An Examination of the Role of the Sports and Exercise Medicine Specialist in Ireland- from Epidemiology to Treatment

---

Éanna Falvey

October 2013

This thesis is submitted for a PhD degree in Medicine
from the National University of Ireland, University College Cork,
School of Medicine and Health.

Supervisor: Professor Michael G Molloy
Head of Department: Professor Fergus Shanahan

Table of Contents

List of Tables

List of Figures

List of Appendices

List of Abbreviations

AOG: airflow obstruction group

BASEM: British association of sports and exercise medicine

BF: Biceps femoris

BHR: bronchial hyper-reactivity

BMI: basal metabolic index

BPM: beats per minute

CI: confidence interval

CO: Colorado

CO_2: carbon dioxide

EBM: evidence based medicine

ECG: electrocardiogram

ED: Emergency Department

EIA: Exercise Induced Asthma

EIB: Exercise Induced Bronchoconstriction

EVH: eucapnic voluntary hyperventilation

FEV_1: forced expiratory volume in 1 second

FIFA: Fédération Internationale de Football Association

FVC: forced vital capacity

GAA: Gaelic athletic association

HIP: A test used in the study where there is a flexed knee, hip flexion and adduction, with the examiner controlling for external loading

HRmax: maximum heart rate

IOC: International Olympic Committee

IQR: Interquartile range

IRB: International Rugby Board

ITB: iliotibial band

ITBS: iliotibial band syndrome

ITBFS: iliotibial band friction syndrome

Kg: kilogram

LFC: lateral femoral condyle

MA: Massachusetts

MVV: maximum voluntary ventilation

NFL: National Hockey League

NAOG: non-airflow obstruction groups

NCAA: National Collegiate Athletic Association

OBER: Ober's test

OECD: Organization for Economic Co-operation and Development

OR: Odds Ratio

PD15: provoking dose (of mannitol) causing a fall in FEV_1 of 15%.

SD: standard deviation

SEM: Sports and Exercise Medicine

SLR: Straight Leg Raise

SRI: Sports and Recreational Injuries

TFL: Tensor Fasciae Latae

U.S.: United States of America

U.S.A.: United States of America

VL: Vastus Lateralis

WADA: World Anti-Doping Agency

WHO: World Health Organization

YIET: the Yoyo intermittent endurance test

Declaration

This thesis is submitted to University College Cork in accordance with the requirements for the degree of Doctor of Philosophy (PhD) in the Faculty of Medicine.

I declare that this thesis is a record of my own work and has not been submitted for any other academic award. All information sources have been fully acknowledged and referenced.

Parts of this work have appeared in the following peer reviewed publications and presentations:

1. **Falvey EC,** Eustace J, Whelan B, Molloy MS, Cusack SP, Shanahan F, Molloy MG. Sport and recreation-related injuries and fracture occurrence among emergency department attendees: implications for exercise prescription and injury prevention. Emerg Med J. 2009 Aug; 26(8):590-5. doi: 10.1136/emj.2008.062315
2. **Falvey EC,** McCarthy C, O'Connor TM, Shanahan F, Molloy MG, Plant BJ. Exercise-induced bronchoconstriction and exercise testing in an international rugby union team. Thorax. 2010 Sep;65(9):843-4.
3. **Falvey E,** McCrory P, Crowley B, Kelleher A, Eustace J, Shanahan F, Molloy MG. Risk factors for hand injury in hurling: a cross-sectional study. BMJ Open. 2013 May 28;3(5).

4. **Falvey EC,** Clark RA, Franklyn-Miller A, Bryant AL, Briggs C, McCrory PR. Iliotibial band syndrome: an examination of the evidence behind a number of treatment options. Scand J Med Sci Sports. 2010 Aug; 20(4):580-7. Epub 2009 Aug 23.
5. Delaney RA, **Falvey E**, Kalimuthu S, Molloy MG, Fleming P. Orthopaedic admissions due to sports and recreation injuries. Ir Med J. 2009 Feb; 102(2):40-2.
6. Clark RA, Franklyn-Miller A, **Falvey E**, Bryant AL, Bartold S, McCrory P. Assessment of mechanical strain in the intact plantar fascia. Foot (Edinb). 2009 Sep; 19(3):161-4.
7. Franklyn-Miller A, **Falvey E,** McCrory P. Fasciitis first before tendinopathy: does the anatomy hold the key? Br J Sports Med. 2009 Dec; 43(12):887-9.

Published abstracts following oral presentation:

1. **Falvey EC,** McCarthy C, O'Connor TM, Shanahan F, Molloy MG, Plant BJ. Exercise-induced bronchoconstriction (EIB) and asthma after field-testing in an International Rugby Union Team. Thorax 2008;63:A18-A21 S39
2. **Falvey EC**, Molloy MS, Whelan B, Cusack SP, Shanahan F, Molloy MG. Promoting safe Sports Participation in Ireland: Results of a Prospective Pilot Study in the Emergency Department. Rheumatology (April 2006) 45 (suppl 1): i191-i197

3. **Falvey EC,** Molloy MS, Whelan BR, Cusack SP, Shanahan F, Molloy MG. Field Sports Result in Fewer Serious Sporting Injuries in Children than Play or Active Recreation: Falvey. Medicine & Science in Sports & Exercise. 38(5):S346, May 2006.
4. **Falvey EC,** Eustace J, Whelan B, Molloy MS, Cusack SP, Molloy MG. Sports and recreation injury, from the playing field to the home; out of the frying pan and into the fire. Br J Sports Med 2008;42:491-548
5. **Falvey EC,** Crowley B, Eustace J, Molloy MG. When is wearing a helmet bad for your health? Hurling injuries and risk compensation. Br J Sports Med 2008;42:491-548

____

Éanna Falvey

October 2013

Acknowledgements

Firstly I would like to thank my supervisor, Professor Michael G Molloy, for mentoring my journey all the way from internship to gaining specialty recognition in sports and exercise medicine. Without your support this journey could not have happened- your enthusiasm, inexhaustible energy and unfaltering belief are an example for all to behold.

I have met many people along this journey who have in their own way made it possible - far too many to thank individually - I owe a debt of gratitude to them all.

Thank you to all of the patients and athletes who generously shared their time and energy over the course of this work.

Thank you to my fellow sufferers in the early days, Mary Daly, Miriam O'Sullivan, Bryan Whelan and all at the department of Rheumatology at CUH. To Professor Joseph Eustace who clarified the unintelligible.

Sinead Kinsella has been a companion from the very start and a champion at the very end she has proven an example in triumph over adversity and has always been a true friend. Thank you for your work, expertise and patience.

To Paul McCrory, Peter Brukner and Andy Franklyn-Miller- you have shown me that if you wish to do something the only person who can really stop you is yourself.

I am grateful to my parents Anna and Jeremiah who fostered a child's thirst for knowledge and gave incessant questions time and attention; they encouraged a pride in hard work and achievement and always believed in me- even when I did not. Thank you to my family Diarmaid, Aoife, Colm and Sinéad who have been my back up from the very start. And my grandfather Tom McCarthy, who told me to be myself.

Thank you to my girls Ava and her enquiring mind, Elsa and Lola who may now have the nappy office back. You all make the hard work worthwhile.

Finally to Nina, you have sacrificed more than anyone to allow this to happen, you are my best friend and supporter. Your quiet confidence has calmed many troubled waters and I would not have completed this work without your love and fortitude.

“If we knew what we were doing, it would not be called research, would it?”

Albert Einstein

Abstract

The expansion of the specialty of sports and exercise medicine (SEM) is a relatively recent development in the medical community and the role of the SEM specialist continues to evolve and develop. The SEM specialist is ideally placed to care for all aspects of physical activity not only in athletes but also in the general population. As an advocate for physical activity the SEM specialist plays a broad role in advising safe effective sports and recreation participation; screening for disease related to sports participation; examining and contributing to the evidence behind treatment strategies and evaluating any potential negative impact of sports injury prevention measures. In this thesis I will demonstrate the breadth of the role the Sports and Exercise Medicine Specialist from epidemiology to in-depth examination of treatment strategies.

In Chapter 2, I examined the epidemiology of sports and recreation related injury (SRI) in Ireland, an area that has previously been poorly studied. We report on 3,172 SRI (14% of total presentations) presentations to the ED over 6 months. Paediatric patients (4-16 yrs) were over represented comprising 39.9% of all SRI presentation compared to 16% of total ED presentations and 18% of the general population. These injuries were serious (32% fractures) and though 49% of injuries occurred during organised competition/practice, 41.5% occurred during recreation- most often at home.

In Chapter 3, I examined risk factors associated with hand injury in hurling. When measures to improve welfare are introduced not only must potential benefits be measured, so too must potential unwanted adverse outcomes. In this study I examined a cohort of adult hurlers who had presented to the ED with a hurling related injury in order to highlight the variables associated with hand injury in this population. I found the athletes who wore a helmet were far more likely (OR 3.15 95% CI (1.51-6.56) p= 0.002) to suffer a hand injury than athletes who did not. Very few of those interviewed (4.9%) used hand protection compared to 65% who used helmet and faceguard. The introduction of the helmet and faceguard in hurling has undeniably decreased the incidence of head and face injury in hurling. However in tandem with this intervention several observational studies have demonstrated an increase in the occurrence of hurling related hand injuries. This study highlights the importance of being cognisant of unanticipated or unintended consequences when implementing a new treatment or intervention.

In Chapter 4, I examined the role of population screening as applied to sport and exercise. This is a controversial area - cardiac screening in the exercising population has been the subject of much debate. Specifically I define the prevalence of exercise induced bronchoconstriction (EIB) using a specifically designed sports specific field-testing protocol. In this study I found almost a third (29%) of a full international professional rugby squad had confirmed asthma or EIB, as compared with 12-15% of the general population. Despite regular medical screening, 5 'new' untreated cases (12%) were elicited by the challenge test and in the group already on treatment for asthma/EIB; over 50% still displayed EIB.

In Chapter 5, I examined the evidence supporting current treatment options for iliotibial band friction syndrome (ITBFS). The practice of sports medicine has traditionally been 'eminence based' rather than 'evidence based'. This may be problematic as some of these practices are based upon flawed principles- for example the treatment of iliotibial band friction syndrome (ITBFS). In this chapter, using cadaveric and biomechanical studies I expand upon the growing base of evidence clarifying the anatomy and biomechanics of the area- thereby re-examining the principles on which current treatments are based.

The role of the SEM specialist is broad; I chose to examine specific examples of some of the roles that they execute. An understanding of the epidemiology of SRI presenting to the ED has implications for individual patients, sports governing bodies and health resource utilisation. Population screening is an important tool in health promotion and disease prevention in the general population. Screening in SEM may have similar less well-recognised benefits. The SEM specialist needs to be conversant in screening for medical conditions concerning physical activity. A comprehensive understanding of the pathophysiology of a disease is required for its diagnosis and treatment. Due to the ongoing evolution of

SEM many treatments are eminence-based rather than evidence-based practice. Continued re-examination of the fundamentals of current practice is essential. An awareness of potential unwanted side effects is essential prior to the introduction of any new treatment or intervention. The SEM specialist is ideally placed to advise sports governing bodies on these issues prior to and during their implementation.

Chapter 1

Background

1.1 Introduction

This work was conceived to examine the role of the specialty trained sport and exercise medicine specialist in the care of the active population- those who are, those who aspire to be and those who should aspire to be! This role has evolved over the last 30 years. Organization for Economic Co-operation and Development (OECD) figures show that 13% of Irelands population are obese (BMI >30)- though significantly lower than our closest neighbours the United Kingdom (23%), this rate is expected to rise by a further 10% over the next 10 years.[1] The role of the specialist in exercise medicine therefore has expanded beyond injury management to a pivotal one in the management of the obesity epidemic gripping our country and the developed world.

Prevention is better than cure. Physical activity, either occupational or recreational is directly linked to cardiorespiratory fitness. Cardiorespiratory fitness is directly proportional to reduced mortality and improvements in metabolic function, body composition, hemodynamics, musculoskeletal, and psychological functioning- conclusively proven by the work of Archer et al.[2]

The sedentary lifestyle of the population of the western world is directly related to increasing levels of cardiovascular disease.[3] Even modest amounts of exercise have been shown to mitigate against proven risk factors such as smoking, hypertension and hypercholesterolaemia.[3] One of the roles of the physician in modern society is to implement physical activity as a treatment similar to, and no less important, than medication or surgical intervention. None of these facts are disputed, however despite the sobering levels of physical inactivity reported recently in Ireland[4] levels of societal rates of physical activity have increased dramatically since the 1950s.[5] Governmental public health policy has directed a good deal of attention toward further increasing physical activity rates with disappointingly modest results. Despite the boost to sport of the London 2012 Olympics physical activity in London rose by only 0.4% between 2005-06 and 2010-11.[6] In fact the London Organising Committee of the Olympic Games set a target at their bid for the Olympic Games in Singapore in 2005. The committee pledged, "by 2012 two million more people would be physically active, a million more people would be playing sports regularly, and 60% of young people would be doing at least five hours of sport per week." It remains to be seen if these targets are even approached- given the mixed pattern of the long term effect of hosting the summer Olympic games in Barcelona, Atlanta, Sydney and Athens- it appears to have been more aspirational than realistic.[7] The London bid was unique however in that it aspired to improve physical activity levels- with governmental backing and no small investment (£162m a year) the benefits were modest. This does not appear to be the most effective means of encouraging physical activity.

Perhaps world sports governing bodies are the answer? The IOC itself published a very comprehensive document in 2011 outlining not only guidelines for physical activity but also some of the means of achieving them.[8] It highlights the role of the WHO, governments, sporting bodies and national educational systems in embedding physical activity not only in the curriculum of schools but in the psyche of the population. Individual sports bodies probably represent a more effective means of capturing the hearts and minds of the general population. The Fédération Internationale de Football Association (FIFA) have very successfully launched the FIFA 11 programme - a means of improving player conditioning and preventing injury. This has been expanded recently to the “FIFA 11 for health” aimed at promoting health and physical activity in the population through the ambassadorship of the sports’ biggest stars.[9]

This laudable approach must be recreated and evolved in Ireland. I have personally seen children forbidden from any physical activity during lunch break in primary school in Ireland. This includes running. An apparent fear of litigation is one of the reasons but we are preventing our children from easy access to physical exercise with their peers - in the winter during the only daylight hours available. This will only be changed when government and educational department guidelines and regulations change.

The role of the sports and exercise medicine specialist is to facilitate this namely by;

- Identifying the most appropriate activities for individuals and populations.
- Identifying activities or behaviours during physical activity which may expose the participant to injury or illness.
- Appropriately preventing injury or illness associated with physical activity.
- Treating illness or injury arising from physical activity in an evidence-based manner.
- Assisting sports governing bodies in the application and development of safety measures to ensure safe sports participation.

1.2 Evolution of Sports and Exercise Medicine

Claudius Galen (129–c200 AD) was arguably the first professional sports physician. Given his medical training in the great center of learning of Alexandria, and his professional role as a physician for the gladiatorial contests in Pergamum in Asia Minor, he was in an ideal position to consider the anatomical aspects of the injuries he saw in his daily professional life-referring to the often horrific wounds seen as "windows into the body". In his four years as physician to the gladiators of the High Priest of Asia only five deaths occurred compared to sixty deaths during his predecessor’s time.[10]

In more recent history the care of athletes has fallen to a wide range of practitioners across both the medical profession and allied health professionals. The management of orthopaedic sports-related injury has by and large been by the orthopaedic surgical community. Orthopaedic medicine was a term popularized by Dr James Cyriax who taught high level diagnostic skills and joint manipulation aimed at the management of soft tissue musculoskeletal injury.[11,12]

The onset of improved imaging such as diagnostic ultrasound and magnetic resonance imaging has improved the understanding of complex overuse injury such as muscle injury and tendinopathy- which in turn has revolutionised the management of these conditions. These advances, in tandem with improved understanding of the physiology and biomechanics of athletic performance and medical issues surrounding physical activity, have formed the basis for the emergence of the specialist sports physician.

The British Association of Sports and Exercise Medicine (BASEM) was founded in 1953 and the American College of Sports Medicine was founded in 1954. BASEM is “dedicated to the promotion of good health through physical activity and the provision of sports medicine expertise to optimise athletic performance at all levels”. In the 21st century many countries have their own sports and exercise medicine faculty with representation at international bodies such as the European Federation of Sports Medicine Associations (EFSMA) and the International Federation of Sports Medicine (FIMS).

The Faculty of Sports Medicine in Ireland was founded in 2002 and is a joint faculty between the Royal College of Physicians of Ireland and the Royal College of Surgeons in Ireland. The discipline of sports and exercise medicine achieved specialty recognition in 2004. The Faculty of Sports and Exercise Medicine (FSEM) was recognised by the Minister for Health and Children and the Irish Medical Council as a body in the State for the purpose of granting evidence of satisfactory completion of specialist training in Sports and Exercise Medicine.

My own journey to speciality in SEM is a circuitous one. I have always been keenly interested in sport. I competed at club level in hurling and football. While at school I ran cross-country in a national schools winning team. My real love however was for boxing. Commencing aged 9 I had my first bout aged 12 at 36kg. I boxed until the age of 14 at St Colman's BC in East Cork and managed to reach the national championships once. I left boxing to play hurling and football but after consecutive meniscal tears returned to boxing after commencing university in Cork. I won national intermediate championship at heavyweight (<91kg) in 1997 and in 2002 I won the national senior super-heavyweight title tipping the scales at 104kg. I had the honour of competing internationally 7 times including a bout against an Olympic and now professional world champion. As a doctor, during my house officer years I gained my membership of the Royal College of Physicians in Ireland and completed a Masters Degree in SEM in Cork. I chose to follow a career in SEM. Though a faculty existed, no formal training programme was in place and I was fortunate to be helped through training and research by Professor M Molloy and the Irish Centre for Arthritis Research and Education. During this time I worked as a team physician in hurling and football, elite boxing and professional rugby. I attended the University of Melbourne and Olympic Park Sports Medicine Centre in Melbourne to complete a clinical/research fellowship. Here, under the stewardship of Associate Professors Paul McCrory and Peter Brukner and in collaboration with Dr Andy Franklyn-Miller I had a highly successful time teaching anatomy, researching the anatomy of fascia of the lower limb and working with the Geelong Cats Australian Rules football team. This led to the writing and publication of 'Clinical Sports Anatomy' a textbook for clinicians on patho-anatomy of sports injury. Here I was introduced to the concept of the clinician researcher in SEM.

On return to Ireland I was appointed team physician to the Irish senior rugby team and the Irish amateur boxing association high performance unit. I became director of sports medicine at the Sports Surgery Clinic in 2009. Working with Dr Andy Franklyn Miller, Dublin City University, Roehampton University and University College Cork, we have 4 PhD students, 3 post-doctoral researchers and a large clinical team of biomechanists, physiotherapists and strength and conditioning coaches researching athletic groin pain, anterior cruciate ligament injury and chronic low back pain among other areas. This is the mechanism through which I would hope to play a substantive role in improving public safety and health during and because of physical activity.

1.3 Sport in Ireland

The Irish public is renowned for their love of sport. Ireland has two national sports: Hurling and Gaelic Football, both of which are played by men and women and have more than 750,000 active participants under the guidance of the Gaelic Athletic Association (GAA). 200,000 people play soccer for Irish football clubs and schools throughout the country. There are 60,000 rugby players on the island of Ireland- rugby is the only professional sport in Ireland with 140 full-time professional players playing in European competition. Ireland has a proud tradition of competing in the Olympic Games with medalists in boxing, athletics and sailing, while Irish athletes have won many medals for the United Kingdom and the United States of America prior to the formation of the Irish State in 1924.

1.4 The role of the Specialist Sports Physician in Ireland.

This work aims to highlight the role of the Specialist Sports Physician by employing specific examples of a number of the major areas outlined above; evaluation of the epidemiology of sports injuries; evaluation of the impact of injury prevention measures; screening for medical conditions that may limit or endanger the population during physical activity; and to review the evidence base for current interventions. These are prefaced in sections 1.4.1, 1.4.2, 1.4.3 and 1.4.4 respectively below.

1.4.1 Epidemiology of sports injury

The data on sports- and recreation-related injury in Ireland are limited. Studies completed prior to 2007 have focused on individual sports and activities such as gaelic football,[13-15] rugby,[16,17] hurling,[18-21] ice-skating,[22] wrestling[23] and boxing.[24] Elsewhere studies focused on specific populations such as school children[25,26] and elite athletes,[17,27] or on specific injury types.[28,29] Two studies from emergency departments in Dublin in the 1980's and 1990's gathered data about all sports injuries presenting to the Emergency Department.[30,31] Walsh et al. studied sports injuries presenting to primary care[32] and O'Rourke et al. examined paediatric emergency department sports injury presentation.[33] On review of this data we noted that while specific areas had been dealt with comprehensively, the data on 'all presentations' was more than 15 years old and required a fresh review.

To elucidate the epidemiology of sports and exercise-related injury in Ireland I studied attendances to the Emergency Department (ED) of a university hospital over a 6-month period for the treatment of sports and exercise-related injury.

