THE EPIDEMIOLOGY OF INJURIES IN ELITE SOCCER AND THE IMPACT OF INJURY-INDUCED DETRAINING ON PHYSIOLOGICAL PERFORMANCE

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ABSTRACT

Injuries in professional soccer are common with the financial cost running into many millions of pounds. The incidence of re-injury on return to competitive action accounts for a high proportion of all injuries with inadequate rehabilitation being identified as one of the major factors when considering re-injury. Methods of improving the rehabilitation outcomes could therefore reduce the overall incidence of injury and may improve team performance through greater player availability.

The accuracy of the available literature addressing the epidemiology of soccer injuries is questionable. It was therefore necessary to establish the incidence of soccer injury and re-injury using comprehensive data collection methods. A data collection method based on the principals of first hand injury observation, comprehensive and immediate recording of injuries and accurately timed exposure for both training and competitive matches was therefore designed. Study 1 used these methods to scrutinise the results of previously published literature. Results showed that the definition of the term 'injury' can have a significant effect on the results from epidemiological studies of soccer injuries. It was also observed that estimating exposure can result in an under-calculation of the injury frequency rate when reporting the rate as new injuries per 1000 training or playing exposure hours. The new methods for collecting epidemiological data clearly showed that methodology is important in generating study results and was adopted for the current thesis.

The data collected to evaluate approaches to data collection indicated that injury is the major cause of absence in professional soccer although this assumption had never been investigated. Study 2 evaluated this supposition by investigating what percentage of total absences soccer injuries were responsible for. The data showed that injury was the major reason for absence with 50% of the incidents of match absence and 65% of training absence being as a result of soccer related injury. Other reasons for absence were also noted. For example, sickness and suspension were areas where it was suggested that availability could be positively affected by reducing the incidence of absence as a result of these two factors.

To highlight the aetiology and severity of soccer injuries at one professional soccer club, a five-season comprehensive epidemiological study was undertaken. Study 3 showed that the average percentage of the playing squad per season sustaining an injury that resulted in more than one days absence from training or playing was 86%. Over the five seasons, an average of 41% of players sustained >1 injury in any one season the severity of which resulted in more than one days absence. Eighty five percent of all injuries throughout the study period were sustained to the lower limb. When analysing both severity and anatomical location of injury, it was found that, during the 5 competitive-season period, 82 injuries were sustained to the lower limbs which resulted in an absence of >14 days. It was suggested that these injuries may necessitate alterations to habitual training patterns

which may, in turn, result in detraining. This detraining may result in players returning to competition ill-prepared for the physical and physiological demands of soccer play at the elite level and increase their susceptibility to re-injury.

In order that the effects of injury-induced absence could be investigated, changes in players performance, using a battery of soccer-specific field tests, was investigated in Study 4. All players absent for >2 d through injury were tested on their return to normal squad training. These results were compared with a data set collected when the players were fit and playing competitive soccer. No significant differences were observed in any performance variables for those players absent for <14 d. The results for players absent for >14d showed no significant differences in tests to evaluate flying 20 m sprint speed, counter-movement jump height and hamstring flexibility. Significant decreases in performance were however, found in estimated $\dot{V}O_{2max}$, and repeated sprint ability (RSA) performance. It was suggested that the decrements in aerobic power and repeated sprint ability may be related with high aerobic power being recognised as a requirement for recovery between bouts of high-intensity exercise. It was also suggested that one of the reasons for the performance decrements may be the changes in habitual soccer training patterns during rehabilitation.

To investigate this hypothesis, study 5 compared the rehabilitation training load and the training load when participating in normal squad training and playing competitive matches. A 17-month investigation to quantify and compare the two modes of training was undertaken. Players who were absent for >14 d due to soccer injury had their rehabilitation training quantified using a number of established methods. On their return to normal squad training, the same methods were used to quantify training for a period similar to the duration of their rehabilitation programme. The period of rehabilitation when players were unable to run, due to the restrictive nature of their particular pathology, lacked the intensity of exercise normally associated with squad training and playing matches. A comparison of other phases of rehabilitation and normal training found no significant differences in average heart rate or ratings of perceived exertion measures. It was therefore demonstrated that the major difference between the two training periods was during early rehabilitation.

In response to the findings of Study 5, Study 6 investigated the effect of increasing the intensity of exercise during early rehabilitation in reducing the effects of detraining following injury. This final study in the thesis investigated the effects of work-matched, constant pace, continuous exercise and intermittent-paced exercise in maintaining aerobic power and RSA performance. Players injured for >14 d (n=16) were randomly allocated to one of the two intervention groups and performed either continuous or intermittent exercise on rowers, cross trainers and cycle ergometers during the period of rehabilitation that they were unable to perform running exercise. Reductions in estimated $\dot{V}O_{2max}$ and RSA were observed in both intervention groups with the reductions in performance of the constant-pace group greater than those observed in the intermittent pace group. None of these differences however, reached significance leading to the conclusion that both intervention programmes were effective in preventing performance decrements in $\dot{V}O_{2max}$ and one was not superior to the other. The only negative changes observed were

in RSA performance decrement for both groups from pre-injury to post-injury tests. Although these changes were found to be significant, the level of change was not sufficiently great to surpass the figure calculated as the smallest meaningful change. This was due to the lack of reliability of this particular test.

Key words: Soccer, injury, performance, detraining.

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Declaration

I declare that the work presented in this thesis is entirely my own. Some of this work has been published in European and International Journals and presented at National and International conferences.

PUBLICATIONS AND COMMUNICATIONS

This thesis has resulted in the following publications and conference communications:

1. Parry, L. and Drust, B. (2006). Is injury the major cause of elite soccer players being unavailable to train and play during the competitive season? Physical Therapy in Sport, 7, 58- 64.

2. Drust, B., Parry, L., Gregson, W. and Lees, A. (2007). Effects of injury related de-training on gross motor performance in elite soccer players. Journal of Sports Science and Medicine, S10, P-060. World conference in Science and Football. Turkey, January, 2007 (Poster presentation).

3. Parry, L., Drust, B., Gregson, W. and Lees, A. Comparing the physical and physiological training loads of injured and uninjured players in professional soccer. 1st World Conference on Science and Soccer, Liverpool, May 2008. (Oral communication)

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LIST OF ABBREVIATIONS

ADP	adenosine triphosphate
ATP	adenosine triphosphate
BF%	percentage body fat
BPM	Beats per minute
°C	degrees Celsius
CK	creatine kinase
cm	centimetres
CS	citrate synthase
CV	cardiovascular
d	day(s)
PFK	Phosphofructokinase
g	gramme
h	hour(s)
HIIE	High-intensity intermittent exercise
HIIT	High-intensity intermittent training
HR	Heart rate
$\%HR_{max}$	Percentage of maximum heart rate
IPG	Intermittent-pace group
kg	kilogram
km	kilometre
LD	lactate dehydrogenase
LT	lactate threshold
m	metres
MD	malate dehydrogenase
min	minute(s)
mg	milligram
ml	millilitre
mm	millimetres
mmol	millimole
$m \cdot s^{-1}$	metres per second
MSS	maximum sprint speed
NAD	β-hydroxy-CoA-dehydrogenase
O_2	oxygen
PC	phosphocreatine
PFK	phosphofructokinase
ROM	Range of motion
rpm	Revolutions per minute
RSA	Repeated-sprint ability
cRSA	Cycling repeated-sprint ability
rRSA	Running repeated-sprint ability
RSA_{PD}	Repeated sprint ability performance decrement

SD	Succinate dehydrogenase
SRRI	soccer-related reportable injury
RT	Rehabilitation training
SD	succinate dehydrogenase
SPG	Steady-pace group
$\dot{V}_{E\,max}$	Maximal pulmonary ventilation
$\dot{V}O_2$	Oxygen uptake
$\dot{V}O_{2max}$	Maximal oxygen uptake
wk	week(s)
y	years(s)
μm	micrometre
<	less than
>	greater than
~	approximately

CONTENTS

1.1 BACKGROUND

An integral component of the responsibilities of a professional soccer club physiotherapist is to identify trends in injury patterns and design prophylactic interventions to reduce the incidence of specific injuries or specific aetiological factors. As a part of this process, the current researcher observed that a number of injuries appeared to be sustained to the same anatomical structure on the player's return to competitive action. On examination of the literature, these injuries would be classed as re-injuries and it appeared that re-injury in professional soccer was one of the major causes of players being unavailable to train and play competitive matches. It was difficult to confirm this observation due to the various methods of collecting and analysing the data which resulted in a wide range of frequencies of re-injury reported across the various studies conducted. A number of researchers concluded that one of the main reasons for these re-injuries was players being poorly rehabilitated and thus insufficiently prepared for the rigours of competition. These conclusions were based on the assumption that the high-frequency of second injuries to the same anatomical location and to the same anatomical structure were most probably caused by players not being sufficiently prepared to return to physical action. No research had been carried out to identify underlying reasons for the incidence of re-injury. This area of work was therefore considered suitable for the current thesis.

Epidemiological literature of injuries in soccer is copious with studies reporting injury rates from the amateur, elite youth, professional and international soccer populations. It was noted that results from many previous studies were based on methodologies, such as estimating time exposed to risk, that may result in the accuracy of the findings being questionable. The first stage of the current process was therefore to confirm that the perceived problem of injury was genuine and, if so, to identify the precise injury frequency rate. As part of the same process, aetiological and pathological information was required to facilitate the investigation of causative factors and incidence of re-injury.

It is possible that a player may experience a high degree of trauma to the same location as a previously pathology that would have damaged the same structure irrespective of previous damage. It is however, not unreasonable to suggest that a player sustaining such an injury can consider themselves as 'unlucky' and is probably why previous researchers have proposed that the major reason for the high incidence of re-injury is poor rehabilitation. Notwithstanding the above, it is unreasonable to attribute all re-injuries to the failure of medical and sports science staff to adequately prepare the player for competition. Pressure from coaching and managerial staff, especially with players who are perceived to be more valuable to the team effort, interfere with the decision making process. Pressure from the player themselves due to personal or financial considerations adds another subjective element to deciding when the player is suitably prepared. Making the decision when to return to action is therefore a miscellany of both subjective and objective factors and not an exact science. It would however, be irresponsible to ignore the possibility that the staff responsible for the rehabilitation of the player may

miscalculate the readiness of the player and declare him ready for competitive action. Those charged with the rehabilitation of the injured player may overestimate the capabilities of the player or underestimate the physical and physiological demands of training and playing. They may also fail to understand the discrete negative physiological adaptations which occur as a result of the changes in the habitual training and competing patterns of the player and the functional impact of these changes.

The negative physiological changes following a cessation or reduction in habitual training patterns is known as detraining. Detraining, following injury, has been identified as a contributing factor to re-injury in professional soccer. It has been demonstrated that periods of reduced activity as short as 10 d could bring about physiological adaptations which may result in performance decrements. In professional soccer, these negative changes could result in players' levels of fitness being compromised which may be detrimental to the player's performance and heath. Injuries to anatomical locations which prevent soccer specific training may increase the impact of detraining due to the inability to stimulate the specific motor units required for soccer performance. The incidence of soccer injuries which affect a player's ability to train normally for extended periods has not been reported. The extent of the underlying problem of injury-induced detraining is therefore unknown.

Changes in soccer-specific performance following injury have gone unreported. In fact, the number of studies that investigate the effects of detraining on sport-specific performance in any elite athletes is limited. To date, researchers have preferred to report discrete physiological and morphological adaptations following detraining and speculate how these changes may impact on athletic performance. However, it is likely that the athlete and the coach are more interested in the impact of detraining on performance. That is to say, athletes may be more concerned with how detraining has affected their ability to run, jump, throw or get to a ball before their opponents. To date, the interest has then focused on what is required to expedite the reinstatement of pre-injury levels of performance. As an alternative to accepting that detrimental changes will occur when injured, a more proactive approach is required to consider prophylactic measures aimed at reducing the effects of detraining. These measures may contribute to maintaining performance throughout the injury period and may also impact on the incidence of re-injury through better preparing players for the rigours of match-play.

1.2 AIMS, OBJECTIVES AND STRUCTURE OF THESIS

1.2.1 The main aims of this thesis are:

1 To determine the incidence and aetiology of injuries at a professional soccer club throughout a 5-season period.

2 To analyse the findings and implement further investigative studies to identify areas of risk which may predispose players to injury.

3 To design and implement interventions to reduce the effects of injury on performance at one professional soccer club.

1.2.2 The above aims will be achieved through the following objectives:

1. To develop a comprehensive data collection method for recording injuries sustained at a professional soccer club.

2. To complete a prospective longitudinal epidemiological study to identify risk factors to injury in professional soccer.

3. To utilising the findings of previous studies, to analyse the data and identify areas of risk on which interventions may have a positive effect on.

4. To investigate the differences in training load between normal squad training and playing and rehabilitation training.

5. To design and implement an intervention to reduce the effects of injury in professional soccer players.

1.2.3 Structure of this thesis

This thesis is organised into 6 chapters. A brief summary of each chapter follows:

Chapter 1 presents the introduction to this thesis. It introduces the background to the reasons for the area of research and presents the aims and objectives of the thesis.

Chapter 2 presents a literature review related to the areas of soccer injuries and detraining. These literature reviews concentrate of research in the field of professional soccer and soccer players and other elite sports people.

Chapter 3 investigates the incidence and aetiology of soccer injuries in professional soccer. Special consideration is given to injuries resulting in extended periods of absence and injuries which result in players being able to perform soccer-specific training. This is made possible by the design of a comprehensive data collection methodology presented at the beginning of the chapter.

Chapter 4 presents the findings of 2 studies. The first study investigates the effects of injury on player's ability to perform soccer-specific tasks. The second compares the training loads of players when they are injured with the training loads when players are training and playing normally.

Chapter 5 explores methods of reducing the effects of injury found in chapter 4 by introducing rehabilitation training interventions.

Chapter 6 presents a synopsis of the work completed for this thesis. Strengths and limitations of each study are discussed. The final part of this chapter proposes areas of work which may benefit from further research.

CHAPTER 2

Literature Review

2.1 The epidemiology of soccer injuries.

2.2 The physiological and physical effects of detraining.

2.1 INJURIES IN PROFESSIONAL SOCCER

2.1.1 Introduction

Association football (soccer) is the most popular sport in the world with an estimated 200 million active players (Stølen *et al.*, 2005; Faunø and Jakobsen, 2006). In the 1999-2000 season the English football leagues became the first in the world to pass £1 billion in income (Hawkins *et al.*, 2001) with £747 million of that sum redirected to player wages (Dietl *et al.*, 2006). Hawkins *et al.* (2001) calculated that absence through injury accounted for as much as 10% of total work time. This could amount to a sum of between £75 and £100 million in medical insurance and paid wages to unavailable players (Rahnama *et al.*, 2002; Woods *et al.*, 2002). If a large number of players from the same club are concurrently injured or the club's better players are injured, there may also be additional costs associated with the performance of the team. These costs cannot be easily quantified but may include reduced income from diminished prize money due to early cup exits and lower league placement (Woods *et al.*, 2002). In view of the increasing primary and secondary costs, as well as the personal suffering of the injured players, an analysis of the problem of injuries in the game is required. These analyses should have the ultimate aim of introducing proactive initiatives to reduce the risk of injury to professional soccer players.

Injury prevention is a term that is used extensively in scientific investigations into soccer epidemiology and aetiology (Inklaar *et al.*, 1993; Ekstrand, 1994b; Hawkins and Fuller, 1998a). The prevention of injury in professional soccer is a complex conundrum with multiple factors worthy of consideration. In theory, if all of these various factors that may predispose players to an increased risk of injury were eliminated, the result would be a sport where the only risks involved would be those that are inherently associated with the playing of the sport itself. In soccer, this level of risk may still be considerable. The combined aim of the amalgamation of scientific investigations into injury prevention should therefore be to identify the variety of factors that predispose players to injury and

their designs. Interventions to reduce the impact of these factors can then be practicably implemented.

The following section of the literature review is split into two sub-sections. The first section explores important considerations within the methodological design of epidemiological studies. This information is analysed in an attempt to perform a critical evaluation as to which are the most effective methods for data collection in this area of research. The second section reports on previous investigations into the epidemiology and aetiology of injuries in professional soccer.

2.1.2 The methodology of collecting epidemiological data in injury investigations related to soccer

2.1.2.1 Background

It is generally accepted that soccer has developed to become faster, with more intensity and aggressive play than seen previously, especially at the professional level (Andersen *et al.*, 2004a; Árnason *et al.*, 2004a). With this increase in intensity, a concomitant increase in participation (Dvorak and Junge, 2000) and the tight scheduling of training sessions and matches (Hägglund *et al.*, 2003) has occurred. Soccer injuries now account for 25-50% of all sport injuries (Hoy *et al.*, 1992; Andersen *et al*, 2004a). The epidemiology and aetiology of soccer injuries has therefore become the object of major medical investigations. Primarily, the aims of these studies have been to establish the extent of the problem in comparison with other populations (Hoff and Martin, 1986; Poulsen *et al.*, 1991; Inklaar *et al.*, 1996; Hawkins and Fuller, 1999; Naunheim *et al.*, 2000; Junge *et al.*, 2004; Giza *et al.*, 2005; Waldén *et al.*, 2005) and to monitor changes across a time scale in the same population (McGregor *et al.*, 2000; Hägglund *et al.*, 2003; Chougle *et al.*, 2005). Identifying common pathologies and their risk factors has also been the focus of several studies (Ekstrand and Tropp, 1990; Hoy *et al.*, 1992; Tucker, 1997; Witvrouw *et al.*, 2003; Dabebo *et al.*, 2004; Volpi *et al.*, 2004; Woods *et al.*, 2004), and to investigate the complex interaction between these risk factors (Ekstrand *et al.*, 1983a; Fuller *et al.*, 2004a). Other research has gauged the effectiveness of interventions

designed to reduce the incidence of specific injuries (Ekstrand *et al.*, 1983a; Pafis *et al.*, 2005) and to investigate the effect of injury on team success (Brynhilsden *et al.*, 1990; Árnason *et al.*, 2004a).

It has been well documented that variations in definitions and methodologies create significant differences in the results and conclusions obtained from studies of injuries in soccer (Junge and Dvorak, 2000; Fuller *et al.*, 2006). This plethora of methodological approaches makes it difficult to compare studies and draw coherent conclusions from the data. The accuracy of reporting can also be questioned due to different methodological processes such as the methods of data collection and the approach used to calculate exposure. For example, if the methods for collecting/calculating exposure data used in an intervention study are not valid or reliable, any inferences as to the effectiveness of the intervention regarding risk per time exposure will be flawed, which will increase the possibility of a type 1 or type II error. The accuracy and significance of other important methodological considerations, for example, incidence of injury with regard to factors such as anatomical, pathological, aetiological and severity parameters is also disparate. It is therefore important that the foundations upon which these studies are built are sound and researchers can be confident that subsequent research is not compromised by an inaccurate data set. Epidemiological research of the aetiology of soccer injuries therefore requires a conceptual model based on sound methodological principles in order to be effective.

When designing the methodology for injury surveillance investigations, the factors highlighted above have to be considered. Such considerations are founded on the definition of the injury, the source of the information collected, and the availability of accurate exposure data (Junge *et al.*, 2004). Each of these considerations are discussed in the sections that follow.

2.1.2.2 Defining injury

Creating a definition of injury is one of the most challenging parts of designing any injury surveillance study (Noyes et al., 1988). Careful consideration must be given to the

aims of the study. The manpower requirements of specific injury definitions must be also accurately assessed as the interpretation of the term 'injury' can have a significant effect on the workload of the researchers. Fuller *et al.* (2006) defined injury as any physical complaint sustained by a player as a result of soccer participation, irrespective of the need for treatment or time absent. This is the precise definition of the word 'injury' but is impractical to use in epidemiological investigations because of the difficulties associated with recording injuries that don't require medical attention. Some studies define injury as "a soccer related incident resulting in medical attention" (Lüthje *et al.*, 1996; Yoon *et al.*, 2004). In England, recording these injuries is a legal requirement under the Reporting of Injuries, Diseases and Dangers Occurrences Regulations (RIDDOR) so information should be readily available (Fuller, 1995). The most common criterion when defining a soccer injury is an absence from training or match(es) (Dvorak and Junge, 2000). Absence criteria range from 1 session (Hägglund *et al.*, 2005), 1 day (Drawer and Fuller, 2002), 48 hours (Hawkins *et al.*, 2001; Le Gall *et al.*, 2006), 3 days (Volpi *et al.*, 2004) and periods between 1 wk (Junge *et al.*, 2000) and 4 wks (Chomiak *et al.*, 2000). Although some of these variations in definition are based on a sound methodological rationale (Chomiak *et al.*, 2000), the inclusion of any 'time out of training/playing' criterion in the definition of 'injury' automatically excludes a stratum of data. This lack of consensus results in there being no universal definition of soccer injury (Dvorak and Junge, 2000).

Defining injury as "any soccer related incident resulting in medical attention" is the most comprehensive definition that is practicable in soccer injury epidemiology. The term 'medical attention' is preferred as using 'treatment' infers that a physical medical intervention occurred, which is not always the case. This definition however, requires a high level of conscientiousness from those responsible for collecting the data and the additional depth of data collected may not be worth the additional work (Fuller *et al.*, 2006).

The time loss criterion of "misses the next training session or match" is a popular approach (Hawkins and Fuller, 1999; Ekstrand *et al.*, 2004b; Hägglund *et al.*, 2005). One

of the advantages of this criterion is that it gives a reflection of interruptions to the player's participation in soccer specific activities. There are however, circumstances when participation could be affected but the inclusion criteria are not met. For example, a player could leave the field of play in the first minute of a competitive match through injury and still be available for the following training session. Using the above definition, this would not be recorded as an injury although it is quite evident that the player's participation, and maybe the team's performance, was significantly affected. Minor injuries and injuries 'cured' by analgesics or other medication are also neglected when using a duration of absence criterion (Junge and Dvorak, 2000). Failure to record these events would reduce the accurate incidence of injury resulting in misleading conclusions.

The decision whether or not to participate in the following session may also raise issues of subjectivity. One player may feel able to play with an injury while another, with the same injury, will feel too impaired to return to play (Noyes *et al.*, 1988). Players may also feel under immense pressure to maintain their team position and to advance their sporting careers (Lewin, 1989) therefore leading to a failure to report injuries. In the case of competitive matches, the importance of a match may have an impact on the player's perception of being available for selection as well as resulting in players not reporting injuries prior to important matches (Waldén *et al.*, 2005). Absence therefore is influenced by psychological factors and is not absolutely objective (Dvorak and Junge, 2000).

The comprehensive study of English professional soccer by Hawkins *et al.* (2001) used the time loss criterion of >48 h not including the day of the injury. Their rationale was that it was impractical to collect data on injuries resulting in <48 h because of the working practices within professional soccer (e.g. the club my have an approach to slight injuries which requires players to miss training for 2 d irrespective of their ability to train). This decision suggests that collection of data on all injuries requires a level of diligence that may not be available when using third party, study detached, volunteer data collectors. They also suggested that the main impact of injuries on the club was players absent from competitive matches so the first 2 d of the injury were not significant. However, periods when matches are condensed, such as Christmas and Easter, could

result in players with injury absences of <3 d being unavailable for a match. There may therefore be periods when injury absence of <3d are significant.

The definition "any soccer related incident which results in medical attention" is the optimal definition of injury in epidemiological studies. This definition will result in a comprehensive data set that enables investigations into the incidence of injury of any severity. It is however recognised that this is a time consuming method and requires a committed and meticulous approach for data collection. It is also recognised that, depending on the aims of the specific study, this definition may produce superfluous information that does not warrant the additional time spent collecting it. If a time loss criterion is utilised in the definition, it should be based on days and not sessions. This will eliminate problems with the differences in time between scheduled sessions so the term 'whether or not a session was timetabled" should be included.

2.1.2.3 Defining re-injury

Defining the term 'injury' is a complex undertaking as discussed above. The definition of the term 're-injury' is no clearer when criterion including a time from the original injury variable and an anatomical location are included. To further complicate the comparison of apparently similar studies, some researchers categorise further injuries, not related anatomically, as re-injuries (Árnason et al., 1996). This interchangability in the terminology defining re-injury and further injury is one reason for the discrepancy in re-injury rates reported in the literature. Further discussion in this section will differentiate between re-injury (a subsequent injury to the same anatomical location and structure) and further injury (a subsequent injury to a different anatomical location and/or structure). When defining re-injury, the criteria of an identical injury to the same side and same anatomical location are consistent throughout studies (Hawkins *et al.*, 2001; Hägglund *et al.*, 2007). The most common timescale for the occurrence of the subsequent injury is two months following the last day of rehabilitation following the first injury (Ekstrand and Gillquist, 1983b; Waldén *et al.*, 2005; Hägglund *et al.*, 2007). A re-injury within this 2 month timescale has also been defined as an 'early recurrence' while the term 'recurrence' is used if an injury is sustained after 2 months (Hägglund *et al.*, 2006).

Hawkins *et al.* (2001) reported a low re-injury rate of 7% when defining re-injury as an identical injury during the same season. This extended timescale could have resulted in re-injuries being up to 9 months after the original injury. This low re-injury rate therefore highlights the subjective element of diagnosing injuries. Physiotherapists may see re-injury as a failure on their part and therefore be reluctant to classify injuries as re-injuries, especially for investigations implemented by their sports governing body. Categorising re-injury into short- and long-term re-injuries allows additional analysis of pathological sequelae which may facilitate the identification of preventative interventions for individuals and groups. However, some sort of time limit is crucial when examining re-injuries (Waldén *et al.*, 2005) and a category of <6 months, in addition to <2 months, may be more effective when examining the timescales associated with re-injury.

Previous injury has also been associated with an increased risk to further injury. Hägglund *et al.* (2006) reported that players with an injury during one season are three-fold more likely to sustain an injury during the following season. The relative risk of injury was also found to increase with the number of injuries that a player sustained during the previous season. Many authors have, however, reported that a high percentage of players (77-100%) sustain an injury at some point during a season (Lewin, 1989; Hawkins and Fuller, 1999; Hawkins *et al.*, 2001; Waldén *et al.*, 2005). This being the case, it is inevitable that there is a long-term association between injury and previous injury and therefore previous injury may not be the causative factor. This highlights the need for an agreed 'cut-off point' when examining the association between injury and further injury. Ekstrand and Gillquist (1983b) found that one third of moderate and major injuries occurred within 2 months of a minor injury. This finding, with its shorter timescale, may justify the additional work of recording minor injuries and categorising further injuries sustained within 2 months of a previous injury irrespective of anatomical location and type of injury.

2.1.2.4 Methods of data collection

Data collection is the most critical methodological variable in soccer injury studies (Poulsen *et al.*, 1991). The methods of data collection strongly influence the validity of

incidence of injury (Inklaar, 1994a). Methods vary considerably ranging from a limited number of studies where one of the authors has daily contact with players (McMaster and Walter, 1978; Lewin, 1989), watching television and reading press reports (Hawkins and Fuller, 1998a), recording injuries treated in an hospital casualty or other medical department (Høy *et al.*, 1992; Goga and Gongal, 2003) and examining soccer injuries for which an insurance claim was made (Cumps *et al.*, 2008). The most popular method of collecting data from a number of different teams is to have a member of each clubs' medical team responsible for completing pro-forma to record injuries and playing exposure on a regular basis (Poulsen *et al.*, 1991; Lűthje *et al.*, 1996; Hawkins *et al.*, 2001; Junge *et al.*, 2004). Consequently, this means that the majority of investigations rely upon third party, study detached volunteer recruits to diligently and regularly complete injury questionnaires or complete retrospective questionnaires (Ekstrand and Gillquist, 1982; Lilley *et al.*, 2002).

Each of the methods detailed above have inherent weaknesses. Using insurance or hospital records limits studies to the more serious injuries with less serious and overuse injuries likely to be missed (van Mechelen *et al.*, 1992). Also in these investigations, the population at risk is unknown. For example, the 715 injuries reported by Høy *et al.* (1992) may have been 1% of the playing population or 100%, the limitations of the study don't allow this information to be calculated. Using video analysis (Hawkins and Fuller, 1996; Hawkins and Fuller, 1998a; Rahnama *et al.*, 2002; Árnason *et al.*, 2004c; Fuller *et al.*, 2004c) limits data to incidents that can be observed on the pitch during matches. Hawkins and Fuller (1996) estimated that, due to the limitations of the cameras following the ball, at least 54 injuries were missed when using video footage to analyse the cause of injuries during the 1994 World Cup. Andersen *et al.* (2004b) supported these ideas stating that, regardless of thorough video analysis, less than half of injuries were identified using this method when compared with prospectively collected medical records. When video is used in isolation therefore, under reporting of the injury frequency rate (IFR) would appear unavoidable and suggests that video is unsuitable for studies that require examination all injuries.

De Loës (1997) suggested that data collection could be a complex task with arduous access to data collection being a hindrance and even some simple methods being very time-consuming. This may be one reason why expecting third party study-detached volunteer data collectors to diligently complete questionnaires results in unreliable and inaccurate returns (Twellaar *et al.*, 1996). The response rate of data collectors has also been questioned. In their study of FIFA and Olympic soccer matches, Junge *et al.* (2004) reported an average response rate of 84% with an inter-team range from 47% to 100%. These findings are from the top elite level of soccer and teams which would be expected to employ numerous medical and backup staff. The response level from lower levels of professional soccer, where staffing levels may not be as high, may be even lower. The reliability of data extrapolated from the injury record sheets competed by volunteer data collectors has also been questioned (Lindenfeld *et al.*, 1988; Twellaar *et al.*, 1996). This is confirmed in a 10-season study of injuries in elite young French soccer players when no effort was made to record the cause of injuries and the time in a match that injuries occurred (Le Gall *et al.*, 2006). The authors stated that this was because they could not be in attendance at all matches, and it was deemed too unreliable to ask coaching staff to record the data and report back. Third-party submission of data is always voluntary and, unfortunately, dropouts and insufficient data registration are common problems in epidemiological studies of soccer injuries (Hägglund *et al.*, 2005).

Using retrospective questionnaires (Lilley *et al.*, 2002) is another method of data collection that can be characterised by inaccurate injury recall and over-estimation of exposure (Nielsen and Yde, 1989; Hägglund *et al.*, 2006). A month after injuries are sustained, under-reporting will start to occur due to recall bias (Inklaar, 1994a). Levy (1988) suggested that retrospective data collected at the end of the season was suspect at best. Junge and Dvorak (2000) quantified the effects of end-of-season retrospective data collection and found that the IFR per player calculated retrospectively was only one third of that calculated on the basis of a weekly follow up. They suggested that the effects of memory as well as psychological defence mechanisms limited the value of retrospective evaluation. Although vulnerable to a number of biases, there are occasions when collecting data retrospectively is the only method available. In professional soccer

however, the medical staff have the opportunity to treat and record injuries from the moment they occur until the day the player is restored to full match fitness. This opportunity should be maximised to collect comprehensive prospective data.

Evidence suggests that population limiting methods of data collection such as video based, third party and retrospective data collection, have weaknesses. It would therefore be appropriate to suggest that the most accurate and precise collection of data can only be obtained by direct supervision and examination of soccer injuries in the field (Poulsen *et al.*, 1991). This approach therefore requires first-hand, prompt, data collection from suitably qualified researchers. This method may not always be feasible but, in the planning stage of the methodology, it should always be the first consideration.

2.1.2.5 Exposure hours

Playing professional soccer has a certain risk of injury. Risk can be expressed as incidence, and can be defined as the number of new cases arising in a defined population during a given period of time (Meeuwisse, 1991). The incidence rate of soccer injuries can therefore be defined as the number of new soccer injuries during a particular time period divided by the total number of soccer players at the start of that period (Lewin, 1989). This definition implies that every soccer player is equally exposed to injury. This is not the case as risk to injury in soccer increases exponentially as time spent participating in competition and training increases. Exposure has therefore to be accounted for to get a true injury rate (Ekstrand *et al.*, 1983b). Having the ability to calculate exposure hours allows results to be reported in terms of injuries per time exposure unit. This can then be reported as an injury frequency rate.

Exposure to risk in soccer is often reported separately in hour's exposure to training and competitive matches. Match exposure has been reported using both estimated and individually timed hours. When estimating match exposure, a popular method is to multiply the number of players per team (e.g. 11) by 100 minutes, to allow for time added at the end of each half (Hawkins and Fuller, 1999; Rahnama *et al.*, 2002; Yoon *et al.*, 2004). This method fails to take into account extra time, sending offs or injury reducing

teams to 10 players and may result in an over-estimation of exposure and thus an under-estimation of IFR. Others estimate match exposure using 11 x 90 minutes (Inklaar *et al.*, 1996; Hägglund *et al.*, 2003) although this method is less common. Fuller *et al.* (2006) recommended individual exposure for match play if the study is investigating relations between the incidences of injury and individual risk factors. This method would likely result in an injury frequency rate that was more accurate and relationships between factors which were more dependable.

Training exposure is also widely reported in the literature (Lűthje *et al.*, 1996; Peterson *et al.*, 2000; Morgan and Overlander, 2001; Hägglund *et al.*, 2005; Árnason *et al.*, 2005). There is, however, insufficient methodological information to compare methods of calculating training exposure and to arrive at a methodology that may be effective. Methodological details of collecting training exposure range from no detail (Hoff and Martin, 1986), recorded by the coach (Árnason *et al.*, 1996) and calculated by multiplying the number of players training by the typical daily training session (Hawkins and Fuller, 1999; Jacobsen and Tegner, 2007). Only one study has attempted to validate estimated training exposure by comparing it with accurately timed exposure (Hägglund *et al.*, 2003). The methods of calculating training exposure were altered across two separate seasons (1982 and 2001) with estimated time (90 min) used in 1982 and accurately timed individual exposure employed in 2001. They suggested that the estimated training exposure of 90 min was a valid assumption because the training sessions for the accurately timed exposure were on average 87 min. A significant playing level change from semi-professional to full-time professional occurred at the club between the two dates so this comparison is not as valid as would first appear. There have been other studies that stated that accurately timed individual players' exposure during training sessions were recorded (Ekstrand and Karlsson, 2003; Hägglund *et al.*, 2003; Kofotolis *et al.*, 2007) but, once again, no detail of methods used to collect this information were published. If accurately timed training exposure is going to be reported, it is important that individual differences in training duration are accounted for. These include players finishing training prematurely or remaining after the majority of other player for additional practice or fitness training. Although the impact of these individual differences

on total exposure time is thought to be minimal (Fuller *et al.*, 2006), no work has been completed to confirm this.

2.1.2.6 Timing injuries

The time of occurrence of match related injuries has been extensively investigated. A number of studies have reported more injuries in the second half than the first half although sometimes this difference does not reach statistical significance (Junge *et al.*, 2006). There have been a number of studies that have segmented competitive matches into 6 x 15 min periods (Rahnama *et al.*, 2000; Andersen *et al.*, 2004a; Árnason, 2004c) or 4 equal quarters (Hawkins and Fuller, 1998a) and compared the IFR during each. Hawkins and Fuller (1998a) attempted to pre-empt time added on at the end of each half by making the first quarter from kick off until 25 min and the second quarter 25 min until half time. If no time is added on at the end of the half, the first quarter is 25% larger than the second making subsequent interpretation of the data questionable as the duration of each period under scrutiny are not equal. The time that can be added on is an unknown variable and it seems more prudent to have it incorporated in the final segment in each half. Identifying temporal peaks in injury incidence may provide possible indicators when investigating injury causation factors and should therefore be included in epidemiological studies. The six-segment method appears to be the most universal method of timing injuries and allows for injuries to be more precisely timed than the four-quarter method.

2.1.2.7 Severity of injury

Closely related to the issue of injury definition is the rational estimation of injury severity (Dvorak and Junge, 2000). In general, the severity of injury is defined according to the length of time that the player is unavailable to train or play competitive matches (Nielsson and Yde, 1989). Severity of injury can also be described in terms of the nature of the injury, tissue damage, and medical costs (Inklaar, 1994a). Table 2.1.1 illustrates the range of time-loss injury severity definitions that have been used in literature. When examining these data, it would appear that the discrepancies between studies, with regard to severity, are focussed on the early stages of the injuries. Most studies comprise of a

moderate and major category of 1 to 4 wks and > 4 wks respectively. Injuries of <1 wk are analysed with a less consistent approach.

Table 2.1.1 Severity of injury classifications utilised in soccer injury studies.

Study	Injury definition	Slight	Minor	Moderate	Major
Ekstrand & Gillquist (1983b)	1 session		< 7d	7-28 d	>28d
Ekstrand *et al.* (1983b)	1 session		1-7 d	8-28d	>28 d
Nielsson and Yde (1989)	1 session		1-7 d	8-28d	>28 d
Engström *et al.* (1990)	1 session		<7 d	7-28d	>28 d
Lüthje *et al.* (1996)	Treatment		< 7d	7-28 d	>28d
Junge *et al.* (2000)	>7 d		7-14 d	15-28 d	>28 d
Hawkins *et al.* (2001)	>48 h	2-3 d	4-7 d	8-28 d	>28 d
Hägglund *et al.* (2003)	1 session	1-3 d	4-7 d	8-28 d	>28 d
Ekstrand *et al.* (2004a)	1 session		1-7 d	8-28d	>28 d
Faude *et al.* (2005)	1 d		< 7d	7-28 d	>28 d
Waldén *et al.* (2005)	1 session	<3 d	4-7 d	8-28 d	>28 d
Le Gall *et al.* (2006)	>48 h	2-3 d	4-7 d	8-28d	>28 d
Junge *et al.* (2006)	Treatment	1-3 d	4-7 d	8-28d	>28 d
Hägglund *et al.* (2006)	1 session	1-3 d	4-7 d	8-28 d	>28 d

In their extensive study of English professional players over 2 competitive seasons, Hawkins *et al.* (2001) defined severity of injury as slight, minor, moderate, or major depending on whether the player was absent from training or competition 2 to 3 d, 4 to 7 d, 8 to 28 d, or >28 d respectively. This is one of the studies that have more than one category for injuries resulting in absence of less than one week. Having a minor category which encompasses all injuries resulting in absences from 1 d to <7 d does not allow for differentiation between minor knocks and those injuries that result in the player missing the whole of a training week. For example, a player who misses 1 d training and a player missing every training session prior to recovering and participating in a competitive match would be allocated to the same severity category. This differentiation may be

useful when analysing re-injury rates and should therefore be included in severity categories.

Major injuries are universally defined as injuries resulting in >28 d absence (Table 2.1.1). Having the most serious injury criterion set at 28 d however, has the potential to coalesce a 35-d hamstring injury and a 52-wk cruciate ligament reconstruction. Categorising the severity of injury is subjective and may be driven by the aims of the study, but an injury severity category that would identify lengthy and possibly career threatening injuries may better reflect the range of injuries sustained in soccer. A category to reflect injuries resulting in months of absence may therefore improve understanding of major injuries in soccer.

2.1.2.8 Categorising traumatic and overuse injuries

Most soccer injuries occur in a traumatic nature (Hawkins *et al.*, 2001), for example an impact injury as a result of being tackled or a muscle strain injury while sprinting for the ball. Traumatic injuries are relatively simple to categorise as there is an event which can be recalled. There are however, a proportion of injuries caused by overuse factors. The percentage of overuse injuries reported in available literature varies from 6% (Lüthje *et al.*, 1996) to 37% (Waldén *et al.*, 2005). It is assumed that the definition of overuse injury is well known and indisputable (Dvorak and Junge, 2000) but this range in the incidence of overuse injury suggests this assumption is ill conceived. The following definitions have been suggested and illustrate the ambiguity when diagnosing an overuse injury;

> "Any pain syndrome of the musculoskeletal system with insidious onset and without any known trauma or disease that might have given previous symptoms" (Orava, 1980).
>
> "Any pain syndrome of the musculoskeletal system appearing during physical exercise without any known trauma, disease, deformity of anomaly that might have given previous symptoms" (Lüthje *et al.*, 1996).
>
> "Any injury as a consequence of repetitive microtraumas" (van Mechelen *et al.*, 1992)

These varied definitions may be the reason for the discrepancy between the percentages of overuse and traumatic injuries reported in the literature. The definition of 'any injury as a consequence of repetitive microtraumas' (van Mechelen *et al.*, 1992) is seldom used but it relates to the clinical understanding of overuse injury. It is however, because of its brevity, open to interpretation. The other two definitions (Orava, 1980; Lüthje *et al.*, 1996) are similar and are based on the absence of a traumatic event. Both definitions also state that the injury may or may not have produced symptoms prior to the point at which the athlete had to cease participation. It is likely that an injury, without previous symptoms, which prevents a player from participating would be categorised, correctly or incorrectly, as a traumatic injury. Examples of the more common overuse conditions are stress fractures, lower leg compartment syndromes (shin splints), achilles tendinosis, adductor tenosynovitis and apophyisitis conditions such as Osgood Schlatter's disease and Sever's disease. Each of these conditions present with symptoms prior to the stage at which the player is unable to continue participating in physical activity. Overuse injuries of severity sufficient to inhibit physical activity are therefore typically characterised by previous symptoms (Slobounov, 2008). An alternative definition to those offered above is therefore suggested;

'Any pain syndrome of the musculoskeletal system with insidious onset and without any known trauma or disease that has given previous symptoms that were initially insufficiently severe to inhibit participation but, over time, the severity increased resulting in the inability to train or play'.

This definition makes the diagnosing of overuse injuries more unambiguous and will be adopted throughout the scientific investigations in this thesis.

2.1.2.9 Intrinsic and extrinsic injuries

In general, the distinction has been made between so-called intrinsic (person related) and extrinsic (environment related) risk factors (Inklaar 1996; van Mechelen *et al.*, 1992; Taimela *et al.*, 1990). The intrinsic risk factors relate to the individual biological and psychosocial characteristics of a person, such as age, joint instability, muscle strength,

muscle tightness, muscle strength and symmetry, previous injuries, adequacy of rehabilitation, and psychosocial stress (Dvorak *et al.*, 2000). Extrinsic risk factors relate to environmental variables such as the level of play; exercise load (amount of competition and practice); amounts and standard of training; position played; equipment such as shin guards, taping, and shoes; playing field conditions; rules; and foul play. Both intrinsic and extrinsic factors can partially influence each other and are therefore not independent of each other (Dvorak *et al.*, 2000). For example, a player who straps an ankle in an effort to reduce the intrinsic risk of a chronically unstable ankle joint may be more prone to ankle injury from extrinsic factors such as being tackled by an opponent. A distinction between intrinsic and extrinsic causative factors may be able to categorise avoidable risk factors and should therefore be included in epidemiological studies. The guidelines of Dvorak *et al.* (2000) on categorising extrinsic and intrinsic injuries factors should therefore be adopted.

2.1.2.10 Reporting the incidence of injury

The incidence of injury has been defined as the number of injuries occurring during the study period (Inklaar, 1994a). Using this approach, previous studies have reported injury in terms of a ratio of new injuries to the total number of subjects (Chomiak *et al.*, 2000). This enables incidents per squad to be reported and then subsequently analysed for variables such as injuries per calendar month, player position and age group (Lewin, 1989; Hawkins *et al.*, 2001; Woods *et al.*, 2003). Methods such as reporting injuries per calendar month may however be limited in their generalisability as they assume an equal number of games per month throughout the season (Sandelin *et al.*, 1985). As a result of most soccer seasons in England beginning mid August and finishing early May, together with weather considerations and cup commitments, this may not be the case. The use of absolute numbers of new injuries and reporting incidence in injuries/population ratio or calendar months may therefore have limited value when determining risk as they will lack a time-at-risk component (exposure) (Lindenfeld *et al.*, 1988).

Frequency based assessments of injury only describe the likelihood that an injury will occur, whereas risk is a function that takes account of both probability and the

consequences resulting from an adverse event (Drawer and Fuller, 2002). The number of participants and the time spent at training and competition are therefore essential epidemiological variables, as they allow the expression of incidence rates per time unit exposure (de Loës, 1997). Injuries are often expressed as an injury frequency rate (IFR) by calculating the number of new injuries to time exposure to the risk of playing soccer matches and training participation (Inklaar, 1994a). The risk denominator for IFR of 1000 playing or training hours appears to be the accepted standard in recent epidemiological studies as it allows comparison with other sports and occupations that utilise the 1000 h exposure denominator (Hägglund *et al.*, 2003; Hawkins and Fuller, 1999; Junge *et al.*, 2004; Hägglund *et al.*, 2005). This denominator also allows comparison with other high-risk occupations (Fuller and Hawkins, 1997).

There are several methods of reporting incidence of injury but the highest and most desirable level of data is that in which time at risk is considered (de Loës, 1997). Recording individual exposure without estimations is crucial to be able to study the actual risk of injury (Waldén *et al.*, 2005). This should be reported, irrespective of the definition of injury, as the number of injuries per 1000 exposure hours.

2.1.2.11 Summary

Injuries in professional soccer are expensive and time-consuming, and thus, preventative strategies and activities are justified on medical as well as economic grounds. Successful injury surveillance and prevention requires valid pre- and post-intervention data on the extent of the problem. The aetiology, risk factors and exact mechanisms of injuries need to be identified. The measure or program for preventing soccer injuries, must include the standardised definition of the injury and its severity, a systematic method of collecting information as well as measurements of the outcome. Valid and reliable measurements of the exposure including exact information about the population at risk and the exposure time are required. However, the subjective component of the injury cannot be ignored, as in almost every study it is the player who decides whether he is injured or not, whether he will report the injury, and when he is ready to return to competition (Ekstrand and Gillquist, 1983b).

In all studies to date, researchers have also been confronted with different methodological and practical problems such as access to players' medical records and data collectors being part-time coaching staff etc. This may compromise the design of methodologies and therefore lead to possible discrepancies between actual and recorded data. In an effort to remedy such issues and quantify the effect they may have on data analysis, a data collection procedure that is not prone to the above limitations is required. To achieve this, every incident resulting in medical attention needs to be immediately dealt with and recorded. Precisely timed and recorded individual player exposure for training and competitive matches is also essential. Such meticulous methods will result in an accurate, reliable and wide-ranging injury surveillance procedure that will produce a valid and reliable information base. These methods will produce the necessary benchmark data for establishing effective injury surveillance strategies which are sufficiently sensitive to monitor the effects of injury prevention interventions. To date few studies have been undertaken with this degree of access, scope and accuracy of injury data collection.

2.1.3 The epidemiology and aetiology of soccer injuries

2.1.3.1 Background

Section 2.1.2 highlighted the problems of comparing methodological approaches in epidemiological studies of injuries in professional soccer. It is therefore apparent that the outcome (results) of these studies in terms of epidemiology and aetiology must also be difficult to compare. There have therefore, been a number of discrepancies in the reported frequencies and types of injuries in the literature as a consequence of different ways of obtaining injury information (Poulsen *et al.*, 1991).

Notwithstanding these difficulties, the recurrent factors influencing soccer injuries need to be identified before any model aimed at reducing the effects of factors affecting injury can be initiated. The conceptual model most commonly applied to epidemiological research of soccer injuries is a stress/capacity model (van Mechelen *et al.*, 1992). In this model, internal (personal) and external (environmental) aetiological factors are identified. These factors must be in balance. Preventative measures must then be designed to achieve or maintain this balance for each individual player. Merely establishing the epidemiological factors such as incidence and prevalence is, however, inadequate if injuries are to be prevented as the aetiology, including causative factors, by which soccer injuries occur must also be identified. The following section therefore evaluates the available literature to examine the epidemiological and aetiological factors of injuries in professional soccer.

2.1.3.2 Frequency of injury

Investigations into the number of soccer injuries in a given period (e.g. one season) tend to be concentrated into earlier studies (Lewin, 1989; McGregor and Rae, 1995). It is however, well documented that studies lacking a time at a risk element are of little value (de Loës, 1997). This has been addressed in later studies such as Peterson *et al.* (2000) and Junge *et al.* (2006). Excellent information on the characteristics and causes of soccer injuries in male professional soccer players has however been presented more recently by Hawkins *et al.* (2001) in their analysis of 6030 injuries in 91 English professional soccer clubs over 2 seasons. These figures corresponded to an average of 1.3 injuries per player

per season. The mean (±SD) number of days absent for each injury was 24 (± 40), with 78% of the injuries leading to a minimum of one competitive match being missed. Competition injuries represented 63% of those reported with significantly ($p<0.01$) more of these injuries occurring towards the end of both halves of the games. August was the period when the greatest number of match injuries occurred ($p<0.05$) suggesting that this may have been a result of players not being physically prepared for the demands of competition. This evaluation may be compromised by the start of the English season being characterised by frequent matches. One of the limitations of the study was the absence of match exposure data which may have reflected the frequency of matches and therefore questioned the suggestion of players being ill prepared. Another reason may have been that the data collectors were still enthusiastic about returning injury registration data for the less serious injuries. Unfortunately, Hawkins *et al.* (2001) did not publish a breakdown for severity of injuries per calendar month so this suggestion cannot be confirmed.

2.1.3.3 Injury frequency rates

Several epidemiological surveys have reported the frequency of injuries in soccer per time unit. Table 2.1.2 illustrates the range of IFR's for training and matches in studies using professional soccer player cohorts. The studies have been grouped so that IFR for similar definitions of injury can be compared. The 73 injuries per 1000 h reported in Olympic soccer by Junge *et al.* (2006) appears to be significantly higher than that reported in professional soccer by others. The injury definition of 'any physical complaint incurred during a match that received medical attention from the team physician, regardless of the consequences with respect to absence from the match or training' may have been the reason for the high IFR in this investigation. Calculating an IFR from their published figures using a definition of 'missed at least one training session or match' resulted in an IFR of 29 injuries per 1000 h which is similar to the other published investigations. This analysis clearly demonstrates the importance of considering the definition of injury when comparing studies for IFR.

Table 2.1.2 Injury frequency rates in studies with adult males as the subjects

Study	Population and sample size	Study period	IFR injuries/1000h	
			Training	Match
Injury definition; Soccer related incident that resulted in the need for treatment				
Inklaar *et al.* (1996)	101 high level Dutch amateurs	2nd half of a season		21.7
Lüthje *et al.* (1996)	263 top Finnish div. professionals	1 season	1.5	16.6
Peterson *et al.* (2000)	21 professionals	1 year	6.5	18.6
Junge *et al.* (2006)	32 Olympic soccer matches	1 Olympics		73
Injury definition; Soccer related incident that resulted in at least 1 session missed				
Nielsen and Yde, (1989)	34 Danish professionals	1 season	2.3	18.5
Engström *et al.* (1990)	64 Swedish professionals	1 season	3	13
Ekstrand and Tropp, (1990)	135 top Swedish division	1 year	4.6	21.8
Poulsen *et al.* (1991)	19 Danish division 1 professionals	1 year	4.1	19.8
Árnason *et al.* (1996)	84 Icelandic professionals	1 season	5.9	34.8
Hawkins and Fuller, (1999)	108 English professionals	3 seasons	3.4	25.9
Ekstrand *et al.* (2004b)	Swedish international players	6 seasons	6.5	30.3
Yoon *et al.* (2004)	411 Asian internationals	1 tournament		45.8
Waldén *et al.* (2005)	310 Swedish elite professionals	1 year	5.7	27.2

The distinction between the incidence of injury in competitive games and practice has been made in several studies (Ekstrand *et al.*, 1983b; Nielsen and Yde, 1989; Ekstrand and Tropp, 1990; Engström *et al.*, 1990). It is well established that the IFR in competitive matches is significantly higher than in training (Table 2.1.2). This is probably because matches involve more intense play and more contact situations than practice (Ekstrand and Gillquist, 1983b). The frequency of injury in training does however appear to be dependent on the coaches' perception of how competitive he expects players to be during training sessions. In their study of severe injuries (absence >4 wks), Chomiak *et al.* (2000) reported that 20% of the injuries reported to have been as a result of foul play were sustained in training. No detail was given as to making the decision on the presence of foul play.

Another factor which affects the incidence of training injuries is when researchers combine pre-season fitness preparation periods and in-season practice exposure (Ekstrand *et al.*, 1983b; Hawkins *et al.*, 2001). Ekstrand *et al.* (1983b) separated pre-season and in-season training and reported an IFR of 21±15 injuries per 1000 h during the pre-season preparation period. The IFR for in-season practice was 8 injuries per 1000 h and for matches 17 injuries per 1000 h. Hawkins *et al.* (2001) reported that training injuries peaked during July ($p<0.05$) which, in England, corresponds to the pre-season preparation period. However, in that study the actual risk of injury is unknown due to absence of exposure data.

Pre-season training periods are by definition, dissimilar to in-season training. Pre-season training is characterised by extended aerobic work and, in the early stages, very little work with the ball (Dunbar, 2002). Pre-season friendly matches are also of a lower competitive nature. Decisions on whether a player with a minor injury, such as a contusion, should continue in a match or training may differ considerably between pre-season and the competitive season. The subjective element of the decision regarding player participation is therefore greater during pre-season when coaches and players may be more concerned with not aggravating an injury than the result of the match. Reporting IFR as a combination of in-season training and pre-season preparation work is therefore questionable and it is reasonable to suggest that pre-season periods and competitive seasons are two, very different variables and, as such, studies into each should be conducted separately.

2.1.3.4 Timing of injuries

Efforts have been made to identify temporal periods in competitive matches when risk of injury is greater than other periods. More injuries are sustained in the second half of matches than in the first half according to Hawkins and Fuller (1999) and Hawkins *et al.* (2001). Sometimes this trend does not reach statistical significance (Junge *et al.*, 2006). The timing of injury has been more accurately reported by Hawkins and Fuller (1998a). In their work comparing the frequency of injury between three levels of professional soccer, they divided matches into 4 equal quarters. Their data, which covered competitive playing levels from international matches at the World Cup to English division one games, showed no obvious pattern of injuries throughout any of the four quarters at any level of competition. These findings were similar to those of Sandelin *et al.*, (1985) who reported the percentage of injuries in each quarter to be 31, 22, 37 and 20% respectively. The percentage of injuries in the final quarter was not significantly lower than the other three quarters.

To identify the timing of injuries more accurately than four quarters, competitive matches have been segmented into 6 x 15 min periods and the frequency of injury compared between each (Rahnama *et al.*, 2002; Hawkins *et al.*, 2001; Rahnama *et al.* 2004). This method provides a more accurate representation of when injuries occur. Rahnama *et al.* (2002) reported no significant difference in frequency of injury between any of the 6 x 15 min segments within a game. The 'potential' for minor injury however, was greater in the first 15 min of each half with the potential for moderate and major injuries being higher in the final 15 min of competitive matches. This study concentrated on incidents involving the ball during 10 English Premier League matches. The small number (n=20) of actual injuries observed, with only 4 having to leave the field of play prematurely, does not allow inferences to the wider population. In a later study, Rahnama *et al.* (2004), observed a significantly greater frequency of actions with the potential for injury in the final 15 min of the first half compared to other periods of the game. These findings however, were based on subjectively determining whether one of 16 actions with potential for injury had occurred and recording them using a hand notation system. The number of matches analysed was one home and one away English Premier League match. To confirm these findings, analysis of more matches is required. The findings in the English FA audit of injuries in professional football, (Hawkins *et al.*, 2001), agreed with those of Rahnama *et al.* (2002) with significantly more injuries were observed during the final 15 min of each half of the match.

Differences in intensity and competitiveness at various stages of matches have been implicated when injury peaks have been reported (Rahnama *et al.*, 2002; Hawkins *et al.*, 2001). There are a greater number of incidences with the potential for both moderate and major injury in final 15 minutes of competitive matches (Rahnama *et al.*, 2002). Player fitness has also been associated with injuries occurring later in matches (Hawkins and Fuller, 1999). This increase in risk may be associated with fatigue of the muscles and other body organs as muscle glycogen stores near completion (Saltin, 1973). Rahnama *et al.* (2003) examined both concentric and eccentric leg muscle strength before, during and following a non-motorised treadmill protocol designed to mimic the demands of match play. They observed a progressive reduction in muscle strength that applies across a range of functional characteristics during the protocol. Player's performance is also negatively affected towards the end of matches as a result of hypohydration (Reilly, 1997).

The above observations suggest that, towards the end of a competitive match, there are more incidents with potential of moderate or major injury and also, players are fatigued. At these later stages of the game, even though the players are tired, the contest may still be intense (Rahnama *et al.,* 2002) with players competing during potential match winning situations. Controlling the competitiveness of matches is not a realistic strategy for injury prevention. Coaches may however, be able to affect the incidence of injury as a result of poor player fitness. It is well recognised that well-trained and prepared players are able to bear the physical stress during the game with a reduced risk of injury (Dvorak *et al.*, 2000). These findings highlight the importance of ensuring effective rehabilitation and physical preparation of players returning to competition following lengthy injury-induced absences.

2.1.3.5 Mechanisms of injury

A precise understanding of the mechanism of injury and situations leading to injury in soccer is important to be able to develop effective preventative measures. Although the mechanism of injury in professional soccer has not been widely reported in the scientific literature (Árnason, 2004b) (Table 2.1.3), the consensus in the available information is that player contact is the major cause of injury (Wekesa, 1995; Hawkins and Fuller, 1999; Peterson *et al.*, 2000; Drawer and Fuller, 2002; Yoon *et al.*, 2004). Player contact has been reported to account for 82% (Peterson *et al.*, 2000) and 87% (Yoon *et al.*, 2004) of all match injuries. Rahnama *et al.* (2002) scrutinised videotapes of 10 top division English matches and

identified being tackled as responsible for 50% of minor injuries, 86% of moderate injuries and 100% of major injuries.

In contrast to the above findings, Chomiak *et al.* (2000) reported that a close examination of the mechanism of injury in their study of severe injuries, revealed that there were almost equal proportions of contact (52%) and non contact injuries (48%). This discrepancy is probably explained by the aims of this study concentrating on injuries that resulted in absences of >28 d. This timescale would have eliminated common contact injuries such as muscle contusions and bone bruising which result in shorter absences but which would be included in most other studies of injuries in soccer. In agreement with Chomiak *et al.*, (2000), Nielsen and Yde (1989) reported that running (54%) was the major mechanism to injury. This study is characterised by the author visiting the team once per week and interviewing the injured players. First hand observations may have expanded the limited range of the mechanics to injury reported which only included tackling, running and shooting. Notwithstanding the above two studies, the evidence suggests that, particularly during match-play, non-contact injuries are less common than contact injuries. It would however, appear that non-contact injuries may result in longer absences. Drawer and Fuller (2002) investigated the mechanism of injury within the 5 main injury locations (thigh, knee, ankle, lower leg and groin). They reported that 39% of the total day's absences through injury were lost as a result of contact and 47% through non-contact injuries.

Table 2.1.3 Percentage of injuries as a function of injury mechanism.

