, optimally prescribing these
loads presents a number of challenges, the most notable of which is choosing an appropriate workload measure.

Workloads can be measured as either external or internal loads. External loads quantify the amount of work performed by the athlete (e.g. distance covered, balls thrown, etc.), while internal loads measure the “relative physiological and psychological stress imposed” on the athlete [96]. A given external load will elicit different internal responses in each athlete, based on the characteristics of how the external load is applied and the athlete’s individual characteristics (e.g. genetics, fitness level, training background, etc.) [97]. Numerous measures have been developed and proposed for internal and external loads from which practitioners can decide (Table 3.1) [96,101]. However, each of these measures aims to quantify the workload completed by the athlete and/or their response to that work. Careful consideration must be given to which type (internal or external) and specific measure(s) of load are most appropriate, given the sport context, goals of load monitoring, logistical and financial constraints, and the psychometric properties (validity/reliability) of the specific measure.
Table 3.1: Measures of external and internal load

<table>
<thead>
<tr>
<th>External Load</th>
<th>Internal Load</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Frequency (/day, /week, /year)</td>
<td>- Session-Rating of Perceived Exertion (RPE*minutes)</td>
</tr>
<tr>
<td>- Time (minutes, seconds)</td>
<td>- HR:RPE Ratio</td>
</tr>
<tr>
<td>- Accelerometer loads</td>
<td>- TRIMP (HR-based training impulse)</td>
</tr>
<tr>
<td>- Distance covered</td>
<td>- Blood Lactate</td>
</tr>
<tr>
<td>- High speed distance covered</td>
<td>- Lactate:RPE Ratio</td>
</tr>
<tr>
<td>- Jumps completed</td>
<td>- Recovery/stress/wellbeing</td>
</tr>
<tr>
<td>- Power output, speed, acceleration</td>
<td></td>
</tr>
<tr>
<td>- Time-motion analysis</td>
<td>questionnaire(s) (e.g. Recovery Stress Questionnaire for Athletes - REST-Q; Daily Analyses of Life Demands of Athletes – DALDA, Profile of Mood States - POMS)</td>
</tr>
<tr>
<td>- Neuromuscular function</td>
<td></td>
</tr>
<tr>
<td>- Weight lifted</td>
<td></td>
</tr>
<tr>
<td>- Throws/Pitches/Bowls performed</td>
<td></td>
</tr>
</tbody>
</table>

Various examples for quantifying both external and internal workload. Examples taken from [96,101,111]

3.5 Monitoring workloads in relation to performance, periodisation, and overtraining

As Borresen and Lambert describe in their review of workloads, physiological responses and performance, “optimising training first involves quantifying what the athlete is currently doing” [101]. Traditionally, this quantification of load has allowed, 1) performance to be predicted, 2) training to be planned, periodised, and tracked, and 3) training readiness and stress to be monitored.

1 – Predicting performance. In the mid-1970s, Banister and colleagues proposed the first systems model of performance, characterising the athlete as a system with input (workload) and subsequent output (performance) [105]. Their landmark model proposed fitness as a positive impulse of training, and fatigue as a negative impulse, acknowledging the dual forces of stress/breakdown caused by a training bout and the potential for positive adaptation with sufficient recovery. Within
this model, both impulses decline exponentially once a session ends and recovery begins, but fatigue decays at a much more rapid rate than fitness.

These basic principles underpin performance strategies like tapering, where a 3% (ranging 0.5-6.0%) improvement in performance may be attained through altering training volume and intensity leading into peak competitions [103,104,148]. While attractive in theory and implemented widely in practice, Banister’s model and similar performance prediction models [106,107] have some limitations in their ability to achieve high levels of precision [149]. These limitations may be partly attributable to individually variable load—adaptation responses to set training regimes [150,151]. It is thus important that these basic principles should be adapted to individual athletes, with the applied load and individual response monitored where possible [82].

2 – Planning, periodising, and implementing training plans. Coaches use the inherent principles of workloads and adaptation to design periodised training plans, with the goal of eliciting optimal adaptation [86,152]. However, workloads must be monitored if coaches are to ensure that their training plan is implemented as intended, as the discrepancy between coaches’ and athletes’ perceptions of training has been demonstrated [102]. Further, workload monitoring is necessary to make and implement refined alterations to training loads. For example, these precise refinements are implemented during tapering phases where training volume may be exponentially reduced while intensity remains high, with the goal of minimising cumulative fatigue and peaking for competition [103,104,148].
3 – Monitoring fatigue to prevent overtraining. Monitoring workloads over time can also be utilised to assess athletes’ stress levels, fatigue, mood, and readiness to train [96]. Both physiological measures (e.g. heart rate variability, heart rate recovery, hormone levels, catecholamine levels) and psychological measures (e.g. REST-Q, POMS, DALDA) have been recommended to monitor athletes’ internal workloads and responses [109,153–155]. In monitoring workloads longitudinally, large individual deviations from normal responses, and discrepancies between internal and external load measures can be used to assess athletes’ responses to training [96]. For example, lower internal loads with a standard external load may indicate fitness improvements, while increased internal loads with the same external load may indicate a state of fatigue [96].

In all of these traditional uses, training loads are imposed on athletes with the goal of maximising performance, acknowledging that while training is necessary to induce the “reward” of positive adaptations, it carries the known “risk” of imposing stress upon the athlete. Thus, every time an athlete trains, they are exposed to the fatiguing effects of training, as well as risks of potential maladaptation through overtraining. However, excluded in these traditional uses and this aforementioned risk – reward balance is the reality that every training and competition load carries the potential for athletic injury [156]. In fact, every athletic injury is sustained while an athlete performs some sort of workload. This begs the question: how do workloads relate to injury occurrence? Is it a simple matter of increased loads leading to increased injury, or is there more at play? Recently, researchers have begun providing some answers in numerous sporting contexts and with various load measures – teasing out this association.
3.5.1 Total workloads and injuries

With each training and competition bout, athletes are exposed to the risk of sustaining an injury, so it may seem intuitive that increased loads should result in increased injury levels. Traditionally, workload-injury investigations focused on this relationship between absolute workloads and injury loads [141,142,157–159]. To highlight a few examples, rugby players running more than 9 m of very high speed running were 2.7 times more likely to experience a non-contact injury than those below that threshold [160]. Australian Football (AFL) players with higher total distance (odds ratio 5.5) and sprint distance (odds ratio 3.7) accumulated over 3 weeks were at a greater likelihood of injury [138]. Furthermore, higher pitching loads in baseball [161,162], and bowling loads in cricket have also been associated with increased injury risk [144].

Gabbett developed an injury prediction model using session-rating of perceived exertion (s-RPE) as a marker of internal load among elite rugby league players over two years [115]. During the subsequent 2 years, the model was used to predict injuries, showing that players who exceeded the weekly workload threshold as determined by the model were 70 times more likely to test positive for non-contact, soft-tissue injuries, while players who did not exceed the threshold were injured 1/10 as often [115]. Collectively, these data indicate that there is an increase in injury risk with absolute workload increases.

This higher workload - higher injury relationship may create the impression that workloads should be kept low in order to minimise injury risk. While a reduction in training load may be appropriate in certain instances [158], two important points should be stated. First, adequate workloads are necessary to induce beneficial physiological adaptations such as high aerobic capacity, optimal
body composition, strength, and repeat-sprint ability [163–165], which are required for high performance, and many of which are associated with decreased injury risk [160,166,167]. Thus, workloads that are too low may not only decrease performance, but may result in lower levels of fitness and preparedness, subsequently increasing injury risk. For example, a ‘dual threshold’ has been shown in cricket fast bowlers, where not only high workloads and short recovery periods associated with increased injury risk, but a low total number of deliveries (<123, relative risk = 1.4) and long recovery periods (>5 days, relative risk = 1.8) also increased injury risk [144].

Second, it may not solely be the total workload that is applied that contributes to injury risk, but the way in which it is applied. Indeed, some investigations show that total workloads are not always associated with increased injury risk, while the rate of change in these workloads over time is a stronger predictor [112,113,136].

### 3.5.2 Acute:Chronic workload ratio and injuries

Specifically, load-injury investigations have recently examined the relationship between acute (1 week) and chronic (4 week) workloads, termed the *acute:chronic workload ratio*, with injury risk [52,113,136]. In the first study of acute:chronic workload ratios, Hulin and colleagues showed that while internal and external loads of cricket fast bowlers in isolation were not significantly related to injury risk, acute:chronic workload ratios of >1.5 increased injury risk by 2–4 times in the subsequent 7 days [136].

High acute:chronic ratios, simply described as training ‘spikes’, similarly increase injury risk in rugby league players [113,168] and professional soccer players [169], with utility for use in
predicting injury (Figure 3.4) as well as subsequent injury during athletes’ return to play [170] (Figure 3.5). In addition to understanding that both total workloads and changes in load over time are related to injury risk, researchers have begun combining these two variables to determine the optimal outcome for both injuries and performance.

Figure 3.4: Injury likelihood associated with various weekly changes in workloads. Unpublished data collected from professional rugby league players over three pre-season preparation periods. Loads were measured using session-rating of perceived exertion, and a total of 148 injuries were sustained. Reused, with permission, from Gabbett [112].
Figure 3.5: Finding the ‘sweet spot’ for acute:chronic workload ratios in return-to-play protocols. Data redrawn from 3 separate sports (cricket, rugby league and Australian football) demonstrating injury likelihoods associated with various acute:chronic workload ratios. With permission [112].

3.5.3 Workload – injury paradox

In contrast to the idea that higher workloads contribute to higher injury incidence, high workloads may contribute to well-developed physical qualities, thereby reducing injury risk [112,171,172]. The training--injury prevention paradox model has recently been defined and reviewed by Gabbett – describing this chronic load effect [112]. This ‘paradox’ of high chronic workloads is their
potential preventative effect, as long as the acute:chronic workload ratio is kept in a moderate range (i.e. training spikes are avoided).

Hulin and colleagues found that elite rugby players who had very high acute:chronic workload ratios as well as high chronic workloads had the largest risk of injury (28.6%). However, they also demonstrated that as long as acute:chronic workload ratios were kept within a moderate zone (0.85-1.35), high chronic workloads were associated with the lowest risks of injuries, other than very low (< 2 SD) acute:chronic ratios (Table 3.2) [113]. In a subsequent rugby league study, the odds of sustaining an injury during the competitive season were decreased by 17% for every 10 pre-season sessions that players completed, controlling for in-season workloads [173].
Table 3.2: Relationships between injury risk and various acute:chronic workloads combined with high and low chronic workloads

<table>
<thead>
<tr>
<th>Acute:chronic workload ratio</th>
<th>Combined with low chronic workload</th>
<th>Combined with high chronic workload</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very-low</td>
<td>0.0 ± 0.0</td>
<td>0.0 ± 0.0</td>
</tr>
<tr>
<td>Low</td>
<td>7.8 ± 4.1</td>
<td>9.6 ± 4.1</td>
</tr>
<tr>
<td>Moderate-low</td>
<td>10.0 ± 2.5</td>
<td>7.5 ± 2.2</td>
</tr>
<tr>
<td>Moderate</td>
<td>9.3 ± 2.6</td>
<td>6.2 ± 2.2</td>
</tr>
<tr>
<td>Moderate-high</td>
<td>11.0 ± 4.9</td>
<td>7.1 ± 4.0</td>
</tr>
<tr>
<td>High</td>
<td>5.9 ± 7.3</td>
<td>12.0 ± 10.7</td>
</tr>
<tr>
<td>Very-high</td>
<td>18.2 ± 14.9</td>
<td>28.6 ± 18.1</td>
</tr>
</tbody>
</table>

Risk of injury (±90 CI) associated with 2-week average acute:chronic workload ratios combined with low (<16 095m) chronic workload, and high (>16 095m) chronic workload in elite rugby league players. With permission, from Hulin et al. [113]

As extensively discussed in Gabbett’s recent review [112], these findings support the notion that workloads should be high enough to produce the beneficial physiological adaptations which may protect against injuries. However, these beneficial effects can be negated if athletes are exposed to large workload spikes, which are likely responsible for a large proportion of non-contact soft tissue injuries [112]. Therefore, the collective findings of workload-injury data indicate that while increased loads are somewhat associated with increased injury rates, optimal load management to minimise injuries entails the accumulation of high chronic workloads, while minimising spikes by maintaining week-to-week changes within approximately 10% [112]. What remains to be explicated are the mechanisms by which these findings link to our current knowledge of injury etiology.
3.6 An updated injury etiology model – incorporating the effects of workloads

Previous injury etiology models neither included workloads within the model, nor explained the strong association of loads with injuries. We have learnt a great deal about the association of workloads with injuries and it is clear that the application of training or competition loads are not inciting events that directly cause injury. An athlete may have a large spike in training and subsequently rupture his or her anterior cruciate ligament, but this injury still occurs via some inciting event (e.g. a dynamic valgus collapse). Overuse injuries, or ‘training load error injuries’[174], may be strongly workload-related, but still have an inciting event of cumulative tissue overload, even though a specific event occurrence may not be identifiable [16,19].

Further, training loads are neither a characteristic of the athlete (internal risk factor), nor an aspect of the environment in which the athlete participates (external risk factor). Rather, training and competition loads are better understood as the ‘vehicle’ in which athletes are exposed to external risk factors and potential inciting events. With this understanding, injuries are not directly caused by workloads. Instead, training and competition loads contribute to injury risk through exposing athletes to potentially injurious situations, as well as through their positive and negative effects on numerous modifiable internal risk factors. Therefore, we propose an updated injury etiology model that explicitly incorporates workloads within the causal chain, and outlines its known effects (See Figure 3.6).
Figure 3.6: The workload – injury etiology model.
Like Meeuwisse’s 2007 dynamic recursive model [4], our model depicts the ever-changing nature of an athlete’s injury risk, as well as the possibility that an athlete may, or may not, experience an injury after being exposed to training or competition loads. We have retained the role of internal and external risk factors in line with previous model iterations [36,71]. However, we have divided internal risk factors into modifiable and non-modifiable factors to differentiate those which can change through adaptations from those that are stable. Further, we have extended previous models by including the ‘Application of Workload' as the primary process whereby an athlete is exposed to various external risk factors, and the potential for inciting events, thereby moving from a ‘predisposed athlete’ to a ‘susceptible athlete’.

Whereas Meeuwisse’s 2007 model specified that an athlete who engages in repeat participation may experience adaptation/maladaptation from training and thereby be at an altered risk [4], we explicitly outline that athlete adaptation comes as the result of each applied workload. As in Banister’s original performance prediction models, we highlight that are both positive adaptations from training (i.e. fitness), and temporary negative effects of training (i.e. fatigue). Whereas Banister and colleagues focused on these adaptations from a performance perspective, we contend they function similarly in dynamically effect injury risk. These adaptation effects alter an athlete’s modifiable internal risk factors so that when they engage in a subsequent training or competition bout, their injury predisposition will be altered.

Lastly, we note that when an athlete experiences an injury, they must engage in the rehabilitation and return to play process. In this scenario, an athlete’s subsequent participation occurs with a modified injury predisposition due to the presence of an index injury [58]. However, in
rehabilitating and preparing to return-to-play, these athlete’s rely on the application of workloads through the same process to restore resilience in the injured tissue and prepare them for the demands of training and competition [170].

3.6.1 The triple role of workloads – ‘exposure’, ‘fitness’, and ‘fatigue’

Within the updated model, a predisposed athlete becomes susceptible to injury when they are exposed to a training or competition load. This workload exposes them to external risk factors and potential inciting events for the duration of the bout. Further, workloads modify subsequent injury risk. This risk modification occurs through both positive and negative adaptations, dictated by the total and relative workload applied. Therefore, workloads affect injury etiology in 3 primary ways.

1 - Exposure – training and competition loads are the medium exposing athletes to external risk factors potential inciting events.

2 - Fitness - positive adaptations associated with training, which improve modifiable internal risk factors, such as aerobic capacity, skill level, or body composition.

3 - Fatigue - negative adaptations associated with training, temporarily causing decreased capacity in modifiable internal risk factors, such as tissue resilience or neuromuscular control.

3.6.2 How do workloads influence injury risk? Implications of the updated model

According to this model, workloads are most likely associate with injuries under three main conditions:
1 – High workloads increase exposure, thereby increasing injury risk.

Since training and competition loads are the medium exposing athletes to potential inciting events and external risk factors, there will always be, to some degree, a higher load – higher injury relationship. No load, and thus no exposure, cannot result in athletic injury, regardless of the internal and external risk factors at play. This aspect of the model explains a great deal of the literature which has found increased injury risks with increased workloads [141,142,157–159].

2 - Workloads which induce high levels of negative changes to modifiable internal risk factors (i.e. ‘fatigue’) increase injury risk.

A given external workload is a poor predictor of fatigue, since individuals vary widely in their internal response to set loads. A better marker of how workload effects fatigue may be the acute:chronic workload ratio, wherein a high ratio indicates that an athlete has been placed under a load that is substantially higher than their recent training has prepared them for. Physiological and psychological measures of internal load can also indicate an athlete’s fatigue levels. In this case, negative changes may occur to a number of modifiable internal risk factors, such as compromised neuromuscular control or reduced tissue resilience, which increase subsequent injury predisposition.

3 – Workloads that maximise positive adaptations while minimising fatiguing effects will help make athletes more robust to injury.

On the other hand, positive adaptations to modifiable internal risk factors help to explain the training-injury prevention paradox. As long as the acute:chronic workload ratio is kept moderate, implicating lower fatiguing effects, higher chronic workloads may be preventative against injury
due to the positive adaptations of training [112]. From a biomechanical perspective, Bahr explains “improved fitness may protect the tissue against injury through the effects of training on its material properties” [36]. Together, acknowledging the recursive nature of workload adaptations with both positive and negative adaptations helps to frame why the acute:chronic workload ratio is strongly associated with injury, and why high chronic workloads may be protective against injury [112,113,136].

3.6.3 Model limitations

As with any model, our proposed injury etiology model is incomplete. A set workload may induce various levels of positive and negative adaptations that are based upon additional athlete stressors (e.g. travel, lack of sleep), which are not explicitly incorporated into the model. Some known risk factors are not explicitly listed (e.g. genotype, psychological state, muscle fibre type, etc.) and some that are yet to be identified. As just one example, workloads may effect certain psychological variables (e.g. commitment to training, self-blame, perceived stress/fatigue, etc.), which could subsequently alter match/training behaviour and change injury risk [155,175]. Moreover, the interactions/confounding relationships between various risk factors are not detailed. Some of these, such as the interaction between previous injury and neuromuscular control are known [36], while others are yet to be explained. However, even just including the known confounding and interacting relationships would make the model nearly illegible due to the number of interrelating connections.

It should be noted that workloads are unlikely to contribute equally to all injuries, on the basis of different tissue characteristics and mechanisms of injury [176]. Different tissues may respond
differently to loads, exemplified in cricket fast bowlers, where high medium term (3 month workloads) are protective against tendon injuries, but a risk factor for bone stress injuries [176].

Further, workloads may contribute less to contact injuries than non-contact injuries. For example, workloads relate to an ice hockey player’s concussion following an illegal body check primarily by providing the exposure to certain external risk factors (e.g. opponent behaviour, a compromised helmet). Conversely, a non-contact injury such as a soccer player’s ACL rupture while changing direction is likely attributable to a greater number of internal risk factors. In this instance, workloads contribute again in this scenario through exposure, but loads in the weeks preceding injury may also have modified certain internal risk factors – tissue resilience, neuromuscular control, etc. – which further predisposed the athlete to injury. Therefore, a number of investigations relating load and injury have focused on non-contact soft-tissue injuries, which are considered ‘preventable’, more sensitive to load changes, and more attributable to internal risk factors [112,115].

Similarly, workload may contribute differently to overuse vs. acute injuries. Whereas workloads may modify athletes’ internal risk such that they are predisposed to an inciting injury event, overuse injuries may occur without a single identifiable injury event. However, the suggested model indicates that this type of cumulative tissue overload could occur with exceedingly high workloads, or spikes in workloads, that result in high levels of fatigue (negative adaptations) without adequate recovery for positive adaptations.
3.7 Where to from here – research implications

The dynamic nature of the current model presents similar challenges to research design and statistical analysis as Meeuwisse’s dynamic recursive model [4]. From a design perspective, although non-modifiable risk factors can be measured once (since they will not change), modifiable risk factors should be measured repeatedly to account for changes over time. Repeated measurement are even more important in the case of training and competition loads, since quantifying both total loads and acute:chronic workload ratios can only be done if workloads are consistently monitored over time. Although repeated measurement presents logistical and financial challenges, repetition is necessary to quantify the dynamic nature of modifiable factors, workloads, their interactions, and athletes’ subsequent injury risk.