Epidemiology is the study of the distribution and determinants of varying rates of diseases, injuries, or other health states in human populations for the purpose of identifying and implementing measures to prevent their development and spread.[34] Hippocrates is often described as the father of epidemiology. His rational thinking and attribution of disease to environmental and natural causes contrasted with the conventions of his time. He proposed empirical treatments such as surgery, diet and herbal remedies.[35] The quality of these theories may explain why 2,500 years later his voice still resonates through modern medicine. Morabia argues however that the first real population-based, mathematically and philosophically founded study of epidemiology began in the 17th century.[35]

Medical advances in the 17th century such as the invention of a microscope strong enough to provide visual evidence of living microorganisms strengthened the argument for the germ theory of disease. The concept that organisms were responsible for sickness rather than vapours and miasma from the local environment did not fully gain credence until the 19th century. During the cholera epidemic in London Dr John Snow linked the spread of cholera to water sources and his treatise lead to the closure of the Broad street pump in 1854.[36] Snow interviewed locals about exposure, showed evidence that contaminated water from the Thames was being pumped to the site, he used a dot map to illustrate the cluster of cases around the pump and used statistics to make the connection between water quality and the spread of disease.

This case is rightly famous as it is one of the first recorded cases of the analysis of the risk factors for disease. Intervention based on this analysis had a positive outcome in the prevention of the disease. Though an undoubted success, the theory behind Snow's work was only reluctantly accepted. Nevertheless, this proved a major step forward in the genesis of modern medical epidemiology.

The speciality of epidemiology initially focused primarily on infectious and communicable disease. In 1949 Gordon recognised that injuries tended to follow patterns or 'biological rules' in a similar fashion to disease; his paper 'the epidemiology of accidents'[37] pointed out that injury could be studied by the application of basic epidemiologic principles.[34] This preceded the application of epidemiological techniques in the investigation of sports injury,[38] marking a transition from the 'clinical series' model.[39,40]

Epidemiology in sports medicine may be descriptive, or analytical. In descriptive epidemiology the researcher attempts to quantify the occurrence of injury; who is injured, how many are injured, how often they are injured.[34] Analytical epidemiology concerns itself with the identification of factors that contribute to the occurrence of sports related injury- why the injury occurred.[41]

While analytical epidemiology is most likely of greater interest to the clinician, descriptive epidemiology is a powerful tool in the understanding of injury prevalence and so allowing useful exercise prescription to the individual and ultimately appropriate resource allocation to the broader population. It may be a useful tool in shaping public health initiatives around physical activity. For example, the work of a number of epidemiologists in the US in the 1970s highlighted how the advent of metal and plastic helmets in American football coincided with a dramatic rise in the incidence of catastrophic spinal and head injury.[42,43] While helmets obviously protected the head and face from direct impact, it was correctly asserted that the protection offered by a helmet - which had evolved from a soft leather structure to a hard metal plastic shell- altered playing and tackling behaviour exposing players to cervical spine and brain injury.[44,45] This ultimately led to a change in laws making certain kinds of tackle and blocking illegal, dramatically altering the rate of serious injury in the sport. The epidemiological work of Crowley et al. in the 1980s[18] and 1990s[19] in Ireland has led to the mandatory use of the helmet and faceguard in hurling. This action by the Gaelic Athletic Association (GAA) - the governing body for hurling- has reduced the rate of head and facial injury in hurling.

To establish a current profile of the epidemiology of serious injury presenting to the emergency department (ED) and to highlight some of the activities most often associated with these injuries, I undertook a 6-month study of all sports and recreation injuries presenting to the ED over the study period. Data were gathered on all presentations, but analysis focused on radiologically confirmed fracture.

The emphasis on physical activity and fitness as a means of improving the health of the nation continues to gain support, not only in medical circles,[2] but from a health economics perspective.[46] While we promote physical activity it is our obligation to do so in a safe manner. If physical activity is inappropriate or unsuitable the results may be adverse and injury may occur. This has the potential to negate the benefits accrued to the patient, and to incur a further burden on the health care system. Health promotion recommendations have called for 'a balance between treatment and prevention so that health gains are maximised'.[47] The promotion of physical activity focuses on increasing energy expenditure and avoiding sedentary behaviour, as this has been shown to attenuate the risk of obesity.[48,49] It is agreed that some exercise is better than none, and that the more acceptable the exercise is the more likely patients are to perform it.[50] Anecdotally the author has seen an increase in informal unsupervised recreational activity such as jogging and cycling, while in a paediatric population, activities such as rollerblading and trampoline[51] have become popular. While injury in formal sport such as hurling,[52] football[14] and rugby[16] are well documented, less emphasis is placed on the injury toll of unsupervised recreational activity.

The sports and exercise medicine specialist is one of the better-placed health professionals to advocate physical activity. Published data highlight the role of physical activity in primary and secondary prevention of cardiovascular illness, hypertension and type-2 diabetes.[53-55] Research by Steven Blair and his group as far back as 1996 in fact identified physical inactivity as the major risk factor for all cause mortality in men and women.[3] Physical inactivity has been described as the biggest public health problem of the 21st century.[56] The sports and exercise medicine specialist must be abreast of data in this area so that they may be effective advocates of safe physical activity based upon international and local injury prevalence data. Restriction of activity is rarely advocated but at the very least it is possible to advise parents and teachers on optimal activity types.

Ideally legislation around exercise safety and health promotion could be positively impacted. The negative impact of physical inactivity on the health services is one we can intuitively understand. A night spent in the Emergency Department (ED) of a trauma centre however highlights the impact of sports and recreation-related injury (SRI) on the health services. A balance must be struck between the most efficacious physical activity and the potential adverse events related to this activity.

In examining the epidemiology of sports related injury in Ireland, my hypothesis was that sports and recreation-related injury (SRI) contributed a significant percentage of presentations of serious injury such as fracture to the ED. I also proposed that unsupervised recreational activity may contribute a significant percentage of these presentations.

1.4.2 Impact of injury prevention measures

In Ireland one of the national sports is hurling. Played by more than 300,000 people nationally it is enjoyed among the Irish expatriate population as far afield as New York, U.S.A. and Sydney, Australia. The game is contested by two teams of 15 players over 60–70 minutes on a pitch with dimensions of up to 145 meters long and 90 meters wide.[52] Each hurler carries an ash stick (camán/hurley) used to propel a hard leather ball (sliotar), of diameter 69–72 mm and weight 110–120 g on the ground or through the air.[57] Similar to other field sports such as field hockey and lacrosse, there are a number of subtle differences. The ball may be struck from the ground or in the air. It may also be caught from the air. Indeed one of the iconic images of the game is of a player fielding a ball while jumping full height amid swinging hurleys.

My study of the presentation of SRI to the ED saw hurling well represented. Not only is the game popular in Ireland and in particular in Cork it is played with considerable enthusiasm and often serious injury to the head or upper limb is accepted and even 'worn' as a badge of honour by players and coaches alike.

Traditionally little in the way of protective equipment was used. The work of Crowley et al. highlighted the issue of head injury in hurling and promoted the use of helmet and face protection.[18] A follow-up study in 1995 by the same group showed improved head and face injury rates following helmet and face guard use.[19] The work of O'Flynn et al. highlighted levels of ocular injury among those hurlers not using face protection.[20] The public awareness raised by these and other works helped to convince the Gaelic Athletic Association to make helmet with faceguard use mandatory for all players under-18, then all players under-21 in 2003 and 2005 respectively. In 2010 helmet and faceguard use was made mandatory for all players of all ages playing or training for hurling. Crowley et al. commented on an apparent rise in hand injury over their period studied when compared to falling rates of head and face injury.[19] It is impossible to comment on whether these were real or statistical rate changes but Kiely et al. also commented on presentations of hurling-related hand injury to a trauma centre.[21]

Private communication with other doctors and healthcare professionals working in hurling confirmed the author's belief that there was a potential for altered behaviour among hurlers due to helmet use. In a reflection of trends seen in American football there is a possibility that perceived safety due to helmet and face protection would allow athletes to engage in more dangerous play, perhaps exposing themselves to injury. Given the unique nature of catching a sliotar from the air while other players attempt to strike it with a hurley- the hand is uniquely vulnerable. My study of the SRI presentations to the ED over a 6-month period reflected existing trends of falling rates of head and face injury in hurling with a persistently high level of upper limb (in particular distal to the wrist) injury. I felt there was significant potential to illuminate some of the injury mechanisms and perhaps behavioural changes at the root of these injury rates. I therefore designed and performed a retrospective telephone questionnaire of a cohort of adult hurlers who had presented to the ED with an injury received during play. This focused on the impact of introduced safety measures in hurling (the helmet with faceguard) on injury rates elsewhere in the hurling population.

Health promotion measures advocate that the adoption of a physically active lifestyle at an early age will lead to considerable secondary benefits throughout life.[58] A consequence of physical activity may be an increase in the exposure to injury. Emery states: "*In both elite and recreational sport populations, every sport entails some degree of injury risk. Therefore,* *strategies to promote safe participation and to prevent injuries are paramount".*[58] The concept of injury prevention has evolved with the increasing awareness that injuries were understood to be predictable and preventable - most injuries are the unintended consequences of individual actions in a risky environment; they are not due to fate or to problem behavior.[59] This understanding led to three fundamental injury prevention strategies, as described in the comprehensive report *Injury in America*[60]*:*

1. Persuade persons at risk to change their behavior,
2. Require behavior change by law or administrative rule,
3. Provide automatic protection through product and environmental design.

Frank Haddon Jr. described these actions as either active or passive, referring to the ''amount of action on the part of the individual required to realize an injury prevention effect.'' An example of an active measure would be promotion of the wearing of a seat belt, whereas a passive one would be the installation of an airbag.[61] Haddon further noted "*adequate success through active approaches has been rare, and requires exceptionally broad understanding and strong motivation on the part of those involved. In sharp contrast, passive approaches, when available, and once initiated, have a spectacularly more successful record".*[62] An excellent example of the merits of this approach were the results of an Australian 10 year study on the use of helmets while cycling.[63] It demonstrated a 30% increase (6% to 36%) in bicycle-helmet use during an 8-year period because of helmet-promotion programs. However, once bicycle-helmet legislation was introduced, helmet prevalence rose from 36 to 73% (a 37% increase) in 1 year. Emery et al. felt that this legislation, the action of a higher level in the 'responsibility hierarchy', was effective for a number of reasons.[58] Firstly, it penalized those who chose to ignore the legislation with a monetary fine. More importantly, regulation served to educate parents that there was good evidence (which should be a prerequisite for policy change) that helmets were effective in preventing head injuries.[58] Perhaps the most important influence of the legislation was that it made it less socially acceptable not to wear a helmet. The policy, therefore, evoked a psychological shift in attitudes towards helmets, resulting in a new social norm: everyone was expected to wear a helmet, and parents who were responsible and caring individuals were expected to ensure their children wore helmets.[58]

So we see that while education and public awareness programmes will play a role in the implementation of safety strategies, a combination of authority-led educated policy change is where the future lies. This approach requires support by quality research into the epidemiology of injury, and measures that improve injury prevention.

The clinician wishing to prevent injury must address the consequences of both traumatic and overuse injury. Traumatic injury may be prevented by educating athletes and coaches, by implementing rule changes preventing particular activities, or by the introduction of protective equipment. Coach and athlete education about training load, training regimes and training methods are the mainstay of prevention of overuse injury, however rule changes regarding load management in training have proven effective in the prevention of some adolescent overuse injury. A limit on the number of overs bowled per week in cricket fast bowlers has been effective in limiting pars articularis stress fracture.[64,65] A limited number of pitches per game and per season in little league baseball has effectively limited the incidence of 'little league elbow',[66] A longitudinal study by Lyman et al. recommended 9 to 14 years old should have a general limit of 75 pitches per game with a maximum season limit of 600 pitches.[67] According to the work of Haddon[62] and Emery[58] strategies to limit player exposure are likely to be most effective when they form part of the legislation around the game rather than merely existing as guidelines. Effective legislation must stay abreast of injury epidemiology and scientific data. The evolution of the American football helmet and the legislation around its use and role in the game provides a fascinating insight into an evolving process which ultimately improved player welfare.

In American football the evolution of the helmet over 50 years from a leather helmet to a metal and plastic hardshell helmet with faceguard drastically changed tackle patterns. The introduction of a plastic shell helmet in the 1940s saw tackle patterns change.[68] Mueller et al. suggested *"Players were wearing full face masks and felt well protected when striking their head."*[68] Traditionally players had been taught to lead with the shoulder and arm (similar to the rugby tackle). Perhaps this lack of perceived risk played a role in the altered playing style which saw the ‘spear tackle’ come to the fore. The ‘spear tackle’ saw players tackle with the head rather than shoulder- this was accompanied by a dramatic rise in catastrophic brain and cervical spine injury. Banning the spear tackle and ensuring helmet specifications led to a 42% decrease in brain and spinal injury over a 5 year period.[45]

The introduction of protective equipment makes intuitive sense. Any activity that raises the threshold for injury in an active population should be supported. However, the potential benefits of any intervention must be interrogated to ensure that, firstly it produces the desired effect (injury prevention) and secondly that it causes no inadvertent harm. Many safety devices are however poorly tested and do not fall under the same scrutiny as for example a new medication in development.[69] This argument is eloquently stated by William Haddon Jr. et al.;[70]

“One can, of course, argue that the introduction of essentially unevaluated accident prevention measures “can’t do any harm,” but two potential dangers in this approach need to be noted. First, the introduction and enforcement of insufficiently evaluated measures may lead to an inappropriate choice of emphasis and may, as a result, dissipate funds, time, and public concern that might be applied to more effective measures. Secondly, the public and its government may conclude that everything that can be done is being done.”

Hagel adds that the possibility exists that the countermeasure in question may have other negative unintended consequences.[69] “*These effects may* *include simply shifting the distribution of injury, a change in the behavior of participants resulting from a false sense of security, to reduced participation in the activity due to public discontent if the safety measure is imposed."*

My hypothesis is that use of protective equipment in hurling may lead to altered player behaviour amongst wearers. I further hypothesised that this has been linked to increased levels of "risky behaviour'. I focused particularly on those players suffering serious hand injury and whether they used helmet and face protection.

1.4.3 Screening in Sports Medicine

A further role of the specialist sports and exercise medicine specialist is in the prevention of injury and illness occurring as a result of physical activity.

The process of screening in medicine has prevented disability and death in millions of people through the early detection of cancer,[71] tuberculosis,[72] diabetic retinopathy,[73] and depression.[74] Although screening may lead to an earlier diagnosis, not all screening tests have been shown to ultimately benefit the person being screened; overdiagnosis, misdiagnosis, and creating a false sense of security are some potential adverse effects of screening.[75] For these reasons, a test used in a screening program, especially for a disease with low incidence, must have not only good sensitivity but an acceptable specificity.[76]

To ensure the accuracy of the screening process and limit any adverse events, the World Health Organization (WHO) published guidelines in 1968 which are still applicable today.[77]

Criteria for Disease Screening

- The condition should be an important health problem.
- There should be a treatment for the condition.
- Facilities for diagnosis and treatment should be available.
- There should be a latent stage of the disease.
- There should be a test or examination for the condition.
- The test should be acceptable to the population.
- The natural history of the disease should be adequately understood.
- There should be an agreed policy on whom to treat.
- The total cost of finding a case should be economically balanced in relation to medical expenditure as a whole.
- Case-finding should be a continuous process, not just a "once and for all" project.

Screening is routinely performed for individuals in sports medicine, particularly in the setting of rehabilitation after illness or in planning a return to sport in a sedentary individual. General population screening is more controversial, for example screening athletes for sudden cardiac death.[78-80] An emotive topic, there is a good deal of disagreement amongst experts as to who should be screened, and by what means they should be screened, leading to differences in international practice. The use of electrocardiography (ECG) in addition to history and questionnaire remains controversial,[80] while in Italy all athletes over 14 years have all of these tests in addition to an echocardiogram.[81] In Ireland many of the main sporting governing bodies abide by the guidelines of either the British Heart Foundation or the American Heart Association.[82] The Gaelic Athletic Association has issued their own guidelines for cardiac screening in athletes over 14 years- "*screening should consist of completion of the GAA's Cardiac Screening Questionnaire, a Physical Examination and an ECG*".[83] There has been little published in the literature on screening in sport in Ireland. Waterhouse et al. evaluated cardiac magnetic resonance imaging as a screening tool for left ventricular hypertrophy in an athletic population.[84] Hogan et al. screened amateur rugby players to exclude degenerative change in the cervical spine.[85] Warrington et al. screened national hunt jockeys for osteoporosis.[86] Moloney et al. developed an exercise challenge to screen for undiagnosed pulmonary fibrosis.[87] The prevalence of obstructive airflow limitation in Irish collegiate athletes was investigated by Smith et al.[88]

My aim was to evaluate a means of screening for a more common condition arising out of sports participation, one which additionally limits athletic performance. I chose to screen for exercise-induced bronchoconstriction (EIB). This condition has been shown to affect those participating in high intensity physical activity in winter conditions. I elected to evaluate the incidence of EIB in a cohort of professional rugby players (a winter sport). Screening for EIB in a professional rugby cohort fulfills the criteria outlined by Wilson.[77]

My hypothesis was that EIB may be more prevalent in a winter sport such as rugby when compared to 'summer sports'. I also hypothesised that due to the purely exercise-related nature of this condition it may be under diagnosed, even in a professional cohort.

Asthma and Exercise Induced Bronchoconstriction

Asthma is a complex disorder characterized by variable and recurring symptoms, airflow obstruction, bronchial hyper-responsiveness, and an underlying inflammation.[89] EIB is an acute, transient airway narrowing that occurs during or after exercise[90] and is objectively defined as a ≥10% decline in forced expiratory volume in 1 second (FEV_1) after appropriate exercise provocation.[91] Exercise-induced asthma refers to such a narrowing in those people with known asthma.[92] In the general population the incidence of asthma is 6-12%[93,94] and EIB has been reported as affecting 12%.[95,96] In those known to be asthmatic, exercise is a common trigger of bronchospasm and bronchial hyper-reactivity (BHR) may occur with exercise in 50-90% of asthma sufferers during or after exercise.[95] In elite athletes EIB has a prevalence of 11 to 50%, depending on the sport.[96]

There are two proposed mechanisms for EIB. The thermal hypothesis states that airway cooling during exercise followed by rapid re-warming of the airways after exercise cause a reactive hyperaemia of the bronchial microvasculature and oedema of the airway wall leading to contraction of bronchial smooth muscle.[97,98] The hyperosmolar hypothesis states that dehydration of the airways on conditioning large volumes of dry air during exercise results in an increase in osmolality in tissue ultimately leading to contraction of bronchial smooth muscle.[99,100] Anderson argues however that neither abnormal airway cooling nor rapid re-warming of the airways is a prerequisite for EIB and that regulatory volume increase, after cell shrinkage (due to airway dehydration), is the key event leading to the release of inflammatory mediators.[98]

The symptoms associated with EIB include fatigue or dyspnoea during exercise and are therefore often incorrectly attributed to deconditioning in athletes.[101-103] EIB is thought to be more common in endurance sports, particularly winter sports[92] where prolonged periods of hyperpnoea in cold, dry air are typical.[104]

Asthma is a recognised cause of sudden death.[105] Lang reported approximately 4,500 deaths annually in the US due to asthma.[106] Unlike sudden cardiac events, death due to asthma during sporting endeavour is rare. However, one retrospective case series recorded all sports-related deaths over a seven-year period.[107] The study collated 61 deaths related to asthma in a sporting population – a rare cause, more common in Caucasian males aged 10-20 yrs. While 91% had a previous diagnosis of asthma 6 cases (9%) did not. Exercise induced bronchoconstriction is however usually associated with diminished athletic performance. In the 2000

Australian Summer Olympic team, the prevalence of EIB was found to be in the order of 21%.[108] Rates of up to 50% have been reported in elite cross-country skiers.[96] The work of Karjalainen et al. has linked chronic EIB to permanent airway remodelling.[109] This may have implications for guidelines around management and treatment of asthma and EIB in professional athletes where chronic exposure has the potential to progress to permanent airway remodelling. This highlights the importance of screening for EIB in the athletic population, especially in those involved in high intensity sports in adverse weather conditions, such as in Ireland.