Study		Contact	Running	Tackled	Tackling	Twisting/ turning	Stretching	Heading	Landing	Kicking	Jumping	Other/unknown
Injury definition; Soccer related incident that resulted in at least 1 session missed												
Hawkins and Fuller (1999)	(Training)	4	18	28	17	5		2	5	6	2	13
	(match)	1	30	10	5	13		1	5	17	3	15
Nielsen and Yde (1989)			54		30					5		
McGregor and Rae (1995)		16		3	12				12			57
Drawer and Fuller (2002)			17	24	13					12		
Injury definition; Soccer related incident that resulted in at least 2 d absence												
Hawkins *et al.* (2001)		12	19	15	9	8	6	1	4	8	2	18

Although the laws of the game seek to protect players from injury through unfair challenges, fouls are responsible for nearly 30% of on-pitch and 20% of post-match medical reports (Fuller *et al.*, 2004b). Junge *et al.* (2006) reported that out of 77 injuries, 65 (86%) were contact injuries of which 41 (67%) were judged to result from foul play. Andersen et al., (2004c) calculated that 1 in 10 of all fouls awarded by the referee were punished by a yellow or red card. In the study by Junge *et al.* (2006) however, the team physician decided if the contact was in breach of the laws of the game. On examination of their data, there was only a 64% agreement between the team physician and the decision made by the referee. It is recognised that the refereeing process is complex and subject to human error (Fuller *et al.*, 2004a), but referees are qualified to officiate and their subjective opinion of the presence of foul play should be more reliable than using staff influenced by own team bias. As part of the Hawkins and Fuller (1998a) study, videotapes of 178 English professional soccer matches over 3 seasons (1994-97) were analysed. They reported that an average of 68% (average for each season, 65, 76, 63%) of match injuries were caused by player to player contact and that between 1.4 - 3% of tackles resulted in the referee giving a foul and 23% of these rule violations resulted in injury. Junge *et al.* (2004) calculated that 88% of match injuries were caused by player contact and 58% of those contacts were deemed to be foul play. The same authors had similar findings in a later study with 86% of injuries being as a consequence of player contact and 67% of these being deemed foul play (Junge *et al.*, 2006). Player contact is one of the major causes of player injury and therefore sub-dividing this parameter to enable further analysis of type of contact is justified. When including a category to indicate the presence of foul play, the criteria should be based on the referee's decision as to the legitimacy of the tackle, even when this is evidently incorrect.

It is not difficult to identify aetiological factors for the majority of injuries. Ekstrand and Gillquist (1983a) suggested they were able to identify factors which had an influence on 75% of injuries. However, injury is often the result of cumulative variables and it is this complex interaction that makes preventing soccer injuries such a convoluted task. Mechanism of injury is however, the most valuable indicator for injury prevention and, as such, comprehensive injury mechanism data should be included in all studies concerned with the aetiology of injury.

2.1.3.6 Anatomical location of injury

It is evident that the majority of injuries in professional soccer affect the lower extremities with the ankle and knee joints, as well as the muscles of the thigh being the anatomical locations most frequently injured (Fried and Lloyd, 1992; Tucker, 1997; Inklaar, 1994b). Inklaar (1994b) concluded from his review of the literature that 61% to 90% of all injuries occurred in the lower extremities. Goalkeepers, however, sustain more head, face, neck, and upper extremity injuries than lower extremity injuries (Sullivan *et al.*, 1980; Wekesa, 1996). These differences in injury patterns highlight the different physical demands (di Salvo *et al.*, 2007) and level of contact between goalkeepers and outfield players. In his study of Kenyan international players over one year (18 matches), Wekesa (1995) reported that lower extremity injuries accounted for 94% of all injuries. The work in this study was concentrated on international friendly (n=10) and competitive (n=8) matches using an injury definition of any medical treatment administered irrespective of time loss. No differentiation was made between the friendly matches and competitive matches which may have provided additional interesting data for comparison. Table 2.1.4 details the percentage of injuries as a factor of anatomical location of injury in studies using professional soccer player populations.

The highest frequency of knee injury (30%) was observed by Chomiak *et al.* (2000). This figure was higher than all other studies. Chomiak *et al.* (2000) however, concentrated their investigations on severe injuries (absence >28 d). It is well documented that injuries to the knee are more likely to result in longer absences than injuries to most other anatomical locations (Engström *et al.*, 1990). This may have contributed to the high percentage of knee injuries observed in this study. The highest frequency reported for ankle injuries was 30% in Danish division two players (Nielsen and Yde, 1989). Although this is a high percentage compared to most other studies, it represents 11 of the total 37 injuries observed during the one-season data collection period. It is also worth noting that 56% of these ankle injuries were sustained when the player was not completely restored from a previous ankle injury. The authors suggested that this was a result of the club coaches putting pressure on players to return to activity prematurely or to continue the game or activity after they had sustained the injury. This may have contributed to the high incidence of ankle injuries reported.

The percentage of thigh injuries reported appears to be influenced by the definition of injury used. Studies that record injuries with no time-loss, or <2 d time-loss, generally report higher percentages of thigh injuries (Nielsen and Yde, 1989; McGregor and Rae, 1995; Lüthje *et al.*,

1996; Waldén *et al.*, 2005). A high proportion of thigh injuries are contusion injuries as a result of contact (Junge *et al.*, 2006). Players with this type of injury may only be absent for 1 or 2 sessions and therefore may not meet the inclusion criteria for studies that analyse injuries with absence >2 d. There are two studies which contradict this hypothesis. Peterson *et al.* (2000) reported a low frequency of thigh injuries (14%) using a definition of injury of all injuries regardless of the consequences with regard to absence. The data collection methods utilised in this study included a weekly visit by the researchers to question players who had sustained injuries. It may therefore be optimistic to expect a player who, 6 days prior to the visit, sustained a 'dead leg' with no time loss, to report the injury. An injury definition with a time-loss component may have been more suitable for this study. The other study which reported a low frequency of thigh injury was Junge *et al.* (2006). They reported that 17% of injuries during the men's soccer tournament in the 2004 Olympic Games were thigh injuries. Examination of the results gives no indication as to why the percentage of thigh injuries may be lower than studies with a similar definition of injury.

Examination of Table 2.1.4 suggests that the percentage of injuries to each anatomical location, with the exception of individual outliers, is reasonably constant. This suggests that the frequency of injuries to each anatomical location reported in the literature is an accurate representation of the injuries sustained in professional soccer. When investigating outliers, there is normally legitimate reasons why the results are dissimilar when comparing what appear to be similar studies. These reasons are typically based around the definition of injury used, the categorising of anatomical locations, methodological variations or study limitations. Examination of the studies in Table 2.1.4 confirms that the majority of injuries in soccer affect the joints of the knee and ankle and the musculature of the thigh.

Table 2.1.4 Percentage of injuries as a factor of anatomical location

Study	Knee	Ankle	Thigh	Shin/calf	Foot/toe	Groin	Hips	Spine/trunk	Head/neck	Shoulder/arms	Other
Injury definition: Soccer related incident that resulted in the need for treatment											
Wekesa (1995)	28	25	34								11
Petersen *et al.* (2000)	18	20	14	10	10	7	0	6	4	5	6
Lüthje *et al.* (1996)	19	17	22	8	8		2	0	0	0	24
Junge *et al.* (2006)	16	12	17	18	4	5			8	5	14
Injury definition; Soccer related incident that resulted in at least 1 session missed											
Hawkins and Fuller, (1999)	15	17	23	15	4	11	3	7	2	3	
Nielsen and Yde, (1989)	11	30	32		8						19
McGregor and Rae, (1995)	23	13	45	4	1	0	1	5	4	3	
Poulsen *et al.* (1991)	23	19	18	2	21	11					6
Junge *et al.* (2004)	13	15	18	17	8	5	1	4	15		5
Yoon *et al.* (2004)	19	20	11	17	6	1	1	14	8		4
Waldén *et al.* (2005)	16	10	23	15	8	16		6	2	0	4
Chomiak *et al.* (2000)	30	19	7	5	4	8		8	0	2	14
Ekstrand & Gillquist (1983b)	20	17	14	12	12	13		5			7
Injury definition; Soccer related incident that resulted in at least 2 d absence											
Hawkins *et al.* (2001)	17	17	23	12	5	10	2	6	0	1	0
Le Gall *et al.* (2006)	14	20	23	5	8	5	2	12		10	1

2.1.3.7 Types of injury

The type of injury, together with the anatomical location, are the most important factors in soccer injury aetiology (Dvorak *et al.*, 2000). The type of injury impacts on the rehabilitation process such that it is the location and type of injury which dictates the type and intensity of training a player is able to perform during rehabilitation. The most common types of injuries in soccer are contusions, sprains, and strains (Dvorak and Junge, 2000). As detailed above, most of these injuries are sustained to the lower extremities compromising the ability to perform weight bearing, soccer-specific exercises. Table 2.1.5 illustrates the types of injury sustained by elite soccer players.

Table 2.1.5 Percentage of injuries as a factor of type of injury

Study	Muscle contusion	Muscle strain	Ligament sprain	Tendon pathology	Laceration	Fracture	Dislocation	Meniscal lesion	Hernia	Other soft tissue	Other
Injury definition; Soccer related incident that resulted in the need for treatment											
Junge *et al.* (2006)	71	6	9	1	4	1	0				6
Injury definition; Soccer related incident that resulted in at least 1 session missed											
Árnason *et al.* (1996)	20	29	22								27*
Hawkins and Fuller, (1999) (Training)	5	53	18		1	4	0			5	14
Hawkins and Fuller, (1999) (match)	24	37	21		2	4				5	7
Engström *et al.* (1990)	13	12	34			4	2			33	2
Nielsen and Yde, (1989)	9	21	49	16		6					
McGregor and Rae, (1995)	9	44	16		1	6	6		3	5	16
Poulsen *et al.* (1991)	8	30	42	9		7					5
Junge *et al.* (2004)	41	23	12	2	7	4	0				11
Ekstrand & Gillquist, (1983b)	20	18	29	23		4	2				4
Yoon *et al.* (2004)	62	10	16		5	0	1				7
Waldén *et al.* (2005)	17	22	14			3	1				41*
Injury definition; Soccer related incident that resulted in at least 2 d absence											
Hawkins *et al.* (2001)	7	37	19	4		4	1	2	2	15	14
Le Gall *et al.* (2006)	30	17	16	8		7	1	2		2	17
Injury definition; Soccer related incident that resulted in at least 3 d absence											
Volpi *et al.* (2004)	29	31	17	10	3	2		2			6

*Overuse injuries classified as other

Table 2.1.5 is characterised by an extended range of muscle contusion injuries (7% to 71%). A high percentage of contusion injuries impacts on the percentage of other injuries and makes comparisons between studies difficult. In the study of the 2004 Olympic Games men's soccer tournament, Junge and colleagues (2006) categorised 55 of 77 injuries (71%) as contusion injuries. Yoon et al. (2004) also reported a high incidence of contusion injuries (62%) during an international Asian tournament and Junge et al. (2004) (41%) during the 2002 soccer

World Cup. The team physicians in each of these studies were responsible for recording all injuries requiring medical attention. They would also have had a responsibility to report any incidents to a player's prospective club emphasising the need for accurate and comprehensive record keeping. It could therefore be expected that some of the injuries recorded were very minor and required minimal, if any, medical intervention. The findings of Hawkins and Fuller (1999) are interesting. They separated training and match play and reported that contusion injuries contributed 5% of training injuries and 24% of match injuries. This highlights the difference in the level of contact between training and match play and emphasises the importance of examining the source activity when comparing studies.

Most injuries sustained in professional soccer are categorised as traumatic but overuse injuries have also been identified as the cause of players being unable to train and play competitive matches. Differences in the interpretation of the term 'overuse injury' have resulted in discrepancies when reporting traumatic and overuse injuries (Dvorak and Junge, 2000). Peterson *et al.* (2000) categorised 23% (n=124) of injuries as overuse in their study of soccer players at different skill levels. Waldén *et al.* (2005) utilised the definition by Orava (1980) when reporting overuse injuries as 37% of all injuries. The majority of these injuries occurred during the pre-season period and included such pathologies as bursitis and tendinitis. No breakdown of overuse and traumatic injuries was reported for the competitive season. Low frequency of overuse injury (7%) has been reported by Hawkins *et al.* (2001). In this study however, the large number of injury mechanism categories made it possible to classify injuries such as tendinitis, thus keeping the frequency of overuse injury low.

2.1.3.8 Severity of injury

The number of studies that have reported the severity of injuries is limited (Table 2.1.6). The available studies are however, fairly consistent in their findings, particularly when the definition of injury is similar. Junge *et al.* (2004) reported a slightly higher percentage of minor injuries resulting in <1 wk absence and a lower percentage of major injuries using a similar injury definition as the study by Morgan and Overlander (2001). As previously discussed (Chapter 2.1.2.7.), the reporting of minor injuries is fraught with problems, especially when volunteers are collecting the data. This may have been the reason for the discrepancies. The reason for the difference in major injuries reported may have been differences in the level of competition between the two studies. The study by Junge *et al.* (2004) used international level competitions while the study by Morgan and Overlander

(2001) investigated injuries during the inaugural season of the American Major League. The high percentage of slight injuries (<3 d, 90%) reported by Junge *et al.* (2006) confirms the previous supposition that meticulous data collection may have been the reason for the high percentage of contusion injuries reported.

Table 2.1.6 Percentage of injuries categorised as a factor of injury severity

	Severity of injury				
	<3d	3-7 d	1-7d	8-28 d	>28 d
Injury definition; Soccer related incident that resulted in the need for treatment					
Morgan and Overlander (2001)			59	28	13
Junge *et al.* (2004)	55	25	70	17	3
Junge *et al.* (2006)	90	4		3	3
Injury definition; Soccer related incident that resulted in at least 1 session missed					
Drawer and Fuller (2002)	14	35	49	38	12
Waldén *et al.* (2005)	32.5	27.5	60	30.5	9.5
Hägglund *et al.* (2006)	30	27	57	33	10

2.1.3.9 Re-injury

Re-injury is a major problem in professional soccer with re-injuries often being more serious than the index injury (Hawkins *et al.*, 2001). Árnason *et al.* (1996) reported that the frequency of re-injury was 35% with 44% of muscle strains and 58% of ligament sprains being registered as re-injuries of the same type and location. Hawkins and Fuller (1999) also stated that it was predominantly muscular strains and ligamentous sprains responsible for a re-injury rate of 22% in their cohort of English professional soccer players. Nielsen and Yde (1989) found that 25% of players who were injured had not fully recovered from a previous injury which was the same type and location. The same authors observed that in 42% of injured players, there had been an injury of the same type and location in the previous year. These data confirm the opinion of others that previous injury and inadequate rehabilitation are the most important well established risk factors for re-injury (Ekstrand and Gillquist, 1983a; Ekstrand and Gillquist, 1983b; Ekstrand and Tropp, 1990; Chomiak *et al.*, 2000).

When analysing the available data, it would seem that certain anatomical locations may be more prone to re-injury. Ekstrand and Gillquist (1983a) attributed 17% (n=44) of their re-injuries to inadequate rehabilitation. They highlighted inadequate knee rehabilitation

following knee ligament injury that left players with persistent mechanical instability as a major cause of re-injury. These findings were later confirmed by Waldén *et al.* (2006). Previous injuries to the ankle ligaments also predispose players to subsequent ankle injuries (Ekstrand and Gillquist, 1983b). Ekstrand and Tropp (1990) calculated that a previous ankle ligament sprain increased the risk of an identical injury by 2.3. This appears to be confirmed by Árnason *et al.* (1996) who reported that 69% of players sustaining ankle injuries had a prior history of ankle injuries. In the study by Nielsen and Yde (1989), every player who sustained an ankle sprain during the study period reported that they had suffered an ankle sprain previously. Some of these findings may be the result of mechanical or functional instability of the ankle joint which, without external prophylactic taping or orthosis, may be difficult to improve sufficiently to reduce the risk (Tropp *et al.*, 1984). However, some of these re-injuries could be as a result of the failure of the rehabilitator to fully restore timing and neuromuscular coordination (Ekstrand and Gillquist, 1983b). Effective rehabilitation may therefore have a role in reducing the incidence of re-injuries of the knee and ankle joints.

Previous injury has also been shown to increase the risk of further injury (Ekstrand *et al.*, 1983a). Using a definition of 'any tissue damage caused by football regardless of the consequences with regard to absence', Peterson *et al.* (2000) reported that 80% of players were injured during a competitive season with 64% sustaining a further injury. Remarkably, 6% of the 216 players in that study were injured on more than five occasions in one season.

Inadequate rehabilitation can leave players susceptible to re-injury and further injury on return to play as a result of a number of factors. As early as 1970, Burkett (1970) identified muscle strength imbalance to predispose soccer players to injury. He found that players with more than 10% difference in isokinetic knee flexion strength bilaterally were more susceptible to hamstring strains in the weaker limb. Non-contact knee injuries have demonstrated reduced muscle strength in the injured leg (Ekstrand and Gillquist, 1983a). They reported that significantly more players who had sustained non-contact knee injuries demonstrated reduced knee-extensor strength in the injured leg compared to uninjured players ($p<0.05$). No other differences in strength indices between injured and uninjured players were shown in this study.

Muscle flexibility asymmetry also appears to contribute to the risk of injury in soccer (Agre and Baxter, 1987). The authors suggested that asymmetry in hamstring flexibility

predisposed individuals to low back injuries. Ekstrand and Gillquist (1983a) reported a correlation between tight muscles and strains of the adductor muscles. Significantly more players with tight adductors (n=34) sustained adductor rupture or tendinitis than those without muscle tightness (n=10). In the same study, no significant influence of range of motion was found with regard to hamstring strains. In complete contrast to these findings, Witvrouw *et al.* (2003) reported that players with a hamstring (p=0.047) or quadriceps muscle injury were found to have significantly lower flexibility in these muscles before their injury compared with the uninjured group. No significant differences in muscle flexibility were found between players who sustained an adductor injury and the uninjured group ($p>0.05$). The lack of statistical power (38%) as a result of the low number of adductor muscle injuries (n=13) in the Witvrouw *et al.* (2003) study or differences in methods of establishing range of motion may have been responsible for the discrepancies between studies. Poor muscle strength and flexibility may therefore have an impact when rehabilitating injured soccer players as failure to re-establish muscle strength and flexibility may leave players predisposed to re-injury.

The high rate of re-injury in soccer suggests inadequate rehabilitation and incomplete healing as risk factors. One of the most important considerations in the treatment of soccer injuries is to fully rehabilitate the injured player (Fried and Lloyd, 1992). Re-injury may occur at the same anatomical location due to sequelae from the original injury. These residual pathologies may include persistent pathological ligamentous laxity, proprioception deficit, decreased muscle strength causing muscle imbalance and localised scar tissue causing persistent discomfort (Engström and Renström, 1998). The occurrence of each of these sequelae however, can be reduced by effective treatment and rehabilitation including allowing sufficient time for full tissue healing and returning the player to the level of physical and physiological readiness required for training and competition.

2.1.3.10 Player position

The incidence of injuries and player position has also being investigated. Several studies have found no significant differences in IFR between players' playing position (McMaster and Walter, 1978; Ekstrand and Gillquist, 1982; Sandelin *et al.*, 1985; Hoff and Martin, 1986; Nielsen and Yde, 1989; Hawkins and Fuller, 1998a; Chomiak *et al.*, 2000). An earlier study by Hawkins and Fuller (1996) however, found that defenders were significantly more likely to sustain an injury than other playing positions. Sullivan *et al.* (1980) suggested it was

goalkeepers who were more likely to be injured due to the level of contact during a high proportion of their actions in matches. Wekesa (1995) reported a higher frequency of injury for midfielders (50%), with defenders (25%), strikers (19%) and goalkeepers (6%) being less likely to sustain injury. This was however a descriptive study with no statistical analysis performed so statistical significance is unknown. It would appear that there is no consensus as to the playing position which carries the highest risk for injury.

The weakness with most of the above studies is that no allowance was made for the number of players playing in each position during matches. For example, one of the most common playing formations is 1 goalkeeper; 4 defenders; 4 midfielders and 2 strikers. If the injury risk were the same among the different positions, the expected distribution would be 9% for goalkeepers, 36% for defenders, 36% for midfielders, and 18% for strikers (Árnason, 2004c). Reporting frequency of injury per player position and drawing conclusions as to level of risk is therefore flawed. Árnason (2004c) allowed for the number of players in each position and, reported that the relative proportion of injuries and incidents to be higher in midfielders and lower in goalkeepers and strikers. Further research is required to confirm whether player position is an influencing factor of injury traumatology and should therefore be incorporated into the design of epidemiological research.

2.1.3.11 Match venue and incidence of injury

It has been suggested that there is an increased risk to injury when playing at an away venue than when playing at home (Ekstrand *et al.*, 2004b). Rahnama *et al.* (2002) investigated the number of playing actions with potential for injury and actual injuries when teams were playing at home and away. They found that, although actual injuries for away teams (n=13) were more frequent than for home teams (n=7), the difference did not reach significance. Actions with potential for injury were also not significantly different between home and away teams. In a later study based at one English Premier League club (Rahnama *et al.*, 2004), a greater number of 'actions with potential for injury' were noted when teams were playing away from home despite there being no differences in actual injuries sustained. Playing at home or away also made no difference to the incidence of injury in the Swedish national team (Ekstrand *et al.*, 2004b). The above data suggest that, although playing away from home results in a team being involved in a greater number of incidents with a potential for injury, there is little difference in actual injuries sustained between playing at home or away.

2.1.3.12 Age

The age of soccer players has been reported to have an effect on the incidence and severity of injury (Keller *et al.,* 1987; Tucker, 1997; Árnason *et al.*, 2004b). This has been demonstrated in a number of studies that observed that professional players sustained significantly more injuries than their apprentice counterparts (Nilsson and Roaas, 1978; Roaas and Nilsson, 1979; Keller *et al.,* 1987; Hawkins and Fuller, 1999). This difference in the risk to injury may be explained in part by a difference in training exposure hours, as well as greater ball and body momentum, and greater intensity and competitiveness in professional soccer (Keller *et al.,* 1987). Analysing whether player's age within adult professional soccer impacts on the risk to injury is a little more difficult due to the limited number of studies which have investigated age as a possible risk factor. An opportunity to publish a definitive statement as to the effects of age and injury in professional soccer was not taken by Hawkins *et al.* (2001). They grouped 2376 players into 4 age-distribution categories (17-22, 23-28, 29-34 and 35+) in their study of injuries in English professional soccer over 2 seasons, but no reference was made to age in the results or the discussion. It may be the case that no significant differences were found so they decided to concentrate on other areas of risk. Subsequent associated studies, using the same data set, however, did give some indication to the effects of age on injury. Woods *et al.* (2002) examined the injuries sustained throughout the pre-season periods and found that older players were less likely to be injured during this training period than younger players. In a later study, players in the older age groups were however, more susceptible to hamstring strains than younger players (Woods *et al.,* 2004). Others have found no link between a player's age and the risk of injury (Morgan and Overlander, 2001; Hägglund *et al.*, 2006). Although Hägglund *et al.* (2006) found no significant impact of age on overall risk to injury, they agreed with the findings of Woods *et al.* (2004) that older players were more susceptible to hamstring injuries.

The link between age and the overall risk of injury is inconclusive. There may however, be specific pathologies, such as hamstring strains, or periods of training, such as pre-season preparation, when age has a greater impact on the risk of injury. These increased risk factors are therefore worthy of consideration when examining the demographic distribution of a club's playing squad with a view to implementing measures to reduce the incidence of injury.

2.1.3.13 Summary

The literature shows that injuries in soccer are common with the risk of injury many times that of recognised high-risk industries. Contact during competitive matches is the main mechanism for injury with this contact often contravening the laws of the game. Injuries to the joints of the knee and ankle, as well as muscle injuries to the thigh, are the most common anatomical location for injuries. Muscle strains and ligament sprains are the most common type of injury observed. Although injuries in soccer are common, it has been considered that many injuries could be prevented (Junge *et al.*, 2002). Investigations have identified chronic joint instability, muscle strength imbalance and muscle flexibility asymmetry as risk factors to injury and previous injury (Ekstrand and Gillquist, 1983a) and inadequate rehabilitation (Dvorak *et al.*, 2000), as major risk factors in the high incidence of re-injury. The presence of these factors is confirmed by the high percentage of re-injuries reported in the literature. Although the identification of possible risk factors is a fairly straight forward process, the identification of individual risk factors can only partially begin to address the question of injury prevention. It is clear that soccer injuries result from a complex interaction of risk factors (Hägglund *et al.*, 2007). Preventing injury is therefore a multifaceted task with complex causal pathways.

2.2 DETRAINING: THE PHYSIOLOGICAL EFFECTS AND CONSEQUENCES ON PERFORMANCE IN SOCCER PLAYERS

2.2.1 Introduction

Soccer is a physically demanding game involving anaerobic periods superimposed on a background of endurance running. The predominant energy demands in soccer are aerobic in nature (Drust *et al.*, 1998). The average level of maximal oxygen uptake ($\dot{V}O_{2max}$) in professional soccer players is about 60 $ml \cdot kg^{-1} \cdot min^{-1}$ (Stølen et al., 2005), but individual figures as high as 73 $ml \cdot kg^{-1} \cdot min^{-1}$ have been reported (Apor, 1988). A moderate to high level of aerobic fitness is therefore important for outfield success in soccer (Shepherd and Young, 2006). The total distance covered by soccer players at the elite level is around 10 km for outfield players (Drust *et al.*, 1998). Distance covered however, can fluctuate for a single player by up to 1 km (Bangsbo *et al.*, 1991a). This fluctuation is believed to be affected by the level of the opposition, the importance of the match, player position, self pacing and fatigue as the game progresses (Bangsbo *et al.*, 1991a; Rico-Sanz, 1999; Mohr *et al.*, 2003; Di Salvo *et al.*, 2007).

As well as distance covered in a match, level of play in soccer is also characterised by the number of changes in intensity of activity with players from higher divisions of national leagues performing longer and more frequent high-intensity bouts (Drust *et al.*, 1998). Bangsbo *et al.* (1991a) and Yamanaka *et al.* (2002) reported a change in activity every 4-6 s. These activities include high-intensity running, tackling, heading, rapid changes of direction and activities whilst in possession of the ball. The number of sprints and high speed runs are in the order of 20 and 60, respectively, with a mean time for every sprint 2.0 s and high speed run 2.1 s (Bradley *et al.*, 2009). Owing to the nature of the game, two sprints could be separated by very short or long recovery periods, consequently affecting the metabolic demands of a match. The intermittent high-intensity pattern of activity during the match requires a high function of both the aerobic and anaerobic energy delivery pathways. The ability to recover from these bouts of high-intensity exercise is one of the most important attributes a professional soccer players possess (Wragg et al., 2000).

As well as high aerobic power and the ability to recover from bouts of high-intensity exercise, other physical and physiological attributes such as good agility, joint flexibility, muscular development, and the ability to generate high torques during fast movements contribute to playing success (Reilly *et al.*, 2000a). Each of these components of fitness are important, though the complexity of soccer makes it difficult to ascertain the relative importance of each to overall match performance (Rösch *et al.*, 2000). As in any sport, players who form part of professional clubs and national teams are those players better suited to cope with the demands of the game through innate capability and optimal training status and are thus considered the elite. To preserve performance and therefore elite status, maintenance of optimal training status is important. Negative changes in habitual training and playing patterns may result in performance decrements. These decrements can compromise the player's performance levels and leave the player susceptible to injury.

This chapter will therefore explore the effects of changes in habitual training habits on the physical and physiological variables that are considered to be important to success in soccer. The number of studies investigating this area of work in soccer *per se* is limited so studies of elite athletes in other sporting disciplines will also be considered.

2.2.1.1 Detraining

Preparation for performance in any sport consists of a series of physiological stresses that bring about, or preserve, specific adaptations to enhance the ability to tolerate the demands of training and competition (Mujika and Padilla, 2000a). Inherent to the concept of training adaptation is the principle of training reversibility or detraining (Wilmore, 1988). Detraining can be defined as the temporary or permanent reduction or withdrawal of a training stimulus that results in the loss of anatomical and physiological adaptations as well as decreases in athletic performance (Faigenbaum *et al.*, 1996). Detraining is a complex physiological state that incorporates several contributing factors that can be linked to observed decreases in performance (Kraemer *et al.*, 2002). Understanding the mechanisms associated with detraining therefore requires an awareness of both the individual responses of specific physiological systems (e.g. neuromuscular, cardiovascular etc.) and the potential interactions that may happen between them following a period of reduced training (Greenleaf, 1997). Detraining can occur for a number of reasons including out-of-season training reductions, loss of motivation, social pressures on training time and injury.

Chapter 2.1 demonstrated the prevalence of injury in elite soccer players. The major consequence of injury is the inability of the injured soccer player to maintain normal training and competition. Recent studies have made it apparent that a short period of reduced training volume (<10 d) may enhance performance (Mujika *et al.*, 2002; Bishop and Edge, 2005; Izquierdo *et al.*, 2007). This performance facilitation will however, change to a deterioration in performance if periods of inactivity are prolonged.

There are several important considerations when examining the available literature with regard to detraining. The pre-injury training status of the subjects and the model of the detraining period require careful examination. The training status of the subjects used appears to have a significant effect on the results as there are considerable differences in the physiological responses to detraining when a population varies between well-trained elite athletes and recently trained individuals who were previously sedentary (Coyle *et al.*, 1984; Wilber and Moffitt, 1994). The model of the detraining period used in the investigation may also be important. The variables that constitute the experimental model used include the duration of the detraining period and the level of activity performed during the detraining period. The observed changes in the variety of different physiological adaptations seem to be time dependant with some changes occurring within days while others take some weeks or even months to demonstrate significant alterations. This may explain why most detraining papers investigate either the short-term (<28 d) or long-term (>28 d) effects of detraining (Mujika and Padilla, 2000b). Significant differences are found in the physiological changes with detraining following a period of immobilisation or bed rest compared to those associated with a reduction in the habitual training patterns completed by athletes (Neufer, 1989; Convertino, 1997; Mujika and Padilla, 2000a).

As this thesis investigates the effects of detraining on elite soccer players, this section will focus on the physiological and performance consequences of detraining in athletic populations. The analysis of the available literature will focus on the duration and level of activity during the detraining period. The periods of detraining that are short-term (<28 d) and long term (>28 d) will be considered as these studies are again, most relevant to the research areas of this thesis

2.2.2 Musculoskeletal responses to detraining

In order that the effects of detraining on muscle in elite soccer players can be examined, an understanding of the muscle morphological characteristics of an elite soccer player is required. In several studies of elite soccer players, a morphological analysis of muscle has been performed on biopsies taken from the vastus lateralis or gastrocnemius muscle. The mean percentage of type I fibres ranged from 40 to 61% in the vastus lateralis and from 49 to 60% in the gastrocnemius (Reilly *et al.*, 2000b). For both muscles, the individual variation in the percentage of type I fibres was large, indicating that a specific distribution between type I and type II fibres is not important for top-class players (Tumilty, 1993).

Muscle capillarisation in soccer players is higher than that observed for untrained individuals, but not as high as that found for elite athletes competing in endurance sports (Kuzon *et al.*, 1990; Bangsbo, 1994a). In addition muscle CS activity and HAD concentration were only slightly lower for professional soccer players than those found for endurance-trained elite Danish athletes. A cross sectional comparison revealed that the muscle CS activity was lower in the vastus lateralis than in the gastrocnemius, while no difference was observed for the activity of HAD (Bangsbo, 1994a). The former difference might be attributed to the gastrocnemius being more involved in endurance exercise during training and match play than the vastus lateralis. The muscle morphology of soccer players would therefore be more comparable to that of endurance athletes than athletes trained for sports in which high-force development is essential for performance such as weight lifters and jumpers (Tesch et al., 1984). Changes in soccer performance therefore may be more influenced by the negative effects of detraining on aerobic metabolism than changes in characteristics that underpin strength performance.

2.2.2.1 Muscle capillarisation

A short-term period of inactivity in athletes leads to declines in capillary density which reduce both the transit time of blood flow through the muscle and the diffusion area (Coyle, 1988). Houston *et al.* (1979) reported a 6.3% reduction in capillary density after only 15 d detraining in 3 experienced endurance runners. A significant decrease in the number of capillaries around type I muscle fibres in 4 professional soccer players was also found following 2 to 21 d of detraining (Bangsbo and Mizuno, 1988). In contrast, Madsen *et al.* (1993) found no significant reduction in capillarisation in 9 endurance-trained runners

following a 28 d detraining period. The contradictory findings may be explained by the level of activity maintained throughout the detraining period in each investigation. Houston *et al.* (1979) didn't record or control the activity patterns of the subjects during the detraining period. Bangsbo and Mizuno (1988) estimated activity at 10% the normal weekly exposure and the athletes in Madsen *et al.* (1993) performed 1 x 45 min high-intensity bout of exercise per week throughout the detraining period as opposed to their normal 6-10 hr of activity. These different approaches to controlling the level of activity will impact on the detraining response and make clear comparisons of the studies difficult.

A longer detraining period of 84 d also failed to significantly affect the number of capillaries per muscle fibre in 7 highly conditioned subjects (Coyle *et al.*, 1984). Capillarisation in this population remained 50% higher than that observed in sedentary controls. The authors suggested that this may have been a consequence of long-term adaptations induced by prolonged training. The mean regular training history was 10 ± 3 years amongst these subjects. This would suggest that they are very well adapted to prolonged endurance type activity thereby preventing transient changes in activity affecting their physiological status. The subjects in the two studies that observed significant decreases in capillary density following short-term detraining however, (Houston *et al.*, 1979; Bangsbo and Mizuno, 1988) had been training for >4 y and >13 y respectively. This would seem to contradict these ideas and indicate that training history is not protective from detraining induced decreases in capillary density. The accuracy of the findings of these two studies (Houston *et al.*, 1979; Bangsbo and Mizuno, 1988) however can be questioned because the total number of subjects investigated in the two studies was low (N=5). The above evidence may therefore be inconclusive and prevent a clear understanding of changes in muscle capillarisation being made.

2.2.2.2 Myoglobin concentration

Information about the effect of detraining on myoglobin is limited. The available research suggests that muscle myoglobin is not affected by the withdrawal of an exercise stimulus (Coyle *et al.*, 1984; Nemeth and Lowry, 1984). Nemeth and Lowry (1984) found that 84 d of detraining resulted in no changes to myoglobin levels in both type I and type II muscle fibres in the vastus lateralis muscle. The results of this investigation must, however, be interpreted with caution as this was an individual case study and so the data may be limited in its generalisability. In a more extensive investigation in which myoglobin content of the

gastrocnemius muscle was examined in trained runners and cyclists at regular intervals during an 84-d detraining period, Coyle *et al.* (1985) reported that the myoglobin concentration at the start of the detraining period (43 $mg \cdot g^{1}$ $protein^{1}$) did not change significantly after detraining (41 $mg \cdot g^{1}$ $protein^{1}$). The limited evidence available would suggest that levels of myoglobin in skeletal muscle are not significantly affected by periods of detraining.

2.2.2.3 Mitochondrial enzyme activity

Mitochondrial marker proteins, which are involved in the utilisation of substrates and oxygen for ATP production, decline rapidly when habitual training is reduced (Mujika and Padilla, 2001a). This is specifically the case for athletes who have trained for several years (Neufer, 1989). For example, skeletal muscle CS activity decreased between 25% and 45% with short-term training cessation in endurance trained athletes (Coyle *et al.*, 1985; Moore *et al.*, 1987; Houmard *et al.*, 1992; Shepley *et al.*, 1992; Houmard *et al.*, 1993; McCoy *et al.*, 1994). Bangsbo and Mizuno (1988) examined gastrocnemius muscle biopsies in 2 of their 4 subjects after 1 week of detraining. They observed a reduction in both CS (17%) and HAD (33%) (46-38 for CS and 52-35 $mol \cdot min^{-1} \cdot g$ dry wt^{-1} for HAD) in these individuals. A decreased muscle oxidative capacity is also reflected in the significant 12 to 27% reduction observed in HAD (Houston *et al.*, 1979; Coyle *et al.*, 1985), malate dehydrogenase (MD) (Coyle *et al.*, 1985) and succinate dehydrogenase (SD) (Houston *et al.*, 1979; Coyle *et al.*, 1985).

Reduced mitochondrial enzyme activity seems to be one of the main characteristics of muscular detraining. Although these adaptations seem to occur in the early stages of detraining, baseline concentrations still appear to remain above those of the sedentary population. Coyle *et al.* (1985) observed that the CS activity of 7 endurance-trained athletes' declined from 10 to 8 $mmol \cdot kg$ $protein^{-1}$ hr^{-1} (25°C) during the first 21 d of detraining. It continued to decline to 6 $mmol \cdot kg$ $protein^{-1}$ hr^{-1} (25°C) by the 8^{th} wk but thereafter stabilised at this concentration. Succinate dehydrogenase, HAD and MD declined roughly in parallel with CS. The timescale of the reductions were also similar with an initial 20% reduction by day 21 and a further 40% decrease by day 56 of detraining. This was followed by a stabilisation at a level that was still 50% higher than that of sedentary controls. These results are very similar to those reported by Chi *et al.* (1983) who observed significant decreases in CS, NAD, MD and SD, of between 25% and 40% after 8 wk of no training in highly-trained endurance runners. Unfortunately, a detailed sequential analysis of these changes was not

available due to the methodological limitations of the study. It is not possible therefore to elicit the point at which these reductions became statistically significant. As in the Coyle *et al.* (1985) study, oxidative enzyme activity remained an average of 40% higher than the control group.

If the effects of detraining in individual human muscle fibres are examined, an interesting picture emerges. This may help to explain the findings discussed above. Mitochondrial enzyme activities decreased to almost untrained levels in type I muscle fibres during 84 d of detraining (Chi *et al.*, 1983). Data from type II muscle fibres showed that CS, HAD and MD activity were still 50 to 80% above the values observed in the untrained. The failure of the whole muscle of trained endurance athletes to fall towards untrained values following 84 d of detraining therefore probably occurs almost entirely because of the persistent 80% elevation levels in the mitochondrial enzyme activity in type II muscle fibres (Chi *et al.*, 1983).

This evidence would suggest that the cessation of prolonged and intense soccer training may result in only a partial loss of mitochondrial enzyme activity. This preservation of enzyme activity levels will result in a persistent elevation above levels observed in the untrained. It would therefore be expected that soccer players undergoing periods of detraining as a result of injury would experience a rapid reduction in aerobic enzyme activity as a consequence of their training status, but the rate of this change would be reduced during a prolonged period of inactivity. This pattern of adaptation may mean that the enzymatic activity of players injured for extended periods may not be significantly lower than players injured for shorter periods.

2.2.2.4 Glycolytic enzyme activity

Changes in the glycolytic capacity of skeletal muscle in the highly-trained are the subject of fewer investigations than the changes in oxidative capacity. In endurance athletes not training for 84 d, hexokinase decreased significantly by about 17% ($p<0.05$) with lactate dehydrogenase increasing significantly by about 20% ($p<0.05$). Phosphorylase showed no change while phosphofructokinase (PFK) increased 16%. These changes were not significant ($p>0.05$) (Coyle *et al.*, 1984). Six top-class rugby league players who detrained for 42 d starting the day after their grand final were the subjects in a study by Allen (1989). In contrast to Coyle et al. (1984), reductions in PFK activity reached significance after 35 d of the detraining period.

One explanation for the contradictions in the findings in the investigations above could have been the type of habitual training undertaken by the athletes prior to detraining. The subjects in the work of Coyle *et al.* (1984) were endurance athletes who had trained for an average of 10 ± 3 years and competed in track events of up to 10000 m. Their training regimen for the 12 months prior to the study was predominantly endurance activity for >4 $d \cdot wk^{-1}$ for ~60 min per day at an exercise intensity corresponding to 70-80% $\dot{V}O_{2max}$. This training programme would have resulted in highly-developed aerobic energy pathways. The absence of any high-intensity work may have restricted glycolytic adaptations. The subjects in Allen *et al.* (1989) were top class full-time professional rugby players and, as such, participated in activity which involves frequent high-intensity work outputs followed by low intensity recovery periods. As a result of this work pattern, significant demands on both aerobic and anaerobic energy sources would have occurred in both training and playing. This would suggest that both the aerobic and anaerobic energy pathways may be highly developed and thus susceptible to negative changes associated with reduced work.

No studies using elite soccer players have investigated the effects of detraining on glycolytic enzyme activity. However, due to the physiological demands of soccer and rugby being similar, the results of Allen *et al.* (1989) may be those that may best relate to the current thesis. The glycolytic capacity of skeletal muscle therefore seems to be more robust to negative changes in training patterns. Extended detraining periods may however, result in negative adaptations of the glycolytic pathways which may limit high-intensity exercise performance.

2.2.2.5 Muscle Fibre Characteristics

Changes in muscle fibre characteristics and individual muscle fibre cross-sectional area changes following short-term detraining periods have also been investigated. Hortobágyi *et al.* (1993) examined changes in muscle fibre cross-sectional area and numbers and percentage of muscle fibre types in 12 power athletes following a detraining period of 14 d. The only significant change was a 6.4% decrease in type II muscle fibre area. Testing at the beginning of the detraining period in this study, however, showed that subjects only achieved 90% and 92% of their personal best in functional performance assessments such as the bench press and the thigh squat. The reason for this sub-maximal performance was not discussed. It could be

suggested that the subjects were not at their optimal training levels at the time of the evaluation of muscle fibre area. This may have resulted in a diminished detraining effect. The results following a slightly longer detraining period of 21 d in soccer players confirmed the above findings (Bangsbo and Mizuno, 1988). They reported a significant reduction in mean cross-sectional area of muscle fibres (5220 to 4868 μm^2). A reduction in cross-sectional area of type II fibres (6022 to 5278 μm^2) was almost entirely responsible for this reduction. It is possible that, once the training stimulus had been removed, the hypertrophied type II fibres become more susceptible to atrophy than type I muscle fibres.

No studies can be found that report a significant change in muscle fibre type following short-term detraining. Hortobágyi *et al.* (1993) found no significant changes in the percentage of muscle fibre types or the number of fibres counted in their study of 12 power athletes after a 14-d detraining period. Fifteen days detraining was also not long enough to induce a significant change in the percentage of muscle fibre types of 6 highly-trained distance runners who wore a lower leg plaster cast for 7 d, then refrained from training for an additional 8 d (Houston *et al.*, 1979). The same was reported in the study of 4 soccer players who detrained for 21 d (Bangsbo and Mizuno, 1988). It would therefore appear that muscle fibre distribution or cross-sectional area should not be affected in soccer players who detrain for periods of <14 d.

Longer-term detraining (>28 d) does, however, induce significant changes in the muscle fibre distribution of athletes (Mujika and Padilla, 2001a). These adaptations appear to be dependant on the type of training (strength or endurance) the athletes were performing prior to the detraining period. An increase in the population of oxidative fibres compared to type II muscle fibres has been found in several studies that have used strength and power trained athletes (Staron *et al.*, 1981; Larsson and Ansved, 1985; Häkkinen and Alén, 1986). The case study of an elite power lifter indicated that the oxidative muscle fibre population was 1.4 times greater after 7 months of detraining indicating a reduction in the percentage of type II muscle fibres (Staron *et al.*, 1981). The percentage of type II fibres was also seen to reduce (66% - 60%) in an elite body builder after 13 months detraining (Häkkinen and Alén, 1986).

In endurance-trained athletes, this trend appears to be reversed with a decrease in oxidative muscle fibre types. Coyle *et al.* (1985) reported a large progressive shift from type IIa to type IIb fibres in endurance runners and cyclists after 84 d of detraining. This change was

associated with an increase in type IIb fibres from 5% in the trained state to 19% in the detrained state. Larsson and Ansved (1985) observed that the proportion of type I fibres in 4 elite oarsman decreased by 24% and 16% in the deltoid and quadriceps muscles respectively during the 4 year period after their retirement. The evidence suggests that the specific muscle fibre adaptations, resulting from the type of prolonged training undertaken, are the most susceptible to the effects of detraining. This may be because the change in the activity patterns of the specific motor units which are habitually stimulated during training is greater than the change in those motor units which are not habitually stimulated during training.

Muscle fibre mean cross-sectional area is also affected by extended detraining periods. Allen (1989) demonstrated that the cross-sectional area of type II fibres decreased to a greater extent than type I fibres after 42 d without training in rugby league players. These findings were confirmed by Häkkinen *et al.*, (1981) who reported a significant reduction in Type II:Type I area ratio following a 56-d detraining period in 14 experienced weight trainers. After 7 months of detraining, an average atrophy of 37% was observed in all muscle fibre types of a power lifter (Staron *et al.*, 1981). The percentage atrophy was most pronounced in the Type II fibres (over 41%) as compared with the Type I fibres (31%). In this study, there was also a significant decrease of 16 cm in the girth of the thigh accompanying the atrophy of the individual muscle fibres. In a similar study, a body builder's thigh (60 to 57 cm) and arm (39 to 34 cm) circumference also reduced significantly following 13 months detraining (Häkkinen and Alén, 1986). An average muscle fibre cross-sectional area decrease (8.3%) was also reported. These data suggest that following extended detraining periods, cross sectional areas of Type II fibres are affected to a greater extent than type I fibres in strength-trained athletes. This selective atrophy of predominantly Type II fibres can be explained by the normal physical activity of the subjects during detraining. Physical activity not involving high muscle tensions is not likely to recruit Type II motor units (Häkkinen *et al.*, 1981). This will lead to a reversal of the training induced Type II muscle fibre hypertrophy.

When considering the effects of detraining on muscle of soccer players, the activity patterns, which include both aerobic and anaerobic bouts, have to be considered. The performance of anaerobic bouts including sprints, jumps, tackles and explosive changes of direction may result in hypertrophy of type II muscle fibres. Although limited when compared to sportsmen such as weight lifters, this hypertrophy may be important to enable soccer players to perform these anaerobic bouts. This was shown by Bangsbo and Mizuno (1988) who observed that the

atrophy in muscle fibre cross-sectional area following detraining, reverted back to pre-detraining levels following 21 d of soccer training. The predominant energy demands of soccer are however, aerobic in nature (Drust *et al.*, 1998) with type I muscle fibres motor units being highly developed. Detraining in professional soccer players will therefore be characterised by negative adaptations in both type I and type II muscle fibres, albeit at different rates. Rehabilitation training therefore has to address the negative responses to detraining in both of these muscle fibre groups.

2.2.2.6 Strength Performance

Muscular strength and power share importance with endurance in soccer (Wisløff *et al.*, 1998). These fitness parameters are important physiological characteristics in order to perform sprinting, jumping, tackling and kicking in a soccer game (Reilly *et al.*, 2000b). In particular, muscle strength of the lower limbs is significantly associated with vertical jump height, sprinting performance (Wisløff *et al.*, 2004) and a reduction in the risk of injury (Fleck and Falkel, 1986). Reilly and Thomas, (1979) monitored the first and reserve teams of an English league team throughout one season. They concluded that an array of strength and power tests successfully discriminated between playing levels. They also suggested that players who were stronger and more muscular were better able to survive the physical demands of the prolonged competition.

Understanding changes in indices of strength as a result of detraining would appear to be vital for those responsible for the reinstatement of the trained status of elite soccer players following periods of inactivity. Different modes of muscular activity induce quite distinct adaptive responses in the muscle tissue (Sjøgaard, 1984; Houston, 1986). The type of long-term exercise undertaken prior to the detraining period will affect selective muscle fibre morphology, central neural drive to the motor units and compensatory hormonal adaptations as previously discussed. Therefore, any discussion of the changes which occur in the muscle during detraining must pay reference to the type of exercise performed (i.e. strength or endurance work) during the preceding training period, and in some cases, during the detraining period.

Following short-term detraining (<28 d) in well-trained athletes, any reductions in strength appear to be minimal (Kraemer *et al.*, 2002). Some studies have even demonstrated improvements following short-term detraining. For example, Shepley *et al.* (1992) observed a

significant increase in isokinetic knee extension (from ~230 to ~265 Nm) following 7 d of a sedentary lifestyle in highly-trained endurance runners (mean $\dot{V}O_{2max}$ 67 $\pm$2 ml·min^{-1}·kg^{-1}) during which no training was completed. One reason for this improvement may be that the voluntary strength of the quadriceps had been suppressed by chronic intensive running training only to be recovered when training was discontinued.

Trained swimmers also maintained muscular strength, as measured on a swim bench, during 28 d of inactivity (Neufer *et al.*, 1987). This strength maintenance did not however transfer to their ability to apply force in the water which was markedly reduced (13.6%). In an excellent study by Hortobágyi and colleagues (1993), 12 strength trained athletes, who had been weight training for an average of 8$\pm$2 years, where assessed using a number of strength and power measures before and after a 14-d detraining period. Results showed slight but non-significant reductions in bench press, squat, isometric and isokinetic concentric knee extension force, and vertical jump height at the end of the detraining period. The only significant finding was an average 12% (114 N) decrease in isokinetic eccentric knee extension at all three angular velocities (-0.87,-2.62,-4.33 rad·s^{-1}). As this was the only eccentric muscle contraction tested, it was suggested that it may be the eccentric component of any muscle contraction, as opposed to the concentric component, that is specifically affected by short-term detraining. This finding is in accord with McCarrick and Kemp (2000) who observed that eccentric muscle contraction performance of the shoulder rotator cuff muscles were more susceptible to detraining than concentric performance. Colliander and Tesch (1992) however, found that eccentric muscle performance was more robust to detraining than concentric muscle action. In a study to elucidate the training frequency required to maintain strength gains acquired by resistance training on hamstring and quadriceps muscles of the thigh, they found that eccentric muscle actions promoted greater and longer lasting neural adaptations to training. It is however, difficult to compare the findings of the studies as each group of subjects in Colliander and Tesch (1992) were specifically trained using eccentric or concentric contraction only protocols whereas no such specificity of training was undertaken in the other studies.

The suggestion that the eccentric component of muscle contraction may be more susceptible to detraining than the concentric component has implications for muscle rehabilitation, particularly following hamstring muscle injury. Hamstring injuries are one of the most

common injuries in soccer (Rolls and George, 2004) with the failure to control the eccentric force of rapid knee extension responsible for a high proportion of hamstring injuries (Askling *et al.*, 2005). Additional vigilance may therefore be required when rehabilitating hamstring injuries to ensure any eccentric control deficits are addressed.

Reductions in concentric force following short-term detraining have been found by others. Gibala *et al.* (1994) observed that a 10 d detraining period lowered the velocity of the concentric peak torque in the elbow flexors of experienced resistance trainers. Peak torque was initially improved with significantly higher values observed on day two. Following this initial super-compensation, values fell to significantly lower levels by day 8 and 10. Maximum isometric and maximum velocity concentric peak torque did not change. The lack of changes to all but one of the variables in this study could be due to the subjects being tested every 48 hours throughout the rest only period (3 x maximal 5 s efforts for isometric contraction and 2 efforts at low and high velocity for concentric contractions) as these tests could have provided sufficient stimuli to prevent detraining and maintain muscle performance.

Longer periods of detraining are accompanied by more pronounced declines in the strength of strength-trained athletes. Indices of strength decline by 7 to 12% during inactivity periods up to 84 d (Häkkinen *et al.*, 1981; Häkkinen and Komi, 1983; Häkkinen *et al.*, 1985). This force decline may involve predominantly neural changes initially (Häkkinen *et al.*, 1985; Häkkinen and Komi, 1983) with a gradual increasing role of atrophy during longer detraining periods (see section 2.2.2.5). Indices of power are also affected by long-term detraining. Hsu and Hsu (1999) reported a significant 12% reduction in swimmer's stroke power following 84 d of detraining. In performance terms, this resulted in an increase in the number of strokes (72 to 78) required to swim 200 m. Godfrey *et al.* (2005) reported performance decrements, in terms of muscular power, following an 56 d detraining period in an Olympic rower. He observed a significant reduction in power of 20% (546 W to 435 W) at peak oxygen consumption. Power at a blood lactate concentration of 2 mmol also decreased from 399 W to 290 W (-27%) after training cessation.

The evidence shows that strength indices are important for soccer players to be able to perform important tasks that are essential for success such as sprinting, tackling, jumping and explosive changes of direction. Although it appears that strength performance may be robust

to short-term detraining, longer (>28 d) detraining may lead to decrements in strength which may affect players performance and also leave them more prone to injury.

2.2.3 Cardiorespiratory responses to detraining

2.2.3.1 Maximal Oxygen Uptake

Maximal oxygen uptake is perhaps the physiological parameter most evaluated in elite soccer players. The average level of $\dot{V}O_{2max}$ is about 60 $ml \cdot kg^{-1} \cdot min^{-1}$ (Stølen *et al.*, 2005), but individual figures as high as 73 $ml \cdot kg^{-1} \cdot min^{-1}$ have been reported (Apor, 1988). Maximal oxygen uptake has been observed to be higher for midfielders than for players in other positions. This higher $\dot{V}O_{2max}$ has been associated with a longer distance covered in the game (Tumilty, 1993). Although a correlation exists between the level of the average $\dot{V}O_{2max}$ of a team and success in soccer (Apor, 1988; Wisløff *et al.*, 1998) the complex interaction between factors other than aerobic power, namely the technical and tactical level make these findings equivocal. Helgerud *et al.* (2001) has, however, showed that gains in aerobic fitness can parallel improvements in individual players' soccer-specific performance on the playing field. This well controlled study, showed that an 11% enhancement of $\dot{V}O_{2max}$ lead to a 20% increase in distance covered, 100% increase in number of high-intensity sprints, 24% increase in involvement with the ball and greater work rate during actual matches. Such high levels of aerobic fitness may also protect against injuries late in games (Drust *et al.*, 1998).

Maximal aerobic capacity is a function of maximal cardiac output and arterial-venous O_2 difference, both of which are enhanced through aerobic training (Lacour and Denis, 1984). It is well documented that maximal aerobic capacity declines by up to 14% with short-term training cessation in highly-trained individuals who possess an initially large aerobic power and an extensive training background (Ehansi *et al.*, 1978; Houston *et al.*, 1979; Coyle *et al.*, 1984; Martin *et al.*, 1986; Houmard *et al.*, 1992). These reductions in $\dot{V}O_{2max}$ are correlated with the training status of the population under consideration (Mujika and Padilla, 2001b). Coyle *et al.* (1984) reported a correlation of 0.93 ($p<0.001$) between the initial level of $\dot{V}O_{2max}$ and the decline in $\dot{V}O_{2max}$ following a detraining period. In that study, the subjects' $\dot{V}O_{2max}$ reduction was in the range of 22 to 10 $ml \cdot min^{-1} \cdot kg^{-1}$ following the 84-d detraining period. The correlation and the reduction in this range of subject's $\dot{V}O_{2max}$ confirms the previous findings that individuals with higher $\dot{V}O_{2max}$ are more prone to the effects of

detraining. The authors suggested, however, that the post-detraining aerobic power levels were still higher than in untrained subjects.

Ehansi *et al.* (1978) evaluated cross-country runners 7 d after completion of their competitive season and found that $\dot{V}O_{2max}$ significantly decreased by 11% from 62 ± 2 to 56 ± 3 $ml \cdot min^{-1} \cdot kg^{-1}$. Interestingly, there were no further decreases in $\dot{V}O_{2\,max}$ over the following 14 d. This rapid loss of aerobic performance has been observed by others. The $\dot{V}O_{2max}$ of endurance athletes decreased by 7% following 12 d of detraining (Coyle *et al.*, 1984) and 4% following 15 d of detraining (Houston *et al.*, 1979). A 28 d detraining period in national level basketball players also resulted in a 10% reduction in $\dot{V}O_{2\,max}$ (57 ± 9 to 49 ± 7 $ml \cdot min^{-1} \cdot kg^{-1}$, $p<0.01$) (Ghosh *et al.*, 1987). The athletes in this study were instructed not to participate in any physical activity during their mid-season break. The only testing sessions were at the beginning and end of the 28-d detraining period. No time course for the adaptations was therefore reported.

These findings of a reduction in $\dot{V}O_{2\,max}$ following short-term detraining are in contrast to those of Shepley *et al.* (1992). They reported no significant changes in $\dot{V}O_{2\,max}$ following a 7-d detraining period in 9 highly-trained middle-distance runners ($\dot{V}O_{2\,max}$ 66-71 $ml \cdot min^{-1} \cdot kg^{-1}$). Other studies have also reported that $\dot{V}O_{2max}$ was maintained by trained athletes following short (10 d) periods of detraining (Cullinane *et al.*, 1986; Claude and Sharp, 1991). Longer periods of detraining (21-28 d) were also insufficient to significantly affect $\dot{V}O_{2\,max}$ in some research (Bangsbo and Mizuno, 1988; Madsen *et al.*, 1993). These differences in the findings of relevant investigations could be related to the amount of physical activity performed by the athletes during the detraining period. For example, the 9 well-trained endurance athletes in Madsen *et al.* (1993) performed one, 35-min high-intensity exercise bout per week as opposed to the normal 6-10 $h \cdot wk^{-1}$ during their 28-d detraining period. This level of high-intensity training load may have been sufficient to maintain $\dot{V}O_{2\,max}$ levels observed in these subjects (trained 66 ± 1, detrained 65 ± 1 $ml \cdot min^{-1} \cdot kg^{-1}$). The instruction to the soccer players in Bangsbo and Mizuno (1988) study during their 21-d detraining period was not to play soccer. They reported no significant changes in $\dot{V}O_{2\,max}$ in 3 outfield players and 1 goalkeeper. No restrictions were, however, placed on them participating in other sports. Players' recorded their own training volume (frequency and duration). Although it was

reported that the detraining training load was ~10% of the normal training load, no record was kept of the intensity level of their activities in the detraining period. This could suggest that subjects completed other types of high-intensity physical activity during the detraining period thereby confounding the results of the investigation.

To illustrate the effects that maintaining a reduced training load can have on $\dot{V}O_{2\ max}$, Rietjens *et al.* (2001) divided 12 well-trained cyclists into two groups. A continuous endurance training group reduced their weekly training intensity for a period of 21 d to approximately 68% of $\dot{V}O_{2max}$ and an intermittent training group maintained intensity but reduced training volume and frequency by 50% and 20% respectively for the same period. Neither group showed changes in maximal workload or $\dot{V}O_{2max}$ following the reduced training intervention. The effects of reduced training on $\dot{V}O_{2max}$ has also being investigated by Neufer *et al.* (1987) who found that the aerobic power of highly-trained swimmers (>5 months, 8000 m/session, 6 sessions/wk) was maintained after a month of moderately reduced training (three sessions/wk, 2700 m/session). The type of training may also be a factor in the length of time a reduced training program is effective in maintaining aerobic power. Performance may be lost at a faster rate when the training is localised to smaller muscle mass, as utilised in cycling compared to running (Hickson *et al.*, 1985). The above evidence demonstrates that $\dot{V}O_{2max}$ can be maintained at the training levels when exercise is reduced. Regulating which components of the training parameters (duration, frequency, intensity or a combination of all three) maintain aerobic power however, is not completely clear from these investigations.

In summary, the $\dot{V}O_{2max}$ of highly-trained athletes decreases rapidly but remains above that of untrained persons even after several weeks of detraining (Fardy, 1969; Coyle *et al.*, 1985). It might therefore be expected that soccer players sustaining serious injury would experience a rapid reduction in $\dot{V}O_{2max}$ over the first ~28 d of detraining, which then falls less dramatically for a further period before reaching a new baseline value for the remainder of the time out injured. There may be little difference therefore in the effect of detraining on $\dot{V}O_{2max}$ between a player injured for 28 d and a player injured for 142 d. The above findings may also have an impact on designing rehabilitation programmes as evidence suggests that maintaining a level of physical activity may help reduce any reduction in $\dot{V}O_{2\ max}$. Due to the rapidness of negative adaptations, interventions aimed at reducing $\dot{V}O_{2max}$ decrements would

seem to have to be implemented soon after the injury event to maximise their effect. The disabling nature of the particular pathology may however, make exercising at the required intensity difficult.