From a statistical analysis perspective, repeated measurement also implicates certain statistical analyses. Simple logistic regression models which assume the same exposure (training and competition load) across individuals may not be appropriate [71], given the large variability in workload measures across individual players. Although Cox proportional hazards regression (time-to-injury event) and Poisson regression (rate of injury per 1000 exposures or per 1000 hours) have been presented as potential models to account for time exposure [4], they often fail to account for the intensity of training which is captured in various load measurements. Though not frequently used, multilevel (i.e. mixed) modelling may allow for these analyses, since they can account for correlated outcomes (repeated measures among players), and include random effects to predict individual athletes’ risks [127,177,178]. The frailty model which also allows random effects for players and the ability to control for the dependencies of recurrent injuries and repeated measures also shows promise [38,44].
3.8 Summary

Training and competition workloads have been an obvious omission from previous injury etiology models. Workloads are neither an internal nor external risk factor for injury, but are the ‘vehicle’ by which athletes either compete in, or train for their respective sports. We propose that our updated injury etiology helps provide a conceptual framework for ‘why’ workloads are strongly associated with injuries. According to this model, increased workloads increase injury risk through exposure to external risk factors and potentially inciting events. However, they also continually modify injury predisposition by inducing physiological adaptations, some positive, and others negative. Therefore, when an athlete experiences a high acute:chronic workload ratio, they likely experience larger degrees of negative maladaptation, modifying a host of internal risk factors and increasing their predisposition to injury in subsequent bouts. Conversely, moderate acute:chronic workloads and the accumulation of high chronic workloads may maximise the positive physiological adaptations to training and thereby reduce injury risk, as suggested by the training load-injury prevention paradox model.

We note that the confounding and interacting effects of workloads and many risk factors are still unknown. However, longitudinal investigations with repeated measures of workloads and modifiable risk factors may begin to unravel these relationships and provide further insight into the dynamic nature of injury etiology.
Chapter 4: Why do workload spikes cause injuries, and which athletes are at higher risk? Mediators and moderators in workload–injury investigations

4.1 Preamble

In chapter 3, I introduced a new athletic injury aetiology model that explicitly included training and competition workloads (Figure 3.6). The model considered traditional internal and external risk factors, but also acknowledged how workloads contribute to the recursive nature of injury risk – through positive physiological adaptations (i.e. fitness) and transient negative effects (i.e. fatigue). Soon after the first multifactorial model of athletic injury, Meeuwisse discussed how different risk factors relate to each other, and how to differentiate the concepts of interaction and confounding [70]. Extending this aetiological thinking to the workload— injury aetiology model, I expanded how traditional risk factors may interact with workload, specifically through mediation or moderation.

The rest of this chapter is a reproduction of text and figures from the following article [179]:


4.2 Introduction

Spikes in training and competition workloads, especially in undertrained athletes, increase injury risk [112]. However, just as attributing athletic injuries to single risk factors is an oversimplification of the injury process [131,180], interpreting this workload— injury relationship
should not be done in isolation. We must further unpack how (i.e. through which mechanisms) workload spikes might result in injury, and what characteristics make athletes more robust or more susceptible to injury at any given workload. In other words, which factors *mediate* the workload— injury relationship, and which *moderate* the relationship.

### 4.3 Domino or dimmer? Differentiating ‘mediators’ and ‘moderators’

Like dominoes being knocked over, *mediators* can be viewed as the intermediary steps that explain the association between an observed variable and an outcome [181]. In this context, mediating variables help to explain ‘why changes in workloads might cause injuries’? For example, it is known that rugby league players exposed to spikes in running workloads, indicated by a high acute:chronic workload ratio, are at an increased risk for non-contact injuries [182]. One potential explanation is that neuromuscular fatigue mediates this relationship, such that increased workloads cause higher levels of neuromuscular fatigue, subsequently precipitating injury (e.g. while performing a cutting manoeuvre) (Figure 4.1).

On the other hand, *moderators* can be viewed as ‘dimmer switches’, modifying the effect of a given variable on an outcome. They are also referred to as *interactions*, or *effect modifiers* [70]. In our context, moderator variables answer the question: ‘what characteristics make certain athletes more robust or more susceptible to injury at given workloads’? For example, in Gaelic football, high aerobic fitness protects against workload spikes [182]. In other words, aerobic fitness protectively moderates the workload effect by ‘dimming’ or reducing the risk of rapid workload increases (Figure 4.1).
Figure 4.1: Examples of how mediating and moderating variables explain the association between a workload spike and subsequent injury.

Top, neuromuscular fatigue is shown as a mediator (i.e. domino) of the effect of workload spikes on injury. In this example, a spike in workload causes an increase in neuromuscular fatigue, and an increase in neuromuscular fatigue leads to an injury. Bottom, aerobic fitness acts as a moderator (i.e. dimmer switch) of the relationship between workload spikes and injury. In this case, a given spike in workload will elicit a different injury risk dependent on an individual’s fitness level.
4.4 Not so simple, not so fast

Admittedly, these previous examples are simple illustrations of how mediation and moderation are conceptualised. In reality, the aetiology of injury is complex, dynamic, multifactorial, and context-dependent [131,180]. Therefore, for certain injuries, the effect of workload on injury is most likely more appropriately viewed as moderated mediation [181]. Combining the two aforementioned examples, a spike in workload may produce increased levels of neuromuscular fatigue, but the strength of that relationship may be moderated by aerobic fitness. In complex systems language, this example may manifest in a ‘risk profile’ which includes workload, aerobic fitness and neuromuscular fatigue as interacting factors within a ‘web of determinants’ – related to an ‘emergent pattern’ of non-contact injuries [180].

Expounding the causal relationship of workloads and injuries and explaining more complex causal pathways presents both methodological and analytical hurdles. First, study design and data collection must be conducted in a way so that mediator and moderator variables are both considered and measured. To reduce the plausibility of alternative explanations, randomised experimental designs should be considered where possible [181]. The a-priori selection and measurement of potential confounding variables, which do not lie on the causal pathway, should be simultaneously conducted [70]. Incorporating these additional variables requires greater sample sizes (i.e. increased power) and a large number of injuries to demonstrate these mediation/moderation effects [181]. Finally, adequate analysis of mediation and moderation often requires both complex designs, and more complex analyses, including structural equation modelling and multilevel modelling, to name a few [181].
4.5 What rewards await at the finish line after jumping the hurdles?

1) Improved understanding, better prevention – Mediation and moderation, as well as their more complicated cases, will improve our understanding of causal mechanisms behind injuries, and subsequent preventative strategies. Moderating variables which are modifiable are key intervention points to ‘dim’ the risk associated with training and competition workloads.

2) Addressing insufficiencies – The inability of screening single risk factors to predict future injuries has been understandably challenged [183]. However, shifting our conceptualisation of these variables from injury predictors to variables that moderate the load— injury relationship provides a different motivation for screening and addressing any shortcomings. For example, while aerobic fitness may not predict injuries, it may increase athletes’ resilience to higher workloads. Therefore, when athletes perform poorly on pre-season fitness tests, practitioners may choose to lower the allowable workload ‘threshold’ for this athlete, while providing individualised attention to address the deficiency.

3) Training smarter and harder – Ultimately, attaining high chronic training loads without rapid spikes in the process is considered current best-practice [112]. However, understanding the characteristics that make certain athletes more robust may allow for more nuanced training load prescriptions. For athletes with a collection of characteristics which ‘dim’ workload-related injury risks (e.g. high aerobic fitness, no previous injuries), practitioners may consider prescribing higher training workloads (e.g. an acute:chronic workload ratio of 1.7) for performance purposes, given that their associated risk is lower than that of the average athlete.
Chapter 5: Getting the most out of intensive longitudinal data: A methodological review of workload— injury studies (Paper 3)

5.1 Preamble

In the previous two chapters I described how athlete workloads and risk factors (internal and external) relate to injury risk. These aetiological models detailed that injury risk changes on a daily or per-session basis as workloads alter athletes’ individual risk profiles (either positively or negatively). Although these provided conceptual frameworks through which workloads relate to injury risk, the recursive, constantly changing nature of injury risk implied by these models makes analysing this association a challenge. Systematic reviews and consensus statements [74–76] and an International Olympic Committee Consensus Statement [65] have synthesised the existing workload— injury literature, but such reviews have not scrutinised the methodologies employed by the authors.

As part of my critical literature review (Chapter 1), I identified challenges in analysing intensive longitudinal data, such as workload— injury analysis. Specifically, I identified generalised estimating equations were often used to account for the dependency created by repeated measurements in these studies despite multilevel (i.e. mixed) modelling approaches having several advantages. This chapter reports my detailed review of this topic. It is in press at BMJ Open (accepted September 4th, 2018) and the rest of this chapter is a reproduction of text and figures from that manuscript [184]:

5.2 Introduction

Intensive longitudinal data (ILD) are being collected more frequently in various research areas [130], catalysed by technological advancements that simplify data collection and analysis [185]. By collecting data repeatedly on the same participants, researchers are enabled to answer more detailed research questions, particularly regarding phenomena that change or fluctuate over time. However, arriving at these answers requires researchers to overcome the challenges of analysing ILD, which include: (1) the dependencies created by repeated measures, (2) missing/unbalanced data, (3) separating between- and within-person effects, (4) time-varying and time-invariant (stable) factors, and (5) specifying the role of time/temporality [186].

The field of exercise and sports medicine provides one specific example that can illustrate principles that apply to the use of intensive longitudinal data broadly. In the field of sports performance, technological advances mean that a plethora of physiological, psychological and physical data are conveniently available from athletes [96,187]. As one example, of 48 professional football clubs that responded to a survey on player monitoring, 100% reported collecting daily global positioning system (GPS) and heart rate (HR) data [98].

One research question that has gained a great deal of interest in the last decade is how athletes’ training and competition workloads relate to injury risk. Since athletes’ training and injury risk continually varies over time, many researchers have used prospective cohort studies to collect and analyse ILD to answer this question [76]. There is moderate evidence from systematic reviews and an International Olympic Committee (IOC) consensus statement suggesting a positive relationship between injury rates and high training workloads, increased risk of injury with low workloads, and
a pronounced increase in injury risk associated with rapid workload increases [65,74–76,188]. However, such systematic reviews do not consider the statistical approaches used in included studies [189]. Choosing the wrong statistical analysis or poorly implementing an otherwise correct one (e.g. violating statistical assumptions) can bias results and create false conclusions. Even a perfectly performed systematic review cannot compensate for poorly designed, or poorly analysed studies [190].

Longitudinal data analysis is most effective when the chosen statistical approach aligns with the frequency of data collection and with the theoretical model underpinning the research question (See Box 1) [126]. Therefore, we used this lens to evaluate the statistical models employed in prospective cohort studies using ILD to investigate the relation between athletic workloads and injury. We had 3 aims: (1) to summarise researchers’ data collection, methodological, statistical, and reporting practices [189,191]; (2) to evaluate the degree to which the adopted statistical analyses fit within Collins’ three-fold alignment [see Box 1]; and (3) to provide recommendations for future investigations in the field.

Box 1: Theoretical model – temporal design – statistical model.

In a landmark, highly-cited paper, Professor Linda Collins described how aligning the (1) theoretical model (subject matter theory), (2) temporal design (data collection strategy/timing), and (3) statistical model (analytical strategy) is crucial when analysing longitudinal data [126]. For example, if researchers (1) theorise that a given physiological variable fluctuates every hour, (2) data must be collected at least on an hourly basis. If researchers measure participants once a day, they will miss virtually all the hourly fluctuations that their theories predict. Once researchers
have collected their hourly data, they should (3) select a statistical strategy that enables them to examine the relationship between these fluctuations and the outcome of interest. As Collins noted, perfect alignment of these 3 components may not be possible, but it provides researchers a target, and readers a lens through which longitudinal research can be evaluated.

5.3 Methods

5.3.1 Article selection

We systematically searched the literature (MEDLINE, CINAHL, SPORTDISCUS, PsychInfo, and EMBASE) (December 10, 2016) to identify systematic reviews and consensus statements that investigated the relationship between workloads and athletic injuries, with the aim of extracting all original articles included in these reviews that met our inclusion criteria. A summary of the systematic search and article selection process is described in Appendix A (Table A.1 and Figure A.1), and the full systematic search is available from the authors.

A priori, we operationally defined ‘workload’ as either external – the amount of work completed by the athlete (e.g. distance run, hours completed, etc.), or internal – the athlete’s response to a given external workload (e.g. session rating of perceived exertion, heart-rate based measures, etc.). We acknowledge that athlete self-reported measures often evaluate how athletes are handling training demands and may be referred to as ‘internal’ load measures, but we considered these perceptual wellbeing measures as a distinct step from quantifying athletes’ internal or external workloads [100]. Athletic injuries have been diversely defined in the literature, so we operationally defined athletic injury as any article that reported measuring ‘injury’, regardless of their specific definition (e.g. time loss, medical attention, etc.).
Two authors (JW + TG) screened the titles/abstracts of the systematic reviews. Where necessary, the full texts were retrieved to determine whether they should be included. A total of 6 systematic reviews [74–76,188,192,193] and 1 consensus statement [65] were identified that included at least 1 article meeting the inclusion criteria.

We extracted and reviewed the full texts of all the original studies included (n=279) in these 7 papers. For our analysis, we included all the original articles that met the following criteria:

1) Original articles were prospective cohort studies that examined the relationship between at least 1 measure of internal or external workload (as defined above) and athletic injury. Since theoretical models describe the recursive nature of injury risk with each training or competition exposure, workloads had to be continually monitored and include both training and match workloads for the same athletes. Although some athletes may have entered or left the group during the study period (e.g. through retirement or trades to other teams) the same team/group of athletes had to be followed throughout the study period, as opposed to repeated cross-sectional snapshots of different cohorts.

2) Articles collected intensive longitudinal data. We defined intensive longitudinal data as >20 observations per athlete [126].

3) Articles studied team sport athletes. We chose team sports because (1) there are high amounts of ILD collected in applied team sport settings [98], and (2) the majority of workload—injury studies are in team sport athletes (Jones, 2016). Military populations and
individual sports (e.g. distance running) were excluded due to the differences in task requirements and operating environment.

5.3.2 Patient and public involvement

As a methodological review, there was no patient or public involvement in this current investigation.

5.3.3 Article coding and description

To describe the methodological, statistical, and reporting approaches utilised in each article, two authors (JW + CA) reviewed all the included papers and extracted 50 items of information for each article. These items included publication year, journal, variable operationalisation (e.g. internal vs. external load measures, injury definition, etc.), methodological approaches, statistical analyses implemented, reported findings, and more. To ensure consistency between coders, 10 articles were randomly selected, coded independently by both reviewers, and compared to assess agreement. Discrepancies were discussed by the two coders and an additional 5 articles were randomly selected and coded independently. The remaining articles were coded by JW and checked by CA.

5.3.4 Assessing how statistical models aligned with Collins’ threefold framework

To evaluate the statistical approaches used in this field, we first identified the key themes and challenges within the theoretical models and temporal design features within the workload—injury field, then developed a qualitative assessment to evaluate the statistical approaches.
5.3.4.1 Collins’ component 1 – The theoretical models that underpin athletic workloads and injury risk (in brief)

Briefly, we identified at least 3 key elements of athletic injury aetiology models. First, *sports injuries are multifactorial* [36,69,71]. Aetiology models since 1994 have all explained between-athlete differences in injury risk by identifying a host of ‘internal’ (e.g. athlete characteristics, psychological wellbeing, previous injury) and ‘external’ (e.g. opponent behaviour, playing surface) risk factors. More recently, Meeuwisse et al.’s dynamic recursive model [4] and the workload—innjury aetiology model [131], have highlighted the recurrent nature of injury risk, meaning each athlete’s injury risk (i.e. within-athlete risk) also fluctuates continually as they train or compete in their sport (Figure 3.6). Thus, a second theme is that *injury risk differs between- and within-athletes*. Finally, more recent injury aetiology models have highlighted *injury risk as a complex, dynamic system* (Figure 5.1) [180,194]. Complex systems, as in weather forecasting or biological systems [195,196], possess many key features, including an open-system, inherent non-linearity between variables and outcomes, recursive loops where the system output becomes the new system input, self-organisation where regular patterns (risk profiles) may emerge for given outcomes (emergent pattern), and uncertainty [180].
Figure 5.1: Complex systems model of athletic injury.

Web of determinants are shown for an ACL injury in basketball players (A), and in a ballet dancer (B)
5.3.4.2 Collins’ component 2 – Temporal design / data collection

The theoretical models relating workloads and injury illustrate a continuously fluctuating injury risk, with many variables that influence risk on a daily or weekly basis [4,131,180]. Thus, if researchers want to investigate the association between workloads and injuries, these data must be collected frequently enough to observe changes in these variables as they occur (temporal design). With technological advances, athletes’ physiological, psychological and physical variables are now often collected on a daily, weekly or monthly basis, along with ongoing injury surveillance data [96,187]. Therefore, in the workload—-injury field, the theoretical models (injury aetiology models that describe regular fluctuation in workloads and injury risk) and the temporal design (frequent, often daily, data collection) are often well-aligned, especially in prospective cohort studies using ILD. This leaves us to consider only whether Professor Collins’ third component – the statistical model – aligns with these first two.

5.3.4.3 Collins’ component 3 – Statistical model

From the theoretical aetiology models underpinning the workload—-injury association, we highlighted three key themes to consider when choosing a statistical model: (1) injury risk is multifactorial, (2) between-athlete and within-athlete differences in injury risk fluctuate regularly, and (3) injury risk may be considered a complex, dynamic system.

From a temporal design perspective, intensive longitudinal data (ILD) are necessary to address these key themes, but they also carry at least 5 challenges that influence the choice of the statistical model.
1) Differentiating between- and within-person effects.

2) ILD include time-varying variables (e.g. workloads) and may also incorporate stable (time-invariant) variables (e.g., sex).

3) The ‘dependency’ created by repeated measurements of the same individuals violates the assumption of ‘independence’ common to many traditional analyses [127,197].

4) Almost all longitudinal datasets have missing or unbalanced data [126].

5) Longitudinal data analysis require researchers to consider the role of time in their analysis [186].

5.3.5 Evaluating statistical approaches

We deliberately tried to align components 1 and 2 of Collins’ framework by describing the theoretical models underpinning the workload—injury association and only including articles that had a temporal design characterised by ILD. To review whether statistical approaches aligned with these two components, two authors (JW + BZ) qualitatively assessed whether the statistical models, as employed in the included studies, (1) were multifactorial, (2) differentiated between- and within-athlete differences in injury risk, and (3) analysed the data as a dynamic system – the three themes highlighted in the theoretical framework. From the temporal design, the same two authors evaluated whether the statistical analyses (4) included both time-varying and time-invariant variables, (5) were robust to missing/unbalanced data, 6) addressed the dependencies created by repeated measures, and (7) incorporated time into the analysis.
5.3.6 **Data synthesis approach**

We first describe the characteristics of the included articles, then present our qualitative assessment of how well the various statistical approaches fit within Collins’ framework.

5.4 **Results**

Thirty-four articles were included in this methodological review (Appendix A). In the first 10 articles coded by both reviewers, there were 10 discrepancies out of 500 total coded entries (10 papers x 50 items/paper), which gave us 98% agreement between reviewers. No item had more than 2 discrepancies. Of the 250 study criteria in the second set of 5 articles coded by both reviewers, there were 8 discrepancies (97% agreement).