Screening for Exercise Induced Bronchoconstriction

Making a diagnosis of EIB has proven to be a challenge, as clinical presentation can be masked and often under-represents the causative pathology. Rundell and co-workers showed that when a group of athletes were exposed to a field exercise challenge test only 61% of athletes who tested positive for airway obstruction reported symptoms while 45% of those negative to the challenge also reported symptoms.[102] Holzer and colleagues described similar findings for a laboratory provocation test.[110] This variation between clinical presentation and pathology means that symptom questionnaires are of limited value. The diagnosis of EIB demands confirmation by demonstration of a decrement in lung function associated with exercise or a surrogate, usually achieved by bronchial provocation testing.[89]

Eucapnic Voluntary Hyperventilation (EVH)

The EVH test is the current challenge test recommended by the International Olympic Committee (IOC) as the optimal laboratory based challenge test for the identification of EIB. [89]

Hyperventilation of cold, dry air has been shown to be an effective stimulus for bronchoconstriction in people with reactive airways.[111] Following inhalation of this air in controlled conditions the resulting bronchial hyper-responsiveness is measured as a fall in $FEV_{1.}$ The work of Philips et al. showed that cooling the inhaled air was unnecessary and a simplified EVH challenge can be done using a single dry gas mixture without the need for cooling inspired gas or monitoring end-tidal fraction of CO_2. This test can be used to identify and study patients with EIB without the requirement for an exercise challenge or the need for elaborate gas conditioning and monitoring equipment.[111] The inhaled dry gas mixture contains 5% carbon dioxide, 21% oxygen, with the balance being nitrogen, for a set duration, depending on the protocol used. This concentration of gas is safe, stimulates ventilation, and has been shown to maintain normal end-tidal CO_2 levels throughout the challenge (unlike exercise).[92]

To overcome the problem of any post-test respiratory muscle fatigue, the FEV_1 should first be recorded at least 3 minutes after challenge. It is usual for the reduction to be sustained over the next five minutes to be consistent with hyperpnoea-induced bronchoconstriction.[91,112]

Though EVH is the ‘gold standard’ it requires a good deal of expensive laboratory equipment and is time consuming. Pharmacological testing is more often used due to ease of access and cost effectiveness.

Pharmacological challenge testing

Methacholine challenge testing utilises a nebulised methacholine solution as a bronchial irritant- the resulting bronchial hyper-responsiveness is measured as a fall in FEV_1. Methacholine is a synthetic choline-ester that acts as a non-selective muscarinic receptor agonist in the parasympathetic nervous system. Concentrations of methacholine of 0.03 mg/ml, 0.06 mg/ml, 0.125 mg/ml, 0.25 mg/ml, 0.50 mg/ml, 1 mg/ml, 2 mg/ml, 4 mg/ml, 8 mg/ml and 16 mg/ml are prepared. Commencing with the most diluted solution, the nebulised solution is inhaled by the patient for 2 minutes and FEV_1 is measured at 30 and 90 seconds. The concentration is increased at 5-minute intervals until a decrease in FEV_1 of 20% or more compared to baseline is noted.

Methacholine challenge testing is more useful in excluding a diagnosis of asthma than in establishing one because its negative predictive power is greater than its positive predictive power.[113]

Pharmacological provocation testing is sometimes performed with pharmacological agents other than methacholine such as histamine, carbachol or adenosine monophosphate. Methacholine and histamine produce bronchoconstriction at nearly equivalent concentrations. Methacholine is more commonly used and is preferred to histamine because histamine is associated with more systemic side effects, including headache, flushing, and hoarseness. In addition, bronchial hyper-responsiveness measurements may be less reproducible when using histamine.[113]

Histamine, carbachol and adenosine monophospate are not accepted as valid tests for the purposes of diagnosis or exclusion of EIB by the IOC[114] and are not considered in this text.

Exercise challenge/provocation testing

Exercise challenge/provocation testing for EIB may be performed in the field or laboratory:

Field exercise challenge tests

Field-based exercise testing has the obvious advantage of having the athlete exercise in their specific sport- thus exposing them to the normal airway irritation. The aim of testing is to have the athlete exercising at 85% maximum heart rate for a minimum of 4 to 6 minutes. Spirometry is then measured at regular intervals for up to 30 minutes following completion of the challenge.[92]

Holzer argues that though this type of challenge is highly specific for the diagnosis of EIB, it has the disadvantages of lack of standardisation of the cardiovascular workload or the environmental conditions of temperature and humidity.[92] Holzer further argues that reliance on suitable weather conditions and on the patient's motivation is a further disadvantage.[92]

Laboratory exercise challenge testing.

Laboratory exercise challenge testing may be performed on a treadmill or cycle ergometer.[92] On the cycle ergometer, 1 of 2 protocols may be used: either a single- load protocol, in which the subject exercises at an intensity of 45% to 60% of predicted maximum voluntary ventilation (MVV) for 6 to 8 minutes, or an increasing protocol, with the intensity starting at 60% of the final load in the first minute, then 75% in the second minute, 90% in the third minute, and 100% in the fourth minute.[115] Once the target level is reached, the workload is held for 4 minutes.[113] The standardised treadmill protocol recommends a speed and grade to produce 4 to 6 minutes at near maximum heart rate, with a total of exercise duration of 6 to 8 minutes.[113] During the first 2 to 3 minutes of exercise, both the treadmill speed and grade are rapidly increased until the subject's heart rate is 80% to 90% of the maximum predicted. Spirometry is then measured at regular intervals for up to 30 minutes following completion of either challenge. Holzer again argues that though this type of challenge test has a high specificity for EIB, it often fails to reach a high enough ventilation rate in trained athletes to induce EIB. Furthermore, it has the following disadvantages: it is not sports-specific, it is not performed in the environment in which the exercise is usually performed, and the equipment is expensive.[92]

To maximise the opportunity for a positive test, the exercise test should be performed breathing dry air for 8 minutes with the intensity of exercise close to maximal for the last 4 minutes. To overcome the problem of any post-test respiratory muscle fatigue, the FEV_1 should first be recorded at least 3 minutes after challenge. It is usual for the reduction to be sustained over the next 5 minutes to be consistent with EIB.[112]

Osmotic Challenge tests

The response to 4.5% saline and the response to mannitol (crystalline sugar alcohol) is usually reported as the dose required to provoke a 15% fall in FEV_1 (PD15) but should also be reported as the maximum fall after the final dose of aerosol.[116] Holzer showed that mannitol had a sensitivity of 96% and specificity of 92% to identify a positive response to eucapnic voluntary hyperventilation (EVH).[117]

If the administration of these challenges results in a reduction in the lung function of the athlete, the presence or absence of asthma or EIB is confirmed.[89]

In all tests a fall in the FEV_1 compared to pre-provocation baseline is the accepted measure. Depending on the type of challenge test, the accepted fall in the FEV_1 for a positive challenge varies from 10% to 20% and must occur within a specified period following the administration of the challenge dose, or within a specific cumulative dose of administered agent.[89] The International Olympic Committee guidelines for provocation testing[114] recommend withholding medications prior to bronchial provocation tests.

Exercise Induced Bronchoconstriction in winter sports

EIB is more common in endurance sports such as cross country skiing, swimming, and long distance running,[92] where prolonged periods of hyperpnoea are the norm.[104] Winter sports are also a risk factor, both cold air[118,119] and low humidity[98] have been linked with a higher prevalence of EIB, perhaps due in part to the increased cooling of airways and the relative increase in reactive hyperemia in the pulmonary vasculature.[95]

Exercise Induced Bronchoconstriction in Rugby

Rugby Union is a contact field sport played in the Northern Hemisphere in the winter and spring months. It is played with 15 players on each side who compete to score points either by 'touching down' over the opposition touchline or by kicking the oval ball over the crossbar of 'H' shaped posts. It is a truly international sport with over 2 million players in 123 countries. The prevalence of EIB in Rugby Union players is unknown despite the popularity of the sport.

I hypothesized that EIB may be under reported in rugby. To address this question, I developed a rugby-specific exercise protocol, questionnaire and spirometry testing procedure to measure the prevalence of Asthma and EIB in a cohort of professional rugby players.

1.4.4 Evidence Based Sports Medicine

Evidence based medicine (EBM) is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients.[120] In practice this means integrating individual clinical experience with the best available external clinical evidence from systematic research. The philosophical roots of EBM extend back to mid-19th century Paris[120] and it has steadily gained support in Western medicine since the mid 1980s. Those who criticize this approach feel it may represent 'cookbook medicine' and seek to replace vital clinical expertise and judgment.[38] It is generally accepted that the modern medical practitioner must practice the most up to date medicine and keep abreast of developments in their field of expertise.

Evidence based medicine in sports medicine has gathered speed similar to other areas of medicine but has perhaps been hampered by a number of specific issues which are unique to sports medicine. Sports medicine is largely practiced in the private medicine setting. While data collection and study of the evidence behind treatment is possible, conducting level 1 research such as a randomised controlled trial is extremely difficult. In a private medicine setting placebo-based studies are not possible unless the study is funded or subsidised. Even when this is possible the athlete is often reluctant to engage in a process where they have the possibility of receiving 'less than the best'. Bernstein eloquently explains that a blinded randomised controlled trial is unlikely to be completed on internal versus external fixation of tibial fracture- making an evidence based approach to managing this problem difficult.[121] It is accepted that evidence based medicine is ideally based on the best level of evidence- Wright[122] noted *"The essence of levels of evidence is that, in general, controlled studies are better than uncontrolled studies, prospective studies are better than retrospective studies, and randomized studies are better than nonrandomized studies."* Where this level of evidence is unavailable the lowest level of evidence (level V) must be relied upon- expert opinion. It is generally agreed that however limited, expert opinion is superior to non-expert opinion.[123] Unfortunately much of the practice in sports medicine and orthopaedics is based upon level V evidence.

The practice of expert based medicine becomes problematic however when it is based upon flawed principles. Patterson describes what many feel to be the first clinical trial.[124] Dr. Pierre Louis investigated the practice of 'blood letting' to cure pneumonia. Patterson writes that in 1836 *"the prevailing conception of illness was that the sick were contaminated by some toxin or contagion or an excess of one humor or another. That understanding of illness contained within it the idea that these conditions could be improved by opening a vein and letting the sickness run out."*[124] Dr. Louis treated people with pneumonia either with early, aggressive bloodletting or less aggressive measures; at the end of the experiment he counted the bodies! This work discredited the process of bloodletting and went some way toward discrediting the principles behind its practice.

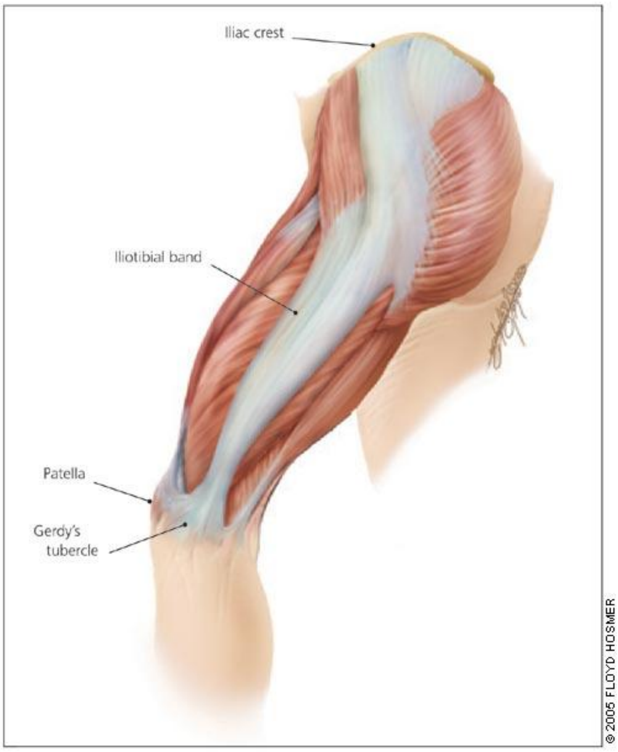
Notwithstanding the difficulties set out above, much excellent work has been performed to improve the evidence base behind decision making in sports medicine. McCrory has led research into concussion and since his first paper on the subject in 1997[125] has worked with others in the field to host the first international meeting around concussion in sport leading to a consensus paper in 2002.[126] Since then McCrory has published 108 articles on concussion, the most recent being the proceedings of the 4th International Concussion in Sport Conference in Zurich in 2012.[127] This work has dramatically shaped the management of concussion in sport and forms the evidence base against which major sporting organisations such as FIFA (Fédération Internationale de Football Association), NFL (National Football League), NHL (National Hockey League), and the IRB (International Rugby Board) create their own regulations and management guidelines. This is just one example where a group of clinicians have worked together to improve the understanding of a condition and therefore improve its management.

Where a body of work of this quality does not exist, the clinician who wishes to practice evidence based medicine may need to make the best of what is available. As Bernstein has stated- "*Limiting one's practice to only proven and established facts is no guarantee of safety: shunning everything unproven forces us likewise to reject the many statements that are true, just not proven.*" Nevertheless the clinician must make efforts to interrogate their management strategies regularly. The clinician must aspire where possible to treat injury and illness arising from physical activity in an evidence-based manner. I chose to assess the evidence base behind distal iliotibial friction syndrome (ITBFS)- the most common cause of lateral knee pain in an active population.[128]

Anatomy of the Iliotibial Band

The iliotibial band (ITB) is a fascial condensation at the lateral aspect of the fascia lata. The ITB as described in classical textbooks is a structure *'over the lateral femoral aspect [where] fascia lata is compacted into a strong iliotibial tract…'.*[129] More current thinking however, focuses on the ITB as a discrete entity, passing from the iliac crest to the lateral (Gerdy's) tubercle of the tibia.

Figure 1.1. A modern representation of the anatomy of the iliotibial band, by Floyd Hosmer. *Contained in the article "Iliotibial Band Syndrome: A Common Source of Knee Pain" by Khaund R et al. Am Fam Physician. 2005 Apr 15;71(8):1545-1550. Permissions requested from the American association of family practitioners (aafp).*[130]



Iliotibial band friction syndrome (ITBFS) is the most common cause of lateral knee pain and a common presentation to the sports physician. This is a pain that occurs when running, particularly downhill, usually after a specific distance. Distal iliotibial band friction syndrome (ITBS) is often aetiologically linked with either friction of the ITB at the lateral femoral condyle prior to insertion into the more distal Gerdy's tubercle of the lateral, proximal tibia.[131,132] Irritation or inflammation of a bursa at the same site is also cited as an aetiological factor in this condition.[128]

The work of Fairclough et al. has challenged much of the basis for these anatomical assumptions.[133] Their work showed dissection and MRI evidence of a firm adherence of the ITB to the lateral femoral condyle. Furthermore they failed to identify a bursa between the distal ITB and femoral condyle in any of their dissections. MRI correlation shows that what is often called a bursa on imaging is in fact a reflection of the joint capsule posteriorly beneath the ITB- an anatomically normal structure.[133,134] The intermuscular septum of the thigh is well described, as is its insertion into the linea aspera of the femur. Fairclough's work shows that the ITB is contiguous with the intermuscular septum and is therefore longitudinally attached to the femur.

These observations mean that many of the treatments recommended for ITBFS – injection of bursa, friction to the distal ITB, stretching of the ITB, are in fact based on a false premise. These treatments are therefore much less likely either to work, or to be of as much benefit to the patient as one might expect.

I chose to further assess these anatomical descriptions through cadaveric dissection. I performed a novel cadaveric biomechanical assessment and an *in-vivo* biomechanical analysis of the strain and movement of the ITB in order to investigate both the mechanism of proposed aetiological factors, and challenge the anatomical principles upon which a number of the traditional treatments of ITBS are based.

My hypothesis is that a number of the traditional treatments aimed at local inflammation and stretching of the ITB derive from an incorrect understanding of the relevant anatomy and pathology.

In summary, the role of the sports and exercise medicine specialist is broad, and extends from a thorough understanding of the epidemiology and pathophysiology of sports and recreation related injury (SRI); to concepts such as screening for disease and injury prevention; a detailed knowledge of the evidence base for all implemented treatments; and an appreciation for the consequences of regulation surrounding sports protective equipment and intervention.

Study Aims and Hypotheses

Specific Aim 1 (Chapter 2): To examine the epidemiology of SRI in Ireland by determining the occurrence of sport- and recreation-related injuries and fracture amongst Emergency Department (ED) attendees by examining all SRI attending the ED of a tertiary referral university hospital.

Hypothesis 1: That sports and recreation-related injury (SRI) contributed a significant percentage of presentations of serious injury such as fracture to the ED. I also proposed that unsupervised recreational activity may contribute a significant percentage of these presentations.

Specific Aim 2 (Chapter 3): To quantify the occurrence of hurling-related hand injury presenting to the ED and examine the variables, which may be associated with hand injury.

Hypothesis 2: That the use of protective equipment in hurling may lead to altered player behaviour and may be associated with increased occurrence of hand injury.

Specific Aim 3 (Chapter 4): To determine the prevalence of exercise induced bronchoconstriction (EIB) in elite rugby players by performing a specifically designed field-based exercise challenge on an intermediate Rugby Union team.

Hypothesis 3: That EIB is under-reported and undertreated in professional Rugby Union.

Specific Aim 4 (Chapter 5): To evaluate the evidence base behind the treatment options for iliotibial band friction syndrome (ITBFS), using novel cadaveric biomechanical assessment and an *in-vivo* biomechanical analysis of the strain and movement of the ITB.

Hypothesis 4: That current treatment options for ITBFS are based on a number of flawed anatomical and biomechanical assumptions.

Chapter 2

Sport and Recreation Related Injuries and fracture occurrence amongst Emergency Department Attendees: Implications for exercise prescription and injury prevention.

This work has been peer reviewed and published as "Sport and recreation-related injuries and fracture occurrence among emergency department attendees: implications for exercise prescription and injury prevention". Falvey EC, Eustace J, Whelan B, Molloy MS, Cusack SP, Shanahan F, Molloy MG. Emerg Med J. 2009 Aug;26(8):590-5. PMID: 19625558. (Extracts reproduced with permission of the publishers).

Introduction

Exercise is promoted to offset many of the adverse effects of modern western lifestyle, but is associated with its own potential complications. Sports and recreation-related injury (SRI) occurrence has risen in tandem with an increased level of public activity.[135] SRI may hamper further activity on the part of the injured, thereby resulting in the sedentary lifestyle that it was hoped to avoid. As with any form of prevention or therapy the ill-effects of an intervention form an integral part in the calculation of its effectiveness.[136]

Regular physical exercise is an essential pre-requisite for normal physical development in children. It is similarly a central public health recommendation for maintaining long term health, avoiding weight gain, and preventing cardiovascular disease in adults. Obesity, resultant adverse medical events, and a diminished quality of life are on the increase in a society bound between excessive caloric intake and physical inactivity into a spiral toward ill-health.

Exercise prescription is very often the role of the primary care physician. While the perceived risks of SRI may limit participation in sports, the risks associated with unsupervised or poorly supervised recreational activities may be underestimated. Health promotion recommendations have called for 'a balance between treatment and prevention so that health gains are maximised'.[47] An accurate assessment of the risks attached to not only sporting, but also recreational activity, is obviously fundamental to this.

Epidemiologists have long struggled to quantify the extent of sports and recreation-related injury.[137-142] Regardless of these limitations, guidelines regarding safe and worthwhile participation in sports and recreation activity will not only be more effective, but will enjoy greater acceptance if they are based on an accurate assessment of the occurrence of complications.[143] To date, much of the information at our disposal has been provided by American and Australian studies, [135,144-147] European data being limited to individual sports, injury types, or age groups.[16,148-151]

I therefore conducted the following prospective survey to quantify the occurrence and resource-utilisation resulting from SRI at a large university hospital-based Emergency Department (ED) in the South of Ireland, and to examine the occurrence of fractures arising from formal sporting as compared to recreational activities amongst ED attendees.

Methods

This was a six-month study based at the ED of Cork University Hospital, the sole orthopaedic trauma centre for Cork city and its surrounding hinterland of one million people. All persons attending the ED for treatment of a physical injury, which the patient identified as resulting from a recreational or sporting activity, had a questionnaire completed by their treating ED physician regarding the nature and circumstances of their injury and their subsequent investigations and management. ED personnel were supported with regular in-service training, and advised to include cases where a doubt arose, allowing a final decision regarding inclusion, to be made by the study investigators.

Fifty-four different activities were volunteered by those participating in the study, which were divided into 6 general categories, ‘Field Sports’ (including soccer, hurling, Gaelic football and rugby), ‘Ballsports’ (including basketball, tennis, and racketball), ‘Non-contact sports’ (including horse-riding), Combat sports (including karate, wrestling and boxing), Recreation (including walking, gardening), and Play.

The location where the injury occurred was summarised as Residential, Public place of sport, and ‘Other’- non formal sporting locations (including school, parks, main roads, and waterways). Mechanism of injury was categorised by the treating ED physician based on a patient narrative as; fall, struck by ball, collision, struck by other player, struck by playing equipment, foul play, and over-exertion. This was subsequently categorised at analysis as ‘Fall’, ‘External force’ (which included injuries resulting from accidental or deliberate contact with a ball, piece of equipment or other player[s]), and ‘Over-exertion’ (injuries due to the subjects own actions, not the result of external force and if associated with a fall, occurring prior to the fall).

Site of injury was categorised anatomically and subsequently grouped into Upper Limb, Lower Limb and Axial (Head, Neck and Trunk). Level of competition was summarised as Organised competition/practice, Informal competition/practice, or Recreational.

In the case of a fracture being noted or suspected, the diagnosis was validated using the subsequent official radiological report attached to the individual ED charts. In keeping with our ED clinical practices those aged 4-16 were defined as paediatric, those 16 and older as adults. Patients under 4 years were excluded from the study; this recognises commencement of schooling and the resultant rise in both group and individual physical activity. Data on all ED attendances over the period was gathered from the hospital electronic medical record system. Review clinic attendances were not included. Population estimates are based on the Irish National Census figures for Cork City and County, 2003.