2.2.3.2 Blood Volume

The hypervolemia that often accompanies endurance training is quickly lost when training is stopped (Convertino, *et al.*, 1997). Coyle *et al.* (1986) found that 28 d of detraining in 8 highly-conditioned cyclists (mean $\dot{V}O_{2max}$ 65 $ml \cdot min^{-1} \cdot kg^{-1}$) who had been performing intense endurance training for an average of 6 years, resulted in a 9% (485 ml) decline in blood volume during upright exercise. They suggested that the majority of the reduction occurred as a result of a 354 ± 66 ml decline in plasma volume and a smaller yet significant decline (131 ± 39 ml) in erythrocyte volume. Other studies have produced similar results showing blood volume and plasma volume reductions of 5% (Houmard *et al.*, 1992) to 12% (Cullinane *et al.*, 1986) in endurance-trained athletes. It is therefore evident that the reductions in blood volume occur rapidly following the cessation of training and may occur within 2 d of inactivity (Cullinane *et al.*, 1986). This rapid reduction is thought to stabilise after ~8 d (Houmard *et al.*, 1992). The rapid onset of reductions in blood volume highlights the importance of early return to physical activity following injury in an effort to prevent these negative changes.

2.2.3.3 Heart Rate

The resting heart rate of top players is typically 48 to 52 $beats \cdot min^{-1}$ (Reilly, 1994a). Maximal heart rate is similar to that observed in the general population of 187-193 $beats \cdot min^{-1}$ (Bangsbo, 1994a). Others have reported lower figures (White *et al.*, 1988) but this probably reflects differences in the mode of exercise utilised for assessment. During match play the average intensity oscillates around the lactate threshold, where the heart rate is 80 to 90% of the maximal heart rate for up to two thirds of the game (Bangsbo, 1994b; Kirkendale, 2000).

A common finding following a reduction in training among athletes is an absence of change in resting heart rate (Ehansi *et al.*, 1978; Kanakis *et al.*, 1982; Cullinane *et al.*, 1986). Rebelo *et al.* (1997) investigated the difference in resting heart rate of 16 professional soccer players at the end of a 28 d holiday (detrained state) and after a 56 d training period. They found no significant change in resting heart rate despite the high-intensity of the re-training period. No

record was kept of the players resting heart rate at the beginning of the detraining period and no records kept of the physical activity levels during the detraining period. Players may have maintained a high level of activity during the detraining period resulting in maintenance of previous heart rate status. The duration of the detraining period may also have been insufficient to elicit physiological adaptations. It would, however, appear that resting heart rate does not change significantly following periods of detraining.

Heart rate during exercise however, increases at sub-maximal (Fardy, 1969; Penny and Wells, 1975; Coyle *et al.*, 1986; Houmard *et al.*, 1992) and maximal (Houston *et al.*, 1979; Coyle *et al.*, 1984; Cullinane *et al.*, 1986; Houmard *et al.*, 1993) intensities following detraining. These increases, of as much as 5 to 10%, are probably due to the decreases in blood volume that accompanies short-term detraining (see chapter 2.3.3.2). Coyle *et al.* (1986) noted that when blood volume was maintained at higher levels through plasma volume expansion, no significant changes in cardiovascular function during exercise were observed.

Maintaining a reduced training load may however, prevent changes in exercise heart rate values. Rietjens *et al.* (2001) found no differences in mean heart rate during exercise when highly-trained cyclists performed a 90 minute steady state cycle test following 21 d of reduced training load (50% of the athlete's normal $hr \cdot wk^{-1}$). A similar training load in early rehabilitation may therefore have a positive effect on maintaining the exercise heart rate of injured soccer players.

2.2.3.4 Stroke Volume

One of the most striking effects of detraining in highly-trained individuals is the rapid decline in stroke volume. Seven well-trained endurance athletes (mean $\dot{V}O_{2max} \approx 62\ ml \cdot min^{-1} \cdot kg^{-1}$) were studied for 84 d during which time physical activity was limited to that required by their sedentary jobs (Coyle *et al.*, 1984). Stroke volume during maximal exercise decreased by 11% within the first 21 d and then subsequently stabilised at 14% below trained levels after 56 d of detraining. These findings were confirmed in a subsequent study by Martin *et al.* (1986). The mean stroke volume of 6 highly-trained athletes prior to detraining was 151 ± 10 ml. This reduced significantly to 136 ± 10 ml and 125 ± 6 ml following detraining periods of 21 d and 56 d respectively ($p<0.005$).

In an attempt to determine the possible role of blood volume on the detraining-induced decline in stroke volume, Coyle *et al.* (1986) re-established plasma volume to trained levels by the infusion of 6% dextran / 0.9% saline in endurance athletes after a 14-28 d detraining period. Blood volume decreased by 9% with detraining, resulting in a 12% reduction in stroke volume during upright submaximal exercise. Plasma volume expansion after detraining returned stroke volume, heart rate and total peripheral resistance to trained levels. This would indicate that following short-term detraining, stroke volume may be more dependent on blood volume than on alterations in cardiac dimensions.

2.2.3.5 Cardiac Output

Cardiac output is the product of stroke volume and heart rate. Alterations in either of these two indices will therefore affect maximum cardiac output. Sections 2.3.3.3 and 2.3.3.4 have detailed how detraining influences both these parameters with the available evidence suggesting that there is an increase in maximum heart rate during exercise and a reduction in stroke volume. Coyle and colleagues (1984) reported an 11% reduction in stroke volume during both steady-state cycling and steady-state treadmill running. This reduction was partially offset by a 5% increase in maximum heart rate. The increase in maximum heart rate was not sufficient to offset all of the reduction in stroke volume, resulting in an 8% decrease in cardiac output during the first 21 d of detraining (29 ± 2 to 26±2 $l \cdot min^{-1}$). Subsequent measures recorded after 56 d and 84 d of inactivity did not differ significantly from values obtained at day 21. This suggests that the greatest reduction in maximal cardiac output occurs in the initial weeks of detraining. Once again, these findings highlight the importance of an early return to activity in soccer rehabilitation to maintain cardiac output levels.

2.2.3.6 Cardiac Dimensions

The debate regarding whether the larger cardiac dimensions of the athlete's heart are an effect of, or a selection factor for, physical performance remains unsolved (Cullinane *et al.*, 1986; George *et al.*, 1991: Naylor *et al.*, 2008). Prolonged and intense endurance training promotes cardiac hypertrophy (Martin *et al.*, 1986). Manetti *et al.*, (1999) evaluated the effects of training and detraining on the heart muscles of 21 professional soccer players over a 13-month period. They reported a significant increase in left ventricular mass during the playing season, evidencing physiological hypertrophy after six months of competitive activity. No subsequent increases were observed over the next two months. After detraining, left

ventricular mass returned to baseline values suggesting that, in this population, the larger cardiac dimensions are an effect of physical activity and as such are sensitive to changes in activity.

Ehansi *et al.* (1978) evaluated 6 male cross-country runners (mean running distance 60-70 miles per week, mean $\dot{V}O_{2max}$ 62 ± 2 $ml \cdot min^{-1} \cdot kg^{-1}$) during a 21-d detraining period immediately after the completion of their competitive season. Echocardiographic data collected after 7 d of inactivity revealed significant reductions in left ventricular internal dimensions (51 ± 3 to 47 ± 3 mm), left ventricular end diastolic volume (65 ± 5 to 57 ± 5 ml/m^2) and left ventricular mass (110 ± 6 to 80.2 ± 6 g/m^2). These decreases first became apparent after 4 d of rest and continued to decline for the remainder of the 21-d detraining period. Others have also observed reductions in cardiac dimensions following periods of detraining. Martin *et al.* (1986) reported that the left ventricular end diastolic dimension of 8 highly-trained cyclists and runners (mean $\dot{V}O_{2max}$ 63 ± 4 $ml \cdot min^{-1} \cdot kg^{-1}$) during upright exercise declined during 56 d of training cessation. In the same study, left ventricular posterior wall thickness decreased progressively by 25%, but left ventricular mass did not change after the initial 19.5% reduction in 21 d.

In an interesting study by Pelliccia *et al.* (2002), 40 former Italian world-class athletes (mean period from cessation of systematic training and competition 6 ± 4 years; range, 1 to 13 years) who, when competing, had previously undergone a medical program that routinely included echocardiography, were re-examined using the same methods. Fifteen of the athletes had completely eliminated any physical activity from their normal routines, whereas the other 25 had been engaged in light recreational exercise programmes. Although left ventricular remodelling was evident in long-term detraining with a significant reduction in cavity size and normalisation of wall thickness, a substantial chamber dilatation persisted in >20% of the athletes. These findings suggest that athletes can inherit, or are genetically predisposed to have, left ventricular cavity enlargement. Therefore, persistently enlarged cavity sizes in some detrained athletes may in fact represent the usual or normal dimensions rather than a residual training effect. These findings therefore have to be considered when analysing the effects of detraining on cardiac dimensions.

In contrast to these findings are observations of no changes in cardiac dimensions following periods of detraining. Cullinane *et al.* (1986) observed no change in cardiac dimensions of 15 distance runners (mean $\dot{V}O_{2max}$ 61 ± 6 ml·min^{-1}·kg^{-1}) following 10 d of training cessation. Pavlic *et al.* (1986) also observed no change in wall thickness or in internal ventricular dimensions following 60 d of inactivity in 36 road-race cyclists (mean $\dot{V}O_{2max}$ 72 ml·min^{-1}·kg^{-1}). It is difficult to understand the reasons for these contradictory findings as all subjects were experienced endurance athletes, the detraining periods were of a similar duration and the subjects in each study were instructed not to participate in physical activity during the detraining phase. The training status of the subjects at the beginning of the detraining period may have affected the subsequent cardiac adaptations although the mean $\dot{V}O_{2max}$ across the studies was similar.

One reason for the discrepancies in the data may relate to the limitations associated with echocardiographic investigations. Conventional echocardiographic techniques are limited by geometric assumptions, image positioning errors, and the use of subjective visual methods (Gopal *et al.*, 1995). Variations in body position, transducer location, and/or the direction of the ultrasonic beam are, for most part, unavoidable with studies that require serial measurements. Thus, findings concerning changes in cardiac dimensions with training/detraining may be viewed as somewhat tenuous (Neufer, 1989; Naylor *et al.*, 2008) and may be responsible for the contradictory findings detailed above.

Notwithstanding the above evidence, the effect that cardiac adaptations have on heart function following detraining can be questioned. Stroke volume can be returned to trained levels by plasma volume expansion (Coyle *et al.*, 1984) suggesting that the ability of the heart to fill with blood is not significantly altered by detraining. If cardiac output does decline, a thinning of the ventricular walls and not a reduction in the diameter of the left ventricle at end diastole is probably involved (Martin *et al.*, 1986). Thus, the large reduction in stroke volume during exercise in the upright position may be largely a result of reduced blood volume and not of a deviation of heart function (Coyle *et al.*, 1986).

2.2.3.7 Endurance Performance

The endurance performance of trained athletes declines rapidly as a consequence of insufficient training stimulus as illustrated by slower performance times for set distances,

greater oxygen uptakes at specific exercise intensities or shorter times to exhaustion at the same intensity of steady-state exercise compared to those prior to detraining (Mujika and Padilla, 2001b). Each of these adaptations will affect a soccer player's ability to perform to the levels achieved prior to any detraining period. Steps to prevent negative changes in these performance indices are therefore important.

Using a cycle ergometer (9000 $ft.lbs^{-1}min^{-1}$), Fardy (1969) reported a 24% decline (419 to 319 s) in time to exhaustion in 5 soccer players following 35 d of detraining. The reduction was partially attributed to a significant reduction in peak ventilation and peak O_2 intake values. Madsen *et al.* (1993) also showed that 28 d of detraining significantly deteriorated (21%) steady state endurance capacity on a cycle ergometer at 75% of $\dot{V}O_{2max,}$ in endurance athletes. The results showed a reduction from 79 ± 4 to 62 ± 4 min following the detraining period. Both of these studies utilised cycle ergometry to investigate time to exhaustion in athletes whose habitual training load was based around running. Investigations that use a running protocol to investigate the same variables may be more relevant to a soccer population and provided a more accurate measure of $\dot{V}O_{2max}$.

Houston *et al.* (1979) used a running protocol on a motorised treadmill (16 $km{\cdot}h^{-1}$, and graded to demand 90% of pre-determined $\dot{V}O_{2max}$) to examine changes in time to exhaustion following a 15-d detraining period. They observed a significant 25% (19 to 14 min) reduction in mean time to exhaustion. The range of changes however (-2.5 to -50%) was extensive. Examination of the data showed that one subject went from producing the group's best performance prior to detraining (~22 min) to the second worst performance (~11 min) following a 15-d detraining period. Although no specific mention of this subject was made in the discussion, it is difficult to attribute a 50% reduction in performance to detraining alone. The estimated reduction in the same subject's $\dot{V}O_{2\ max}$ was small (~4.6 to 4.4 $l{\cdot}min^{-1}$) suggesting other reasons for the performance decrement such as a lack of effort during the second testing session. In another treadmill based assessment, Houmard *et al.* (1992) reported a significant 9% reduction in time to exhaustion (12 ± 0.5 to 10 ± 0.5 min). These results easily surpassed the ±2% (~20 s) variability the authors calculated for the sub-maximal test (75% $\dot{V}O_{2max}$) for distance runners.

2.2.3 Metabolic responses to detraining

The high levels of aerobic energy production in soccer and the pronounced anaerobic energy turnover during periods of match-play are associated with the consumption of large amounts of substrates (Bangsbo, 1994a). Prolonged physical training, such as in elite soccer, induces marked changes in the energy metabolism in muscle. These changes include an increase in the availability of carbohydrate and lipid as substrate to meet cellular needs for ATP resynthesis with one of the most consistent effects being an increase in the capacity to oxidise carbohydrates and fats (Chi *et al.*, 1983). Other changes include a rise in high density lipoprotein cholesterol (HDL-C) levels, a lowering of triglycerides and low density lipoprotein cholesterol levels, and high insulin sensitivity to fasting glucose (Petibois *et al.*, 2004).

2.2.4.1 Substrate Availability and Utilisation

Short-term detraining is characterised by an increased respiratory exchange ratio (RER) at both sub-maximal (Coyle *et al.*, 1984; Moore *et al.*, 1987; Madsen *et al.*, 1993) and maximal (Houmard *et al.*, 1992) exercise intensities. This is indicative of a shift towards a higher reliance on carbohydrate as a substrate for exercising muscles at the expense of lipid metabolism. This reliance on carbohydrate results in a concomitant reduction in aerobic performance. Such changes were demonstrated in endurance runners in the study by Houmard *et al.* (1992) who reported an increase in RER from 1.03 to 1.06 following 14 d of training cessation. These results are similar to those of Coyle and colleagues (1985) who investigated exercise at the same relative intensity to that prior to 21 d of inactivity. Detraining resulted in a significant increase in RER (0.93 to 0.97) indicating a significantly greater proportion of the total energy expenditure coming from carbohydrate oxidation in the detrained state. The contribution of fat oxidation to the total energy utilised during exercise decreased from 24% to 7%. A slightly longer training cessation period of 28 d also resulted in a significant increase in RER from 0.89 to 0.91, when cycling at 75% of $\dot{V}O_{2max}$ (Madsen *et al.*, 1993). These results, when evaluated together, are a clear indication of a shift towards an increased reliance on carbohydrate as an energy substrate for exercising muscles with a concomitant decrease in lipid metabolism following periods of detraining.

Petibois and Déléris, (2003) studied 10 highly-trained rowers throughout a 52-wk detraining period. Their sub-maximal test consisted of rowing 18 km at 75% of maximum aerobic velocity determined 2 days prior to each testing session. They found that only 35 d of detraining resulted in lower adipose tissue triglyceride delivery during exercise. This reduction did not however, represent a direct metabolic limit to exercise as the increase in liver triglyceride delivery allowed total fatty acid concentration to remain unchanged. Following the 52-week detraining period, their main finding was an altered metabolic response to exercise with a decrease in fatty acid availability. The authors suggested that this reduction in fatty acid availability was responsible for the increase in glycogen utilisation observed. The only other available data on the metabolic response to long-term detraining in highly-trained athletes also showed higher respiratory exchange ratio values during a steady state exercise test, indicating an increased utilisation of carbohydrate (Coyle *et al.*, 1985).

The decrease in lipid metabolism has been reported to be partially due to sensitivity to insulin-mediated whole-body glucose uptake which decreases rapidly with inactivity (Mikines *et al.*, 1989). This results in a reduction in the ability to metabolise lipids (Costill *et al.*, 1985a; McCoy *et al.*, 1994). Mikines *et al.* (1989) investigated the effects on insulin action of 5 d of inactivity in 7 trained endurance athletes. They observed that sensitivity for insulin-mediated whole-body glucose uptake significantly decreased after detraining to levels comparable with the sedentary population. This was paralleled by a decrease in whole-body glycogen synthesis at sub-maximal insulin concentrations and in glycogen synthase activity in the muscles. From the above evidence it is clear that the aerobic performance of soccer players in the detrained state will be affected by their inability to conserve muscle glycogen and plasma glucose. This is due to an increased reliance on carbohydrate as an energy substrate for exercising muscles with a concomitant decrease in lipid metabolism following periods of detraining. No studies can be found which investigate the time-course of the return to training levels following these negative adaptations in soccer players.

2.2.4.2 Blood Lactate Kinetics

The increased utilisation of carbohydrate during exercise of the same absolute and relative intensities before and after detraining is accompanied by a greater accumulation of lactate in the blood (Coyle *et al.*, 1985; Claude and Sharp, 1991). Significantly larger increases in blood lactate at the same relative exercise intensity have been observed after as little as 12 d of detraining in previously endurance-trained runners and cyclists performing a sub-maximal

exercise test (Coyle *et al.*, 1985). Penny and Wells (1975) found a significant increase in blood lactate levels after 21 d of a 63-d detraining period suggesting that blood lactate changes, in response to a steady-state exercise test, occur relatively quickly after training cessation. These findings were confirmed by Costill *et al.* (1985b). They observed that, after a standard 183 m swim at 90% of the swimmers' best time for that distance, blood lactate rose from a mean of 4.2 (±0.8) in the trained state to 9.7 (±0.8) $mmol.l^{-1}$ following 28 d of detraining. This increase was accompanied by a lowered bicarbonate level, resulting in a base deficit and a higher post exercise acidosis.

The above results are indicative of a reduction in the muscles oxidative capacity. This reduction is due to reduced metabolic efficiency with changes in blood flow and oxygen delivery and a resultant reduction in lactic acid removal (Hurley *et al.*, 1984). Similar findings have been observed by Neufer *et al.* (1987), also in competitive swimmers. The increase in blood lactate during a submaximal 200 m paced swim after 28 d of detraining was 3.0 and 4.5 $mmol.l^{-1}$ higher than 2 groups which continued to train once or three times per week respectively. This suggests that maintaining a reduced training load during rehabilitation may moderate the increases in blood lactate at submaximal levels. This may be achieved through partially maintaining capillary density which preserves optimal exchange of substrates and metabolites.

The lactate threshold also occurs at a lower percentage of $\dot{V}O_{2max}$ in the detrained state. Coyle *et al.* (1985) reported that the percentage $\dot{V}O_{2max}$ at lactate threshold declined progressively with inactivity. They found that the largest decrease was most evident in the first 57 d of detraining (79% to 74% of $\dot{V}O_{2max}$); thereafter they reported only a small further decline up to 90 d. Following this time point it stabilised at a level that was still higher than that of untrained control values of 62%. The same authors showed that the decline in $\dot{V}O_2$ at lactate threshold was closely related to both the decline in mitochondrial enzyme activity (r = -0.98) and the increase in total lactate dehydrogenase activity (r = -0.99). In a more recent meta-analysis study, Londeree (1997) investigated the importance of training intensity for improving and maintaining lactate threshold. Although only 4 studies could be found that utilised elite athletes as subjects, it was confirmed that there is a significant decline in the lactate threshold with training cessation. The intensity of a significant proportion of an elite soccer match is played around the lactate threshold so any reduction in a player's lactate

threshold following injury may have a significant effect on performance on returning to competition.

2.2.4.3 Muscle Glycogen

Fatigue during a soccer match is partially related to the availability and the ability to utilise muscle glycogen (Rico-Sanz *et al.*, 1999). The type of exercise and the length of a soccer match make it quite obvious that the level of muscle and liver glycogen stores will be important in both performance and endurance in soccer, particularly in the second half of the game (Ekblom, 1986). Any decrease in glycogen stores will therefore impact on the player's ability to withstand the demands of a competitive match for the full 90 minutes. This may result in performance decrement and may increase the likelihood of injury.

Glycogen concentration in skeletal muscle is higher in the trained compared to the untrained and declines rapidly with detraining (Hawley, 1987). This was demonstrated by Costill *et al.* (1985) who noted a 20% decline (153 ± 3 to 123 ± 2 mmol$\cdot$kg^{-1}) in levels of muscle glycogen in the deltoid muscle after only 7 d of training cessation in competitive swimmers. Glycogen concentration levels continued to decline by 8 to 10% per week in the remaining weeks of a 28-d detraining period (111 ± 8 and 93 ± 7 mmol.kg^{-1} after 2 and 28 d respectively). This may be due to the decrease in glucose to glycogen conversion and glycogen synthase activity (Madsen *et al.*, 1993).

These findings highlight the importance of structured rehabilitation training following injury. This type of programme may contribute to the re-establishment of maximal muscle glycogen concentration levels prior to return to competitive match performance and training following lengthy absence. Without this, the ability to maintain match performance or training load may be compromised and the risk of injury increased.

2.2.4.4 Endocrine responses to detraining

Literature regarding the effect of detraining on endocrine response to detraining is limited and has focused primarily on insulin and the catecholamines both at rest and during exercise (Houmard *et al.*, 1989). Insulin stimulates the uptake of glucose and amino acids by muscle cells and also promotes the conversion of carbohydrate into fat (Iozzo *et al.*, 2003). Exercise stimulates sympathetic nervous system activity, which leads to increases in plasma adrenaline and noradrenaline (Harmer *et al.*, 2000). In turn, elevated levels of plasma catecholamines

induce several cardiorespiratory and metabolic responses including increases in heart rate and ventilation, inhibition of pancreatic β-cell secretion of insulin and mobilisation of glucose and lipids as energy substrates (Stuart *et al.*, 2005)

When exposed to a glucose load, physically trained individuals secrete less insulin than untrained subjects (Liu *et al.*, 2008). This would suggest that the insulin response is more sensitive in trained versus untrained individuals. This is important as it promotes more efficient glucose, lipid, and amino acid metabolism (Liu *et al.*, 2008). There have been a number of reports of a rapid decline in sensitivity for insulin-mediated whole-body glucose uptake during short-term inactivity (Mikines *et al.*, 1989; Vukovich *et al.*, 1996). Three d (LeBlanc *et al.*, 1981) and 5 d (Mikines *et al.*, 1989) without training are enough to decrease insulin sensitivity in endurance athletes to levels found in untrained subjects. Burstein *et al.* (1985) found that a decrease in insulin binding to erythrocytes after 60 h of detraining was almost entirely accounted for by a decrease in the number of insulin receptors. This would suggest that the changes in insulin sensitivity may be at least partially mediated by changes in insulin receptor numbers. Sensitivity to whole body, insulin mediated, glucose uptake may also be as a result of reduced GLUT-4 content as this was shown to decrease in the first 6-10 d (Vukovich *et al.*, 1996). These findings suggest that the increase in peripheral insulin action seen in trained athletes is rapidly reversed.

The effects of short-term detraining on hormones is also characterised by similar decreases in catecholamines levels. This more efficient catecholamine response has been associated with higher catecholamine clearance (Kjaer *et al.*, 1992) as well as a reduced sympathetic nervous system response. These adaptations result from less relative stress and better ability to maintain plasma glucose (Mazzeo, 1986). Using the hyperglycaemic clamp technique to assess catecholamine response in endurance trained athletes, Mikines *et al.* (1989) reported that the plasma concentrations of epinephrine and norepinephrine were similar before and after 5 d of detraining. A time course for changes in catecholamines response has been shown by Coyle *et al.* (1985). After 21 d of inactivity epinephrine and norepinephrine concentrations did not differ from the trained values. After 84 d of detraining, however, epinephrine (244 ± 55 vs. 316 ± 128 pg/ml) and norepinephrine (2346 ± 324 vs. 2521 ± 584 pg/ml) levels were significantly greater than trained values suggesting a less efficient catecholamine response during submaximal exercise.

2.2.5 Effects of detraining on other performance parameters relevant to soccer

2.2.5.1 Changes in Body Composition

The average body fat of top players is between 8-10% during the playing season (Mangine *et al.*, 1990; Morgan *et al.*, 2005), but can rise to 19 to 20% between seasons (Reilly, 1994b). Given that about 5% body fat is common in distance runners (Shephard, 1999), it may be inferred that soccer players have traditionally carried more than an optimal amount of bodyfat, and that between seasons, in particular, it is important to limit its accumulation. Body composition is an important aspect of fitness in soccer (Junge *et al.*, 2000) with excess fat mass a disadvantage to performance (Morgan *et al.*, 2005) because of the negative effect on energy expenditure and sprinting performance (Árnason *et al.*, 2004a). Body fat percentage is also a discriminatory factor between levels of competition in soccer (Brewer and Davis, 1991). Krustrup *et al.* (2006) suggested that body composition also played a role in intermittent high-intensity, predominantly aerobic performance in soccer players.

Ostojic (2003) investigated changes in percentage body fat and 50 m sprint time in 30 professional soccer players throughout the preparation and competitive phases of a Serbian soccer season. He reported a strong correlation between changes in body fat percentage and 50 m sprint time ($r=1$, $p<0.05$). The players' percentage body fat was significantly lower and sprint times significantly quicker at the end of the season when compared with the beginning of the preparation phase (9.6 ± 2.5% vs. 11.5 ± 2.1% and 7.1 ± 0.5 s vs. 7.5 ± 0.6 s respectively) confirming the link between excess body fat and performance decrement.

The findings with regard to changes in body mass and estimated percentage body fat following short-term training cessation in trained athletes is equivocal. Hortobágyi *et al.* (1993) found no significant differences in body mass and estimated percentage body fat following a 14-d detraining period in power athletes. These findings agreed with Houston *et al.* (1979) and Simsolo *et al.* (1993) who also both found no notable body mass or percentage body fat changes in trained endurance runners following a similar period of detraining. Even over longer detraining periods of 21 d (La Forgia *et al.*, 1999), 28 d (Houmard *et al.*, 1990), 42 d (Allen, 1989; Kraemer *et al.*, 2002), and 18 wk (Green *et al.*, 1980) no changes in body fat percentage or body mass in trained individuals have been reported.

Although athletes may not be inclined to increase their body fat percentage as a result of short-term detraining, performance can suffer in those who do. It has been demonstrated that an athletes' percentage body fat may impact on performance. Árnason *et al.*, (2004a) examined 306 male soccer players from 17 teams in the two highest divisions in Iceland. They suggested that there was a trend ($p<0.07$) between the soccer teams average percentage body fat and team success. These findings were confirmed by Kalapotharakos *et al.* (2006) in their review of the physiological characteristics of elite Greek professional soccer teams. Estimated body fat percentage in the team which finished top of the division, as determined using skinfold callipers, was significantly lower than a team which finished mid-table and another which finished bottom (9 ± 2, 11 ± 2, 11 ± 2 % respectively). When dealing with injured soccer players therefore, it may be prudent to take steps to ensure body fat percentage does not increase. This can be achieved by engaging in a moderate level of activity and controlling the diet (Liu *et al.*, 2008).

2.2.6 Summary

This chapter has demonstrated that detraining is a multifaceted physiological state with multiple mechanisms interacting to contribute to changes in athletic performance. It can be observed from the data included that there are contradictory findings throughout the detraining literature. These discrepancies highlight the difficulties in comparing detraining studies. The length and model of the detraining period, the training history, trained status, the type of training undertaken by the subjects and testing procedures all impact on the outcome of physiological responses to the reduction in habitual training.

A consensus of outcomes which characterise the effects of detraining in the elite athlete population however, gives an appreciation of the probable consequences of alterations in systematic training patterns. Significant declines in cardiovascular function and the muscles metabolic potential occur within days of cessation of training (Neufer, 1989). These early responses to detraining appear to include an early reduction in $\dot{V}O_{2max}$. A rapid reduction in blood and plasma volumes which, in turn, limits cardiac filling and stroke volume contributes to this decrease in $\dot{V}O_{2max}$. Even though heart rate increases at both submaximal and maximal levels, this increase is not sufficient to counterbalance the reduced blood volume and therefore maximal cardiac output declines. Cardiac dimensions and ventricular volume may also be affected by detraining. The total oxygen carrying capacity is therefore reduced as

a result of a combination of these factors. Concomitant with this reduction in blood flow is a rapid reduction in oxidative muscle enzymes (e.g. citrate synthase, β-hydroxyacil-CoA dehydrogenase, succinate dehydrogenase and malate dehydrogenase) reducing the muscle's oxidative capacity. Although these adaptations result in a rapid decline in endurance performance in trained athletes, the decline levels off and remains above that of sedentary or recently trained subjects.

Skeletal muscle morphological responses to detraining appear to be a little more robust than cardiovascular indices with strength performance generally retained for up to 28 d. Extended periods of detraining may be required to significantly affect muscle fibre type and cross-sectional area which appear to be unchanged during the initial weeks of inactivity. The cross-sectional area of type II muscle fibres is more sensitive to reductions in training than type I fibres with the greater percentage of muscle atrophy found in type II fibres. With detraining periods of up to 56 d there may be a decreased proportion of type I fibres and a large shift from type IIa to type IIb in endurance athletes and an increased oxidative fibre population in strength-trained athletes. Reduction in capillary density however, may occur within 21 d of training cessation and may be a contributory factor to early aerobic performance decrements. Muscle glycogen concentration also declines rapidly within as little as 10 d of detraining.

Metabolically, the working muscle's capacity to utilise lipid energy stores, thus sparing glycogen supplies, are also negatively affected within 21 d of training cessation as indicated by an increased respiratory exchange ratio. An increase in insulin sensitivity and a reduction in muscle GLUT-4 transporter content results in a reduction in whole-body glucose uptake. In addition muscle lipoprotein lipase activity decreases. Detraining also affects blood lactate concentrations, increasing concentration at submaximal levels and lowering the percentage of $\dot{V}O_{2max}$ at which the lactate threshold occurs.

To put these findings into the context of injured soccer players, it would appear that most physiological adaptations occur in the early stages of detraining with further negative adaptations being less pronounced. This may mean that some negative adaptations in players injured for 28 d may not be significantly different to those injured for 56 d. This being the case, every effort should be made to reduce the effects of injury as early in the rehabilitation programme as possible.

CHAPTER 3

The epidemiology and aetiology of injuries in professional soccer

3.1 The development of the methodological procedures for investigation into the epidemiological of soccer injuries.

3.2 The influence of first hand data collection and accurately timed exposure on injury frequency rate.

3.3 Is injury the major factor for absence in professional soccer? An investigation into players being unavailable to train and play matches.

3.4 The injury profile of an English professional soccer club over 5 seasons.

Chapter 2.1 highlighted problems with comparing epidemiological studies of injuries in professional soccer. A comprehensive data collection method was required to determine the injury frequency rate in the population utilised in this thesis. The following chapter details the design of such methods. To demonstrate the issues of methodological inconsistencies, previous published literature were retrospectively analysed. The new methods were then utilised to provide a picture of the accurate incidence of injury in soccer with special reference to injuries which limit physical activity for extended periods.

3.1 THE DEVELOPMENT OF METHODOLOGICAL PROCEDURES FOR THE INVESTIGATION INTO THE EPIDEMIOLOGY OF SOCCER INJURIES.

The basis for investigations into the incidence of injury in English professional soccer players is a sound data base of the aetiology and epidemiology factors of injuries. In order to compile this data base, an accurate and comprehensive record of epidemiological and aetiological information is required. It is important that this data base is sufficiently wide ranging, detailed and accurate to provide any data for requirements for subsequent experimental chapters. This section details the methodological processes utilised to ensure the availability of accurate and comprehensive data.

3.1.1 Method of data collection

The period covered by the experimental investigations in this thesis is 9th August 2003 to the 31st December 2008. The study period for each discrete experimental investigation started on the first day and ended on the final day of each relative competitive season, unless otherwise stated. In all cases the start of the season was the second Saturday in August. In all but one season, when the club qualified for the league play-offs, the final match was the first Saturday in May. Players injured at the start of any of the study periods were not included in the relevant data set until they had returned to playing competitive matches. Absence data were collected on all injured players until the last day of the relevant study period. Each player was identified by a randomly assigned personal code in order to protect their confidentiality.

The basis for all epidemiological data collected was a comprehensive daily register (Appendix i). This was a paper based procedure and was required as part of the club's official record keeping procedures. The information collected was subsequently transferred onto a personal computer (Microsoft Excel, Microsoft Corp, Redmond, Washington). In general, attendance (when a players presence was required by the club for any reason) and absence (when a players attendance was not required by the club or if the player was unable to attend, irrespective of the reason for non-attendance) were recorded in the daily register. The details of the daily register included information on player participation in training or competitive

matches (including timed exposure in hours). These exposure hours were recorded for all contracted professional players throughout the period covered by the experimental investigations in this thesis (August 2003 to December 2008). Any reasons for a player's unavailability to train or play (e.g. injury, suspension, rest days off, illness, international duty, personal reasons and other absences) were also recorded. Clear definitions were agreed for all categories of player unavailability (Table 3.1.1).

Table 3.1.1 Definitions of absence categories

Reason for absence	Definition
Injury	*Any musculo skeletal condition or tissue damage, irrespective of cause, which prevents the player from participating in squad training or matches.*
Soccer injury	*Any soccer-related incident that resulted in medical attention regardless of the consequences with respect to absence from training or matches.*
Match suspension	*Not being available for first team selection as a result of a sanction from the relevant authority prohibiting match participation.*
Day off	*A day when attendance at the football club for training or match play was not required.*
Illness	*A day's absence as a result of an illness or disease. A doctor's confirmation note was not required.*
International duty	*A day's absence as a result of the player being called up to train with, or represent his country.*
Loan	*Occasions when a player was temporarily loaned out to another football club. A loan-day was recorded for each day's absence.*
Personal	*A days absence for personal or social reasons including family illness, child birth, bereavement etc.*

The researcher was in attendance at all training sessions and all competitive matches of both the first team and reserve team throughout the period covered by the experimental investigations. A training session was defined as 'any coach directed scheduled activity carried out with the team' (Hägglund *et al.*, 2006). All training incidents requiring medical attention, irrespective of subsequent time unavailable, were immediately recorded on the daily register. During competitive matches, all incidents resulting in on-pitch medical attention, which was defined as 'any attention provided to a player during a match by the team physiotherapist or doctor' was recorded. Medical attention at half time and after the match were also recorded. Incidents of match ending injuries were also recorded.

As timing of match injuries is considered important (see chapter 2.1.2.6.), on-pitch medical attention was accurately timed using a stopwatch (Uwin 100 series, Reydon Sports,

Nottingham). Only the first medical attention per match was recorded if the same injury required further treatment. If a player did not receive on-pitch medical attention but subsequent data from that injury were required, video analysis of match footage was used. The researcher and the injured player studied the official footage (Panasonic FRX 250, Germany) in an attempt to identify the circumstances and time that the incident that led to the injury took place. If these procedures did not permit an identification of the incident leading to the injury, the injury was recorded in a specific category labelled 'unknown timing'. This only occurred on 8 occasions during the 5 seasons covered by this thesis (season 1, 2003-04, n=1; season 2, 2004-05, n=2; season 3, 2005-06, n=2; season 4, 2006-07, n=3).

3.1.2 Calculating exposure

When considering injuries in soccer, the number of participants and the time spent at training and competition are essential epidemiological variables (de Loës, 1997). This information allows the expression of incidence rates per time unit exposure. It was therefore decided to record individual exposure as this has been shown to be crucial to be able to study the actual risk of injury (Waldén *et al.*, 2005).

3.1.2.1 Match exposure

Match exposure hours for all first team and reserve team competitive matches were calculated. To facilitate this, each individual player's participation in matches was accurately timed on a hand held stop watch (Uwin 100 series, Reydon Sports, Nottingham) and recorded. This individual exposure included 'additional' time added on by the referee for stoppages at the end of each half. Players not completing the full match because of being substituted or sent off had their actual participation exposure noted. In the event of matches going to 'extra' time (normally 2 periods of 15 min addition play) the additional exposure for each player was accurately timed and recorded. First team and reserve team match exposure were recorded separately. This enabled them to be analysed discretely or in combination. To allow injury frequency rate to be compared with previous literature that estimated exposure (Hawkins and Fuller, 1999; Rahnama *et al.*, 2002; Yoon *et al.*, 2004), an 'estimated match exposure' was also calculated using the equation;

100 minutes x 11 (players per team).

3.1.2.2 Training exposure

The researcher accurately timed each training session from the start of the warm-up to players leaving the training pitch. A hand-held stopwatch (Uwin 100 series, Reydon Sports, Nottingham) was used to time each session. Training exposure was then calculated by multiplying the number of players participating in the training session by the session duration (min). Individual differences in the duration of squad training, due to early completion of the session or additional training, was then added or subtracted. Dividing the total by 60 gave accurate training exposure hours for the session. During competitive matches, the warm-up and cool-down periods were timed separately and recorded as training exposure (Fuller *et al.*, 2006). Matches between players at this club or friendly matches against other clubs were deemed non-competitive matches and recorded as training (Fuller *et al.*, 2006).

3.1.3 Diagnosing and recording type and severity of injuries

The definition of, and the type and severity of injury are important considerations in epidemiological investigations (de Loës, 1997). The anatomical structures injured, anatomical location and the severity of the injury also have a direct impact on the type and intensity of training a player is able to perform during rehabilitation. The following section discusses each of these variables.

3.1.3.1 Definition of soccer injury

The definition of soccer injury used in the investigations in this thesis as discussed in chapter 2.1.2.2 is:

> *"Any soccer-related incident that resulted in medical attention regardless of the consequences with respect to absence from training or matches"*

3.1.3.2 Diagnosing injuries

All injuries were prospectively diagnosed and recorded by the researcher who is a chartered physiotherapist specialising in sports medicine for 20 years. This procedure reduced bias through differences in injury interpretation, recall and changes in observation methods between practitioners. If, as a result of further diagnostic protocols such as imaging procedures, the diagnosis was revised, the relevant category in the records was amended.

3.1.3.3 Severity of injury

Severity of all injuries were categorised to reflect the time-lost from normal soccer training and playing matches. The categories were based on those used in previous literature (Hawkins *et al.*, 2001; Árnason, *et al.*, 2004c) with one adaptation. Major injuries are universally defined as injuries resulting in >28 d absence (Table 2.1.1). Having the most serious injury criterion set at 4 wk however, has the potential to coalesce a 5-wk hamstring injury and a 52-wk cruciate ligament reconstruction. Categorising the severity of injury is subjective and may be driven by the aims of the study, but the addition of an injury severity category of > 2 months would identify lengthy and possibly career threatening injuries. Another additional category of >60 d was therefore added in the current thesis to enable identification of those more serious, potentially career threatening injuries. The following definitions of injury severity (Table 3.1.2.) as discussed in 2.1.2.7, are therefore used throughout this thesis.

Table 3.1.2 Definitions of injury severity

Transient injury	*A soccer-related incident that required medical attention but did not result in loss of training or match participation apart from that lost in the training session or match in which the injury occurred.*
Non-recordable injury	*A soccer-related incident that resulted in 1 day's absence irrespective of a training session or match being timetabled.*
Slight injury	*A soccer-related incident that resulted in between 2 and 6 days absence irrespective of a training session or match being timetabled.*
Minor injury	*A soccer-related incident that resulted in between 7 and 14 days absence irrespective of training sessions or matches being timetabled.*
Moderate injury	*A soccer-related incident that resulted in between 15 and 28 days absence irrespective of training sessions or matches being timetabled.*
Major injury	*A soccer-related incident that resulted in between 29 and 60 days absence irrespective of training sessions or matches being timetabled.*
Severe injury	*A soccer-related incident that resulted in >60 days absence irrespective of training sessions or matches being timetabled.*

Collectively, all soccer injuries resulting in >1 d absence, not including the day of the injury, were termed soccer-related reportable injuries (SRRI). This timescale related to the legal requirement to report such injuries under the Reporting of Incidence, Disease and Dangerous Occurrence Regulations (1994). Using these guidelines, transient and injuries of <2 d absence were therefore non-reportable soccer injuries. A non-soccer related injury was recorded if a

player sustained an injury that was not related to training or match participation. Examples of these are accidents in the home or road traffic accidents, preventing them from training or competing.

3.1.3.4 Recording reportable injuries

In the event of a player not being available to fully participate for the second day following an injury, irrespective of whether a training session or match was timetabled, additional data were recorded on a specifically designed Injury Data Collection Form (IDCF). In order that the full impact of match ending injuries could be examined, an IDCF was also completed for all match-ending injuries irrespective of subsequent absence. This would help identify the types of injury causing premature cessation of match participation and may identify risk factors that interventions may be successful in reducing. The flowcharts for recording injuries for training (Figure 3.1.2) and matches (Figure 3.1.3) were followed. This information was then transferred onto a specially designed Excel spreadsheet (Microsoft Corp., Redmond, Washington) on a personal computer. Figure 3.1.5 provides an example of a blank IDCF. This figure clearly illustrates all categories utilised in the detailed recording of SRRI's. These categories included basic descriptive data such as date of injury and activity being undertaken when injured such as training or playing in a competitive match (source activity). Other categories enabled the classification of the cause of injury into traumatic or overuse injuries. A definition of overuse injury based on Orava (1980), as discussed in chapter 2.1.2.8, was used. This was;

> '*Any pain syndrome of the musculoskeletal system with insidious onset and without any known trauma or disease that has given previous symptoms that were initially insufficiently severe to inhibit participation but, over time, the severity increased resulting in the inability to train and/or play'.*

The IDCF also contained data such as categories for the mechanics of traumatic injuries based on Waldén *et al.* (2005) (Figure 3.1.5). Injuries sustained to the player's dominant or non-dominant side were noted. The player's dominant side was deemed to be the preferred kicking leg. A differentiation between intrinsic (person-related) and extrinsic (environment/third person related) causative factors was also made and recorded using categories suggested by Dvorak and Junge (2000). In match play, the outcome of any contact with an opposing player was categorised to indicate if foul play had occurred. Foul play was

defined as a situation during the game that was interrupted by the referee and deemed to be against the rules of play and that led to a free kick to either team (Jacobsen and Tegner, 2007). The referee's decision relating to any further sanction for the player responsible for the foul (in the form of a yellow or red card) was also noted.

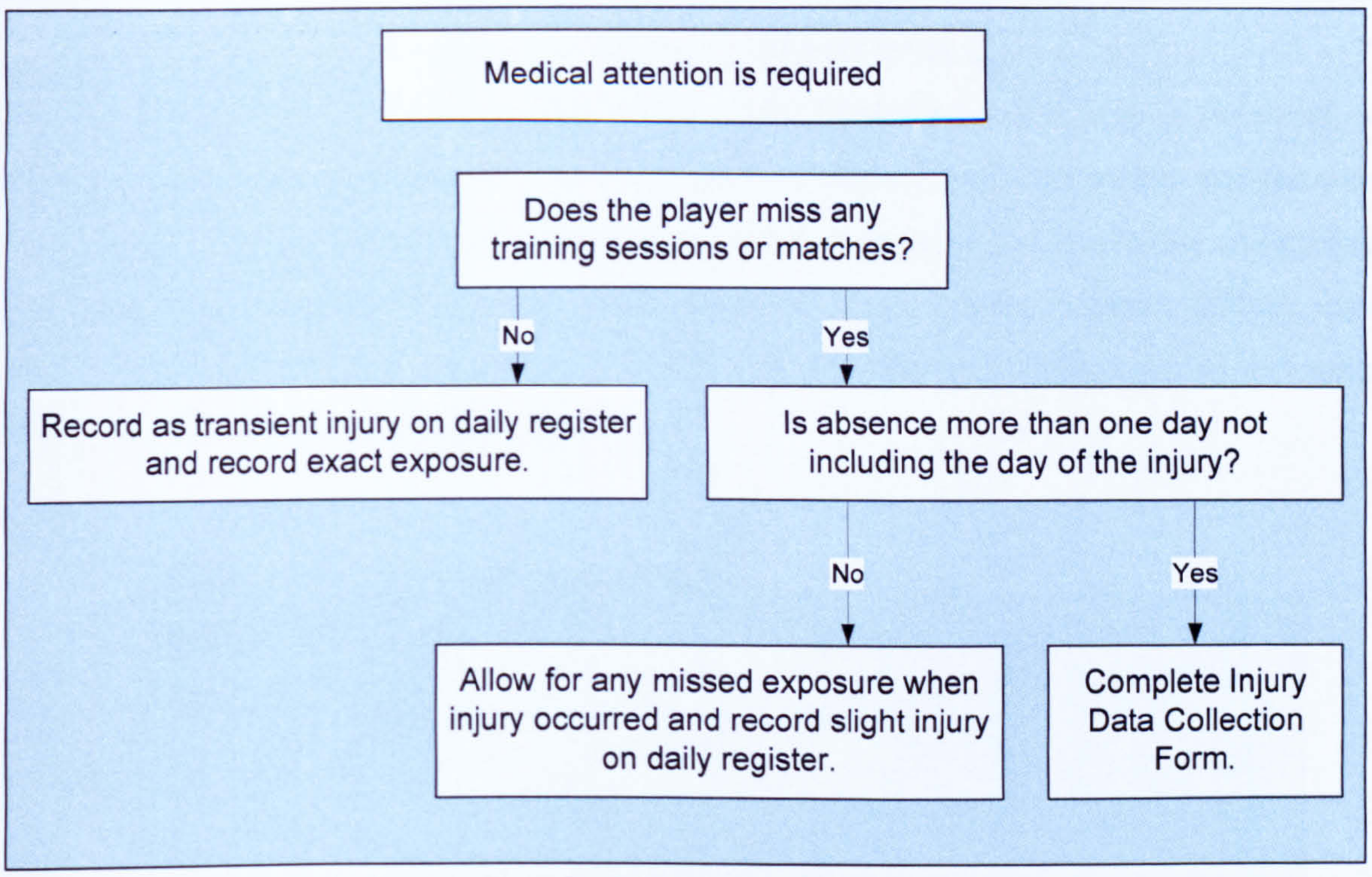


Figure 3.1.1 Flowchart for the recording of training injuries

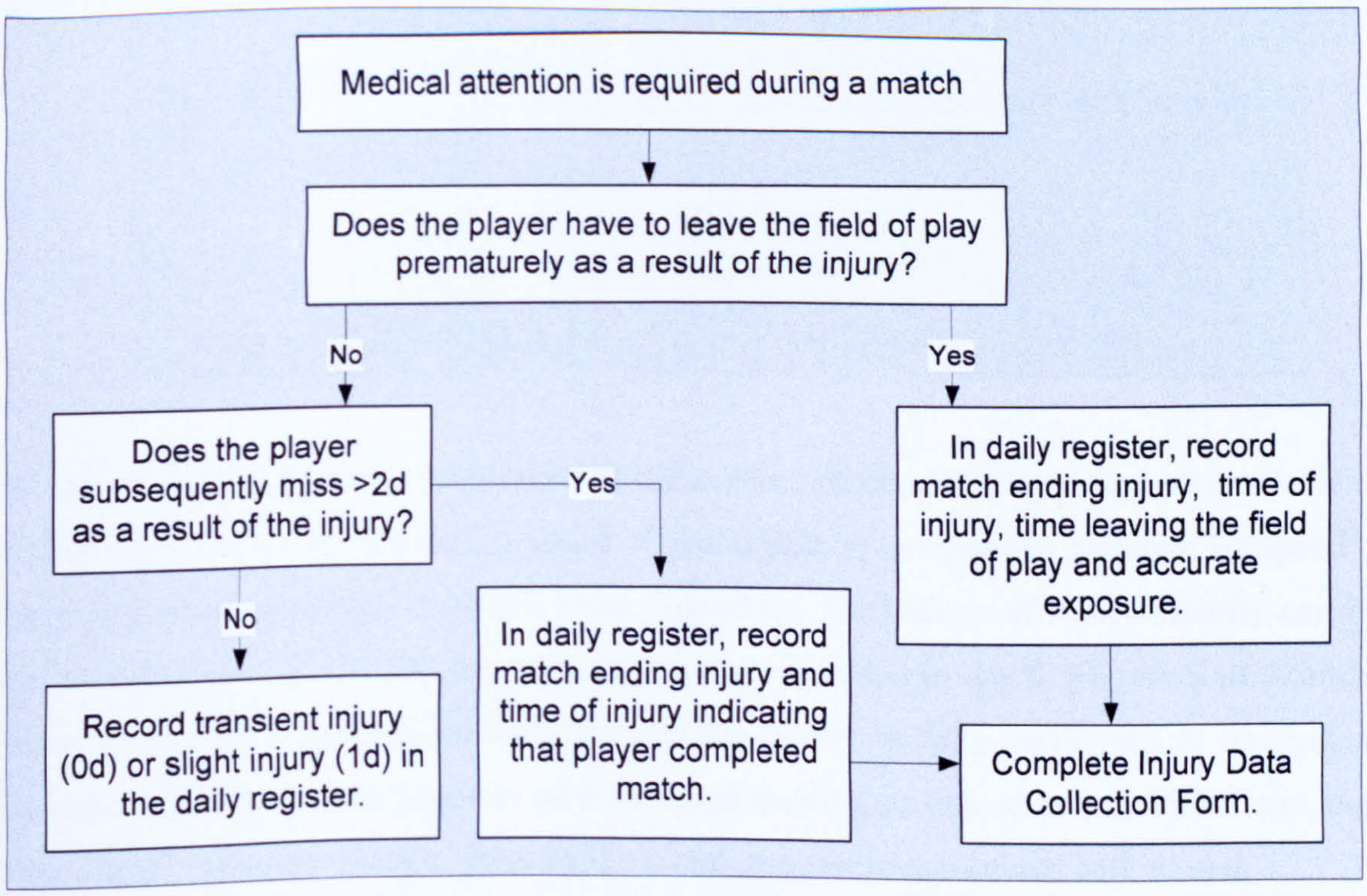


Figure 3.1.2 Flowchart for the recording of match injuries

The timing of match injuries was also recorded on the IDCF. The time of the specific incident leading to the injury and the time of the player leaving the field of play were noted following timing information recorded from a stopwatch. In many cases these were identical but on 50 occasions (season 2003-04 n=13; 2004-05 n=11; 2005-06 n=10; 2006-07 n=9; 2007-08 n=7) players sustained SRRI's and were able to stay on the field of play until later in the match or until the completion of the match. The above method enabled both injury time and the time the player left the field of play or completed the match to be recorded. To enable comparison with similar studies (Rahnama *et al.*, 2002; Andersen *et al.*, 2004a; Árnason, 2004c), each match was sub-divided into 8 sectors (Table 3.1.3). No injuries occurred during any extra time periods (n=2) during the study period.

Table 3.1.3 Match segment categories

Match sector	Timing
1	Start of the match to 15 minutes
2	16 to 30 minutes
3	31 minutes to the half time break
4	Half time break
5	Start of the second half (timed as 46 minutes) to 60 minutes
6	61 to 75 minutes
7	76 to end of normal 90 minute match
8	After the match

The severity of SRRI's was determined by the number of days the player was not available to train normally with the rest of the squad or participate in competitive matches, irrespective whether a training session or match were timetabled. Definitions of injury severity can be found in Section 3.1.3.3. The day of the injury was recorded as day 0. Players were deemed to be injured until the medical staff allowed the player to fully participate in training or matches. If a player participated in parts of squad training or trained in a modified way, e.g. other players were discouraged from tackling him, they were considered still injured.

In order that short-term and long-term sequelae could be investigated, links to previous injury were noted by examining previous injury records. Injuries which were deemed to be inopportune such as muscle contusions, lacerations and concussions were not considered when defining re-injury. The definitions that relate to re-injury, as discussed in chapter 2.1.2.3, that will apply to all investigations in this thesis are detailed in Table 3.1.4.

Table 3.1.4 Definitions for subsequent injuries.

Early re-injury	*Any soccer-related injury of the same type and to the same anatomical location within 2 months of the final day of rehabilitation of the index injury.*
Late re-injury	*Any soccer-related injury of the same type and to the same anatomical location within 2 to 6 months of the final day of rehabilitation of the index injury".*
Further injury	*Any soccer-related injury irrespective of type or anatomical location within 2 months of the final day of rehabilitation of the index injury.*

3.1.3.5 Player's age and playing position

Players' ages and age category (Hawkins *et al.*, 2001) were recorded. The categories were 18-21 y; 22-28 y; 29-34 y and >34 y. Player's normal playing positions were also determined prior to the beginning of each season. Each individual coach (n=7, all UEFA 'A' or 'B' Licence coaches) specified each player's normal playing position as either goalkeeper, defender, midfielder or striker on a standard form. These individual player positions were used throughout the relevant player's time at the club.

3.1.3.6 Anatomical location of injury

To enable comparative analysis with other studies which categorise the anatomical site of injury in a different way, the anatomical location of injury was categorised twice. One set of categories was broad with 10 categories. The second set of categories was more detailed and included 24 categories. For example, the 10-category method may categorise an injury as a thigh injury and the 24-category method would diagnose the injury into a more specific quadriceps, hamstring or adductor injury (column 4, Figure 3.1.5). Injuries were also categorised according to the nature of tissue damage sustained. Examples included strain, sprain, muscle contusion, etc. (Table 3.1.5).

Table 3.1.5	Definitions for categories of tissue damage
Sprain	*Acute distraction injury of ligaments or capsules*
Joint injury	*Acute chondral and meniscus lesions*
Strain	*Acute distraction injury of muscles or tendons*
Contusion	*Tissue bruising without concomitant injuries classified elsewhere*
Fracture	*Traumatic break of bone*
Dislocation	*Partial (subluxation) or complete (luxation) displacement of the bony parts of the joint*
Laceration/cut	*A cut or tear of the skin.*
Concussion	*An injury to the brain produced by a violent blow and followed by a temporary or prolonged loss of function.*

The above prospective data collection methods were designed in an effort to ensure that data could be compared with the wide range of methodologies utilised in other studies and the effectiveness of future investigations in this thesis was not compromised by inadequacies in the available epidemiological information. It was therefore envisaged that there would be epidemiological data collected that would not be utilised in any of the subsequent experimental investigations. For further clarity, Figure 3.1.4 uses examples of injuries to illustrate how the relevant data would be recorded.

Incident	Register entry
A player is kicked in training and has to sit out for 30 min. He recovers and completes the session and trains normally the following day.	The 30 min missed is allowed for in the player's individual training exposure and the injury is recorded as 'transient' on the daily register.
A player is injured and receives on pitch attention in a match but finished the match. The next day is a rest day but he still requires attention and wouldn't have been able to train. He trains the following day.	The full match exposure and the time of the medical attention are recorded, the injury is categorised as a non-reportable soccer injury and the following day's absence is recorded on the daily register.
A player sustains a match ending injury and is absent for the following 3 weeks.	The exact match timing of the injury and the match exposure is recorded and the injury are recorded as an SRRI of moderate severity on an IDCF.
A player stubs his toe in the shower causing a fracture which results in a 6 week absence.	The injury is recorded as a non-soccer related injury and daily absence is recorded on the register.
A player injures his ankle in the first half of a match and receives on pitch attention. In the second half he requires further attention on the same ankle and later in the match he is winded and requires attention. He doesn't miss any training sessions.	The injuries are recorded as 2 x transient injuries. The ankle counts as one injury although he received attention twice. The timing of both injuries is noted with the timing of the first ankle injury being recorded.
A player had been complaining about a shin problem for some weeks but had not missed any training sessions or matches. During a training session the pain became intolerable and he had to stop. He didn't participate in training or matches for a further 3 months.	The player's precise exposure for that training session is recorded. The injury is recorded on an IDCF as an SRRI of severe severity and categorised as an 'overuse' injury.

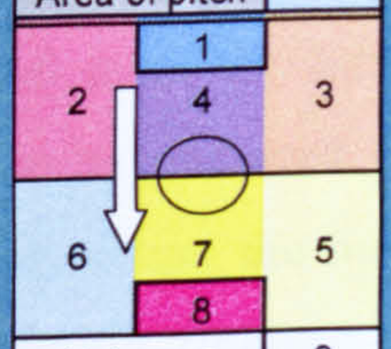
Figure 3.1.4 Examples of recording different circumstances leading to injuries.

Player:		Squad No.		Age:		Age Band	
Injury Date		Date 1st match				Data sheet No.	

Player Position	
Keeper	1
Defender	2
Midfielder	3
Striker	4

Source activity	
Training	0
First team	1
Reserves	2
Other match	3
Other cause	4

Venue	
Home	1
Away	2
Not in match	3
Injury sector	
Off sector	
Warm up	0
0-15 min	1
16-30 min	2
30-h/t	3
Half time	4
45-60 min	5
61-75 min	6
75 min-end	7
Finished Match	8
Unknown timing	9
Not in match	10

Area of pitch	
	
Unknown	9
Not in match	10
Player contact?	
Yes	1
No	2
N/A	3
Foul given?	
Yes, no card	1
Yes Yellow	2
Yes Red	3
No	4
N/A	5

Trau/overuse	
Traumatic	1
Overuse	2
Other	3

Ext/Intrinsic	
Extrinsic	1
Intrinsic	2
Other	3

Dom/non-dom	
Dominant	1
Non	2
N/A	3

Training	
Warm up	0
1st quarter	1
2nd quarter	2
3rd quarter	3
4th quarter	4
Cool down	5
Not in training	6
Surface	
Grass	1
Astroturf	2
Gym	3
Other	4
Footwear	
Rubbers	1
Studs	2
Blades	3
Trainers	4

Referrals	
Consultation	1
Minor Surgery	2
GA Surgery	3
Injection	4
Immobilisation	5
Plain x-ray	6
CT/isotope/MRI	7
Other referral	8
None	0
Absence code	
SRRI	1
non-SRRI	2
Non SR injury	3
Other	0

Mechanism	
Running	1
Twisting/turning	2
Tackling	3
Tackled/kicked	4
Collision/impact	5
Heading	6
Jumping	7
Kicking	8
Stretching	9
Viol conduct/elbow	10
Falling/landing	11
Indeterminate	12
Long standing	13
Other	14

Structure	
Muscle	1
Ligament	2
Tendon	3
Capsule/fascia	4
Bursa	5
Meniscus	6
Other soft tis	7
Bony	8
Other	9

Tissue damage	
Strain	1
Sprain	2
Muscle contusion	3
Fracture	4
Dislocation	5
Tissue bruising	6
Tear	7
Hernia	8
Cut/laceration	9
Degeneration	10
Inflammatory	11
Concussion	12
Illness	13
Infection	14
Other	0

Limb/joint		
Scalp	1	1
Face	2	
Neck	3	
Shoulder	4	2
Arm	5	
Elbow	6	
Wrist/Hand	7	
Ribs	8	3
Spine	9	
Abdoms	10	
Groin	11	4
Hips	12	
Pelvic girdle	13	
Adductor	14	5
Hamstring	15	
Quad	16	
Knee	17	6
Calf	18	7
Shin	19	
Ankle	20	8
Achilles	21	9
Foot	22	
Toe	23	
Other	24	0

Links to prev injury	
Same injury prev 2 mth	1
Same injury prev 6 mth	2
Diff injury prev 2 month	3
Diff injury prev 6 months	4
No injury link	5

Estimated grade	
<2 days = non reportable	0
2d-6d = Slight	1
7d -14d = Minor	2
15d-28d = Moderate	3
29d-60d = Severe	4
>60d = Major	5

Comments:

Figure 3.1.5 Injury Data Collection Form (IDCF)

3.2 STUDY 1: THE INFLUENCE OF FIRST HAND DATA COLLECTION AND ACCURATE TIMING OF EXPOSURE ON INJURY FREQUENCY RATE.

3.2.1 Introduction

The literature review highlighted that professional soccer players suffer from relatively high rates of injury compared with participants in other sports. Researchers in the field of soccer injury epidemiology suggest that studies on the aetiology of soccer injuries require a conceptual model based on sound methodological principles (van Mechelen *et al.*, 1992). Such considerations are founded on the definition of the injury, the source of the information collected, and the availability of accurate exposure data (Junge *et al.*, 2004). Without this reliable information base, the accuracy and significance of other considerations such as anatomical, pathological, aetiological and severity parameters can be questioned.