Included articles were published from 2003 – 2016, with 78% of the studies published since 2010. Sports studied included rugby league (n = 10), soccer (n = 7), Australian football (n = 6), cricket (n = 5), rugby union (n = 2), multiple sports (n = 1), and basketball, handball, and volleyball (n = 1 each). Studies included an average of 96 athletes (median = 46), ranging from 12 [141] to 502 athletes [198]. The observation period for these cohort studies ranged from 14 weeks [157] to 6 years [136]. Most studies investigated male athletes (n = 30), with 2 studies on female athletes, and 2 on both sexes. Table 5.1 summarises the included articles’ basic characteristics, while the full data extraction table is available from the authors upon request.
Table 5.1: Summary of included workload—injury investigations, sorted by sport then publication year

<table>
<thead>
<tr>
<th>Reference</th>
<th>Journal</th>
<th>Study Length</th>
<th>Sport</th>
<th>n</th>
<th>Level</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Rogalski et al., 2013) [140]</td>
<td><em>J Sci Med Sport</em></td>
<td>1 season</td>
<td>AFL</td>
<td>46</td>
<td>Elite</td>
<td>Male</td>
</tr>
<tr>
<td>(Colby et al., 2014) [138]</td>
<td><em>JSCR</em></td>
<td>1 Season</td>
<td>AFL</td>
<td>46</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Duhig et al., 2016) [199]</td>
<td><em>BJSM</em></td>
<td>2 Seasons</td>
<td>AFL</td>
<td>51</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Murray et al., 2016-2020) [201]</td>
<td><em>IJSPP</em></td>
<td>1 season</td>
<td>AFL</td>
<td>46</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Veugelers et al., 2015) [139]</td>
<td><em>J Sci Med Sport</em></td>
<td>15 weeks</td>
<td>AFL</td>
<td>45</td>
<td>Elite</td>
<td>Male</td>
</tr>
<tr>
<td>(Anderson et al., 2003) [141]</td>
<td><em>JSCR</em></td>
<td>21 weeks</td>
<td>Basketball</td>
<td>12</td>
<td>Sub-elite competitive</td>
<td>Female</td>
</tr>
<tr>
<td>(Dennis et al., 2003) [145]</td>
<td><em>J Sci Med Sport</em></td>
<td>2 Seasons</td>
<td>Cricket</td>
<td>90</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Dennis et al., 2004) [202]</td>
<td><em>J Sci Med Sport</em></td>
<td>1 Season</td>
<td>Cricket</td>
<td>12</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Dennis et al., 2005) [203]</td>
<td><em>BJSM</em></td>
<td>2002-2003 cricket season</td>
<td>Cricket</td>
<td>44</td>
<td>Sub-elite competitive</td>
<td>Male</td>
</tr>
<tr>
<td>(Saw et al., 2011) [204]</td>
<td><em>BJSM</em></td>
<td>1 season</td>
<td>Cricket</td>
<td>28</td>
<td>Elite</td>
<td>Male</td>
</tr>
<tr>
<td>(Hulin et al., 2014) [136]</td>
<td><em>BJSM</em></td>
<td>43 indiv. seasons / 6 years</td>
<td>Cricket</td>
<td>28</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Bresciani et al., 2010) [205]</td>
<td><em>Eur J Sport Sci</em></td>
<td>1 season (40 weeks)</td>
<td>Handball</td>
<td>14</td>
<td>Elite</td>
<td>Male</td>
</tr>
<tr>
<td>(Gabbett, 2004a) [158]</td>
<td><em>BJSM</em></td>
<td>3 years</td>
<td>Rugby league</td>
<td>220</td>
<td>Sub-elite</td>
<td>Male</td>
</tr>
<tr>
<td>(Gabbett, 2004b) [142]</td>
<td><em>J Sports Sci</em></td>
<td>1 season</td>
<td>Rugby league</td>
<td>79</td>
<td>Semi-professional</td>
<td>Male</td>
</tr>
<tr>
<td>Reference</td>
<td>Journal</td>
<td>Study Length</td>
<td>Sport</td>
<td>n</td>
<td>Level</td>
<td>Sex</td>
</tr>
<tr>
<td>----------------------------------</td>
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<td>----------------</td>
<td>----------------------</td>
<td>----</td>
<td>-------------</td>
<td>-------------</td>
</tr>
<tr>
<td>(Gabbett, 2010) [115]</td>
<td><em>JSCR</em></td>
<td>4 years</td>
<td>Rugby league</td>
<td>91</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Gabbett &amp; Jenkins, 2011) [143]</td>
<td><em>J Sci Med Sport</em></td>
<td>4 years</td>
<td>Rugby league</td>
<td>79</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Gabbett &amp; Ullah, 2012) [160]</td>
<td><em>JSCR</em></td>
<td>1 Season</td>
<td>Rugby league</td>
<td>34</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Hulin et al., 2015) [113]</td>
<td><em>BJSM</em></td>
<td>2 Seasons</td>
<td>Rugby league</td>
<td>28</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Hulin, et al., 2016) [168]</td>
<td><em>BJSM</em></td>
<td>2 Seasons</td>
<td>Rugby league</td>
<td>53</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Windt et al., 2016) [173]</td>
<td><em>BJSM</em></td>
<td>1 season</td>
<td>Rugby league</td>
<td>30</td>
<td>Elite</td>
<td>Male</td>
</tr>
<tr>
<td>(Killen et al., 2010) [157]</td>
<td><em>JSCR</em></td>
<td>14 weeks</td>
<td>Rugby league</td>
<td>36</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Brooks et al., 2008) [198]</td>
<td><em>J Sports Sci</em></td>
<td>2 seasons</td>
<td>Rugby Union</td>
<td>502</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Cross et al., 2015) [52]</td>
<td><em>IJSSP</em></td>
<td>1 Season</td>
<td>Rugby Union</td>
<td>173</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Arnason et al., 2004) [206]</td>
<td><em>AJSM</em></td>
<td>1 season</td>
<td>Soccer</td>
<td>306</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Brink et al., 2010) [207]</td>
<td><em>BJSM</em></td>
<td>2 Seasons</td>
<td>Soccer</td>
<td>53</td>
<td>Elite</td>
<td>Male</td>
</tr>
<tr>
<td>(Clausen et al., 2014) [209]</td>
<td><em>AJSM</em></td>
<td>1 Season</td>
<td>Soccer</td>
<td>498</td>
<td>Recreational</td>
<td>Female</td>
</tr>
</tbody>
</table>
**Table 5.1 continued:** Summary of included workload— injury investigations, sorted by sport then publication year

<table>
<thead>
<tr>
<th>Reference</th>
<th>Journal</th>
<th>Study Length</th>
<th>Sport</th>
<th>n</th>
<th>Level</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Owen et al., 2015) [210]</td>
<td><em>JSCR</em></td>
<td>2 consecutive seasons</td>
<td>Soccer</td>
<td>23</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Bowen et al., 2016) [211]</td>
<td><em>BJSM</em></td>
<td>2 Seasons</td>
<td>Soccer</td>
<td>32</td>
<td>Elite Youth Players</td>
<td>Male</td>
</tr>
<tr>
<td>(Ehrmann et al., 2016) [169]</td>
<td><em>JSCR</em></td>
<td>1 Season</td>
<td>Soccer</td>
<td>19</td>
<td>Professional</td>
<td>Male</td>
</tr>
<tr>
<td>(Malisoux et al., 2013) [212]</td>
<td><em>J Sci Med Sport</em></td>
<td>41 weeks</td>
<td>Varied</td>
<td>154</td>
<td>High-school</td>
<td>Both (65% males)</td>
</tr>
</tbody>
</table>
5.4.1 Data collection

5.4.1.1 Injury definitions

Injury definitions varied across articles, with exact wording outlined in the Online Supplementary Material. In Table 5.2, we have categorised the definitions into more discrete injury categories (and subcategories) in accordance with recognised consensus statements [9]. Where studies used multiple injury definitions, we categorised them according to the definition used for the primary analysis.

Table 5.2 Broad injury definitions used in workload—-injury investigations

<table>
<thead>
<tr>
<th>Injury Definition</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time-Loss</strong></td>
<td></td>
</tr>
<tr>
<td>All time-loss</td>
<td>13</td>
</tr>
<tr>
<td>Match time-loss</td>
<td>2</td>
</tr>
<tr>
<td>Non-contact time-loss</td>
<td>7</td>
</tr>
<tr>
<td>Non-contact match time-loss</td>
<td>1</td>
</tr>
<tr>
<td><strong>Medical Attention</strong></td>
<td></td>
</tr>
<tr>
<td>Medical attention</td>
<td>7</td>
</tr>
<tr>
<td>Player-reported pain, soreness, or discomfort</td>
<td>1</td>
</tr>
<tr>
<td>Non-contact medical attention injuries</td>
<td>1</td>
</tr>
<tr>
<td>Clinical diagnosis of jumper’s knee</td>
<td>1</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
</tr>
<tr>
<td>Injury scale on the Recovery-Stress Questionnaire for Athletes (REST-Q)</td>
<td>1</td>
</tr>
</tbody>
</table>
5.4.1.2 Subsequent or recurrent injuries

Of the 34 articles, 30 did not define or include subsequent or recurrent injuries. Of those that explicitly addressed subsequent injuries, two defined these injuries as those occurring at the same time and occurring by the same mechanism [208,210]. Two articles explicitly stated that they only considered time until first injury, meaning no injuries were subsequent or recurrent [145,203].

5.4.1.3 Workload definitions

Workload variables varied widely across articles and are summarised in Table 5.3. For a more detailed description of each article’s load measures, see the Online Supplementary Material. Many articles used workload metrics to derive additional variables from workload distribution over time (e.g. monotony, strain, acute: chronic workload ratios).

Table 5.3: Independent variables used in workload—-injury investigations

<table>
<thead>
<tr>
<th>Workload Measure</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal</td>
<td></td>
</tr>
<tr>
<td>- sRPE</td>
<td>15</td>
</tr>
<tr>
<td>- Heart rate zones</td>
<td>2</td>
</tr>
<tr>
<td>External</td>
<td></td>
</tr>
<tr>
<td>- Balls bowled or pitched</td>
<td>5</td>
</tr>
<tr>
<td>- GPS / accelerometry</td>
<td>10</td>
</tr>
<tr>
<td>- Hours</td>
<td>6</td>
</tr>
</tbody>
</table>

* If articles included more than one type of workload variable they are counted more than once. sRPE scores could be the original Foster scale or modified. GPS – Global positioning system. sRPE – Session-rating of perceived exertion (calculated as the product of session intensity on a 1-10 Borg Scale and activity duration in minutes).
5.4.1.4 Measurement frequency

Most included articles (n=32) collected workload data at every session that athletes completed, while 2 studies recorded workload on a weekly basis [209,213].

5.4.1.5 Handling missing data

Twenty-three of the 34 articles (67%) did not report any strategies for missing data. Of those that did, 5 used listwise or casewise deletion, and 6 used estimation. Estimation methods for players missing data included techniques such as: using the full team average values for the drills a player completed [211], using an individual’s mean weekly value [207], and multiplying player’s pre-season per-minute match data by the number of minutes they played in a match [138].

5.4.2 Statistical analysis and reporting in included articles

5.4.2.1 Data binning/aggregation

Although 32 articles collected daily workload measurements, many aggregated data for analysis. Most (n = 16) summed workload metrics for a total or average weekly workload. Three studies aggregated workload data for the entire year, 3 aggregated data into season periods, 2 aggregated data monthly, and 3 used multiple aggregation strategies.

5.4.2.2 Analysis methods

Table 5.4 summarises the statistical practices of applied researchers investigating the relationship between workload and injury. Although some studies had analysed other primary or secondary objectives, we recorded only the analyses used to investigate the workload – injury relationship.
Table 5.4: The number of studies using various statistical analysis techniques

<table>
<thead>
<tr>
<th>Analytical Method</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression modelling</td>
<td></td>
</tr>
<tr>
<td>- Logistic</td>
<td></td>
</tr>
<tr>
<td>- Regular</td>
<td>10</td>
</tr>
<tr>
<td>- Generalised Estimating Equation</td>
<td>5</td>
</tr>
<tr>
<td>- Multilevel</td>
<td>1</td>
</tr>
<tr>
<td>- Linear</td>
<td></td>
</tr>
<tr>
<td>- Regular</td>
<td>2</td>
</tr>
<tr>
<td>- Poisson</td>
<td></td>
</tr>
<tr>
<td>- Generalised Estimating Equation**</td>
<td>1</td>
</tr>
<tr>
<td>- Multinomial regression</td>
<td></td>
</tr>
<tr>
<td>- Regular</td>
<td>1</td>
</tr>
<tr>
<td>- Cox proportional hazards model</td>
<td>1</td>
</tr>
<tr>
<td>- Frailty model</td>
<td>1</td>
</tr>
<tr>
<td>Correlation</td>
<td></td>
</tr>
<tr>
<td>- Pearson</td>
<td>9</td>
</tr>
<tr>
<td>- Spearman</td>
<td>1</td>
</tr>
<tr>
<td>Relative risk/rate ratio*</td>
<td>8</td>
</tr>
<tr>
<td>T-tests</td>
<td></td>
</tr>
<tr>
<td>- Paired and independent samples</td>
<td>4</td>
</tr>
<tr>
<td>- Independent samples only</td>
<td>2</td>
</tr>
<tr>
<td>Chi-square tests</td>
<td>1</td>
</tr>
<tr>
<td>Repeated measures ANOVA (one or two-way)</td>
<td>5</td>
</tr>
</tbody>
</table>

If articles used more than one statistical method to analyse workload and injury, they are included more than once in the table. We only report analyses used to analyse workload— injury associations, not other analyses reported in the articles (e.g. ANOVA to test for differences in total workloads at separate times of the season).

* Relative risk here refers to the use of RR as a primary analysis based on risks in different categorical groups, not as an effect estimated from another model. For example, comparing risks among different load groups like Hulin et al., (2014, 2016, 2016) are counted here, whereas Gabbett and Ullah (2012) derived RR from their frailty model, and Clausen et al. (2014) derived RR from their Poisson model, but neither are included in the count for RR.

** Clausen et al. (2014) also report fitting multilevel models, but do not report any of the results – presenting only their GEE findings in their results and discussion.
5.4.2.3 Typical uses of statistical tools

Regression approaches were used most commonly (22/34 studies). The most common approach was logistic regression (binary injury status as the outcome variable), independently or jointly modelling workload variables as independent variables. Generalised estimating equations were used in 5 studies to account for the clustering of observations within players and were used very similarly to simple logistic regression approaches.

Correlation was the second most common method (10/34 studies). Most studies that used correlation (7/10) measured the association between weekly or monthly workloads and injury incidence at the team level. Of those that used correlation at the individual level, two compared the number of completed pre-season sessions with the number of completed in-season sessions [131,200], while the final compared workload with injury operationalised as a numerical score on the injury subscale of the Recovery-Stress Questionnaire for Athletes (REST-Q) [205].

Relative risk approaches were generally used in one of two ways. First, workload categories were established for the entire year, like cricket bowlers who averaged <2, 2-2.99, 3-3.99, 4-4.99 or >5 days between bowling sessions up until an injury, or for the entire year if they did not sustain an injury [145]. Risks were calculated as the number of injuries/number of athletes in a given group, and relative risks were calculated to compare across groups [204]. In the second approach, athletes contributed exposures on a weekly basis, and thus contributed to multiple workload classifications. In this case, the likelihood/risk was the number of injuries/number of weekly player exposures to that workload category [136,168,211].
Group differences were sometimes evaluated using t-tests, ANOVAs or chi-square analyses. Typically, unpaired t-tests contrasted workload variables (e.g. mean sessions/week) between athletes who sustained an injury during the year, to those who did not [145,203]. Paired t-tests and repeated measures ANOVAs (one- or two-way) were most often used to contrast the workloads of the same athletes at different time periods. For example, workloads in an ‘injury block’ (like the week preceding an injury), were contrasted with non-injury blocks, like other weeks in the season [145,204], or the 4 weeks preceding the injury block [212].

5.4.2.4 Justifications for statistical approaches

Authors of 15 of the included articles (44%) did not cite any sources to support their analytical choices. Of those who did, most (n=14) cited previous literature in the sports medicine field. Eight articles referenced statistics or methodology articles, 4 cited articles on Prof. Will Hopkins’ website (www.sportssci.org), and 3 cited statistical textbooks [214–216].

5.4.2.5 Addressing analysis assumptions and model fit

More than half (n = 20) the included articles did not report on the assumptions underlying their statistical analyses. Among those that did report on analysis assumptions, checks included checks for normality, collinearity of predictor variables in regression analyses [52], sphericity for repeated measures ANOVA [205], overdispersion [209], or correlation structures for generalised estimating equations [139].
When authors reported checking for normality, Shapiro-Wilk [200] or Kolmogorov-Smirnov tests [157] were referenced. Regression modelling was the most common analysis to investigate the workload—injury association. In 8/10 instances where simple logistic regression was chosen, the authors appear to have conducted the analyses using weekly observations without accounting for the dependencies created by repeated-measures across players. In all instances where regression was used, it was uncommon for authors to report that model assumptions were checked. Where multiple regression approaches were used, multicollinearity checks were rarely reported – an important consideration since multicollinearity can cause imprecise estimates of regression coefficients when multiple workload variables are simultaneously modelled [52,217,218].

Of the papers that modelled data using regression or similar techniques, six described how they assessed model fit. Some authors assessed specificity/sensitivity, or receiver operating characteristics, either on the current data set [213], or future data set [115]. Other in-sample model fit indices $R^2$ values [210], Aikeke Information Criteria (AIC) and Bayesian Information Criteria (BIC), which were sometimes mentioned as guiding the model selection process [173].

5.4.3 Alignment of authors’ statistical models with theoretical model and temporal design challenges

In Table 5.5 (a more detailed table – Table A.2 - is available in Appendix A), we qualitatively evaluated whether the statistical approaches chosen by the authors in our current review effectively addressed the key themes/challenges presented by the theoretical model and the temporal design (intensive longitudinal data). This table is an analytical tool to guide the reader through the discussion. It highlights the themes/challenges of the theoretical model and temporal design, as
well as the strengths/weaknesses of the statistical tools used in included studies. The table has the challenges/themes in columns and statistical tools in rows. The reader can follow a row to see how well a given statistical tool addressed key challenges as used by researchers in our included articles, or they can choose a challenge and follow the column down to see which analyses were used in a way that addressed that challenge adequately. The rows are ordered according to their qualitative ‘score’. As one proceeds down the rows, the statistical tools address more of the temporal design and theoretical model challenges.

We caution the reader that (1) not every possible statistical tool is included in the table, only those used in at least 1 article in our review, and (2) the evaluation is based on whether researchers of our included papers used a test in a way that addressed a given challenge, not necessarily whether the test is capable of being used in a way that meets that challenge. For example, a logistic regression analysis conducted using a generalised estimating equation framework can include multiple explanatory/predictor variables, thereby allowing for a multifactorial model. However, some authors used GEEs and only included one predictor variable [159,202], in which case the GEE did not address the multifactorial theme.

**5.5 Discussion**

We used the workload—injury field of medical research to examine whether statistical approaches analyse intensive longitudinal data optimally. By design, the theoretical models underpinning the workload—injury field and the temporal design (ILD) were aligned in all the included articles, but common statistical approaches varied in how adequately they addressed the key themes needed to align them with the other two components.
5.5.1 Consideration #1 – Theoretical theme – multifactorial aetiology

Sports injury aetiology models of the last 2 decades have highlighted the multifactorial nature of athletic injury [69,71]. We asked whether the burgeoning body of research relating workloads and injury, is using modern statistical methods to capture workloads while incorporating known risk factors. Few articles in this review incorporated previously identified risk factors and workload into the same analysis. In some instances, the analytical approach prevented this from being an option. For example, simple analyses like t-tests, correlations, and chi-square tests do not allow for multiple variables to be included. In other instances, the statistical approaches allowed a multifactorial approach (e.g. generalised estimating equations) but researchers opted to focus on the effects of workloads in isolation [159,202].

Including known risk factors in workload—injury investigations is important from an aetiological perspective in at least two ways. First, failing to control for known risk factors may mean that key confounding variables are not included in the analysis and the relationship between workloads and injury are spurious. For example, women have a 2-6 times higher risk of ACL injury in soccer than their male counterparts [3,219]. If a study included both male and female soccer players and did not account for sex in the analysis, then differences in workload may be spuriously correlated with injury rates if male and female players performed varying levels of workload. Depending on the injury type and sporting group, previous injury, age, sex, physiological and/or biomechanical variables may all be important to include.
Secondly, by including additional risk factors into the analysis, the investigator may be able to identify moderation or effect-measure modification to better understand how risk factors and workload jointly contribute to injury risk [179,220]. As a reminder, there are subtle, but important differences between mediation, moderation, and effect measure modification that will influence analytical choices [221,222]. Effect modification occurs when the effect of a treatment or condition (e.g. a given workload demand), differs among different athlete groups. Interaction (or moderation), although similar, examines the joint effect of two or more variables on an outcome. Finally, mediation is concerned with the pathway of exposure to a given outcome, and what are potentially intermediate variables. Previously identified risk factors may aetiologically relate to workload in each of these three ways and may be explored through different modelling strategies.
Table 5.5: Evaluation of the degree to which authors’ use of statistical tools addressed theoretical and temporal design challenges

<table>
<thead>
<tr>
<th>Method</th>
<th>Themes of theoretical model</th>
<th>Themes of temporal design - intensive longitudinal data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Multifactorial aetiology</td>
<td>Between and Within-Athlete Differences</td>
</tr>
<tr>
<td>Correlation (Pearson and Spearman)</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Unpaired t-test</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Chi-square tests</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Relative risk calculations</td>
<td>0</td>
<td>X</td>
</tr>
<tr>
<td>Regression (logistic, linear, multinomial)</td>
<td>0</td>
<td>X</td>
</tr>
<tr>
<td>Paired t-test</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Repeated measures ANOVA (one or two-way)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Generalised Estimating Equations (Poisson and logistic)</td>
<td>0</td>
<td>X</td>
</tr>
<tr>
<td>Cox proportional hazards model</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Multilevel modeling</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Frailty model</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

Qualitative assessment performed on a three-tiered scale. An ‘X’ (red formatting) means that none of the authors using this tool adequately addressed that specific challenge. In some cases, this may be because the statistical model was unable to address it, and other times it may be because of the way they used it. An ‘O’ (yellow formatting) indicates that some authors addressed that challenge while others did not. This generally happened when the statistical tool could address a challenge but the authors sometimes chose not to use it in that way. A ‘’ (green formatting) indicates that all authors using this statistical tool addressed that challenge adequately. *Missing/unbalanced data here is that caused by intensive longitudinal data – meaning a different number of observations for each athlete during the observation period, some of which may be missing.
Statistical approaches that allow multivariable analyses enabled researchers to examine the effects of workloads while controlling for known risk factors. Malisoux et al. (2013) used a Cox-proportional hazards model to control for age and sex while examining the effects of average training volume and intensity. Gabbett and Ullah’s frailty model (2012) incorporated previous injury – a proven injury risk factor – into the evaluation of the influence of different GPS workloads on injury risk. When investigating multifactorial phenomena, statistical approaches that enable multiple explanatory variables provide a more appropriate option.

### 5.5.2 Consideration #2 – Theoretical theme - between and within-athlete differences

One of the primary benefits of ILD is that it enables researchers (when using certain analyses) to differentiate within-person and between-person effects [186]. In the sports medicine field, this would correspond to researchers asking, (1) why do some athletes suffer few injuries (between-person inquiry) while others appear ‘injury-prone’? and on the other hand, (2) at what point is a given athlete (within-person inquiry) more likely to sustain an injury? The simpler statistical approaches used by researchers in our included studies (correlation, t-tests, ANOVAs, regular regression) are limited in the number of variables they can include, and consequently cannot differentiate risk between- and within-athletes. Tests of group differences (independent sample t-tests and one-way ANOVAs) only differentiate between athletes (e.g. injured vs. uninjured), while repeated measures tests (repeated measures ANOVA and paired t-tests) only examine within-athlete differences (e.g. loads preceding injuries vs. loads during non-injury weeks).