Statistical analysis

Distributions were summarised using means (standard deviations) or medians (intra-quartile range) as appropriate. Proportions were compared using Chi square tests. Univariate associations of fracture occurrence were examined using logistic regression. The presence, strength, independence, and significance of fracture occurrence with activity category was quantified using multivariate logistic regression, adjusting simultaneously for age, gender, site and mechanism of injury. As place of injury was highly correlated with activity type, the former was excluded from the multivariate model. Analysis was performed using SPSS V13, (SPSS Inc, Chicago, Illinois) using a 2 sided type I error rate of 0.05. The study was funded from clinical revenues by the Department of Rheumatology, Sports

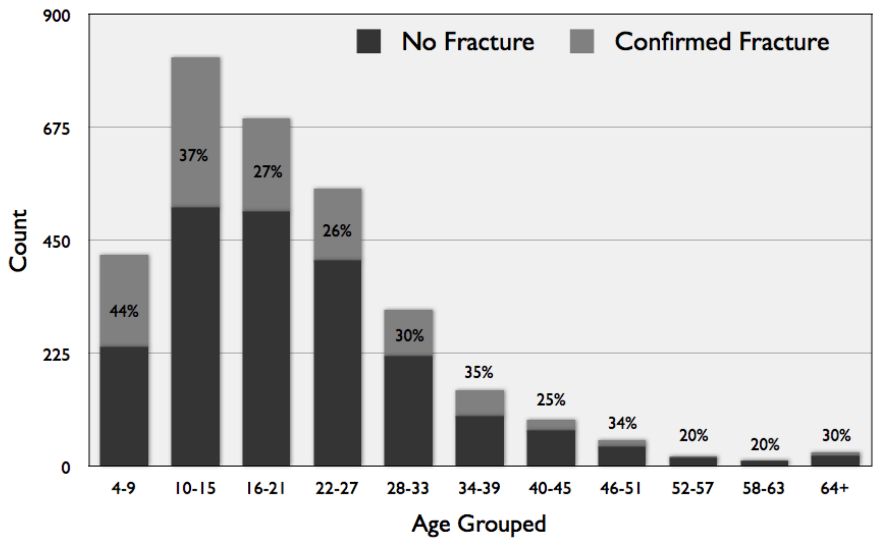
& Exercise Medicine Programme, Cork University Hospital, Cork, Ireland. Ethical approval was secured from the Cork Teaching Hospitals Ethics Committee.

Results

Sports and Recreational Injuries

Of the 22,465 ED attendances over the study period, 3172 (14.1%) were identified by the patient as being sports- and recreation-related injury (SRI). Children aged 4-16 comprised 39.9% of SRI attendances as compared with 16.5% of all-cause ED attendances, and 18% of the underlying Cork population. SRI attendees were more likely to be male (75%) as compared to the overall ED population (58.2%) or the underlying population (45.9%). Attendance figures peaked for the 10-15 year age group (26% of SRI attendees) and thereafter declined (Figure 2.1).

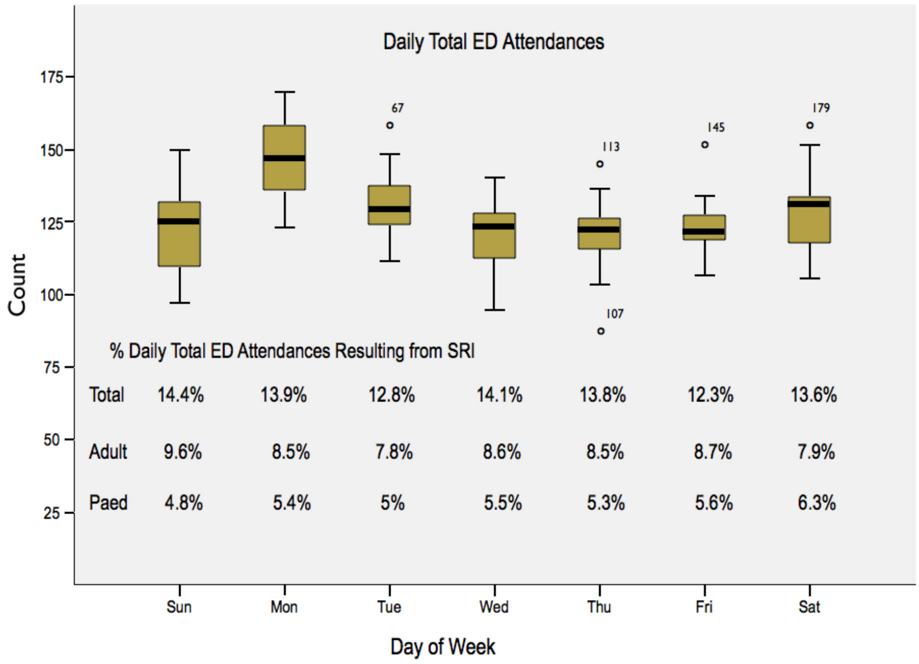
Figure 2.1: Age distribution of subjects attending the Emergency Department with Sports and Recreational Injuries and age-group specific fracture rate.



Resource utilisation

Average daily ED attendances for all injuries were highest on a Monday, but the proportion of ED attendances due to SRIs remained relatively constant over the week with a mean daily proportion (SD) of 13.6% (0.7), Figure 2.2. On average 58% of patients attending the ED with SRI were referred by their primary care physician.

Figure 2.2: Box plot of attendees with Sports and Recreational Injuries at a regional Emergency Department per day of the week, showing the median (solid line) and intraquartile range (box) for each distribution.



Emergency admission to hospital from the ED was required in 8.7% of cases. Plain radiology was performed in the majority of cases (89%), with other forms of radiology utilised in a further 2% of cases. Outpatient hospital follow-up was arranged in 57% of cases, including Orthopaedic follow-up in 34% of the overall group, ED follow-up in 9%, and physiotherapy in 7%. Thirty three percent of SRI attendees were referred back to their primary care physician and only 10% of cases were discharged without planned follow-up.

Characteristics of Sports and Recreation Related Injury

Of the 54 individual activities reported, 75% of all presentations were due to 9 activities; soccer, hurling, gaelic football, rugby football, playground, trampoline, bouncy castle, cycling, horse riding (Table 2.2). Field sports were the commonest cause of SRI (46.5%). Although 61% of SRIs occurred in a public place of sport, 19% occurred in a residential setting, including 47% of playground equipment injuries, 83% of Bouncy Castle-injuries, and 98% of trampoline injuries. Organised competition/practice was the commonest activity underlying SRI (49%), closely followed by recreation (41.5%), informal competition/practice being the cause in 9.4%, (Table 2.1). The commonest mechanism of injury resulted from *an external force* (48.1%), *fall* in 35.2% of cases, and *overexertion-type injury* in 16.7%. Thirty two percent of SRI attendees were diagnosed with a fracture, 29% with a sprain and/or strain, and a further 149 (4.7%) with an uncomplicated dislocation.

Table 2.1 Characteristics of Sports and Recreational Injuries among Emergency Department Attendees.

Variable	Total	Adult		Paediatric	
	N (%)	N (% of adults)	*% of variable*	N (%)	% of variable
Specific Sport/ recreational activity (n=3115)					
Field Sport	1799 (57.8%)	1238 (66.2%)	(68.8%)	561 (45%)	(31.2%)
Play	402 (12.9%)	43 (2.3%)	(10.7%)	359 (28%)	(89.3%)
Recreation	172 (5.5%)	155 (8.3%)	(90.1%)	17 (1.4%)	(9.9%)
Non-contact	401 (12.9%)	199 (10.6%)	(49.6%)	202 (16%)	(50.4%)
Combat	132 (4.2%)	96 (5.1%)	(72.7%)	36 (2.9%)	(27.3%)
Ballsport	209 (6.7%)	138 (7.4%)	(66.0%)	71 (5.6%)	(34.0%)
Mechanism of injury (n=3095)					
External	1489 (48.1%)	968 (52.3%)	(65.0%)	521 (41.9%)	(35%)
Fall	1090 (35.2%)	484 (26.1%)	(44.4%)	606 (48.8%)	(55.6%)
Overexertion	516 (16.7%)	400 (21.6%)	(77.5%)	116 (9.3%)	(22.5%)
Site of injury (n=3172)					
Upper limb	1476 (46.5%)	764 (40.1%)	(51.8%)	710 (56.1%)	(48.2%)
Lower limb	1111 (35.0%	775 (40.7%)	(69.8%)	336 (26.6%)	(30.2%)
Axial	585 (18.5%)	366 (19.2%)	(62.6%)	219 (17.3%)	(37.4%)
Level of participation (n=2800)					
Recreation	1162 (36.6%)	508 (30.1%)	(43.7%)	654 (58.8%)	(56.3%)
Informal	263 (8.3%)	171 (10.1%)	(65.0%)	92 (8.3%)	(35.0%)
Organised	1375 (43.3%)	1008 (59.8%)	(73.3%)	367 (33.0%)	(26.7%)
Place of injury (n=2738)					
Residential	505 (15.9%)	366 (34.5%)	(72.5%)	139 (8.3%)	(27.5%)
Public Place of Sport	1681 (53.0%)	1262 (75.3%)	(75.1%)	419 (39.5%)	(24.9%)
Other	552 (17.4%)	276 (26%)	(50.0%)	276 (16.5%)	(50.0%)

Fracture occurrence

Of the 1,029 diagnosed fractures 69.8% (n = 718) were upper limb injuries, 22.3% (n = 229) lower limb injuries, and 8% (n = 82) were axial. The highest rate of fracture occurred in those aged 4-9 (44%), followed by the 10-15 years old group at 37% (Figure 2.1). There was a subsequent progressive reduction in fracture occurrence in older subjects though fracture rates remained substantial at older age groups, e.g. 35% for 34-39 year olds. The proportion of patients who sustained a fracture during a specific activity is shown in Table 2.2. Of the commonest activities, approximately one quarter to one third of patients with SRI who attended the ED due to field sports injury had a fracture. Rates of up to 40% were seen for several recreational, play, and non-contact sporting activities, including 40% SRI due to trampoline use and 39% of SRI due to ‘Bouncy Castles’. Of note, some activities (though rare causes of SRI overall) have an appreciable potential for serious injury. Thus rollerblading, though the cause of only 16 SRI over the study period, was associated with 12 fractures.

Table 2.2 Percentage occurrence of sports and recreational injuries amongst the 12 commonest individual activities (expressed as a percentage of all attendees with sports or recreational injuries*) and the percentage of attendees for that specific activity who were diagnosed with a fracture.*

Field Sports		**Play**		**Non contact Sports**	
Soccer (22%)	*26%*	Playground (7.3%)	*47%*	Cycling (5%)	*38%*
Hurling (18%)	*34%*	Trampoline (3.6%)	*40%*	Horse riding (3%)	*39%*
Gaelic football (10%)	*30%*	Bouncy castle (1%)	*39%*	**Combat sports**	
				Martial arts (1%)	*22%*
Rugby football (6%)	*34%*	**Recreation**		**Ball Sports**	
		Walking (2%)	*39%* (2%)	Basketball (2%)	*30%*

On univariate analysis (Table 2.3) the paediatric age group was associated with a significantly increased odds of fracture compared to the adult group. Women had a non-significant higher odds ratio of fracture, OR (95% CI) 1.15 (0.97 – 1.36). Although most SRI had occurred in a public place of sport, a residential location or other non-formal public place of sport was associated with a significantly higher OR for fracture. Of the activities themselves, field sports did not have a significantly higher odds ratio of fracture compared with the presumed lower-risk ball sports, while play activities, non-contact sports and combat sports had (Table 2.3). As expected, external force (which included both deliberate and accidental forms) had an over 2.0-fold increased odds of fracture relative to overexertion type injuries, $p<0.001$, though suffering a fall had an even higher, 3.7-fold increased odds, $p<0.001$. In keeping with the strong association with falls, the odds of fracture were significantly higher for upper limb injuries (OR 5.8, $p<0.001$) compared with axial injuries, with lower limb injuries having a weaker though still highly significant association (OR 1.59, $p=0.001$)

Table 2.3 Univariate association of fracture occurrence among 3,712 Emergency Department Attendees with sports and recreational Injuries (SRI)

		% Fracture*	**OR (95% CI)**	**p-value**
Age group	Paediatric	40%	1.71 (1.47-1.99)	<0.001
	Adult	28%	1 (reference)	
Sex	Female	35%	1.15 (0.97-1.36)	0.11
	Male	32%	1 (reference)	
Place of Injury	Residential	38%	1.47 (1.19-1.80)	<0.001
	Other ⨍	37%	1.42 (1.16-1.74)	0.001
	Public Place of Sport	30%	1 (reference)	
Site of injury	Upper Limb	49%	5.83 (4.52-7.52)	<0.001
	Lower Limb	21%	1.59 (1.21-2.1)	0.001
	Axial	14%	1 (reference)	
Mechanism of Injury	External Force #	31%	2.34 (1.8-3.02)	<0.001
	Fall	42%	3.74 (2.88-4.87)	<0.001
	Overexertion ǂ	16%	1 (reference)	
Activity	Field sport	30%	1.32 (0.95-1.83)	0.1
	'Play'	45%	2.45 (1.69-3.55)	<0.001
	Recreation	29%	1.24 (0.79-1.95)	0.36
	Non-contact sport	35%	1.64 (1.13-2.38)	0.01
	Combat sport	35%	1.62 (1.0-2.6)	0.048
	Other ball sports	25%	1 (reference)	

∗ Percent of patients who present to ED with an SRI, who have a fracture.
⨍ Other = Main road, water, seaside, school, other.

ǂ Overexertion = Injury occurring due to patients own actions not involving external force, objects, or persons.

External Force = Collision, struck by ball, player, playing equipment, foul play.

The same general relationships persisted in multivariate analysis (Table 2.4). Upper limb injuries remained strongly and significantly associated with the odds of fracture (adjusted OR: 5.8, $p<0.001$) and while the adjusted OR for 'Falls' relative to over-exertion type injuries was attenuated, it remained highly significant (adjusted OR: 2.15, $p<0.001$). Despite adjusting simultaneously for age, gender, and the site and mechanism of injury, 'play' was significantly and independently associated with fracture occurrence as compared to ball sports, with an adjusted Odds Ratio that exceeded that of field sports and was equivalent to that of combat sports (Figure 2.3).

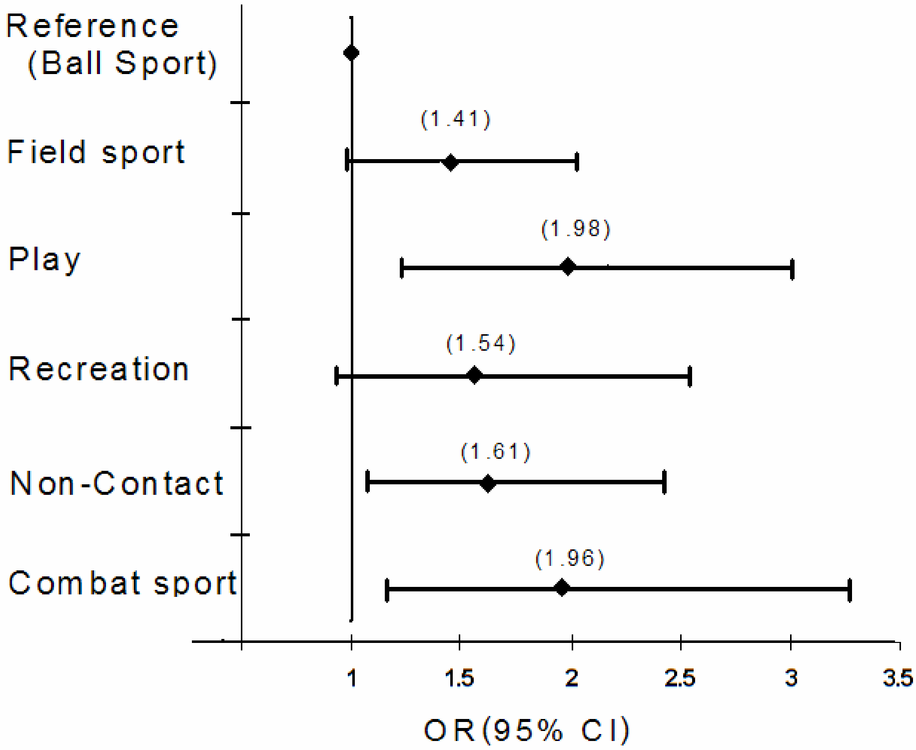
Table 2.4 Multivariate association of fracture occurrence among 3,712 Emergency Department Attendees with Sports and Recreational Injuries (SRI)

Category	Reference	OR (95% CI)	p-value
Age group	Paediatric Vs Adult	1.21 (1-1.45)	0.045
Sex	Female Vs Male	0.99 (0.81-1.21)	0.91
Site of injury	Upper Limb Vs Axial	5.82 (4.47-7.57)	<0.001
	Lower Limb Vs Axial	1.87 (1.4-2.5)	<0.001
Mechanism of Injury	External force # Vs Overexertion ‡	1.59 (1.2-2.13)	0.002
	Fall Vs Overexertion ‡	2.15 (1.6-2.89)	<0.001
Activity	Field sport Vs Ball sport	1.41 (0.98-2.02)	0.06
	'Play' Vs Ball sport	1.98 (1.23-3.01)	0.001
	Recreation Vs Ball sport	1.54 (0.94-2.53)	0.09
	Non-contact sport Vs Ball sport	1.61 (1.07-2.43)	0.02
	Combat sport Vs Ball sport	1.96 (1.17-3.27)	0.01

‡ Overexertion = Injury occurring due to patients own actions not involving external force, objects, or persons.

External Force = Collision, struck by ball, player, playing equipment, foul play.

Figure 2.3: Forest plot of adjusted odds ratio of fracture occurrence amongst Emergency Department attendees with Sports and Recreational Injuries, adjusted for age, gender, site and mechanism of injury.



Discussion

This work represents the first attempt to examine all-cause sports and recreation injuries resulting in ED attendance, in both paediatric and adult patients in a European population. My results highlight that SRI may potentially attenuate the secondary health benefits of exercise within a substantial proportion of subjects.

The mean daily proportion (SD) of ED attendances resulting from SRI was 13.6% (0.7).

Nearly one in five SRIs occurred in the residential setting. When the injury involved playground equipment, this rose to 46.5% and almost all ‘Bouncy Castle’- and trampoline- related injuries occurred in the home.

Fall was the mechanism of injury in more than a third of cases. Those who suffered a fall had a 3.7-fold increased odds of a fracture compared to the reference group (Table 2.3). A similar relationship persisted despite adjustment for age, gender, and site and mechanism of injury, between ‘play’ and fracture occurrence (adjusted OR: 2.45, p<0.001).

Fractures accounted for almost a third of total SRIs (32.4%). They most often occurred in the upper limb (69.8%).

The overall fracture rate is higher than the previously published figures (20%-22%).[135,144,147] The paediatric fracture rate was 40.5%, similar to some [152] but higher than other reports.[144,153]

Historically, SRIs were seen as trivial injuries that were managed but which were not a primary focus of the ED workload. My data agrees with several other studies from the U.S. and Australia[135,144,154] in showing that SRI are responsible for a substantial proportion of the current ED workload. This workload persists throughout the week, and thus is not primarily a weekend or Monday morning phenomenon. With the increased development of sports medicine as a sub-speciality, whether this work could be equally or perhaps more optimally managed in a specific sport injury clinic by individuals with specific training in both the acute management and subsequent rehabilitation of sports injuries, is worthy of investigation.

This analysis demonstrates that falling is a major mechanism contributing to SRI associated fractures. In the paediatric group, this may be contributed to at the individual level by underdeveloped balance due to higher centres of gravity [152] and poorer awareness of danger.[155] Initiatives have been aimed at the prevention of injury due to fall in a playground setting.[137,152] Falls were particularly associated with upper limb fractures presumably due to falling on the outstretched arm in an attempt by the subject to save themselves. Awareness, education, and training to limit injuries sustained from falling, may potentially impact on this association.

Tinsworth & McDonald found that the playground was second only to the home as a cause of accidental paediatric injury.[156] This is borne out in this work. Further, I noted that only 56 public playgrounds are available to the population in the area surveyed. Though public playgrounds are in themselves a source of many injuries, they are subject to quality control, supervision, and maintenance. This may not be the case in the home, where many of the recreation-related injuries occurred and where the potential for injury of several recreational activities such a trampolining may be underappreciated. The American Academy of Paediatrics has issued guidelines regarding the potential risks of injury and unsuitability of some playground equipment in the home, particularly trampolines.[157] Their use continues to increase however, as do the injuries resulting from them.

One in twenty (4.9%) attendees to the ED with a SRI are currently due to either bouncy castle or trampoline use, of which 40% suffered a fracture. The potential danger of poorly serviced, installed, and unsuitable equipment in a residential setting as source of injury cannot be overstated, and, as shown in this data, represents a substantial public health concern.

Though several studies have looked at recreational injury in a paediatric population, information regarding recreational, as distinct from sporting injuries, in the adult population is scant. As the population ages, people are less likely to be involved in the more popular team sports due to higher physical demands and time commitments required for these activities. It would appear, however, that though more senior 'recreators' appear to suffer fewer serious injuries/fractures than their younger counterparts, these types of injuries are more common than previously reported.

Several methodological limitations may be present in this work.