The interpretation of the term 'injury' and the data collection system are the most critical methodological variables in soccer injury studies (Poulsen *et al.*, 1991). The definition of injury, especially the time absent element of the definition, affects the number of cases that meet the inclusion criteria and can subsequently have a significant effect on the results presented. Soccer injuries are often expressed as an injury frequency rate (IFR) by calculating the number of new injuries per 1000 h participation in soccer matches and training (Inklaar, 1994a). Both the definition of injury and the accuracy of timing exposure can however, be affected by the method of data collection (Chapter 2.1.2.4). When calculating IFR therefore, it is therefore important that the most comprehensive methods for collecting injury and exposure data.

The aim of the present study was therefore to determine which methods of collecting data and calculating exposure were the most appropriate for the current population. This was

achieved by using a comprehensive data set to compare previously published methods of data collection and exposure calculation as detailed in Chapter 3.1. Results from this study formed the basis for calculating accurate incidence and injury frequency rates in the remaining investigations in this thesis.

3.2.2 Methods

This study was conducted at an English Division One club during the 2003/2004 domestic season. All contracted professional players (n=28) (age 24 ± 5 years; height 1.8 m ± 0.1 m; body mass 81 kg ± 7 kg) agreed, in writing in accordance with Liverpool John Moores University ethical procedures, to allow the use of their injury and physical activity data to be included in the present study.

At the beginning of the study, all contracted players (n=25) were able to train and play. During the study, 3 players left the club and 3 joined. At the end of the study period one player was injured and unable to train. His data was included until the last day of the study period. A total of 274 d spanned the first and last competitive match of the season, 22 (88%) players were at the club for all 274 d. The 6 players who joined or left the club averaged 154 ± 64 d.

The general methods for this study are described earlier (Chapter 3.1). Only methods specific to this study will be presented in detail here in this chapter.

3.2.2.1 Defining injury

In the current study a soccer-related reportable injury (SRRI) was defined as an incident during training or match-play that prevented a player from participating in normal training or competition for >1 d, not including the day of injury (Lewin, 1989; Hawkins *et al.*, 2001; Woods *et al.*, 2003). In order to meet the aims of the study, the effect of different definitions of injury had to be explored. These definitions differed mainly as a consequence of the temporal component of their injury definitions. Three separate

definitions of injury, based on those most frequently employed in other epidemiological studies, were applied to data analysis. These definitions were;

Definition A.

"Any football related injury resulting in a player requiring medical treatment, irrespective of subsequent time absent" (Lüthje et al., 1996; Dvorak and Junge, 2000; Junge et al., 2004; Yoon et al., 2004).

In the current study this would include all injuries.

Definition B.

"Any football related injury resulting in a player missing the next training session or match" (Ekstrand and Gillquist, 1983b; Nielsen and Yde, 1989; Engström et al., 1991; Lilley et al., 2002; Hägglund et al., 2003; Witvrouw et al., 2003; Ekstrand et al., 2004a; Árnason et al., 2004c; Hägglund et al., 2005).

In the current study, this would include SRRI's and non-reportable injuries and exclude transient injuries.

Definition C.

"Any football related injury resulting in a player missing more than 48 h not including the day of injury" (Lewin, 1989; Hawkins et al., 2001; Woods et al., 2003).

In the current study, this would only include SRRI's and exclude non-reportable and transient injuries.

3.2.2.2 Methods of data collection

The researcher completed a comprehensive daily register for all contracted professional players throughout the 2003-4 competitive season. All attendance, including training and match exposure, and absence were recorded (Chapter 3.1.2.1). Any player injured at the start of the study period was not included in the study until they had returned to playing

competitive matches. Data were collected and included on injured players until the last day of the study period.

3.2.2.3 Timing exposure

Accurate match and training exposure were recorded according to the methods outlined in Chapter 3.1.2. Briefly, both individual player training sessions and match exposure were accurately timed using a stopwatch (Uwin 100 series, Reydon Sports, Nottingham). For comparison with studies that estimate exposure hours, an 'estimated match exposure' was calculated as 100 minutes x 11 (players per team). For comparison with studies that utilise monthly incidence of injuries to denote exposure (Lewin, 1989; Hawkins *et al.*, 2001; Woods *et al.*, 2003). The month of May's data were excluded from total match and training exposure hours. This was then divided by the nine months of the study and recorded as 'mean match exposure' and 'mean training exposure' (The study period ended on the 7th May).

3.2.2.4 Recording injuries

Injuries were recorded using methods of outlined in Chapter 3.1.1. Briefly, all training incidents requiring medical attention, irrespective of subsequent time unavailable, were immediately recorded. During competitive matches, all incidents resulting in on-pitch medical attention and incidents of match ending injuries were accurately timed (Uwin 100 series, Reydon Sports, Nottingham) and recorded.

Statistics

All analyses were performed using SPSS Version 11 for Windows (Chicago, Illinois, USA). All data were expressed as mean $\pm SD$. Paired-sample t-tests were conducted to investigate the different methods of calculating exposure. Differences in calculating IFR using the 3 definitions of injury was conducted using a one-way between groups Analysis of Variance. Statistical significance was accepted as $p < 0.05$ unless stated otherwise.

3.2.3 Results

3.2.3.1 *Exposure to training and competitive matches.*

During the study period 57 first team and 32 reserve team competitive matches were played generating 1391 match exposure hours (First team 990 h, reserve team 401 h). Accurately timed mean exposure to competitive match-play for individual players was 35 ± 30 h for first team and 15 ± 15 h for reserve team matches. The mean duration for all competitive matches including time added on at the end of each half was 94 ± 1 minutes. There were 6 matches (8%) when a player was sent off and therefore 11 players did not finish the match. The mean exposure for these players was 57 ± 24 minutes.

When comparing the effects of estimating match exposure, total estimated match exposure hours (1507 h) was 8% greater than total accurately timed match exposure hours (1391 h). Mean monthly estimated match exposure hours were greater than accurately timed match exposure hours (estimated 151 ± 15 h, accurately timed 139 ± 14 h; $t(9) = -7.0$, $p<0.0005$) (Figure 3.2.1).

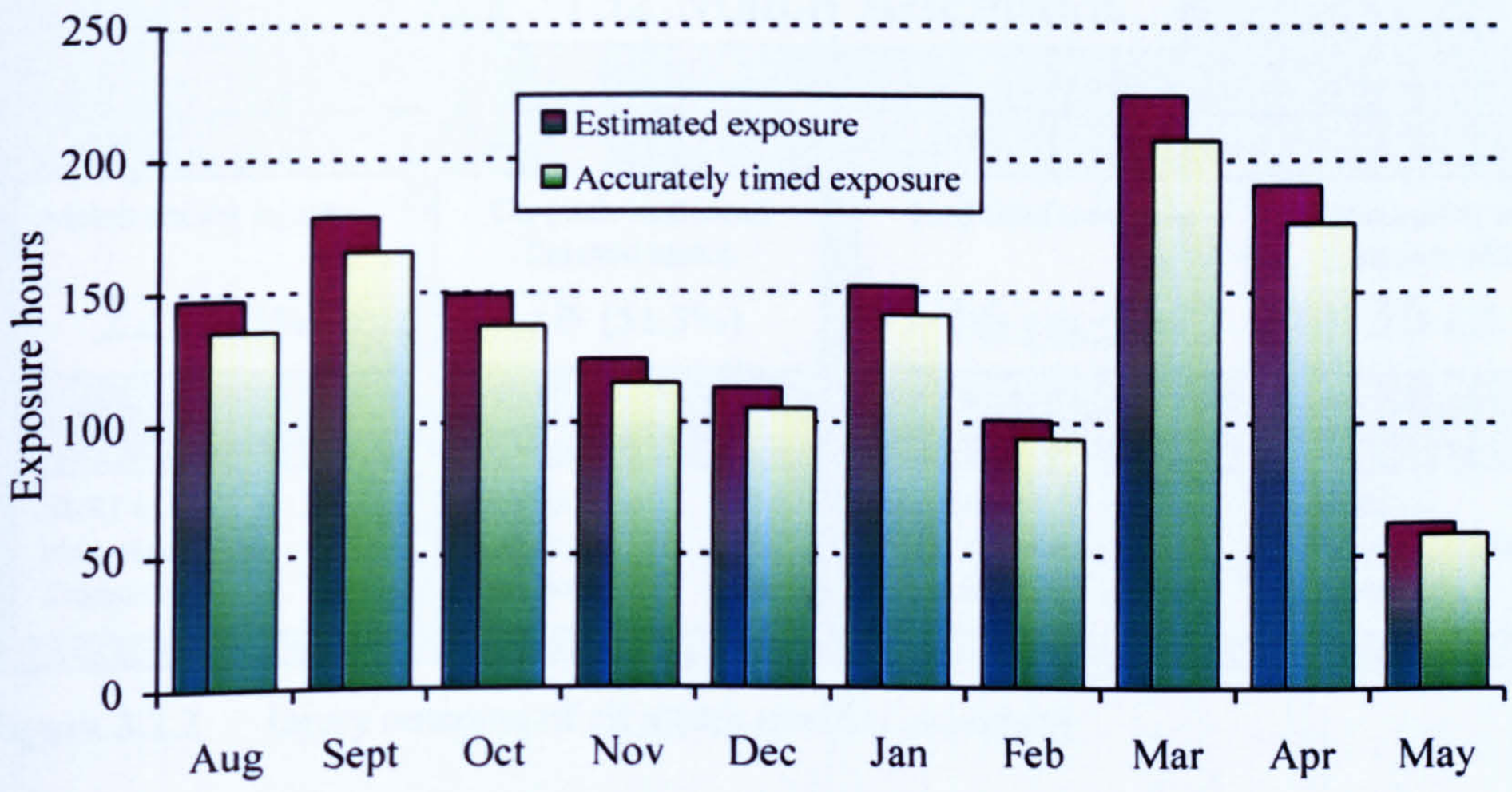


Figure 3.2.1 Monthly distribution of estimated and accurately timed match exposure hours.

In the same period, 144 squad training sessions generated 3195 individual participations or 5970 training exposure hours. The mean duration of training sessions was 1.9 h. There were 17 occasions when a player did not complete the training session.

3.2.3.2 Incidence of injury and IFR

During the season, a total of 201 injuries according to the definitions used in this thesis (SRRI's n=39, non-reportable n=28, transient n=134) were sustained and recorded. Injuries resulting in absences of <2 d accounted for 76% of all injuries. There were 100 incidents (1.1 per match) of players requiring on-pitch medical attention. A further 52 players required medical attention at half time and after matches giving a total match IFR of 109 per 1000 h or an average 1.7 soccer injuries per match. Figure 3.2.2 details the outcome of these 152 incidents. Thirty one players required medical attention for transient injuries sustained during training (5.2 per 1000 h), and eight sustained non-reportable injuries (1.3 per 1000 h).

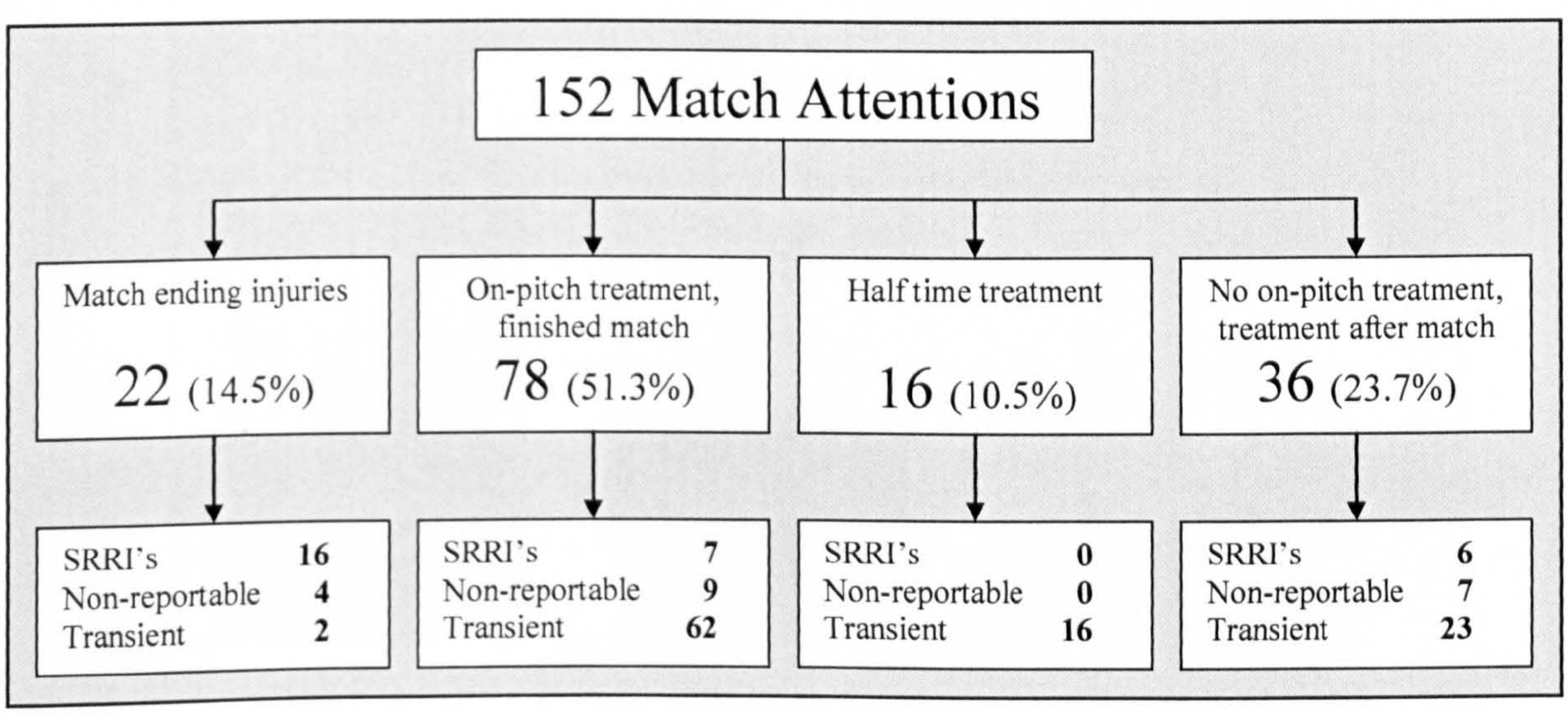


Figure 3.2.2 Injury outcome of all match medical attentions

Table 3.2.1 details the incidents and IFR for all injuries using definitions A, B and C. Analysis of the effect of injury definition on frequency of match and training injuries

demonstrated that utilising definition 'A' resulted in significantly more injuries than when utilising definitions 'B' and 'C' ($p<0.005$) (Figure 3.2.3). No difference was found between definition 'B' and 'C' ($p>0.05$).

Table 3.2.1 Number of injuries and IFR for each injury definition using accurately timed exposure

Definition	A		B		C	
	n	IFR	n	IFR	n	IFR
All matches	152	109.3	49	35.2	29	20.9
1st Team matches	129	131.2	35	35.4	20	20.3
Reserves matches	23	56.2	14	35	9	22.2
Training	49	8.2	18	3	10	1.7
Total	201	27.3	67	9.1	39	5.3

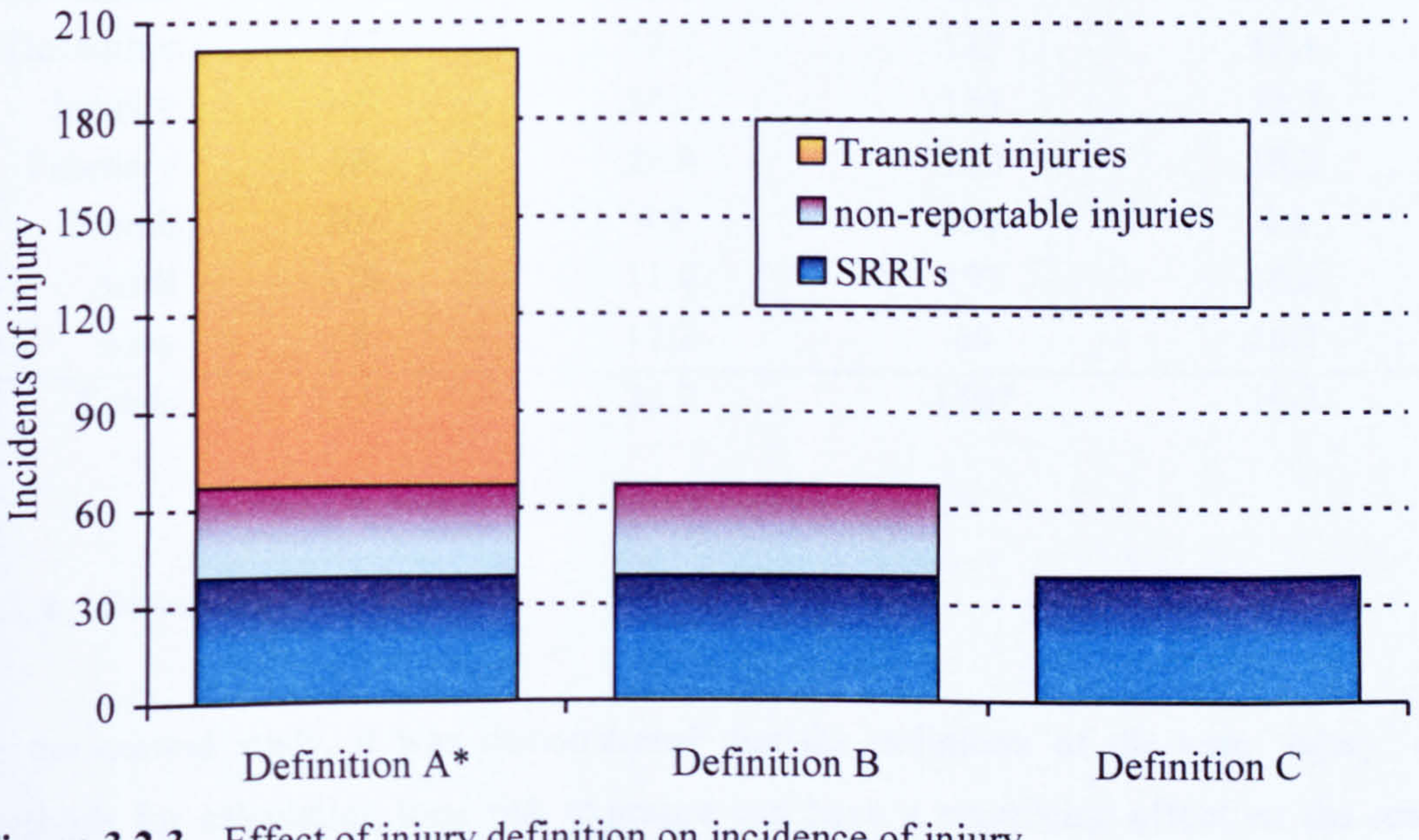


Figure 3.2.3 Effect of injury definition on incidence of injury
*Definition 'A' generated significantly more injuries than definitions 'B' and 'C' ($p<0.005$).

Table 3.2.3 utilises definition 'C' to illustrate the effect of calculating monthly IFR using accurately timed and estimated match exposure. Definition 'C' was chosen as this

definition is the same as the definition of SRRI in the current thesis. There was a statistically significant decrease in IFR from accurately timed (21 ± 4 injuries per 1000 h) to estimated mean monthly match exposure (19±4 injuries per 1000 h; t (9) =5.3, p<0.001). Further statistical analysis when averaging out accurately timed match exposure hours across calendar months found no differences between monthly exposure hours or monthly IFR (p>0.05). The completion of a similar analysis to evaluate the impact on IFR on training exposure hours across calendar months and accurately timed training exposure hours also found no differences in training hours (p>0.05).

Table 3.2.3 Influence on IFR of estimating and averaging monthly match exposure.

	Accurately timed		Estimated	
Month	Hours	IFR	Hours	IFR
August	145	27.6	160	25.0
September	185	10.8	195	10.26
October	152	46.1	162	43.2
November	118	25.4	130	23.1
December	107	18.7	117	17.1
January	147	34.0	153	32.7
February	96	20.8	110	18.2
March	207	4.8	227	4.4
April	176	11.4	193	10.4
May	58	17.2	60	16.7
Totals	1391	20.9	1507	19.2

3.2.4 Discussion

In the current study, it was demonstrated that the definition of the term 'injury' and methods for calculating total risk exposure can have a significant effect on the results collected in epidemiological studies on soccer injuries. The comprehensive data collection and analysis techniques applied in this investigation highlighted the effects of a number of different methodological approaches used in previous investigations on the frequency of

soccer injuries. The present data suggests that previous investigations that have estimated exposure may have reported an underestimation of the injury frequency rate. The current methods therefore seem to provide a more appropriate methodology for collecting data than previous approaches for this thesis.

3.2.4.1 Defining injury

Defining, categorising and quantifying injury in professional soccer is complex. This complexity may explain the lack of consensus as to a universal definition of soccer injury (Dvorak and Junge, 2000). The most common criterion for any definition of injury is an absence from training or match(es) (Witvrouw *et al.*, 2003). The current study recorded every incident that resulted in medical attention and categorised them according to recognised methods in the literature to enable comparisons with other epidemiological studies. Figure 3.2.3 illustrates the effects of utilising various definitions of injury on the incidence of injury. These data clearly illustrate that the definition of injury can have a significant effect on epidemiological indices such as IFR. Reporting the incidence of injury, using the same data set, can result in IFR ranging from 109 injuries (definition A) per 1000 h to 21 injuries (definition C) per 1000 h depending which definition is used. These differences highlight the importance of noting injury definition when comparing studies. The advantages of a uniform definition of injury would be ease of between-study data comparison and better knowledge of the actual injury rate in professional soccer.

Ekstrand and Karlsson (2003) stated that every definition of injury has advantages as well as disadvantages and that the choice of definition may depend on the purpose of the study. This can be illustrated using the current study as an example. Definition 'A' will identify all injuries but is both time consuming and resource intensive. The major benefit from using definition 'A' is that it enables comprehensive analysis that can be further manipulated for comparative analysis using definitions 'B' and 'C'. Definition 'B' identifies injuries that result in training or match absence but does not record transient injuries. These may be important as they could be a risk factor to subsequent injuries (Ekstrand and Gillquist, 1983b). Definition 'C' is the least resource intensive but will not

identify minor injuries resulting in <2 d absence, once again, ignoring what could be a significant risk factor to further injury. Data collection utilising definition 'A' is therefore recommended and time absent can then be utilised to categorise injury severity.

3.2.4.2 Risk denominator

The risk denominator for IFR of 1000 playing or training hours appears to be the accepted standard in recent epidemiological studies (Hägglund *et al.*, 2003; Hawkins and Fuller, 1999; Junge *et al.*, 2004; Hägglund *et al.*, 2005). The match IFR in the present study using definition 'B' (35 per 1000 h) was similar to that reported by Árnason *et al.* (1996) in their study of elite Icelandic players but lower than 46 per 1000 h reported by Yoon *et al.* (2004). Some studies use a slightly different approach than utilising exposure hours by examining the new-injury to total population ratio and reporting incidents in calendar months (Lewin, 1989; Hawkins *et al.*, 2001; Woods *et al.*, 2003). Although there may be occasions when these methods can be justified, the highest and most desirable level of data is that in which time at risk is considered (de Loës, 1997). The methods of reporting incidence in injuries/population ratio or calendar months may therefore have limited value when determining risk (Lindenfeld *et al.*, 1988). A risk denominator of 1000 h that is accurately timed is therefore recommended.

3.2.4.3 Incidence of injury

The current findings of 39 SRRI's for one competitive season appears to compare with 39.1 injuries per English club per season reported by Hawkins *et al.* (2001). However, their data included pre-season injuries, the current data did not. The discrepancy may be due to the high number of slight injuries in the current study. When examining the current data, the percentage of slight (<7d) injuries was 44% compared with 33% in Hawkins *et al.* (2001). This difference may reflect a reluctance of medical staff to complete lengthy questionnaires for short-term injuries and may highlight the advantages of first-hand data collection. The reason for the similarity may therefore be that the level of under-reporting may have matched the number of injuries sustained in pre-season. In the current study 79% of all players suffered at least one SRRI during the season. This compares well with

injuries to 82% (Peterson *et al.*, 2000; Dvorak *et al.*, 2000) and 86% (Lewin, 1989) of all playing staff during one competitive season using a similar definition. These data reinforce the belief that injuries in professional soccer are common with an average of 4 out of 5 players sustaining an injury each season. On-pitch treatments per match of 1.7 compares well with 1.7 reported by Wekesa (1995). These comparisons demonstrate that the base data of the present study compare well with other work and is therefore suitable for methodological comparisons.

3.2.4.4 Exposure hours

It is common in other studies to estimate match exposure hours, and in doing so, discount factors that can have an effect on match duration, i.e. calculating exposure time by multiplying 11 players per team by 100 minutes (Hawkins and Fuller, 1996; Rahnama *et al.*, 2002; Yoon *et al.*, 2004). In the current study, estimated exposure hours were significantly increased (8%) over accurately timed exposure. For example, one player in the present study completed 56 full matches resulting in an accurately timed exposure to risk of 88 h compared to 93 h that would be assumed if estimated exposure were used. This would result in an under calculation of the IFR as demonstrated in Table 3.2.2 and subsequently underestimate the extent of the injury problem being investigated.

There is insufficient information in the literature to compare methods of calculating training exposure. Methodological details of collecting training exposure range from no detail (Hoff and Martin, 1986), to 'recorded by the coach' (Árnason *et al.*, 1996) and calculated by multiplying the number of players training by the typical daily training programme (Hawkins and Fuller, 1999). The number of participants and the time spent at training and competition, including injured and uninjured players, are essential epidemiological variables, as they allow the expression of incidence rates per time unit exposure (de Loës, 1997). A risk denominator of accurately timed hours appears to be the most precise method to report the frequency of soccer injuries and will be used throughout this thesis.

3.2.4.5 Methods of data collection

Data collection is the most critical methodological variable in soccer injury studies (Poulsen *et al.*, 1991). Methods vary considerably ranging from daily contact with players (Lewin, 1989) to watching television and reading press reports (Hawkins and Fuller, 1998a). The current researchers were fortunate to be able to record all soccer injuries immediately and to accurately time match and training exposure. Logistics and manpower do not always make this approach possible so some studies have to rely upon third party, study detached recruits to complete regular protocol sheets (Hawkins *et al.*, 2001; Junge *et al.*, 2004; Yoon *et al.*, 2004). This method however, may result in incomplete returns. In their study of FIFA and Olympic soccer matches, Junge *et al.* (2004) reported an average response rate of 84% ranging from 47% to 100%. The reliability of data extrapolated from protocol sheets has also been questioned (Lindenfeld *et al.*, 1988; Twellaar *et al.*, 1996). It may therefore be possible that data collectors are reluctant to complete lengthy questionnaires or questionnaires for minor injuries.

To illustrate the effects of incomplete returns using current data, if only 50% of non-reportable injuries were recorded (in the current study 14 of 28), the overall IFR using definition 'B' would be reduced from 9 injuries per 1000 h to 7 injuries per 1000 h. Using retrospective questionnaires (Lilley *et al.*, 2002) is another popular method of data collection that can be characterised by inaccurate injury recall and over-estimation of exposure (Nielsen and Yde, 1989). Levy (1988) suggested that end-of-season data acquisition was suspect at best and Junge and Dvorak (2000) found that the IFR per player calculated retrospectively was only one third of that calculated on the basis of a weekly follow up. They suggested that the effects of memory as well as psychological defence mechanisms limited the value of retrospective evaluation.

3.2.5 Conclusion

The risk of injury in professional soccer is undoubtedly high. The aim of the current study was to determine which methods of collecting injury data was most appropriate for the current thesis. It was found that for the most precise calculation and comparative analysis of IFR, all soccer related medical treatments, irrespective of subsequent time absent, had to be recorded and player's individual exposure time accurately timed. It is however, recognised that the resources to construct a methodology of this extent is beyond the scope of many studies, and may not be required to meet the aims of some studies. However, the methods utilised in the current study have been found to be accurate and suitable for the extensive requirements of the current thesis and will therefore be adopted throughout this thesis.

3.3 STUDY 2: IS INJURY THE MAJOR FACTOR FOR ABSENCE IN PROFESSIONAL SOCCER? AN INVESTIGATION INTO PLAYERS BEING UNAVAILABLE TO TRAIN AND PLAY MATCHES.

3.3.1 Introduction

The proportion of a professional soccer squad available for first team selection can be affected by numerous factors including injury, illness, suspension and personal circumstances. Important players, players in key playing positions or an accumulation of players being unavailable for selection can tax playing resources. Such unavailability of players is frequently blamed for unsuccessful performances (Frost, 2002; Bech, 2003; Doherty, 2003; West, 2003).

Injury is often cited as the major reason for player unavailability in professional soccer (Hawkins *et al.*, 2001; Rahnama *et al.*, 2002; Witvrouw *et al.*, 2003), although no studies can be found to confirm this statement. There is a plethora of epidemiological studies that analyse injuries in elite (Junge *et al.*, 2004), amateur (Goga & Gongal, 2003), youth (Kakavelakis *et al.*, 2003) and female, (Engström *et al.*, 1991) soccer players and report the effects of injuries on availability of players for team selection. These studies do not however, examine the effect of other factors on player availability choosing instead to determine the impact of soccer injuries on player availability in isolation. There appears therefore to be little, if any, current data that acknowledges that there may be various reasons why soccer players may not be available for first team selection or to train with the rest of the squad. These reasons may include such diverse factors as non-soccer related injuries, illness, suspensions, loan periods to other clubs, international call-ups and social and family related reasons.

This prospective study was performed to examine the reasons, and their respective incidences for all absences from training participation and competitive match availability at an English Division One club. This data would quantify the cost, in terms of loss of training and match availability, of all absences and allow analysis of the significance of absence due to soccer related injury to be determined. This may also highlight potential areas where comprehensive proactive initiatives could be developed that would minimise non-injury related player absence and hence increase the availability of players for competitive games.

3.3.2 Methods

The detailed methodology for this study can found in Chapter 3.1 of this thesis. This study was conducted over two competitive soccer seasons, 2003-04 (S1) and 2004-05 (S2). Data were collected between the first and final competitive match of each season. Absences from both training and matches during this time period were recorded. All contracted professional players (Table 3.3.1) agreed, in writing in accordance with Liverpool John Moores University ethical procedures, to allow the use of their injury, physical activity and attendance data to be included in the present study. Each player was identified by a randomly assigned code in order to protect their confidentiality.

Table 3.3.1 Descriptive details of the sample of players included in each season (mean ± SD).

Season	N	Age (years)	Height (m)	Weight (kg)	Playing position			
					Goalkeeper	Defender	Midfielder	Striker
2003-04	28	24 ± 5	1.8 ± 0.1	81 ± 7	2	12	8	6
2004-05	27	25 ± 6	1.8 ± 0.1	83 ± 7	2	9	9	7

In season one, 25 players were at the club at the start of the study period, 22 (88%) of the players who started the season were still at the club on the final day. The 6 players who

joined or left the club averaged 154 ± 64 days. In season two, 27 players were at the club at the start of the season, 23 (85%) of the players who started the season were still at the club on the final day of the season. The 4 players who left the club during the season averaged 141 ± 14 days. One of the 4 players was eliminated from the study due to mid-season work permit problems making him ineligible to play competitive soccer. His data was included until the day he became ineligible to play.

3.3.2.1 Recording absences

Details of collection methods used can be found in Chapter 3.1. A brief overview of those methods and specific methods for this study are provided here. All player attendance and absence were recorded by the club physiotherapist on a comprehensive daily register throughout the study period. An absence was defined as an inability to train with the rest of the squad or play competitive first team matches irrespective of the cause. Possible reasons for absence were pre-selected by examination of the previous 2 years absences. The categories selected were injury (soccer related and non-soccer related), suspension, rest-days, loans to other clubs, illness, international duty and personal circumstances. Injuries were classified as; transient (no loss of participation other than the session in which the injury was sustained); non-reportable (missed only one day not including the day of the injury) and soccer related reportable injury (SRRI) (resulted in >1 days absence not including the day of the injury). Any non-soccer related injuries , such as accidents in the home, that resulted in a player missing at least one days training or playing were recorded as 'other-injury'.

Statistics

All analyses were performed using SPSS for Windows Version 11 (Chicago, Illinois, USA). Descriptive statistics were presented and data expressed as mean ± *SD*. Differences between groups (age band, playing position) were compared using one-way between-groups analysis of variance with Post-hoc comparisons using the Tukey HSD test. Pearson

product-moment correlation coefficients were used to compute the relationship between age and all absence variables and player position and all absence variables. Statistical significance was accepted as $P < 0.05$ unless stated otherwise. Injury frequency rate (IFR) was stated as the number of new SRRI's per 1000 hours participation in soccer.

3.3.3 Results

3.3.3.1 Player attendance and exposure

Data from 55 (S1, 28:S2, 27) players (Table 3.3.2) were collected during the two study periods. A total of 274 and 284 days spanned the first and last competitive match of each season. During the study period there were 110 (S1, 57:S2, 53) first team matches and 60 (S1, 32:S2, 28) reserve team matches generating 2604 (S1, 1390:S2, 1214) match exposure hours. In the same period there were 287 (S1, 144:S2, 143) squad training sessions generating 6245 (S1, 3195:S2, 3050) individual participations or 10740 (S1, 5970:S2, 4770) training exposure hours. There were 4071 (S1, 1938:S2, 2133) individual days off with 134 days (21%) when >80% of players were given the day off. Descriptive statistics of player attendance and exposure patterns are shown in Table 3.3.2.

Table 3.3.2 Player attendance and exposure details

	Season 2003/04		Season 2004/052	
	Mean (SD)	Range	Range	Mean (SD)
Days at club	246 ± 56	84-271	121-284	263 ± 52
Training sessions	113 ± 32	32-141	43-145	113 ± 28
Days off	69 ± 17	22-87	28-107	79 ± 19
1st team match involvements	27 ± 20	0-56	0-54	27 ± 20
Reserve team involvements	10 ± 10	0-27	0-27	7 ± 8
1st team minutes	2106 ± 1790	0-5317	0-4849	2120 ± 1911
Reserve minutes	873 ± 894	0-2490	0-2343	618 ± 733
Total match minutes	2979 ± 1312	1076-5317	147-4849	2737 ± 1506

3.3.3.2 Absences

Table 3.3.4 details all reasons for absence throughout the study period. There was a season average of 12 incidents of player suspension. This accounted for almost one seventh of the total players being unavailable for first team selection. During the two seasons, 10 players were suspended for sending offs and 14 for an accumulation of individual bookings. Illness was also a noteworthy reason for absence. During the 2 seasons, 11% of match absences and 9% of training absences were due to illness. No significant differences were observed among player position or age groups in any of the absence variables ($p>0.05$).

3.3.3.3 Injury incidence and injury frequency rates.

Twenty two (79%) players sustained 39 SRRI's (mean 1.4 ± 1 per player) during season one. During season two, 20 (74%) players sustained 44 SRRI's (mean 1.8 ± 1 per player). In total, match injuries (n=64) accounted for 77% of SRRI's, (1st team n=51, reserves n = 13), while training injuries (n=19) accounted for 23%. These injuries resulted in a total of 731 training days lost (2.6 ± 1 per training session) and 199 players being unavailable for 1st team selection (1.8 ± 1.3 per match). Each SRRI resulted in a mean training loss of 9 ± 9 days and 2.5 ± 4 first team matches.

Although the IFR for first team matches (27/1000 h) was higher than reserve team matches (19/1000 h), this difference did not reach significance ($p>0.05$). Table 3.3.3 details IFR for all exposure hours.

Table 3.3.3 Injury frequency rates for SRRI's

	Exposure hours	Reportable injuries	Injuries per 1000 h
Training	10742	19	1.8
1st team	1920	51	26.6
Reserves	684	13	19.0
Total match	2604	64	24.6
Totals	13346	83	6.2

Table 3.3.4 Causative factors for all training and match unavailability

Unavailable for:	Season	Incidents	1st team matches N	1st team matches %	Training (sessions) N	Training (sessions) %
SRRI's	03-04	39	84	49.1	351	60.5
	04-05	44	115	55.5	392	45.5
Non reportable soccer injury	03-04	28	0	0.0	28	4.8
	04-05	36	0	0.0	36	4.2
Illness	03-04	21	11	6.4	33	5.7
	04-05	25	11	5.3	32	3.7
Other injury	03-04	4	27	15.8	101	17.4
	04-05	5	12	5.8	60	7.0
Personal reasons	03-04	10	6	3.5	9	1.6
	04-05	19	5	2.4	23	2.7
Suspensions	03-04	14	33	19.3	0	0.0
	04-05	10	20	9.7	0	0.0
Internationals	03-04	6	3	1.8	33	5.7
	04-05	14	6	2.9	146	17.0
Loan to another club	03-04	3	7	4.1	25	4.3
	04-05	5	38	18.4	172	20.0
Totals	03-04	125	171	100	580	100
	04-05	158	207	100	861	99.9

3.3.3.4 Analysis of non-reportable soccer injuries.

A total of 64 (S1, 28:S2, 36) training days were lost due to injuries that resulted in only one training day being missed. The major cause of single day absences were injuries sustained in competitive matches (n=53, 20.4 per 1000 h). Eleven single-day absences were recorded as a result of injuries sustained in training sessions (1/1000 h).

3.3.3.5. Distribution of absences

Mean monthly distribution of training days lost due to SRRI's and the total of all other absences (non-SRRI's) are shown in Figure 3.3.1. After excluding May (both seasons terminated on the first Saturday in May) no significant differences were found between any months in training days lost due to either SRRI's or non-SRRI's ($p>0.05$).

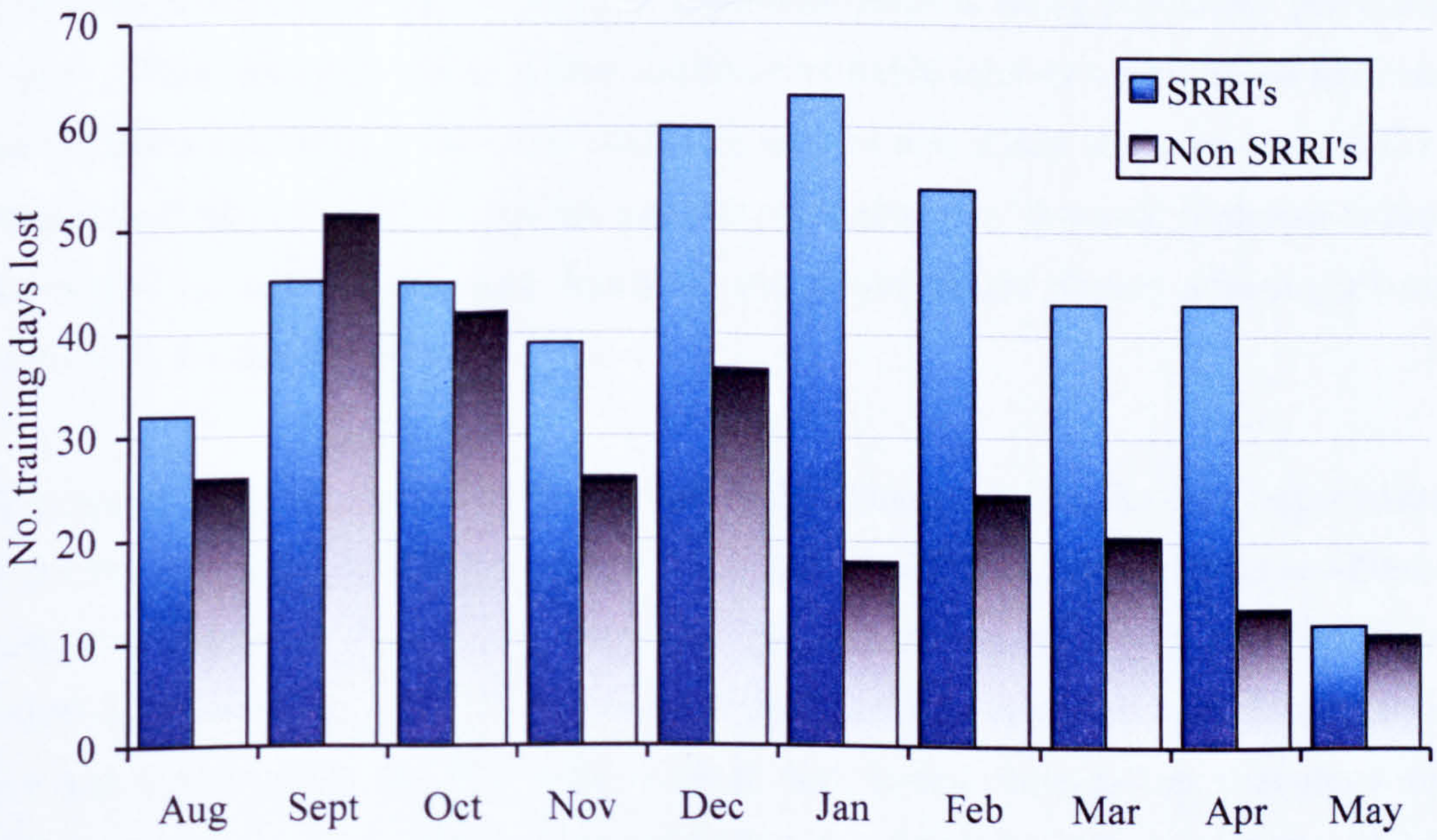


Figure 3.3.1 Comparison of mean monthly training days lost due to SRRI's and non-SRRI's.

3.3.3.6 Non-soccer injuries

During season one, 4 players suffered 4 'other' injuries. The injuries were; a road traffic accident, explorative abdominal surgery, an ankle injury whilst away from the club and an arthrodesis to a player's first metatarsal phalange joint due to disease based arthritic

changes. In season two, five players sustained 'other' injuries. The injuries sustained were; foot surgery following a domestic accident, lower leg infection following a laceration sustained at home, a hamstring strain rising from bed, retirement due to effects of delayed-union of a previous road traffic accident fracture from which the player had returned to competitive play and one player was occasionally unable to train due to long standing arthritic changes in a knee.

3.3.4. Discussion

This prospective study found that 6 out of 10 incidents of players being unavailable for first team selection can be attributed to reportable injuries using previously published criteria. Other absences such as illness and non-reportable injuries which would not meet the inclusion criteria in some other studies as well as non injury absences such as loan periods and international call ups account for other absences. It would therefore appear acceptable to acknowledge that injury is the major single factor affecting player availability for this data set.

The current study has, however, shown that injury reduction is not the only major factor in maximising player availability. It was found that suspensions, other injuries and illness were also important factors in determining the availability of players for first team selection and training (Table 3.3.3). No other data currently compares total training days and match availability lost for reasons other than injury. Suspensions accounted for almost one in seven of all player unavailability for first team selection. Although no analysis was undertaken with regard to reasons for suspension, it could be presumed that some of the bookings and sending offs would have been attributable to poor discipline and were therefore avoidable. Proactive initiatives to improve player discipline may therefore reduce the number of avoidable bookings and could have a positive effect on the number of suspensions.

The mean number of matches (n=12) and training days (n=33) lost per season to illness is another area where preventative strategies may have reduced player availability. Investigation into initiatives such as regular health checks and inoculations may be justified in an effort to reduce the incidence of these absences. Illness and suspensions combined accounted for 1 in every 4 incidents of players not being available for first team selection.

The findings of the present study highlight some of the limitations in considering soccer injuries as isolated factors when investigating player availability. Health-related absences (SRRI's, non-reportable soccer injuries, other injuries, sickness) accounted for approximately 7 out of 10 first team and training absences. Many studies based their definition and quantification of injuries on time absent from participation as a result of soccer related incidences (Hawkins & Fuller, 1999; Chomiak *et al.*, 2000). Others recorded every absence or treatment administered but limited their data to injuries sustained whilst playing or training (Lüthje *et al.*, 1996; Peterson *et al.*, 2000). Manipulating the present health-related data to include only soccer-related injuries resulting in more than one day's absence reduced gross data by 150 (28.8%) training days and 31 (23%) matches lost. Manipulating the data to include only soccer-related injuries reduced the training days lost by 118 (22.6%) and matches by 31 (23%). It is therefore suggested that the use of these definition bases do not reflect the effects of health-related absences in professional soccer.

When investigating if the present population is representative of the wider population in professional soccer, injury data compared well with other published data. The mean percentage (76%) of players injured per season is very similar to a number of other studies (Engström *et al.*, 1991; Peterson *et al.*, 2000). Injury frequency rate during competitive matches (25 per 1000 h) was also the same as reported by others (Hawkins & Fuller, 1999; Árnason *et al.*, 2004c) using a similar definition of injury. Mean SRRI's per player per season (1.5) was slightly higher than the average 1.3 per season observed by others. Hawkins *et al.* (2001) reported an average injury rate of 39 injuries per English club per season. This compares well with the 41 reported here. The IFR for training

(2/1000 h) is slightly lower than reported elsewhere (Poulsen *et al.*, 1991; Hägglund *et al.*, 2003) although this parameter is highly dependant on training methods and coaching attitudes to intensity and contact during training sessions (Ekstrand *et al.*, 1983a). Comparing IFR for training therefore may not result in the variables being measured being comparable.

3.3.5 Conclusion

The current study confirmed that soccer related injury is the major single factor affecting a player's availability for first team selection and training. However, strategies to maximise player availability by targeting player discipline and general health initiatives may be effective in reducing substantial time lost through suspension and illness. Clubs have no control over other factors such as international call ups and many personal and social events so reduction of player unavailability due to these reasons, may not be possible. It would appear that initiatives to help reduce the incidence, severity and recurrence of injuries may be able to play a key role in maximising player availability and improving the performance of the team.

3.4 STUDY 3: A PROSPECTIVE EPIDEMIOLOGICAL STUDY OF INJURIES AT ONE ENGLISH PROFESSIONAL SOCCER CLUB OVER 5 COMPETITIVE SEASONS.

3.4.1 Introduction

Study 2 demonstrated that injury is the major cause of professional soccer players not being available to train and play competitive soccer. These absences have an effect on team selection which may, in turn, have a significant effect on team performance and results. As well as the effects on team success, the financial implications of injury in respect of players being unavailable to compete can run into many millions of Euros (Hawkins *et al.*, 2001). A reduction in injury rate would therefore not only have a positive effect on player welfare but also on the financial wellbeing of professional soccer clubs.

Several studies have attempted to identify injury risk factors in an effort to suggest preventative strategies which may reduce the incidence of injury (Ekstrand and Gillquist, 1983a; Heidt *et al.*, 2000). These studies have been invaluable in highlighting specific causative factors. The data presented in each research has resulted in the development of successful prophylactic counter-measures such as balance training (Gioftsidou *et al.*, 2006) and strength training (Croisier *et al.*, 2008). It is well documented that the most important risk factor in injury in soccer is previous injury (Árnason *et al.*, 2004b; Kucera *et al.*, 2005). For example, Ekstrand and Gillquist (1983a) showed that one third of moderate and major injuries occurred within 2 months of a minor injury. This increased risk has been explained by factors such as poor rehabilitation and premature return to competitive action (Dvorak and Junge, 2000).

In professional soccer, it is normally the role of the physiotherapist or sports rehabilitator to ensure that players returning from injury are sufficiently rehabilitated to withstand the stresses of training and competition. One of the major difficulties encountered during

rehabilitation is the necessity to adapt training due to the high proportion of injuries being sustained to the lower limbs (Chomiak *et al.*, 2000). As soccer-specific training is normally characterised by running exercises, the enforced alteration to habitual training patterns may lead to a change in specificity and a reduction in the training load that may lead to inadequate rehabilitation. These modifications may be specific to individual pathologies as a direct consequence of the inter-relationship between the specific restraints of the injury. No studies can be found that describe the incidence of injuries in terms of their impact on a players' inability to perform soccer-specific training such as small-sided games and directional fitness drills.

Absence from normal training and competitive match-play may result in players detraining which can affect their ability to withstand the physical and physiological demands of match play on their return (Rhodes *et al.*, 1986). This detraining is a complex physiological state (Kraemer *et al.*, 2002) with changes occurring rapidly following either cessation, or a significant reduction, of habitual training. Aerobic indices such as mitochondrial enzyme activity (Costill *et al.*, 1985a; Bangsbo and Mizuno, 1989), reductions in blood volume (Coyle *et al.*, 1984), significant decreases in muscle capillary density (Moore *et al.*, 1987; Bangsbo and Mizuno, 1989) and a rapid reduction in $\dot{V}O_{2max}$ (Klausen *et al.*, 1981; Dahlström *et al.*, 1987) have all been reported to occur within 14 days of training cessation. Evidence therefore suggests that a period of detraining > 14 days may result in subtle physiological adaptations that could contribute to performance decrements. The result of these negative adaptations may lead to a reduction in players' capacity for work which could leave players susceptible to further injury. No studies can be found that specifically explore the incidence of injury-induced periods of altered training stimuli of sufficient duration to result in possible detraining in elite soccer players.

Previous research that describe the epidemiology and risk factors of injury in professional soccer players are typically conducted over one season or one competition (Nielsen and Yde, 1989; Ekstrand *et al.*, 1990; Lüthje *et al.*, 1996; Árnason *et al.*, 1996; Peterson *et al.*, 2000; Waldén *et al.*, 2005; Junge *et al.*, 2006) with some only including a part of a

season (Inklaar *et al.*, 1996; Hägglund *et al.*, 2005). Whereas injury incidence and injury pattern is known to vary within one season (Hawkins *et al.*, 2001), little is known about the natural variations between seasons. Only a few published investigations have included data from two seasons (McGregor and Rae, 1995; Hawkins *et al.*, 2001; Hägglund *et al.*, 2006) or longer (McGregor *et al.*, 2000; Hawkins and Fuller, 1999; Ekstrand *et al.*, 2004b; Chougle *et al.*, 2005). No studies can be found which analyse comprehensive prospective data including accurately timed exposure and first hand data collection, over a period as extensive as 5 seasons.

The aims of the present study were therefore to utilise the comprehensive data collection techniques detailed in Chapter 3.1 to:

(i) Comprehensively describe the injury profile of an English 1st division club over five competitive seasons.

(ii) Analyse the injury frequency rate of injuries as a function of severity, nature and anatomical location across the five seasons with particular focus on injuries that may impact on players' ability to undertake soccer-specific physical exercise for periods that may result in detraining.

3.4.2 Methods

This investigation was conducted over 5 competitive soccer seasons at one English League One club throughout the period August 2003 to May 2008. It was important that the data collected was not susceptible to misinterpretation related to the fluctuations in incidence and aetiological factors associated with short-term data collection periods. All contracted professional players (Table 3.4.1) agreed, in writing, in accordance with Liverpool John Moores University ethical procedures, to allow the use of their injury and physical activity data to be included in the present study. Each player was identified by a randomly assigned code in order to protect confidentiality. The greater number of players during the 2005-2006 season was a result of a severe injury to a goalkeeper which resulted in 3 additional goalkeepers being brought in to cover the player for the season.

Table 3.4.1 Player details for each season (mean ± SD) and player position frequencies.

Season	N	Age (years)	Height (m)	Mass (kg)	Playing position			
					Keeper	Defender	Midfielder	Striker
2003-04	28	24 ± 5	1.79 ± 0.1	81 ± 7.2	2	12	8	6
2004-05	27	25 ± 6	1.80 ± 0.1	83 ± 7.5	2	9	9	7
2005-06	34	24.5 ± 6	1.79 ± 0.1	81 ± 7.5	5	11	11	7
2006-07	26	26 ± 6	1.77 ± 0.1	79 ± 8	4	8	10	4
2007-08	26	25 ± 6	1.79 ± 0.1	79 ± 6.4	2	9	10	5

To meet the aims of this study, comprehensive data collection methods were utilised. This enabled a thorough analysis of the injury patterns to be determined. The current investigation was carried out prospectively with a cohort design using the methods detailed in Chapter 3.1.1. Briefly, the club physiotherapist completed a comprehensive daily register for all contracted professional players throughout each competitive season of the study duration. All attendance, including training and match exposure, and absence were recorded (Chapter 3.1.2.1). All training and match incidents requiring medical attention, irrespective of subsequent time unavailable, were immediately recorded. Injuries resulting in absence of >1 d were recorded on a ICDF (Figure 3.1.5). Any player injured at the start of the study period was not included in the study until they had returned to playing competitive matches. Data were collected and included on injured players until the last day of the study period.

The severity of injury in the current study were reported using total days absence (the total number of days from the day following the injury until the day the player fully participated in training or a match) and training days missed (the number of days when a squad training session took place in which the player was not available to participate). The calculation of the total day's absence allowed comparison with other epidemiological studies in the literature. Training days missed, together with first team match absence, gave an indication of the impact that the injury had on the number of soccer-specific

activity sessions that the player was unable to participate in. In addition, injuries to the lower limb which may affect a player's ability to perform soccer-specific training, and injuries resulting in >14 d absence which have the potential to induce negative physiological adaptations, and their integration are highlighted. The data for players' remaining injured on the last day of any study period were not included in this study. This occurred on 5 occasions.

Statistics

All analyses were performed using SPSS Version 14 for Windows (Chicago, Illinois, USA). All data were expressed as mean ± *SD*. Statistical methods applied included frequencies and descriptives. Anthropometric data for age, height and body mass, which were normally distributed, and inter-season differences, were compared using one-way analysis of variance (ANOVA). Categories for age and playing position were compared for qualitative variables with the chi squared test (χ^2). Quantitative variables were compared between groups using student's t-test. In the case of heterogeneity of variance (Levene's test) or non-normal distribution (Kolmogorov-Smirnov test), Mann-Whitney *U*-test or Kruskal-Wallis test were used. Statistical significance was accepted as $P < 0.05$ unless stated otherwise. Injury frequency rate was calculated as the number of injuries per 1000 exposure hours.

3.4.3 Results

3.4.3.1 Attendance and exposure

Attendance, exposure and basic injury data for the 5 seasons are presented as total for 5 seasons (mean of 5 seasons ± SD). The combined data collection days for the 5 seasons totalled 1376 d (275±5 d) (Table 3.4.2). The greater number of days for the 2004-05 season was as a result of the team getting to the Division One league play-off semi final.

In each season, the percentages of players who were at the club on the first and last days of the season and for who a full season's data were collected were; 2003-04 = 88%;

2004-05 = 85%; 2005-06 = 68%; 2006-07 = 85%; 2007-08 = 77%. There was no difference between seasons in player's mean age, height or body mass ($p>0.05$).

During the 5-season study period, there were 265 first team matches and 139 reserve team matches generating 5965 match exposure hours (Table 3.4.2). These match exposure hours were composed of 4623 1st team hours and 1342 reserve team hours. No differences were found in any match exposure variables between any of the five seasons ($p>0.05$). Figure 3.4.1 illustrates the distribution of the mean monthly exposure hours for first team and reserve team matches. There were no differences in first team or reserve team match exposure hours in any corresponding months across the 5 seasons ($p>0.05$).

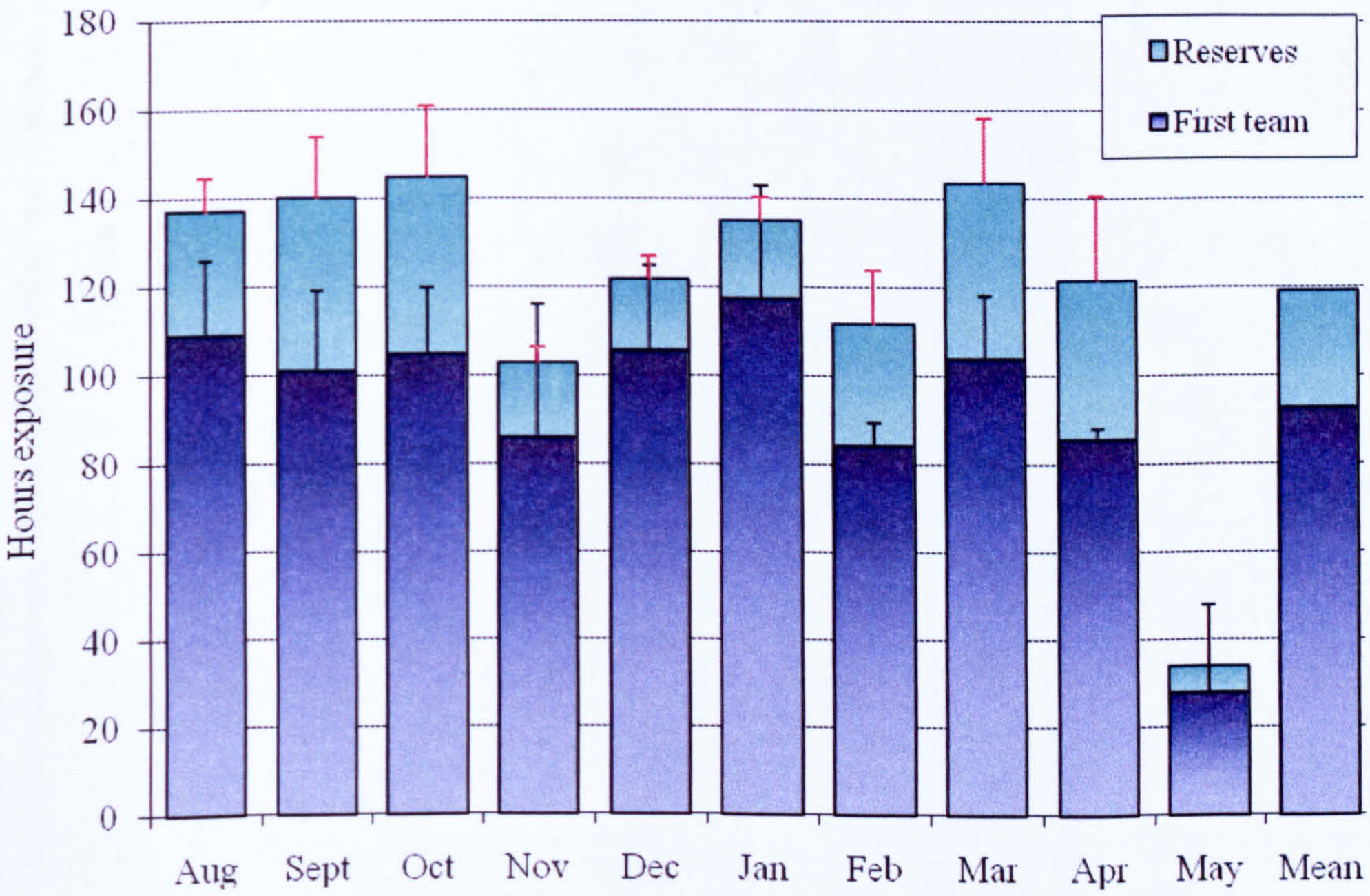


Figure 3.4.1 Mean monthly first team and reserve team match exposure for 5 seasons (Error bars +1SD).

Table 3.4.2 Descriptive statistics of 5-season exposure for training and matches.