Generalised estimating equations (GEE) were commonly used to address some of the longitudinal data challenges. Although this approach accounts for the clustering within-persons, it assumes the
effects of predictor variables are constant across all athletes [223]. Simple Cox proportional hazards models (Malisoux et al. 2013) are common in survival analyses, but do not differentiate between- and within-person effects [224].

Only two statistical tools were used in a way that examined between- and within-athlete differences in injury risk. The frailty model by Gabbett and Ullah (2012) modelled each athlete as a random effect with a given frailty. The multilevel model by Windt et al., (2016) incorporated athlete-level variables (age, position, pre-season sessions) and observation-level variables (weekly workload measures). In the latter case, athletes’ weekly distances did not affect their risk of injury in the subsequent week (OR = 0.82 for 1 standard deviation increase, 95% CI = 0.55 to 1.21) – a within-athlete inquiry. However, controlling for weekly distance and the proportion of distance at high speeds, athletes who had completed a greater number of pre-season training sessions had significantly reduced odds of injury (OR = 0.83 for each 10 pre-season sessions, 95% CI = 0.70 to 0.99) – a between-athlete inquiry. These two examples highlight that certain analyses carry a distinct advantage of allowing researchers to tease out differences both between- and within- study participants.

5.5.3 Consideration #3 – Theoretical theme - injury risk as a complex dynamic system

Complex systems are defined, among other things, by the interaction between multiple internal and external variables that interact to produce an outcome. Simple analyses (t-tests, correlations), which cannot incorporate multiple variables, cannot examine the interaction between multiple factors. However, even other traditional analyses which are more effective in handling the
challenges of longitudinal data (e.g. generalised estimating equations, Cox-proportional hazards models) were not used to incorporate non-linear interactions between predictor variables.

The most recent reviews of athletic injury aetiology have highlighted complex systems models [180,225]. None of the analyses included in our review analysed intensive longitudinal workload—-injury data with statistical analyses that fit within a complex systems framework. This lack of research may reflect the fact that the suggestion that injury aetiology fits within a dynamic, complex systems framework is still relatively ‘new’ in this field. It remains to be seen whether a complex systems approach and the analyses recommended in such reviews (e.g. self-organising feature maps, classification and regression trees, agent based models, etc.) are more effective for evaluating the association between workloads and injury [180].

5.5.4 Consideration #4 – ILD challenge – including time-varying and time-invariant variables

Tying back to the theoretical model of workloads and injury, some relevant factors may be relatively stable (time-invariant) over the course of an observation period (e.g. height, age), while others are time-varying (e.g. workload). Some analyses can incorporate both time-varying and time-invariant variables, while others are limited in this respect. All analyses that cannot or did not address the multifactorial nature of injury cannot include time-varying and time-invariant variables concurrently. Group difference tests (t-tests, ANOVAs, etc.) may collect time-varying measures, but must aggregate them into a single average for analysis.
Including time-varying and stable variables in the same analysis links closely to between- and within-athlete differences – with the frailty model [160] and multilevel model [173] both used in a way that allowed the researchers to include both. The one exception in our included studies was the generalised estimating equation approach. As mentioned earlier, the GEE assumes an ‘overall’ effect for each explanatory variable, such that between- and within-athlete differences cannot be differentiated [226]. However, the major benefit to a GEE is that it accounts for the repeated measures for each participant and can therefore include both time-invariant and time-varying variables for each participant.

5.5.5 Consideration #5 – ILD challenge – handling missing and unbalanced data

Dealing with missing and unbalanced data is a near certainty when collecting ILD, and is common in applied workload-monitoring settings [227]. Such missing data decreases statistical power and increases bias, and may be missing at completely at random (MCAR), missing at random (MAR), or missing not at random (MNAR). When analysing aggregated data or using analyses that require balanced data, strategies may include complete-case analysis, last observation carried forward, or various imputation methods [228,229]. Multiple imputation methods, of which there are many, involves replacing missing values with values imputed from the observed data and is preferred over single imputation. Finally, if interactions are included in regression analyses, the transform-then-impute method has been recommended [230].

However, these missing data approaches are not recommended for longitudinal analyses, since researchers have statistical analyses that are robust to missing and unbalanced data at their disposal [128]. Statistically, four types of analyses used in this review are robust to missing and unbalanced
data – Cox proportional hazards models, GEEs, multilevel models, and frailty models, where all observations can be included in the analysis, and athletes can have different numbers of observations. Since mixed/multilevel models have less stringent assumptions for missing data (i.e. missing at random) than generalised estimating equations (i.e. missing completely at random), they have been suggested over GEEs [128].

While the statistical concerns related to unbalanced data may be addressed with these analyses, missing data may also affect derived variables, which are common in workload—iinjury research. These derived variables include rolling workload averages (e.g. one-week, ‘acute’, workloads, four-week average, ‘chronic’, workloads, etc.) [136,211], ‘monotony’ (average weekly workload divided by the standard deviation of that workload) or ‘strain’ (the monotony multiplied by the average weekly workload) [141]. Since these measures are all calculated from workloads accumulated over time, failing to estimate workloads for these missing sessions (that end up being treated as ‘0’ workload days) means inferences from these derived measures may be underestimated and unreliable. Few authors discussed how they handled missing data. In these instances – it is important that researchers report how they accounted for missing data, whether they be strategies employed in the past – e.g. full team average values [211], weekly individual averages [207], player specific per-minute values by time played [138] – or whether through other advanced imputation methods recommended for ILD [228,230].

5.5.6 Challenge #6 – ILD challenge – dependencies created by repeated measures

Collecting ILD in applied sport settings means repeated (often daily) measurements of the same athlete – such that observations are clustered within athletes. Comparisons of independent groups,
through chi-square tests, independent sample t-tests and one-way/two-way ANOVAs all assume participants contribute a single observation to the analysis and force an aggregated variable (e.g. average number of balls bowled in a week) to conduct the analysis [145]. Similarly, correlation and simple regression (in its linear, logistic, and multinomial forms) assume independence of observations [231]. Paired t-tests and repeated measures ANOVA were used to deal with repeated measurements by comparing the same athletes’ workloads at different periods (e.g. the week before injury vs. weeks that did no precede injury).

Of the analyses that addressed this challenge, GEEs were used most commonly (6 studies). GEE’s ability to handle clustering was also used in one article to control for players clustering within teams [209]. Cox proportional hazard models, used in one article [212], can handle repeated measurements for participants [232]. Multilevel models [173] and frailty models [160] – an extension of the Cox-proportional hazards model – were also used in a single instance each, where repeated measures were clustered within players through a random player effect.

As mentioned in our introduction, there may be additional data dependency created by recurrent injuries [38]. Previous recommendations to handle the recurrent injury challenges have included frailty models [44], and a multistate framework [40]. However, as so few articles reported collecting information on recurrent injuries (n = 5), we focused primarily on the dependencies caused by repeated measures across participants.
5.5.7 Challenge #7 – ILD challenge – incorporating time into the analysis / temporality

One of the most relevant questions in ILD analyses is the way that time is accounted for [130,186]. Some authors used one-way [157] and two-way repeated measures ANOVAs [158] to compare loading in different seasons or season-periods – a very simple way of accounting for time. Repeated measures ANOVAs [169,200,212] and paired t-tests [145,204] also account for time by categorising time-periods as pre-injury blocks or non-injury blocks. Multilevel models have been used to examine change through the interactions of variables with time, but the one multilevel model used in this review did not include time as a covariate [173]. Survival analyses explicitly account for time by calculating the effects of variables on the predicted time-to-event [160,212]. Notably, only one analysis – the frailty model ([160]) – adjusted the probability of long-term outcomes (e.g., injury) based on variations after an initial capture of risk, something few traditional analyses accomplish [195].

Temporality is also vital in considering potential causal associations. While making causal inferences from observational data is a topic beyond the scope of this paper, temporality is a well-accepted component of causality dating back, at least, to Bradford Hill’s ‘criteria’ [233]. Without temporality – where a postulated cause precedes the outcome – directional associations cannot be made [234,235]. A lack of temporality can also skew associations since it allows for reverse causality. In the workload—injury field, findings that high weekly workloads are sometimes associated with lower odds of injury in a given week [139,173] may be in part because players who get injured in a given week are less likely to accumulate high weekly workloads. Trying to account for temporality, some researchers have included a latent period – where workload variables are examined for their association with injury occurrence in a given proceeding
time window, like the subsequent week [136,173]. While recent work has noted that the length of the latent period may affect model findings [236], it is clear that without some type of latent period, any directional inferences between workloads and injury cannot be made.

5.5.8 Methodological, statistical and reporting considerations

5.5.8.1 Data aggregation

Data aggregation was common, whether in data preparation, or forced through the analysis. In some cases, researchers aggregated individual level data into team-level measures (total/average workload and injury incidence). Although 32/34 articles collected daily data, most aggregated these daily data into weekly measures, potentially contributing to temporality problems if no latent period was included. Finally, certain analyses (e.g. paired t-tests, simple logistic regression) aggregated data for athletes across an entire year so that workload measures were used to control for exposure [206,213]. Differences in analyses make it impossible to measure the effect of fluctuations in workload and potential impact on injury risk. Further, with no latent period, the directionality of the relationship is unclear. For example, players with high exposure throughout the year were at a lower injury risk than the intermediate group, but it could be interpreted that players who do not sustain an injury throughout the year are more likely to accumulate high total training and match exposures (i.e. higher workload) [206]. Aggregated data may be easier to analyse but comes at the cost of losing some of the inherent benefits of collecting ILD, such as the changes in injury risk that occur at a daily level. As a result, theory-driven questions that relate to daily workload fluctuations and injury risk will become challenging, or impossible to answer.
5.5.8.2 Checking model assumptions and fit

While many studies may have under-reported how they assessed model assumptions or fit, others [52] provide an example for other researchers to emulate. In fitting a GEE to account for intra-team and intra-player clustering effects, they explained how they selected an appropriate autocorrelation structure, reported how potential quadratic relationships were assessed in the case of non-linear associations, and described checking for potential multicollinearity with defined thresholds (variance inflation factor >10) and for their GEE.

5.5.9 Researcher ‘trade-offs’, consequences of misalignment

We used the workload—injury field to highlight seven themes that relate to theoretical injury aetiology models and temporal design (ILD). In many cases, published studies’ statistical models either could not, or were not used in a way that addresses these themes. In some cases, misalignment may carry a severe cost – like assumption violations that may bias study results [197]. This is akin to building conclusions on an unstable foundation. Other times, researchers have properly employed their chosen statistical approach, but the approaches themselves were limited, and unable to answer research questions that ILD can address. This is more akin to having a grand building plan and all the necessary supplies, but only using a screwdriver to construct the building.

Simple regression models provide an ideal example of researchers’ trade-offs when using traditional statistical analyses on ILD, and the potential costs of misalignment. Although 13 papers used regular regression to analyse the association between workloads and injury outcome, they chose one of three paths when dealing with ILD. First, many proceeded to analyse each daily or
weekly data point as an independent observation – not addressing the violation of the independence assumption [136,140,168]. Second, some researchers aggregated the workload data into an average weekly workload or total workload exposure over the course of the year, such that each participant contributed only one observation to a classic logistic regression [206,213]. Although the regression assumptions were not violated, workload was aggregated into a single metric, the temporal relationship between workload and injury was lost, and there was then no way to analyse the effects of workload fluctuations on injury risk. Third, some researchers converted individual data to team level data and examined team workloads with team injury incidence in a linear regression [142,210]. In this final case, no differentiation could then be made between players or within-players, and inferences were only possible at the team level. This may be sufficient to inform research on the association of workloads and injury at the team level, but the theoretical model underpinning team injury rates may differ from those that underpin individual athletes’ injury risk.

5.5.10 Review limitations

Previous systematic reviews investigating the workload— injury relationship have documented the challenges of identifying articles through classic systematic review search strategies [74,76]. Heterogeneous keywords and the breadth of sporting contexts have meant previous systematic reviews include many articles post-hoc that were not originally identified by their systematic searches (e.g. 29 of 67 articles in Jones [76], 12 of 35 articles in the paper by Drew and Finch [74]). Therefore, although we worked to identify articles through 6 systematic reviews [74–76,188,192,193] and the 2016 IOC consensus statement on athletic workloads and injury [65], we may have missed potentially eligible articles.
We used the cut-off for intensive longitudinal data (>20 observations) proposed by Collins (2006). However, there is no universal cut-off for ILD, with previous thresholds of ‘more than a handful’ [130], ten observations [237], or forty [186].

In some instances, authors’ analytical choices may have been attributable to factors outside of statistical considerations. For example, in lower level competitions, or in organisations with lower budgets, it may not have been feasible to collect multiple variables longitudinally with the available equipment or staff. In these types of instances, authors would be unable to employ a multifactorial approach, instead of choosing not to use one. Such external factors may have influenced the findings of this methodological review.

Finally, it was beyond the scope of this review to list every challenge posed by ILD, and we were not exhaustive in our discussion of different analyses and their capacity to handle the challenges. Where possible, we tried to identify the themes that are most common within the research field of Sport and Exercise Medicine field. Ultimately, our call to action is that statistical tools be chosen more thoughtfully so that the extensive work put into theory building and data collection is not short-changed by a sub-optimal statistical model.

5.5.11 Longitudinal improvements in ILD analysis

Methods and statistical analyses evolve over time, as with all scientific inquiry. Therefore, it is possible that we were a little unfair to some earlier papers. For example, researchers may have chosen analyses that aligned with ‘their’ theoretical model at the time, not what is considered the most current theoretical model. However, most papers were published since 2010 – the dynamic,
recursive aetiology model was introduced in 2007, and the multifactorial nature of injury risk has been highlighted since 1994 [69]. As complex systems approaches are the most recently proposed theoretical model [180,225], it is not surprising that none of the included articles analysed the data within this type of framework, with the first analysis of its kind in sport injury research only appearing recently [238]. Further, some techniques for longitudinal data analysis have been developed and grown in popularity recently, so researchers may not have been aware of alternative approaches at the time of their studies.

As more statistical methods are developed and refined for longitudinal data analysis, researchers will continue to gain awareness and skills with these analyses and their implementation is likely to become more common. Some evidence for that progression can be seen in this review. If we were to assign a ‘method’ score to each analytical approach outlined in Table 5.1, assigning 0 for each red box, 0.5 for each yellow box, and 1 for each green box (e.g. correlation would score 0, while generalised estimating equations would score a 3.5), and then assign that score to each paper in the study, we could obtain a rough estimate of whether analytical approaches were improving over time. Breaking the papers roughly into four periods, the ‘average score’ for papers up to 2005 (n = 6) is 1.6, papers between 2006 and 2010 (n = 7) score an average of 1.9, papers between 2011-2015 (n=11) score 1.7, and papers since 2016 (n = 10) score an average of 2.3. Moreover, since the search for this current review was conducted, there have been promising developments in the sports medicine field and a continued improvement in longitudinal analysis. Recent publications have applied statistical models that more appropriately take advantage of the strengths inherent to ILD, and better align with the theoretical frameworks [239–244].
Mediation, effect measure modification, and interaction/moderation are all causal models which may also contribute to aetiological frameworks [181]. We recently proposed that traditional intrinsic and extrinsic risk factors may act as moderators or effect measure modifiers of the workload— injury association [179]. If that is true, the most appropriate statistical model would include workload measures as the independent variable of interest, and incorporate other risk factors such that these causal models can be investigated, whether by stratifying effects across different levels of these risk factors, or including an interaction term within regression [220]. While no included articles performed such an analysis, recent studies (not included in this review because it was published after our search) have started to adopt these approaches [241,245,246]. For example, Møller et al. used a frailty model with weekly workload fluctuations (decrease or <20% increase, 20-60% increase, and >60% increase) as the primary predictor variable in a frailty model. Known shoulder risk factors were treated as ‘effect measure modifiers’, so the model was stratified based on the presence or absence of a given risk factor (e.g. scapular dyskinesis) [222]. In so doing, the researchers used a statistical tool (Component #3) that addressed all the challenges inherent to longitudinal data (Component #2), conducting a multifactorial analysis that clearly differentiated both within- and between-athlete injury risk – key aspects of the theoretical model (Component #1).

5.5.12 Future directions and recommendations for ILD analysis

Researchers in the sports medicine field should be encouraged that the increased availability of ILD may improve understanding of athletes’ fluctuating injury risks – as articulated by their theoretical models. More advanced statistical techniques for longitudinal data are increasingly being developed and implemented across disciplines. This will enable sports medicine researchers
to more accurately answer their theory-driven questions by taking advantage of the benefits of ILD. To capitalise on this understanding, researchers must choose statistical models that most closely align with their theory and that address longitudinal data challenges. Generalised estimating equations, a Cox proportional hazards model, a multilevel logistic model, and a frailty model were the 4 analyses that most closely approached this alignment within our included papers. However, there remains some clear room for improvement in the future.

First, although mixed modelling was only used in one study, these forms of analyses have inherent values over GEE methods and have been recommended for this reason [247]. Because of sample structure, mixed models prevent false positive associations and have an applied correction method that increases the power of the analysis [248]; a finding that is useful with the commonly smaller samples. Mixed models also carry a less stringent missing data assumption (missing at random) when compared with GEEs (missing completely at random). Further, whereas GEEs require the correlation structure to be chosen by the researcher (which may be wrong), mixed models model the correlation structure so that it can be investigated. Finally, GEEs assume a constant effect across all individuals in the model, while mixed models allow for individual level effects and for differentiating these individual effects.

To borrow an example from another field and demonstrate the flexibility and utility of mixed effect models, Russell et al. used daily stressor values from students during their first 3 college years to demonstrate that students consumed more alcohol on high-stress days than low-stress days (within-person fixed effect) [249]. However, a significant random effect between students suggested that some students experienced this increase in alcohol consumption, while others did not. Finally,
those students with a tendency to increase alcohol consumption with stressors were more likely to have drinking-related problems in their 4th year [249]. For more information on multilevel/mixed effect models for longitudinal analysis, readers are referred to other helpful resources [127,128,130,250,251].

Time-to-event models are another family of statistical models that have become a very common in clinical research articles – reported in 61% of original articles in the New England Journal of Medicine in 2004-2005 [252] – but were used infrequently within our included articles. Notably, these models answer a different research question – when does an event occur? These approaches can account for many of the ILD challenges [253–255]. Time-to-event models account for censoring, can incorporate time-varying exposures, time-varying effect measure modifiers, and time-varying changes in injury status, and may be used to control for competing risks [253]. As with other modelling techniques, the appropriate number of events per variable has been investigated, and at least 5-10 events per variable are recommended for these types of models to prevent sparse data bias [256]. As long as this and other model assumptions are met, more advanced time-to-event models may be a valuable tool for researchers analysing ILD [232,257,258].

Lastly, computational modelling methods, which involves computer simulation has both pros and cons where modelling injuries. On one hand, they may provide insight on the best ways to model certain predictor variables [259], and open the door to more complex systems modelling (e.g. agent-based modelling) [238]. Though they show promise, such simulation studies are based on artificially generated data and must be interpreted carefully [260].
More analytical approaches are available for ILD, but a full discussion of each of these is beyond the scope of this paper. For the interested reader, functional data analysis [261], machine learning approaches [239,242], time series analysis [129], and time-varying effect models [262] all show promise. Such analyses and others for ILD can be found in Walls and Schafer’s landmark ILD textbook [130], and more recently, in the work of Bolger and Laurenceau [250].

We believe intensive longitudinal data provide an exciting opportunity for applied researchers and statisticians to collaborate moving forward. As the field continues to progress to more advanced analytical approaches that may better suit ILD, the need for collaboration with statisticians will be vital. In our included papers, few researchers referenced methodological or statistical references to justify their analytical approaches. In some instances, this may be attributable to using common, relatively simple analyses – one likely does not expect a citation for a t-test. Where such references existed, they were often to previous papers in the field, not statistical sources. In future longitudinal analyses, we encourage researchers to partner with a statistician, psychometrician, epidemiologist, biostatistician, etc. [263]. Such fruitful collaborations may lead to statistical approaches that take full advantage of intensive longitudinal data by aligning theory, data collection and statistical analyses as seamlessly as possible.

5.6 Conclusion

We used studies investigating the relationship between workloads and injury as a substrate to highlight to researchers how important it is to align their theoretical model, temporal design, and statistical model. In longitudinal research, thoughtfully chosen statistical analyses are those
grounded in subject matter theory and that maximise the utility of the collected data. The three most common analyses in our included papers (logistic regression, correlations, and relative risk calculations) addressed one or none of the 3 key theoretical themes, and one or fewer of the 4 inherent challenges of intensive longitudinal data. In this example discipline, researchers have developed sophisticated theories and frequently collect data that enable them to test these theoretical models. The missing step, and future opportunity for researchers, is to avail themselves of all the tools at their disposal – choosing statistical models that address the ILD challenges and answer theory-driven research questions.
Chapter 6: Does player unavailability affect football teams’ match physical outputs? A two-season study of the UEFA champions league (Paper 4)

6.1 Preamble

In Chapter 5, I reviewed 34 original prospective cohort studies that analysed intensive longitudinal data to investigate the workload—-injury association. Authors selected generalised estimating equations most frequently to account for the dependency created by repeated measurements in these studies. Multilevel (i.e. mixed) modelling approaches have distinct advantages over GEEs, as described in section 5.5.12, and only one of the articles used a mixed modelling framework (Chapter 8).