Using presentation to the ED as an inclusion criterion may underestimate injury occurrence. Not all SRI, even among those of moderate severity, present to the ED for assessment, estimated in one series to be of the order of 40% of those SRIs actually coming to medical attention.[144]

In this analysis I focus on fracture-occurrence as this is a clinically important outcome in its own right, with substantial implications for long term health and resource utilisation. In addition, recognising the difficulties of under-presentation of sports injuries to the ED, it is likely that a higher percentage of fractures will present to the ED, ensuring more complete case ascertainment. Some of these higher rates may result from the studied ED being a referral centre for orthopaedic injuries leading to a higher proportion of more serious injuries, as compared to more minor injuries, being referred to us, especially from more remote areas. The percentage of fractures relative to other soft tissue-related sports injuries may therefore be somewhat inflated. However, the actual number of fractures that occurred is an important observation in its own right and the above concerns should not in themselves bias my analysis of the factors associated with fracture occurrence amongst ED attendees. As this research is based in the South of Ireland, it includes injuries related to Gaelic games as well as more traditional European sports, I do not believe however, (but within the current dataset clearly cannot prove) that the inclusion of Gaelic games should unduly bias the associations of the other activities seen in my study.

It is impossible to define with any precision the number of subjects participating in sporting and recreational activities or the duration and intensity of this exposure, thereby preventing the calculation of appropriate event-rates. Difficulties surrounding event-rates have always been troublesome in sports epidemiology.[135,138,140,146,154]

I echo the Wanless report's call for a greater parity between prevention and cure.[47] Given that many soft tissue injuries are managed in the primary care setting, improved undergraduate and postgraduate training in musculoskeletal medicine, recognised as a poorly taught area,[158] may help to optimise injury management. Effective prevention however is at least equally as important but it must be evidence-based. In our current resource-starved health environment, the development and widespread application of cost-effective analysis is essential.

Conclusion

This data demonstrates several important observations. Recreational activity is a major source of SRI, especially for several childhood play activities such as trampolines and 'Bouncy Castles'. These findings concur with other studies[135,152,159] that the majority of SRI presenting to the ED relates to team balls sports. In the absence of participation rates, and purely on the basis of event rates my findings suggest that recreation in an unstructured environment, particularly the home, may be a significant cause of SRI. More than one half of all SRI presentations to the ED had seen their primary care physician prior to attending the ED and a third were referred back to them post injury. There is vast potential for patient education dealing with safe exercise/recreation, injury prevention, injury treatment, and the prevention of reoccurrence of injury. The primary care physician and the Emergency Department are ideally placed to address both prevention and cure but the sports physician is well placed to highlight these issues and to collate and develop advice for best practice.

Chapter 3

Risk Factors for Hand Injury in Hurling

This work has been peer reviewed and published as "Risk factors for hand injury in hurling: a cross-sectional study". Falvey E, McCrory P, Crowley B, Kelleher A, Eustace J, Shanahan F, Molloy MG. BMJ Open. 2013 May 28;3(5). PMID: 23793666. (Extracts reproduced with permission of the publishers).

Introduction

Hurling is the national sport of Ireland and is also played throughout the world among members of the Irish diaspora in North America, Europe, Australia, New Zealand, South Africa and Argentina.[160] Thought to predate Christianity, hurling has been a distinct Irish pastime for at least 2000 years. Stories of the hurling feats of Irish mythological heroes such as Setanta are recorded in ancient 12th century texts such as *Lebor Laignech* (The Book of Leinster).[161] One of Irelands' native Gaelic sports, it shares much with Scottish *shinty*,[162] *cammag* played on the Isle of Man, and *Bando* in Wales and England.[163] Hurling was played in Ireland in ancient times by teams representing neighboring villages. Games involved hundreds of players, which would last several hours or even days. In 1904, hurling was an unofficial demonstration sport in the St Louis Summer Olympic games and in the final; Chicago (Fenian FC) defeated St Louis (Innisfails FC).[164]

Reputedly one of the fastest team field sports, this amateur game is played by two teams of fifteen players who compete for a leather-bound ball (*sliotar*) using a metre-long piece of ash wood (*hurley*) as a bat (Figure 3.1).

Figure 3.1: Typical action in a game, a player rises to catch the *sliotar* despite the attentions of opponents. *Courtesy of Dan Sheridan, inpho photography.*

The standard hurling pitch is 135 – 145 meters long and 80 – 90 meters wide. Two posts, which are set 6.4 meters apart, and connected above the ground by a crossbar set at a height of 2.5 meters, form the goals at each end. A ball hit over the bar is worth one point. A ball that is hit under the bar is called a *goal* and is worth three points.[57]

Hurling differs from field hockey and lacrosse in that the *sliotar* can be caught in the hand and carried for not more than four steps, struck in the air, or struck on the ground with the hurley. Further, when the ball is struck for longer distances one of the greatest arts of the game is to jump and field the ball- while opponents are free to strike the ball with their hurley

(Figure 3.1). The player may kick or slap the ball with an open hand (the hand pass) for short-range passing.

In a 1984 study of Emergency Department (ED) presentations due to hurling injuries, Crowley et al noted that 28% of presentations were facial and head injuries and 36% were hand injuries.[18] Nine years later following the voluntary introduction of helmet and face protection the absolute number of presentations to ED due to hurling injury had almost halved.[19] The ratio of presentations of site of injury had also changed with 20% of presentations due to head injury and 56% due to hand injury. This relative rise in hand injury was also noted in a further study by Kiely.[21]

The most widely used, dedicated hand protection for hurling, commercially available was the Ashgard™ glove by O'Dare (Figure 3.2). This is constructed of neoprene and elasticised fastenings; this apparatus focuses primarily on protecting the metacarpal bones. This was the most commonly used device at the time of this study. Anecdotally, and in discussion with other physicians caring for the hurling community, (personal communication Prof M G Molloy) I observed relatively poor levels of use of this equipment, this despite published ED injury presentations and recommendations.[19,21]

Figure 3.2: Typical action, showing players with helmet, helmet and face protection, and hand protection, the Ashgard™ hand glove is shown in inset. *Courtesy of Dan Sheridan, inpho photography*.

This study aims to quantify the occurrence of hurling-related hand injury presenting to the ED and examine the variables, which may be associated with hand injury. In particular, to assess the association of helmet and facial protection with the occurrence of hand injury in this population, and to describe the impact that this has on time lost from play and work.

Methods

Consecutive hurling-related injuries over a 3-month period, July to September, in 2006 presenting to the ED of a university hospital were recorded. At the time of each patient's assessment a questionnaire was completed by their treating emergency room physician regarding the nature and circumstances of their injury, and their subsequent investigations and management.

In total 430 hurling-related injuries presented to the ED in the defined period. Due to the enforcement of the use of helmet and face protection by many juvenile clubs (catering for players of 16 years and younger), I excluded this population (n=231). This enabled a true reflection of equipment use in the adult/voluntary setting. The remaining 199 patients were contacted for a telephone interview. Prior to the interview, patients were contacted by telephone to give their consent to their participation in the study. Interviews were completed within 90 days of initial presentation to the ED (Mean 68 days (15-88)). The subjects also received background information about the study based on the Ethics Committee approval as well as a plain language statement. Telephone calls followed a scripted protocol to avoid investigator bias. (Appendix 1) The questionnaire consisted of questions focusing on:

- Site of injury;
- Mechanism of injury;
- Protective equipment in use at the time of injury;
- Previous injury;
- Previous use of protective equipment;
- Reasons for discontinuing use of protective equipment.

Those who had tried but discontinued hand protection were given five potential options as to why they discontinued use of hand protection:

- Discomfort
- Ineffective protection
- Limitation in performance
- Poor aesthetics
- Expense

Those players who had discontinued use of hand protection were asked if they would consider trialling different protection if it were to become commercially available.

Previous injury was defined as a physical injury, suffered while playing hurling, resulting in at least one game missed. To aid analysis of data, upper limb injuries were classed as proximal or distal. A proximal upper limb injury occurred at the wrist or in the upper limb proximal to the wrist (forearm, elbow or shoulder); a distal upper limb injury described all upper limb injury distal to the wrist. An injury which resulted from an action of an opposing player which was penalised by the referee was documented as 'foul play'.

I was particularly interested in exploring the use of protective equipment and whether or not this impacted on injury presentations to the ED. Based on the hypothesis that use of protective equipment has been linked to increased levels of “risky behaviour’ I focused particularly on those with serious hand injury and whether they used helmet and face protection.

The study proposal was approved by the Clinical Research Ethics Committee of the Cork Teaching Hospitals.

Statistical analysis

Tests for normality were performed using Shapiro-Wilks test. All variables in the analysis were normally distributed and therefore described using means and standard deviations. Proportions were compared using Chi square tests. Univariate associations of upper limb injury were examined using logistic regression analysis. The presence, strength, independence, and significance of upper limb injury with the use of helmet with faceguard was quantified using logistic regression. This was adjusted simultaneously for age, previous hand injury, being struck directly by a hurley and foul play. Variables that were significant using Pearson chi-squared test were included in the multivariate logistic regression model as were those variables deemed clinically important. The final model examines the association of upper limb injury with use of helmet and face-guard, adjusted simultaneously for age, previous hand injury, being struck directly by a hurley and foul play. The factors associated with hand injury were analysed by comparing those with confirmed upper limb injury (n=100) with those injured elsewhere (n= 63). Analysis was performed using SPSS version 12 with a 2 sided type one error rate of 0.05, (Chicago, Illinois).

Results

Hurling-related injuries for 430 patients were reviewed from 3172 consecutive sports injuries presenting in the defined period. Of 199 identified and suitable patients, 27 subjects were uncontactable, and 9 declined to participate. The total response rate was 82% of possible subjects. Data on 163 patients were included.

Interviews were conducted with 17 women and 146 men (n= 163). Average time to follow-up was 39 weeks (range 28 – 48 weeks) post injury. Patient's ages ranged from 17-39 years (mean 23.52 yr). The majority of injuries occurred in organised competition or supervised practice, n = 155 (95%).

Injury site and mechanism of injury

The most commonly injured site (Table 3.1) was the upper extremity distal to wrist, 85 (52.1%) followed by lower limb 30 (18.4%), with 27 head injuries (16.6%). A statistically significant number of the distal upper limb injuries sustained from a blow of a hurley were fractures n=46/74 (62%), compared to soft tissue injury (laceration, ligamentous injury) n=28/74 (38%), [Pearson Chi square $p<0.001$]. The most commonly injured digits were the 1st (n= 16, 35%) and 5th (n=15, 33%). The metacarpal bones were most commonly fractured (n=17, 37%) followed by the proximal phalanges (n=15, 32%), the middle phalanx was least likely fractured (n=4, 8%) and the distal phalanx was fractured in 10 cases (22%).

Table 3.1 Patient demographics, protection used, injury severity

Patient Characteristics	Total n=163 (%)	Hand Injury n= 100 (%)	No Hand Injury n=63 (%)	P value
Age, yrs				
Range	17-39yr	17-33yr	17-39yr	
Mean (sd)	23.51 (4.1)	23.51 (4.2)	23.52(4.1)	
Gender				
Female	17 (10)	10 (10)	7 (11)	
Male	146 (90)	90 (90)	56 (89)	
Site Injured				
Distal Upper limb	85 (52.1)	85		
Proximal upper limb	15 (9.2)	15		
Lower limb	30 (18.4)		30 (48)	
Axial	33 (20.3)		33 (52)	
Protection used				
Helmet with faceguard	106 (65)	74	32 (51)	0.002
Hand protection	8 (5)	4	4 (6)	NS
Injury severity				
Fracture	74 (45.4)	60	14 (22)	<0.001
Variables associated with Injury				
Foul play	26 (15.9)	16	10 (16)	NS
Struck by hurley	104 (63.8)	74	30 (60)	NS
Previous hand injury	82 (50.4)	57	25 (40)	0.03

Previous injury

Most patients had suffered at least one injury in the past, n = 116 (71.2%), two thirds of patients had between 1 and 5 previous injuries (n = 108, 66.3%). Eight patients (4.9%) had more than six previous injuries. Fifty percent (n = 82) of patients had previously suffered an upper limb injury, 39% (n = 64) had suffered a prior head injury, and a fifth (20.9% (n = 34)) had experienced both. One third (35%) of those presenting with a fracture to the hand or fingers had suffered a prior fracture to the area. A history of previous upper limb injury was associated with further injury of the area, OR 1.31 (95% CI1.02-1.68).

Protection used

Only 8 (4.9%) used hand protection (Ashgard™ by O'Dare, Figure 3.2), while 149 (91.4%) had tried it in the past. Helmet with face protection was used by 65% (n = 106). At the time of study helmet and faceguard use was voluntary in adult participants. Previous trial of helmet with face-guard, and hand protection was reported by 154 (94.5%). Given this high trial-rate, yet poor uptake, respondents were asked why they had discontinued use. Most respondents, n=123 (75.4%), described poor utility citing issues such as bulkiness and diminished dexterity. More than half, n= 95 (58.3%) felt protection was inadequate, rendering the hand protection ineffective. When asked about potential interest in new protective equipment, 121 (74.2%) felt they would try a newly designed glove.

Univariate associations with hand injury

Univariate analysis of the variables associated with hand injury demonstrated a statistically significant association between prior injury, wearing a helmet and faceguard and being struck by a hurley (Table 3.2). The latter two relationships persisted on multivariate analysis respectively, independent of adjusted variables. (Table 3.3)

Table 3.2: Univariate associations with hand injury in hurling

	Upper limb injury n =100	**No upper limb injury** n = 63	**OR (95% CI)**	**p-value**
Helmet with faceguard n=106	74 (69.8%)	32 (30.1%)	2.76 (1.42-5.37)	0.003
Previous hand injury n=82	57 (69.5%)	25 (30.5%)	1.88 (1.46-4.94)	0.032
Age less than mean - 24yrs n=52	31 (59.6%)	21(40.4%)	1.05 (0.56-1.97)	0.88
Struck by a hurley n=104	74 (71.2%)	30 (28.8%)	2.31 (1.23-5.22)	0.009
Foul play n=26	16 (61.5%)	10(38.5%)	1.01 (0.43-2.4)	0.983

OR= Odds Ratio, CI= Confidence Interval

Table 3.3: Logistic Regression analysis of the association of hand injury with helmet and faceguard use (OR, 95% CI).

Category	OR (95% CI) for Upper Limb injury	p-value
Helmet with faceguard	3.15 (1.51-6.56)	0.002
Struck by a hurley	1.99 (1.24-3.8)	0.013
Age from mean	0.82 (0.4-1.68)	0.59
Previous hand injury	1.73 (0.90-2.6)	0.73
Foul Play	1.32 (0.49-3.5)	0.98

OR= Odds Ratio, CI= Confidence interval

Impact of hand injury

A week or more of play was lost by 152 (93.3%) of those injured, 89 (54.6%) lost more than 4 weeks. Due to their injuries, 71 (43.6%) people missed work, with 26 (16%) people missing more than 4 weeks of work.

Discussion

I report that in this retrospective cross-sectional study of 163 hurling players presenting to a university hospital emergency department with hurling –related injury, hand injury was significantly associated with use of helmet and facial protection, independently of age, previous hand injury, being struck directly by a hurley and foul play. While in this cross-sectional study I cannot demonstrate causality, this finding raises interesting questions regarding the epidemiology of hurling-related hand injuries in the era of voluntary helmet and face protection use in hurling.

Published data on the incidence of hurling-related hand injury is sparse. The available literature however suggests that while the occurrence of head and facial injury in hurling has fallen, the proportion of players presenting with hand injury remains essentially unchanged. Crowley et al. reported that 52% of ED presentations for hurling injury were injuries to the hand. Eight years later this proportion was similar at 56%,[19] and is comparable to the 62% observed in the current study. Despite hand injury being a common occurrence, only 8% of adults reported use of commercially available hand protection, similar to the 9.8% reported by Kiely et al. in a 2003 study.[21] No rules are enforced in hurling regarding the use of hand protection. In the U.S. the National Collegiate Athletic Association (NCAA) dictates that gloves be worn in intercollegiate stick-handling sports (men's lacrosse, women's lacrosse, and men's ice hockey).[165-167] These sports have many similarities to hurling. The major difference between these sports and hurling is that the puck or ball is not handled by outfield players. Therefore a bulky glove may be worn without affecting dexterity or impeding play. The technical requirements of a hand protection device in hurling therefore differ and at the time of study had not gained acceptance among those players presenting to the ED.

Previous injury patterns reported by patients may provide some insight into the role of an individual's behaviour in exposure to further injury. I report that 50% of patients had previously suffered an upper limb injury, 39% had suffered a prior head injury, with 21% experiencing both in the past. Sixty-five percent of this cohort wore helmet and face protection voluntarily, demonstrating risk awareness regarding potential head and facial injury. A similar usage of hand protection was not observed. Why the majority of players would adopt head and face protection while discontinuing hand protection use cannot be addressed in this cross-sectional study. This may in part be explained by the large emphasis placed on head protection [18-20] by the sports body and injury commentators. Little emphasis has been placed on hand injury and protection.[21] The utility and function of commercially available hand-guards may also play a role. The Ashgard™ model was described as 'uncomfortable' and 'bulky' by players, and did not protect beyond the first phalanx, 30% of fractures were seen beyond this site (n=14). A more anatomically correct model (Mycro Long Finger Glove™) has been available on the market in more recent times (Figure 3.3). This glove protects the metacarpals, and offers greater protection for the phalanges, utilising hardened plastics over the phalanges – providing protection without loss of dexterity.

Figure 3.3: The more recent Mycro Long Finger Glove™ offering greater protection to the phalanges

The significant relationship of a number of variables such as helmet use; being struck by a hurley; and previous hand injury may represent altered behaviour on behalf of both the injured party and the party causing the injury. It could be argued that the use of helmet and face protection has altered player behaviour leading to more hazardous playing style. The concept of risk 'compensation' or 'homeostasis' has been debated following the introduction of many safety measures in many sports such as American football,[44,45] cycling[168] and even on the introduction of the automobile seatbelt.[59,69] In American football the evolution of the helmet over 50 years from a leather helmet to a metal and plastic hardshell helmet with faceguard drastically changed tackle patterns. The 'spear tackle' saw players tackle with the head rather than shoulder- this was accompanied by a dramatic rise in catastrophic brain and cervical spine injury. Banning the spear tackle and ensuring helmet specifications led to a 42% decrease in brain and spinal injury over a 5 year period.[45] It has been argued that cyclists are less likely to ride cautiously when wearing a helmet owing to their feeling of increased security.[168] A level of perceived safety has been postulated to lead to increased levels of 'risky behaviour'[59]- in hurling it could be postulated that helmet with face protection increases the likelihood that a player will attempt a more risky aerial catch such as seen in Figure 3.1.

The majority of the injuries reported upon in this study occurred during organised competition or supervised practice at club events. The apparent success of the introduction of head and facial protection occurred because this level of regular supervision allows the enforcement of mandatory use laws. The use of helmet and facial protection was made mandatory for initially all players under-18, then all players under-21 in 2003 and 2005, respectively. I studied this group in the period prior to 2010 when it became mandatory to wear this protection for all players. Players are not insured to train or play at their clubs without the correct head and face protection. Further prospective studies evaluating the effect of hand protection on the occurrence of hurling-related hand injury are warranted to determine if the mandatory use of such protective equipment would result in a comparable decrease in injury.

This data describes the impact of upper limb and hand injury both on return to sport, and time lost from work. Almost one-fifth of all hurling related hand injuries resulted in more than 4 weeks off from work. Though upper limb injury is often regarded as being less serious than head injury such as eye injury, studies have shown that hand is likely to take longer to return to pre-injury activity than injury to other parts of the body.[169,170] Trybus et al. showed more than 50% of hand injuries presenting to a specialist centre suffered persistent post-traumatic disability.[170]

Limitations of this study included the retrospective nature of the self recorded data obtained by telephone interview; however the initial ED presentation data were gathered prospectively with follow-up performed to investigate factors associated with these injuries. The 82% response rate which may have resulted in selection bias in the data provided- non-responders may have had different attitudes regarding hand protection. This work investigates hurling-related hand injury presenting to the ED (and compares to other studies gathering data by the same means), it may therefore bias the analysis toward serious injury. Two prospective studies on 74 and 127 players, revealed hand injury rates of 33% and 15.2% respectively.[52,171] These lower rates may represent a 'dilution' of more serious injury among less serious, minimal time loss injury. This work emphasises the high occurrence of hand injury, which remains in hurling with a significant fracture rate. The study has attempted to highlight factors associated with this, and, I feel, poses some important questions as to the behavioural changes that may accompany the introduction of safety equipment. Answers to these questions may help to inform future decisions regarding safety equipment development and use in hurling.

Chapter 4

Exercise Induced Asthma and Exercise Testing in an International Rugby Union Team

This work has been peer reviewed and published as “Exercise-induced bronchoconstriction and exercise testing in an international rugby union team”. Falvey EC, McCarthy C, O'Connor TM, Shanahan F, Molloy MG, Plant BJ. Thorax. 2010 Sep; 65(9):843-4. PMID: 20805187. (Extracts reproduced with permission of the publishers).