	Season						
	2003-04	2004-05	2005-06	2006-07	2007-08	Total	Mean±SD
Total days of season	274	284	273	275	270	1376	275±5
Total training sessions	144	145	134	133	134	690	138±6
Total training hours (h)	322	272	265	229	232	1320	264±38
Total training exposure (h)	5973	4831	5547	4374	4466	25191	5038±697
First team matches	57	54	51	51	52	265	53±3
Reserve team matches	32	28	26	27	26	139	28±3
Total match exposure (h)	1391	1214	1153	1126	1081	5965	1193±121
1st team match exposure (h)	990	930	895	898	909	4622	925±39
Reserve match exposure (h)	401	284	258	228	172	1343	269±85

In the same period there were 690 squad training sessions. These generated 14862 individual participations or 25191 training exposure hours. The mean ± SD monthly distribution of training exposure is shown in Figure 3.4.2. No differences were observed in training exposure in the corresponding months across the 5 seasons (p>0.05).

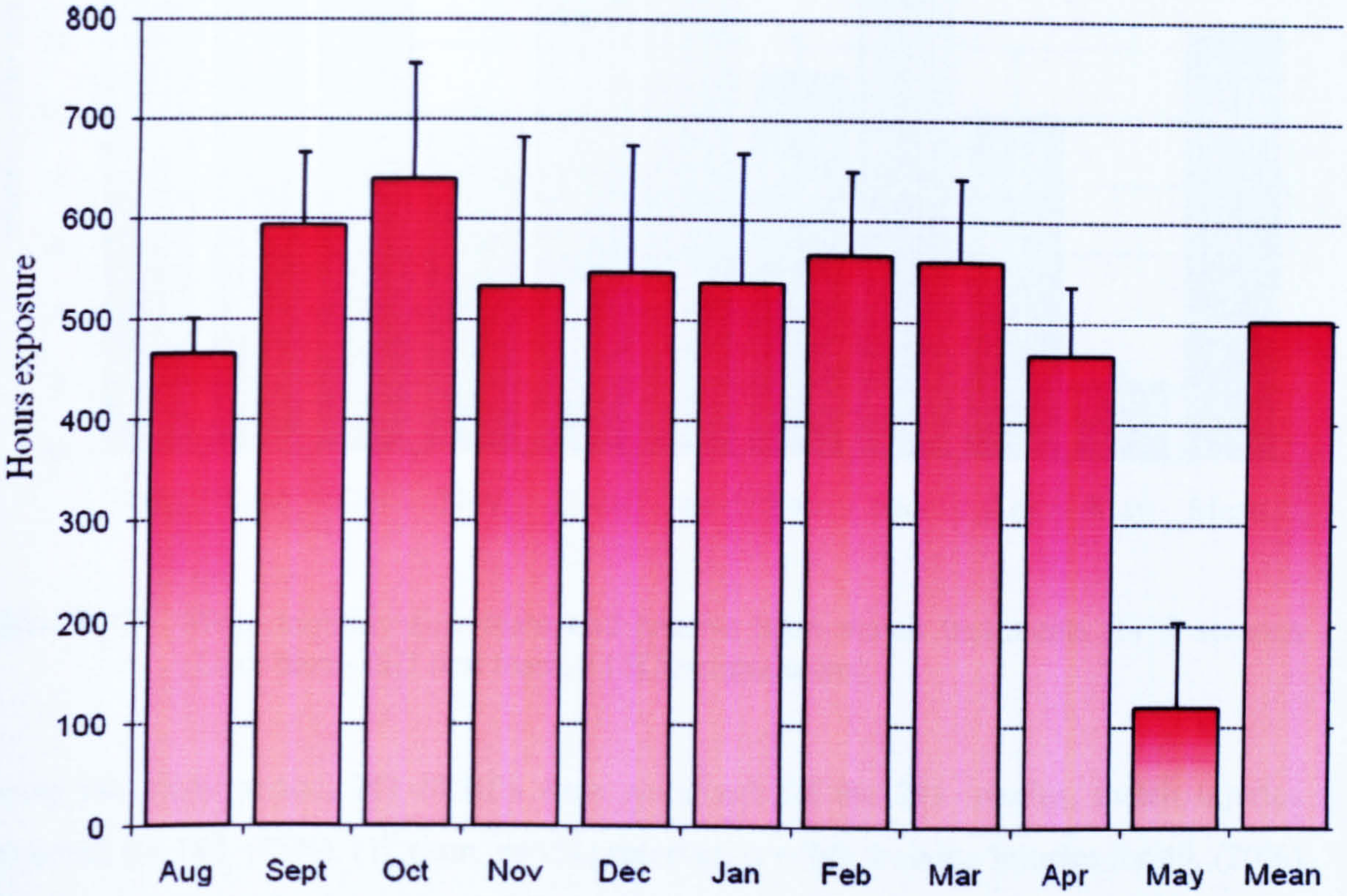


Figure 3.4.2 Mean monthly training exposure hours for 5 seasons (error bars + 1SD)

3.4.3.2 Incidents and temporal distribution of injuries

During the five-season study period, 136 incidents of players requiring medical attention during or after training were observed (mean 3±1.3 per month). During competitive matches, 608 players required medical attention (first team, n= 550; reserve team, n=72) (Figure 3.4.3). The monthly mean ± SD for frequency of players requiring medical attention was 11±6 per month for first team matches and 1.4±1 per month for reserve team matches. In the 5-season period, 205 transient injuries and 162 (38±14) non-reportable injuries were observed and recorded on the daily register.

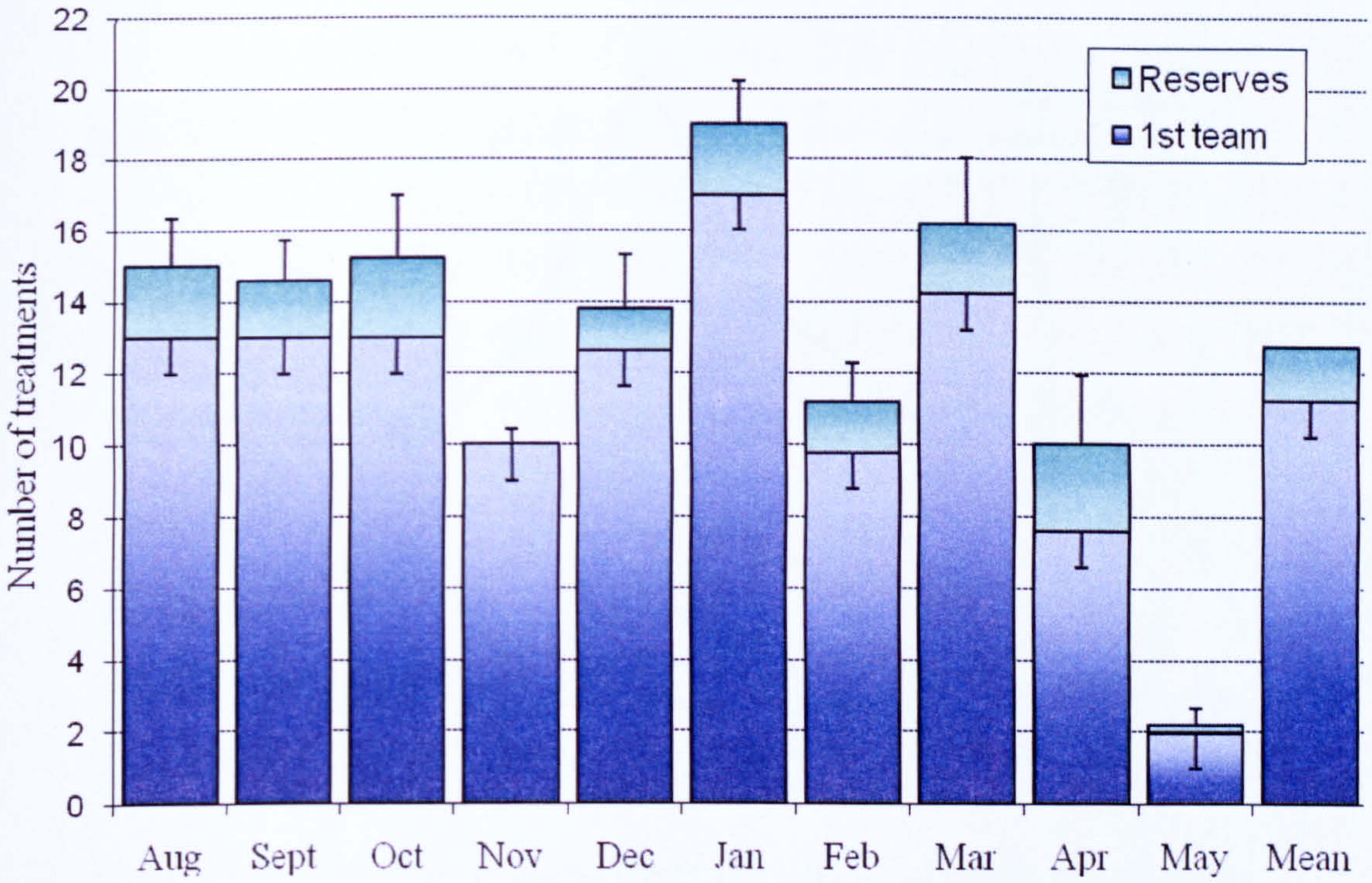


Figure 3.4.3 Mean monthly first team and reserve team match treatments for 5 seasons (Error bars -1SD first team, +1SD reserve team)

During the study period, 241 SRRI's were sustained. Of the 241 injuries, match injuries accounted for 182, (75%), (1^{st} team, n=151; reserves, n = 30), training injuries for 49, (20%) and overuse injuries for 11 (5%). SRRI's resulted in a total of 3689 days absence (15.3 ± 20 per SRRI) and 2180 squad training days absence (9 ± 12.6 per SRRI). SRRI's sustained in first team matches resulted in an average of 13.7 ± 21 d absence and SRRI's sustained in reserve team matches 16.9 ± 17 d absence. SRRI's sustained in training resulted in 16 ± 16 d absence. The 241 SRRI's resulted in a total of 601 incidents of players being unavailable for 1^{st} team selection (mean 2.5 ± 4.3 per SRRI). There was no differences in the severity of injuries, as determined by total days absence, sustained in either training or matches ($P>0.05$). There was also no difference in the number of injuries between any of the 5 seasons ($p>0.05$). Figure 3.4.4 illustrates injury source activity and total monthly frequency of all SRRI's. Although the total number of injuries sustained in December appears to be greater than any other month, the only month where this difference reached significance was May ($p<0.05$).

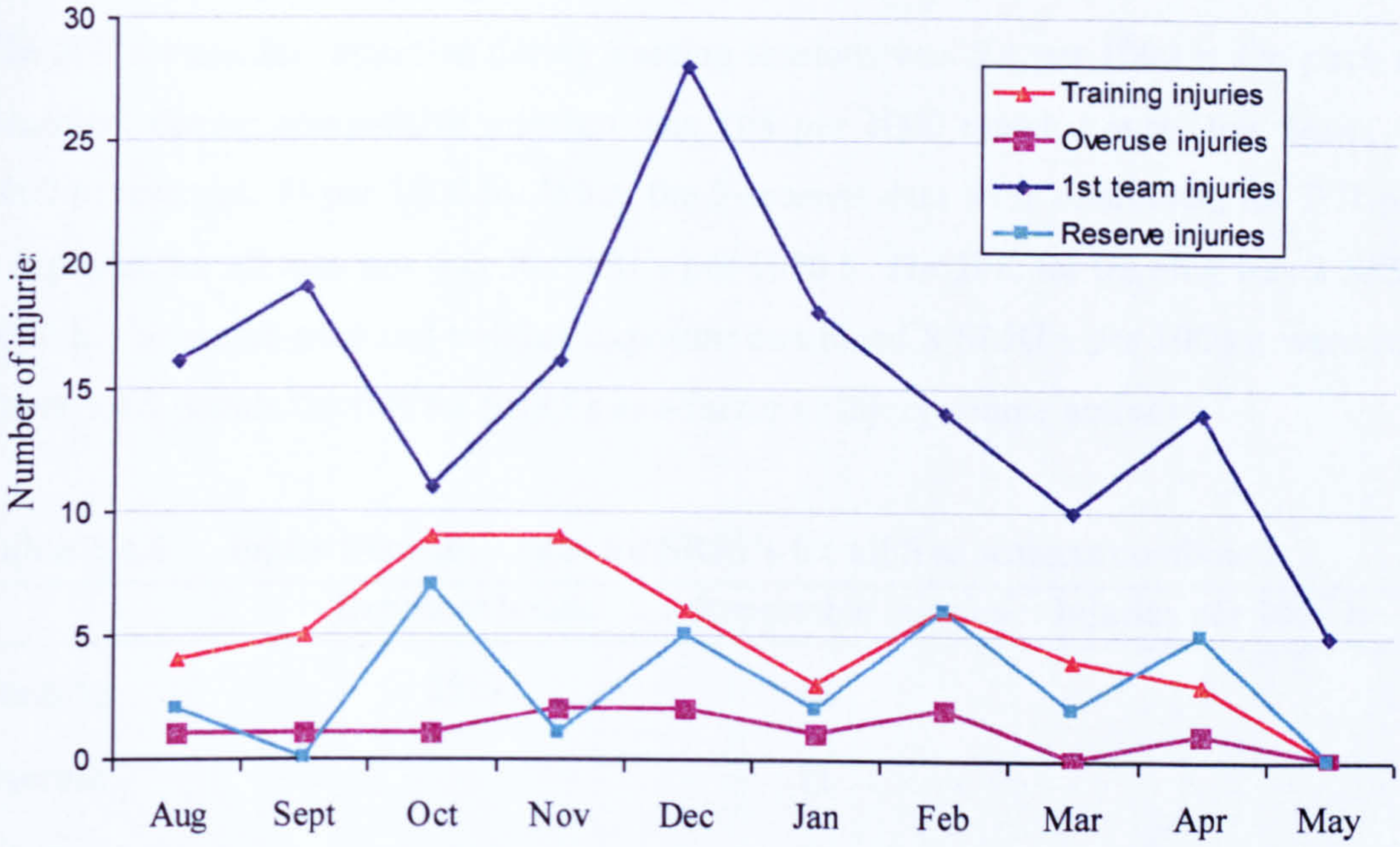


Figure 3.4.4 5-season monthly totals for SRRI's as a factor of injury source activity

Table 3.4.3 details the frequency of the number of times that players were injured each season. Over the 5 seasons 86% of player sustained at least 1 SRRI during the competitive seasons. This represented an average of 41% of the playing squad sustained >1 SRRI each season (46%, 33%, 33%, 58%, 38% for the 5 seasons). Analysis of the 17 players sustaining >3 SRRI's in one season showed that 15 of the 17 players had a least 2 injuries categorised in absence of <14 days.

Table 3.4.3 Frequency of number of times individual players were injured

	03-04	04-05	05-06	06-07	07-08
Percentage of squad sustaining at least 1 SRRI	82	100	74	85	92
Average SRRI per player per season	2	1.8	1.6	2.5	1.7
Number of players sustaining 1 SRRI	9	18	14	7	14
Number of players sustaining 2 SRRI's	7	2	8	6	6
Number of players sustaining 3 SRRI's	3	3	1	3	2
Number of players sustaining 4 SRRI's	1	3	2	3	1
Number of players sustaining >4 SRRI's	2	1	0	3	1

3.4.3.3 Injury frequency rate

The IFR for medical attention during training sessions was 5.4 per 1000 h. On pitch medical treatment during competitive matches was 104 per 1000 match hours (first team, 119 per 1000 h; reserves, 54 per 1000 h). When the 5-seasons data were combined, the IFR per 1000 h exposure for all matches was 30 SRRI's per 1000 h. The IFR for training was 2 SRRI's per 1000 h. For match-play and training exposure combined 8 SRRI's per 1000 h were recorded. Table 3.4.5 details the IFR for SRRI's as a factor of injury source activity.

Table 3.4.4 Injury frequency rates for SRRI's for all five seasons combined

	Exposure hours	Reportable injuries	Injuries per 1000 h
Training	25191	48	1.9
Overuse		11	
Total match	5965	182	30.5
1st team	4623	151	32.7
Reserves	1342	31	23.1
Totals	31156	241	7.7

Figure 3.4.5 details the monthly IFR for matches per 1000 exposure hours for each season. Although February 2006-07 appears to have been a particularly bad month for injuries, no difference was found between this month and the February of the other 4 seasons ($p>0.05$). There were also no differences when comparing all other months across the five seasons ($p>0.05$).

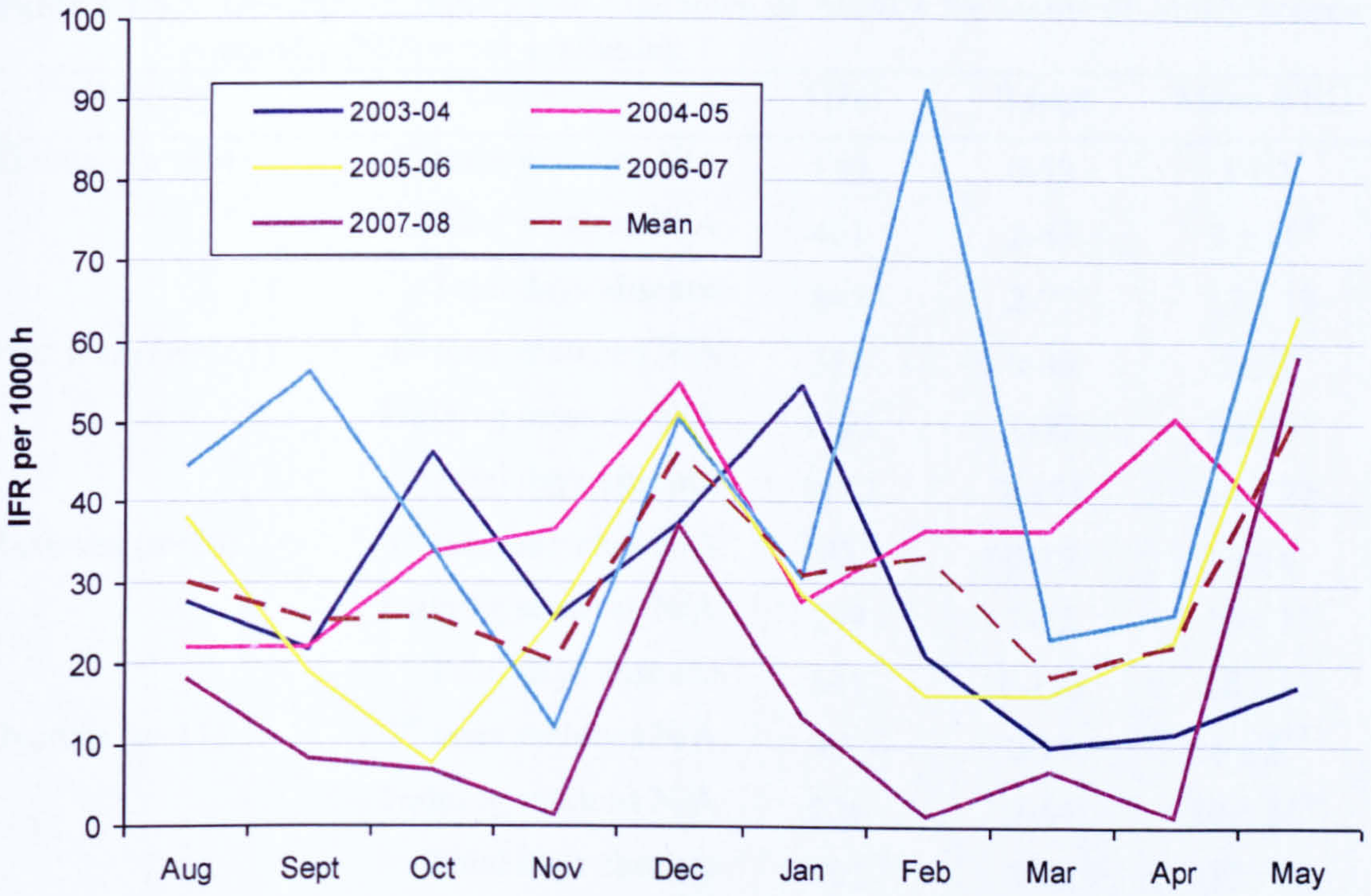


Figure 3.4.5 Mean monthly match IFR for 5 seasons

3.4.3.4 Severity of injuries

In professional soccer, injury severity is often described in terms of length of absence from training sessions and unavailability for first team match selection. Table 3.4.5 describes the severity of SRRI's in terms of match and training absence by factor of injury source activity. Overuse injuries resulted in a greater number of first team matches missed ($F(3, 238) = 2.5$, $p<0.05$) and training sessions missed ($F(3, 238) = 3.5$, $p<0.01$) than both training injury and first team match injury categories. No difference was found between overuse injuries and reserves team match injuries ($p>0.05$). No other differences were found in training days missed, total days absence or first team match unavailability between any injury source activity categories ($p>0.05$).

Table 3.4.5 Descriptive statistics of outcomes of SRRI's by factor of injury source activity (N/A = not available).

Activity	Outcome	Total	Range	Mean ± SD
Training (n=49)	1st team matches N/A	139	0-13	3 ± 3*
	Training sessions N/A	461	2-43	9 ± 10$
	Total days absence	845	2-77	17 ± 19
First team (n=151)	1st team matches N/A	317	0-40	2 ± 5#
	Training sessions N/A	1195	1-92	8 ± 13+
	Total days absence	2132	2-191	14 ± 22
Reserves (n=30)	1st team matches N/A	83	0-19	3 ± 4
	Training sessions N/A	299	1-53	10 ± 11
	Total days absence	541	1-113	18 ± 23
Overuse (n=11)	1st team matches N/A	62	0-15	6 ± 5*#
	Training sessions N/A	224	3-44	20 ± 16$+
	Total days absence	402	3-82	37 ± 30

*# = significantly greater number of first team matches missed as a result of both training injuries and first team match injuries ($p<0.05$). $+ = Significantly greater number of training sessions missed as a result of both training injuries and first team match injuries ($p<0.01$).

Table 3.4.6 details the mean training and match loss as a factor of injury severity. When analysing injuries of < 15 d (n=150) the mean absence was 6±3 d. For injuries of >14 d (n=91) mean total days absence was 35±27. For the same injuries, mean absence from training days was 22±16 and mean number of first team match unavailable was 7±6 matches.

Table 3.4.6 Five season frequency of injury severity with mean participation loss

			Total days absence		Squad training sessions missed		First team matches missed	
	N	%	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
2-6 d, slight	105	44	2-6	4±1	1-4	2±1	0-2	0.2±0.4
7-14 d, minor	45	19	7-10	8±1	2-6	4±1	0-2	1±0.6
15-28 d, moderate	53	22	11-28	19±6	4-19	10±4	1-5	3±1
29-60 d, major	27	11	29-57	43±9	11-38	25±7	4-11	7±2
over 60 d, severe	11	5	68-191	93±37	32-92	50±21	9-40	19±0.5

Differences in the severity of injuries sustained in each month were investigated. The average number of subsequence day's absence for injuries sustained in each month was calculated. There were no significant differences between the average severity of injuries sustained in any months ($F(8) = 0.45$, $p>0.05$).

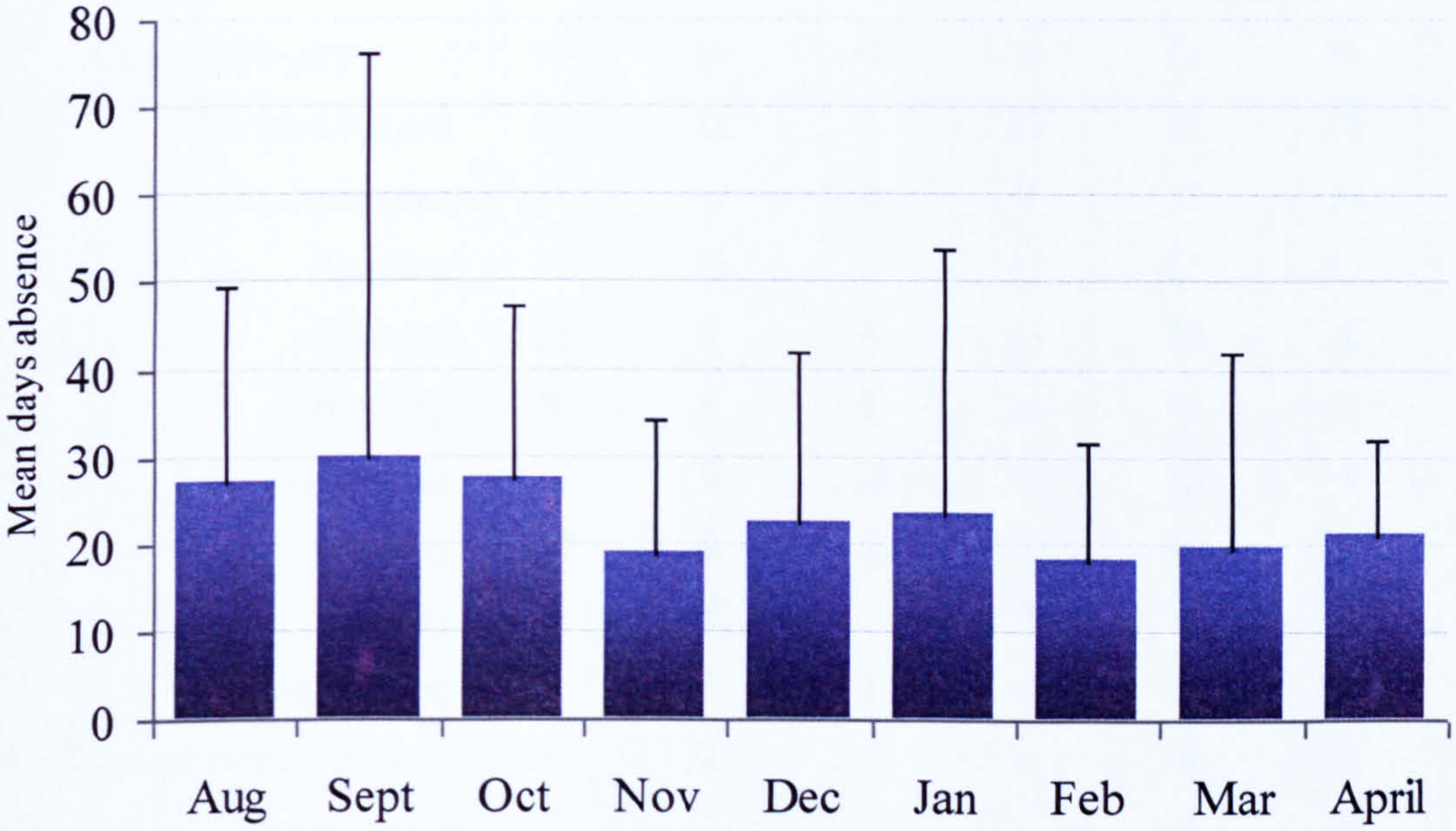


Figure 3.4.6 Average number of day absence for injuries sustained in each calendar month

When considering the IFR as a factor of injury severity, slight or minor (<15 d) (n=150) injuries accounted for 20 injuries per 1000 match exposure hours. When injury source activity was considered, the IFR for first team match injuries resulting in absence of <15 d was 22 injuries per 1000 h, 11 injuries per 1000 h for injuries sustained in reserve team matches and 1 injury per 1000 training hours. IFR's categorised as moderate, major or severe (>14 d) (n=91) were 11 per 1000 match exposure hours and 1 per 1000 training exposure hours.

3.4.3.5 Aetiology of injuries.

Of the 241 SRRI's, 230 (95%) were categorised as traumatic and 11 (5%) categorised as overuse. Extrinsic causative factors accounted for 125 injuries (52%) with 116 (48%) being categorised as having intrinsic causative factors. There was little difference in the number of dominant and non dominant sided injuries (Dominant n=113, 47%, non-dominant n=96, 40%,

non applicable n=32, 13%). Table 3.4.7 details the causative factors of all SRRI's that were not overuse injuries. There were no differences in the total days absence among any cause of injury category ($\chi^2 = 12.3$, $df = 10$, $p = 0.27$).

Table 3.4.7 Mechanics of recordable injuries

	All injuries		Training		Match	
Cause of injury	N	%	N	%	N	%
Tackled/kicked	59	25	7	13	52	28
Collision/impact	41	17	4	8	37	20
Running	31	13	6	13	25	13
Kicking	21	9	6	13	15	8
Stretching	9	4	5	10	4	2
Twisting/turning	22	9	10	21	12	6
Tackling	20	8	5	10	15	8
Jumping	7	3	1	2	6	3
Falling/landing	6	3	3	6	3	2
Violent conduct/elbow	4	2			4	2
Long standing	2	1			2	1
Indeterminate	8	4	2	2	6	3
Totals	230		49		181	

It is interesting to note that only 11 of the 230 injuries (5%) were sustained as a result of contact during training (tackled/kicked, collision/impact, violent conduct) while 93 (40%) were sustained as a result of contact during competitive matches. This relates to an IFR for contact injuries of 0.4 injuries per 1000 training hours and 16 injuries per 1000 match hours. There was no differences between seasons in the number of contact injuries during training ($p>0.05$). In the categories, tackled/kicked and violent conduct during match-play (n=56), the referee gave a free kick against the opposing team on 41 occasions (73%) (7 per 1000 match hours) indicating that the offending tackle was in breach of the laws of the game. This related to 31% of all match SRRI's resulting from foul play. Of the 41 fouls, 20 players (50%) were further sanctioned by being shown a yellow card and 1 (2%) a red card.

3.4.3.6 Risk factors

3.4.6.3.1 Playing position

There was no difference when comparing total days absence per injury and playing position (p>0.05). Although no significant differences for any absences between any playing position were observed (p>0.05), the data suggests that goalkeepers sustain more serious injuries than other playing positions. The low number of goalkeeper injuries was probably the reason the difference did not reach statistical significance.

3.4.6.3.2 Age

Analysis of age categories and absence showed no difference in total days absence (p>0.05). The average number of total day's absence per injury for each age category were similar. They were; 18-21 y, 17 ± 24 d; 22-28 y, 16 ± 19 d and 29-34 y, 13 ± 15 d.

3.4.6.3.3 Re-injury

During the 5-season study period, 5 injuries (2%) were classified as early re-injury, 6 (2%) as late re-injury and 26 (11%) classified as further injury (Table 3.4.8). Two hundred and four injuries (56%) had no link with any previous injury. When comparing early and late re-injuries with all injuries with no link, absence from training (20±12 versus 9±12 d) and total days absence (27±19 versus 15±20 d) were greater for the injured with a link to a previous injury (p>0.05). Match unavailability was also greater for injuries with a link to a previous injury (5±3 versus 2.4±4 first team matches) although this did not reach significance (p=0.07).

Table 3.4.8 Training and match absence as a function of re-injury category

		Range	Mean(SD)
Early re-injury (N=5)	1st team matches N/A	1-9	5±3
	Training sessions N/A	2-32	18±11
	Total days	5-38	21±12
Late re-injury (N=6)	1st team matches N/A	0-10	5±4
	Training sessions N/A	2-44	21±14
	Total days	4-72	33±24
Further injury (N=26)	1st team matches N/A	0-10	2±3
	Training sessions N/A	1-37	6±8
	Total days	2-45	10±8
No injury link (N=204)	1st team matches N/A	0-40	3±5
	Training sessions N/A	1-92	9±13
	Total days	2-191	15±20

3.4.3.7 Anatomical location of injuries

When injuries were categorised into 18 different anatomical locations almost 85% of SRRI's were to the lower extremities (94% of training injuries and 78% of match injuries). Table 3.4.9 details the frequency of injury to each anatomical location as a factor of injury source activity. No differences were found when investigating whether injuries to specific anatomical locations were different between seasons ($\chi^2 = 13.1$, $df = 8$, $p=0.11$). That is to say that inter-season fluctuations in the frequency of specific injuries such as knee injuries or thigh strains did not seem to be a major factor in the pattern of injury.

Table 3.4.9 Distribution of injuries by anatomical location and factor of injury source activity.

	All injuries		Training		Match		Overuse	
	N	%	No.	%	N	%	N	%
Scalp	10	4			10	5		
Face	12	5			12	6		
Shoulder	1	0			1	1		
Wrist/hand	2	1			2	1		
Ribs	2	1			2	1		
Spine	7	3	3	6	4	2		
Abdomen	3	1			2	1	1	10
Groin/hips	3	1	2	4	1	1		
Adductor	16	7	3	6	13	7		
Hamstring	35	15	12	25	22	12	1	10
Quad	23	10	6	12	17	9		
Knee	47	20	8	17	27	20	3	30
Calf	19	8	1	2	18	9		
Shin	10	4	1		7	3	2	10
Ankle	28	12	7	15	19	10	2	20
Achilles	5	2			5	3		
Foot	11	5	5	10	5	3	1	10
Toe	7	3	1	2	5	3	1	10
Totals	241	102	48	99	193	100	11	100

Table 3.4.10 details the severity of injury as a factor of anatomical location. Thirty four percent of all SRRI's (n=82, mean 16.4 ± 3.4 per season) sustained to the lower limbs resulted in absence >14 d. Only 9 of 91 injuries (10%) resulting in absence of >14 d were sustained to the upperbody. Three of these injuries were fractured zygoma and one was a shoulder reconstruction. Although unable to train, all 4 players were able to participate in running type training within 2 weeks of sustaining their injuries.

Table 3.4.10 Severity of injury as a factor of anatomical location.

	Actual grade of injury				
	2-6 d, slight	7-14 d, minor	15-28 d, moderate	29-60 d, major	> 60 d, severe
Head and neck	18		1	3	
Shoulders and arms			2	1	
Torso	8	2	2		
Pelvic area	2	1			
Thigh	32	21	20	2	
Knee	14	5	10	12	4
Lower leg	17	4	8		1
Ankle	10	8	5	7	2
Foot	4	4	5	2	4
Lower limb	77	42	48	23	11
total	105	45	53	27	11

Absence from first team matches and training sessions were significantly greater for knee (mean 4 ± 7 matches not available, 15 ± 17 d total absence) and foot (mean 5 ± 5 matches not available, 17 ± 18 d total absence) injuries than for thigh injuries (mean 1 ± 2 matches not available, 6 ± 5 d total absence) (p<0.05). Absence for training days was also greater for knee and foot injuries than head and neck injuries (mean 4 ± 7 d total absence) (p<0.05). No other differences in absences as a factor of anatomical location of injuries were found (p>0.05).

There was no differences in the percentage of each anatomical structure damaged between training and match-play (p>0.05) (Table 3.4.11). Match-play however, produced a more diverse range of injuries with most injuries in training affecting muscular or ligamentous structures.

Table 3.4.11 Distribution of anatomical structures injured as a factor of source activity.

	All injuries		Training		Match	
Structures injured	N	%	N	%	N	%
Muscle	108	45	25	52	83	43
Ligament	57	24	16	33	41	21
Tendon	5	2			5	3
Capsule/Fascia/bursa	2	1	1	2	1	.5
Bony	29	12	5	10	24	12
Meniscus	1	.5			1	.5
Other soft tissue	30	12			30	16
Other	9	4	1	2	8	4
Totals	241		48		193	

3.4.3.8 The frequency of injuries that may result in detraining.

When investigating the anatomical location of injuries, injuries to the lower limb accounted for 201 of the 241 SRRI's (83%) (Table 3.4.12). Almost half of these injuries (47%) were sustained to the muscle complexes of the thigh and calf. Muscle strains accounted for 80% of all thigh injuries and 69% of all calf injuries. Ligamentous structures were the primary injury in 27% of the cases with a similar percentage of knee and ankle/foot ligament pathologies. Ligamentous injuries accounted for 64% of all knee injuries with 70% (n=21) of these injuries resulting in absence of >14 d. Nine (41%) of the 22 ligamentous ankle injuries resulted in absence >14 d. In total, 82 of the 201 lower limb injuries resulted in absence of >14 d. Therefore, a season average of 16 injuries were sustained, the anatomical location of which almost certainly affected the player's ability to maintain previous intensity of training. The average number of day's total absence for these injuries was 30.3 ± 27 d. The average number of players absent at any time during a competitive season with an injury causing absence of >14 d and with a pathology that would affect their ability to train at previous intensity could therefore be calculated using the formula;

$$\frac{\text{Mean frequency of players absent for >14 d x mean absence}}{\text{Mean season duration (d)}} = \frac{(16 \times 30.3)}{275} = 1.8$$

Using the current data set therefore, it was calculated that an average of almost 2 players who had sustained a lower limb injury were absent on any day during the competitive season with an injury of a severity that would prevent them from training normally for a period of >14 d. In the current study, this relates to 8% of the current population being absent on any day during the competitive season with an injury which limits the scope of exercise possible.

Table 3.4.12 Cross-tabulation of lower limb structure injured and anatomical location

	Anatomical location					
Structure injured	Thigh	Knee	Calf/shin	Ankle	Foot	Total
Muscle	74	5	20	0	0	99
Ligament	0	30	0	22	3	55
Tendon	0	1	0	4	0	5
Meniscus	0	1	0	0	0	1
Other soft tissue	0	8	6	0	6	20
Bony	0	2	3	7	9	21
Total	74	47	29	33	18	201
Percentage	37	23	14	16	9	99

3.4.4 Discussion

The current study utilised comprehensive data collection procedures to accurately describe the injury patterns in an English Division One professional soccer club over a 5-season period. These data showed that the risk of playing competitive matches, as demonstrated by injuries per 1000 h exposure, was significantly greater than the risk of injury in training. Injuries were predominantly sustained to the lower limbs and to structures which may have resulted in joint stability or the ability to run being compromised. Further analysis noted that an average of 18 injuries occurred per season which were of a severity which may have resulted in detraining. Of these 18 injuries, 16 were sustained to anatomical structures which affected the player's ability to undertake soccer-specific training.

3.4.4.1 Incidents of injury and temporal distribution of injuries

Throughout the study period, 75% of injuries occurred during competitive matches. This is higher than the 66% reported by Lewin (1989), 67% identified by Hawkins and Fuller (1999) and the 63% reported by Poulsen *et al.* (1991) and Hawkins *et al.* (2001). The reason for

these differences is probably that the length of the data collection period in the current investigation being longer than the above studies thus giving a clearer indication of injury patterns. It is quite evident from current and previously published data that the risk of injury during matches is significantly greater than during normal squad training. The increased intrinsic risk is probably due to the competitive element of the match increasing player effort. This may mean that players are performing closer to, and on occasions trying to exceed, their maximum potential thereby increasing the possibility of injury. The current data shows that the extrinsic risk factor increase during matches appears to be almost entirely due to player on player contact, particularly being tackled by an opponent.

The present data shows that an average of 86% of all players suffered at least one SRRI during each competitive season. This compares well with injuries to 82% (Peterson *et al.*, 2000) and 86% (Lewin, 1989; Dvorak *et al.*, 2000) of all playing staff during one competitive season when using a similar definition of injury. Hawkins *et al.* (2001) reported an average injury rate of 39 injuries per English club per season, compared to 48 in the current study. The data from Hawkins *et al.* (2001) also included pre-season injuries. Injuries in this stage of the competitive cycle were not included in the current study, the difference in the reported incidence of injuries is even greater. This will likely increase the disparity between our data and the data of Hawkins *et al.* (2001). Hawkins *et al.* (2001) relied upon third party medical staff to diligently complete and return injury report forms for all injuries resulting in >1 d missed training. The significant difference in the percentage of slight (<7d) injuries (current study 44%; Hawkins *et al.* 2001, 33%) observed here may reflect a reluctance of medical staff to complete lengthy questionnaires for short-term injuries. When considering the number of injuries observed during their study, Hawkins *et al.* (2001) concluded that injury in soccer is a major problem. The current data suggests that the problem may be more serious than previously thought with a high percentage of the total playing squad sustaining a participation-limiting injury every season.

3.4.4.2 Injury frequency rate

The IFR in the present study during competitive matches (30.5 per 1000 h) was higher than the 20-26 injuries per 1000 h reported in other studies using a similar definition of injury (Poulsen *et al.*, 1991; Hawkins and Fuller, 1999; Waldén *et al.*, 2005). As demonstrated in Chapter 3.2, this may be due, in part, to the methods of data collection and accuracy of timing match exposure. Notwithstanding the above differences, the current data confirms that the

risk to injury in soccer is higher than the risk in recognised high-risk occupations (Fuller and Hawkins, 1997). Nielsen and Yde (1989) reported the lowest match IFR (19 injuries per 1000 match hours) while Árnason *et al.* (1996) observed the highest (35 injuries per 1000 match hours). Both of these studies used professional soccer players as subjects but both relied on coaching staff to time and report exposure data. If these exposure data were not accurate, it could have a significant effect on IFR. Both studies were also of one season duration so their findings may be the result of inherent season-on season-variability.

The IFR for training in the current study (1.9 injuries per 1000 hours) is slightly lower than reported elsewhere (Poulsen *et al.*, 1991; Hawkins and Fuller, 1999). It has previously been suggested that training IFR is highly dependant on training methods and coaching attitudes to the intensity of training and the contact that is permissible between players during training sessions (Ekstrand *et al.*, 1983a). The low IFR may therefore be a reflection of this particular club's philosophy with regard to training intensity and contact in training drills. However, there were no significant differences between seasons in the frequency of training injuries despite the club employing 3 different managers during the study period (August 2003 to October 2004; November 2004 to October 2006; October 2006 to end of study period). Each of these managers would have had their own, and different, training philosophy. The incidence of injury in training therefore, may not be dependant on coach attitudes to intensity as previously reported but may reflect inherent differences in intensity and level of competitiveness between training and match play.

3.4.4.3 Severity of injuries

The current study found no differences in the severity of injuries sustained in training or matches ($p<0.05$). The average number of days absence per SRRI (15 ± 20) was considerably lower than the 24 d (Hawkins *et al.*, 2001) and the 20 d (Ekstrand *et al.*, 1983) observed in other studies. This was probably due to methodological differences previously highlighted.

3.4.4.4 Previous injury as a risk factor

When investigating links between injury and previous injury (Table 3.1.4), it was observed that almost 5% of all SRRI's were sustained to the same anatomical location and structure within 6 months of the index injury. Expanding the data to include re-injuries to different anatomical locations and structures for injuries within 2 months of the index injury, increased the percentage of re-injuries to 13%. If all SRRI's are analysed, an average of 41% of

players were injured on more than one occasion during any one season. Each of these methods of reporting re-injury have been used in previous literature. This illustrates the difficulty in comparing re-injury rates across the literature. This discrepancy is due to the definition of re-injury containing an anatomical criterion, a timescale and the definition of the original injury. Hawkins *et al.* (2001) reported a re-injury rate of 7% when using the definition "an injury of the same nature and location involving the same player in the same season". This compares well with the 5% in the current study. Others have reported re-injury rates as high as 50-60% using a definition of re-injury similar to 'further injury' in the current study within the same season (Árnason *et al.*, 1996; Peterson *et al.*, 2000).

The available literature demonstrates that previous injury is a significant risk factor to further injury (Árnason *et al.*, 2004b). Re-injuries may also be more severe than the index injury as injured players that had been previously injured in the same season have been shown to be absent significantly longer than for the index injury (Hawkins *et al.*, 2001; Waldén *et al.*, 2005). These findings are similar to the current study. Analysis of severity of injuries in each 'link to same injury' category showed a significant, greater number of training days and total days absence for subsequent injuries than injuries with no injury link. The current findings would therefore appear to support the idea that previous injury is not only a risk factor for further injury but that subsequent injuries can lead to an injury of a more severe nature than the index injury. This may be due to the impact of a previous injury resulting in greater tissue damage or a more cautious approach to returning to competition. This highlights the importance of reducing re-injury in professional soccer by ensuring players are sufficiently rehabilitated and physically prepared to return to competitive match-play.

3.4.4.5 Anatomical location and structures injured.

The anatomical structure damaged as a result of injury can impact on a player's ability to perform soccer-specific exercise. The percentage of lower limb injuries observed in the current study was 83% which is in agreement with others (Nielsen *et al.*, 1989; Engström *et al.*, 1991; Chomiak *et al.*, 2000 Hawkins *et al.*, 2001). Pathologies to structures related to locomotion such as muscle or structures responsible for static joint stability, such as ligaments, may prevent players from exercising at intensities completed pre-injury. Injuries to these structures may also prevent soccer-specific exercise due to the ability to complete the required directional demands included in training and match-play. In the current study, the

most common tissue damage was muscle strain (45%). Thigh and calf muscle strains accounted for 87% of these injuries. Ligamentous injuries accounted for 24% of all SRRI's. Knee and ankle injuries accounted for 95% of ligament injuries. The findings of the current study therefore suggest that a high proportion of injuries are sustained to structures that affect a player's ability to run or affect a player's joint integrity. The likely result of these injuries is the inability of injured players to perform soccer-specific exercise at an intensity possible prior to the injury, especially during early rehabilitation.

Although it would appear that a high proportion of soccer injuries result in the inability of the injured player to participate in any part of normal squad training, this is not always the case. For example, in the current study a player sustained a fracture to his zygoma and was unable to participate fully in training for 8 weeks. He was however, able to run on day 4 following the injury and participate in normal squad training from day 8 of his injury. His squad training was however, modified by discouraging other players from tackling him and he was also unable to head the ball in training. Another player sprained his lateral ankle ligaments resulting in an absence of 4 weeks. In contrast to the above player, he wasn't able to run until day 19 of his injury and had to rely on non-weight bearing CV work such as cycling and rowing. These two cases highlight the effects of the anatomical location of injury on the training that players are able to perform during rehabilitation. Lower limb injuries are therefore more likely to affect the player's ability to perform soccer-specific training and therefore players with lower limb injuries may be more susceptible to the effects of detraining.

3.4.4.6 Injuries and possible detraining

It has been previously discussed that detraining occurs rapidly following reductions in habitual training patterns. Negative physiological adaptations have been shown to occur following detraining periods of <14 d (Bangsbo and Mizuno, 1988). When investigating injuries of sufficient duration to result in detraining however, the current study utilised periods of >14d. This duration was selected to reflect a length of absence to match the majority of short-term detraining studies (Houmard *et al.*, 1992; Hortobágyi *et al.*, 1993). From the current results, it can be calculated that, on average, over 2.3 players were absent at any one time with an injury resulting in them being unable to train normally for >14 d. This is over 8% of the current population being absent with an injury, the severity of which, could result to the player detraining, at any one time during the competitive season.

When investigating the anatomical location of injuries resulting in absence >14 d (n=91), 90% were sustained to the lower limbs. These figures average out at almost 2 players injured throughout the 5-season study period with an injury to the lower limb of >14 d severity. In a squad size averaging 24 players over the study period, this loss through injury could have a significant effect on player availability and even team success. It is also probable that these injuries affected the players' ability to perform soccer-specific training. Therefore, it would be expected that those players absent for >14 d after sustaining injuries to the lower limb would be markedly prone to the effects of detraining. Maintaining pre-injury levels of fitness with players who are not able to perform soccer-specific running-type exercises at previous intensities however, may be difficult. A method of evaluating the effects of soccer injury on performance indices important to soccer is therefore required. Although there is a plethora of studies into the effects of detraining, no studies can be found in the available literature that investigated the effects of injury-induced alterations in habitual training patterns in groups of athletes.

3.4.5 Conclusion

The current study found that the risk of injury, especially during first team matches, is high. Soccer injuries predominantly affect the lower limb and involve structures which affect the mobility and joint stability of the injured player. Injuries resulting in absence of sufficient duration to make detraining a possibility occur approximately 18 times per season. Of these injuries, 16 are sustained to the lower limb. This means that, at any point during a competitive season, a typical division one club will have, on average, almost two players who have sustained an injury to a lower limb, anatomical structure which prevents soccer-specific exercise. The effects of these injuries could include the inability to maintain training load and/or training intensity which could result in detraining.

CHAPTER 4

The effect of injuries on performance indices in professional soccer and the contributory factors to detraining.

4.1 Study 4: The effects of detraining on gross motor performance.

4.2 Study 5: A quantitative comparison of the training loads of professional soccer players during rehabilitation and normal squad training.

The previous chapter showed that detraining may be a significant problem for professional soccer players following injury. This chapter investigates whether detraining results in decrements in soccer-specific performance and explores reasons why the inability to train normally may result in negative physiological adaptations.

4.1 STUDY 4: THE EFFECTS OF DETRAINING ON GROSS MOTOR PERFORMANCE.

4.1.1 Introduction

The previous chapters have shown that injuries in soccer are common and that injuries sustained to the lower limb, of a severity which may result in detraining, occur on average 16 times per season in this population. Chapter 3.4 also showed that 13% of all SRRI's were sustained within 2 months of a previous injury. This may suggest that there could be a link between the activities completed during rehabilitation and future injury risk.

One reason for this possible failure to fully rehabilitate players may be a lack of appreciation of the subtle physiological adaptations that may have occurred during the period of injury. These adaptations may be as a consequence of the injured player's altered training stimulus. This lack of awareness may result in unrealistic training expectations of the injured player and premature return to normal squad training and competitive action. An understanding of the extent of any possible functional and/or performance decrements, as a result of detraining may therefore be invaluable when designing a safe and progressive rehabilitation programme and thus reducing the incidence of re-injury (Parkkari *et al.*, 2001). Limited information is currently available on such performance decrements as the majority of previous detraining studies have relied upon limited subject groups (non athletes) following a training period/detraining period design. No research can be found that investigates professional soccer players who, as a result of injury, undergo periods of detraining of varying duration and with injuries that will permit rehabilitation protocols of different intensities and designs.

In professional soccer, it is normally the role of the physiotherapist or sports rehabilitator to ensure that players returning from injury are suitably prepared to withstand the stresses of training and competition. In professional soccer, players must possess moderate to high aerobic and anaerobic power, have good agility, joint flexibility and muscular development and be capable of generating high torques during fast movements (Reilly *et al.*, 2000b). Sprint ability over short distances (Reilly *et al.*, 2000b) and vertical jump height (Wisløff *et al.*, 2004; Kalapotharakos *et al.*, 2006) have also been reported to be fitness prerequisites for professional soccer players.

Physical ability tests, targeting some of the above sub-components of fitness, are often used to determine soccer players' level of readiness (Bangsbo and Linquist, 1992; Rösch *et al.*, 2000; Aziz *et al.*, 2000; Chamari *et al.*, 2004; Chamari *et al.*, 2005), identify possible talent (Reilly *et al.*, 2000a) or differentiate between competitive levels of play (Dunbar and Power, 1995; Edwards *et al.*, 2003). These outcome tests could also be utilised to provide a quantitative indicator of an injured player's readiness to return to squad training and competition as players returning from injury must be suitably rehabilitated to withstand all of the physical and physiological demands of competitive play. Testing components of fitness as a quantitative measure of readiness may therefore play an important role in determining the extent of this readiness and therefore reducing any potential risk of re-injury. The aim of the current study is therefore to determine the effects, using gross motor performance tests, of injured players' inability to participate in normal squad training and play competitive matches due to pathologies of varying severity.

4.1.2 Methods

4.1.2.1 Subjects

This study was conducted over one soccer season between August 2004 and May 2005. All professional players contracted throughout the study period (n=27) at one English League One soccer club agreed, in writing in accordance with the Liverpool John Moores University ethical procedures, to allow the use of their injury and physical activity data to be included in the present study. Each player was identified by a randomly assigned code in order to protect confidentiality.

At the beginning of the current study period, players were tested for a number of fitness indices considered to be important for soccer performance. Players who sustained injuries during the study period were re-tested on their return to normal training to determine the extent of any changes in the same specific fitness parameters. This information would give an index of the player's loss of capability due to the injury.

Section 3.1 details the methodology for the collection of attendance and absence data. At the beginning of the study period, all contracted players (n=25) were able to train and play. During the study period, 2 players left the club and 2 joined. At the end of the study period

one player was injured and unable to train. His data was excluded from the current study. A total of 284 days spanned the first and last competitive match of the season, 22 (79%) players were at the club for all 284 days. The 4 players who joined or left the club averaged 154 ± 64 days.

4.1.2.2 Categorising injury severity.

Injury severity was categorised using the total number of days players were unavailable to train normally or play due to injury. An injured player was considered injured until he was able to fully participate in all aspects of squad training (Hägglund *et al.*, 2005). If a player participated in squad training in a modified manner, e.g. missed out on a full-contact component of the session, they were considered to be still injured. In order that possible effects of different durations of absence through injury could be investigated, injury severity categories were classified as detailed in Table 4.1.1. These categories are similar to those utilised in previous detraining investigations: ~14 d (Ready *et al.*, 1981; Coyle *et al.*, 1985; Hortogagyi *et al.*, 1993), 4 weeks (Yuza *et al.*, 2000), and 60 d (Klausen *et al.*, 1981; Ready and Quinney, 1982).

Table 4.1.1 Definitions of injury severity

Slight injury	*A soccer-related incident that resulted in between 2 and 6 days absence irrespective of a training session or match being timetabled.*
Minor injury	*A soccer-related incident that resulted in between 7 and 14 days absence irrespective of training sessions or matches being timetabled.*
Moderate injury	*A soccer-related incident that resulted in between 15 and 28 days absence irrespective of training sessions or matches being timetabled.*
Major injury	*A soccer-related incident that resulted in between 29 and 60 days absence irrespective of training sessions or matches being timetabled.*
Severe injury	*A soccer-related incident that resulted in >60 days absence irrespective of training sessions or matches being timetabled.*

4.1.2.3 Testing

All professional players were tested within 2 weeks of the start of the 2004-05 competitive season using a battery of soccer-specific performance tests. This was to ensure base data was taken when players were fit and playing competitive soccer. In an effort to keep data current, fit players were re-tested throughout the season when training schedules allowed. Although the on-going re-testing was not timetabled, all fit players were re-tested in December which is approximately half way through the competitive season. All testing always followed the methods detailed below. For data analysis, a player's most up to date performance data in any test were used (T1). Players joining the club were tested as soon as was practicable within 2 weeks of joining the club. Players returning from injury were retested immediately prior to rejoining squad training (T2). If an injured player had returned from a previous injury 3 months prior to the subsequent injury, the data for the second injury were not included in the study. This was to ensure that players included in the study were not still suffering the effects of a previous injury.

All testing took place in the same indoor training area with a hard carpeted floor. This eliminated some of the extraneous variables normally encountered outdoors. The training area floor was permanently marked to ensure consistency of timing gates and start/finish line position. Players were informed about the most appropriate footwear (trainers), and instructed to ensure that the same type of footwear was used for all testing. Players were verbally informed to keep to their normal diet and not to consume food or any liquid apart from water for 3 hours prior to testing. They were also instructed to refrain from alcohol, drinks containing caffeine or any ergogenic aid during the 24 h prior to testing. Coaches also ensured that players abstained from high intensity exercise on the day prior to testing. A standardised 15 min jog and stretch warm up preceded all tests. All players were verbally encouraged throughout all tests. This verbal encouragement was kept simple and as consistent as possible for each of the players during all testing.

4.1.2.4 Familiarisation

Familiarisation testing took place as an integral part of the pre-season training programme. All players had completed each test on at least three occasions prior to the first testing session. Players joining the club also completed three familiarisation sessions prior to initial testing.

4.1.2.5 Performance tests

All players completed a battery of soccer-specific field tests designed to evaluate gross motor performance in a number of components of fitness considered important to soccer. These included anthropometric measurements including height in m (using a wall mounted stadiometer, Seca, 222, Hamburg) and total body mass in kg (using counterbalance scales, Avery Berkel, Fereday and Sons, Walsall, UK). Estimated body fat percentage (% BF) was calculated using the sum of 4 skinfolds: triceps, biceps, sub-scapula and supra-iliac, using Harpenden skinfold callipers (Quinton Instruments, Seattle) as described by Durnin and Womersley (1974). The Multistage Fitness Test (MSFT) (Léger *et al.*, 1988), agility T-sprint (Semenick, 1990), 'flying' 20 m sprint, 6 x 20 m repeated sprint ability test (RSA), countermovement jump (CMJ) and active knee extension (AKE) to determine hamstring flexibility (Gajdosik and Lusin, 1983) were also completed.

Test order was maintained for all testing sessions to reduce systematic error. The testing day was divided into morning and afternoon sessions. Players were given at least 10 minutes rest in between each test. Players were instructed to recover actively between attempts for a given test by walking around the perimeter of the sports hall.

4.1.2.5.1 Morning session

Multistage fitness test (Léger and Lambert, 1982). The MSFT involved running between two lines set 20 m apart at a pace dictated by a CD recording emitting tones at appropriate intervals. Velocity was set at 8.5 $km \cdot h^{-1}$ for the first minute, increasing by 0.5 $km \cdot h^{-1}$ every minute thereafter. The test score achieved by the subject was the number of stages (one minute sections) and single 20 m laps completed before the subject either withdrew voluntarily from the test or failed to be within 3 m of the end lines on 2 consecutive tones. A performance matrix, which accompanied the CD, was used to transform the stage and number of shuttles to an estimated $\dot{V}O_{2max}$. To provide an additional variable of the players' endurance performance, the total distance covered was also calculated. To calculate total distance, the total number of shuttles completed was multiplied by 20 m. This test has been validated as a predictor of $\dot{V}O_{2max}$ by Léger *et al.* (1988) and by Ramsbottom *et al.* (1988). Each player's heart rate was monitored continuously during the MSFT at 5-s intervals using short-range radio telemetry, (Polar Team System. Polar Electro Oy, Kempele, Finland) to help ensure a maximal effort was produced by each individual.

Active knee extension (AKE) test (Gajdosik and Lusin, 1983). Prior to performing the AKE test, all players completed a standardised warm up in a gymnasium adjacent to the room in which testing took place. In addition to previous warm-up and activity, players performed a 5 minute warm up on a cycle ergometer (Aerocycle, Universal inc. USA) at a cadence of ~80 rpm followed by standardised stretching. This was to ensure optimum hamstring extensibility. Figure 4.1.1 illustrates the setup for the AKE. The subject lay in a supine position on a bench with one thigh strapped tightly to the bed to prevent hip flexion and to fix the pelvis. The other leg was positioned against a sturdy vertical jig so that the lateral centreline of the thigh was as close to vertical as possible. This jig was secured to the bed to prevent movement. The subject's arms were placed across his chest and he was instructed to maintain contact with the bed with his head. This setup was to minimise peripheral body movement. At this point, self-adhesive markers (1 cm diameter) were fastened to the skin of the vertical leg to provide anatomical datum points. These points were located on the centre of the greater trochanter, the lateral femoral epicondyle and the tip of the lateral malleolus. The thigh was then held in position against the jig by a third person and the player was asked to straighten his knee as far as he could and to hold the position while a digital photograph was taken. Two cameras (Olympus DX435) were set up 3 m either side of the player, level with the vertical thigh and at approximately the height of the lateral epicondyle on the knee joint. This process was then repeated for the contra-lateral leg. These images were analysed using the anatomical datum points (Visio 2003 software, Microsoft Corp., Redmond, Washington) and range of motion (degrees short of full knee extension) was recorded.