In this chapter, I demonstrate the utility of mixed modeling using an intensive longitudinal data set. The investigation, which I carried out in partnership with the Football Research Group¹, altered the explanatory and outcome variables – asking whether teams’ player unavailability (because of athletic injuries) was associated with match physical outputs (e.g. distance run, number of high velocity sprints). The repeated matches played by each team exposed the analytical challenges highlighted in Chapter 5, with match observations clustered within each team. Given the benefits of mixed modelling approaches over GEEs and other analyses ill-suited for ILD, I employed a mixed model to investigate whether player unavailability was associated with any changes in teams’ match physical outputs.

¹ Football Research Group (FRG) is a collaborative, international research group anchored at Linköping University, partnering with the European Football Union and elite football clubs, including Real Madrid, FC Barcelona, Juventus and Manchester United. Since its inception in 2001, 55 clubs from 18 countries have participated. The FRG database contains over 22,000 injuries and over 2 million hours of exposure, making it the world’s largest database on male professional football players. The FRG’s goal is that exercise and football participation should be safe, with minimal injury risk and side effects.
The rest of this chapter is a reproduction of text and figures from the following article [264]:


### 6.2 Introduction

Injuries are common in professional football [265,266] and negatively affect team performance [53,56,134]. While the link between injuries and team performance has been established [56], the mechanisms through which injuries affect teams’ match play and subsequent performance has not been investigated. Given the multifaceted nature of team sport performance, having more players unavailable may cause teams to alter their tactics, as less skilled players are selected may result in worse technical proficiency and altered physical performances during match play. While all these avenues provide future research opportunities, we will focus on the association between player unavailability and football teams’ match physical outputs.

Individual players’ physical outputs – including their total distance run, high speed distance run, number of sprints performed, time spent at different velocities, number of repeated sprint demands, and many other variables – are now readily quantified. This has enabled researchers to examine intra- and inter-individual variation over successive matches [267–269], as well as the effects of player position [270], team formation [271], team ranking [272], and fatigue (transient and end-game) [273]. To our knowledge, whether player availability due to injury is related to any changes in football teams’ physical outputs has not been examined.
If player unavailability is associated with changes in match physical outputs, sports medicine and science staff may benefit in two ways. First, the impact of injuries may be more readily quantified – improving preventative efforts. Second, teams with more players unavailable for selection may better understand what to expect in coming matches and therefore prepare appropriately. Our objective was to investigate whether player availability was associated with any changes in teams’ match physical outputs. Theoretically, having more players unavailable due to injury may cause less ‘match fit’, or more inexperienced players to be selected for match play, and reduce teams’ ability to utilise squad rotation during times of fixture congestion. Therefore, we hypothesised that player unavailability would be associated with a decrease in teams’ physical outputs.

6.3 Methods
The UEFA Champions League (UCL) injury study is an ongoing prospective injury surveillance study of male professional football that started in 2001 [274]. The current study cohort followed the teams participating in the UCL injury study who competed in the 2014-2015 and 2015-2016 UEFA Champions League competitions. Only UCL matches were included, without any league matches. UCL fixtures are generally played every 2-3 weeks on Tuesdays and Wednesdays between September and May. Teams play 6-12 matches depending whether they advance to the knockout stage and how long they remain in the competition.

Data collection procedures and definitions followed the UEFA guidelines [274]. and were consistent with the consensus statement for injury surveillance in football [9]. All players
completed informed written consent and the study design was approved by the UEFA Medical Committee.

First teams’ medical staff members logged all injuries on a standard injury form, sent to the study group each month. A time-loss injury definition was used for this investigation, with injuries recorded whenever a player’s physical complaint prevented full football training or match play [9]. Players were considered injured until the medical staff allowed full participation in training and availability for match selection. The number of players unavailable due to injury was calculated for each match as the ‘count’ of how many players were designated as ‘injured’ on the date of the match and therefore unavailable for selection. Players unavailable due to international duty, yellow cards, or other reasons were not included in this count.

Performance data were collected through video tracking and analysed by Deltatre. These are the same data Deltatre analyses for UEFA and reports to teams during and after matches [275]. The analysis algorithm used to calculate physical outputs was consistent across seasons, but the video tracking hardware provider changed between seasons – Stats in the 2014/2015 UCL Season and ChyronHego in the 2015/2016 Season. Therefore, we chose to analyse each season separately.

Physical output data included total distance run (km); distance run (km) and time spent (minutes) at low, medium, and high-speeds; number of sprints performed (n). Speed thresholds were set at $< 0 - 10 \text{ km/h}$ for low intensity, $<10 – 14 \text{ km/h}$ for medium intensity, $>14 \text{ km/h}$ for high intensity, and $20 \text{ km/h}$ for sprinting [267,276]. We specifically selected three physical output variables for this analysis – 1) total distance run, 2) number of sprints, and 3) percentage of total distance performed
at high speeds (>14 km/h). These outcome variables were chosen to examine both match volume (total distance) and match intensity (sprint count and % of distance above high speeds). Two matches were excluded due to technological errors (no sprints were recorded by the video tracking software), and 9 matches were excluded which went to extra-time, as the physical outputs of these matches were outliers for all analyses.

Both match and team characteristics were collected and included in the multilevel models as covariates. Match characteristics included ball possession (% of match time), score differential (e.g. -3, -1, +2, etc.), venue (Home/Away), and competition stage (Group Stage/Knockout). Team characteristics included average team age and team quality (determined by UEFA Club Coefficients).

Descriptive analyses and visualisation were completed in the open-source statistical software, R (Version 3.2.2), using the dplyr and ggplot2 packages. Multilevel models were constructed using HLM 7.01 for Windows [277]. Magnitude-based inferences for correlations and t-distributed statistics were calculated using a custom spreadsheet [278]. In accordance with previous approaches, we defined the smallest worthwhile change in each output variable as 0.2 times the between-team standard deviation, and as 0.1 for correlation coefficients. Finally, we examined the association between player absences and each outcome variable by analysing the change associated with a 2 standard deviation change in the number of players absent [279].

Results are expressed as means ± standard deviations. To examine the association between player unavailability and physical output, two approaches were used. First, across the season, between-
team differences were calculated using Pearson correlation coefficients, examining the relationship between teams’ average player unavailability and their average physical outputs. Second, within-team effects of player unavailability were examined using multilevel (mixed/random effect) linear regression – modelling the three physical outputs as outcome variables, with player availability as the explanatory variable of interest.

Multilevel models were performed with match observations (Level 1) clustered within teams (Level 2). This modelling approach was chosen for its ability to handle the data dependency created by repeated measures of the same teams, the unbalanced data formed as teams participated in different numbers of matches throughout the competition [127,280], and recent recommendations for mixed models to be used in these types of analyses [281]. Following previous recommendations, we included all level 1 covariates as random effects, including level 1 covariates in raw metrics if they held a meaningful 0 point (score differential, 0 = draw), while centering other continuous variables (ball possession) at the group mean (a.k.a. centering within cluster) – recommended when trying to examine the effect of another Level 1 variable [282]. The full model building strategy can be seen in Appendix B, and all the multilevel model outputs are included as supplementary material. Where sample size allowed, we used the robust standard errors calculated in HLM.

6.4 Results

In 2014-2015, 23 teams were included with 180 match observations. In 2015-2016, 20 teams were included, with 158 match observations. Fourteen teams participated in both UEFA seasons (total
n = 29). The breakdown of teams’ demographic information as well as injury breakdown is included in Appendix B.

Player unavailability ranged from 0 to 8 players unavailable due to injury on match day, with an average of 2.1 players unavailable in 2014/2015 (SD = 1.91) and 2.7 players in 2015/2016 (SD = 1.84). Rounding these standard deviations to the nearest integer (2), we calculated the changes in our outcome variables associated with 4 players unavailable for selection (2 x SD) [283]. The distribution of player unavailability for each team across each month of play is shown in Appendix B (Figure B.1).

The average physical outputs as well as the within and between-team variability of various physical outputs are recorded in Table 6.1. Due to the differences in physical outputs between the two seasons, remaining analyses were conducted with each season independently.
Table 6.1: Average and variability of teams’ physical outputs in the 2014-2015, 2015-2016 UCL seasons

<table>
<thead>
<tr>
<th>Physical Output Measure</th>
<th>2014/2015 Season</th>
<th>2015/2016 Season</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean*</td>
<td>Range*</td>
</tr>
<tr>
<td>Total Distance (m)</td>
<td>113 451</td>
<td>99 813 – 125 086</td>
</tr>
<tr>
<td>High Speed (14-20 km/h) Distance (m)</td>
<td>21 765</td>
<td>16 660 – 6 537</td>
</tr>
<tr>
<td>Very High Speed (&gt;20km/h) Distance (m)</td>
<td>10 285</td>
<td>6 917 – 15 108</td>
</tr>
<tr>
<td>Sprints (number of efforts &gt;20km/h)</td>
<td>417</td>
<td>266 – 562</td>
</tr>
<tr>
<td>% of Total Distance Performed at High + Very High Speeds (&gt;14km/h)</td>
<td>28.2</td>
<td>23.3 - 33.1</td>
</tr>
</tbody>
</table>

* Average and ranges calculated for all match observations.
** Within-team standard deviation is calculated as the standard deviation of the level 1 residuals.
*** Between-team standard deviation is calculated as the standard deviation of the random intercept term in a null (random intercept only) model with no independent variables.
Teams’ average player unavailability and average physical outputs were correlated for each season. Figure 6.1 displays the results from each of these analyses. Likely positive relationships existed between average player unavailability and average number of sprints per game for both seasons (Pearson’s r = 0.38 and 0.37, respectively) and a likely positive increase was seen in % of distance at high speeds with player absences in the 2014/2015 Season. All other associations were unclear.

Estimated associations of player unavailability and each of the 3 physical outcomes, controlling for match and team characteristics, are presented in Figure 6.2. Having 4 additional unavailable players unavailable for selection was associated with an estimated increase of 20.8 sprints in 2015/2016 (p = 0.040), and an 0.60% (p = 0.035) and 0.77% (p = 0.028) increase in the percentage of distance ran at high speeds (>14 km/h) in 2014/2015 and 2015/2016, respectively. There was a ‘possibly positive’ and ‘likely positive’ association between player unavailability and total distances, with estimated increases of 597 meters and 1395 meters for 4 additional unavailable players (Figure 6.2). All qualitative inferences were positive, except for an unclear association between player unavailability and sprint count in the 2014/2015 Season. A summary table of model findings can be found in Appendix B, and full HLM model outputs are available as Online Supplementary Material. HLM model assumptions were examined through residual plots and no issues warranting considerations were found.
Figure 6.1: Teams’ average player unavailability for each season and their average: 1) Distance per game (m), 2) Number of sprints (efforts >20km/h) per game, and 3) Percent of total distance covered above 14km/h per game.
Figure 6.2: Estimated effects of 4 additional unavailable players on physical output measures
Row 1 = total distance (m), row 2 = sprint count (efforts above 20km/h), and row 3 = percentage of total distance above 14km/h. Shaded area represents the smallest worthwhile change in the physical output (calculated as 0.2 times the between-team standard deviation for the physical output variable). For interpretability, effect estimates were calculated as the 90% CI for the effect of 4 additional unavailable players. Magnitude-based inferences were calculated to interpret the probabilities for this effect as negative, trivial or positive from a custom spreadsheet [284].
* Centre value is the model intercept – estimated physical output for a team playing a group stage match wherein the venue is away, teams have their average ball possession time, and the match finishes as a draw.
6.5 Discussion

Player unavailability was associated with increased match intensities in the 2014/2015 and 2015/2016 seasons. First, teams’ average player unavailability was positively correlated with their average match sprint counts in both seasons, and with the percentage of distances covered at high speeds in the 2014/2015 season (Figure 6.1). However, since correlation does not equal causation, and these were averaged over the whole season, we cannot determine the direction of this relationship. Increased sprint counts may contribute to increased injury counts, or increased injury counts may contribute to increased sprint counts. It could also be the case that both effects could be present. Some evidence to suggest that player unavailability influences match intensities is presented in the multilevel models, since they account for player unavailability entering match play, controlling for match and team characteristics. These models indicated that higher player unavailability was associated with an increased percentage of distance covered at high speeds in both seasons, and with an increased sprint count in the 2015/2016 Season (Figure 6.2).

Player unavailability was also associated with ‘possibly’ and ‘likely’ positive increases in match distances (Figure 6.2). The fact that these associations were not statistically significant likely stems from the multifaceted nature of football match play – effected by team and opponent strategy, environmental conditions (heat/altitude), match importance, match outcome, and many more factors [281,285,286]. Controlling for as many of these factors as possible in our current modelling approach, player unavailability may also be associated with increased match volumes.

These results were contrary to our original hypothesis that there would be a negative association between player unavailability and physical outputs. Although currently speculative,
these findings may extend previous research comparing physical outputs between teams. Top-ranked teams engage in less high-intensity activities than lower than lower-ranked opponents [272]. Our results indicate that this may also be true within the same team, where increased player unavailability contributes to a relatively weaker line-up and concomitant higher match intensities. The reasons for these higher intensities remain unknown, but could be due to increased efforts to regain possession, or stem from decreased technical and tactical proficiency with ball possession, causing an increased reliance on physical outputs.

These findings should also be considered pragmatically within the context of professional football [271]. For example, if 4 players are unavailable for selection, teams would be estimated to run an additional 0.60-0.77% of their total match distances at high speeds, and as a team complete 21 additional sprints. Knowing what to expect during such matches when multiple players are unavailable may help sports medicine and coaching staff to more appropriately prepare for the upcoming fixture. For example, under-exposure to high-speed running increases athletes’ injury risk when they experience rapid workload increased, so ensuring a sufficient high speed running chronic workload may be important, especially for players with less frequent match exposures [114].

We analysed the association between player unavailability and match physical outputs at the team-level, across entire matches – not individual player outputs or outputs during different match segments. Examining individual player outputs and the time course of these outputs could provide more specific findings unaccounted for in our analysis. For example, although the intensity of teams’ physical outputs may increase with fewer players available for selection, we did not determine if those changes happened for certain players – like the players selected in place of injured athletes, or for certain positions (e.g. central defenders or fullbacks).
Similarly, although there was no relationship between match distance and player unavailability at the team level, it may be that the distribution of that distance varies across players or positions when fewer players are available for selection. Given the breadth of research that compares the physical outputs of players based on positions or physical fitness levels [270,287], future investigations should analyse the effects of player availability on player/position-specific physical outputs. Finally, investigating fatigue and pacing in elite team sports examine the time course of these match outputs — it may be that player unavailability is associated with an altered physical output distributions over time [273,281].

As highlighted previously, physical match outputs are multifaceted and we did not have all relevant variables collected in our current investigation (e.g. environmental conditions, team strategy, etc.). Knowing that player unavailability leads to poorer team performance [53,134], we focused on whether player unavailability was related to teams’ physical outputs. Whether a change in relative intensity or other physical output variable is involved in mediating the injury—team performance relationship is a question for further study. Future studies may also want to include measures of technical performance associated with match outcomes — like corners, passing percentage, shots on goal, etc. [288] — or more advanced performance analytics [289], in conjunction with the variables we included. Incorporating such variables may assist in assessing the ways in which player availability effects match performances and team success.

6.6 Summary and conclusions
As teams in the 2014/2015, 2015/2016 UEFA Champions League had fewer players available for match selection, they experienced increased match intensities — based on the estimated
percentage of total match distances run above 14 km/h and the number of sprints the team performed. Increased player unavailability was also associated with increased total match volumes. Knowing that match physical outputs may be increased when multiple players are unavailable for selection may equip sports medicine and coaching staff to better prepare their players for those fixtures.
Chapter 7: Is it all for naught? What does mathematical coupling mean for acute:chronic workload ratios? (Paper 4)

7.1 Preamble

Longitudinal workload monitoring has increased in prominence within the sports medicine and sport science field. The acute:chronic workload ratio (ACWR) is often at the center of these discussions – most often as an indicator of potential injury risk. To my knowledge, at least 26 original studies of workload— injury have incorporated the ACWR since it was first published under this name in 2014 (See Supplementary Material 1. Note Malisoux et al [212] used what would be an uncoupled ACWR, but didn’t refer to it as an ACWR). Although the ACWR has consistently been associated with injury risk in a range of sports, there are a range of opinions about its utility. Editorials have criticized it [227,290–292], suggested modifying it [293,294], and supported its use [295–298].

Criticisms of the ACWR include: 1) the challenges of monitoring the ACWR in some professional team contexts – particularly football (soccer) [227], 2) using rolling averages as opposed to exponentially weighted averages [291,292], and 3) the ‘mathematical coupling’ that occurs (in the traditional 1:4 rolling average ACWR approach) because the most recent training week is included in both the acute and chronic loads [293].

These latter 2 criticisms are mathematical/statistical in nature, and fundamentally question the validity of using an ACWR in longitudinal monitoring. While Lolli and colleagues identified that mathematical coupling existed, with a clear correlation between the acute and chronic workloads in simulated data, they did not explicitly define what this correlation did to the ACWR calculation. Other than ‘uncouple’ the acute and chronic periods, they also did not
unpack how practitioners and researchers should respond to ‘mathematical coupling’. Therefore, my purpose in this paper was to answer these two questions. First – what does mathematical coupling between the acute and chronic workloads do to the ACWR calculation. Second, what does this mean for applied sport science/medicine research and practice. My aim was to bridge the mathematical discussion with the applied research and practice realm.

The rest of this chapter is a reproduction of text and figures from the following article [299]:


7.2 Introduction

Traditional calculations of the acute:chronic workload ratio (ACWR) are ‘mathematically coupled’, as the most recent week is included both the acute and chronic workload estimates (Figure 7.1). As Lolli and colleagues rightly point out, this induces a spurious correlation between the acute and chronic loads of ~ 0.50 (r=0.52 in their simulated data of 1000 athletes) [293]. They suggest that the simplest solution is to use uncoupled ACWRs (where the acute load is not part of the chronic load) instead.

Notably, at least two studies have already used uncoupled ACWR calculations, both demonstrating that rapid workload increases are associated with higher injury risks [212,241]. To this end, Lolli and colleagues’ suggestion warrants consideration – should we use ‘uncoupled’ ACWRs instead of ‘coupled’? We have 2 aims in this editorial: 1) to further comment on how mathematical coupling affects ACWR estimates, and 2) to encourage
researchers and practitioners to use a critical approach to load management, wherein ACWRs may play a part.

**Figure 7.1:** The distinction between traditional ‘coupled’ and ‘uncoupled’ estimates of the acute:chronic workload ratio. The inclusion or exclusion of the most recent week in ‘chronic load’ calculations is the key distinction.

### 7.3 Comments on mathematical coupling and ACWRs:

1) **Defining coupled and uncoupled ACWRs.** We define mathematical coupling in the same manner as Lolli et al. – where a number is represented in both the numerator and denominator of a ratio, contributing to a spurious correlation. In the case of the ACWR, both coupled and
uncoupled calculations convey whether recent workloads are increasing or decreasing compared to prior workloads (ACWR >1 = increasing, ACWR < 1 decreasing). However, including the most recent week in the chronic loading window changes the interpretation/definition of the ACWR.

<table>
<thead>
<tr>
<th>Coupled</th>
<th>Uncoupled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formula</td>
<td></td>
</tr>
<tr>
<td>$\frac{A}{0.25 \times (A + W2 + W3 + W4)}$</td>
<td>$\frac{A}{0.333 \times (W2 + W3 + W4)}$</td>
</tr>
<tr>
<td>Definition</td>
<td>The ratio between the most recent week of work with the average of the most recent 4 weeks.</td>
</tr>
<tr>
<td></td>
<td>The ratio of the most recent week of work with the average of the 3 preceding weeks.</td>
</tr>
</tbody>
</table>

*Where ‘A’ is the acute workload and the last four weeks are represented by $A$, $W2$, $W3$, and $W4$, respectively.*

Another way to think of the traditional, coupled ACWR is ‘what proportion of the total 4-week load is provided by the acute load?’ At constant loading conditions, the acute load makes up $\frac{1}{4}$ of the total 4-week load. As workloads spike, the acute workload constitutes a greater proportion (Table 7.1). The ACWR equals how many times greater this proportion is than 0.25. This number can approach, but never reach 4, since the acute load can constitute virtually all of the chronic load, but never all of it.
Table 7.1: Coupled and uncoupled (rolling averages over 4 weeks) ACWR calculations

<table>
<thead>
<tr>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
<th>Week 4 (Acute)</th>
<th>Chronic (with Acute)</th>
<th>Chronic (without Acute)</th>
<th>Traditional (Coupled) ACWR</th>
<th>Uncoupled ACWR</th>
<th>Total 4 Week Load</th>
<th>Proportion of 4 Week Loads made up by Acute</th>
<th>How many times greater is the acute proportion than 0.25?</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>10</td>
<td>10</td>
<td>0.01</td>
<td>7.5</td>
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<td>9.4</td>
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<td>0.75</td>
<td>37.5</td>
<td>0.200</td>
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<td>1.00</td>
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<td>10</td>
<td>10</td>
<td>14.5</td>
<td>11.1</td>
<td>10</td>
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<td>1.45</td>
<td>44.5</td>
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<td>1.80</td>
<td>48</td>
<td>0.375</td>
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<td>10</td>
<td>10</td>
<td>10</td>
<td>30</td>
<td>15.0</td>
<td>10</td>
<td>2.00</td>
<td>3.00</td>
<td>60</td>
<td>0.500</td>
<td>2.00</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>10</td>
<td>50</td>
<td>20.0</td>
<td>10</td>
<td>2.50</td>
<td>5.00</td>
<td>80</td>
<td>0.625</td>
<td>2.50</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>10</td>
<td>90</td>
<td>30.0</td>
<td>10</td>
<td>3.00</td>
<td>9.00</td>
<td>120</td>
<td>0.750</td>
<td>3.00</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>10</td>
<td>210</td>
<td>60.0</td>
<td>10</td>
<td>3.50</td>
<td>21.00</td>
<td>240</td>
<td>0.875</td>
<td>3.50</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>10</td>
<td>100000</td>
<td>25007.5</td>
<td>10</td>
<td>4.00</td>
<td>10000</td>
<td>100030</td>
<td>1.000</td>
<td>4.00</td>
</tr>
</tbody>
</table>
2) **The coupled correlation is expected.** The ‘spurious’ correlation of approximately \( r = 0.5 \) between acute and chronic loads is exactly what we would expect based on the ACWR calculation. Squaring this correlation \( (r^2 = 0.5^2 = 0.25) \) demonstrates that, on average, \( \sim 25\% \) of the total variation in our outcome (chronic loads) is explained by its linear relationship with the acute load. This aligns with the fact that the acute load makes up one fourth of the chronic load calculation. Extending this logic, if a coupled ACWR was calculated over a 5-week window, the correlation would approximate \( r=0.45 \) \( (r^2 = 0.20) \).