Introduction

Exercise Induced Bronchoconstriction (EIB) is an acute, transient airway narrowing that occurs during or after exercise[90] and is objectively defined as a ≥10% decline in FEV_1 after appropriate exercise provocation.[91] In the general population the incidence of asthma is 6-12%[93,94] and EIB has been reported as affecting 12%.[95,96] In those known to be asthmatic, exercise is a common trigger of bronchospasm and bronchial hyper-reactivity (BHR) may occur with exercise in 50-90% of asthma sufferers during or after exercise.[95] In elite athletes EIB has a prevalence of 11 to 50%, depending on the sport.[96]

In the general population asthma is a cause of unexpected sudden death[105]. A recent study highlighted that in the general population less than 25% of asthmatics carry rescue medication when exercising[172] and in one series over seven years asthma-related death occurred in 61 cases over a 7–year period.[107] There is increasing concern that poorly controlled EIB can result in recurrent airway inflammation resulting in permanent airway remodelling.[109]

The symptoms associated with EIB include fatigue or dyspnoea during exercise and are therefore often incorrectly attributed to deconditioning in athletes.[101-103] EIB is thought to be more common in endurance sports, particularly winter sports [92] where prolonged periods of hyperpnoea in cold, dry air are typical.[104]

The prevalence of EIB in Rugby Union players is unknown despite the popularity of the sport, with over 2 million players in 123 countries. A professional sport since 1998, elite international players now play, on average, 45 games per year.[173] The importance of the potential influence of EIB in rugby is highlighted by this dramatic rise in popularity, the advent of professionalism, and the rugby season spanning winter months.

Reasons for the under-investigation of EIB in certain sports may include unfamiliarity with the diagnosis, the presumption that players performing at the highest level have been 'naturally selected' precluding underlying diagnoses such as EIB and the lack of access to lab-based tests such as eucapnic voluntary hyperventilation (EVH).

General guidelines for EIB pulmonary function testing include an exercise challenge in ambient laboratory conditions of 6–8 min duration at an intensity of ~85% of predicted peak heart rate (HRmax) [174,175] , however in many cases symptoms do not appear unless the exercise intensity approaches competition level (90–100% HRmax).[176] Traditional standardised field tests such as the Coopers 12-minute run[177] or the modified progressive 20-m multistage fitness test - the Yoyo intermittent endurance test (YIET)[178] do not translate into certain sports including rugby, particularly given that modern professional methods of training focus both on prolonged aerobic activity and high intensity anaerobic bursts. Long regarded as a sport that caters for all 'shapes and sizes' rugby sees backs as light as 75kg play against forwards of more than 125 kg. The variable physical demands of play phases are reflected in player anthropometry. Play phases such as the scrum, rucks, mauls (generally the domain of the larger forwards) and tackling require significant recruitment of trunk and upper limb musculature, whilst certain backline players must emphasise high-intensity explosive recruitment of lower limb musculature (and are usually of smaller stature).

To reflect this level of exertion any functional test, proposed as a sports specific exercise challenge, needs to recruit not only lower limb musculature but must encompass whole body activity. The unique upper body demands of rugby are not easily recreated but I recognised similarities between the training and conditioning regimens employed in rugby union and amateur boxing. In conjunction with the high performance unit of the Irish Amateur Boxing association I altered an exercise programme of theirs to meet this requirement.

In order to address the hypothesis that EIB may be under-reported and undertreated in rugby, I developed a rugby-specific exercise protocol (Appendix 2). This in conjunction with a questionnaire (Appendix 3) and spirometry testing, were used to determine the prevalence of Asthma and EIB in a cohort of professional rugby players.

Methods

Participant recruitment

All players in the Irish Senior Rugby squad, attending pre-season training camp gave informed consent (Appendix 4) for their participation in this study. Those subjects who had previously been placed on asthma medications (following confirmation of diagnosis via provocation testing as per International Rugby Board guidelines) were allowed to use them as normal. All test subjects were advised to refrain from caffeine and a warm-up exercise on the day of the testing session.

Symptom questionnaire

Each participant at recruitment completed a questionnaire based on a previously validated sports-specific questionnaire for asthma and exercise induced respiratory symptoms and atopy prior to exercise testing.[110,179] (Appendix 3)

Whole body exercise test

An exercise programme was developed in conjunction with the high performance unit of the Irish Amateur Boxing Association to reflect the level of exertion experienced by elite rugby players where both lower limb and whole body musculature are recruited intermittently.[180] This test is a combination of exercise and sprinting with a 4 kg exercise ball. Three athletes performed the test at a time to promote competition and tester supervision by the first author ensured correct task completion. The test was designed to last for at least 8 minutes with actual test completion time varying depending on the speed of execution**.** The full test protocol is provided in Appendix 2.

Perceived exertion was measured at test completion on a visual analogue Borg scale.[181] All athletes wore heart rate monitors (Polartec®, Lawrence, MA). Serum lactate was measured from a whole blood sample 1 minute after exercise cessation using the Lactate Pro Meter Set (Biomedic Labs, Durango, CO).

Pulmonary function measurement

Pulmonary function was measured using a calibrated, computerized, pneumotachograph spirometer (MicroLab 3500, Micro Medical Ltd., Chatham, Kent, United Kingdom), which was validated by the Respiratory Function Laboratory at Cork University Hospital each day prior to testing, conforming to international guidelines.[182] Spirometric analysis recorded individual Forced Expiratory Volume in first second (FEV_1), Forced Vital Capacity (FVC), FEV_1/FVC ratio. A fall in FEV_1 $\geq$10% from baseline was considered positive for EIB. Temperature and humidity readings were recorded by the Irish National Meteorological Service at the time of each testing.[183]

Statistical analysis

For the purpose of analysis players were grouped into 2 cohorts. An airflow obstruction (AOG) group (Any player with a previous diagnosis of asthma or EIB or those with positive baseline spirometry), and a non-airflow obstruction (NAOG) group (no previous history of asthma, EIB or negative spirometry). Proportions were compared using Chi square tests. Analysis was performed using SPSS V13, (SPSS Inc, Chicago, Illinois) using a 2 sided type I error rate of 0.05.

Results

Forty-two players were tested, Table 4.1 characterises the subjects' characteristics, heart rate and levels of perceived exertion and serum lactate. These did not differ significantly between both groups. Of the 42 recruited, 12 (29%) demonstrated airflow obstruction. There was a trend towards an increased prevalence of airflow obstruction in players whose normal position in the team was as a back (42%) compared to forward (17.4%) (p=0.078).

Table 4.1; Player Anthropometry, heart rate and levels of perceived exertion.

	Total (n=42)	**NAOG** (n = 30)	**AOG** (n=12)
Mean Age (yrs)(range)	26.5 (20-33)	26.1 (20-33)	27.6 (23-30)
Mean Height (m)(range)	1.86 (1.72-1.98)	1.87 (1.77-1.96)	1.83 (1.72-1.98)
Mean Weight (kg)(range)	99 (73-116)	101 (83-116)	95 (73-116)
Position Back	19	11	8
Forward	23	19	4
Mean Heart Rate (bpm)(range)	175 (156-195)	174 (156-195)	176 (162-192)
Mean Perceived exertion (range)	15.6 (14-18)	15.5 (14-17)	15.8 (15-18)
Mean Lactate(range)	11.3 (5.7-16.2)	11.3 (8.1-16.2)	11.2 (5.7-15.8)

AOG= Airflow obstruction group, NAOG= Non-airflow obstruction group

Rugby-Specific Questionnaire

Table 4.2 summarises the results from the rugby specific questionnaire. Wheeze was reported by 42% (n=5) of the AOG, and 7% (n=2) of the NAOG (p=0.006). Four of the AOG (33%) had been woken from sleep by dyspnoea, none of the NAOG reported this symptom (p=0.001). Exercise increased dyspnoea (42% versus 10%; p=0.015) and cough (58% versus 20%; p=0.047) was reported in the AOG compared with the NAOG.

Table 4.2: Summarised results of study questionnaire.

	NAOG (n = 30)	AOG (n=12)	**Sensitivity (%)**	**Specificity (%)**	**p-value**
Symptoms					
Wheezing	2 (7%)	5 (42%)	42	93	0.006
Woken by Dyspnoea	4 (33%)	0	33	100	0.001
Attack of Dyspnoea	4 (33%)	1 (3%)	33	97	0.001
Atopy	2 (17%)	4 (13%)	17	87	0.78
Pneumonia	2 (17%)	2 (7%)	17	93	0.318
Exercise specific symptoms					
Dyspnoea post exercise	5 (42%)	3 (10%)	63	79	0.015
Cough post exercise	7 (58%)	6 (20%)	54	83	0.047

AOG= Airflow obstruction group, NAOG= Non-airflow obstruction group

Pulmonary function tests

Table 4.3 summarises results of pulmonary function testing. Seven players had been previously diagnosed with asthma and were on therapy at the time of study. Of this group, 4 (9.5%) had a greater than 10% drop in FEV_1 post exercise challenge.

A further 3 athletes who had a positive exercise challenge test had had a diagnosis of asthma made as children but no longer took any medication. One of these had obstructive spirometry even prior to testing, a second had a strongly positive response to exercise challenge (FEV_1 decreased by 18%). Two athletes with no previous history of asthma or airway obstruction had a significant drop in FEV_1 after exercise challenge, demonstrating EIB.

Table 4.3: Spirometric analysis pre- and post-exercise challenge

	AOG (n=12) Pre-exercise	AOG Post-exercise	NAOG(n=30) Pre-exercise	NAOG Post-exercise
FEV_1 (**L** $\pm$ SD) (**% predicted** $\pm$ SD)	4.53 ± 0.96 (98 ± 14.9)	4.14 ± 0.93 (90 ± 15.7)	4.8 ± 0.62 (100 ± 11.8)	4.86 ± 0.62 (101 ± 12.3)
FVC (**L** $\pm$ SD) (**% predicted** $\pm$ SD)	5.79 ± 1.04 (105 ± 11.2)	5.52 ± 1.06 (100 ± 11.7)	5.92 ± 0.68 (102 ± 10.4)	5.93 ± 0.68 (103 ± 10.6)

AOG= Airflow obstruction group, NAOG= Non-airflow obstruction group, FEV_1 = forced expiratory volume in 1 second, FVC = forced vital capacity.

Climactic conditions were stable throughout the study. Temperature 14.3˚(11˚-16˚), and Humidity 84% (75-92%) were provided by the Irish Meteorological Bureau [Mean(range)].[183]

Discussion

This is the first paper to document the prevalence of EIB in a group of elite rugby players. I also propose a sports specific field test as a screening tool in professional rugby. Almost a third (29%) of a full international professional rugby squad had confirmed asthma or EIB, as compared with 12-15% of the general population.[184] This group undergo annual occupational medical screening. Despite this, 5 "new" untreated cases

(12%) were elicited by this challenge test and in the group already on treatment for asthma/EIB; over 50% still displayed EIB.

Exercise performance was a poor indicator of airway limitation. It is important to highlight that athletes with airway obstruction had similar perceived exertion, heart rate and serum lactate scores to the control group. This supports the theory that elite athletes may function at a very high level despite suboptimal treatment. Asthma and EIB do not preclude elite participation, nor does elite participation preclude the presence of asthma or EIB. Whether treating EIB enhances performance is of great interest but is beyond the remit of this study.

While the presence of EIB may not impact on an athlete's ability to perform in the present, it may have implications for long-term health. A drop in FEV_1 after exercise is explained due to exercise induced airway inflammation. Histological studies in cross-country skiers have supported the concept that untreated EIB may cause future irreversible airway remodelling.[109] The importance of carrying rescue medication for asthmatics during exercise cannot be overemphasised. A recent study highlighted that in the general population less than 25% of asthmatics carry rescue medication when exercising[172] and in one series over seven years asthma-related death occurred in 61 cases over a 7–year period [107]. A number of the athletes in this study who had a prior diagnosis of asthma as children were no longer being treated for asthma and a significant number had EIB despite treatment. This has implications for player safety.

The questionnaire used was specific but not sensitive, in keeping with previous studies.[92,102] My study supports that wheeze, being woken from sleep by dyspnoea and cough after exercise are important symptoms in rugby players which if present warrant further investigation. The trend towards an increased incidence of EIB in backs warrants further study to truly delineate whether this is a real finding.

I do not know how the field test used in this study and spirometry compares to eucapnic voluntary hyperventilation (EVH), or other laboratory-based tests to confirm EIA/EIB as recommended by the International Olympic Committee (IOC). It should be noted that no gold standard test exists.[185] There are many benefits to this sport-specific test; all players completed the protocol and the testing system was portable, easy to use, instruction for other testers is straightforward and it is an extremely cost effective screening tool.

Field testing is recognized by the world anti-doping agency (WADA) as an appropriate provocation test for the diagnosis of EIB.[186] General guidelines for EIB pulmonary function testing include an exercise challenge in ambient laboratory conditions of 6–8 min duration, at an intensity of ~85% of predicted peak heart rate (HRmax). [174,175] In many cases however symptoms do not appear unless the exercise intensity approaches competition level (90–100% HRmax).[176] My test achieved this level of exercise intensity. Traditional standardized field tests such as shuttle runs or 'Cooper's run' do not translate into certain sports including rugby, particularly given that modern professional methods of training focus both on prolonged aerobic activity and high intensity anaerobic bursts. The variable physical demands of play phases are reflected in player anthropometry. Play phases such as the scrum, rucks, mauls (generally the domain of the larger forwards) and tackling require significant recruitment of trunk and upper limb musculature, whilst certain backline players must emphasize high-intensity explosive recruitment of lower limb musculature (and are usually of smaller stature). The combination of exercises and sprints with a 4 kg exercise ball used in my study simulated this and address many of the criticisms of exercise and field testing highlighted previously.[89] In contrast to other field challenges this both resembles game activity and is a standardised challenge for each player.

My data may under-report the prevalence of EIB amongst professional rugby players. Exercise load,[104,176] relative humidity,[98] and air temperature[119] are identified as the three major factors that may influence the sensitivity of field testing. These tests were performed in the preseason, during summer in humid conditions and may therefore underestimate the problem.[187] Though the professional season spans the winter months, club and other international commitments, as well as highly tailored individual training programmes and injury meant that the preseason ‘summer camp’ was the only time the entire squad trained together.

Exercise induced cough or dyspnoea are useful clinical screening questions for EIB in this cohort. Wheezes, dyspnoea at rest, awakening from sleep due to dyspnoea, and a paroxysmal attack of dyspnoea as symptoms of exercise induced airway obstruction, whilst specific, had poor sensitivities and are of limited value in the clinical assessment of EIB.

In conclusion I believe that all professional rugby players should be screened for EIB. Useful questions for exercise-induced symptoms are the presence of cough and dyspnoea after exertion. Eucapnic voluntary hyperventilation (EVH) remains the most recommended laboratory test for EIB but ease of use in a team environment suggests that the 8-minute all-out field test in conjunction with portable spirometry is a worthwhile alternative. Definitive studies comparing the efficacy of this method against validated laboratory tests are required. In the meantime however, this work provides an effective cheap and acceptable means of testing for EIB in a rugby population.

Chapter 5

Iliotibial Band Syndrome: An examination of the evidence behind a number of treatment options.

This work has been peer reviewed and published as “Iliotibial band syndrome: an examination of the evidence behind a number of treatment options”. Falvey EC, Clark RA, Franklyn-Miller A, Bryant AL, Briggs C, McCrory PR. Scand J Med Sci Sports. 2010 Aug; 20(4):580-7. PMID: 19706004. (Extracts reproduced with permission of the publishers).

Introduction

Iliotibial band (ITB) syndrome (ITBS) is the most common cause of lateral knee pain in athletes, occurring with a reported incidence as high as 12% in runners and up to 22% in military recruits.[128,132,133,188-190] ITBS also presents in athletes who participate in running, cycling, dancing, volleyball, tennis, football, skiing, weight lifting, and aerobics.[128,133,189,191-194] It is also a common presentation in exercising adults, occurring in up to 15% of women and 7% of men.[195]

The ITB as described in classical textbooks is a structure *'over the lateral femoral aspect [where] fascia lata is compacted into a strong iliotibial tract…'.*[129] More current thinking however, focuses on the ITB as a discrete entity, passing from the iliac crest to the lateral (Gerdy's) tubercle of the tibia.

Patients with ITBS complain of pain along the lateral aspect of the knee, specifically at the lateral femoral condyle.[196] It is often assumed that ITBS results from inflammation secondary to friction of the band across the lateral femoral epicondyle in flexion and extension.[128,193,197] Pain is most acute at 30˚ flexion, particularly affecting the more posterior fibres,[198] and an accompanying bursitis is also described. The gross pathology and histopathology of ITBS has been described in tissue obtained at surgery.[199] Features of chronic inflammation characterize the macroscopic appearance of tissue between the distal ITB and the lateral femoral condyle, but a true bursa does not appear to be characteristic of the pathology.[199-201] These findings have been supported by reports of magnetic resonance imaging findings in patients with ITBS.[196,198]

More recently a prospective study performed in a healthy athletic population found that a greater strain rate was seen in the ITB of those athletes who developed ITBS when compared to those who did not.[202] Numerous biomechanical risk factors have been proposed (e.g. genu varus, abnormal lower limb alignment, foot biomechanics and body type etc) but well-conducted scientific studies to link these factors in a causal relationship to the development of ITBS in runners are limited.[128,133,189,203,204]

A number of treatment modalities have been suggested for the management of ITBS. These include rest, pool running, reducing the amount and intensity of running, ice, stretching and strengthening of hip abductors, podiatric assessment, massage, and oral non-steroidal anti-inflammatory drugs.[128,131,134,189,205-207] There are only two published randomised controlled trials regarding appropriate treatment which suggest a role for injected corticosteroid ITBS[203] and a combination of anti-inflammatory/analgesics together with physiotherapy[208] in early (< 2 week duration) ITBS. A consensus of opinion regarding best practice in the treatment of ITBS has however not been reached.

Given the ambiguity surrounding aetiological factors for ITBS, the authors noted the relative lack of evidence-based treatment of ITBS. I therefore undertook a novel cadaver-based study coupled with an *in vivo* biomechanical analysis of ITB strain during movement to investigate both the mechanism of proposed aetiological factors, and to examine the anatomical principles upon which a number of the traditional treatments of ITBS are based. My hypothesis is that a number of the traditional treatments aimed at local inflammation and stretching the ITB derive from an incorrect understanding of the relevant anatomy and pathology.

Methods

This investigation was composed of three independent sub-experiments in an attempt to answer the proposed questions via a natural progression of research. This work was completed over a 5-month period from May to October 2007 (EF and AFM worked exclusively on this project over that period). The initial study consisted of mapping the anatomical landmarks and structure of the ITB. Based on these observations, the second experiment determined the location for a mechanical strain sensor and incorporated an assessment of three different proposed ITB stretches. These two experiments were performed using cadavers. The final *in vivo* study examined strain in the ITB during tensioning in professional athletes.

Study 1: Cadaveric anatomical studies of ITB

Cadaveric specimens

20 adult, formalin fixed cadavers were examined (age: 79±12yr, height: 1.66±0.14m, body mass: 69.4±14.9kg). Cadavers were preserved with standard formalin embalming fluid under routine process. The Department

of Anatomy and Cell Biology, University of Melbourne supplied all material. Information on age, gender, and cause of death was provided in accordance with the University of Melbourne Human Ethics Committee approval of applied and clinical investigations utilising cadaver tissues. Information regarding occupational history or previous physical activity levels was not available.

Cadaveric Manipulation

Limb alignment was assessed by measuring Q angle with an angle of $<13^0$ (male) and $<18^0$ (female) confirming a normal configuration.[209] All limbs were fully flexed at hip and knee prior to positioning in extended, supine anatomical position. Dissection was performed initially in prone then supine positions, to allow removal of all skin and subcutaneous fat from the lower limb. Superficial attachments of the fascia lata and ITB were noted and recorded.

Outcomes

Deep dissection was then performed to investigate:

1. Origin of the ITB and the relationship to Tensor Fasciae Latae (TFL)
2. Location of the insertion of Gluteus Maximus into the ITB
3. Location of the longitudinal attachment of ITB to the linea aspera
4. Site of attachment of ITB to the lateral femoral condyle

Study 2: Cadaveric ITB strain

Subjects

The Department of Anatomy and Cell Biology, University of Melbourne provided 5 unembalmed fresh-frozen cadavers (age: 76±10yr, height: 1.74±0.08m, body mass: 73.4±18.6kg) in accordance with the University of Melbourne Human Ethics Committee approval of applied and clinical investigation using cadaver tissues. All cadavers were thawed for 24-36 hours at 4˚C prior to testing to ensure complete thawing.