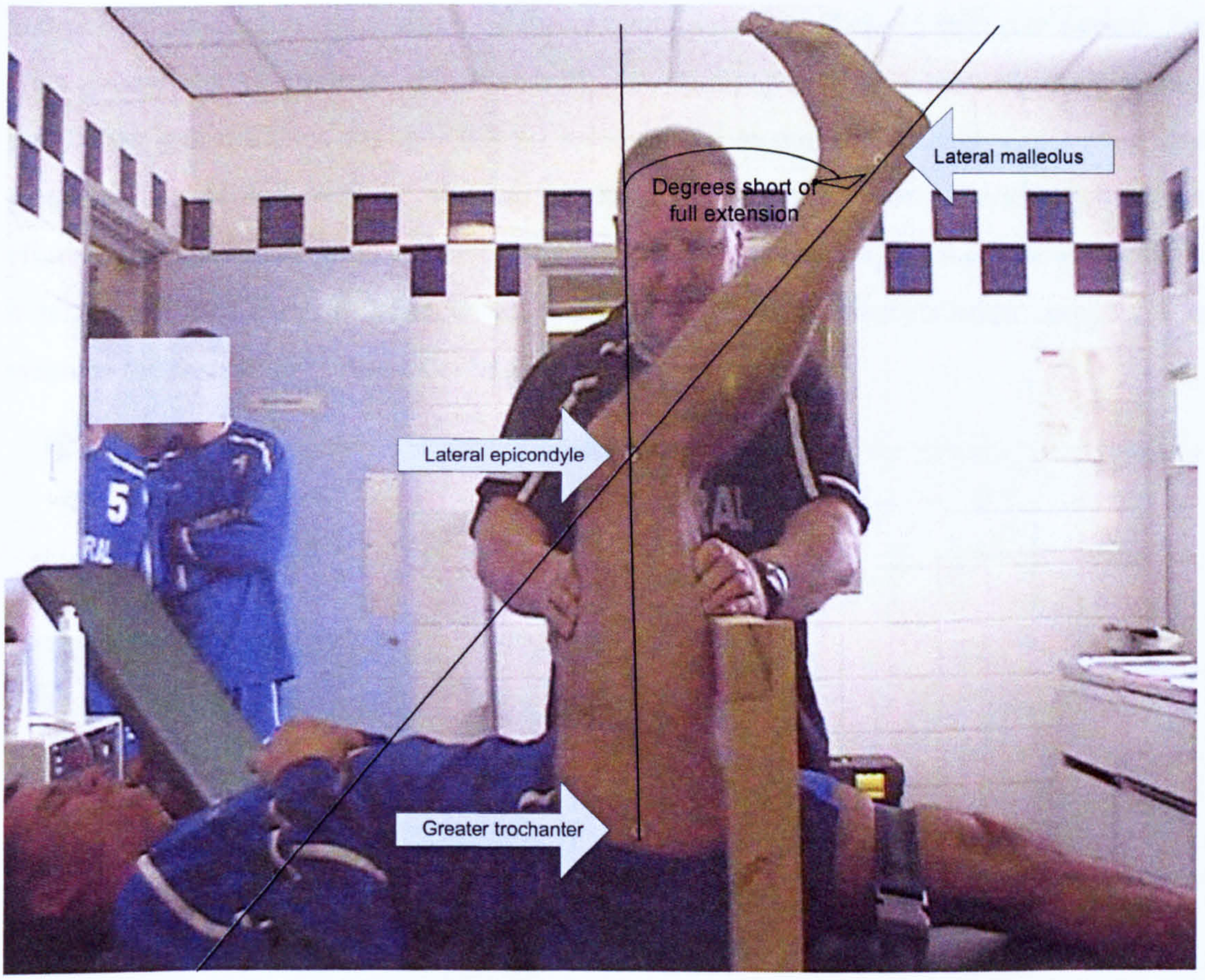


Figure 4.1.1 Illustration of the set up for the Active Knee Extension test (Gajdosik and Lusin, 1983). With a flexed knee, the thigh was held vertically and the player asked to straighten the leg as far as possible. The number of degrees short of full extension was recorded.

5 m T-sprint. This test was first published by Semenick (1990) and found to be highly reliable by Pauole *et al*. (2000). In the current study, the legs of the 'T' were shortened from 10 yards to 5 m in order that the total distance covered matched that of the 20 m flying sprint test. Figure 4.1.2 illustrates the set up for the T-test. Players started 0.3 m behind the timing gates and to eliminate any influence of reaction time, started the test when they were ready. Players sprinted forwards 5 m (1) before side-striding to either the left or right for 2.5 m (2). This was followed by a 5 m side-stride in the contra-direction (3), changing direction again for a 2.5 m side stride (4) and a 5 m backward run through the timing gates (5) to finish. The total movement distance was 5 m forwards, 5 m side stride to the left, 5 m side stride to the right, 5 m backwards. Players were instructed to alternate between right and left for the initial side-stride element for each trial. During the lateral elements of the test, players were instructed to bring their feet together in-between steps and not to cross feet. Violation of this

instruction resulted in termination of the test and a re-run after a 5 min rest period. Players were encouraged to maintain maximal backward running until they were all the way through the timing gates. Cones (height, 1.0 m) were placed at the end of each component and the player was expected to touch the top of the cone on completion of each component. To eliminate any doubts regarding the player touching the cone, players were instructed to touch it with sufficient force to produce an audible 'slap'. Five attempts were completed and a mean of the fastest three was calculated and used for analysis.

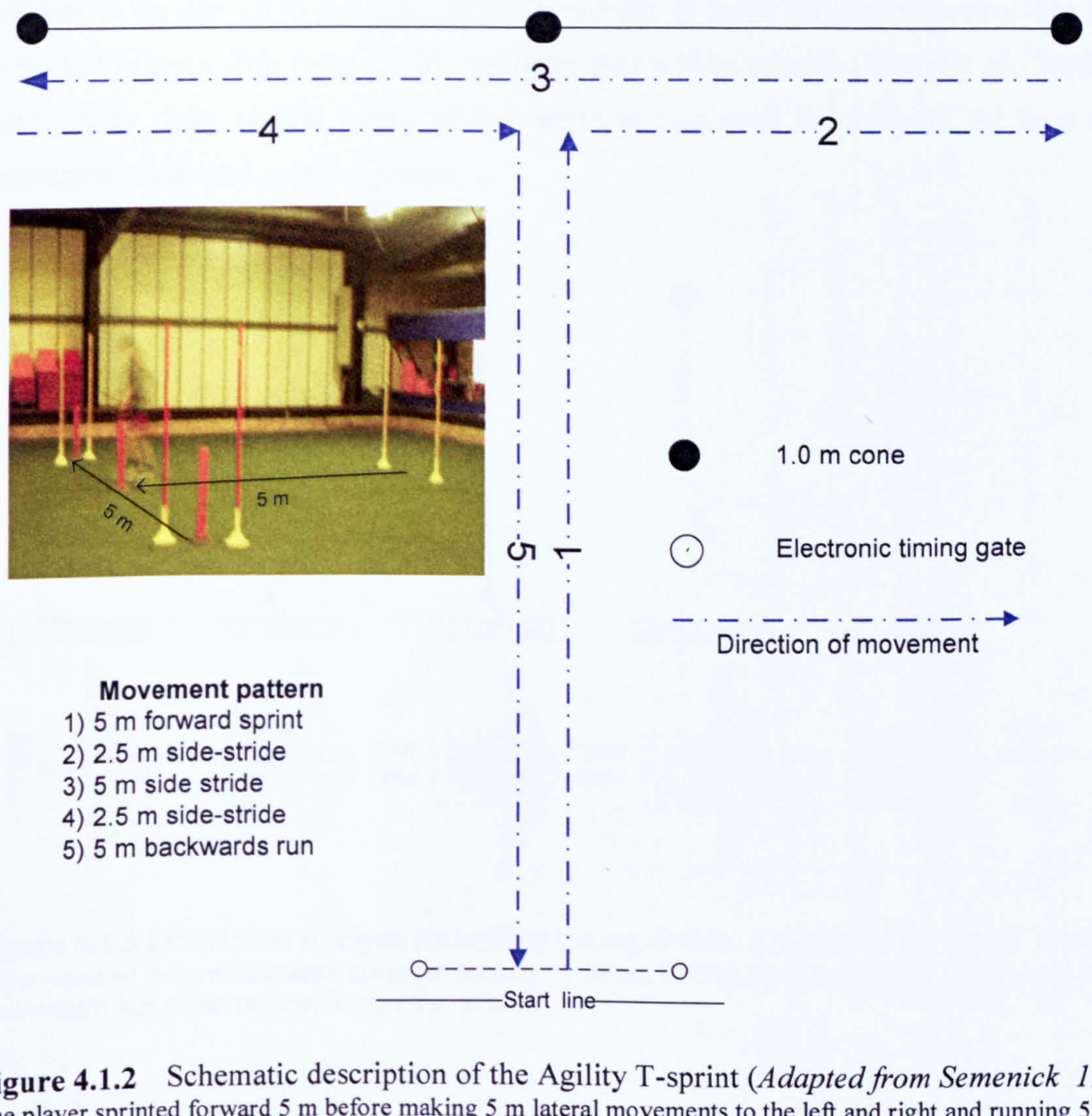


Figure 4.1.2 Schematic description of the Agility T-sprint (*Adapted from Semenick 1990*). The player sprinted forward 5 m before making 5 m lateral movements to the left and right and running 5 m backwards through the starting gates.

Countermovement jump. Players performed five attempts of a 'freestyle' CMJ on a device that allowed the measurement of flight time (GPL PlyoTrainer; Gold Performance UK. Ltd.) (Figure 4.1.3.). This apparatus consisted of a digital timer (± 0.01 s) connected by a cable to a reaction mat. The timer was triggered by the feet of the player at the moment of release

from the mat and stopped at the moment of touch down. The electronic timing device indicated flight time (ms) and jump height (mm). This method of calculation assumes that the position of the jumper was the same at take off and landing. Players were therefore instructed to land with extended knees and hips to replicate their anatomical position at the point of take-off and so eliminate extended flight time. Players were encouraged to squat until their knees were flexed to ~90°. Players were instructed not to repeatedly swing their arms prior to takeoff with the exception of the one backward movement on lowering down to the squat position. In an attempt to maintain soccer-specificity of tests, no further control was placed on the use of arms. This form of CMJ has been shown to be reliable (Slinde *et al.*, 2008). The mean of the three highest jumps of five attempts was used for analysis. At least 2 min recovery was allowed between jumps.

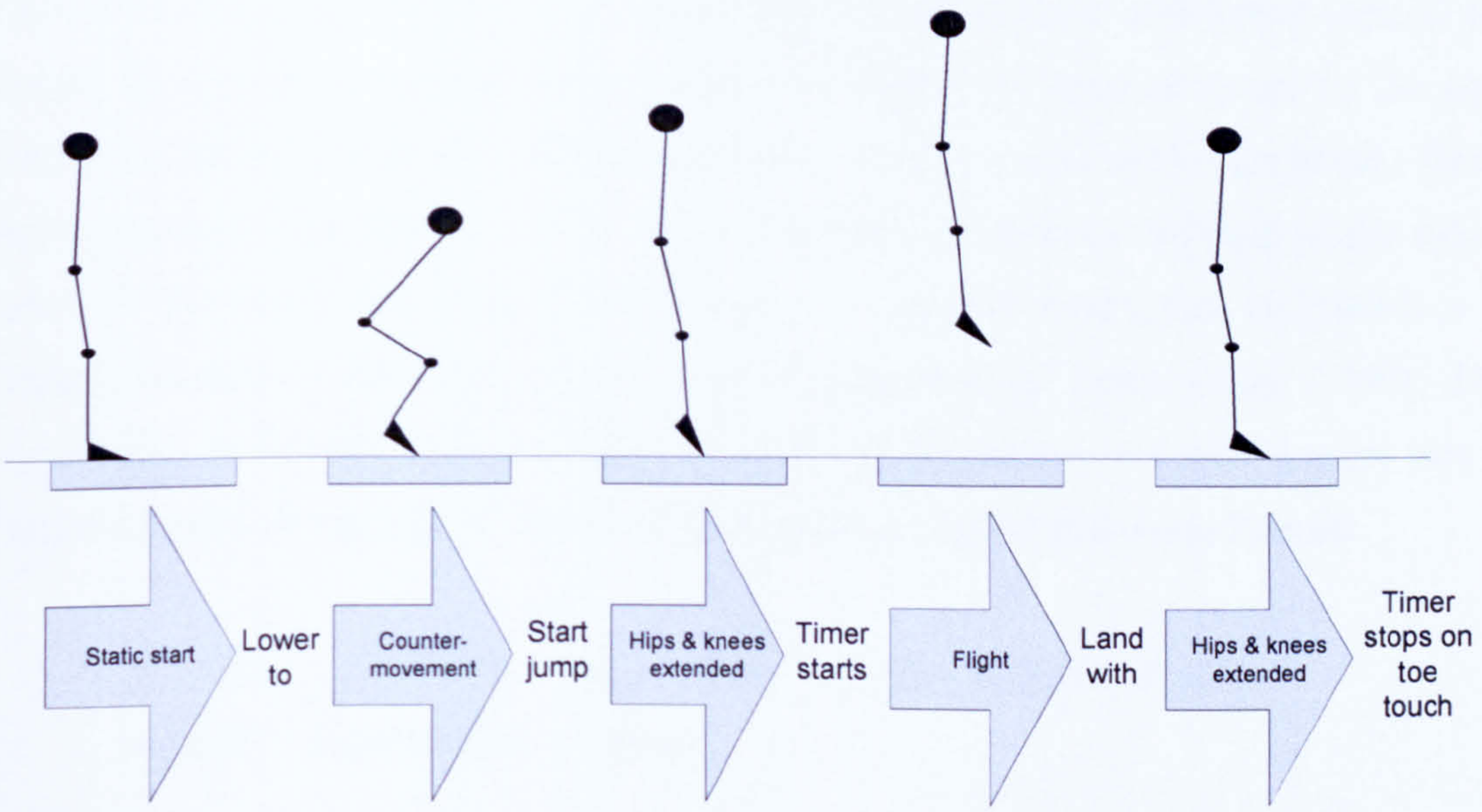


Figure 4.1.3 Description of Plyotrainer jump timing device. *Adapted from Slinde et al. (2008).* The player squatted down until knees were approximately 90 flexed, he then jumped as high as possible and landed with straight legs to recreate the taking off position.

4.1.2.5.2 Afternoon session

20 m flying sprint: Electronic timing gates (Eleiko Sport, Type S5812. Sweden) were set up 20 m apart. The photocells were positioned 1.2 m above the ground to avoid triggering the timing gates prematurely during the sprints. A start line was marked 20 m before the starting gates to give a 20 m acceleration distance and therefore produce a 'flying' 20 m sprint. Players were verbally encouraged to maintain maximum velocity through the second timing

gate and not to decelerate until they were passed through. Five attempts were made with at least 3 minutes active recovery between attempts. The mean of the best three attempts was used for analysis.

Repeated sprint ability test. Timing gates (Eleiko Sport, Type S5812. Sweden) were set up as for the flying 20 m sprint using the same floor markings with the addition of a taped start line positioned 0.3 m outside each set of gates. This was to reduce the risk of the timing gates registering any random body movements prior to the start of each sprint. This helped reduce the risk of the timing gates registering a premature start. The repeated sprint ability test consisted of 6 x 20 m maximal sprints from a standing start with a 10 s recovery between each run. The 10 s recovery period began as soon as the subject finished the previous 20 m sprint. The recovery period was timed using a hand held electronic stop watch (Unwin, 100 Series. Nottingham, England). The players started their sprints at alternating ends of the 20 m as the 10 seconds allowed between sprints would not have been sufficient for the players to return to the same start line. All subjects were verbally encouraged throughout. This verbal encouragement was kept as simple and as consistent as possible for each of the subjects on each of the testing dates. Total time (s) for each set of 6 sprints was calculated. A fatigue index was also calculated using the formula suggested by Baker *et al.* (1993). This was calculated as the difference between the mean of the fastest two and slowest two sprints expressed as a percentage of the fastest two sprints using the following formula.

$$(S1+S2)/2 - (F1+F2)/2 = \text{Diff} \quad \text{then} \quad (\text{Diff} \times 100)/ (F1+F2)/2$$

where S = slowest and F = fastest

Higher fatigue indices represented a greater level of fatigue during the test. To allow for a quantitative comparison of the physiological response during each of the repeated sprint tests, heart rate was monitored continuously at 5-s intervals using short-range radio telemetry, (Polar Team System. Polar Electro Oy, Kempele, Finland).

4.1.2.6 Test retest reliability

A group of 18 apprentice soccer players (age 17 ± 1 years) were used to determine test-retest reliability for, 20 m sprint, T-sprint, repeated sprint, MSFT, CMJ and AKE (Table 4.1.2). All players were fully familiarised having completed all tests on at least 3 occasions previously.

Two testing days, with a rest day in between, were used as a model to collect test retest data. The methods and pre-test controls as described in 4.1.2 were used.

Student's t-test analyses were performed to investigate the presence of any significant change in the mean of the two tests for all performance variables. Statistical procedures employed to assess reliability were intraclass correlation coefficient (ICC) to determine the degree to which individuals maintain their position in a sample with repeated measures (Atkinson and Nevill, 1998) and Typical Error (TE) which allows the investigator to appreciate the extent to which the observed score would deviate from the true score (Spencer *et al.*, 2006a). Analysis was performed using the spreadsheet for reliability provided by Hopkins (2000) and SPSS Version 12 for Windows (Chicago, Illinois, USA).

Table 4.1.2 Test-retest reliability co-efficients all performance tests.

			95% CI		
	Change in mean	TE	Lower (%)	Upper (%)	Intraclass *r*
T-sprint (s)	-0.13	0.15	0.12	0.21	0.81
Flying 20 m sprint (s)	-0.01	0.02	0.01	0.02	0.99
RSA fatigue index (%)	-0.8	1.06	0.72	2.04	0.6
RSA total sprint time (s)	0.55	0.27	0.19	0.45	0.95
MSFT ($ml \cdot kg^{-1} \cdot min^{-1}$)	0.8	0.37	0.21	0.49	0.94
CMJ (cm)	-1.85	10.6	8.4	14.5	0.91
AKE (°)	-0.27	1.36	1.06	2.01	0.97

The performance variables of agility T-sprint, flying 20 m sprint, RSA total time, RSA fatigue index, MSFT, AKE, and CMJ all had a high degree of reliability in this group of young professional soccer players assessed with ICC. Using the guidelines suggested by Cohen (1988), the ICC was large for all variables indicating that players maintained their position in the sample. There was no difference in player performance between the reliability test days when compared using Student's paired t-tests ($p>0.05$ for all variables). The small typical error for T-sprint, flying 20 m sprint, RSA total time, MSFT and RSA fatigue index suggest that inconsistency of measurement in these variables would occur in an acceptably

small range. We believe that the ICC, Student's t-test and TE reported indicate that all performance tests provided a reliable measure of performance.

Statistics

All data was processed on a Dell computer (Dell Inc., Wicklow, Ireland) using Microsoft Office (Microsoft Corp., Redmond, Washington). The statistical procedures were completed using SPSS Version 12 for Windows (Chicago, Illinois, USA). Methods applied were frequencies, crosstabulations, descriptives, and means ± standard deviation (SD). Verification that the data were normally distributed was provided by the Kolomogorov–Smirnov normality test and visual inspection of the normality plots. This assumption was not violated. Due to small subject numbers in each severity category, 95% confidence intervals (CI) of the difference are also expressed where appropriate. In order that relationships between changes in key performance variables could be investigated, Pearson product-moment correlation coefficients were used to compute the relationship between changes in T1 and T2. One-way analysis of variance (ANOVA) was used to test differences between injury severity groups between T1 and T2 in all performance variables. One-way analysis of variance was also used to investigate sprint performance times between T1 and T2 for all 6 sprints. Statistical significance was accepted as $p < 0.05$ unless stated otherwise.

4.1.3 Results

During the study period there were 49 incidents of players missing at least 2 training days due to injury. Analysis of slight (n=15), and minor (n=19) injuries showed no significant differences in any of the performance tests between T1 and T2 ($p > 0.05$) (Table 4.1.3). The only performance measure that approached a significant change was the agility T-test (change in mean = -0.05 ± 0.13 s, p=0.06). It would therefore appear that absences <15d do not result in performance decrements in fitness parameters important in soccer. Further results will therefore ignore injuries resulting in <15 d absence (slight and minor categories) from training and focus on injury periods that are >14 d.

Table 4.1.3 Student's paired t-test results for T1 and T2 for all performance measures of players absent through injury for <15 d.

	Paired Differences			
	Change in mean (± SD)	95% CI Lower	95% CI Upper	Sig. (2-tailed)
Mass (kg)	-0.2 ± 1.3	-0.7	0.31	0.43
BF%	0.23 ± 0.9	-0.14	0.59	0.22
Flying 20 m sprint (s)	0.01 ± 0.06	-0.02	0.03	0.83
Agility T-sprint (s)	-0.05 ± 0.13	0.00	-0.02	0.06
Est. $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	0.6 ± 2.7	-0.3	1.6	0.19
RSA fatigue index (%)	-0.3 ± 2.2	-1.2	0.6	0.46
RSA total sprint time (s)	-0.07±0.2	0.12	0.33	0.81
CMJ (cm)	0.3 ± 2.7	-.72	1.37	0.53
AKE (°)	0.8 ± 2.8	-.29	1.89	0.14

During the same period there were 15 incidents of players not being available to train for >14 d (Severity of injuries; moderate n = 6; major n = 7; severe n = 2). Table 4.1.4 details the anatomical location of the injuries as a factor of injury severity. Thirteen of the 15 injuries (87%) were sustained to the lower limbs. The mean number of days absence in each category was 21 ± 3 d for moderate; 40 ± 9 d for major and 70 ± 10 d for severe injuries.

Table 4.1.4 Crosstabulation for anatomical location of injuries of >14 d as a factor of injury severity

General anatomical area of injury	Severity of injury 15-28 d	29-60 d	> 60 d	Total >14 d
Head and neck	0	0	1	1
Shoulders and arms	0	1	0	1
Thigh	3	3	0	6
Knee	1	3	0	4
Ankle	1	0	0	1
Foot	1	0	1	2
Total	6	7	2	15

Player's mean performance data for T1 and T2 is shown in Table 4.1.5. When these data were analysed (Table 4.1.6), no significant differences between T1 and T2 were observed for body mass, estimated % BF, flying 20 m sprint time, RSA total sprint time, CMJ and hamstring flexibility ($p>0.05$). Significant decreases (2%) in Agility T-sprint performance (T1, 6.2 s ± 0.3; T2, 6.3 s ± 0.3, $t = -2.6$, $p=0.019$) and RSA % fatigue index (12%) (T1, 7.3 ± 5%; T2, 8.0 ± 6%, $t = -2.84$, $p=0.01$) were observed between T1 and T2. When investigating the effect of sprint number on 20 m sprint time, sprint number 1 and 2 were significant quicker than sprints 4, 5 and 6 during both T1 and T2 ($p<0.05$).

Table 4.1.5 Descriptive statistics for T1 and T2 for all performance variables for players injured >14 d (Mean, SD).

	Test 1		Test 2	
	Mean	SD	Mean	SD
Flying 20 m sprint (s)	2.5	± 0.3	2.5	± 0.3
Agility T-sprint (s)	6.2	± 0.3	6.3	± 0.3
Est. $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	56.1	± 5.2	54.5	± 4.9
RSA fatigue index (%)	7.4	± 5.5	8.3	± 6.3
RSA total sprint time (s)	20.09	±0.9	20.13	±0.9
CMJ (cm)	41.8	± 3.9	41.9	± 3.9
AKE (°)	18.8	± 8.9	18.9	± 8.9

Estimated $\dot{V}O_{2max}$ was also significantly reduced (3%) for T2 (T1, 56 ± 5 $ml \cdot kg^{-1} \cdot min^{-1}$; T2, 55 ± 5 $ml \cdot kg^{-1} \cdot min^{-1}$, $t = 2.25$, $p=0.038$). Substituting estimated $\dot{V}O_{2max}$ with the distance covered (m) in the MSFT also showed reductions between T1 and T2 (T1, 2420 ± 362 m; T2, 2303 ± 341 m, $t = 2.6$, $p=0.022$). Examination of the difference between T1 and T2 as a function of injury severity was also performed. No differences were observed between any of the severity categories for any of the performance variables ($p>0.05$).

Table 4.1.6 Paired Samples Test for anthropometric and all performance variables for T1 and T2

	Paired Differences			
	Change in	95% CI		Sig.
Performance variable	mean ± SD	Lower	Upper	(2-tailed)
Mass (kg)	-0.4 ± 1.0	-0.81	0.20	0.21
% BF	-0.01 ± 0.8	-0.41	0.39	0.70
Flying 20 m sprint (s)	-0.02 ± 0.04	-0.04	0.01	0.13
Agility T-sprint (s)	-0.1 ± 0.1	-0.16	-0.02	0.03*
Est. $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	-1.6 ± 2.5	0.24	3.0	0.03*
RSA fatigue index (%)	-0.9 ± 1.1	-1.31	-0.19	0.01*
RSA total sprint time (s)	-0.04	0.17	0.48	0.59
CMJ (cm)	0.06 ± 2.8	-1.53	1.10	0.94
AKE (°)	-0.1 ± 1.9	-1.01	0.79	0.85

* Significant difference between T2 and T1, $p<0.05$

4.1.4 Discussion.

In the current study, significant performance decrements were found in aerobic power, agility and repeated sprint ability following periods of absence from normal training of > 14 d. No significant differences were found in body mass, % BF, flying 20 m sprint time, CMJ height and hamstring flexibility. Analysis of decrements using the injury severity categories moderate, major and severe demonstrated no significant differences in any absence duration category. This was probably due to the small numbers in each category. These decrements may leave players returning from injury ill prepared to withstand the physical demands of competitive soccer. This, in turn, may lead to re-injury.

Maximal aerobic capacity is generally accepted as the most valid measure of overall cardiorespiratory fitness (Saltin and Åstrand, 1967) and is an important attribute in professional soccer (Reilly, 1994a). A significant reduction in estimated $\dot{V}O_{2max}$ from T1 to T2 was observed in the current study. It has been previously suggested that participants performing the MSFT may be influenced by subjects dropping out prematurely and that some athletes have a predetermined idea as to what stage they are going to reach and so may voluntarily drop out early (Wilkinson *et al.*, 1999). This may suggest that players failing to

give maximal effort in T2 could explain the significant reduction in the estimated $\dot{V}O_{2max}$ observed. If a submaximal effort had been responsible for the performance decrement, it would be expected that players who were absent for <15 d would also be prone to the same lack of self motivation and hence a similar performance decrement would have been observed for those players. In the current study, there was no such significant performance decrement in the MSFT for players injured <15 d ($p>0.05$) suggesting that this did not occur. An analysis of mean heart rate for the final one minute of the MSFT showed no significant differences between T1 and T2 (mean 196 ± 11 bpm versus 196 ± 7 bpm, $t = 0.19$, $p=0.85$). This suggests that players re-produced a similar level of effort in each test. It is therefore unlikely that a significant difference in player effort between T1 and T2 was a contributory factor to the performance decrement observed in players absent >14 d.

The effects of detraining on the aerobic energy pathways and aerobic performance are well documented and as such, these findings are not surprising. A consensus of opinion points towards a rapid (<10 d) reduction in $\dot{V}O_{2max}$ (Klausen *et al.*, 1981; Dahlström *et al.*, 1987) predominantly as a result of a reduction in oxidative enzymatic activity (Coyle *et al.*, 1985; Linossier *et al.*, 1997). A rapid decline in the activity of aerobic enzymes will therefore result in a reduction in the efficiency of the aerobic energy pathway and a concomitant reduction in aerobic performance (Hawley, 1987).

Previous work has suggested that maintaining a reduced work load during training can result in the maintenance of aerobic performance (Hickson *et al.*, 1985; Houmard *et al.*, 1989). Fleck (1994) found that reducing the duration of training by two thirds and training frequency by a half provided a sufficient training stimulus to maintain $\dot{V}O_{2max}$ and sub-maximal performance after 15 weeks. This statement would seem to contradict the current findings as injured players underwent adapted training programmes of similar frequency and duration to normal training yet still lost fitness. It has also been demonstrated that the intensity of each exercise session within a training programme is a major factor when the aim is to maintain $\dot{V}O_{2max}$ in athletes (Neufer, 1989). In the current study, the intensity of rehabilitation training was not quantified. It may therefore be that the intensity of rehabilitation training was significantly lower than normal squad training. This may lead to reductions in the indices of aerobic fitness as we have seen here. It would be beneficial to evaluate the intensity of rehabilitation training in future studies to see if there is a significant difference between the

intensity of rehabilitation training and normal squad training. This may also allow investigation into the extent to which differences in training loads may explain the performance decrements we have observed.

Soccer entails intermittent exercise with bouts of short, intense activity punctuating longer periods of moderate intensity exercise (Drust *et al.*, 2000). Although high intensity sprinting accounts for approximately only 5% of the time during a match (Reilly, 1997), with the majority of sprints performed being less than 20 m or approximately 3.5 s (Bishop *et al.*, 2001), they are related to the key, result determining moments of games. It would therefore appear that the ability to reproduce maximal short distance sprints with little rest may be one of the more important determinants of success in soccer (Wragg *et al.*, 2000). In the current study, a significant increase in the fatigue index of the RSA was found between T1 and T2. Examination of sprint 1 times between T1 and T2 showed no significant differences (mean ± SD T1, 3.26 ± 0.13 s, T2, 3.2 ± 0.12 s) suggesting players started both tests with similar performance and effort. Many researchers suggest that a high level of aerobic fitness is a prerequisite for a superior anaerobic performance during sustained intermittent activity (Aziz *et al.*, 2000). The major role of aerobic metabolism during multiple sprints appears to lie in its contribution to the restoration of homeostasis during the intervening recovery periods (Glaister *et al.*, 2005). This would primarily involve replenishing anaerobic energy supplies such as ATP and phosphocreatine (PCr) (Reilly 1997; McMahon *et al.*, 1998; Aziz *et al.*, 2000). It is therefore possible that the significant decrement in aerobic power found in this study also had an effect on the players' ability to re-synthesise ATP/PCr and their ability to repeat high-intensity efforts.

Due to the limitations of the number of timing gates available and the unavailability of a method for instantaneously recording the acceleration and velocity patterns of all 6 sprints, such as a radar velocimeter, no investigation into the individual phases of each sprint during the RSA test was undertaken. Had 2 additional timing gates been available and placed at a 5 m mark, it would have been possible to obtain an indication of performance in the initial 5 m acceleration phase and the final 5 m phase. It may therefore have been possible that only the acceleration phase or the maintenance of peak velocity was significantly affected in each sprint. The short sprint distance and 10 s turn-around between sprints may also have encouraged the players to decelerate prematurely, despite verbal encouragement. If this did occur, it would have had a significant effect on measures of fatigue. For those subjects who

maintained maximal velocity through the finish gates, the effort required to decelerate quickly and get them back to the start line within 10 s may also have had a significant effect of sprint times. Lakomy and Haydon (2004) investigated the effects of deceleration on fatigue indices during repeated sprint exercise. They found that, using a 6 x 40 m repeated sprint protocol with and without deceleration being limited to 6 m and departing every 30 s protocol, the deceleration phase could have a significant effect on fatigue. Evidence suggests that rapid deceleration in RSA protocols exacerbates the fatigue indices (Clarkson and Sayer, 1999). This deceleration induced fatigue, may therefore have had a greater effect on fatigue indices in the current study which utilised a much shorter 10 s recovery period. An RSA of longer distance and longer recovery periods may therefore better reflect a player's RSA. The fatigue index utilised in the current study (Baker *et al.*, 1993) utilised the quickest two and slowest two sprint times to calculate a fatigue index. It has been observed that there is often an increase in power or speed in the final sprint of RSA protocols (Glaister *et al.*, 2005; Glaister *et al.*, 2006; Glaister *et al.*, 2008) making any formula using the last sprint data questionable. Even though the current formula attempted to control for this effect by using the means of the quickest two and slowest two sprints, the degree of improvement in reliability may be insufficient to support the validity of this approach. The design of an RSA protocol specific to soccer and a more reliable indication of fatigue are other areas of methodology where further investigations may be warranted.

Reilly *et al.* (2000b) suggested that agility performance was a pre requisite in soccer, given the players are frequently involved in sudden directional changes in order to be effective during the game. Raven *et al.* (1976) rated agility as a soccer player's greatest asset. The current data showed a significant increase in mean T-sprint times between T1 and T2. No significant differences were found in the 20 m flying sprint times which suggests that the reductions in the ability to generate energy through the anaerobic energy pathways may not be a major contributory factor to this performance decrement. Explosive leg strength can also contribute to the ability to perform agility tests (Pauole *et al.*, 2000; Young *et al.*, 2002; Miller *et al.*, 2006). A failure to see significant reductions in jump performance between T1 and T2 may also suggest that changes in dynamic leg strength are also not a contributory factor to the decrement in agility performance.

It has been suggested that players in the final stages of rehabilitation may not have recovered psychologically from their injury (Smith *et al.*, 1990) and this could affect performance. This

may suggest that the current findings of a significant reduction in agility may have been as a consequence of a lack of confidence in the ability to perform maximally when making explosive turns and accelerations following absence through injury. The decrements in agility performance of players injured <15 d (mean change -0.5 s), which almost reached significance (p=0.06), may be indicative of this lack of confidence following injury and support this hypothesis. No work was carried out to elicit the player's psychological state during testing as it was beyond the scope of this thesis.

Good acceleration and maximum sprint speed are vital in professional soccer (Bangsbo, 1993). Success or failure may depend on the ability to reach a certain position or make contact with the ball before the opposition (Dowson *et al.,* 1999). In the current study, no significant changes were observed in 20 m sprint times. These findings concur with others (Chi *et al.*, 1983; Amigo *et al.*, 1998). Linossier *et al.* (1997) investigated short sprint performance before and after a 7-wk detraining period. They found that all the beneficial adaptations made to sprint training were maintained following detraining. These included a 28% increase in maximal power output, a 16% increase in peak force and considerable hypertrophy in type IIb muscle fibres. They concluded that a 7-wk detraining period was too short to have an effect on anaerobic performance potential. These findings regarding detraining and sprint performance, together with those of the present study, suggest that short sprint performance (~20 m) is more resilient to the effects of detraining and players may be able to withstand extended absence with no significant changes in sprint performance.

The number of subjects in the current study was dictated by the number and severity of injuries sustained within one season. This resulted in statistical problems associated with dealing with a variety of absence durations and low case numbers. This limitation also makes the study unique. The only other detraining studies found which used injured athletes as subjects have been case studies of single elite athletes (Nichols *et al.*, 2000; Godfrey *et al.*, 2005). The use of injured players also resulted in the inability to establish a time course for performance changes. This was due to the disabling effect of the various lower limb pathologies resulting in not being able to maximally test injured players at strategic points throughout their rehabilitation.

4.2.5 Conclusion

The current study was able to utilise established physical ability tests to identify gross performance decrements in sub-components of fitness considered to be important to soccer. Reductions in estimated $\dot{V}O_{2max}$, repeated sprint ability and agility were observed following periods of detraining >14 d. No performance decrements were found in jump height, flying 20 m time, RSA total sprint time or hamstring flexibility suggesting that these fitness parameters are more robust to the altered training stimuli delivered during rehabilitation. The current study draws no conclusions as to causative effects of these performance decrements although a difference in the physical and physiological demands of rehabilitation and normal squad training may be the most likely explanation. Further research is required to explore differences in normal squad training and rehabilitation training that may lead to these decrements.

4.2 STUDY 5: A QUANTITATIVE COMPARISON OF THE TRAINING LOADS OF PROFESSIONAL SOCCER PLAYERS DURING REHABILITATION AND NORMAL SQUAD TRAINING.

4.2.1. Introduction

Chapter 4.1. showed that injury periods of >14 d can result in significant decrements in physiological variables that are important for soccer performance such as aerobic power and repeated sprint ability. These physiological changes may mean that players return to competitive match-play lacking match sharpness (Doherty, 2007), unable to perform optimally (Ogden, 2007) and with an increased susceptibility to re-injury (Dvorak and Junge, 2000). If the reasons for these negative adaptations are to be elucidated, a thorough analysis of the rehabilitation process needs to be completed. Such analysis will facilitate a systematic comparison between the normal soccer participation associated with squad training and playing competitive matches (SMT) and that completed during rehabilitation from injury (RT).

A number of methodological approaches have been utilised in an attempt to quantify the load associated with SMT. Few, if any of these techniques however, have been used during periods of rehabilitation training following injury. Simple evaluations of the frequency and duration of the session, the product of which has been used by Houmard (1991) to give a time exposure or training volume per time unit represent the simplest methods used to quantify training load. Physiological markers such as heart rate (HR) (Little and Williams, 2007a), rating of perceived exertion (RPE) (Impellizzeri *et al.*, 2004) and blood lactate (Mujika *et al.*, 2004) are also popular as they provide some indication of the overall physiological stress of a training session. Researchers have also combined methodologies or further developed specific approaches beyond a simple representation of the data recorded. For example, training impulses (Trimps) first introduced by Banister *et al.* (1975) and subsequently modified (Mujika *et al.*, 1996; Foster *et al.*, 2001) are a popular method of utilising heart rate data to produce quantitative arbitrary units to assess the amount or volume of training undertaken in any one given bout. Another method of indicating the high intensity components of training work is the summated heart rate zone score method (SHRS)

(Flanagan and Merrick, 2002). With this approach, heart rates are separated into intensity zones and subsequently multiplied by the duration of time for which these HR's are maintained during the session to give an indication of the overall intensity. Ratings of perceived exertion can also be multiplied by session duration to give a session-RPE (SRPE).

All of these methods represent well established field-based protocols for quantifying SMT when the mode of training is soccer-specific (i.e. includes activities such as running drills, technique work and small-sided-games). Rehabilitation programmes are not, however, always characterised by functional soccer training. The restrictive nature of injury leads to the necessity to employ a wide range of exercise modalities and intensities to ensure that physical activity is undertaken within the constraints of the injury. Quantifying RT is therefore difficult due to both the miscellany of non-specific training modalities that may be incorporated and their potential impact on the validity of the approaches that are currently used to indicate training load.

These factors may explain why methodological evaluations of rehabilitation training have not been a focus in the available published literature. Investigations to date have limited their analysis to discrete elements of rehabilitation such as knee stability (Shelbourne and Davis, 1999), proprioception (Eils and Rosenbaum, 2001), strength training (Askling *et al.*, 2003) and outcome test measures (Fuller and Walker, 2006). It would therefore seem relevant to attempt to use recognised techniques to systematically quantify the internal training load throughout rehabilitation and compare it with the training and playing profile of the same players when not injured. This analysis would also enable the appropriateness of these approaches to quantify non-specific training to be evaluated. Such data would not only provide an understanding of the effectiveness of approaches to monitor training load in a wider context but also be valuable in evaluating rehabilitation programmes. This will enable the development of more effective interventions following injury for this specific population.

The purpose of this study was therefore to;

1) Utilise established methods of quantifying soccer training to quantify the global training load of a player's rehabilitation programme and compare it with the training load of the same player on return to normal soccer participation.
2) Using the same methods, systematically quantify and compare the soccer-specific and non-soccer-specific modes of exercise employed during rehabilitation.

4.2.2. Methods

4.2.2.1 Subjects

This study was conducted over a period between January 2006 and May 2007 with data being collected during the competitive phases of this period. This period covered the second half of the 2005-6 season (January 2006-May 2006) and all of the 2006-7 season (August 2006-May 2007). All contracted professional players (n=41; age, 25 ± 6 y; height, 1.84 ± 0.05 m; body mass 79 ± 7 kg; goalkeepers n=4, defenders n=13, midfielders n=15, strikers n=9) at one English League 1 club agreed, in writing in accordance with Liverpool John Moores University ethical procedures, to allow the use of their injury and physical activity data to be included in the study.

During the study period, 22 players sustained 27 injuries resulting in absences of >14 d. Insufficient data were collected for 2 injuries due to them extending into the non-competitive period at the end of the season. Five injuries were eliminated from the study as the second of 2 injuries sustained by the same players leaving 20 injuries for analysis (age, 25 ± 6 y; height, 1.8 ± 0.05 m; body mass 80 ± 7 kg; HR_{max} 190 ± 7 beats·min^{-1}; defenders n=8, midfielders n=7, strikers n=5). Analysis of the injuries showed that 93% of the original 27 and 100% of the final 20 were sustained to the lower body.

4.2.2.2 Experimental design

To profile and quantify relevant RT at the club, session duration, HR and RPE data were collected for all injured players for every training session throughout their rehabilitation programme following injury. The same measurement techniques were then subsequently used to evaluate the training load of the same players when returning to SMT. This analysis was completed for a similar period to that of the absence for RT. It was, however, considered that a 4-week period would be sufficient to reflect the habitual training patterns of uninjured players. As a consequence, a maximum observation period of 4 weeks was used for the analysis of SMT. Comparisons were made between the training load associated with SMT and RT to examine any differences between the training completed by players when fit and when injured. This data also provided the basis for a comparison of the loads associated with soccer-specific and non-soccer-specific exercises completed during both RT and SMT.

4.2.2.3 Data collection and inclusion criteria

In order that a thorough record of both SMT and RT exposure could be recorded, a comprehensive daily register as detailed in chapter 2.1 was completed for all players throughout the study period. A player was considered injured when he was unable to fully participate in all aspects of squad training (Hägglund *et al.*, 2005). In chapter 4.1 it was demonstrated that absences of <15 d do not have a significant effect on fitness parameters important to soccer performance. As a consequence, only players that were unable to train for a period >14 d with a soccer-related injury were included in the study. This would normally mean that the player was not available for at least one first-team match. To eliminate the impact of previous injury on subsequent injury data, each player was used only once during the study period. If a player met the inclusion criteria on more than one occasion, the data from the first absence were used in analysis.

4.2.2.4 Quantification of training load

Injured players had their RT data recorded for every session immediately following their injury until they were again able to fully participate in squad training. Within 4 weeks of returning to squad training, players had their SMT data recorded for a period similar to that of the injury. All training for both RT and SMT were recorded and included in the analysis regardless of whether the session was focused on soccer-specific training or other forms of RT (e.g. swimming and weight training).

Baseline HR data were established for all players at the beginning of the study period (Polar team system, Kempele, Finland). Resting heart rate (HR_{rest}) was taken as the mean of the lowest 15-s continuous period of heart rate with players relaxing in a semi-recumbent position in a quiet room for a period of 15 min. Following a familiarisation session, maximum heart rate (HR_{max}) was determined using an incremental treadmill protocol (Helgerud *et al.*, 2001). For all training sessions, heart rate (Sampled at 5-s) were recorded using the team polar system (Polar team system, Kempele, Finland). The total duration (min) of activity was also calculated using data extrapolated from the associated software.

It was considered vital that the overall physiological load associated with SMT was comprehensive. Competitive matches were therefore also included in the training load for SMT. It was not however, possible to monitor HR for first team matches due to the regulatory constraints of the competitive league in which the players played. The demands of match-

play for players returning from injury were therefore estimated using HR values collected during competitive league and cup matches played for the squad's reserve team. This information was used as an estimation of the physiological strain and intensity of effort during all competitive matches (first team or reserves) that the player participated in during their observation period. In order that the impact of playing competitive matches on the training load could be evaluated separately, match data was also extracted from SMT to give data related only to squad training (ST).

Training impulses (Banister *et al.*, 1975) and, SHRS (Flanagan and Merrick, 2002) were calculated from the data collected for all training sessions and matches. Heart rate data from each session was used to calculate average heart rate (HR_{ave}) and average $\%HR_{max}$ for the session. To enable periods of high cardiovascular load to be identified, a 'high-intensity heart rate zone' of 80% to 100% of HR_{max} was also calculated from the heart rate data.

After the conclusion of each training session and competitive match, the players rated the entire training session's subjective intensity using a modification of the Borg CR10-scale (Foster *et al.*, 1995). Approximately 30 minutes after each training session and match they were shown a printout of the scale (appendix ii) and asked "How was your workout?". This was to ensure that the perceived effort referred to the whole session and not the terminal session intensity (Foster *et al.*, 2001). A session-RPE was obtained by multiplying the RPE by the total duration of the training session in minutes (Foster *et al.*, 1995).

4.2.2.5 Mode of rehabilitation

To enable second aim of the investigation to be completed, the type of training undertaken during rehabilitation was also categorised and quantified. In order that an indication of the type of training undertaken during rehabilitation could be reported, modes of exercise were categorised (Table 4.2.1.). These pre-determined categories were merely intended to give an indication of the logical progression through rehabilitation back to full contact training and also categorise any supplementary training that may be performed. Following every session, the club physiotherapist and the player(s) involved in the session discussed which category best suited the session and this was recorded. At the current club, no organised supplementary squad training took place such as weight circuits or water-based activity. All normal squad training was therefore full function soccer-specific activity similar to category 7 in Table 4.2.1.

Table 4.2.1. Categories of rehabilitation training sessions.

Category	Activity description
1	Non-weight bearing, gym CV work performed. Cycling, swimming etc.
2	A gym based weight training session. May include arm and/or leg resistance work.
3	A gym session that combines arm and leg work and also includes CV work, boxing etc.
4	Light running work, straight line jogging performed at approx <40% pace.
5	Extended aerobic pace work (Approx 40-80% pace) and basic directional work performed.
6	Straight line sprint work. Testing but sub-maximal agility and ball work. No contact between players.
7	Full function work, up to max soccer specific random agility drills, ball work with contact, tackling etc.

To enable further analysis, the RT categories were also grouped to reflect the specificity of the training. These groups were non soccer -specific rehabilitation (NSR) (Categories 1,2,3,4) and a soccer-specific running-based group of categories (SSR) (Categories 5,6,7).

Statistics

The statistical procedures were completed using SPSS Version 14 for Windows (Chicago, Illinois, USA). Descriptive statistics, mean ± standard deviation (SD) were calculated for all variables. Before using parametric tests, the assumption of normality was verified using the Kolmogorov-Smirnov test. Differences between RT and SMT in session frequency and duration, $\%HR_{max}$, percentage time spent in selected heart rate zones, SHRS, Trimps, RPE and SRPE were analysed using paired t-tests. An independent samples t-test one-way was used to investigate whether differences existed between ST and the RT categories SSR and NSR with the dependant variables being session duration, $\%HR_{max}$, SHRS, Trimps, RPE and SRPE. Where appropriate, effect sizes ($\acute{\eta}^2$) were calculated and values of 0.01, 0.06 and > 0.15 were considered small, medium and large respectively (Cohen, 1988). Statistical significance was accepted as $p < 0.05$ unless stated otherwise.

4.2.3 Results

4.2.3.1 Session details

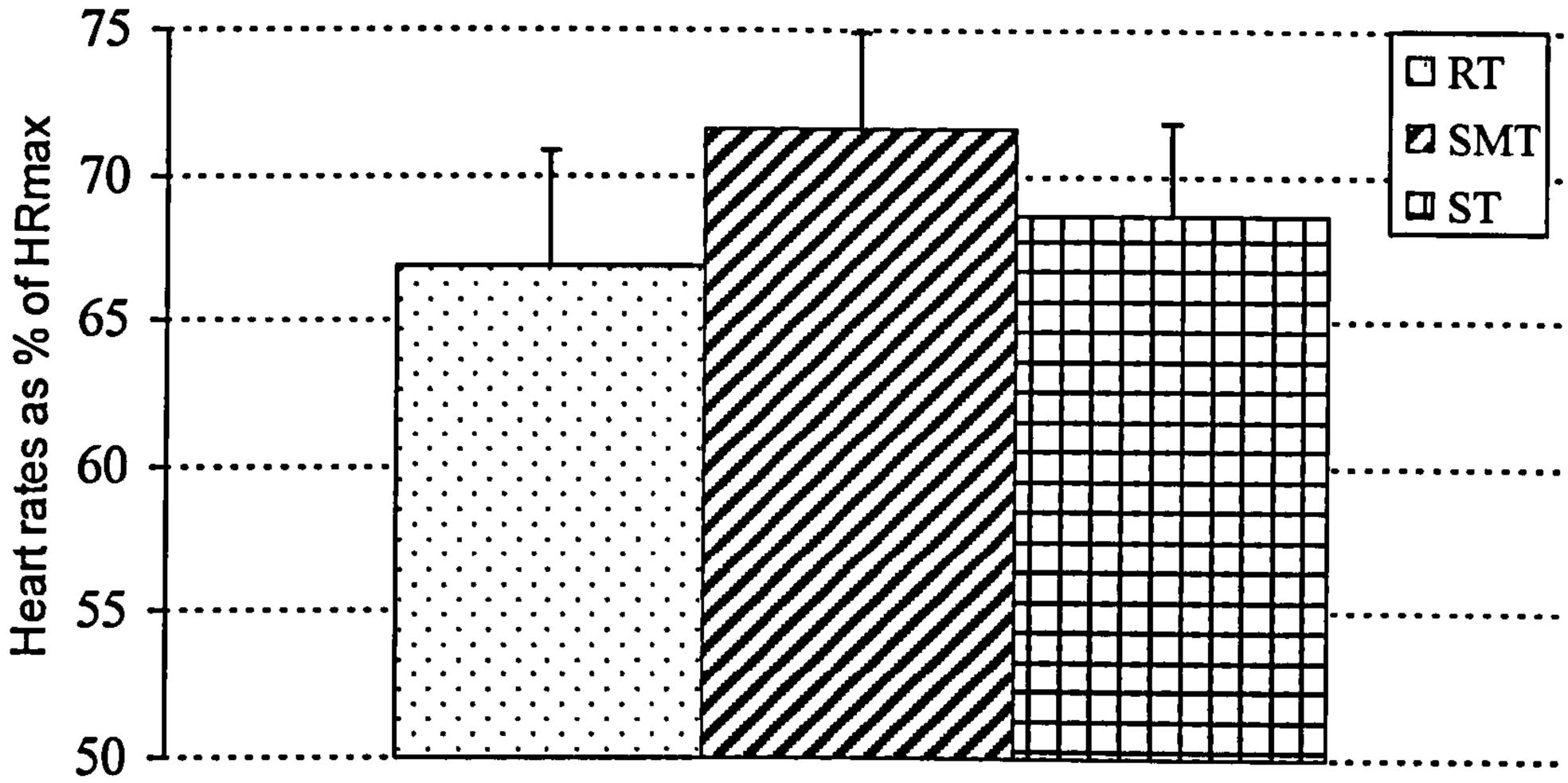
The mean ± SD for observation periods and total training days of RT and SMT are shown in Table 4.2.2. A typical squad training week consisted of 4 ± 1 training days; 2 ± 1 competitive matches and 2 ± 1 d off. A typical rehabilitation week consisted of 5 ± 1 training sessions per week and 2 ± 1 d off. The average number of rehabilitation sessions per day (0.8 ± 0.05) was greater than the number of sessions completed in SMT (0.7 ± 0.04) ($t(19) = -7.2$, $p<0.001$. Effect size $\acute{\eta}^2=2.7$). There was no difference in session duration between RT (96 ± 7 min) and SMT (96 ± 8 min) ($p>0.05$). There were no differences in observation periods, hours of exposure or duration of sessions ($p>0.05$) between SMT and RT.

Table 4.2.2. Details of observation periods for RT and SMT (mean ± SD per injury, n = 20)

	RT	SMT
Total observation days recorded	465 (23 ± 10)	430 (22 ± 5)
Total number of sessions recorded	381 (19 ± 8)	307 (15 ± 4)
Total training hours recorded	613 (31 ± 13)	491 (25 ± 6)
Total match exposure (h)		103 (5.2 ± 1.5)
Mean duration of sessions (min)	96 ± 7	96 ± 8

4.2.3.2 Session intensity

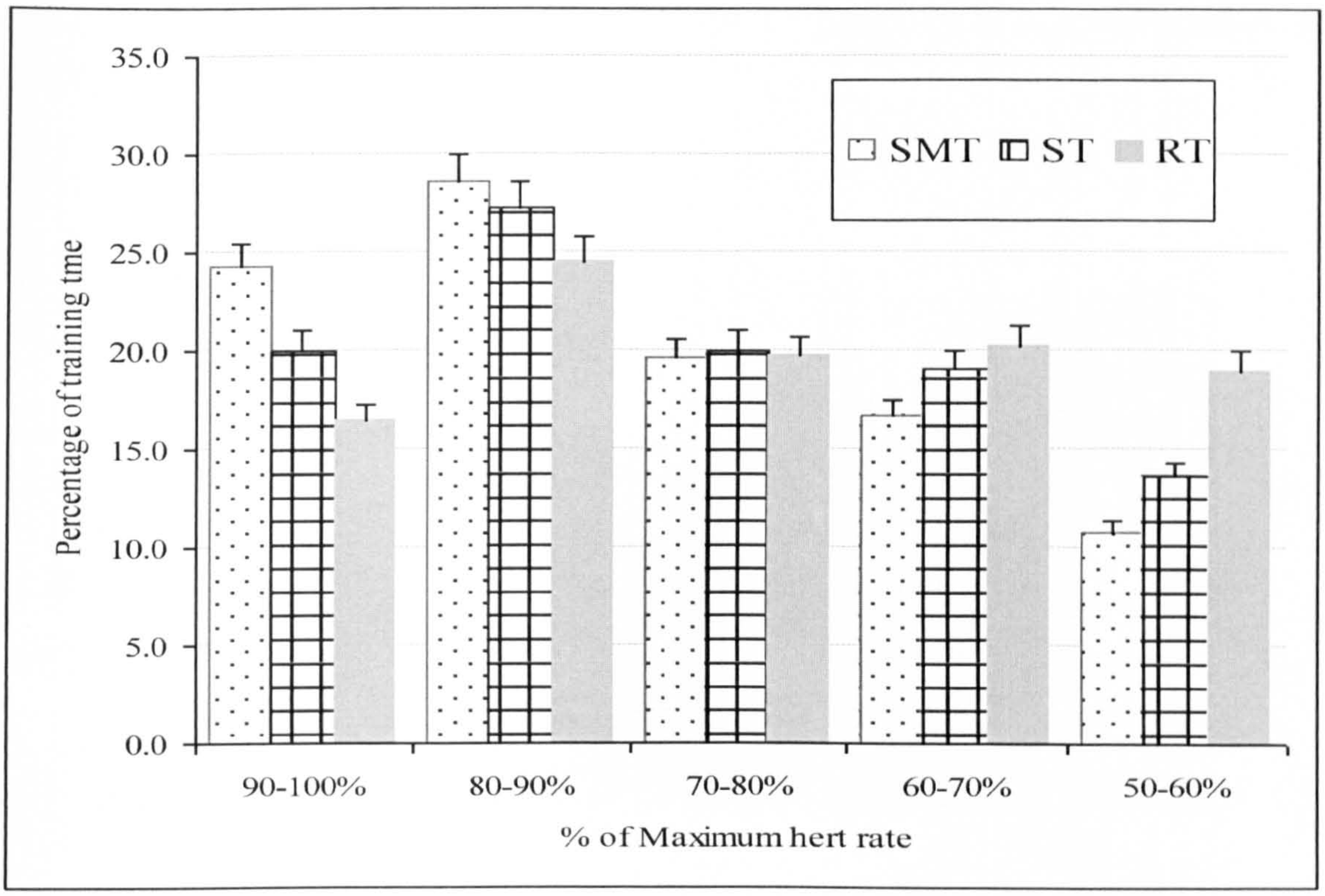
The intensity of training sessions were analysed using heart rate data. The $\%HR_{max}$ (72 ± 3% HR_{max}) and HR_{ave} (135 ± 6 beats·min^{-1}) for SMT were greater than RT (67 ± 4% HR_{max}) ($t(19) = 5.1$, $p<0.001$, effect size $\acute{\eta}^2=0.63$). The $\%HR_{max}$ for SMT was also greater than ST (mean 69 ± 3% HR_{max}) ($t(19) = 16.7$, $p<0.001$, effect size $\acute{\eta}^2=0.93$). The $\%HR_{max}$ during RT elicited similar $\%HR_{max}$ to that observed during ST ($p>0.05$) (Figure 4.2.1.).



*Significant difference between RT mean $\%HR_{max}$ and SMT, t (19) =5.1, $p<0.01$, effect size $\acute{\eta}^2=0.63$.

Figure 4.2.1. Percentage of maximum heart rates for RT, SMT and ST (mean ± SD).

Figure 4.2.2 shows the percentage of time spent in each HR zone for RT, SMT and ST. Time in the high-intensity zone (80-100% HR_{max}), was lower for RT (41 ± 7% HR_{max}) than both SMT (53 ± 7% HR_{max}, t (19) = -5.4, $p<0.01$, effect size $\acute{\eta}^2=0.61$) and ST (47 ± 8% HR_{max}, t (19) = -2.9, $p<0.01$, effect size $\acute{\eta}^2=0.31$). Computations for Trimps and SHRS (Table 4.2.3) also demonstrated greater values for SMT than RT ($p<0.05$). No differences for Trimps were however, found between RT (283 ± 32) and ST (293 ± 40) ($p>0.05$).



* Percentage time spent in the 90-100% HRmax zone for RT was significantly lower than both ST, t (19) = 2.3, $p<0.001$, effect size $\eta^2=0.36$ and SMT, t (19) = 4.6, $p<0.01$, effect size $\eta^2=0.31$. # Percentage time spent in the 90-100% HRmax zone for ST was significantly lower than SMT, t (19) = 6.1, $p<0.01$, effect size $\eta^2=0.5$. $ Percentage time spent in the 80-90% HRmax zone for RT was significantly lower than SMT, t (19) = 0.2, $p<0.01$, effect size $\eta^2=0.36$

Figure 4.2.2. Percentage of time spent in each heart rate zone for RT, ST and SMT.

Table 4.2.3. Mean ± SD of calculated training loads for RT, SMT, ST.

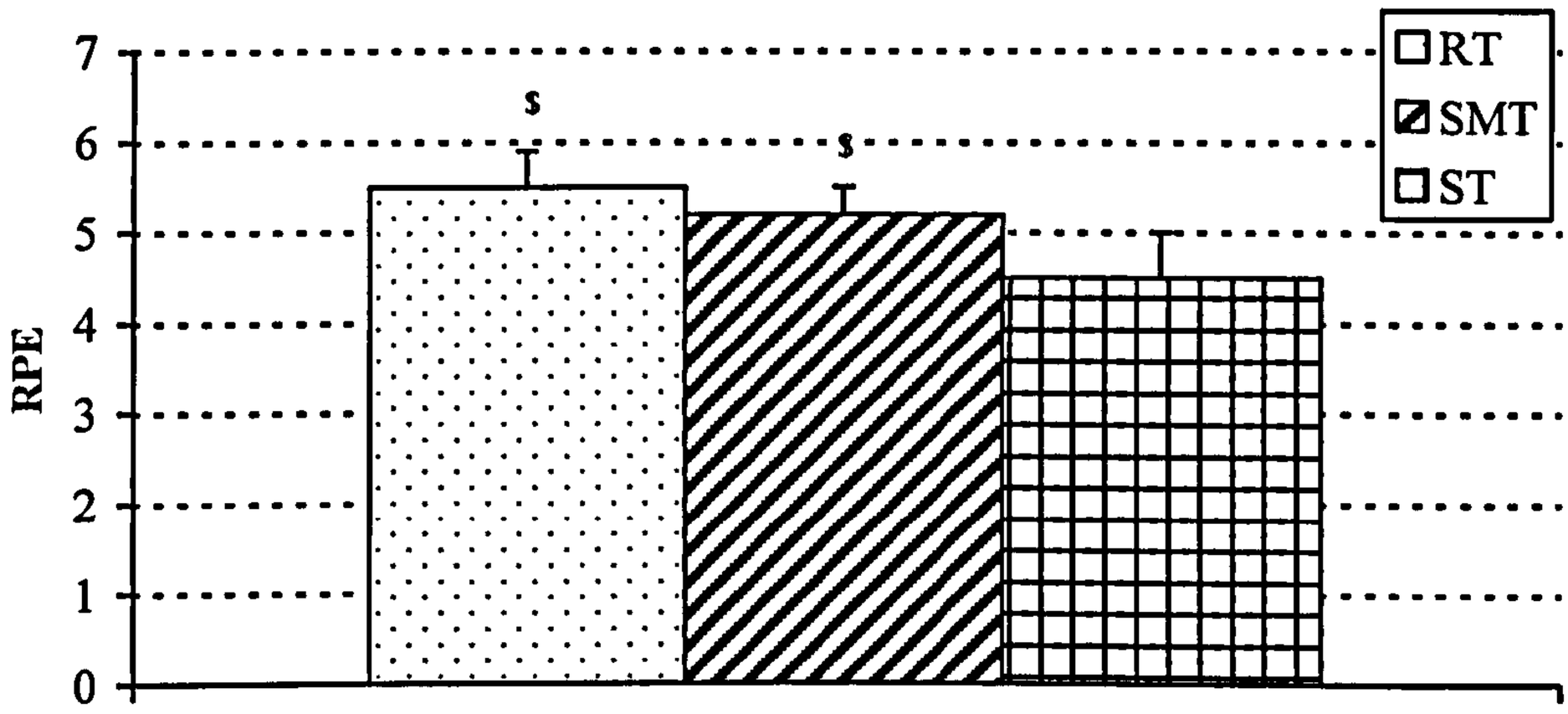
			Mean	SD	Std. Error Mean
RT	n=381	Trimps	87*	19	4
		SRPE	540■	64	14
		SHRS	283▲	37	8
SMT	n=307	Trimps	134*	36	8
		SPRE	518	52	12
		SHRS	319▲	35	8
ST	n=248	Trimps	92	24	5
		SRPE	428■	74	17
		SHRS	293	40	9

* Significant difference P<0.001, $\eta^2=15.9$; ■ Significant difference $p<0.001$, $\eta^2=2.0$; ▲significant difference $p<0.005$, $\eta^2=15.9$.