3) **One can convert between coupled and uncoupled ACWRs.** Since both ratios discussed by Lolli et al. are calculated from the same four training weeks, one can convert between the two via the following formula (Appendix C).

\[
\text{*Uncoupled ACWR} = \frac{3 \times 'Coupled'ACWR}{(4-'Coupled'ACWR)}
\]

4) **Effects of mathematical coupling on ACWRs.** Lolli et al. note that a ‘coupled’ ACWR of 1.45 equals an ‘uncoupled’ ACWR of 1.71. Thus, mathematical coupling ‘suppresses’ traditional ACWRs compared with uncoupled ACWRs. The degree to which the ratio is suppressed increases as the ACWR increases. The ratios are equal at 1.0, vary slightly at first (1.5 vs. 1.8), and progressively diverge (2.0 vs. 3.0, 3.0 vs. 9.0). The coupled ACWR never exceeds 4, regardless of how high the uncoupled ACWR gets (Figure 7.2) so a coupled ACWR should not be considered a continuous measure. The way that these scaling properties affect research estimates and the appropriateness of including them in different analyses (e.g. regression modelling) deserves further consideration.
Figure 7.2: The association between coupled and uncoupled estimates of the acute:chronic workload ratio with increasing workload progression.

While the coupled ACWR asymptotes at 4, uncoupled estimates continue to increase in a linear fashion.
7.4 What now? A call for critical, thoughtful monitoring

Mathematical coupling may support uncoupled ACWRs in the future. However, rather than suggest one particular approach, we propose that researchers and practitioners should focus on a thoughtful, critical approach to load monitoring, which may, or may not include ACWRs. We highlight 3 practical applications below:

1) Don’t use any ACWR in isolation

Athlete monitoring is a multifaceted process with information gleaned from external and internal loads, athlete wellness, readiness, and other information [100]. One factor which may be of interest to practitioners and researchers is the rate of workload change. An ACWR (coupled or uncoupled, rolling average or exponentially weighted) provides a single measure that indicates how recent loads are changing relative to previous loads. Whatever method (coupled or uncoupled) is chosen, it must not be interpreted in isolation. Instead, it must be interpreted in conjunction with other information – e.g. training modalities, absolute workload changes, how the athlete is tolerating training, the athlete’s chronic load, and the athlete’s internal risk profile (e.g. age, aerobic fitness level, strength, etc.) [5, 6].

2) Understand the implications of how any given ACWR was calculated.

Different acute and chronic time windows [236], exponentially weighted moving averages [294], and week-to-week changes [300] have been proposed in the literature to monitor training progression. Each of these calculations will result in different numbers given the same workload sequence. Notable ‘thresholds’ will also differ depending on how an ACWR is calculated. The proverbial ‘sweet spot’ of 0.8 to 1.3 for the ACWR has been largely based on coupled, rolling average estimates. If uncoupled (1-week:4-week) ACWR estimates were used,
these same thresholds would correspond to different ratios (0.75 to 1.45). It is up to the user to critically select which variation, if any, they will use, and how they will interpret the values.

3) **Explicitly detail how the ACWRs were calculated.**
Finally, if researchers choose to use ACWRs, they must explicitly detail how they were calculated, facilitating interpretation and replication. These details should include whether calculations were rolling averages or exponentially weighted [294], the length of the acute:chronic windows, and whether acute and chronic workloads were coupled or not.

Ultimately, developing robust, high-performing athletes depends on building high chronic workloads, but increasing these workloads too quickly will likely increase athletes’ risks of injury [74,241]. In the context of a thoughtful, critical athlete monitoring approach, ACWRs may help by estimating workload progression – however they are quantified.
Chapter 8: A training load--injury paradox: Is greater pre-season participation associated with lower in-season injury risk in elite rugby league players? (Paper 6)

8.1 Preamble

In the chapters 3-7, I outlined how workloads conceptually relate to injury, called researchers to thoughtfully consider what workload metrics are used, and reviewed the statistical approaches that best analyse intensive longitudinal data. In this final data chapter, I investigated whether rugby league players who participated in a greater number of pre-season sessions were better able to resist in-season injury. As detailed in Chapter 5, this research question deals with between- and within-athlete differences. The primary research question – whether completing more pre-season sessions is associated with a greater ability to stay healthy during the season – differentiated between athletes, as each athlete completes a distinct number of pre-season sessions. I also needed to control for the within-athlete exposures to workloads throughout the competitive season. Multilevel modeling allowed me to cluster weeks (level 1) within each athlete (level 2), so that weekly workload variables could be included in the model, along with athlete characteristics.

The rest of this chapter is a reproduction of text and figures from the following article [173]:

8.2 Introduction

Athletic injuries are common in team sports [132,133], compromising team success [52,53,134] and posing a significant financial burden to organisations [54]. High training loads and substantial spikes (rapid increases) in training volume have been associated with increased injury rates [52,113,115]. Both external (work completed) [136,138,160] and internal (physiological response such as perceived exertion or heart rate) [157,159,210,301] training load measures have been used to identify the association between workloads and injury risk.

Traditionally, workload-injury investigations focused on absolute workloads and injury [142,157], and higher workloads were associated with greater rates of injuries [142]. However, high training loads are necessary for beneficial physiological adaptation such as increased aerobic capacity, strength, and repeat-sprint ability, along with optimal body composition [163,164], many of which are associated with decreased injury risks [160,166,167].

Recently, load-injury investigations have highlighted that the relationship between acute (1 week) and chronic (rolling 4-week total averaged to one week) workloads, termed the acute:chronic workload ratio, may better predict injury risk than total workloads [52,113,136]. Moreover, Hulin and colleagues demonstrated that as long as players' acute:chronic workload ratios were kept within a moderate level (0.85-1.35), high chronic workloads may reduce injury risk in rugby league players – the training load-injury paradox [112,113].
Pre-season training provides several physical benefits for sporting teams. It allows players to reach high chronic workloads [113], as well as to develop the physical capacities associated with reduced injury risks [160,166,167]. Indeed, pre-season periods often include higher training loads than in-season periods [52,159]. Theoretically, players who have a more ‘successful’ pre-season may be more resilient to injury when faced with the demands of the competitive season.

To our knowledge, no study has investigated whether pre-season training provides a foundation which decreases in-season injury risk in elite team sport athletes. Therefore, we investigated whether elite rugby league players who participated in a greater number of pre-season sessions were less likely to miss games due to injury throughout the competitive season, while accounting for their external training loads during the competitive season.

8.3 Methods

8.3.1 Study design

We prospectively followed thirty rugby league players (mean ± SD age, 25 ± 3 years) from one elite rugby league club throughout their 17-week pre-season period and 26-round competitive season. All participants provided informed written consent and received a clear explanation of the study. All experimental procedures were approved by the Institutional Review Board for Human Investigation at Australian Catholic University.

8.3.2 Measures

We collected both time-varying and time-invariant variables. Time varying variables were summarised weekly and included injury status and daily training load variables. Time invariant
variables included the number of pre-season sessions completed, player position, and age at the start of the pre-season. The competitive season was divided into 3 time periods for descriptive purposes, around the representative (i.e. ‘State of Origin’ interstate series) period: ‘pre-origin’ (Weeks 1-8), ‘origin’ (weeks 9-17), and ‘post-origin’ (weeks 18-26).

8.3.2.1 Injury status

The team’s medical staff (including physician and physiotherapist) diagnosed injuries, while the team physiotherapist updated and maintained the injury reports. For the purpose of this investigation, an injury was defined as any injury that resulted in a loss of match-time – “match time loss only” [18].

8.3.2.2 Pre-season attendance:

For each training day throughout the pre-season and competitive season, players’ participation in training was recorded as ‘full’, ‘modified’, ‘rehab’, or ‘away’. Players’ individual pre-season participation levels were quantified as the number of “full” pre-season sessions they completed.

8.3.2.3 Quantifying in-season training loads

External workloads were obtained using global positioning system (GPS) devices (GPSports, SPI-HPU 5 Hz (interpolated 15 Hz), Canberra, Australia). Load variables collected included total distance, high speed (>5 m/s) distance covered, and the percentage of total distance completed at high-speeds. Our analysis included all field training sessions and National Rugby League matches throughout the 2015 season.
8.3.3 Data collection and analysis procedures

Data were categorised into weekly blocks from Monday-Sunday throughout the 26-week season. If GPS data were missing for players who were recorded as attending the ‘full’ training session, load data was estimated by calculating the average workload for players of the same position who participated in the full session. Since models were fitted to determine the likelihood of sustaining a time-loss injury in a given week or subsequent week, players’ data for a given week were excluded if they were already injured, suspended, or released from the team.

8.3.4 Statistical analysis

All data were analysed in the open-source statistical software, R (Version 3.2.2). Separate univariate random effect logistic regression models were fitted for each time-varying and time-invariant variable, with the likelihood of sustaining a time loss injury as the outcome variable, and random-intercepts for each player. These models were used to determine which variables were associated with an increased or decreased risk for injury throughout the season, not controlling for other covariates. Random effect models were chosen for their ability to handle unbalanced data with varying number of follow-up observations, their capacity to generate individual specific-predictions, and for their recommended use in analysing repeated-measures designs with correlated data.

In fitting the regression models, all training load variables were standardised, due to the different scales of the measures and subsequent failure of the models to converge in the statistical software with unadjusted predictor variables. Odds ratios were calculated to determine the effect size associated with a one standard deviation increase in training load variables. For pre-season
participation, odds ratios were calculated to examine the effect sizes associated with an increase of 10 “full” pre-season sessions. Statistical significance was set at p<0.05 for all analyses, and odds ratios were calculated as an effect size for all models.

Two separate multilevel logistic regression models were fit to determine the effect of pre-season participation on injury likelihood, controlling for training loads. One model was fit to determine the likelihood of injury in the current week. A second model was fit to determine the likelihood of injury in the subsequent week. The final models were first fitted by including variables shown to be significant predictors from univariate models. From here, all other training load variables, as well as time invariant covariates (age, position, season period), and interaction terms were added to the model to optimise model fit. Model fit was assessed by minimising the values of model diagnostics criteria (AIC/BIC) and the standard deviation of the random intercepts. Variables that did not improve the model fit were excluded from the final models.

8.4 Results

8.4.1 Injuries

A total of 40 injuries were sustained during the competitive season (29.0/1000 h). These led to 241 total matches missed. There were no significant differences in injury likelihood when comparing positions (p = 0.73) or season period (p = 0.46).
8.4.2 Pre-season participation

During the pre-season period (November 3, 2014 to February 27, 2015), the team had 87 pre-season training sessions. Players completed an average of $64 \pm 19$ ‘full’ pre-season sessions (range 12 - 86).

8.4.3 Pre-season loads and injury risk

Over the course of the season, there was a significant correlation between the number of full pre-season training sessions that players completed and the number of full in-season sessions completed ($r = 0.59$, $p<0.001$). Further, there was a significant association ($r = -0.40$, $p < 0.05$) between the number of pre-season sessions players completed and the percentage of games they missed due to injury (Figure 8.1). Without adjusting for training loads, greater pre-season participation was associated with a decreased likelihood of injury throughout the competitive season during the current (OR = 0.82, 95% CI = 0.69-0.97) and subsequent week (OR = 0.80, 95% CI = 0.68-0.94).
Figure 8.1: The association of pre-season participation with games missed due to injury. $r = -0.40$, $p < 0.05$. Percentage of games missed due to injury was calculated by dividing the number of games missed due to injury by the number of games that players were eligible to play. Games were excluded from calculation if players were ineligible due to suspension or being traded during the season and therefore not eligible to play.
8.4.4 Training loads and injury risk

Training load measures over the course of the competitive season are summarised in Table 8.1. The average distance as well as injury incidence for each week of the competitive season are displayed in Figure 8.2.

Table 8.1. Descriptive statistics for players’ average workload and injuries over the duration of the study.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Phase of season</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-Origin</td>
<td>Origin</td>
</tr>
<tr>
<td>Injuries (n)</td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>Distance (m)</td>
<td>9574 (4099)</td>
<td>10815 (5311)</td>
</tr>
<tr>
<td>High Speed Distance (m)</td>
<td>677 (462)</td>
<td>668 (467)</td>
</tr>
<tr>
<td>High Speed Distance/Total Distance (%)</td>
<td>7.2 (4.8)</td>
<td>6.1 (2.4)</td>
</tr>
</tbody>
</table>

Data are mean (SD) for load measures and count for injuries. Phase of season divides the season into three periods around the ‘State of Origin’ interstate series: ‘Pre-origin’ (Weeks 1-8), ‘origin’ (weeks 9-17), and ‘post-origin’ (weeks 18-26).
Figure 8.2: Weekly training loads and injury incidence
Higher acute (1 week) distances were associated with lower injury likelihoods in the current week (OR = 0.64, 95% CI = 0.46-0.90), but not in the subsequent week. Conversely, a greater percentage of total distance completed at high speeds was associated with an increased likelihood of injury both in the current (OR = 1.34, 95% CI = 1.03-1.73) and subsequent week (OR = 1.07, 95% CI = 1.06-1.08). Absolute high-speed running distance was not associated with injury likelihood in either the current or subsequent week.

Chronic workloads were not significantly associated with injury risk in either the current or subsequent weeks. Similarly, acute:chronic workload ratios for all training load variables were not associated with significant changes in injury likelihood. Table 8.2 summarises all models of single training load variables and their associated effects on injury risk.
Table 8.2 Association of training load variables with injury likelihood in the current and subsequent week.

<table>
<thead>
<tr>
<th>Load calculation</th>
<th>1 SD</th>
<th>Effect of 1 SD increase on current week injury likelihood OR (95% CI)</th>
<th>Effect of 1 SD increase on subsequent week injury likelihood OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute (1 Week Loads)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance</td>
<td>5205 m</td>
<td>0.64* (0.46-0.90)</td>
<td>0.86 (0.61-1.22)</td>
</tr>
<tr>
<td>High Speed Distance</td>
<td>461 m</td>
<td>0.83 (0.58-1.20)</td>
<td>0.83 (0.57-1.19)</td>
</tr>
<tr>
<td>High Speed Distance/Total Distance (%)</td>
<td>3.7%</td>
<td>1.34* (1.03-1.73)</td>
<td>1.07* (1.06-1.08)</td>
</tr>
<tr>
<td><strong>Chronic (Rolling 4 Week Average Loads)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance</td>
<td>3389 m</td>
<td>0.80 (0.58-1.11)</td>
<td>0.82 (0.57-1.18)</td>
</tr>
<tr>
<td>High Speed Distance</td>
<td>346 m</td>
<td>0.86 (0.60-1.22)</td>
<td>0.99 (0.70-1.40)</td>
</tr>
<tr>
<td>High Speed Distance/Total Distance (%)</td>
<td>3.1%</td>
<td>1.13 (0.83-1.54)</td>
<td>1.22 (0.90-1.66)</td>
</tr>
<tr>
<td><strong>Acute:Chronic Workload Ratios</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance</td>
<td>0.72</td>
<td>0.72 (0.48-1.07)</td>
<td>0.89 (0.63-1.27)</td>
</tr>
<tr>
<td>High Speed Distance</td>
<td>0.90</td>
<td>0.91 (0.64-1.31)</td>
<td>0.88 (0.61-1.27)</td>
</tr>
<tr>
<td>High Speed Distance/Total Distance (%)</td>
<td>0.42</td>
<td>1.64 (0.83-3.24)</td>
<td>1.13 (0.53-2.42)</td>
</tr>
</tbody>
</table>

* p < 0.05, SD = Standard Deviation
8.4.5 Full injury prediction models

Two multivariate injury prediction models quantified the effect of pre-season participation on injury risk, controlling for training loads (Table 8.3). Training load variables included in the final models were those that were found to have a significant association with injury risk in univariate models, specifically 1-week total distance and the 1-week proportion of distance performed at high speeds. The fit of these final models was not improved with the addition of any other variable, nor with the addition of a random slope to the model, so none were included.

Table 8.3. Effect of pre-season participation on injury likelihood in current and subsequent week, controlling for training load variables.

<table>
<thead>
<tr>
<th>Model</th>
<th>Variable</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1:</td>
<td>10 Pre-Season Sessions</td>
<td>0.85 (0.70-1.02)</td>
</tr>
<tr>
<td>Injury Likelihood in Current Week</td>
<td>High Speed Distance/Total Distance</td>
<td>1.27 (0.99-1.63)</td>
</tr>
<tr>
<td>Model 2:</td>
<td>10 Pre-Season Sessions</td>
<td>0.83* (0.70-0.99)</td>
</tr>
<tr>
<td>Injury Likelihood in Subsequent Week</td>
<td>High Speed Distance/Total Distance</td>
<td>1.07 (0.79-1.45)</td>
</tr>
<tr>
<td></td>
<td>Acute Distance</td>
<td>0.82 (0.55-1.21)</td>
</tr>
</tbody>
</table>

* p<0.05

Model 1 predicts the likelihood of injury in the current week. Controlling for training load, increased pre-season participation was still associated with a reduced odds of injury, though this was no longer statistically significant (OR = 0.85, 95% CI = 0.70-1.02). Similarly, a greater percentage of distance run at high speeds appeared to be associated with an increased injury risk, but the effect was no longer significant when controlling for pre-season participation and acute distance (OR = 1.27, 95% CI = 0.99-1.63). Finally, as with univariate models, greater acute distance was associated with a significantly reduced likelihood of injury (OR = 0.56, 95% CI = 0.36-0.87).
Model 2 predicts the likelihood of injury in the subsequent week with a given pre-season participation, acute distance, and acute percentage of distance ran at high speeds. In this model, when controlling for distance and percentage of distance at high speed, increased pre-season participation was associated with a reduced likelihood of injury (OR = 0.83, 95% CI = 0.70-0.99). Within this model, neither acute distance nor percentage of distance ran at high speeds were significantly associated with injury risk in the subsequent week (Figure 8.3).
Figure 8.3: Predicted injury probabilities based on high speed running percentage and pre-season participation.

Injury probabilities were predicted from model 2, predicting the probability that a player would sustain a match time-loss injury in a subsequent week, controlling for the total distance and percentage of total distance ran at high speeds in the current week, as well as pre-season participation. Pre-season participation has been divided into three equal tertiles, such that “Low” equals < 59 sessions (n=10), “Moderate” equals 59 – 75 sessions (n=10), and “High” equals >75 sessions (n=10).
8.5 Discussion

In this sample of rugby league players, greater pre-season participation was associated with a decreased injury risk during the competitive season. Players who participated in a greater number of full pre-season sessions had a reduced likelihood of injury throughout the competitive season, completed more in-season training sessions and missed fewer games due to injury. This reduced injury likelihood in the subsequent week was maintained even when controlling for training load variables (acute distance and acute percentage of total distance completed at high speeds).

8.5.1 Training loads and injury likelihood

In contrast to recent studies [52,113], neither acute:chronic workload ratios nor chronic workloads in isolation were significantly associated with injury. It may be that the current sample size was too small to detect these effects. However, two separate measures of acute (1 week) training loads, 1) distance covered, and 2) percentage of total distance covered at high speeds, were significantly associated with changes in injury likelihood.