Cadaveric Manipulation

The cadaver was positioned supine in the anatomical position on a metal dissection table. The three tests performed are shown in Figure 5.1:

1. Control Variable - Straight leg raise to 30^{o} (SLR[210])
2. Experimental Variable 1 - Modified Ober test[211] (OBER)
3. Experimental Variable 2 - Hip flexion, adduction and external rotation, with added knee flexion (HIP)

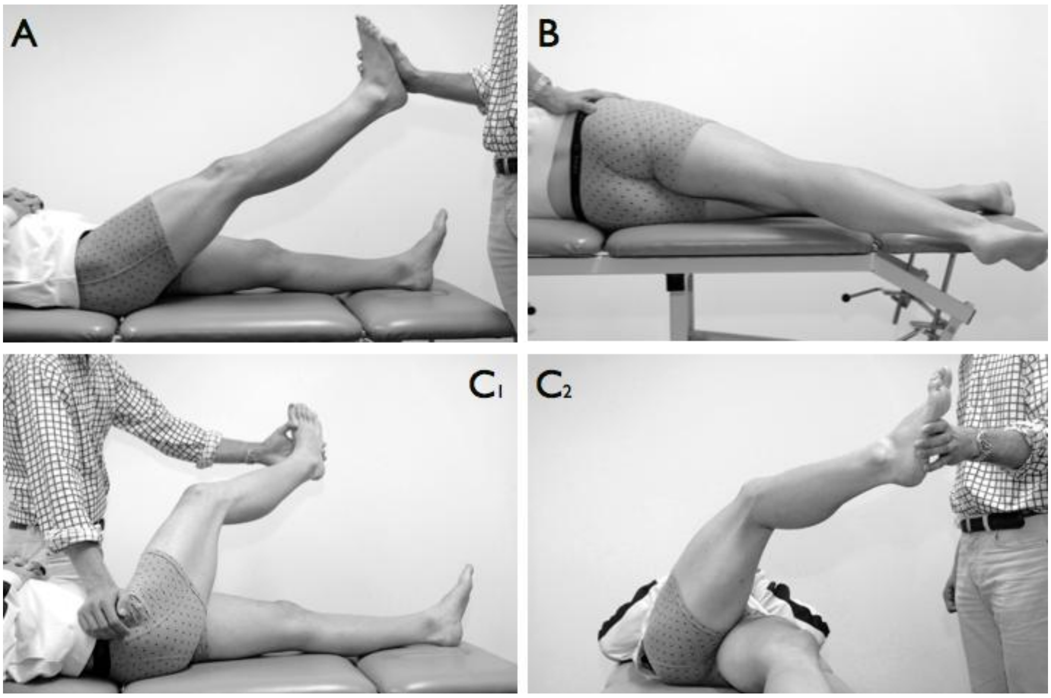


Figure 5.1: Each of the three testing positions; A = Straight leg raise to 30˚, B = Modified Ober test, C = HIP Flexed knee, hip flexion and adduction, with the examiner controlling for external loading (C_1 lateral view, C_2 cranial view)

The hip was maintained in the anatomical position by applying load superior to the greater trochanter on the contralateral limb. External loading was applied to the knee by the examiner to force hip adduction, and this was set at 100N using a manual muscle tester controlled by the examiner. All stretches were held in position for 30s, separated by a one-minute interval. All tests were performed on both limbs of each cadaver. Detached fascia specimens subjected to controlled loading showed little evidence of plastic deformation given the rest intervals (1 minute) employed. Pre-stretching of all tested limbs was performed in an attempt to ensure that this deformity was minimized. Randomisation of stretching was precluded due to the technicalities of strain gauge placement protection during dynamic manoeuvres. Accordingly the SLR and Ober test were performed first and last as the least and most perturbing stretches.

The SLR test, which replicates the joint angles occurring during heel contact in human locomotion, was included as a control variable. This allowed for the determination of strain occurring during the two experimental tests, which were ITB specific stretching exercises, which does not occur during typical human locomotion.

Strain Assessment

Insulated, 10mm, 120Ohm foil-type microstrain gauges (BCM Sensor Technologies, Antwerp, Belgium) were attached to the external surface of the ITB using a gauge specific cyanoacrylate adhesive (TML, Tokyo, Japan) prior to the performance of the ITB stretches (Figure 5.2). Data were acquired at 50Hz via an USB-based CompactDAQ system, and was normalised and calibrated using a combination of Signal Express 2.0 and Labview 8.5 software (National Instruments, Austin, Texas, U.S.A.). This strain acquisition protocol has been found in a separate study performed in our laboratory to produce a high (r>0.90) correlation with gold standard force/displacement measures of tissue, and to have a satisfactory level of repeatability (ICC: R=0.76).

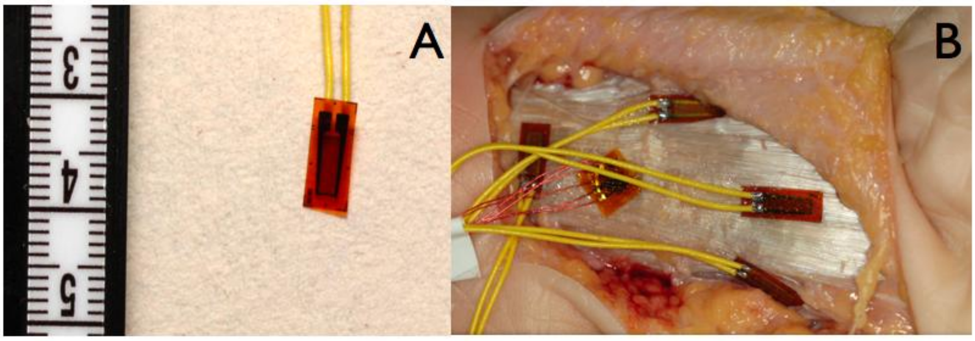


Figure 5.2: A: 10mm, 120Ohm foil-type microstrain gauges (BCM Sensor Technologies, Antwerp, Belgium) B: Microstrain gauge external fixation to fascia with gauge specific cyanoacrylate adhesive (TML, Tokyo, Japan).

The sensor was placed 8cm proximal to the lateral femoral epicondyle, ensuring it was undisturbed during manoeuvres, and assessed the longitudinal strain on the superficial surface of the ITB. The peak strain value during each test, which consistently occurred immediately after the testing position was assumed, was then determined using a custom written Labview analysis program (National Instruments, Austin, Texas, U.S.A.). The data obtained from the gauges in this study do not provide the actual magnitude of strain in percentage terms, as they have not been calibrated for the tissue to which they are joined. However, the results do provide a relative measure of strain in each of the three stretching protocols, allowing for an accurate comparison of the relative stretch occurring in the ITB during each of the tests. Previous studies have implanted buckle[212] or linear displacement gauges[213-215] into the fascia. While these sensors provide many benefits, including pre-calibrated displacement and ease of use, their limited ability in detecting dual-axis strain (in this case longitudinal and bending) restricts their application in situations where the transfer of strain may not be uni-axial. In addition, because the stretches incorporated in this study consist of complex, non-linear movements, the minute thickness of foil-type strain gauges securely and non-invasively attached to the fascia provide the benefits of reduced potential for tissue deformity and the consequent measurement error during the trials (Figure 5.3). It is important to note that the microstrain (me) results reported in this study are based on the manufacturers calibration factors, performed on rigid metal during mechanical testing. Therefore, the reported data does not provide a definitive true strain value, however, as this study was of a within-subject test-retest design the values recorded from the strain gauge allowed for a direct comparison of the results for each stretching intervention.

Figure 5.3: Instrumented (multiple microstrain gauges and electronic goniometer) cadaveric limb in completion of HIP test.

Data Analysis

Statistical analyses were performed comparing each testing procedure. Multiple Wilcoxon signed-rank tests at a significance threshold of $p<0.05$ were performed for each combination of the testing protocols (SPSS, Illinois, U.S.A.).

Study 3: In vivo measurement of ITB displacement

<u>Subjects</u>

Nineteen professional rugby league players (mean age: 19.00 $\pm$ 0.5 yr, mean height: 1.83$\pm$0.07m, mean body mass: 90.7$\pm$8.8kg) volunteered to participate in this study. The University of Melbourne Human Ethics Committee granted ethical approval.

Testing protocol

In an attempt to assess whether stretching does occur in the ITB, ultrasound assessment of the Tensor Fasciae Latae (TFL)/ITB aponeurosis interface was performed during a functional task designed to induce strain in the ITB. This task was a maximal voluntary contraction (MVC) of the TFL, which if stretching occurs, should invoke a shortening of the muscle and consequent lengthening of the ITB. This would produce a proximal shift in the TFL/ITB interface, which would be evident visually during the ultrasound assessment of the task. This protocol has been used extensively in assessment of muscle/tendon interfaces, particularly of the gastrocnemius/Achilles tendon.[216,217]

The subject was instructed to lie on their right side on a plinth, with their hips at 180^{o} extension. The subjects then abducted their left leg to a position horizontal with the ground. A manual muscle tester was used to weigh the limb in this position, providing the force level necessary to resist gravity. A custom-made leg cuff and rigid chain were then used to secure the subjects limb to a load cell mounted on the floor, which allowed for an isometric hip abduction with the limb parallel to the ground to be performed. During this test the subjects were required to maintain contact of their heel with a vertical guidepost, in an attempt to isolate the contraction primarily to the TFL. The movement consisted of the subjects lifting their limb to the horizontal position, deemed the baseline level, holding the limb in this position for three seconds and then performing a three second MVC. During the test a 10 MHz, 38mm linear ultrasound probe (Mindray DP-6600, Shenzhen, China) was fixed to the skin directly superior to the TFL/ITB insertional junction. The excursion of this junction was recorded throughout the entire test, providing an indirect measure of the elasticity of the ITB. The displacement of the junction from the baseline position to the position of peak force was measured using an image analysis program (ImageJ v. 1.36b, National Institute of Health, Bethesda, MD, U.S.A.). This method replicates previous anatomical junction studies performed in our laboratory.[218] Three MVC tests of the left limb of each subject were performed, separated by a 30 second rest interval.

Data Analysis

The one-tailed nature of the experimental protocol meant that even minor shifts in the anatomical landmark would create positive results for ITB displacement. Therefore data analysis was limited to assessments of the magnitude of the effect size using the Cohen's d equation with the initial resting length as the measure of standard deviation. An estimation of the strain occurring in the ITB was performed by measuring the length of the ITB using callipers, based on the mapping performed in Study 1, which thus designated the initial length of the ITB. The displacement of the junction was then added to this initial ITB length to create a measure of ITB length at MVC. The effect size of the percentage difference in length, deemed the ITB strain, was then calculated. The strain occurring in the ITB was then determined using the equation:

ITB strain (%) = $((L_{MVC} / L_i)-1) \times 100$

Where: L_{MVC} =ITB length at MVC, and L_i = ITB length at rest.

Results

Study 1: Cadaveric anatomical studies of ITB

Our anatomical findings confirmed that the ITB is in fact a thickening of the facia lata which completely envelopes the leg. In all cases it was connected to the femur along the linea aspera from the greater trochanter (by the intermuscular septum) to, and including, the lateral epicondyle of the femur by coarse fibrous bands. I failed to demonstrate a bursa interposed between the ITB and distal lateral femur on a single cadaver. The Tensor Fasciae Latae (TFL) muscle was completely enveloped in fascia, its origin formed by fascia lata arising from the iliac crest. TFL inserted directly into ITB, the latter structure behaving as an elongated tendon insertion of TFL. A substantial portion of Gluteus Maximus inserted directly into ITB, independently of the portion of muscle that inserts into the greater trochanter. These findings are shown in cadaveric form in Figure 5.4 and schematically in Figure 5.5.

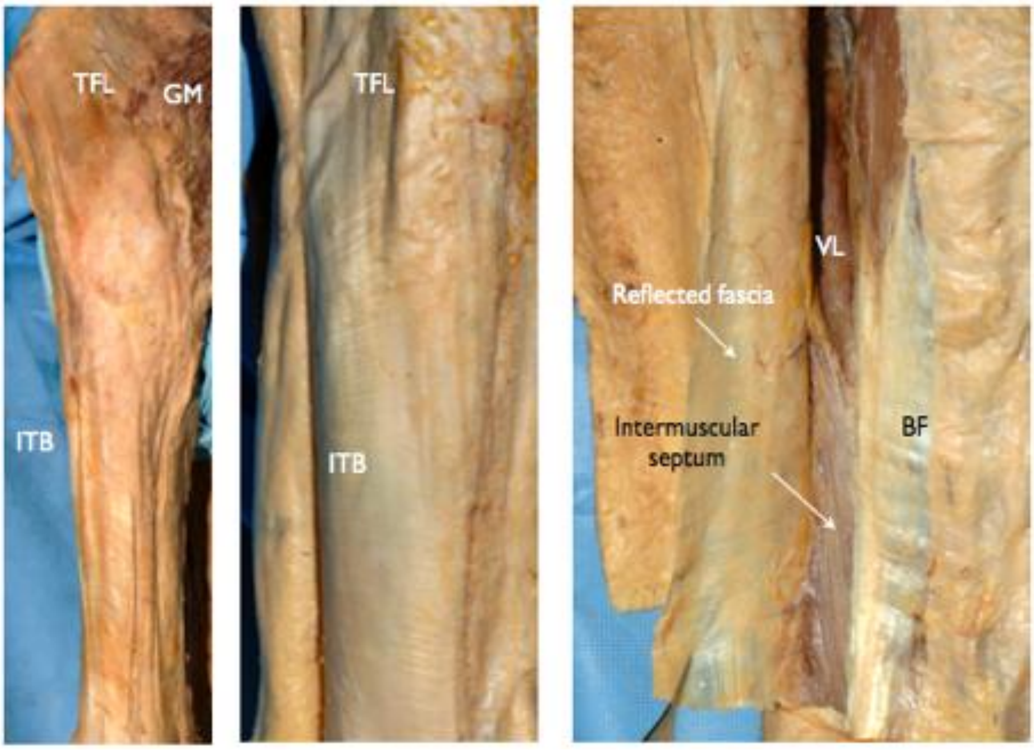


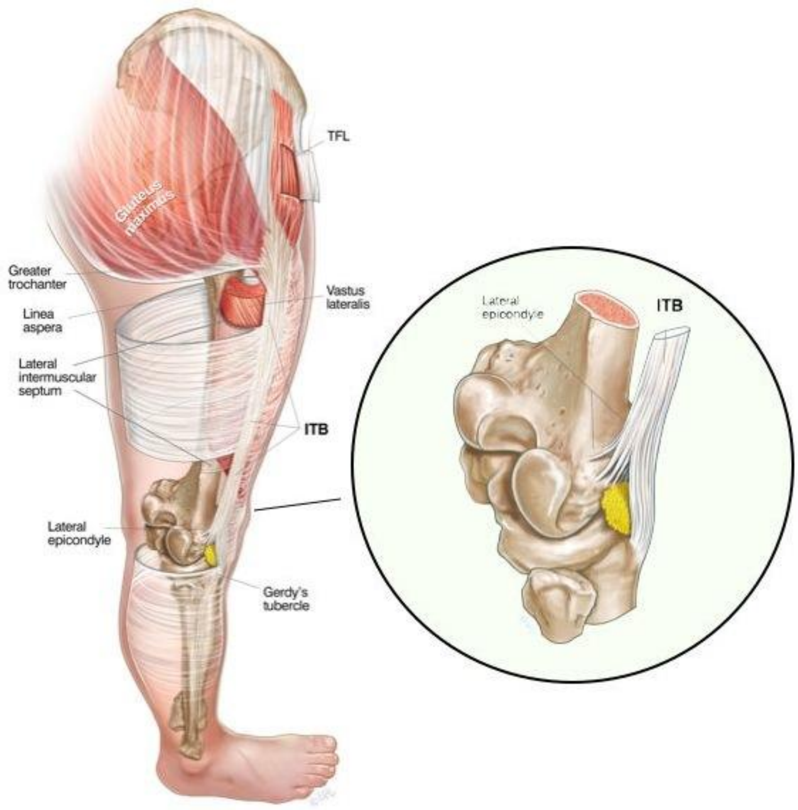
Figure 5.4: Dissected specimens of the iliotibial band on the left leg viewed posteriorly.

(a) The circumferential nature of the fascia lata is demonstrated, the position of iliotibial band (ITB) is shown. Tensor Fasiae Latae (TFL) is completely enveloped in fascia, the fascial insertion of Gluteus Maximus is also highlighted.

(b) The iliotibial band (ITB) as a lateral thickening of the fascia lata rather than a distinct entity.

(c) The fascia lata dissected to reveal the intermuscular septum attaching the iliotibial band to the femur, separating Vastus Lateralis (VL) from Biceps Femoris (BF).

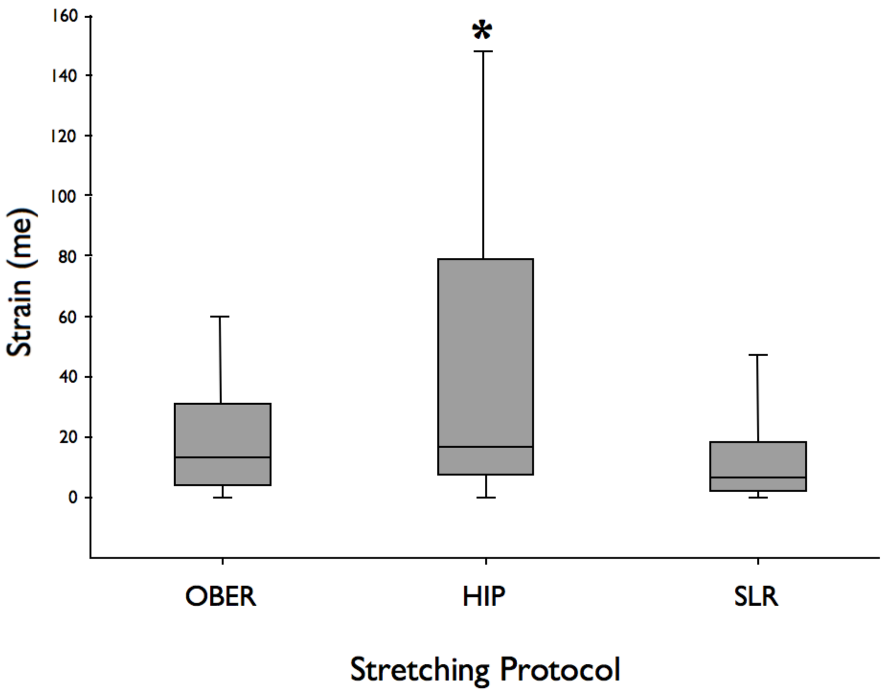
Figure 5.5: A schematic representation of the anatomy of the Iliotibial band (ITB). TFL = Tensor Fasciae Latae, by Dr Levente Efe. Contained in the article "Iliotibial band syndrome: an examination of the evidence behind a number of treatment options." Scand J Med Sci Sports 20(4): 580-587.[219] Courtesy of Scandinavian Journal of Medicine and Science in Sport.



Study 2: Cadaveric ITB strain

The microstrain (me) values (median (IQR)) for the OBER (15.4(5.1-23.3) me), HIP (21.1(15.6-44.6) me) and SLR (9.4(5.1-10.7) me) showed a marked disparity in the optimal inter-limb stretching protocol. These peak intralimb strain values occurred during the OBER, HIP or SLR test in three, four and two of the limbs respectively. Statistical analysis revealed that the HIP stretch invoked significantly (Z=2.10, p=0.036) greater strain than the SLR trial. No other significant differences were observed. These results are provided in Figure 5.6.

Figure 5.6: Strain measured in the ITB during three different testing protocols. * Indicates a significant (p<0.05) increase in strain during the HIP (Flexed knee, hip flexion and adduction) stretch in comparison with the SLR (straight leg raise).



Study 3: In vivo measurement of ITB displacement

The TFL/ITB junction displacement during the MVC was 2.0 ± 1.6mm. The mean ITB length was 87.7 ± 4.6cm. This resulted in a mean strain of 0.23 ± 0.18% strain. This negligible change in length occurred despite a mean hip abduction force of 235.6 ± 58.3N.

Discussion

Many of the traditional treatments for ITBS are based on the presence of a bursa between the ITB and the LFC, an ability to stretch the ITB, and the development of friction between the ITB and the LFC due to transverse motion. This work's findings challenge these anatomical and pathological principles. Two of the common treatments of ITBS focus on treating local inflammation of the distal ITB and putative 'bursa' and stretching the ITB.[131,199,220]. The effectiveness of these two modalities should be questioned given the lack of support for the presence of a lateral bursa and the low magnitude and disparate strain occurring during stretching and MVC found in this study.

In regard to treatment of the 'bursa' this routinely utilises non-steroidal anti-inflammatory drugs or corticosteroid injection in the belief that a bursitis or local inflammation is the basis of the condition.[220] The gross anatomical studies performed by the authors (EF and AFM) failed to demonstrate a bursa interposed between the ITB and distal lateral femur on a single cadaver. These findings correlate closely with the works of Fairclough et al.[133,134] who have suggested that a richly innervated and vascularised loose connective tissue, (containing pressure-sensing pacinian corpuscles) represents the pain generating structure in the area. This is also suggested by the surgical specimens and imaging findings previously discussed.[198,200] Local inflammation in the area may be related to compression of this connective tissue.[133]

Physiological fibrous bands were found to extend from the ITB to the lateral femoral condyle confirming the findings of Evans[221] and Fairclough et al.;[134]. These bands, and the fact that the ITB is a lateral thickening of the circumferential fascia lata prevent lateral movement over the condyle, result in the 'friction' usually reported unlikely from an anatomical standpoint.