4.2.3.3 Ratings of perceived exertion

Mean RPE for RT (6 ± 2) was greater than both SMT (5 ± 2) and ST (5 ± 1) ($p<0.05$) (Figure 4.3.3). Incorporating the duration of the session in order to calculate SRPE resulted in no difference in SRPE between SMT and RT (p>0.05). SRPE was however, significantly greater

during RT (mean 540 ± 64) than ST (mean 428 ± 74) (t(19)= -6.2, p<0.01, effect size $\acute{\eta}^2$=0.7).



$ significant difference between mean RPE during RT and SMT, t (19)=-2.9, p<0.05, effect size $\acute{\eta}^2$=0.3. • significant difference between mean RPE during RT and ST, t(19)=-8.3, p<0.01, effect size $\acute{\eta}^2$=0.78)

Figure 4.2.3. RPE for RT, SMT and ST (mean ± SD).

4.2.3.4 Soccer-specific and non soccer-specific rehabilitation

Table 4.2.3. details the frequency of data collected for each rehabilitation category. The number of NSR sessions was 232 and the number of SSR sessions 145. When investigating differences between SSR and NSR sessions during RT, it was observed that there were no significant differences in session duration (95 ± 34 vs. 97 ± 31 min).

No differences were observed between NSR and SSR for RPE (6 ± 2 vs. 5 ± 2) or SRPE (544 ± 259 vs. 530 ± 282) (p>0.05) . The mean %HR_{max} during SSR (71 ± 9% HR_{max}) was, however, greater than that for NSR (mean 62 ± 10% HR_{max}; t(375)=78, p<0.01, effect size $\acute{\eta}^2$=0.12). Significantly higher SHRS values were also observed during SSR (mean 311 ± 107 vs. 258 ± 106; t(375)= 23, p<0.01, effect size $\acute{\eta}^2$=0.06). This same pattern was also observed for Trimps (120 ± 96 vs. 67 ± 45; t(375)=48, p<0.01, effect size $\acute{\eta}^2$=0.11).

Table 4.2.4. Frequency, duration, $\%HR_{max}$ and RPE of each rehabilitation category recorded

	Rehabilitation categories	Sessions (n)	Percent	Mean duration (min)	$\%HR_{max}$	RPE
1	Non-weight bearing, gym CV work performed. Cycling, swimming etc.	69	18.3	91±33	67±10	4.7±1.2
2	A predominantly based weight training gym session. May included upperbody and/or leg resistance training.	49	13.0	95±36	55±10	6.4±1.7
3	A gym session that combines arm and leg work and may also include CV work, boxing etc.	72	19.1	103±33	62±8	6.3±1.5
4	Light running work, straight line jogging performed, approx <40% pace.	42	11.1	89±27	67±9	4.9±1.4
5	Extended aerobic pace work (Approx 40-80% pace) and basic directional work performed.	58	15.4	95±33	73±8	6±1.6
6	Straight line sprint work. Testing but sub-maximal agility and ball work. No contact between players.	42	11.1	100±39	74±8	6.2±1.8
7	Full function work, up to max soccer specific random agility drills, ball work with contact, tackling etc.	45	11.9	97±21	70±8	5.1±1.3
	Total	377	100.0	96±32	67±10	5.5±1.6

4.2.4. Discussion

The main findings of the current study indicated that injured players trained more frequently per day than when they were not injured. Rehabilitation programmes did, however, lack the cardiovascular intensity of normal soccer training. Individual player subjective ratings of effort during RT were significantly greater than those observed for both SMT and ST indicating that players perceived rehabilitation training to be more demanding than normal squad training despite the lower levels of cardiovascular strain. The nature of the exercise during rehabilitation training was therefore hypothesised to be an important factor in these findings. When RT was divided into SSR and NSR our data demonstrated that the physiological stress, as indicated by all heart rate variables, was significantly greater during SSR than NSR. Taken together, these findings suggest that reductions in physiological load during RT are probably responsible for the reductions in fitness that are demonstrated by players following injury. This may be due to the injured players' inability to perform SSR. The data also suggests that care should be taken when using RPE as an indicator of training load when the exercise programme is novel.

In the current study, all heart rate variables for SMT were significantly greater than those found for RT. This is probably due to the disabling effects of the various pathologies encountered as a consequence of injury making training at a high intensity difficult. Training intensity is the major parameter influencing the effects of training and cardiorespiratory fitness (Wilmore, 1988; Fleck, 1994; Helgerud *et al.*, 2001) so it is logical to assume that this decrease in intensity may lead to reductions in important physiological performance parameters for soccer such as aerobic power. This may suggest that it is important to try and increase the cardiovascular intensity of RT sessions within the functional limitations of the current status of injury. Such increases in intensity may provide better preparation for players to tolerate the increase in exercise intensity associated with normal squad training on their return to fitness. This may also have the additional positive benefit of reducing the risk of re-injury.

A significant functional difference between injured and uninjured players is the ability to play competitive matches. As the physiological demands of match-play are an important component of the overall training load of a soccer player (Mohr *et al.*, 2003) it seemed pertinent to evaluate this specific aspect of a players training load in isolation. In the current study, the intensity of match play, utilising reserve team match data, was significantly greater than RT and ST in all variables used to quantify training load. These findings concur with Rohde and Espersen (1988), who compared HR of training and matches. They found that time in a high-intensity zone (HR_{max} – 15 beats·min^{-1}) during training (4%) was significantly less that during matches (26%). The actual intensity of training may have been even lower as HR measurements were taken on the same day each week. This day was reported to be the most intense training day of five. Playing competitive matches is therefore, a considerable component of the training load in this sample of players when they are uninjured. These differences may also represent an underestimation of the actual importance of the contribution of match-play to the overall training load as the use of reserve team match data is most likely lower than the true physiological stress associated with first team matches (Drust *et al.*, 1998). The current data (mean match HR 160 ± 8 beats·min^{-1}) does however, compare well with other published data for first team games (Bangsbo, 1994b) suggesting that the cardiovascular stress observed in the games in our sample was similar to that observed at a higher level of play. These differences in physiological stress may have important implications for RT as it would appear that the physiological stresses associated with match-play are so high that replicating these demands during rehabilitation is not realistic given the restrictions of an individual's functional ability. It should be noted that these findings may also impact on those fit players who are not playing regular competitive soccer. The lack of stimulus through match-play may therefore result in detraining and leave the player with the same problems encountered by the injured player on their return to competition.

As matching the demands of match-play may not be possible during RT, a more practical aim may be to maintain a level of intensity similar to that of normal squad training. Although the highest levels of intensity are observed during competitive match-play, the majority of a soccer player's total physical activity load, when fit, involves soccer-

specific activities aimed at improving the technical, tactical and physical ability of players and the preparation for forthcoming games (Bangsbo, 1994b). A comparison between RT and ST will therefore provide a valid evaluation of the demands of RT and give an indication of the highest physiological stress that RT may be able to simulate. Although average %HR_{max} for RT (67%) and ST (69%) were very similar, the percentage time spent in the high-intensity HR zone (80-100% HR_{max}) during RT was significantly lower than during ST. These findings would suggest that the current approach to RT did not provide the high-intensity exercise stimulus associated with ST. Further comparisons between components of RT and ST may identify specific elements of RT that lack the cardiovascular intensity associated with either SMT or ST. The RT incorporated in this study was characterised by a wide range of exercises and intensities. A more detailed comparison between components of RT and ST may help identify specific elements of RT that lack the cardiovascular intensity associated with ST.

The current investigation attempted to examine this question by splitting the activities completed in RT into two categories; SSR and NSR. There were no differences in the total number of sessions that were categorised as SSR and NSR during RT (SSR=187; NSR=194 sessions). This would suggest that the approach to rehabilitation included an equal proportion of both general RT and functionally specific RT. It was however, evident from the data that the cardiovascular demands of SSR were significantly greater than those associated with NSR. This is probably a result of the predominant form of exercise in SSR being running focused as opposed to the non-ambulatory modes of exercise that dominate NSR. It is well recognised that HR responses from running are significantly greater than non-locomotion forms of exercise such as static cycle ergometers and rowers (McArdle *et al.*, 1973).

The current definition of SSR is not widely recognised in the literature. As such it's accuracy as a definition may be questioned. Data from our investigation does seem to suggest that the distinction made here between NSR and SSR is appropriate and as a consequence supports the use of our current definition. No significant differences in any heart rate derived variables (% HR_{max}, Trimps, SHRS) were observed for SSR when

compared with ST ($p>0.05$). This would suggest that the definition of SSR provides a valid representation of the physiological demands of training undertaken during normal squad training. This would indicate that our data provides a suitable comparison between different forms of RT in our current population.

The specific stage of rehabilitation at which SSR and NSR training was undertaken in the RT programme was not analysed. Logical considerations with regard to the effects of the various pathologies on the reduction of the physical capabilities of injured players would suggest, however, that the majority of NSR sessions occurred early on in the RT programme. Early RT sessions, which are normally characterised by non- or partial-ambulatory exercises based around cross trainers, cycle ergometers, rowing machines, water-based activities or very light jogging sessions were, however, found to be lacking in cardiovascular intensity in the current study. Physiological adaptations resulting in performance decrements may therefore occur during these earlier stages of rehabilitation. Modes of exercise that elicit high $\%HR_{max}$, and are suitable for use during early rehabilitation, are therefore required to limit these reductions in fitness found in previous studies (Chapter 4.1).

In the current study, RPE was found to be significantly greater during RT than both ST and SMT. Rating of perceived exertion has been widely and successfully used to monitor training intensity in resistance training (Day *et al.*, 2004) and intermittent type sports (Impellizzeri *et al.*, 2004). Investigations are, however, normally carried out using athletes participating in their habitual activity. No literature can be found which compares the use of RPE during habitual and non-habitual activity. Rehabilitation training involves substantial amounts of NSR which won't be habitual for soccer players. For example, activities such as weight training and water-based activities are infrequently completed by players during their normal activities (Bangsbo, 1994b). This is in direct contrast to the modes of exercise during SSR which are regularly completed by fit players. This would suggest that players may become habituated to the sensation of cardiovascular stress in SSR, but are not so familiar with the more localised perceptions of effort associated with unfamiliar RT activities. It may therefore be possible that RPE during RT

reflects an artificially high perception of effort due to a large proportion of the training modes being unfamiliar. Ratings of perceived exertion may therefore not be a suitable method of quantifying rehabilitation training because of the range of unfamiliar modalities utilised. This may be especially applicable during the early phases of rehabilitation when more non-familiar exercise modes are undertaken.

It was recognised that rehabilitation training would differ significantly between clubs and that this may have an impact when analysing the results of this study. With this in mind, 96 questionnaires (Appendix iii) were sent to ex players who were asked "On a scale of 1-10 please compare the difficulty of rehab training at ******** with your other clubs. A score of 5 to 6 indicates that it was, on average, the same intensity as other clubs. A score from 1-4 indicates that it was generally easier than other clubs and a score of 7-10 indicates it was more difficult". The number of seasons at the club and the number of other clubs the player had been injured at were also recorded.

Out of the 96 questionnaires sent out to ex-players, 58 (57%) responded (Table 4.2.3.). Of the 58, only 3 (5%) ex-players gave a mark of <5 (scores of 1-4 indicated that rehabilitation training at this club was easier than experienced at other clubs).

Table 4.2.5. Questionnaire returns answering question "How difficult is rehab at ****"

Returns	Other clubs played at		Intensity score	
N	Total	Average	Average	SD
96	150	1.4	7.4	1.3

As previously discussed, session intensity is a major contributory factor to the magnitude of physiological adaptations following injury. It may be that one reason for the findings of this study was that the lack of cardiovascular intensity during RT at the current club was significantly less than at other similar professional soccer clubs. The questionnaire response from ex-players, however, would indicate that this is not the case. It is recognised that recall bias may affect retrospective responses but the mean intensity score of 7.4 ± 1.3 would indicate that the rehabilitation at the current club is significantly more

difficult than at other clubs. Although no inference is made to suggest that this is the case, the data would indicate that RT at the current club is at least similar to that at other clubs. Players at other professional soccer clubs may therefore be experiencing similar effects of detraining to those at the current club.

4.2.5 Conclusion

In the current study it was found that, although players perceived rehabilitation training to be more demanding, it lacked the cardiovascular intensity of normal squad training and match play. The mode of training during rehabilitation has a significant affect on the cardiovascular demands of normal soccer participation as non-soccer specific activities do not lead to the same increases in HR as soccer training or match-play. It may be difficult to recreate the intensity associated with soccer-specific exercise early in the rehabilitation programme though methods that attempt to increase the cardiovascular intensity of early rehabilitation programmes may be suitable interventions in reducing the effects of injury.

CHAPTER 5

Preventing performance decrements following injury in professional soccer.

5.1 Study 6: Comparing the effects of constant steady-pace and variable intermittent exercise protocols in maintaining aerobic power and repeated sprint ability in injured elite soccer players.

The previous chapter found that, following injury, player's aerobic power and repeated sprint ability was significantly affected by the changes in habitual training patterns. It was suggested that these changes could be the result of the lower intensity of exercise performed during the early phase of rehabilitation. This chapter investigates methods of increasing the training load during early rehabilitation whilst exercising within the constraints of injury.

Study 6 COMPARING THE EFFECTS OF CONSTANT STEADY-RATE AND INTERMITTENT EXERCISE PROTOCOLS IN MAINTAINING AEROBIC POWER AND REPEATED SPRINT ABILITY IN INJURED ELITE SOCCER PLAYERS.

5.1 Introduction

Chapter 4.1 showed that elite soccer players who were absent from training for >14 d due to injury demonstrated a significant reduction in estimated $\dot{V}O_{2max}$ and significant increase in fatigue index during an RSA protocol. High aerobic power (Matkovic *et al.*, 1993; Wisløff *et al.*, 1998; Helgerud *et al.*, 2001) and the ability to recover quickly between repeated bouts of high-intensity intermittent exercise (HIIE) (Nagahama *et al.*, 1993; Wragg *et al.*, 2000; Cometti *et al.*, 2001; Mohr *et al.*, 2003) are considered to be important characteristics of elite soccer players. These performance decrements may therefore have a significant effect on a player's ability to perform, at the levels observed prior to injury, on their immediate return to competitive play. This also may negatively impact on the success of the team.

Chapter 4.2 suggested that the inability to exercise at high-intensity during early rehabilitation may be the major reason why players detrain following injury. Early rehabilitation is frequently associated with the player being unable to perform soccer-specific activities such as running. This rehabilitation period is therefore characterised by training using non- or partial-weight bearing exercise machines such as static cycle ergometers, rowing machines and cross-trainers. The intensity of such exercise may not replicate that associated with normal soccer training. This may result in negative performance changes. It is therefore possible that increasing cardiovascular demand during these early periods of rehabilitation may have a positive effect on maintaining aerobic power and RSA and hence reduce the decrements in performance observed following injury.

High-intensity intermittent training (HIIT) improves $\dot{V}O_{2max}$ (Tabata *et al.*, 1996; Rodas *et al.*, 2000; Helgerud *et al.*, 2001; Edge *et al.*, 2005) and repeated sprint ability (Dupont *et al.*, 2004; McMillan *et al.*, 2005). These type of exercise models could therefore provided a potential means to reduce decrements in performance if utilised during rehabilitation. The performance of short, maximal-effort bouts of exercise during early rehabilitation may not however, be feasible due to the specific limitations of the various pathologies incurred by players. Training at a moderate-intensity (60-70% $\dot{V}O_{2max}$) can also improve aerobic capacity and maximal mitochondrial enzyme activities (Hickson *et al.*, 1982; Holloszy, 1984; Gollnick *et al.*, 1984; Tabata *et al.*, 1996; Edge *et al.*, 2005). The effects of moderate-intensity constant steady-rate exercise have also been investigated on RSA (Tabata *et al.*, 1996; Edge *et al.*, 2005). The available data from these studies would seem to suggest that moderate-intensity training can also induce improvements in both $\dot{V}O_{2max}$ and RSA despite reductions in the overall exercise intensity. These results should however, be incorporated with caution as the subjects in each of these studies were sedentary or recreational athletes. Moderate-intensity exercise may not provide a sufficient stimulus to elicit similar in elite athletes populations. These ideas can be supported in Chapter 4 which reported performance decrements in $\dot{V}O_{2max}$ and RSA despite such moderate-intensity steady-rate training being performed during rehabilitation.

Intermittent exercise may provide an alternative solution to the prescription of moderate-intensity constant steady-rate exercise. This type of exercise includes periods of exercise of two or more different intensities that are performed alternately (Christensen, 1960) and, as such, provides an opportunity to deliver a short period of intense exercise that is followed by a recovery period. As a consequence, intermittent exercise appears to elicit a different physiological response than constant-pace exercise performed at the same average intensity. Intermittent training impacts associated physiological markers of aerobic performance such as blood lactate thresholds and can improve endurance performance (Edwards *et al.*, 1973; Esfarjani and Laursen, 2007). Drust and colleagues (2000) compared a soccer-specific intermittent exercise bout with continuous exercise of

the same average workload. Average heart rate for the intermittent protocol was higher than the continuous protocol (168±10 and 162±1 beats·min^{-1} respectively). Intermittent exercise (15 s exercise, 15 s rest) has also been shown to result in considerable fluctuations in ATP and CP levels, whereas only minor changes were observed during work-matched continuous exercise (Essén, *et al.*, 1977). These data suggest that intermittent exercise elicits a higher heart rate, higher lactate levels and increased utilisation of ATP and CP than work loads matched to continuous exercise.

These physiological changes associated with intermittent exercise provide a basis for its application as a possible rehabilitation intervention. Intermittent training at sub-maximal exercise intensities has not however, been as widely researched as HIIT. Chesley *et al.* (1996) reported that daily sessions of moderate-intensity, intermittent training (MIIT) (60-70% $\dot{V}O_{2max}$, 2 h·d^{-1}) for only 6 to 10 d improved aerobic capacity. A similar training protocol also resulted in an increase in mitochondrial enzyme activities and reduced blood lactate levels at the same absolute work rate (Spina *et al.*, 1996). These data may indicate that intermittent exercise may therefore be a more potent mode of exercise intervention than continuous exercise. This may therefore lead to intermittent exercise being a more effective method of reducing the effects of detraining associated with injury. No studies can be found that has investigated whether a sub-maximal intermittent training protocol is more effective than a work-matched constant steady-rate protocol at maintaining fitness and therefore reducing the impact of injury on performance decrements in aerobic power and RSA.

The aim of this study was therefore to compare the effects of intermittent and constant steady-rate training protocols, matched for total work and time, during early stage rehabilitation, on elite soccer player's estimated $\dot{V}O_{2max}$ and RSA performance.

5.2 Methods

The current study investigated the effects of intermittent training during early rehabilitation on injured professional soccer players. Aerobic power and RSA were tested

on return to normal activity in any player who sustained an injury resulting in an absence of >14 d. These results were compared with previous test results recorded when players were deemed fit prior to injury. The impact of different rehabilitation strategies was determined by comparing two separate interventions carried out during early rehabilitation. One intervention was characterised by intermittent exercise. The other utilised constant steady-rate training.

All professional players age >17 at an English League One professional soccer club during the competitive phases of the period from August 2007 to December 2008, agreed to participate in this study (Table 4.2.1). This period consisted of the whole of the 2007-08 season and the first half of the 2008-09 season. Attendance, absence and injury data were recorded using methods detailed in Chapter 3.1.

5.2.1 Baseline testing

All players underwent baseline testing when training and playing competitive soccer to determine anthropometric data (Height, body mass, body fat %), estimated $\dot{V}O_{2max}$ and RSA. Players were tested within 2 weeks following the beginning of each competitive season and then periodically during each season. These in-season testing sessions were not timetabled but each player was tested on at least one further occasion at the mid-point of the 2007-08 season. A player's most up-to-date testing data were used in the event of that player sustaining an injury. This was termed 'test 1' (T1).

Players completed all testing sessions on days following rest days from competition and training. The time of day for T1 was recorded for all players. Any further testing took place at approximately (±1 h) the same time of day. Players were tested >3 hours postprandial and asked to refrain from consuming any energy drink or drink containing caffeine on the day of the test. Players were asked about their health and any player reporting illness was tested when asymptomatic.

5.2.1.1 Anthropometry

Subject's height (Seca 220 Stadiometer, Birmingham, England) and body mass (Seca ά 770 Digital Low Form scale, Birmingham, England) were taken and recorded. Body fat percentage using Harpenden skinfold callipers (Quinton Instruments, Seattle) were calculated from the sum of 4 skinfolds (triceps, biceps, sub-scapula and supra-iliac), as described by Durnin and Womersley (1974).

5.2.1.2 Estimated $\dot{V}O_{2max}$

All players completed the multistage fitness test (Léger and Lambert, 1982) to provide an indicator of aerobic fitness. This test has been validated as a predictor of $\dot{V}O_{2max}$ by Léger *et al.*, (1988) and by Ramsbottom *et al.* (1988). Details of the test protocol can be found in Chapter 4.1. The reliability of this test was found to be good (ICC, r = 0.94, $p<0.05$, TE, 0.37 $ml \cdot kg^{-1} \cdot min^{-1}$, 95% CI 0.21 – 0.39 $ml \cdot kg^{-1} \cdot min^{-1}$) (see Chapter 4.1 for details). The SMC calculated between the two tests was ± 1.03 $ml \cdot kg^{-1} \cdot min^{-1}$.

5.2.1.3 Repeated sprint test

All players completed a repeated sprint test. Briefly, this consisted of 6 x 40 m sprints. Each sprint departed every 30 s and were initiated from a 4 s rolling start.

5.2.1.3.1 Protocol design

Evaluation of the RSA test protocol in the discussion of Chapter 4.1 suggested that, despite verbal encouragement, the short sprint distance and 10 s turn–around between sprints may have encouraged some players to decelerate prematurely. For those subjects who did manage to maintain maximal velocity through the finish gates, the effort required to decelerate quickly and get back to the start line within 10 s may have had a significant effect of subsequent sprint times (Lakomy and Haydon, 2004). These factors would suggest that the RSA protocol previously employed in this study was not suitable for this investigation. An experimental RSA protocol which incorporated components of repeated sprint ability that were more specific to soccer and alleviated the problems highlighted above was therefore required.

When considering each component of the RSA test protocol, the demands of competitive match play were taken into account. Elite soccer players spend 3 times more time walking in a match than standing still (Kirkendale, 2000; Bloomfield *et al.*, 2007). In their time–motion analysis study, Spencer *et al.* (2004) observed that 95% of the recovery between sprints within a repeated sprint bout was active, consisting primarily of jogging. Passive recovery may therefore not be truly representative of repeated sprint bouts during competitive matches (Rampinini *et al.*, 2007a; Impellizzeri *et al.*, 2008). It was decided that the recovery phase of the protocol should include walking at ~1 $m \cdot s^{-1}$. Bloomfield *et al.* (2007) observed that most sprints in soccer are initiated from a moving start and Svensson and Drust (2005) suggested that sprint tests that begin from a standing start may not be specific to soccer. A rolling start from a jogging speed (~2.5 $m \cdot s^{-1}$) was therefore selected for the protocol to increase test specificity. A single cycle of the RS protocol was therefore; walk at ~1 $m \cdot s^{-1}$ for 20 s, jog at ~2.5 $m \cdot s^{-1}$ for 10 m leading into an all–out 40 m sprint. This 30 s cycle was repeated 6 times to form the RSA protocol. Although this protocol is a simplification of the complex activity patterns noted in soccer, it represents useful progression in assessing RSA in elite soccer players.

5.2.1.3.2 Experimental protocol

This experimental set was used for all elements of RSA assessment incorporated in this study. This included the evaluation of the reliability of the protocol and all subsequent evaluation of a player's RSA. An indoor test area with a concrete floor was permanently marked with an oil paint marker to ensure consistency of equipment set up. Sprint times were measured to the nearest 0.01 s for each sprint using 3 sets of electronic photocells connected to an electronic timer (Polifemo, Radio photocell, Microgate, Balzano, Italy). In order to ensure consistent results, the photocells were placed 1.2 m above the ground. The timing gates were set up at 0 m, 20 m and 40 m (Figure 5.1.1). A line was marked 10 m outside the end of each timing gate. An area 8 m by 8 m was marked outside the 10 m line to restrict the distance that subjects could travel from the start line during the walk phase (Figure 5.1.1).

The protocol consisted of two specific elements. Subjects firstly completed a standardised 15 min warm up prior to the determination of their maximum sprint speed (MSS) over 40 m. During the warm up, the pace of the walk and jog were controlled in order to re–familiarise the subjects with the requirements of the protocol. All subjects then completed 2 x single maximal 40 m sprints from a jogging start following the warm-up. Maximum sprint speed was taken as the quickest of these 2 sprint times. Following a three minute rest during which the subjects were encouraged to walk and stretch, the RSA test took place. The MSS was used as a criterion score to assess potential pacing. Subjects were expected to obtain a performance for their quickest time for any sprint during the RSA test that was within 2% of their MSS time. If the criterion score was not achieved, the test was stopped immediately and subjects were required to repeat the RSA test with maximum effort after a rest period of at least 10 minutes. This occurred on 1 occasion during all testing sessions.

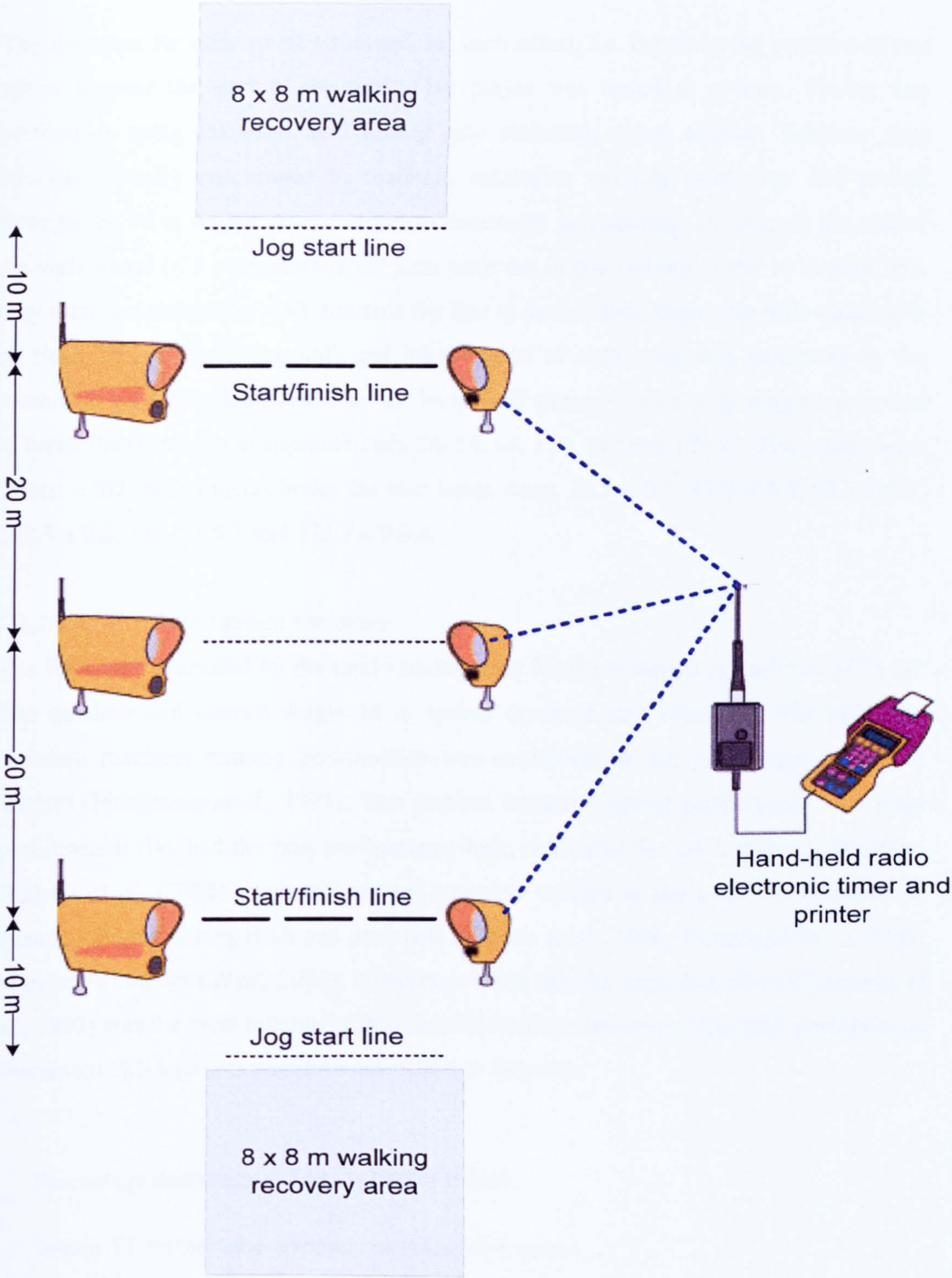


Figure 5.1.1 Schematic diagram of the setup for the RSA.

The direction for each sprint alternated for each effort, i.e. the finishing position of one sprint became the start of the next. One player was tested at a time. Timing was continuous using the hand held timing gate electronic timer display. Subjects were strongly verbally encouraged to maintain maximum velocity until they had passed through the 40 m timing gates and not to decelerate prematurely. If, towards the end of the walk phase (< 5 s remaining), subjects were not in the vicinity of the 10 m start line, they were encouraged to walk towards the line to ensure they began the following cycle on time. The pacing of the walk and jog elements of each cycle was facilitated by the researcher providing feedback from the hand–held timing device with subjects expected to break the first beam at approximately 24, 54, 84, 114, 144 and 174 s. The actual times (mean ± SD) that subjects broke the start beam were; 23.7 ± 0.3, 53.7 ± 0.3, 83.7 ± 0.3, 113.8 ± 0.2, 143.8 ± 0.3 and 173.7 ± 0.3 s.

5.2.1.3.3 Performance variables

The RSA was evaluated by the total running time for the 6 sprints in each test (TT) (s). The quickest and slowest single 40 m sprints (s) were also identified. The ability to maintain maximal running performance was evaluated by the percentage decrement method (Fitzsimons *et al.*, 1993). This method compares actual performance with ideal performance (i.e. had the best performance been replicated for all 6 sprints). Recently, Glaister *et al.* (2008) evaluated several formulae utilised in the available literature to quantify fatigue during RSA test protocols (Brooks *et al.*, 1990; Psotta and Bunc, 2005; Mendez–Villanueva *et al.*, 2008). It was concluded that the formula below (Fitzsimons *et al.*, 1993) was the most reliable of those available in the literature. The RSA performance decrement (RSA_{PD}) was therefore calculated as follows:

Percentage decrement = 100 - ((TT/IT) x 100).

Where TT = total time taken to complete all 6 sprints

Ideal time (IT) = quickest time x number of sprints (6).

Comparative differences between total sprint time (s) for the 0–20 m component (TT_{0-20}) and the 20–40 m component (TT_{20-40}) were also recorded. This allowed the researcher to investigate the specific phase of the sprints that was most affected if significant differences were observed in TT. In order to identify if fatigue during the first 20 m or second 20 m of each 40 m sprint were affected differently during the RSA protocol, the same formula was utilised to calculate RSA_{PD} for the first (RSA_{PD20})and second 20 m (RSA_{PD40}) of each run.

Heart rate was monitored continuously throughout the RSA test at 5–s intervals using short–range radio telemetry, (Polar Team System. Polar Electro Oy, Kempele, Finland). Ratings of perceived exertion, using a modification of the Borg CR10-scale (Foster et al., 1995), were taken immediately following the final sprint.

5.2.1.3.4 Protocol reliability

The ultimate objective of any performance test is to detect genuine changes in an athletes' capabilities. When monitoring an intervention, it is crucial these performance outcomes reflect changes in performance that are attributable to the intervention as opposed to random variation or systematic bias in the measurement. The test performance should yield consistent and reliable results that do not fluctuate by more than an acceptable degree from one test to another (McGawley and Bishop, 2006). The main reason for assessing the test–retest reliability of measures such as fastest sprint, total time and fatigue is therefore to determine the degree of within–subject variation in these measurements. High degrees of reliability improve the precision of single measurements and enhance the ability to monitor changes in performance as a result of experimental interventions (Glaister *et al.*, 2004).

Due to organisational constraints, it was not possible to investigate the reliability of the RSA protocol using the professional playing squad. Twelve healthy male elite apprentice soccer players were therefore used to evaluate the reliability of the RSA test protocol (age, 18 ± 1 years; height, 1.8 ± 0.1m; body mass 70 ± 5 kg; HR_{max} 191 ± 9 beats·min^{-1}; estimated $\dot{V}O_{2max}$ 58 ± 3 ml·kg^{-1}·min^{-1}, goalkeepers n=1, defenders n=5, midfielders

n=4, strikers n=2). Although the absence of any learning effects of this type of multi–sprint protocol has recently been reported (Glaister *et al.*, 2007), all subjects completed 2 familiarisation trials, 2 h apart, 2 d prior to the first testing session. Analysis of the 2 trials using paired student's t-test showed no significant differences between performance in any of the sprints or total sprint time ($p>0.01$). Intraclass correlation between the 2 trials was $r = 0.91$, ($p<0.01$) suggesting high correlation and an absence of systematic bias indicating no learning effect.

For testing the reliability of the protocol, all subjects performed the RSA test on three separate occasions. In order to test the reliability of the protocol *per se*, short-term reliability was investigated by the subjects performing the test twice in two sessions separated by two hours (RT1 and RT2). A 2 h recovery period was selected to ensure that there was no systematic change as a result of any fatigue induced by RT1. To test long–term reliability, all subjects performed a further test (RT3) 14 d following the first test. This duration was selected to reflect the minimum injury duration for study inclusion. A rest day proceeded the day of all testing sessions. Subjects were tested >3 hours postprandial and were verbally instructed to abstain from products containing caffeine or alcohol in the 24 hours prior to testing. Subjects were allowed to drink water *ad libitum* throughout the testing session and in–between the two short–term reliability tests. Subjects were asked to prepare similarly for each test and wear the same footwear. All tests were performed at approximately the same time of day between the hours of 10 am and 12 noon to prevent the effects of circadian variations on variables measured (Racinais *et al.*, 2005). The test order of the subjects was kept the same for all tests.

5.2.1.3.4.1 Analysis of RSA protocol reliability

The statistical procedures employed to assess reliability were a single factor, within-subjects (repeated-measures) ANOVA, intraclass correlation coefficient (ICC) and Typical Error (TE). The ANOVA was used as an inferential test of mean differences across trials as an assessment of systematic error. If this investigation showed significant differences between trials, further trials would have been performed until results showed no significant differences. Intraclass correlation determines the degree to which

individuals maintain their position in a sample with repeated measures (Atkinson and Nevill, 1998) and, when combined with ANOVA and TE, have been suggested to be the most appropriate measures of reliability (Denegar and Ball, 1993; Greig and McNaughton, 2005).

The TE allows the investigator to appreciate the extent to which the observed score would deviate from the true score and has the same units as the measurement of interest (Spencer *et al.*, 2006). The TE can be used to determine the coefficient of variation and Limits of Agreement. The TE can also be used to define the smallest meaningful change (SMC) needed between separate measures on a given subject. The smallest meaningful change was calculated as follows (Ellaszlw *et al.*, 1994; Greig and McNaughton, 2005).

$$SMC = TE \times 1.96 \times \sqrt{2}$$

Once the SMC is calculated, then any change in a subject's score, either below or above the previous score, that is greater than the SMC is considered 'real'. More precisely, for all subjects whose differences on repeated testing are at least greater than or equal to the SMC, 95% of them would reflect real differences (Greig and McNaughton, 2005).

5.2.1.3.4.2 Statistical analysis of RSA reliability

Data are presented as mean ± standard deviation (SD). Verification that the data were normally distributed was provided by the Kolomogorov–Smirnov normality test and visual inspection of the normality plots. The differences in sprint times for each player for corresponding sprint numbers across the 3 trials were evaluated with an analysis of variance (ANOVA) with repeated measures. If significant ($p<0.05$) between–trial differences were observed, a Bonferroni–adjusted post hoc analysis was used to determine where the differences occurred.

The dependant variables tested for reliability were TT, TT_{0-20}, TT_{20-40}, quickest 40 m sprint in each test, RSA_{PD} and RPE. The following criteria were adopted for interpreting the magnitude of correlation (*r*) between measures: an ICC of >0.9 was considered almost perfect, 0.7–0.9 very large, 0.5–0.7 large, 0.3–0.5 moderate, 0.1–0.3 small and

<0.1 trivial (Pyne *et al.*, 2008). Because of the small number of subjects, ICC and TE were expressed with associated 95% confidence intervals (CI).

5.2.1.3.4.3 Reliability of total sprint time and individual sprint performance

Total sprint time was not different (mean 32.1 ± 1.2 s, 32.6 ± 1.1 s, 32.2 ± 1.0 s) ($F(2,33) = 0.8$, $p>0.5$) between RT1, RT2 and RT3 respectively. There were also no differences in sprint times when comparing each player's equivalent sprint number across the three trials ($p>0.05$). This suggests an acceptable level of systematic bias. When investigating any between–trial differences in $TT_{0–20}$ and $TT_{20–40}$, there were also no differences between the three trials ($p>0.05$). The ICC and TE for short- and long-term reliability of TT are shown in Table 5.1.1. The SMC in TT for this population, calculated from trial 1 and 3 = ± 0.8 s.

Table 5.1.1. Typical error, and ICC values with their respective lower and upper 95% confidence intervals for short–term and long–term reliability as a factor of total sprint time for all 6 sprints

	Change in mean (s)	95% CI			95% CI	
		TE (s)	lower	Upper	ICC (r)	Lower	Upper
RT1 and RT2	0.55	0.23	0.19	0.45	0.95	0.8	0.99
RT1 and RT3	0.25	0.18	0.21	0.5	0.87	0.60	0.96

5.2.1.3.4.4 Fatigue measures

Analysis of variance for RSA_{PD} showed no differences between RT1, RT2 or RT3 ($F(2, 33) = 1.8$, $p>0.05$). Table 5.1.2 shows the ICC and TE with associated 95% confidence intervals. The SMC for RSA_{PD} calculated from the TE from RT1 and RT3 was ± 2.2%.

Table 5.1.2 Short–term and long–term test–retest reliability measures for RSA performance decrement

	Change in mean (%)	TE (%)	95% CI		ICC (r)	95% CI	
			lower	Upper		Lower	Upper
RT1 and RT2	0.04	1.2	0.9	2.1	0.6	-0.01	0.86
RT1 and RT3	0.3	0.8	0.6	1.4	0.33	-0.3	0.76

This analysis would suggest that only one session prior to testing was necessary to ensure full familiarisation. The inclusion of this one session would reduce the chances of type I errors due to systematic bias. Short– and long–term test–retest reliability for TT, TT_{0-20} and TT_{20-40} and RPE variables were also found to be robust. RSA_{PD} was found to be less reliable with a change of ±2.2% required to suggest a 'real' change in performance. The mean ± SD RSA_{PD} for all 3 trials combined was 4.8 ± 1.8%. A smallest change of 2.2% therefore compares to a performance decrement change of almost 50% between trials before any change could be attributed to an intervention.

5.2.2 Eligibility criteria and group allocation

Although eligibility criteria was for soccer players sustaining injuries resulting in >14 d absence, any player injured to such a severity that their absence was prospectively estimated to be >10 d were randomly allocated to a constant steady-rate training group (SPG) or an intermittent training group (IPG). This was to ensure that an underestimation of severity did not lead to loss of suitable subjects. If subsequently, injured players were not absent from training and playing for >14 d, their data were not included in the study. This happened on two occasions during the study period. On no occasions did a player, whose injury was estimated as < 10 d, meet the inclusion criteria. As the aim of this study was to evaluate the effect of intermittent training in early rehabilitation, any player sustaining an injury that resulted in lower limb immobilisation or the inability to perform at least one of the activities to the desired intensity for a period >14 d were eliminated from the study. To ensure that the intervention and control groups had a similar spread of injury severities and player positions, group allocation was undertaken using minimisation (Evans, 2006). Injury severity was categorised as detailed in Chapter 3.1.

5.2.3 Training intervention

All rehabilitation sessions were quantified using methodologies detailed in Chapter 4.2. At the current soccer club, it was normal for injured players who were unable to run during early rehabilitation to perform cardiovascular (CV) exercise on two of three cardiovascular exercise machines (static cycle, cross-trainer and rowing machine). This exercise was only one component of early rehabilitation and was performed in 30 min segments at a steady cadence and resistance selected by the physiotherapist after considering the specific pathology and severity. The current intervention controlled the internal training load limiting inter-player variations in the training load during early rehabilitation.

During the study period, both groups of injured players participated in normal rehabilitation training relevant to their pathologies with one significant difference. The SPG performed CV exercise at a constant steady-rate and the IPG performed CV exercise using an intermittent exercise pattern. To facilitate consistency of data, all CV training was completed on a cycle ergometer (Life Fitness, Illinois, USA), cross-trainer (Life Fitness, Illinois, USA) or rowing machine (Concept II rowing machine, Nottingham, UK) for periods of 30 minutes. All players wore heart rate monitors set to sample at 5 s (Polar Team System. Polar Electro Oy, Kempele, Finland) for all rehabilitation sessions. Players were supervised by the same physiotherapist for all sessions and were verbally encouraged throughout each exercise bout to ensure consistent effort. All injured players were instructed not to participate in any additional physical exercise when not at the club.

During CV sessions, the SPG maintained a consistent cadence while the IPG exercised using an intermittent cadence which fluctuated above and below the cadence used for the SPG. Pilot work was undertaken to investigate the level of difficulty and cadence for each exercise ergometer that would elicit a heart rate response of 75-80% HR_{max} for constant steady-rate exercise. Heart rate responses of 75% of HR_{max} and 80% HR_{max} represent an intensity of exercise of approximately 60% and 67% of $\dot{V}O_{2max}$ respectively

(Swain *et al.*, 1994). This work was undertaken over a period of 3 months prior to the study commencing using apprentice and professional soccer players. The pilot data collected suggested that the appropriate range of intensities for the cycle ergometer were level 15 of 20 and a cadence of 70-90 rpm. The corresponding exercise prescription for the cross-trainer and the rowing machine were level 15 of 20 at 65-85 rpm and level 10 of 10 at a 1 min 50 s – 2 min 10 s per 500 m pace respectively. Additional pilot sessions explored the percentage increase that players could tolerate if an intermittent exercise pattern was performed. The increase in cadence from that required to elicit 75-80% HR_{max} during the continuous protocol was set at +20% for all modalities. This increase was found to be sufficient to make it not possible for the majority of players to continue constant steady-rate exercise for a period >10 min yet maintain the desired workloads for the 30 min of the exercise session. To facilitate the matching of the workload to that of the constant steady-rate protocol, the lower intensity for the intermittent protocol was set at 20% below, and the higher intensity at 20% above, that of the cadence of the constant steady-rate protocol.

To determine the individual cadence settings of the CV equipment, each injured player undertook a preliminary incremental test on each piece of equipment as soon as their particular pathology permitted. The aim was to determine a cadence which elicited the required 75% to 80% of the player's HR_{max}. The player wore a Polar HR monitor (Polar RS100, Polar Electro Oy, Kempele, Finland) with a wrist watch that was set to indicate, when the player had reached the target HR. This was identified by means of an audible bleep from the HR monitor. Following a short (~3 min) recovery, every player completed one 30 min bout of constant steady-rate exercise on each piece of equipment with >30 min recovery between each exercise bout. The constant steady-rate cadence was used for all subsequent SPG training completed by the player. This parameter associated with this session was also used to calculate the cadence for the IPG protocol (Table 5.1.3). For example, in the preliminary test, to elicit a heart rate of ~75% HR_{max}, a player performed on the cross-trainer at an intensity of level 15 and a cadence of ~70 rpm. If this player was allocated to the intermittent protocol, he would perform 30 min on the cross trainer at an intensity of level 15 with alternating 30 s periods of ~84 and ~56 rpm. Figure 5.1.2

illustrates the heart rates for a constant steady-rate and intermittent cycle ergometer protocol. Using this method the number of repetitions on each piece of equipment should also be matched. As a further indicator of the two protocols being matched, the distance covered was also recorded and compared with the preliminary constant steady-rate exercise session for the relevant piece of equipment.

Table 5.1.3 Constant steady-rate and intermittent cadences for static cycle ergometer, cross trainer and rowing machine.

Cycle level 15	RPM	RPM	RPM	RPM	RPM
Constant steady-rate	60	70	80	90	100
Intermittent upper rate	72	84	96	108	120
Intermittent lower rate	48	56	64	72	80
Cross trainer L15	RPM	RPM	RPM	RPM	RPM
Constant steady-rate	60	70	80	90	100
Intermittent upper rate	72	84	96	108	120
Intermittent lower rate	48	56	64	72	80
Rowing machine L10	s/500 m	s/500 m	s/500 m	s/500 m	s/500 m
Constant steady-rate	140	130	120	110	100
Intermittent upper rate	168	156	144	132	120
Intermittent lower rate	112	104	96	88	80

Players completed training on 2 of the 3 pieces of exercise equipment during each rehabilitation training day. Specific exercise prescriptions were timetabled to vary between modes of exercise to ensure an equal number of exercise bouts on each piece of equipment were performed across the early rehabilitation training period. When players had recovered sufficiently to be able to start running, they discontinued the intervention protocol and reverted back to following a normal rehabilitation programme. This permitted a clear analysis of the differences between the distinct intervention strategies used in the early stages of rehabilitation to be evaluated.

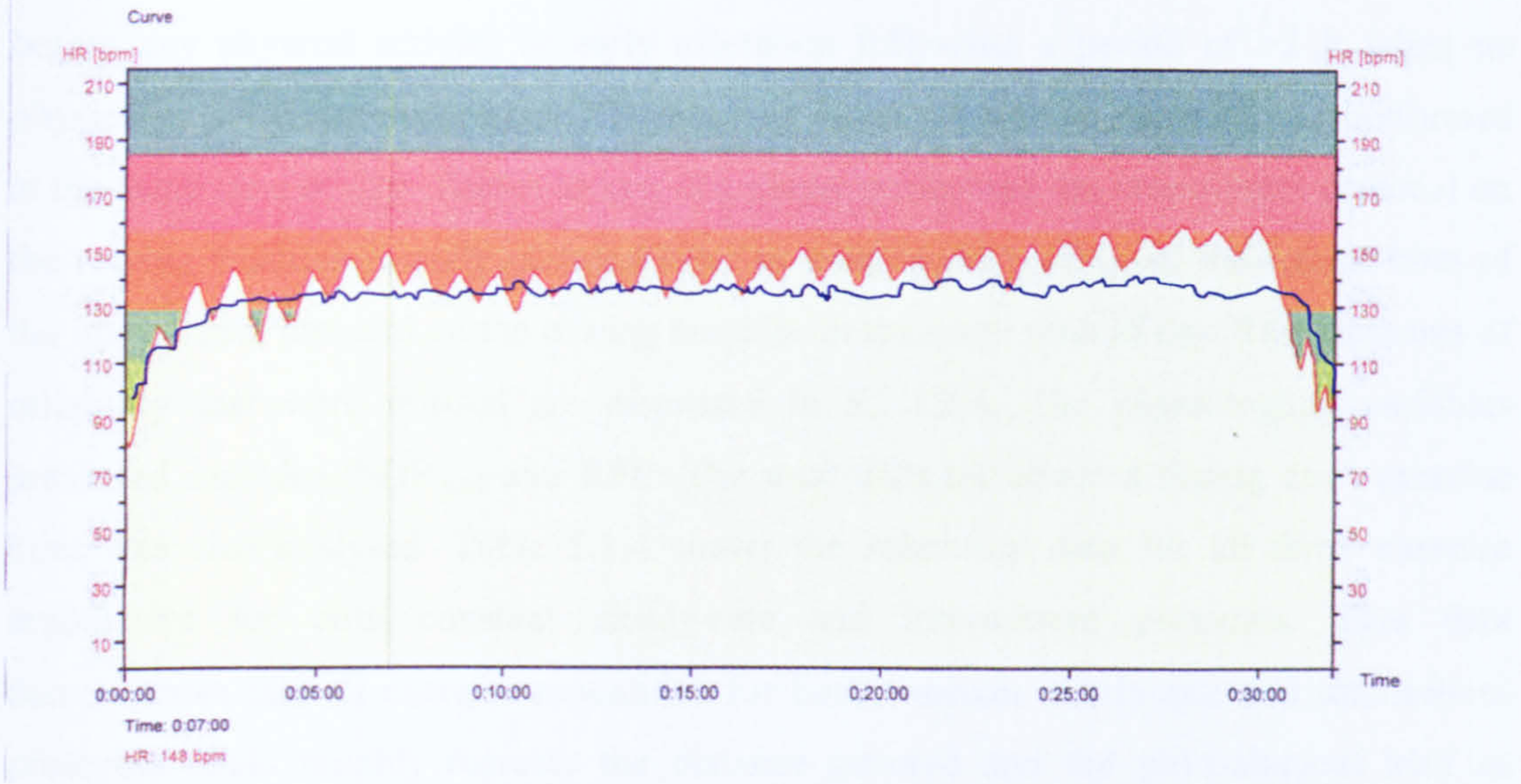


Figure 5.1.2 Illustration of Polar heart rate tracings for 30 min intermittent (red line) and constant steady-rate (blue line) cycle ergometer exercise.

To compare the training load associated with SPG and IPG during rehabilitation, each individual rehabilitation programme was split into 2 phases. These two phases were differentiated into early and late rehabilitation phases. Early rehabilitation was the stage of rehabilitation when the player was unable to run at a jogging pace. This corresponded to the intervention stage. Late rehabilitation was the point at which the player was able to run at a jogging pace until the stage at which he was able to participate fully in normal squad training sessions. This approach allowed for any significant differences in training load during either early or late rehabilitation between SPG and IPG to be identified. The variables used to quantify this training load included heart rate expressed as percentage of maximal heart rate ($\%HR_{max}$), training impulses (Trimps) and RPE. Methods of collecting, calculating and recording this data can be found in Chapter 4.1.

5.2.4 Test-retest reliability of intermittent intervention

The reliability of the workload prescription for the 3 separate pieces of CV equipment was determined using test-retest methods. Twelve elite apprentice soccer players (age 17±1) performed on each exercise ergometer on 4 occasions, the constant steady-rate protocol twice and the intermittent protocol twice. The 12 trials for each player were

completed within a 2-wk period. Each exercise bout was performed either early morning before any physical activity or early afternoon following a period of >2 h when no physical activity was undertaken. The exercise bouts on each ergometer were performed at the same time of day. That is to say, if a player performed the intermittent protocol on the rowing machine in early morning prior to training, he performed the second bout of the intermittent protocol on the rowing machine at the same time of day. The measures of reliability that were utilised are discussed in 5.2.1.2.4. The physiological variables measured included $\%HR_{max}$ and RPE. The total distance covered during each exercise bout was also analysed. Table 5.1.4 shows the reliability data for all three exercise ergometers for both constant steady-rate and intermittent protocols. This data demonstrates that all exercise modalities for both constant steady-rate and intermittent protocols could reliably recreate the distance covered and the physiological load as indicated by $\%HR_{max}$. The data for ratings of perceived exertion showed that this variable was not as reliable for all three modalities but still fell within an acceptable range.

5.2.5 Retesting

All players absent through soccer injury for >14d were retested immediately prior to return to full squad training using the same test protocols (T2). If this was not possible, T2 was performed within 2 days of returning to squad training. On no occasion throughout the study period was it not possible to retest players within 2 d of their return to squad training.

Table 5.1.4 Reliability statistics for constant steady-rate and intermittent protocols on cycle ergometer, rowing machine and cross trainer.

		Constant steady-rate					Intermittent rate				
		Change in mean	Typical Error	95% CI Lower	95% CI Upper	ICC	Change in mean	Typical Error	95% CI Lower	95% CI Upper	ICC
Rowing machine	$\%HR_{max}$	0.02	0.2	0.1	0.5	0.7	0	1.8	1.1	4.4	0.87
	Distance (m)	5.8	21	13	52	0.8	-22	41	26	101	0.95
	RPE	-0.2	0.41	0.33	0.65	0.31	0	0.32	0.21	0.6	0.33
Cycle ergometer	$\%HR_{max}$	0	0.02	0.01	0.04	0.86	0.9	0.01	0.01	0.02	0.84
	Distance (km)	0.07	0.07	0.05	0.18	0.88	0.02	0.1	0.06	0.2	0.83
	RPE	-0.2	0.22	0.15	0.45	0.55	0	0.44	0.25	0.7	0.5
Cross trainer	$\%HR_{max}$	0.04	0.1	0.08	0.3	0.81	0.02	0.1	0.05	0.2	0.5
	Distance (km)	0.02	0.1	0.15	0.6	0.9	0.03	0.1	0.06	0.2	0.9
	RPE	-0.3	0.4	0.2	0.7	0.43	0	0.45	0.32	0.78	0.5

(ICC = Intraclass correlation, (shown with 95% confidence limits), TE = Typical Error)

Statistics

All data was processed on a Dell computer (Dell Inc., Wicklow, Ireland) using Microsoft Office (Microsoft Corp., Redmond, Washington). The statistical analyses were completed using SPSS Version 15 for Windows (Chicago, Illinois, USA). Normality of distribution for all variables was checked with the Kolomogorov-Smirnov normality test. Methods applied were frequencies, descriptives, and means ± SD for anthropometric, injury severity and days to running data. One-way analysis of variance (ANOVA) was used to determine if there were any differences between SPG and IPG for anthropometric data,

length of absence, days to running and results of T1 performance tests. One-way ANOVA were also utilised to examine the differences between SPG and IPG for intervention RPE, HR_{ave} and Trimps during early rehabilitation and for each individual exercise ergometer. A two way between-groups ANOVA (two groups x two tests) with repeated measures for time were used to test for interaction and main effects for the independent variables measured. In addition, the effect size was calculated and Cohen's conventions for effect size were used for interpretation, where effect size = 0.2, 0.5, and 0.8 are considered small, medium and large, respectively. Pearson product-moment correlation coefficients were used to compute the relationship between changes in T1 and T2 between all performance tests. Pearson product-moment correlation coefficients were also used to compute the relationship between duration of player absence and performance changes. Statistical significance was accepted as $p < 0.05$ unless stated otherwise.

5.3 Results

5.3.1 Subjects

During the study period 18 injuries were sustained which met the inclusion criteria of a soccer-related injury resulting in >14 d absence. Two injured players remained injured at the end of the study period and were therefore excluded from the study. The playing positions of the 16 included players were goalkeeper (n = 1); defender (n = 4); midfielder (n = 4); and striker (n = 7) (Table 5.1.5). Five of the injuries were sustained in training, 8 in first team matches and 3 in reserve team matches. Eight injuries were allocated to the SPG (goalkeeper n = 1, defender n = 2, midfielder n = 2 and striker n = 3) and 8 to the IPG (defender n = 2, midfielder n = 2 and striker n = 4). No differences for any anthropometric variables between the subjects allocated to the SPG and IPG were observed ($p>0.05$). The severity of injuries in each group is shown in Table 5.1.6.

Table 5.1.5 Descriptive details of all injured players and injured players as a factor of intervention group.

	Variable	Minimum	Maximum	Mean	SD
All injured players (n = 16)	Age (y)	18	33	25.1	5.6
	Mass (Kg)	67	88	80	5.9
	Height (m)	1.68	1.93	1.8	0.1
	Est. Body fat %	9.6	12.5	11.1	0.8
	HR_{max}	180	204	190.4	7.5
Constant steady-rate group (n = 8)	Age (y)	18	32	24.6	6.1
	Mass (Kg)	67	86	78	5. 6
	Height (m)	1.68	1.93	1.8	0.1
	Est. Body fat %	9.6	12.5	11.1	0.9
	HR_{max}	182	204	192	7.8
Intermittent rate group (n = 8)	Age (y)	19	33	25.6	5.5
	Mass (Kg)	67.2	87.7	81.3	6.4
	Height (m)	1.68	1.93	1.8	0.1
	Est. Body fat %	10.0	12.0	11.1	0.7
	HR_{max}	180	202	189	7.5

Table 5.1.6 Severity of injuries in terms of length of absence for all injuries and as a factor of intervention group

	Total days absence	Frequency	Percent	Mean absence (d)	SD
All injured players (N = 16)	15-28	9	56.3	22	2.8
	29-60	5	31.3	39	6.4
	>60	2	12.5	80	7.8
Constant steady-rate group (n = 8)	15-28	4	25.0	22	3.8
	29-60	3	62.5	37	3.2
	>60	1	12.5	85	
Intermittent rate group (n = 8)	15-28	5	50.0	21	2.2
	29-60	2	37.5	42	10.6
	>60	1	12.5	74	

The total number of day's absence for the 16 injuries was 547 (mean 34.2±20 days absence per injury; SPG 35±21 day's absence per injury, IPG 34±20 day's absence per injury). The 16 injuries also resulted in 104 occasions when the player was not available for first team selection (mean 6.5±3.8 matches unavailable. SPG 7±4 matches unavailable; IPG 6±4 matches unavailable). No differences between SPG and IPG were observed for any absence variables ($p>0.05$).

5.3.2 *Test one results*

Table 5.1.7 details the performance variables for T1 for all players included in the study and as a function of intervention group. An analysis of the differences between SPG and IPG showed no differences in any performance variable before the training intervention ($p>0.05$).