Higher 1-week total distances were associated with a reduction in injury likelihood in the current week, but not in the subsequent week. Similarly, previous data in rugby league players showed that greater distances completed at lower intensities were associated with a reduction in injury risk [160]. However, it should be noted that the reduced likelihood of injury associated with higher distances in the current week may be partly attributable to players sustaining an injury earlier in the week. In this case, it may be that increased distances are not preventing injury, but are accumulated by players who are healthy.
In contrast to total distance, the percentage of total weekly distance performed at high speeds was associated with an increased risk of injury in the current and subsequent week. This increased injury risk with greater high-speed running loads has been previously seen in team sport athletes [138,160]. Notably, when controlling for players’ pre-season attendance, the percentage of running performed at high-speeds was no longer significantly associated with injury. This may indicate that players who had accumulated more of the benefits of a successful pre-season were better able to tolerate the stress of the competitive season. Similarly, it has previously been shown that team sport athletes who perform greater than 18 weeks of training before sustaining initial injuries were at a reduced risk of sustaining a subsequent injury [302]. Collectively, the present and previous [23] results demonstrate the protective effect of pre-season training in team sport athletes.

8.5.2 Pre-season participation and in-season injury – providing protection or revealing underlying differences [i.e. identifying the robust players]?

We speculate as to potential mechanisms responsible for the associated reduction in injury likelihood with increased pre-season participation. From a physical standpoint, pre-season participation may be protective by allowing players to accumulate high chronic workloads [113], and develop greater strength and aerobic capacity [302]. Further, players who participate in a greater proportion of pre-season training sessions may also be better prepared mentally and tactically within the team environment. On the other hand, increased pre-season participation may merely identify players who are inherently more robust to injury, and therefore more likely to handle both the pre-season training loads as well as the rigors of the competitive season. Thus, the association between pre-season training and in-season injury risk may stem from both protective and revelatory effects of the pre-season.
8.5.3 **Preparation through pre-season participation – practical implications**

Our findings do not necessarily indicate a need for high training loads during the pre-season. Rather, pre-season training should be conducted so that players are able to participate in the highest proportion of team sessions possible. This is especially pertinent given that the majority of training-related rugby league injuries occur during the pre-season periods [158]. Even though 24% of a rugby union team’s annual training occurred during the pre-season period, 34% of training related injuries occur during this time [303]. High injury incidence during the pre-season period may be partly attributable to training loads, which are generally higher in pre-season periods compared with in-season periods [52,302]. Moreover, a reduction in pre-season training loads significantly reduced injury incidence in rugby league players [158]. From our experience (TG), those who manage team training loads should aim to design pre-season training periods which induce positive physiological adaptations while minimising injury risk and maximising player availability. Accurate season-by-season records of injury and training loads will help teams find their ‘sweet spot’.

8.5.4 **Potential limitations**

The present study included a sample size (30 players), and total injury occurrences (40), which limits the number of variables that could be included in the injury prediction models and reduces the sensitivity of the models. Due to the limited availability of GPS devices, the number of players who could be monitored was restricted to 30, as opposed to all members of the rugby league club. Although more injuries would have been captured with a broader definition of injury, “match time-loss only” is accepted as an accurate and reliable definition used in team sport contexts [18].
Although we used GPS-derived total and high speed running distances, the inclusion of other external load variables (e.g. accelerations, decelerations, collisions) would likely add value to any investigation of the relationship between pre-season training load and in-season injury risk in rugby league players [96,101]. However, as discussed in recent load injury investigations [113,168], the ability of GPSports technology to accurately measure these variables is limited. Further, internal load measures (e.g. session-RPE or heart-rate) may also be useful to investigate pre-season and in-season training loads. Finally, while we quantified pre-season participation in sessions, future investigations may quantify pre-season training loads to further distinguish characteristics of pre-season training that facilitate the development of athlete resilience.

8.5.5 Summary and conclusions

In this first study to investigate the association of pre-season training participation and injury likelihood during the competitive season, players who completed a greater number of pre-season sessions were less likely to be injured during the competitive season, even when controlling for their external training loads. Total distance covered was associated with a decreased likelihood of injury in the current week, while players who completed a greater percentage of their total distance at high-speeds were at increased risk of injury in the current and subsequent week.
Chapter 9: Conclusion

9.1 Integrating my findings into the field, and contributions of my dissertation

Workload and injury research has proliferated in the last decade. From the time I commenced this dissertation in 2015 until today, the field has developed substantially. As just one example, the acute:chronic workload ratio was first introduced by this name in 2014. Just four years later, approximately 30 original cohort studies have investigated workloads and injury using this derived measure. Discussions have progressed to nuances of how to best calculate workload progression [293,294], and differentiating its association with injury from its predictive power [304,305]. Therefore, integrating this dissertation within the current research landscape is analogous to parking on a rapidly moving freeway.

9.1.1 Conceptual contribution

My extension of the sports injury aetiology literature – the workload—injury aetiology model [131] (Chapter 3, Figure 3.6) – provided the first athletic injury aetiology model that explicitly incorporated training and competition workloads. It provided researchers and applied sports medicine/science practitioners with a framework to understand how workloads may contribute to injury occurrence, specifically through 1) exposure to potential inciting events, 2) positive physiological adaptations which improve athletes’ individual risk profiles, and 3) negative fatiguing effects on an athletes’ individual risk profiles.

Although this workload—injury aetiology model was the first to incorporate workloads explicitly into athletic injury development, at least three other frameworks have since extended the aetiological literature [180,306,307]. These extensions included 1) differentiating physiological
Differentiating physiological and biomechanical adaptation timelines extends the overall workload—injury aetiology model. Vanreentghem and colleagues introduced a similar ‘fitness’ and ‘fatigue’ athlete monitoring framework aimed at team sports, but distinguished ‘physiological’ from ‘biomechanical’ adaptations [306]. The former refers to adaptation from a central, cardiovascular perspective (e.g. lactate threshold, oxygen consumption) and the latter to the musculoskeletal system (e.g. bone, tendon, joint). Thus, a given workload progression may allow sufficient adaptation from a cardiovascular perspective, but exceed the ability of the biomechanical system to adapt following training stimuli. Where the workload—injury aetiology model focused on injury development as the outcome, Vanreentghem and colleagues quantified external loads and internal loads with a spectrum of underloading to overloading as their outcome. Notably, both these models conceptualised training and adaptation over time and across multiple sessions, with less emphasis on intra-session injury aetiology.

Intrasession injury aetiology among runners was the central focus of Bertelsen and colleagues’ running injury framework [307]. Within their framework, injury occurs when the load on a specific structure exceeds the capacity of that structure. The rest of the model details the factors contributing to the structure-specific loading (divided into load magnitude and load distribution per stride) and the structure-specific capacity. Like my model, they considered internal risk factors (e.g. previous injury, sleep, age, etc.) and external risk factors (e.g. shoes, terrain). Risk factors can alter structure-specific capacity (e.g. a previous injury may have worsened tissue integrity) or
structure-specific loading – through the load magnitude per stride (e.g. body weight) or through the load distribution per stride (e.g. terrain, anthropometrics). On one hand, the framework’s increased granularity provides more clarity on how risk factors contribute to injury risk at a tissue level. On the other hand, there is no recursive element, so the structure-specific adaptation across sessions is not incorporated into the model.

Complex systems thinking presented a third development in the aetiological literature. Extending other’s work [225], Brazilian PT/PhD Natalia Bittencourt and colleagues introduced a complex systems approach to athletic injury aetiology that fundamentally shifted the discussion away from internal and external risk factors to a ‘web of determinants’ made up of these factors (Figure 5.1) [180]. Within this framework, athletic injury is an ‘emergent pattern’, which can occur through multiple ‘risk profiles’. Risk profiles are made up of ‘regularities’ where certain risk factors interact and contribute jointly to injury risk. Complex systems analyses are conceptually appealing yet challenging to analyse, and statistical approaches (e.g. agent-based modeling) require datasets that are larger than those currently collected in sporting settings. To address this challenge, simulated data can be used to explore injury aetiology using complex systems analyses, as demonstrated in the first of these studies [238]

Extending the workload— injury aetiology model, mediation and moderation are causal models that I suggested may help explain the workload— injury association (Chapter 4). Within the last three years, several original investigations have supported this notion [182,241,245,308]. Aerobic fitness moderated the workload— injury association in both elite Gaelic football and professional soccer [182,309], with fitter players more robust to workload spikes measured with an ACWR.
Heart rate variability also moderated workload spike tolerance in a small sample of Crossfit athletes over a 16-week period [245]. Finally, scapular dyskinesis moderated the relationship between throwing workload and shoulder injury in 679 elite handball players [241].

Using the handball study, Nielsen and colleagues extended the moderation discussion by drawing from their running aetiology framework [307]. They presented a directed acyclic graph (DAG) to demonstrate the potential causal pathway to shoulder injury [220]. In this DAG they again differentiated how workloads impact injury through structure-specific loads exceeding that structure’s capacity. In this case, scapular dyskinesis modifies the distribution of structure-specific loads onto certain muscles and contributes to injury at a workload where an athlete with normal scapular kinematics would be distributing the load more evenly and thus be protected against injury.

9.1.2 Methodological contributions

Although several systematic reviews and an international consensus statement on workloads and injury have been published, none had critically appraised the statistical approaches that authors employed. I reviewed and then applied US Professor Linda Collins’ threefold framework (1 – conceptual model, 2 – temporal design/data collection frequency, and 3 – statistical model) [126] as a lens to assess these statistical approaches (Chapter 5). The inclusion criteria meant that the theoretical aetiology models which described daily fluctuating injury risk were aligned with the daily data collection in all of the included prospective cohort studies, leaving the statistical model to consider.
Very few sport workload studies had utilised statistical approaches that can adequately handle the challenges imposed by intensive longitudinal data. One of these challenges – the dependency created by repeated measures – increases the risk of false-positive findings when analysing workload—-injury data [259].

Several statistical approaches (e.g. correlation, t-tests, ANOVA) used in the past also cannot answer research questions begged by existing aetiological frameworks – such as whether risk factors moderate the workload—-injury association. I recommended multilevel/mixed modeling and time-to-event modeling as well-suited approaches that have been underutilised in the field. Given the superiority of multilevel modeling over generalised estimating equations, I provided two examples of mixed/multilevel modelling studies using longitudinal data in Chapter 6 and Chapter 8. Since ILD are not limited to sports medicine, methodologically reviewing the strengths and weaknesses of analytical approaches may inform future research across disciplines.

Methodological and statistical considerations for longitudinal workload data continue to move to the forefront of research – this is a dynamic field. Therefore, other statistical approaches and methodological considerations that I did not discuss in detail within this thesis are likely to contribute to the field in the future. Simulation research has recently used complex systems approaches (specifically, agent-based modeling) to explore the workload—-injury association in distance runners [238], and to demonstrate how non-linear approaches such as regression splines are more appropriate than discretization for modeling the ACWR [259].
9.1.3 Applied contributions

Given the advances in research methodology in athlete monitoring, there is an opportunity to apply these considerations into applied practice (e.g. working with teams). In Chapter 7, I responded to published criticism of the ACWR [293] by describing how mathematical coupling influences the derived calculation of the ACWR, and what this effect means for applied sport scientists and sports medicine researchers. I concluded that researchers must explicitly report what measures they chose and think critically about the implications. The same workload progression will result in different ACWR values depending on how it is calculated, and the variable itself may change from linear, unbounded variable (uncoupled) to an upper-bounded non-linear variable (coupled). Therefore, the effect of any specific ACWR value will greatly depend on how it is derived (coupled vs. uncoupled, rolling averages vs. exponentially weighted). Similarly, applied practitioners often use decision support systems or ‘traffic light systems’ to alert them to ‘notable’ findings [226,310]. Since given workload progressions give different ACWR values, practitioners must critically consider what each calculation method means for various ‘threshold’ calculations and use this to inform what findings they deem ‘notable’.

To my knowledge, Chapter 8 was the first study to investigate whether a successful pre-season – judged by the number of sessions completed – helped to prepare athletes for regular season demands. It was also the first prospective cohort study in the workload—innjury field to use a mixed/multilevel model. By using a mixed modeling approach, as recommended in Chapter 5, I clustered weekly workload observations (Level 1) within each athlete (Level 2). By structuring the data in this multilevel format, athlete characteristics (e.g. age, # of preseason sessions) could be modeled together with workload, with injury as the outcome.
Looking through the lens of the framework in Chapter 5, this modeling approach allowed me to address two major themes from theoretical aetiology models (like the workload—injury aetiology). I considered a multifactorial injury aetiology and included both within-athlete (workloads) and between-athlete (pre-season sessions) variables. This modeling approach also addressed three longitudinal data analysis challenges – 1) it controlled for the repeated measures dependency by clustering observations (weeks) within athletes, 2) it adequately handling unbalanced data as athletes had different numbers of observations, and 3) it included time-varying (weekly workloads) and time-invariant variables (e.g. age and number of pre-season sessions) in the model.

Mixed modeling enabled me to show that athletes who completed more preseason sessions were able to complete a greater proportion of in-season sessions and were better prepared for in-season demands. These preseason benefits have since been documented in Australian football [201,311] and in American football [312]. Collectively, these studies suggest that players who participate in a greater proportion of a team’s planned pre-season may be more robust during the regular season. This could relate to a ‘survival of the fittest’ phenomenon, or to athletes successfully building up a sufficient chronic workload and adaptation during the preseason period. As these published studies examined single teams, it remains to be seen whether this extends to between-team comparisons – namely, whether teams with more pre-season sessions have lower injury rates than those with fewer pre-season sessions.
9.2 Dissertation strengths

The first strength of this dissertation is its methodological focus. Workloads are an important causal factor related to injury risk. However, understanding how workloads relate to injury and the strength of this relationship depends on the strength of the analyses employed in the original manuscripts. Although there have been many original investigations and now a reasonable number of systematic reviews in our field, methodological appraisals of these studies had been entirely lacking. The methodological emphasis came at a time where increasing amounts of longitudinal data were being collected. My review of these original studies through an intensive longitudinal data analysis framework should inform future analyses in sports medicine and has the potential to provide insights for other health researchers who benefit from intensive longitudinal data. As one example, Prof. Collins’ lab recently used multilevel models to better understand how stress affected drinking behaviour in college students, based on daily diary data [249].

Using multilevel modeling to account for the repeated-measures nature of the data included in the original prospective cohort studies represented a step forward in the modelling procedures in many workload—injury studies. Subsequent simulations have demonstrated that failing to account for the repeated-measures nature of the data in longitudinal workload monitoring increases the false-positive rate [259].

9.3 Dissertation limitations - what I would do differently

My studies described in Chapters 6 and 8 were among the first to incorporate multilevel modelling with workload and injury data, and among relatively few that controlled for the repeated measures nature of longitudinal data. Reporting and interpreting the random effects from mixed models is
another benefit of mixed models. In hindsight, I would have incorporated these to a greater effect in both mixed modeling chapters. Further, more advanced statistical techniques have continued to be developed, particularly time-to-event models that account for competing risk and multi-state transitions. Moving forward I would utilise these methods in prospective cohort studies, if there were enough events per variable to power these models.

I modeled ACWR as a continuous variable in my pre-season analysis (Chapter 8), instead of through non-linear approaches. This may contribute to why ACWR was not associated with injury risk. Most of the studies that have included ACWR to injury risk, explored potential non-linear associations through discretization/categorisation of the ACWR [259] and suggest a non-linear (u-shaped) association between the ACWR and injury risk. Carey and colleagues simulated workload data and demonstrated how regression splines or non-linear modelling may be more appropriate than discretization [259]. I would revisit our modeling to incorporate ACWR in this way.

9.4 Future research directions

Global (internal) workload measures (e.g. session RPE) and ‘standard’ workload progression measures (e.g. 1- to 4-week rolling average ACWRs) are unlikely to be ideal for all sporting environments and injury types. Refining which load measures and derived workload variables most closely relate to injury risk will continue. Sport-specific load measures may include jump count for sports such as volleyball [313,314], or throw counts for sports such as handball or baseball [241,315]. Researchers may also differentiate whether running (e.g. high-speed running distance) or change of direction (e.g. accelerations, decelerations) metrics are most relevant in team field/court sports [317].
Ideal derived variables may also vary across sporting contexts or injury types. Standard 1-week to 4-week rolling averages may be less sensitive than ratios with different acute/chronic time-windows or exponentially weighted approaches [236,246,317]. Since the timeline for adaptation differs based on the specific system (e.g. physiological/biomechanical), different acute:chronic ratios relate more closely to specific injury types based on tissue type. Finally, given the criticisms of rolling average ACWR approaches [290,291,293], other derived variables may be developed or refined that more accurately capture workload progression.

By using appropriate multivariable statistical approaches, future workload—disease investigations may better understand athletic injury aetiology by asking theory-driven questions. Which risk factors moderate the workload—disease association? Do multiple risk factors interact with each other or with workload progression to alter injury risk?

Injury prediction remains the ‘holy grail’ of athletic injury research [319]. Athlete screening with single physical tests cannot be used to accurately predict injury at an individual athlete basis [183]. Existing analyses using workload data have also been relatively poor predictors to date [304,305,319]. However, these poor predictive capabilities may largely be expected because many used only one derived workload variable (e.g. ACWR) to predict injuries. Since injury risk is multifactorial, as datasets grow to include multiple risk factors along with workload data, future investigations should undoubtedly deploy more complex predictive models, the performance of which remain to be seen.
Injury aetiology – a cornerstone of my dissertation – is the second step of van Mechelen’s sequence of prevention model [5]. Although there is an association between workloads and injury, injury prevention can only occur when this knowledge informs preventative interventions. An important next step will be for researchers to (i) develop a preventive intervention that includes workload management (e.g. controlling the number of times athletes are exposed to rapid workload increases while building high chronic workloads to maximise positive adaptation) and (ii) test its efficacy can be evaluated in a research context (e.g. randomised control trial). I understand that such a trial has been registered in Norway.

As the TRIPP model [63] and recent views on the van Mechelen model [321] remind us, the implementation context of interventions is key. What is needed for successful intervention? When considering workload management to prevent injuries, it will be critical to understand which professionals dictate athlete loads in specific sporting environments (e.g. usually a head coach – not a health professional or a sports scientist). This has led to claims that the team coach/manager is the biggest injury risk a player faces. Future research will likely address whether their coaches’ behaviours can be modified. As I was finalising this thesis, a paper gained a lot of attention showing that the quality of communication between the coach/manager and the health professional team influenced team injury rates in professional football.

In conclusion, the workload—-injury field has several avenues for future research. Treating this thesis as a hub, I see the potential for at least four spokes in the domains of: (i) sport-specific workload measures and derived variables (e.g., jumping in volleyball), (ii) aetiological investigations that incorporate workloads in addition to traditional risk factors and apply
appropriate longitudinal data analyses, (iii) injury prediction models that incorporate multiple variables from larger datasets as technological advancements continue, and (iv) clinical trials that employ best practice workload management.

Broadly speaking, my 2015-18 PhD experience provided me invaluable lessons in both research and life. In research, I learned that the right question makes all the difference; in life, I learned that the right team makes all the difference.
Bibliography


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Sampson JA, Fullagar HHK, Murray A. Evidence is needed to determine if there is a better way to determine the acute:chronic workload. Br J Sports Med 2017;51:621–2. doi:10.1136/bjsports-2016-097085


Appendices

Appendix A - Additional tables and figures from Chapter 5

Table A.1 – Example search categories and terms used in chapter 5.

<table>
<thead>
<tr>
<th>Category</th>
<th>Search Terms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population</td>
<td>Athlet*, sport*</td>
</tr>
<tr>
<td>Method</td>
<td>Systematic review, Consensus statement</td>
</tr>
<tr>
<td>Outcome (Injury)</td>
<td>Injur*, illness*, strain, sprain, incidence, overuse, overreach*, accidents, stress, wellness, recover*</td>
</tr>
<tr>
<td>Workload</td>
<td>Training, resistance training, external load, internal load, workload, acute:chronic workload ratio, congested calendar, physical exertion, session RPE, global position systems, accelerometry, intensity, duration, physical fitness, fatigue</td>
</tr>
</tbody>
</table>
Figure A.1: Flow chart of included articles for Chapter 5.
Table A.2: Expanded table of 3-fold alignment (with descriptions)

<table>
<thead>
<tr>
<th>Method</th>
<th>n</th>
<th>Themes of theoretical model</th>
<th>Themes of temporal design: intensive longitudinal data</th>
<th>Statistical summary and typical uses</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Themes of theoretical model</td>
<td>Themes of temporal design: intensive longitudinal data</td>
<td>Statistical summary and typical uses</td>
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<tr>
<td></td>
<td></td>
<td>Multifactorial aetiology</td>
<td>Missing/unbalanced data due to rep. measures</td>
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<td></td>
<td></td>
<td>Between and within-athlete differences</td>
<td>Statistical summary and typical uses</td>
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<td></td>
<td></td>
<td>Complex system</td>
<td>Repeated measure dependency</td>
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<td>Incorporates time in the analysis</td>
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<td></td>
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</tr>
<tr>
<td>Correlation (Pearson and Spearman)</td>
<td>10</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Anderson - 2003</td>
<td></td>
<td>Can only handle one x and one y variable.</td>
<td>Correlation could be at the team level (training load and # of injuries), or on individual level with quantitative outcome, but cannot differentiate within/between athlete differences.</td>
<td>No interactions between multiple predictors</td>
</tr>
<tr>
<td>Bresciani - 2010</td>
<td></td>
<td></td>
<td>No, assumes independent observations.</td>
<td></td>
</tr>
<tr>
<td>Books - 2008</td>
<td></td>
<td></td>
<td>Assumes one observation per research participant/study unit, so participants couldn't have different numbers of observations.</td>
<td>In this case the correlation assumes independent observations so dependency is not taken into account.</td>
</tr>
<tr>
<td>Gabbett - 2004</td>
<td></td>
<td></td>
<td>No, assumes independent observations.</td>
<td></td>
</tr>
<tr>
<td>Killen 2010</td>
<td></td>
<td></td>
<td>In this case the correlation assumes independent observations so dependency is not taken into account.</td>
<td>Could, through having time as one of the variables, but cannot account for temporality</td>
</tr>
<tr>
<td>Mallo Della - 2012</td>
<td></td>
<td></td>
<td>No, assumes independent observations.</td>
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<tr>
<td>Murray Gabbett - 2016</td>
<td></td>
<td></td>
<td>No, assumes independent observations.</td>
<td></td>
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<tr>
<td>Windt – 2016</td>
<td></td>
<td></td>
<td>No, assumes independent observations.</td>
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</tr>
<tr>
<td>Unpaired t-test</td>
<td>6</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Dennis et al., 2003</td>
<td></td>
<td>Can only handle one x (grouping) and one y (outcome) variable</td>
<td>Only between-athlete differences (injured vs. uninjured)</td>
<td>No interactions between multiple predictors</td>
</tr>
<tr>
<td>Dennis et al., 2005</td>
<td></td>
<td></td>
<td>No interactions between multiple predictors</td>
<td></td>
</tr>
<tr>
<td>Duhig et al. - 2016</td>
<td></td>
<td></td>
<td>Independent samples t-test compare group means on either a time-varying (e.g. average workload) or time-invariant variable (e.g. height), not both.</td>
<td>No, assumes one observation per research participant/study unit, so by design forces a balanced set (1 observation per participant)</td>
</tr>
<tr>
<td>Owen et al., 2015</td>
<td></td>
<td></td>
<td>No, assumes one observation per research participant/study unit, so by design forces a balanced set (1 observation per participant)</td>
<td>By definition, assumes independence</td>
</tr>
<tr>
<td>Saw et al., 2011</td>
<td></td>
<td></td>
<td>By definition, assumes independence</td>
<td>None included time in the analysis,</td>
</tr>
<tr>
<td>Visnes Bahr., 2013</td>
<td></td>
<td></td>
<td>By definition, assumes independence</td>
<td></td>
</tr>
</tbody>
</table>
Table A.2 (continued): Expanded table of 3-fold alignment (with descriptions)

<table>
<thead>
<tr>
<th>Method</th>
<th>Themes of theoretical model</th>
<th>Themes of temporal design: intensive longitudinal data</th>
<th>Statistical summary and typical uses</th>
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<tbody>
<tr>
<td></td>
<td><strong>Themes of theoretical model</strong></td>
<td><strong>Themes of temporal design: intensive longitudinal data</strong></td>
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<td></td>
<td>Multifactorial aetiology</td>
<td>Between and within-athlete differences</td>
<td>Complex system</td>
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<tr>
<td>Chi-Square Tests</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Murray - 2016 - IJSPP</td>
<td>Only examines load groups and injury incidence across different load groups only</td>
<td>Examines differences in injury incidence between multiple predictors</td>
<td>Included load groups and injury incidence only</td>
</tr>
<tr>
<td>Relative Risk Calculations</td>
<td>O</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Bowen - 2016 Dennis - 2003 Dennis - 2005 Hulin - 2014 Hulin - 2016 Hulin - 2016 Murray - 2016 - Scand.</td>
<td>In some cases, authors examined relative risks of loading groups after subdividing across another variable, like chronic workload, making it multifactorial. Other authors only examined risks across load groups.</td>
<td>No differentiation, and independence assumed</td>
<td>No interactions between multiple predictors</td>
</tr>
</tbody>
</table>

Many of these RR approaches seem to use RRs that traditionally require independence, but do not account for this in their analysis.
Table A.2 (continued): Expanded table of 3-fold alignment (with descriptions)

<table>
<thead>
<tr>
<th>Method</th>
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<th>Themes of theoretical model</th>
<th>Themes of temporal design: intensive longitudinal data</th>
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<td>Between and within-athlete differences</td>
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<td></td>
<td>Complex system</td>
<td>Includes time-varying and time-invariant variables</td>
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<td></td>
<td></td>
<td>Missing/unbalanced data (due to rep. measures)</td>
<td>Repeated measure dependency</td>
<td>Incorporates time in the analysis</td>
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</tbody>
</table>

**Regression (logistic, linear, multinomial)**

<table>
<thead>
<tr>
<th>Method</th>
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<th>Themes of theoretical model</th>
<th>Themes of temporal design: intensive longitudinal data</th>
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<td>Missing/unbalanced data (due to rep. measures)</td>
<td>Repeated measure dependency</td>
<td>Incorporates time in the analysis</td>
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**Paired t-test**

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<tr>
<th>Method</th>
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<th>Themes of theoretical model</th>
<th>Themes of temporal design: intensive longitudinal data</th>
<th>Statistical summary and typical uses</th>
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<td>Multifactorial aetiology</td>
<td>Between and within-athlete differences</td>
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<tr>
<td></td>
<td></td>
<td>Complex system</td>
<td>Includes time-varying and time-invariant variables</td>
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<td></td>
<td></td>
<td>Missing/unbalanced data (due to rep. measures)</td>
<td>Repeated measure dependency</td>
<td>Incorporates time in the analysis</td>
</tr>
</tbody>
</table>

**Notes:**
- Some authors include multiple variables, others only single load measurements independently.
- Assumes 1 observation per unit.
- Visnes & Bahr (2013) do take it into account through logistic regression, but do not have the benefit of ILD.
- Assumes 1 observation per unit.
Table A.2 (continued): Expanded table of 3-fold alignment (with descriptions)

<table>
<thead>
<tr>
<th>Method</th>
<th>n</th>
<th>Themes of theoretical model</th>
<th>Themes of temporal design: intensive longitudinal data</th>
<th>Statistical summary and typical uses</th>
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<tbody>
<tr>
<td>Repeated measures ANOVA (One- or two-way ANOVA allowing for between- and within)</td>
<td>5</td>
<td>- Multifactorial aetiology</td>
<td>- Between and within-athlete differences</td>
<td>- Complex system</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>- Includes time-varying and time-invariant variables</td>
<td>- Missing/unbalanced data (due to rep. measures)</td>
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<td></td>
<td>- Repeated measure dependency</td>
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<td></td>
<td></td>
<td>- Incorporates time in the analysis</td>
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<tr>
<td>Ehrman - 2016</td>
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<tr>
<td>Gabbett - 2004</td>
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<tr>
<td>Malisoux - 2013</td>
<td></td>
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<tr>
<td>Murray - 2016</td>
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<tr>
<td>Killen - 2011</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Murray (2016) compared part of season by training load group, Malisoux (2013) and Ehrman (2016) only compared injury and pre-injury blocks</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In some cases (Murray, 2016), a two-way repeated measures ANOVA can examine between and within athlete differences in risk</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>No interactions between multiple predictors</td>
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<td></td>
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<tr>
<td>Murray (2013) compared load group by season period</td>
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<tr>
<td>Assume sphericity</td>
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<tr>
<td>Yes, by definition</td>
<td></td>
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<tr>
<td>Yes, as season period, or as pre-injury and injury period</td>
<td></td>
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<tr>
<td>Cox proportional hazards model</td>
<td>1</td>
<td>√</td>
<td>√</td>
<td>√</td>
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<td></td>
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<tr>
<td>Malisoux - 2013</td>
<td></td>
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<tr>
<td>Included volume and intensity of training along with age and sex</td>
<td></td>
<td>Cox PH conducted at the team/school level examined between-athlete differences</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No interactions between factors</td>
<td></td>
<td>Only included average weekly load and average intensity</td>
<td></td>
<td>Can handle unbalanced data</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>By using time-to-event as the outcome, Cox-PH robust to this dependency</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Uses time to event in analysis</td>
</tr>
<tr>
<td>Method</td>
<td>Themes of theoretical model</td>
<td>Themes of temporal design: intensive longitudinal data</td>
<td>Statistical summary and typical uses</td>
<td></td>
</tr>
<tr>
<td>------------------------------------------</td>
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<td>------------------------------------------------------</td>
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<td></td>
</tr>
<tr>
<td>Method</td>
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<tr>
<td>Generalised estimating equations</td>
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<tr>
<td>equations (modeled through logistic and</td>
<td></td>
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<td></td>
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<tr>
<td>poisson regression)</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Most authors used multiple variables</td>
<td></td>
<td></td>
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<tr>
<td>with GEEs, although some only used GEE</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>to account for repeated measures</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>(Dennis, 2004, Gabbett Domrow, 2007)</td>
<td></td>
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</tr>
<tr>
<td>Provides an ‘average’ effect for all</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>athletes, but controls for the clustering</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>No complex interactions between factors</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Some authors only predicted injury (y/n)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>based on workload variables</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>GEEs handle unbalanced data well</td>
<td></td>
<td></td>
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<tr>
<td>GEE accounts for clustering</td>
<td></td>
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<td></td>
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<tr>
<td>Do not incorporate time explicitly into</td>
<td></td>
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</tr>
<tr>
<td>the modelling process, and none of the</td>
<td></td>
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</tr>
<tr>
<td>authors included time in the model.</td>
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</tr>
<tr>
<td>Multilevel Modeling</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Multiple physical outputs included</td>
<td></td>
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<tr>
<td>and pre-season training</td>
<td></td>
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<tr>
<td>Within-athlete risks determined from</td>
<td></td>
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<tr>
<td>level 1 variables, between-athlete from</td>
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<td></td>
</tr>
<tr>
<td>level 2</td>
<td></td>
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</tr>
<tr>
<td>No complex interactions between factors</td>
<td></td>
<td></td>
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<tr>
<td>Pre-season and player variables along</td>
<td></td>
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<td></td>
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<tr>
<td>with training variables</td>
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<tr>
<td>Multilevel models are robust to</td>
<td></td>
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<tr>
<td>missing/unbalanced data</td>
<td></td>
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<td></td>
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<tr>
<td>Via random effects for each player</td>
<td></td>
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<tr>
<td>Analysed weekly risk and subsequent</td>
<td></td>
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<tr>
<td>week risk; did not directly incorporate</td>
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<tr>
<td>time</td>
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</tr>
<tr>
<td>Frailty model</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes, previous injury and physical outputs</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Unclear (I think yes due to the frailty</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>term)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No complex interactions between factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes (injury history)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes (robust to unbalanced data)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Yes (frailty model allows for dependency</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>of recurrent events)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes, since this is a time-to-event</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>analysis.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gabbett Ullah - 2012</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Included a many physical output metrics that were dichotomised into high/low categories and calculated RRs. Multicollinearity was not considered.
Appendix B - Additional tables and figures from Chapter 6

**Table B.1:** Expanded table of 3-fold alignment (with descriptions)

<table>
<thead>
<tr>
<th>Level of data</th>
<th>Purpose in model</th>
<th>Variable</th>
<th>Scale</th>
<th>Units</th>
<th>Treatment in models(a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 2</td>
<td>Unit of analysis</td>
<td>Team</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Covariate</td>
<td>Average team age</td>
<td>Continuous</td>
<td>Years</td>
<td>Centred on grand mean</td>
</tr>
<tr>
<td></td>
<td>Covariate</td>
<td>UEFA Club Coefficient</td>
<td>Continuous</td>
<td>Arbitrary Units</td>
<td>Centred on grand mean</td>
</tr>
<tr>
<td>Level 1</td>
<td>Unit of analysis</td>
<td>Match observation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Outcome variable (Model 1)</td>
<td>Total distance</td>
<td>Continuous</td>
<td>Meters</td>
<td>Raw</td>
</tr>
<tr>
<td></td>
<td>Outcome variable (Model 2)</td>
<td>% of distance covered &gt; high speed (&gt;14 km/h)</td>
<td>Continuous</td>
<td>Percent</td>
<td>Raw</td>
</tr>
<tr>
<td></td>
<td>Outcome variable (Model 3)</td>
<td>Number of sprints</td>
<td>Continuous</td>
<td>Sprints (count)</td>
<td>Raw</td>
</tr>
<tr>
<td></td>
<td>Independent variable of interest</td>
<td>Player unavailability</td>
<td>Continuous</td>
<td>Players (count)</td>
<td>Raw (Random)</td>
</tr>
<tr>
<td></td>
<td>Score differential</td>
<td>Continuous</td>
<td>Goals</td>
<td>Raw (Random)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Venue</td>
<td>Categorical</td>
<td>0 = Away</td>
<td>Raw (Random)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1 = Home</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Covariates</td>
<td>Stage</td>
<td>Categorical</td>
<td>0 = Group Stage</td>
<td>Raw (Random)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 = Knockout</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ball possession</td>
<td>Continuous</td>
<td>% of game time</td>
<td>Centred within cluster (Random)</td>
</tr>
</tbody>
</table>

Level 2 Variables can be included in the model (1) in the raw metric or (2) centred at the grand mean; Level 1 Variables can be included (1) in the raw metric, (2) centred at the grand mean, or (3) centred within the cluster (at the team average). Additionally, Level 1 variables can be fixed (where the effect is assumed to be the same for all teams) or random (where the effect is allowed to vary between teams).
Table B.2 – Multilevel linear regression model outputs

<table>
<thead>
<tr>
<th>2014–2015 season</th>
<th>Model 1 — total distance</th>
<th>Model 2 — number of sprints</th>
<th>Model 3 — percentage of distance covered &gt;14 km/h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed effects</td>
<td>Coefficient</td>
<td>S.E.</td>
<td>P-Value</td>
</tr>
<tr>
<td>Intercept</td>
<td>$\beta_{00}$</td>
<td>113.276.8</td>
<td>706.5</td>
</tr>
<tr>
<td>Player absences</td>
<td>$\beta_{10}$</td>
<td>596.7</td>
<td>638.3</td>
</tr>
<tr>
<td>Stage (Knockout)</td>
<td>$\beta_{20}$</td>
<td>−957.4</td>
<td>799.1</td>
</tr>
<tr>
<td>Venue (Home)</td>
<td>$\beta_{30}$</td>
<td>245.9</td>
<td>641.0</td>
</tr>
<tr>
<td>Score</td>
<td>$\beta_{40}$</td>
<td>514.7</td>
<td>182.2</td>
</tr>
<tr>
<td>Ball possession</td>
<td>$\beta_{50}$</td>
<td>79.0</td>
<td>65.7</td>
</tr>
<tr>
<td>UEFA CC</td>
<td>$\beta_{01}$</td>
<td>−30.7</td>
<td>70.3</td>
</tr>
<tr>
<td>Team age</td>
<td>$\beta_{01}$</td>
<td>−525.89</td>
<td>420</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Random effects</th>
<th>Standard deviation (p-Value)</th>
<th>Standard deviation (p-Value)</th>
<th>Standard deviation (p-Value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>$\tau_{00}$</td>
<td>2632.4 (p = 0.022)</td>
<td>24.5 (p = 0.080)</td>
</tr>
<tr>
<td>Player absences</td>
<td>$r_{10}$</td>
<td>221.2 (p = 0.246)</td>
<td>6.34 (p = 0.119)</td>
</tr>
<tr>
<td>Residual</td>
<td>$\tau_{11}$</td>
<td>3122.9</td>
<td>44.2</td>
</tr>
</tbody>
</table>
Table B.2 (continued): Multilevel linear regression model outputs

<table>
<thead>
<tr>
<th>2015–2016 season</th>
<th>Model 1 — total distance</th>
<th>Model 2 — number of sprints</th>
<th>Model 3 — percentage of distance covered &gt;14 km/h</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed effects</strong></td>
<td><strong>Coefficient</strong></td>
<td><strong>S.E.</strong></td>
<td><strong>P-Value</strong></td>
</tr>
<tr>
<td>Intercept</td>
<td>$\beta_{00}$</td>
<td>106,003.5</td>
<td>1250.1</td>
</tr>
<tr>
<td>Player absences</td>
<td>$\beta_{10}$</td>
<td>1395.3</td>
<td>1085.9</td>
</tr>
<tr>
<td>Stage (Knockout)</td>
<td>$\beta_{20}$</td>
<td>2010.6</td>
<td>1209.0</td>
</tr>
<tr>
<td>Venue (Home)</td>
<td>$\beta_{30}$</td>
<td>589.7</td>
<td>670.2</td>
</tr>
<tr>
<td>Score</td>
<td>$\beta_{40}$</td>
<td>17.9</td>
<td>219.6</td>
</tr>
<tr>
<td>Ball possession</td>
<td>$\beta_{50}$</td>
<td>−62.4</td>
<td>70.9</td>
</tr>
<tr>
<td>UEFA CC</td>
<td>$\beta_{60}$</td>
<td>55.1</td>
<td>77.2</td>
</tr>
<tr>
<td>Team age</td>
<td>$\beta_{70}$</td>
<td>−213.1</td>
<td>580.8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Random effects</strong></th>
<th><strong>Standard deviation (p-Value)</strong></th>
<th><strong>Standard deviation (p-Value)</strong></th>
<th><strong>Standard deviation (p-Value)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>$\tau_{00}$</td>
<td>4454.5 (p &lt; 0.001)</td>
<td>19.1 (p = 0.015)</td>
</tr>
<tr>
<td>Player absences</td>
<td>$\tau_{10}$</td>
<td>808.3 (p = 0.059)</td>
<td>5.5 (p = 0.036)</td>
</tr>
<tr>
<td>Residual</td>
<td>$\tau_{11}$</td>
<td>3159.0</td>
<td>34.7</td>
</tr>
</tbody>
</table>

Bolded rows highlight Player Unavailability Estimates. The estimated ‘average’ effect of player unavailability is the ‘fixed’ effect coefficient, while the variation of this effect across teams is found in the random effects section of the table.

*p < 0.05.
Table B.3. Team information about injuries and player availability during the 2014–2015/2015–2016 UCL seasons.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Range</td>
</tr>
<tr>
<td>Age</td>
<td>25.1 ± 1.3</td>
<td>22.3–27.1</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>182.0 ± 1.53</td>
<td>177.8–185.3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>77.8 ± 2.3</td>
<td>73.1–82.9</td>
</tr>
<tr>
<td>Squad size (n)</td>
<td>31.2 ± 5.0</td>
<td>23–40</td>
</tr>
</tbody>
</table>

Injury totals (n)

- Overall        | 41.2 ± 14.1     | 17–70           | 38.7 ± 12.8     | 19–66           |
- Match          | 23.3 ± 9.2      | 11–45           | 22.5 ± 8.0      | 8–39            |
- Training       | 17.9 ± 8.0      | 3–40            | 16.2 ± 7.7      | 3–32            |

Injury incidence (/1000 h)

- Overall        | 5.7 ± 2.3       | 1.6–10.8        | 6.1 ± 2.4       | 2.9–12.8        |
- Match          | 20.6 ± 7.9      | 8.8–40.5        | 20.8 ± 6.8      | 8.9–34.0        |
- Training       | 3.0 ± 1.3       | 0.3–6.3         | 3.1 ± 1.5       | 0.43–6.9        |

Injury type

- Acute          | 26.2 ± 10.3     | 9–42            | 23.6 ± 11.3     | 6–45            |
- Overuse        | 15.0 ± 8.9      | 4–38            | 15.1 ± 7.6      | 4–32            |
Figure B.1: The average number of players unavailable due to injury for games in each month of competition play. Note — there were no matches played in January.
Appendix C: Note – thanks to Carson Grose and David Taylor for assistance with this proof.

\[ \text{Uncoupled} = \frac{W_1}{0.33(W_2 + W_3 + W_4)} \quad \text{Coupled} = \frac{W_1}{0.25(W_1 + W_2 + W_3 + W_4)} \]

\( W_1 = \text{Acute} \)

\( \mu = W_2 + W_3 + W_4 \)

Therefore:

\[ \text{Coupled} = \frac{A}{0.25(A + \mu)} = \frac{4A}{(A + \mu)} \]

\[ \text{Uncoupled} = \frac{A}{0.33(\mu)} = \frac{3A}{(\mu)} \]

\[ A = \frac{\text{Uncoupled} \cdot \mu}{3} \]

Therefore:

\[ \text{Coupled} = \left( \frac{4 \cdot \text{Uncoupled} \cdot \mu}{3} \right) + \left( \frac{\text{Uncoupled} \cdot \mu + 3\mu}{3} \right) \]

\[ \text{Coupled} = \frac{4 \cdot \text{Uncoupled} \cdot \mu}{\text{Uncoupled} \cdot \mu + 3\mu} \]

\[ \text{Coupled} = \frac{4 \cdot \text{Uncoupled} \cdot \mu}{\mu(\text{Uncoupled} + 3)} \]

\[ \frac{\text{Coupled}}{\text{Uncoupled} + 3} = 4 \cdot \text{Uncoupled} \]

\[ \text{Coupled} \cdot \text{Uncoupled} + 3 \cdot \text{Coupled} = 4 \cdot \text{Uncoupled} \]

\[ 3 \cdot \text{Coupled} = 4 \cdot \text{Uncoupled} - \text{Coupled} \cdot \text{Uncoupled} \]

\[ 3 \cdot \text{Coupled} = \text{Uncoupled}(4 - \text{Coupled}) \]

\[ \text{Uncoupled} = \frac{3 \cdot \text{Coupled}}{4 - \text{Coupled}} \]