Table 5.1.7 Test 1 performance variables for all players and as a function of intervention group

Variable		Mean	SD	Min	Max
Total sprint time (s)	All players	32.0	1.38	29.9	35.8
	SPG	32.5	1.67	29.9	35.8
	IPG	31.6	0.91	29.9	32.4
Quickest sprint (s)	All players	5.17	0.21	4.82	5.77
	SPG	5.25	0.26	4.9	5.77
	IPG	5.1	0.13	4.82	5.28
Performance decrement (%)	All players	-3.36	0.95	-5.41	-1.72
	SPG	-3.18	0.55	-3.89	-2.15
	IPG	-3.53	1.24	-5.41	-1.72
Estimated $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	All players	59.5	3.24	54.8	65.1
	SPG	59.9	2.76	55.4	63.2
	IPG	59.0	3.81	54.8	65.1
MSFT metres completed (m)	All players	2698	242	2360	3120
	SPG	2728	210	2380	2980
	IPG	2668	282	2360	3120

5.3.3 *Rehabilitation and intervention training*

The average number of days following the injury to players being able to perform the intervention protocols was 2.3±1.3 d. The average number of day's absence from the day of the injury to the day that players were able to perform running exercise was 17±7. The mean number of day's absence to being able to run expressed as a percentage of the total

absence duration was 52±13%. These data suggest that early and late rehabilitation phases were of approximately the same duration. Table 5.1.8 shows the days to run details for all players and as a function of intervention group. There was no difference between SPG and IPG for the number of days to a player being able to run or the number of days expressed as a percentage of the total absence ($p>0.05$)

Table 5.1.8 Number of days from the day of injury until players able to participate in their first running session.

		Mean	SD	Min	Max
All players	Number of days to first jog	16.6	7.1	8	32
	% of total absence to first jog	52	13	27	70
SPG	Number of days to first jog	17.5	6.8	11	28
	% of total absence to first jog	54	14	33	70
IPG	Number of days to first jog	16	7.8	8	32
	% of total absence to first jog	51	13	27	65

One hundred and twenty five training sessions associated with the intervention were performed by all players (mean number of intervention sessions per player 8±13). The average heart rate for these sessions was 149±5 beats·min^{-1} which corresponded to a mean value of 78±0.2 $\%HR_{max}$. Data for the individual modalities for all injuries and for both SPG and IPG are shown in Table 5.1.9. During the early phase of rehabilitation the IPG protocol resulted in significantly more Trimps than the SPG protocol (IPG, 140±25 Trimps; SPG, 122±17 Trimps, $t(1) = 9.5$, $p<0.05$). There were no differences between SPG and IPG for $\%HR_{max}$ or RPE in the early rehabilitation phase ($p>0.05$). There were also no differences between SPG and IPG for $\%HR_{max}$, RPE or Trimps in the late rehabilitation phase ($p>0.05$) (Figure 5.1.3).

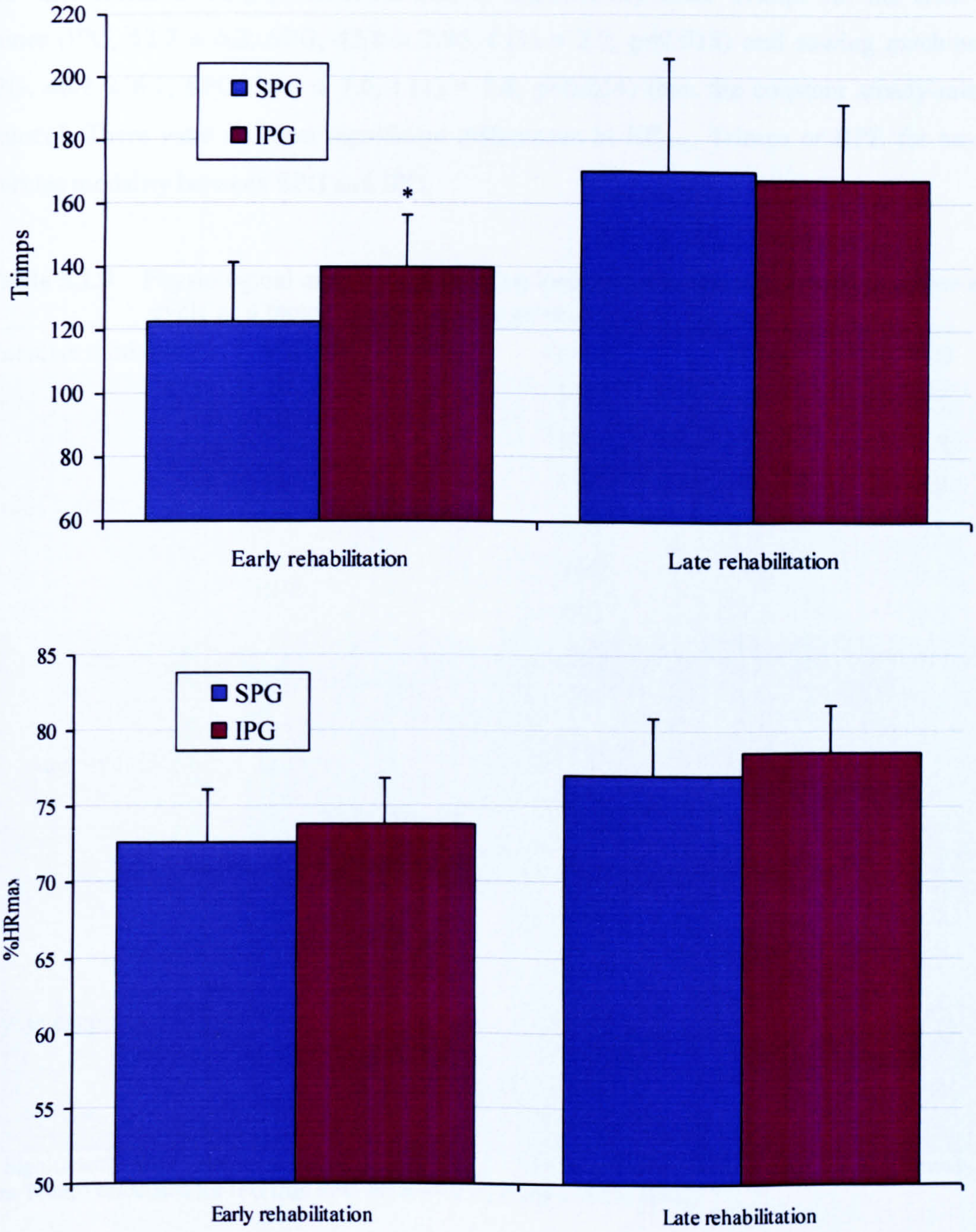


Figure 5.1.3 Training load for the phases of rehabilitation as a factor of intervention group.
* Significantly more Trimps during IPG than SPG $p<0.05$.

The intermittent training protocol resulted in significantly more Trimps for the cross-trainer (IPG, 52.7 ± 6.2; SPG, 42.8 ± 2.96, t (1) = 7.7, p=0.015) and rowing machine (IPG, 48.2 ± 6.1; SPG, 38.6 ± 7.6, t (1) = 7.8, p=0.014) than the constant steady-rate protocol. There were no other significant differences in HR_{ave}, Trimps or RPE for any exercise modality between SPG and IPG.

Table 5.1.9 Physiological measures of training load on cross-trainer, rowing machine and cycle as a factor of intervention group.

Exercise modality	Variable	Group	Mean	SD
Cross-trainer	HR_{ave} (beats·min^{-1})	SPG	149	3.0
		IPG	152	6.2
	Trimps	SPG	43.4	8.3
		IPG	52.7*	5.9
	RPE	SPG	7.6	0.2
		IPG	7.5	0.3
Rowing machine	HR_{ave} (beats·min^{-1})	SPG	146	3.0
		IPG	151	6.1
	Trimps	SPG	38.6	7.6
		IPG	48.2$^{\$}$	6.1
	RPE	SPG	7.3	0.3
		IPG	7.0	0.2
Cycle ergometer	HR_{ave} (beats·min^{-1})	SPG	146	2.6
		IPG	149	6.7
	Trimps	SPG	38.0	7.1
		IPG	44.6	5.9
	RPE	SPG	7.1	0.3
		IPG	6.9	0.3

* = Significantly more Trimps calculated for IPG than SPG for cross-trainer exercise bout. $^{\$}$ = Significantly more Trimps calculated for IPG than SPG for rowing machine exercise bout.

5.3.4 Test two results

All players completed T2 prior to returning to normal squad training or within 2 days of returning to normal squad training. Table 5.1.10 details the performance data for all players for T2 and as a function of intervention group.

Table 5.1.10 Test 2 performance variables for all players and as a function of intervention group.

Variable		Mean	SD	Min	Max
Total sprint time (s)	All players	32.5	1.6	30.3	37.2
	SPG	33	2.0	30.3	37.2
	IPG	32	0.9	30.5	32.8
Quickest sprint (s)	All players	5.2	0.23	4.8	5.9
	SPG	5.2	0.30	4.9	5.9
	IPG	5.1	0.13	4.9	5.3
Performance decrement (%)	All players	-4.5	1.13	-6.1	-2.0
	SPG	-4.9	0.74	-5.9	-3.8
	IPG	-4.2	1.4	-6.1	-2.0
RPE for RSA protocol	All players	8.3	0.7	7	9
	SPG	8.5	0.5	8	9
	IPG	8	0.8	7	9
Estimated $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	All players	58.4	3.1	54.3	63.2
	SPG	58.1	2.7	54.3	61.5
	IPG	58.6	3.5	54.3	63.2
MSFT metres completed (m)	All players	2618	223	2320	2980
	SPG	2598	196	2320	2840
	IPG	2638	259	2320	2980

There were no significant main effects in estimated $\dot{V}O_{2max}$ between intervention groups ($F(1, 293) = 0.03$, $p = 0.87$, $\eta=0.01$) or between T1 and T2 ($F(1, 293) = 0.9$, $p = 0.35$, $\eta=0.03$). There was also no significant differences when the interaction between intervention group and test number was investigated ($F(1, 293) = 0.37$, $p = 0.55$, $\eta=0.01$).

There were no significant differences in RSA performance, as indicated by TT, between intervention groups ($F(1, 59) = 3.3$, $p = 0.08$, $\eta=0.11$), test number ($F(1, 59) = 0.9$, $p = 0.42$, $\eta=0.02$) or the interaction between intervention group and test number ($F(1, 59) = 0.2$, $p = 0.89$, $\eta=0.00$). Fatigue during the RSA test, which was measured using RSA_{PD},

increased in both intervention groups. A significant increase from T1 to T2 was identified (mean T1 = -3.2±0.6%; mean T2 -4.9±0.7%), ($F(1, 58) = 10.02$, $p < 0.01$, $\eta=0.26$). No significant differences however, were observed between intervention group ($F(1, 58) = 0.23$, $p = 0.63$, $\eta=0.01$) or the interaction of intervention group and test number ($F(1, 58) = 2.03$, $p = 0.17$, $\eta=0.07$).

Changes between T1 and T2 in players' quickest and slowest sprint times during the RSA test were also investigated. Players' quickest sprint times did not change significantly between T1 and T2 ($F(1, 28) = 0.1$, $p = 0.9$, $\eta=0.00$). There was also no difference between the two intervention groups ($F(1, 28) = 3.27$, $p = 0.08$, $\eta=0.1$) or in the interaction between intervention group and test number ($F(1, 28) = 0.04$, $p = 0.84$, $\eta=0.00$). Although there was an increase in the mean time of players' slowest sprint (mean T1 = 5.5±0.26 s; mean T2 = 5.62±0.3 s) this effect was not different between intervention groups ($F(1, 28) = 2.5$, $p = 0.13$, $\eta=0.08$) or between T1 and T2 ($F(1, 28) = 1.65$, $p = 0.21$, $\eta=0.06$.

Figure 5.1.4 illustrates the percentage changes in performance for RSA total sprint time, RSA_{PD} and estimated $\dot{V}O_{2max}$ as a factor of intervention group. Investigating correlations between changes in performance from T1 to T2 showed no significant correlations in both SPG and IPG for any performance variables ($p>0.05$). Pearson product-moment correlations were also used to investigate if there was a correlation between the number of days that players were absent for, and changes in performance indices. No significant correlations were observed ($p>0.05$).

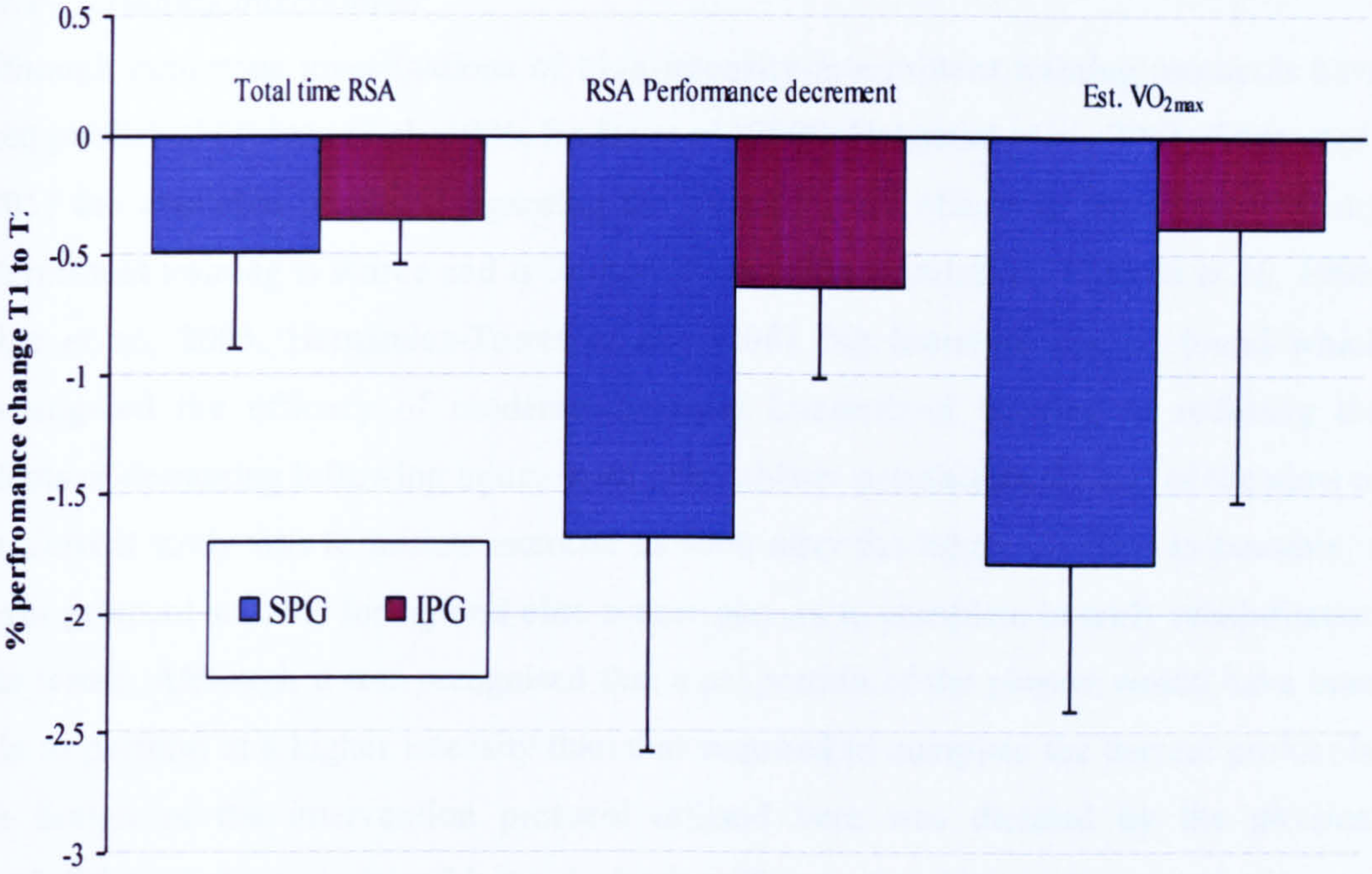


Figure 5.1.4 Performance changes from T1 to T2 as a percentage of T1.

5.4 Discussion

This study is the first to compare the effects of different exercise strategies, during the early rehabilitation of injured elite soccer players, on $\dot{V}O_{2max}$ and RSA performance. The major finding of the current study was that there was no significant differences in the impact of utilising either a constant steady-rate training protocol or a work-matched moderate-intensity intermittent training protocol during early rehabilitation. When comparing the training loads of both groups during early rehabilitation, the only significant difference was a greater number of Trimps completed in IPG than SPG suggesting that the training loads of SPG and IPG were similar during early rehabilitation. No significant differences in training load for late stage rehabilitation were observed between the two groups. These findings would suggest that constant steady-rate cardio-vascular training at an intensity of ~65% $\dot{V}O_{2max}$ produces similar results as work-matched intermittent training when the aim is to reduce the effects of injury-induced detraining on indices of aerobic power and RSA_{PD}.

5.4.1 Training intervention

Although numerous investigations of high-intensity-intermittent training protocols have been published (Tabata *et al.*, 1996; Rodas *et al.*, 2000; Helgerud *et al.*, 2001; Edge *et al.*, 2005) the available literature regarding the physiological effects of moderate-intensity intermittent training is scarce and is limited to non-elite populations (Tabata *et al.*, 1996; Edge *et al.*, 2005; Hernández-Torres *et al.*, 2008). No literature can be found which investigated the efficacy of moderate-intensity intermittent training in reducing the effects of detraining following injury in an elite athletic population. As one of the aims of the current study was to initiate exercise as soon after the injury incident as possible, a novel protocol suitable for injured elite soccer players to complete in early rehabilitation was tested. Although it was recognised that a proportion of the players would have been able to perform at a higher intensity than that required to complete the current protocols, the design of the intervention protocol utilised here was dictated by the physical capabilities of the majority of injured players. The current intervention protocols were able to facilitate this early participation as demonstrated by the low average number of days following injury to players being able to perform either of the intervention protocols.

Investigations into the heart rate responses during early rehabilitation showed that there were no significant differences during early rehabilitation in average heart rate between IPG and SPG. The $\%HR_{max}$ levels observed were greater than those observed during similar training activities in category 1 rehabilitation training (average 67% HR_{max}) presented in Chapter 4.2. Players' heart rates during both intervention protocols were also ~90% of the average heart rate observed by Drust *et al.* (2000) during a soccer-specific exercise protocol which was considered to accurately reflect the physiological demands of a soccer match. Heart rate levels during early rehabilitation were also slightly higher than those experienced during normal squad training (140 beats·min^{-1}). It should however, be noted that average heart rate and $\%HR_{max}$ only give a representation of the average intensity of a training session and no indication of any fluctuations in heart rate due to periods of greater physiological load. For example, normal training, which is

characterised by bouts of very high-intensity exercise interposed with periods of rest, demonstrated an average $\%HR_{max}$ which was similar to that that of early rehabilitation. The mode of exercise during early rehabilitation in the current study however, resulted in a heart rate response pattern that lacked the peaks and troughs associated with normal training. It would therefore appear that, although the HR_{ave} of normal training and early rehabilitation are similar, care should be taken when drawing conclusions as to the similarity of the physiological responses to the two training methods.

To ensure that the two intervention groups were comparable, confounding variables were investigated. Pre-injury and injury severity data for both intervention groups were very similar. No significant differences between SPG and IPG were found for severity of injury (total days absence) or the number of days from the injury to the day players were able to jog ($p>0.05$). Results from T1 also showed that there were no significant differences between the intervention groups for any performance variable. Current performance data for T1 also compares well with other studies using 40 m sprints as a performance measure (Brewer and Davis, 1991; Azhar and Schmidt, 1998) and $\dot{V}O_{2max}$ in elite soccer players (Reilly *et al.*, 2000b; Strøyer *et al.*, 2005). The above data suggest that there were no significant differences between the two intervention groups prior to undertaking the intervention protocols.

When investigating significant differences between the intervention protocols, there was no significant difference in $\%HR_{max}$. In fact, the only significant difference observed between the 2 protocols was the number of Trimps calculated for IPG being significantly greater than for SPG ($p<0.5$). This was probably due to the intermittent training pattern of IPG. Trimps are quantitative arbitrary units that were devised to assess the amount or volume of training undertaken in any one given bout (Morton, 1997). They are based, in a non-linear fashion, on the extent to which the training raises the heart rate between resting and maximum. Trimps have the advantage of loading outcomes in favour of high intensity work and this is probably why the intermittent protocol resulted in significantly more Trimps that the continuous protocol. No significant difference between SPG and IPG for RPE was also observed during early rehabilitation ($p>0.05$).

Player's late-stage rehabilitation was also quantified. This phase was initiated when players were able to commence running based rehabilitation exercise. During this phase, no significant differences were observed between SPG and IPG for Trimps, $\%HR_{max}$ or RPE (all $p>0.05$). These data suggest that the two groups contained a similar spread of injury severity and followed a comparable rehabilitation programme.

5.4.2 Performance changes

Both protocols resulted in a reduction in estimated $\dot{V}O_{2max}$ with the reduction in estimated $\dot{V}O_{2max}$ for the SPG (-1.8 ± 0.6 ml·kg^{-1}·min^{-1}) being greater than the 1.03 ml·kg^{-1}·min^{-1} calculated for the SMC. These reductions however were not sufficient to be significant between intervention groups or between T1 and T2. These findings suggest that there was little, if any, difference between the SPG and IPG protocols when the aim is to prevent negative changes in estimated $\dot{V}O_{2max}$ as a result of detraining.

The above results indicate that the estimated $\dot{V}O_{2max}$ of all players returning to squad training was not significantly different from data collected prior to the players sustaining injuries. Methodological limitations however, only allowed for $\dot{V}O_{2max}$ to be determined just prior to players returning to squad training. This was due to the restriction of being unable to determine $\dot{V}O_{2max}$ throughout the course of the rehabilitation period as a consequence of the disabling effects of the various pathologies encountered. It may therefore be possible that there was a reduction in either or both intervention group's aerobic power during the early rehabilitation period which subsequently improved close to pre-injury levels following late rehabilitation training. Although the $\%HR_{max}$ during late rehabilitation was not found to be significantly greater than that during early rehabilitation, late rehabilitation is characterised by soccer-specific training which is predominantly high intensity periods of exercise interposed with periods of rest. This mode of exercise may have resulted in an average $\%HR_{max}$ that was similar to that of early rehabilitation but with a completely different heart rate response pattern due to the different physiological demands. It is therefore possible that, although the $\%HR_{max}$ of early and late rehabilitation are similar, the physiological responses to each phase are

significantly different. A method of establishing aerobic power at the end of the early rehabilitation phase may therefore identify any fluctuations in $\dot{V}O_{2max}$. Notwithstanding the possibility that late rehabilitation training may have contributed to reclamation of aerobic power, both intervention protocols were of sufficient intensity to be able to prevent significant reductions in $\dot{V}O_{2max}$.

These findings however, contradict the observations of others. It has been reported that intensity of exercise requiring ≥90-100% of $\dot{V}O_{2max}$ had to be maintained to prevent reductions in $\dot{V}O_{2max}$ following changes in training load (Hickson and Rosenkoetter, 1981; Wilmore, 1988; Fleck, 1994; Welsch *et al.*, 1994; Helgerud et al., 2001). The intensity of both protocols in the current study was estimated at ~65% $\dot{V}O_{2max}$. This indicates that a training protocol utilising a much higher percentage of $\dot{V}O_{2max}$ than that observed in the current study is required to prevent $\dot{V}O_{2max}$ performance decrements. Others however, have agreed with the current findings that exercise at a much lower percentage of $\dot{V}O_{2max}$ is sufficient to maintain performance (Rietjens *et al.*, 2001).

The current study observed a significant increase in RSA_{PD} following the rehabilitation period (mean T1 = -3.2±0.6%; mean T2 -4.9±0.7%). There were however, no significant differences between the intervention groups or the interaction of group and test number. The percentage change in mean RSA_{PD} for SPG (1.8±0.6%) was below the 2.2% calculated for any change to be considered 'real'. This is not surprising as quantifying fatigue in an RSA protocol is widely recognised to be problematic (Glaister *et al.*, 2008). For example, in the current study, the 2.2% change calculated to be necessary to indicate a 'real' change would represent a change of >50% of the mean RSA_{PD} calculated for T1 (-3.4±1%). The use of formulae to quantify fatigue during tests of RSA can therefore be questioned. However, an indicator of the drop-off in performance of successive sprints during repeated sprint performance is required to enable this important fitness parameter to be quantified. The formula utilised in the current study (Fitzsimons *et al.*, 1993) has been found to be the most suitable of the various formulae utilised in the literature to date

(Glaister *et al.*, 2008). Until a more robust formula to measure fatigue is available, it is necessary to continue using this formula.

The negative changes observed in RSA_{PD} were more marked in SPG (-1.8±0.6%) than IPG (-0.4±1.1). Although these changes were not significant, the results highlighted a possible weakness concerning the use of quickest sprint time in the formula utilised to calculate RSA_{PD}. This weakness may impact on future research, and may result in a type I error when rejecting the null hypothesis, even if significant changes are observed. The current observations, and those found in Chapter 4.1, of maximal sprint speed being maintained following periods of detraining is in agreement with others who have suggested that anaerobic performance is more robust to the effects of detraining than aerobic indices (Chi *et al.*, 1983; Linossier *et al.*, 1997; Amigo *et al.*, 1998). In the current study, player's average quickest sprint time for T1 and T2 changed very little (T1, 5.167 ± 0.05 to T2, 5.173 ± 0.06). The effect however, of even the smallest of changes in players' quickest sprint time on RSA_{PD}, can be significant. The SPG average quickest time for T2 was slightly quicker than T1 (-0.014 s) and the IPG slightly slower (+0.025 s). Although these changes appear to be very small, the 'ideal time' numerator in the RSA_{PD} formula is based on this figure being multiplied by six. The change in the difference between the numerators in the SPG and the IPG would therefore be 0.014 + 0.025 = 0.039 x 6 = 0.24 s. These minimal changes in quickest sprint time being in opposite directions in the two intervention groups, was therefore a contributory factor to the differences in RSA_{PD} observed between SPG and IPG in the current study. Examination of changes in quickest sprint times is therefore required when analysing results of RSA tests using the current performance decrement formula which utilise subject's quickest sprint times.

When comparing RSA TT between intervention groups, once again, the increase was more noticeable in the SPG (0.5 s) than the IPG (0.4 s). Analysis however, demonstrated this difference was not significant. Differences between test number and the interaction between group and test number were also not significant. The SMC, calculated from the reliability work, was 0.8 s which also suggests that neither group's performance,

measured in total time, changed sufficiently to be able to be certain that any 'real change' took place. These data suggest that, even though negative changes in both SPG and IPG for RSA_{PD} and TT were observed, the level of changes were insufficient to be able to draw any conclusion when comparing the effectiveness of either protocol at preventing negative RSA performance.

The findings of an absence of significant changes in performance in both aerobic power and RSA_{PD} are not surprising. The ability to recover from repeated sprints and $\dot{V}O_{2max}$ are related, with high levels of aerobic power associated with enhanced recovery during HIIE protocols (Dawson *et al.*, 1993; Balsom *et al.*, 1994; McMahon and Wegner, 1998; Aziz *et al.*, 2000; Krustrup and Bangsbo, 2001; Tomlin and Wegner, 2001; Barbero *et al.*, 2005). It may therefore be unexpected to observe a significant improvement or decrement in one fitness parameter without the other. This is because subjects with elevated $\dot{V}O_{2max}$ are more effective at transporting O_2 to the muscle and in facilitating its movement into the mitochondria where it is used as an electron receptor during ATP production (McMahon and Jenkins, 2002). At the onset of high-intensity exercise, the major pathways for adenosine triphosphate (ATP) resynthesis are the breakdown of phosphocreatine (PCr) and the degradation of muscle glycogen to lactic acid (McCartney *et al.*, 1986). With repeated bouts of high-intensity exercise with limited rest periods, the contribution of these processes to ATP turnover declines, and there is an increase in the aerobic contribution to support the energy requirements of exercise (Balsom *et al.*, 1993; Bangsbo, 2000b). Absence of changes in both aerobic power and RSA performance observed in the current study suggests that there may be a link between the two fitness parameters.

There may also have been considerations that weren't allowed for in the current study. Intermittent exercise results in higher lactate levels than work-matched constant pace exercise (Edwards *et al.*, 1973; Esfarjani and Laursen, 2007). Levels of ATP and CP have also been shown to result in considerable fluctuations during intermittent exercise but only demonstrate minor changes during work-matched continuous exercise (Essén, *et al.*, 1977). Although each of these studies utilised protocols of higher $\%HR_{max}$ than the

current study, lactate levels or levels of ATP and CP were not measured in the current study. Had they have been, they may have given an indication as to subtle differences in the physiological responses to the SPG and IPG protocols.

5.5 Conclusion

The current study found that the effectiveness of intermittent training and work-matched constant-pace exercise during early rehabilitation was similar when the aim was to reduce the effects of detraining on aerobic power and RSA. Both training regimens were effective in preventing significant performance decrements in estimated $\dot{V}O_{2max}$. It would appear that neither protocol was effective in preventing RSA_{PD} performance decrements although the scale of the changes observed suggest that further investigations into this performance variable are required.

CHAPTER 6

Synthesis of findings.

This chapter presents an overview of the findings of the current thesis. The chapter concludes with suggestions for future research.

Chapter 6 SYNTHESIS OF FINDINGS.

6.1 Introduction

The aim of this chapter is to review and integrate the experimental findings from studies 1-6. This analysis will provide a basis for an evaluation of the successful completion of the aims and objectives of the thesis. Important methodological limitations to all the experimental chapters are also provided as well as an overall conclusion to the thesis presented. The final section relates to the recommendations for future research.

6.1 Achievement of aims and objectives

Aim 1: To determine the incidence and aetiology of injuries at a professional soccer club throughout an extended period.

This aim was achieved through the completion of studies 1, 2 and 3. In study 1 a method of comprehensively recording soccer injuries was used to compare the accuracy of the current data with the data presented previously in the published research. Study 2 utilised these data collection methods to confirm that injury was the major cause of absence in professional soccer. The results of study 3 were then able to identify the major aetiological factors related to soccer injury and to quantify the incidence of injuries which may result in absences that could lead to detraining.

Implications

The implications arising from the findings of the above 3 studies are wide ranging. The accuracy of the data utilised enabled thorough analysis of previous epidemiological methodologies. Assessing the effectiveness of interventions designed to reduce the incidence of specific injuries requires accurate data collection methods which are sufficiently sensitive to detect small changes. The accuracy of the current methods originates from a dedication to, and enthusiasm for, the pursuit of the optimal soccer injury surveillance methods. The time demands of these methods may prove prohibitive

if a wider, multi-club study was proposed. However, some of the findings of the current studies would suggest that methods such as accurately timing match exposure could be substituted with estimating match exposure as 95 minutes. It was shown in study 1 that the mean duration of a division one soccer match was 95 minutes and not the 100 minutes commonly used to estimate match exposure (Hawkins and Fuller, 1999; Rahnama *et al.*, 2002; Yoon *et al.*, 2004). This simple development will improve the accuracy of IFR by ~5%, and if adopted universally, would improve the validity of inter-study comparisons.

Aim 2: To analyse the findings and implement further investigative studies to identify areas of risk which may predispose players to injury.

This aim was achieved through the completion of studies 4 and 5. In study 4 it was observed that injuries of >14 d duration resulted in performance decrements in aerobic fitness, agility and RSA in soccer players. Study 5 identified reductions in the intensity of exercise during rehabilitation training, especially during early rehabilitation, as a possible causative factor in these performance decrements.

Implications

The findings of these two studies have substantial implications for the work of physiotherapists and sports rehabilitators. Detraining and 'loss of match fitness' due to injury has been the subject of much debate and little research over the past decades. Evidence that a previously injured player had lost match fitness was based upon the assumption that a player 'must have lost fitness because he hasn't trained with the squad' or 'looks to be off the pace in a match' has been based on uninformed conjecture and subjective observations. These studies show that some components of fitness are robust to changes in habitual training and that it may be possible to eliminate detraining by introducing maintenance work early in an injured player's rehabilitation. They also show that the cardiovascular demands of normal squad training can be replicated by using non soccer-specific training modalities. The over-riding message from these studies is that rehabilitators should take every step to start cardiovascular work as early in a player's rehabilitation as possible.

The results of study 4 suggested that competitive match play formed a significant proportion of a player's weekly training load and that the intensity of match play could not be replicated on the training pitch during squad training or during rehabilitation. It was proposed that the inability to train at match intensity during rehabilitation may lead to a loss of some components of fitness. Although addressing this limitation during rehabilitation would not be feasible, the finding did highlight a possible area of concern for uninjured players. It was proposed that players who were not injured but did not participate in competitive matches due to being substitutes in first team matches and not playing in reserve team matches, may experience detraining due to insufficient training stimulus. This may leave these players prone to injury and it would therefore be prudent for those responsible for the fitness of players to pay special attention to monitoring the fitness of players whose participation in competitive matches is limited.

Aim 3: To implement interventions aimed at reducing the detrimental effects of injury at one professional soccer club.

This aim was achieved through the completion of study 6. In study 6 it was observed that an intermittent exercise intervention, in which the intensity of training was frequently elevated, was no more effective that a continuous steady-pace training programme during early rehabilitation in maintaining an injured player's aerobic power and RSA.

Implications

The methods employed in this study meant that injured players began moderate intensity rehabilitation work as soon as was possible considering the particular pathologies. The result of this early activity was that injured players were able to maintain fitness levels in components of fitness that were considered important for success in soccer. It may be that rehabilitators in professional soccer are reluctant to risk aggravating an injury by prematurely exposing the player to demanding training protocols. The levels of ingenuity demonstrated to circumvent exposing injured players to risk whilst facilitating the completion of cardiovascular exercise is not known. Techniques such as one-leg cycling and rowing can be utilised in early rehabilitation even when the patient has sustained a significant lower-limb injury. This study demonstrated that training to a moderate

intensity in early rehabilitation can prevent detrimental physiological adaptations and should be adopted at every opportunity.

6.2 General discussion of findings

The studies undertaken in this thesis were designed to investigate both the injury and rehabilitation process in professional soccer. The outcome of this investigation was to attempt to ascertain reasons for ineffective rehabilitation and to design interventions that may reduce the effects of injury on performance. Before this could be undertaken, a robust and comprehensive method of accurately recording injuries had to be designed and implemented.

At the time of conception of this thesis and early data collection, the methods of collecting epidemiological data in professional soccer were many and varied with no consensus as to the type of data to be collected and/or the methods for its collection. This was recognised by researchers from the Fédération Internationale de Football Association Medical Assessment and Research Centre. They suggested that variations in the definitions and methodologies across studies had created differences in the results and conclusions obtained from the data. A group of researchers recognised as the world's leading authorities in this field were therefore brought together with an aim to establish a consensus on definitions and methodology and implementation and reporting standards for investigations into the epidemiology of injuries in soccer. This culminated in the 2006 consensus statement, published by Fuller *et al.* (2006). The current data collection methods used in this thesis appear to meet the methods outlined in this consensus statement. It could also be argued that they extend these proposed strategies by collecting additional data such as the precise timing of exposure and accurately timing injury occurrence. As a result, we believe that the methods used throughout this thesis are amongst the most comprehensive and accurate records of injury that have been attempted in this research area. These methods are, however, very time intensive and require first-hand attendance by the researcher at all training sessions and attendance at all first team and reserve team matches. This commitment is probably beyond the scope of the majority

of researchers investigating the incidence of injury at a single club. This may make them less appropriate for investigations that seek to investigate the incidence of injury across a number of soccer teams.

It is also acknowledged that studies 2 and 3 were limited to a single professional soccer club. Although a number of studies previously published have utilised one soccer club, these have tended to be older studies in the literature (Lewin, 1984; McGregor and Rae, 1995). More recent epidemiological studies have attempted to gain a broader picture of the incidence of injury by undertaking multi-club studies (Hawkins *et al.*, 2001; Junge *et al.*, 2004). It is acknowledged that one-club studies may lead to problems in the validity of the data as the specific circumstances that lead to the injury pattern observed at one club in one country may not be similar to that observed in other clubs in either the same or different countries. However, the improved study control of a one-club study may partially address any methodological limitations that are associated with such a specific focus. It is well known that relying on third party, study detached recruits to collect such comprehensive data results in a loss of study control and therefore inaccurate returns due to unreliable data collection (Twellaar *et al.*, 1996). This is discussed in Chapter 2.1. The tighter methodological control of the epidemiological data in the current thesis may therefore reflect more accurate results but within a more restricted population. The 5-season duration of the study and the number of observed injuries do, however, probably make an interpretation of the data presented in this thesis generalisable to a wider population. This will particularly be the case if these generalisations are to other English clubs playing at the same competitive level.

The effect that injury has on a professional soccer player's fitness has received limited attention in the literature. Such an area of research is very important, especially in applied practice, as maximising playing time for all of the players within the club is probably an important factor in determining the success of the team. An applied "real world" research model was adopted in this thesis as this approach was considered to provide the "best fit" for the nature of the specific research questions under consideration. This approach, while advantageous in a range of areas also has some inherent drawbacks.

These are frequently associated with a lack of rigorous experimental control. It is acknowledged that at times in this thesis a more experimental approach may have provided some additional insights. The potential for this extra information should not, however, be included at the expense of a loss of relevance to the football world both in relation to the methodologies that are employed and the knowledge that is created. As such the experimental procedures here represent an individual's attempt to adopt an effective strategic position on the "applied" to "basic" research continuum.

Physical inactivity as a result of injury will lead to physiological adaptations that will ultimately reduce an individual's physical performance. Obtaining specific data on such physiological responses to de-training (e.g. enzyme concentrations) was not practical for the subject population under consideration. As a consequence Study 4 utilised field tests to investigate the effects of detraining on fitness indices considered important for success in soccer as these can provide a suitable proxy for the underlying physiology. These indices included aerobic power, RSA, 20m sprint speed and countermovement jump height. The inability to find evidence of reductions in fitness until periods of de-training were < 15 d may partly be related to the methodological approach that was utilised, more specifically the sensitivity associated with some of the field tests. An inability of the selected tests to detect subtle changes in performance clearly has the potential to lead to erroneous conclusions related to the impact of injury on fitness. .

The RSA assessment included in this thesis provide a good specific example of some of the sensitivity issues highlighted above. The detailed discussion of this specific test here will hopefully illustrate important considerations that could be of relevance to other testing parameters included in the experimental chapters. The RSA test was first used in Study 4 to investigate the changes in the ability of players to perform repeated sprints following injury. As part of the process of designing the methods for Study 6, methods utilised in Study 4 were examined. It was decided to change a number of the test variables in light of literature published after Study 4 was completed. For example, the formula utilised to quantify fatigue (Baker *et al.*, 1993) was found to be less reliable than other formulae as a consequence of the specific sprint bouts that were used in the

calculation (i.e. 2 quickest and 2 slowest sprint times ,Glaister *et al.*, 2007). It was also considered that the short (10 s) turn around may have encouraged early deceleration in order that players could get back to the starting point in time for the start of the next sprint (Lakomy and Haydon, 2004).

The reliability of quantifying fatigue has been shown to be poor (Glaister *et al.*, 2008). The method utilised in the current study is one of the most reliable methods but was susceptible to the small changes in the player's quickest sprint times. The level of change in performance required before a 'real' change can be observed to have occurred is also substantial. To compare within-subject changes in RSA, a method which combines changes in the range of individual sprint times and/or total time is required. For example, a player could perform 6 sprints in times of 6; 6.2; 6.4; 6.6; 6.8; 7 s with an RSA_{PD} of -9% using current methods. Following injury the same player can produce 6 sprints in times of 8; 8.2; 8.4; 8.6; 8.8; 9 s with an RSA_{PD} of -7%. This would suggest that he is better able to cope with the fatigue associated with repeated sprints following de-training. This would clearly make little physiological sense in light of the available data. A formula to calculate performance decrement therefore has to incorporate either a multiplier based on total time or quickest time for it to be useful in monitoring changes in RSA.

As well as improving the reliability of fatigue formulae, investigating the different components of RSA (acceleration, maximum velocity, deceleration) may have revealed which components, if any, were affected to a greater extent than others. A laser speed-gun (Laveg LDM 300C) may therefore have helped identify if any section of the 40 m sprints were more affected by fatigue. That is to say, the laser gun would have been able to accurately record acceleration over the first 5 m for example and this could have been compared across the 6 sprints to determine if acceleration was affected by fatigue. The data from a laser speed-gun may have added a valuable variable to the analysis of players' repeated sprint ability. No RSA studies can be found which have utilised this piece of equipment. Future RSA studies may therefore benefit from investigating which component of short sprints is most affected by fatigue.

One possible potential strategy to improve the accuracy of the physiological test procedures used in this thesis would be to adopt laboratory-based assessment models rather than those in the field-based. These strategies would provide the additional benefits of greater control of the extrinsic factors that could influence the data and also enable the application of more detailed methodological techniques. The use of laboratory-based tests was considered throughout relevant studies in this thesis. Initially it was intended to use laboratory based tests to evaluate both $\dot{V}O_{2max}$ and RSA in Study 6. Several considerations resulted in the proposed tests being changed to field tests. All members (n=26) of the professional squad included in the study were required to be tested prior to the beginning of both seasons covered by the study. The organisational restrictions associated with lab testing in relation to time, particularly with regard to familiarisation, was prohibitive. It was however, considered that this could be overcome with familiarisation sessions taking place prior to the commencement of pre-season. It then became evident that more familiarisation sessions were required for the laboratory based RSA non-motorised treadmill protocol when using the on-line gas analyser system (Medical Graphics, CPX/D) as players' performance appeared to be significantly affected by the wearing of the tight face mask when comparing against performance without it. The non-motorised treadmill was also mechanically unreliable. The hardware/software interface caused the software programme to freeze during 75% of all tests. One player started 8 RSA tests and was unable to complete any as a result of this computer problem. Taking all of these issues into consideration, resulted in a decision to continue with the use of strictly controlled field-tests. If similar future research required laboratory tests, it may be more appropriate to test injured players at the point at which they return to training following injury and then test the same players again when they had been training normally and were injury free for a predetermined time. This would reduce the number of laboratory tests required and reduce the demands of testing the whole squad.

Another possible reason for the absence of change in players' performance assessments may have been the limitations associated with the scheduling of the physiological assessments used in the investigation. The physiological evaluations during study 6 took

place when the players were fit and playing competitive soccer and again just prior to them returning to squad training following injury. These testing sessions did not allow for a clear determination of the time scale of possible reductions and improvements in fitness as a consequence of injury and rehabilitation to be determined. A test to determine a player's fitness at the end of the early rehabilitation phase would have provided this information and would therefore have been a valuable addition to the current methodology. The complexities of the various pathologies however, make it difficult to timetable maximal tests at such strategic points throughout a player's rehabilitation. A reliable test of fitness, that did not require a maximal effort, may therefore have been a suitable addition to the protocols here. Clearly the development and validation of such tests is difficult and would require substantial research.

The above paragraphs clearly highlight some of the potential limitations that are associated with adopting a more "applied" approach to the generation of knowledge. A number of these issues seem to relate to the specific methodological procedures, and their ability to generate specific types of data, that can be employed in non-laboratory based investigations. Such factors were also considerations for the studies completed in this thesis that required the determination of individual player training loads. The procedures that were used for data collection needed to be relatively simple and unobtrusive as they had to be functional during normal squad training as well as during rehabilitation sessions. Both the frequency and duration of training sessions were recorded to enable exposure to training to be calculated. Heart rate data was also collected to give an indication as to the physiological stress of each discrete training session. While these approaches provide us with a range of useful information as to the training load that players completed they do permit us to gain a full understanding of the specific external and internal loading placed on individuals. While it could be argued that such a "complete" picture of an individual's training is impossible to obtain modern technological advances such as Global Positioning Satellite (GPS) may have provided additional information. At the start of the current series of studies, GPS technology was still in its infancy and has only recently been found to be reliable (Hill-Haas *et al.*, 2008). This technology, if applied in the current studies, would have enabled distance covered

and average velocity to be recorded for all outside, running-type training. This would have added a valuable indictor of the external training load of soccer players and enabled further comparison between rehabilitation and normal training. Other methods of quantifying the physiological response to exercise were considered such as portable gas analysers, but were considered inappropriate for the study. The inclusion of such methods in future investigations would therefore seem to represent an important area for progression in research design.

The appropriateness of the specific training loads used to prescribe exercise interventions also seem of relevance to the areas within this thesis that attempted to modify the rehabilitation process. In study 6, the intervention was based on a maximum intensity of 75-80% HR_{max}. This intensity was decided following pilot work in which the highest exercise intensity that could be used for the early stages of rehabilitation was evaluated. It was not envisaged however, that this would result in players in both SPG and IPG maintaining aerobic power performance. Study 4 and study 5 had suggested that early rehabilitation training at an intensity of ~65% HR_{max} resulted in performance decrements in $\dot{V}O_{2max}$ and RSA. A constant steady-pace protocol at ~65% HR_{max} and a work-matched intermittent protocol may therefore have been more suitable in exploring whether the intermittent protocol was more effective as it would have been expected that the steady-pace protocol would have resulted in performance decrements as found in Study 4. Future studies should explore the maximum intensity of exercise that a player is able to perform that results in performance decrements as a result of detraining and then use an intermittent protocol that is work-matched to the same intensity as the constant steady-pace protocol. These methods will then properly evaluate the intermittent protocol.

6.3 Recommendations for future research

There are several potential areas of future research which have become apparent as a consequence of critically analysing the experimental chapters in this thesis. These are outlined below:

6.3.1 *Initiate a multi-club, multi-level epidemiological longitudinal study with the aim of elucidating an accurate IFR and the precise aetiological factors of injury in soccer.*

The findings of the current thesis suggest that previous investigations into soccer injury may not be accurate as a result of methodological weaknesses. These weaknesses include limited aetiological information, questionable data collection methods and ambiguous exposure data. A longitudinal study using data collectors who share a common desire to produce research of the highest standards would produce a piece of epidemiological research the accuracy and wide ranging implications of which have not been seen previously. These data collectors will also recognize the value of gathering data in improving their own practice and, as such, will take ownership of the research.

6.3.2 *Investigate the injury frequency rate of players who are not injured but play limited competitive soccer matches.*

As a result of club or manager policy, some soccer clubs do not play senior players in reserve team matches. Some of these players will occupy the substitute's bench during first team matches yet seldom enter the field of play. It has been suggested that players who do not include the training load of competitive matches in their weekly training load may experience detraining which may leave them susceptible to injury. It may therefore be sensible to investigate the possibility of these players being at increased risk of injury during training. If this was found to be the case, additional training could be prescribed to alleviate this increase in risk.

6.3.3 *Quantifying fatigue in RSA test protocols.*

The various formulae employed to quantify RSA fatigue are known to be unreliable leading to researchers questioning their value (Glaister *et al.*, 2008). A test of repeated

sprint ability without a measure of fatigue is however, a sprint test. Total time can be utilised to give an indication to how quickly a player is able to complete a number of sprints of a set distance. However, this variable gives no indication as to the pattern of performance each player produces. For example, player 1 performs 6 sprints in 6,6,6,6,6 and 6 s giving a total time of 36 s. Player 2 performs 6 sprints in 5,5,6,6,7 and 7 s also giving a total time of 36 s. Total time does not allow for differentiating between the two quite obviously different performance patterns. An indicator of fatigue is therefore vital when investigating RSA. Further work should investigate the construction of an RSA fatigue formula that is reliable and valid. This may be based on total time or quickest time but a criterion for an acceptable pattern of sprint times should also be considered. That is to say, the data of a subject producing a quickest sprint time in the final sprint of a series should not meet the criteria for using the formula.

6.3.4 *Identifying which components of RSA sprints are most affected by fatigue.*

No research can be found which explores the possibility that some components of RSA may be differently affected by fatigue than others. During repeated sprints, it may be that, as a factor of sprint number, acceleration is affected more than either maximum velocity or the maintenance of maximum velocity. These parameters may also be affected differently for sprint distances of varying lengths. Identifying which components are most affected may enable training protocols with the aim of addressing these weaknesses to be developed.

6.3.5 *Determine a minimum intensity of steady state exercise required for injured soccer players to maintain levels of aerobic power.*

When professional soccer players are injured, every consideration has to be given to the particular pathology when designing the rehabilitation programme. One of the aims of rehabilitation is to maintain cardiovascular fitness, usually using non-running exercise modes. The frequency, duration and intensity of this work is generally based on empirical experience as no research can be found which indicates the minimum training load required to maintain aerobic power under these conditions. A piece of research which indicated that to maintain aerobic fitness an injured professional soccer player would

need to perform steady-pace cardiovascular exercise; f x per week; for d x minutes; at i x intensity would have wide ranging implications for the sport.

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Appendices

Tranmere Rovers F.C. Players' Attendance Register

Date		Day	M T W T F S S			
	AM		PM		EVE	
	Code	Exposure	Code	Exposure	Code	Exposure
Player 1						
Player 2						
Player 3						
Player 4						
Player 5						
Player 6						
Player 7						
Player 8						
Player 9						
Player 10						
Player 11						
Player 12						
Player 13						
Player 14						
Player 15						
Player 16						
Player 17						
Player 18						
Player 19						
Player 20						
Player 21						
Player 22						
Player 23						
Player 24						

TRAINED	T
INJURED	I
SICK	S
PERSONAL	P
OTHER	O
SUSPENDED	B
1st Team match	1
Reserve match	2
Other match	3

Comments

Appendix 1

Ratings of Perceived exertion

0 Rest

1 Very, very easy

2 Easy

3 Moderate

4 Somewhat hard

5 Hard

6

7 Very hard

8

9

10 Maximal

Appendix ii

Tranmere Rovers Football Club

Dear

Hope you are well. I am completing a study at Liverpool JMU looking at the difference between normal training and rehabilitation. Ultimately I want to discover why players lose fitness when they are injured. As part of this study, I need to give an indication of how hard rehabilitation training is compared to other clubs. I would therefore ask that you complete the tear off section of this page and send it back to me as soon as possible. I have not asked you to identify yourself on the sheet as I feel that this will result in a more accurate reply.

Take care

Les

How many seasons were you at Tranmere? ---------

Apart from Tranmere, how many
other clubs *have you played* at? ---------

On a scale of 1-10 please compare the difficulty of rehabilitation training at Tranmere Rovers with your other clubs.

A score of **5 to 6** indicates that it was, on average, the same intensity as other clubs.

A score from **1-4** indicates that it was generally easier than other clubs

A score of **7-10** indicates it was more difficult

LIVERPOOL JOHN MOORES UNIVERSITY

FORM OF CONSENT TO TAKE PART AS A SUBJECT IN A RESEARCH PROJECT

Title of project/procedure: Is injury the major factor for absence in professional soccer?.

I, ...agree to take part in the above
(Subject's full name)*

named project/procedure, the details of which have been fully explained to me and described in writing.

Signed ..... Date.....
(Subject)

I, LES PARRY certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed ..... Date.....
(Investigator)

I, SHAUN GARNETT certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed..... **Date** .....
(Witness)

NB The witness must be an independent third party.

* Please print in block capitals

Appendix iv

Title of Project: A prospective epidemiological study of injuries at one English professional soccer club over 5 competitive seasons.

Name of Researcher and School/Faculty: Les Parry
The Research Institute for Sport and Exercise Sciences (Rises).

You are being invited to take part in a research study. Before you decide it is important that you understand why the research is being done and what it involves. Please take time to read the following information. If there is anything that is not clear or if you would like more information please contact Les before proceeding further. Take time to decide if you want to take part or not.

1. What is the purpose of the study?
The purpose of the research is to investigate the reasons for injury and how long players are absent through injury to different parts of their bodies or for different ways of being injured such as running or tackling.

2. Do I have to take part?
You are not obliged to allow me to use your records in this study. If you do take part you will be asked to provide consent where your name will be recorded should you wish to withdraw at a later date. Your involvement is voluntary and you may withdraw at any time. If you decide to take
part you are still free to withdraw at any time without giving a reason.

3. What will happen to me if I allow my records to be included?
This study is an extension to records already kept at the club by Les Parry in his role as the club physiotherapist. Les wants to use the records to investigate patterns of injury. You will not be aware that you are taking part in the study as nothing will change. No additional information to TRFC normal medical records will be kept.

4. Are there any risks / benefits involved?
There are no identifiable risks to taking part in the study.

5. Will my taking part in the study be kept confidential?
If you consent to take part in this research, the information you provide will be kept strictly confidential. Your name and any other personal identifiable information submitted will be kept secure. You are requested to provide the researcher with your name on a dated consent form. This will be kept by the researcher separate from any other information you may provide.

Contact details of researcher
If you have any questions about the research study please contact: Les Parry
Tranmere Rovers F.C., Prenton Park, Birkenhead, Merseyside
Phone: 0151 6084194 ; Email lesp@tranmererovers.co.uk
Appendix v

EC3

LIVERPOOL JOHN MOORES UNIVERSITY

FORM OF CONSENT TO TAKE PART AS A SUBJECT IN A RESEARCH PROJECT

Title of project/procedure: **The effects of detraining on gross motor performance**

I, ...agree to take part in the above
(**Subject's** full name)*

named project/procedure, the details of which have been fully explained to me and described in writing.

Signed ..... Date.....
(Subject)

I, LES PARRY certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed ..... Date.....
(Investigator)

I, SHAUN GARNETT certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed..... **Date** .....
(Witness)

NB The witness must be an independent third party.

* Please print in block capitals

Appendix vi

Title of Project: The effects of detraining on gross motor performance.

Name of Researcher and School/Faculty: Les Parry
The Research Institute for Sport and Exercise Sciences (Rises).

You are being invited to take part in a research study. Before you decide it is important that you understand why the research is being done and what it involves. Please take time to read the following information. If there is anything that is not clear or if you would like more information please contact Les before proceeding further. Take time to decide if you want to take part or not.

1. What is the purpose of the study?
The purpose of the research is to investigate which components of fitness you lose when you are injured and unable to train normally.

2. Do I have to take part?
You are not obliged to allow me to use your records in this study. If you do take part you will be asked to provide consent where your name will be recorded should you wish to withdraw at a later date. Your involvement is voluntary and you may withdraw at any time. If you decide to take part you are still free to withdraw at any time without giving a reason.

3. What will happen to me if I participate in this study?
You will not notice anything different as you will follow a normal rehabilitation programme and perform tests which you are familiar with. Agreeing to participate allows me to use the data I normally collect to investigate how being unable to train affects your fitness.

4. Are there any risks / benefits involved?
There are no identifiable increased risks to taking part in the study.

5. Will my taking part in the study be kept confidential?
If you consent to take part in this research, the information you provide will be kept strictly confidential. Your name and any other personal identifiable information submitted will be kept secure. You are requested to provide the researcher with your name on a dated consent form. This will be kept by the researcher separate from any other information you may provide.

Contact details of researcher
If you have any questions about the research study please contact: Les Parry
Tranmere Rovers F.C., Prenton Park, Birkenhead, Merseyside
Phone: 0151 6084194 ; Email lesp@tranmererovers.co.uk

Appendix vii

EC3

LIVERPOOL JOHN MOORES UNIVERSITY

FORM OF CONSENT TO TAKE PART AS A SUBJECT IN A RESEARCH PROJECT

Title of project/procedure: A quantitative comparison of the training loads of professional soccer players during rehabilitation and normal squad training.

I, ...agree to take part in the above
(**Subject's** full name)*

named project/procedure, the details of which have been fully explained to me and described in writing.

Signed ..... Date.....
(Subject)

I, LES PARRY certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed ..... Date.....
(Investigator)

I, SHAUN GARNETT certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed..... **Date** .....
(Witness)

NB The witness must be an independent third party.

* Please print in block capitals

Appendix viii

Title of Project: **A quantitative comparison of the training loads of professional soccer players during rehabilitation and normal squad training.**

Name of Researcher and School/Faculty: Les Parry
The Research Institute for Sport and Exercise Sciences (Rises).

You are being invited to take part in a research study. Before you decide it is important that you understand why the research is being done and what it involves. Please take time to read the following information. If there is anything that is not clear or if you would like more information please contact Les before proceeding further. Take time to decide if you want to take part or not.

1. What is the purpose of the study?
The purpose of the research is to investigate why players lose fitness when they are injured. This will be done by recording your activity levels when you are training normally and again when you are injured and comparing the two.

2. Do I have to take part?
As such, you are not doing anything different by taking part in the study. If you agree or not, you will follow the normal training and rehabilitation training programmes. You are allowing me to use your training data. You are not obliged to allow me to use your records in this study. If you do take part you will be asked to provide consent where your name will be recorded should you wish to withdraw at a later date. Your involvement is voluntary and you may withdraw at any time. If you decide to take part you are still free to withdraw at any time without giving a reason.

3. What will happen to me if I take part in the study?
You will not realise you are taking part in a study as nothing in your training programme will change. It is important for the study that you train and rehabilitate normally.

4. Are there any risks / benefits involved?
There are no identifiable increased risks to taking part in the study. You will train and rehabilitate normally.

5. Will my taking part in the study be kept confidential?
If you consent to take part in this research, the information you provide will be kept strictly confidential. Your name and any other personal identifiable information submitted will be kept secure. You are requested to provide the researcher with your name on a dated consent form. This will be kept by the researcher
separate from any other information you may provide.

Contact details of researcher
If you have any questions about the research study please contact: Les Parry
Tranmere Rovers F.C., Prenton Park, Birkenhead, Merseyside
Phone: 0151 6084194 ; Email lesp@tranmererovers.co.uk

Appendix ix

EC3

LIVERPOOL JOHN MOORES UNIVERSITY

FORM OF CONSENT TO TAKE PART AS A SUBJECT IN A RESEARCH PROJECT

Title of project/procedure: **Comparing the effects of constant steady-pace and variable intermittent exercise protocols in maintaining aerobic power and repeated sprint ability in injured elite soccer players.**

I, ...agree to take part in the above
(**Subject's** full name)*

named project/procedure, the details of which have been fully explained to me and described in writing.

Signed ..... Date.....
(Subject)

I, LES PARRY certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed ..... Date.....
(Investigator)

I, SHAUN GARNETT certify that the details of this project/procedure have been fully explained and described in writing to the subject named above and have been understood by him.

Signed..... **Date** .....
(Witness)

NB The witness must be an independent third party.

* Please print in block capitals

Appendix x

Title of Project: Comparing the effects of constant steady-pace and variable intermittent exercise protocols in maintaining aerobic power and repeated sprint ability in injured elite soccer players.

Name of Researcher and School/Faculty: Les Parry
The Research Institute for Sport and Exercise Sciences (Rises).

You are being invited to take part in a research study. Before you decide it is important that you understand why the research is being done and what it involves. Please take time to read the following information. If there is anything that is not clear or if you would like more information please contact Les before proceeding further. Take time to decide if you want to take part or not.

1. What is the purpose of the study?
The purpose of the research is to find out if we can prevent or reduce you losing fitness when you are injured.

2. Do I have to take part?
You are not obliged to allow me to use your records in this study. If you do take part you will be asked to provide consent where your name will be recorded should you wish to withdraw at a later date. Your involvement is voluntary and you may withdraw at any time. If you decide to take part you are still free to withdraw at any time without giving a reason.

3. *What* will happen to me if I agree to participate?
Your performance during *rehabilitation will* be recorded, this happens irrespective of your inclusion in the study but you are allowing me to use *this data to* investigated if steady pace or intermittent work is better for maintaining fitness.

4. Are there any risks / benefits involved?
There are no identifiable increased risks to taking part in the study.

5. Will my taking part in the study be kept confidential?
If you consent to take part in this research, the information you provide will be kept strictly confidential. Your name and any other personal identifiable information submitted will be kept secure. You are requested to provide the researcher with your name on a dated consent form. This will be kept by the researcher separate from any other information you may provide.

Contact details of researcher
If you have any questions about the research study please contact: Les Parry
Tranmere Rovers F.C., Prenton Park, Birkenhead, Merseyside
Phone: 0151 608 4194 ; Email lesp@tranmererovers.co.uk

Appendix xi