While reducing the inflammatory response may be useful in controlling acute symptoms, addressing the underlying biomechanical/anatomical abnormality is more likely to result in long-term benefit. Current recommended strategies aimed at stretching the ITB have been tested in this study.[222]

This work also highlighted important structural characteristics central to understanding the difficulties in achieving any real stretching of the ITB. The longitudinal and firm attachment (0.3mm average thickness) of the ITB to the full length of the femur means that the potential for physiological lengthening is limited. This would appear at odds with a number of authors, which have stretched,[223,224] and even quantified, the lengthening of the ITB.[223] This is likely to represent an apparent, rather than true lengthening, related to the lengthening of TFL rather than the ITB itself.

Results from the ultrasound measurements provide *in vivo* proof that the ITB in fact stretches minimally during isometric contraction of TFL. The average length of ITB in subjects is 88cm with the average measured movement representing a 0.2% increase in length during a MVC. In addition, ITB strain measurements during three ITB stretching manoeuvres demonstrated that in the absence of muscle tone (in cadaveric subjects) the stretches exert a different strain on the iliotibial band. Though of small magnitude, the strain generated by the HIP test (a cadaveric simulation of some of the more dynamic stretches such as Fredericson's 'modified matrix exercise'[128]) was significantly higher than the control test. These findings highlight the tensioning role of Gluteus maximus (working synergistically with TFL) in ITBS, concurring with other studies noting the substantial contribution of Gluteus maximus to the ITB.[133,134]

There are a number of potential limitations in this study. The cadaveric work included here was performed on a much older age group, unlikely to partake in regular exercise. Information regarding occupational history or previous physical activity levels was not available. Given the age profile of the cadavers muscle bulk was not equivalent to fit healthy athletes. The measurement reliability of the Q angle in the supine position is only moderate.[225] In acknowledging these limitations I must point out that I studied the ITB in the absence of muscle tone, which allows us to comment on the ITB free of muscular influence. Study 3, which did require muscular involvement, was performed on elite rugby players, in whom the TFL muscle was a considerable structure. I also recognise that TFL contraction alone could not be assured in this test, the relative role of gluteus medius in abducting the hip between individuals was not measured. However, for the purpose of this study movement or lack of movement of the ITB/TFL junction was measured accurately. This work did not image or investigate symptomatic patients with ITBFS. I am aware of some exciting but as yet unpublished work using dynamic ultrasound and ITBFS in other centres which may prove an enlightening expansion on this work.[226]

The findings reported in this study highlight the limited role lengthening of the fascial component (ITB) has in any lengthening of the ITB/TFL complex, which may instead result from a decrease in stiffness in the muscular components (TFL, Gluteus maximus) of the system. The anatomical evidence for the current treatment regimens discussed previously appears insufficient. While local treatment measures may have roles in temporarily easing symptoms, they appear to treat symptoms rather than cause. Given that it appears that the muscular component of the complex plays an important role in tensioning of the ITB, treatment should be directed at TFL and Gluteus maximus. Soft tissue therapy such as massage therapy or dry needling of myofascial trigger points may be more valid as these interventions are supported by this construct, however, well-designed randomised controlled studies on current, and proposed, management strategies in a pathological population are needed before any conclusion may be made.

Conclusion

This work revisits the anatomy of the iliotibial tract, combining gross anatomy with modern techniques for measuring strain and stretch in order to evaluate the evidence base for traditional treatment regimens. These results suggest that measures aimed at treating local inflammation and stretching the ITB[128,131,189,205] are based on an incorrect understanding of the relevant anatomy and pathology. Future studies must focus on the efficacy of treatment of the muscular component of the ITB/TFL complex. Soft tissue measures such as massage and dry needling may be utilised to decrease muscular stiffness and effect a functional lengthening of the complex.

Chapter 6

Conclusions and future directions

This thesis was designed to examine the role of the specialist sports physician working in sports medicine in Ireland. The discipline of sports and exercise medicine in Ireland was granted speciality recognition in 2004, and the faculty of sports and exercise medicine was recognised as the body in the State for the purpose of granting evidence of satisfactory completion of specialist training in Sports and Exercise Medicine. Despite this, the role of the sports and exercise specialist is unclear to the medical fraternity and general public as a whole. This work seeks to elucidate and clarify the role of the specialist in sports and exercise medicine, and to outline the potential benefits of this skill-set not only to the patient, but also to other areas of the medical community and to the general public.

I examined specific examples of a number of the areas in which the specialist sports physician may improve individual and population health through the safe promotion of physical activity. I established the following areas of importance:

- The identification of the most appropriate activities for individuals and populations.
- The identification of activities or behaviours during physical activity which may expose the participant to injury or illness: the evaluation of sport- and recreation-related injuries and fracture occurrence amongst emergency department attendees (Chapter 2)

- The assessment of the unanticipated potential consequences of the safety measures used in sport: the evaluation of the risk factors for hand injury in hurling (Chapter 3).
- The appropriate screening for disease associated with physical activity: the evaluation of the prevalence of EIB in a cohort of professional rugby players (Chapter 4).
- The appropriate treatment of illness or injury arising from physical activity in an evidence-based manner: the evaluation of the anatomy and function of the ITB to assess the evidence base for current treatment (Chapter 5).

In this thesis, I examined the role and responsibilities of the sports and exercise specialist encompassing the breadth of the specialty. This was achieved by performing an epidemiological review, evaluating the consequences of health related intervention in sport, screening for activity related disease, and undertaking primary research into the basis of treatment of injury. The prevalence of sports related injury (SRI) and emergency department presentations at a tertiary referral university hospital were reviewed. There was an exploration of the relationship between SRI and fracture, place of injury, activity at the time of injury and site of injury suffered. I then investigated the impact of protective equipment on the injury profile and player behaviours in a particular sport. The introduction and subsequent mandatory use of helmet and face protection in hurling saw a steady decrease in head, facial and ocular injury. It has previously been observed that rates of hand injury increased dramatically in tandem with this decrease in head and facial injury. I investigated this apparent increase in hand injury and the potential association with head and facial protective equipment use and also possibly altered behaviours arising from that use in hurling. Screening was performed for an illness secondary to physical activity, specifically, a professional rugby team (with a sports specific field test) for exercise induced bronchoconstriction. Finally I investigated the evidence base behind some of the recommended treatments for a commonly presenting cause of anterolateral knee pain in athletes. I performed a cadaveric study of the anatomy of the iliotibial band (ITB); a further cadaveric study- using novel strain gauge technology- to measure the efficacy of commonly used stretches for ITB pain and an *in vivo* biomechanical measurement of ITB movement.

In Chapter 2, I examined 22,465 presentations to a university hospital emergency department over a 6-month period where 3,172 injuries were found to have arisen from sport and recreation. This work was the first attempt to examine all-cause sports and recreation injuries resulting in ED attendance, in both paediatric and adult patients in a European population.

One in 7 ED presentations are due to SRI (13.6%, SD 0.7), nearly one in five (19%) presentations were for injuries occurring in the residential setting. Children appear particularly vulnerable. Children aged 4-16 comprise

16.5% of all-cause ED attendances, and 18% of the underlying Cork population. This group comprised 39.9% of SRI attendances. Though these figures may represent a greater participation rate in all activities and most particularly in unsupervised activity, it may be pertinent to note that the supervision which is mandatory in team/field or structured sport may have a 'protective' effect. This paediatric cohort is of particular import however as significant injury in this group may preclude physical activity later on in life.

In tandem with this risk to future physical activity the burden on health services should not be underestimated. It is established that undergraduate and subspecialty training in musculoskeletal medicine is suboptimal.[158] Some of the most vulnerable and frequently injured exercisers in our population may be better served by a specialist sports medicine service similar to an orthopaedic 'fracture clinic' or plastic surgery 'soft tissue' clinic.

Public education around the risk of unsupervised playground equipment use in the residential setting should be a target for public health groups.

Ireland does not have a unique problem but a number of areas require further focus to prevent unnecessary injury, and where it does occur, to treat it effectively with as little impact on future activity levels as possible.

Future directions of real value from this work would be to take a modified model of the questionnaire and disseminate it to Emergency Departments around the country. This study was designed as a pilot study for just such a purpose and indeed the quality of the data generated suggests an expansion of its scope would have a significant role in informing health policy in this country. If collection of exposure data to sporting activity in different sports were allied to this work an extremely powerful prospective epidemiological study on risk factors for SRI would be possible - one of truly international merit. Ireland's small population and public healthcare system are uniquely helpful in making this possible.

In Chapter 3, I explored the relationship between the use of head and face protection in hurling, and hand injury. This was an expansion of some of the work in Chapter 2. My hypothesis was that use of protective equipment in hurling may lead to altered player behaviour amongst wearers. I further hypothesised that this has been linked to increased levels of "risky behaviour'. I focused particularly on those players suffering serious hand injury and whether they used helmet and face protection. In a cohort of adult hurling players those players using helmet and facial protection were statistically more likely (Odds Ratio 2.76, 95% confidence interval (1.42-5.37), p<0.003) to suffer significant hand injury than players who do not use this equipment. Player uptake of hand protection was extremely poor, only 8 (4.9%) used hand protection (Ashgard™ by O'Dare, Figure 5.2), while 149 (91.4%) had tried this equipment in the past. The introduction of head and face protection in hurling has seen a significant drop in injury to this area. The rate of hand injury has not decreased however and this work suggests that the introduction of mandatory hand protection may lead to a similar drop in hand injury as has been seen in head and facial injury. There are lessons here for further planned interventions in hand protection; the poor uptake of commonly available products is closely linked to the hand injury rate. The efficacy of newer hand protection has not been ascertained and a well designed prospective study to follow hand injury patterns in two cohorts, one with and the other without this hand protection, would provide significant insight into this issue.

In Chapter 4, I performed a sports specific field test to determine the prevalence of exercise induced bronchoconstriction (EIB) in an international professional rugby team. The specialist in sports and exercise medicine must be aware of the potential medical complications of physical activity, and screening for any preventable medical conditions is of course paramount to ensuring athletes' safety and performance. I chose to investigate exercise induced bronchoconstriction as it is an area which has not achieved as much attention, or degree of investigation and publication as cardiovascular illness. Many practitioners in sports and exercise medicine may therefore be less *au fait* with EIB than would be optimal. I looked at EIB in a professional rugby team as it is a winter sport and there had previously been no published prevalence or incidence of EIB in rugby.

Nearly one third of players (29%) in the cohort tested had confirmed asthma or EIB significantly higher than 12-15% reported in the general population.[184] In a group of professional rugby players who are medically screened annually 5 'new' cases were diagnosed and half of asthma/EIB diagnosed players were under treated. Exercise induced bronchoconstriction may therefore represent not just an undiagnosed limitation to athletic performance, but in professional sport a potentially occupational hazard if undiagnosed and untreated. This work highlights not only a higher than reported prevalence of EIB in rugby, but also proposes a sports specific field test for making the diagnosis of EIB. This test must be validated in its own right but it fulfils the WADA criteria as an appropriate provocation test for the diagnosis of EIB,[186] and was designed specifically to elicit recommended intensity, duration and attained heart rate.[174,175] The test is acceptable to the athletes, is easily arranged and does not require either expensive equipment or time away from training venues.

Medical conditions affect athletes just as they do the general population and physician trained specialists should play a future role in determining adequate and appropriate medical screening and governing body health policy.

In Chapter 5, I examined the science and evidence behind some of the accepted treatments of iliotibial band friction syndrome (ITBFS). My hypothesis was that a number of the traditional treatments aimed at local inflammation and stretching the ITB derive from an incorrect understanding of the relevant anatomy and pathology. My cadaveric and biomechanical studies supported that hypothesis. I confirmed the work of Fairclough et al.[133,134] which demonstrated a fat pad between the distal ITB and femur and not a bursa. I demonstrated the ITB is firmly attached to both the medial femoral condyle and the length of the linea alba. All of these anatomical features make the accepted friction syndrome with the distal ITB impacting on a bursa an implausible cause for the exertional distal lateral leg pain seen in ITBFS. This work questions the utility of traditional dogma around the management of distal ITBFS and refocuses attention to potentially more efficacious treatment and rehabilitation. The specialist in sports medicine must attempt to vigorously challenge the science behind treatments offered for particular conditions.

As the physician-trained specialist should play a role in health policy this work illustrates the need to vigorously interrogate the evidence behind treatment strategies. Current continued professional development standards dictate that all clinicians perform an annual audit. It would be worthwhile to consider adding regular assessment of the evidence behind treatment practices to this requirement. The faculty of sports medicine would be well placed to guide this practice.

In conclusion the sports and exercise specialist must play an important role in maintaining the health and activity levels of the active population. Sport and recreation-related injury may be more common and of a higher morbidity than I had previously supposed. Screening for illness and medical conditions related to physical activity is a vital part of keeping a population active. The practice of evidence-based medicine is sometimes difficult in sports medicine given the dearth of research in a number of areas. Evidence-based medicine rather than historical opinion-based medicine must however be the level to which the sports medicine community aspires. While injury prevention measures should be promoted during physical activity, care must be taken to ensure that these measures are as rigorously assessed for safety as other health interventions. The medical community must be vigilant for any unwanted secondary injury patterns following the introduction of any novel injury prevention strategy.

Obesity, heart disease, and type II diabetes are often described as western diseases. Their prevalence is linked to affluence, inactivity and poor dietary habits. Our medical system caters well for those suffering from disease, but is poorly equipped to prevent illness. Sport and physical activity is in itself a potent primary preventative measure. Attending a population while active or preparing to be active is an ideal time to achieve the aims outlined in this text. An aspirational level of physical activity for the youth of modern society is 5 hours per week.[6] It is likely that very few Irish children and adolescents attain this. This is where we need to start. The specialist in SEM and the Faculty of Sports and Exercise Medicine has primary roles in achieving this. This work on the epidemiology of SRI in Ireland, of attitudinal change secondary to the use of protective equipment, of screening in SEM and evaluation of the evidence supporting treatment options, demonstrates how the specialist in sports and exercise medicine may influence health service resource allocation and guide national and international sports governing bodies in ensuring the general population achieves and maintains optimal levels of safe physical activity.

References

1. Sassi F. Obesity and the Economics of Prevention: Fit not Fat. 2010.http://www.oecd.org/els/health-systems/obesityandtheeconomicsofpreventionfitnotfat.htm (accessed 06.04.2013 2013).

2. Archer E, Blair SN. Physical activity and the prevention of cardiovascular disease: from evolution to epidemiology. *Progress in cardiovascular diseases* 2011; **53**(6): 387-96.

3. Blair SN, Kampert JB, Kohl HW, 3rd, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *JAMA* 1996; **276**(3): 205-10.

4. Affairs MfCaY. Growing up in Ireland. In: Affairs DoCaY, editor. Dublin: Government Publications; 2013.

5. Taubes G. The elusive benefits of exercise. Why we get fat. New York: Random House Inc.; 2010: 40-57.

6. Campbell D. Will London's Olympic public health legacy turn to dust? *BMJ (Clinical research ed* 2012; **344**: e4207.

7. Weed M, Coren E, Fiore J, et al. Developing a physical activity legacy from the London 2012 Olympic and Paralympic Games: a policy-led systematic review. *Perspectives in public health* 2012; **132**(2): 75-80.

8. Mountjoy M, Andersen LB, Armstrong N, et al. International Olympic Committee consensus statement on the health and fitness of young people through physical activity and sport. *British journal of sports medicine* 2011; **45**(11): 839-48.

9. Steffen K, Emery CA, Romiti M, et al. High adherence to a neuromuscular injury prevention programme (FIFA 11+) improves functional balance and reduces injury risk in Canadian youth female football players: a cluster randomised trial. *British journal of sports medicine* 2013; **47**(12): 794-802.

10. Nutton V. The chronology of Galen's early career. *Classical quarterly* 1973; **23**(1): 158-71.

11. Cyriax JH. Manipulative Treatment of Intestinal Spasm. *British medical journal* 1938; **1**(4025): 445-7.

12. Cyriax J. Treatment by Movement. *British medical journal* 1944; **2**(4365): 303-5.

13. Newell M, Grant S, Henry A, Newell J. Incidence of injury in elite Gaelic footballers. *Irish medical journal* 2006; **99**(9): 269-71.

14. Wilson F, Caffrey S, King E, Casey K, Gissane C. A 6-month prospective study of injury in Gaelic football. *British journal of sports medicine* 2007; **41**(5): 317-21.

15. Cromwell F, Walsh J, Gormley J. A pilot study examining injuries in elite gaelic footballers. *British journal of sports medicine* 2000; **34**(2): 104-8.

16. Shelly MJ, Butler JS, Timlin M, Walsh MG, Poynton AR, O'Byrne JM. Spinal injuries in Irish rugby: a ten-year review. *The Journal of bone and joint surgery* 2006; **88**(6): 771-5.

17. Watson AW. Incidence and nature of sports injuries in Ireland. Analysis of four types of sport. *The American journal of sports medicine* 1993; **21**(1): 137-43.

18. Crowley PJ, Condon KC. Analysis of hurling and camogie injuries. *British journal of sports medicine* 1989; **23**(3): 183-5.

19. Crowley PJ, Crowley MJ, Dardouri H, Condon KC. Value of wearing head protection gear while playing hurling. *British journal of sports medicine* 1995; **29**(3): 191-3.

20. Flynn TH, Fennessy K, Horgan N, et al. Ocular injury in hurling. *British journal of sports medicine* 2005; **39**(8): 493-6; discussion 6.

21. Kiely PD, Ashraff M, O'Grady P, Dawson MJ, O'Beirne JG. Hurling-related hand injuries. *Injury* 2003; **34**(8): 561-3.

22. Clarke HJ, Ryan D, Cullen I, Cusack S. The impact of a temporary ice-rink on an emergency department service. *European journal of emergency medicine : official journal of the European Society for Emergency Medicine* 2006; **13**(4): 204-8.

23. McCarthy T, Crushell E, Synnott K, Kiely P, McCormack D. Play-wrestlers and their injuries. *Irish medical journal* 2005; **98**(3): 84.

24. Porter M, O'Brien M. Incidence and severity of injuries resulting from amateur boxing in Ireland. *Clin J Sport Med* 1996; **6**(2): 97-101.

25. Abernethy L, MacAuley D. Impact of school sports injury. *British journal of sports medicine* 2003; **37**(4): 354-5.

26. Abernethy L, MacAuley D, McNally O, McCann S. Immediate care of school sport injury. *Inj Prev* 2003; **9**(3): 270-3.

27. Watson AW. Sports injuries related to flexibility, posture, acceleration, clinical defects, and previous injury, in high-level players of body contact sports. *International journal of sports medicine* 2001; **22**(3): 222-5.

28. Carroll SM, Jawad MA, West M, O'Connor TP. One hundred and ten sports related facial fractures. *British journal of sports medicine* 1995; **29**(3): 194-5.

29. Quigley F. A survey of the causes of sudden death in sport in the Republic of Ireland. *British journal of sports medicine* 2000; **34**(4): 258-61.

30. Burke P, Buckley N, McShane D, O'Connor P. Sports injuries and the Casualty Department. *Irish medical journal* 1983; **76**(3): 127-9.

31. Murphy AW, Martyn C, Plunkett PK, O'Connor P. Sports injuries and the accident and emergency department--ten years on. *Irish medical journal* 1992; **85**(1): 30-3.

32. Walsh PJ. A survey on sports injuries in general practice. *Irish medical journal* 2002; **95**(9): 279-80.

33. O'Rourke KP, Mun S, Browne M, Sheehan J, Cusack S, Molloy M. A retrospective study of the demographics of sport and exercise injuries in 1143 children presenting to an Irish emergency department over a 6-month period. *European journal of pediatrics* 2005; **164**(7): 421-6.

34. Caine C. An Epidemiologic Approach to Sports Injuries. In: Caine D, ed. Epidemiology of Sports Injuries. Champaign, Illinois: Human KInetics; 1996: 1-13.

35. Morabia A. Epistemology. In: Morabia A, ed. A history of epidemiologic methods and concepts. Basel: Birkhauser Verlag: 92-5.

36. Magnússon G. Health and Human Rights—A Public Health Perspective. In: Gunn SWA, Masellis M, eds. Concepts and Practice of Humanitarian Medicine. New York: Springer Science+Business Media, LLC; 2008: 9-13.

37. Gordon JE. The epidemiology of accidents. *American journal of public health and the nation's health* 1949; **39**(4): 504-15.

38. Wallace RB. Application of epidemiologic principles to sports injury research. *The American journal of sports medicine* 1988; **16 Suppl 1**: S22-4.

39. Haddon W, Jr., Ellison AE, Carroll RE. Skiiing injuries. Epidemiologic study. *Public health reports* 1962; **77**: 975-85.

40. Romano RL, Burgess EM, Andrews CB. Medical implications of water-skiing. *Clinical orthopaedics* 1962; **23**: 140-5.

41. Lysens R, Steverlynck A, van den Auweele Y, et al. The predictability of sports injuries. *Sports medicine (Auckland, NZ* 1984; **1**(1): 6-10.

42. Maroon JC, Steele PB, Berlin R. Football head and neck injuries--an update. *Clinical neurosurgery* 1980; **27**: 414-29.

43. Torg JS, Vegso JJ, Sennett B. The National Football Head and Neck Injury Registry: 14-year report on cervical quadriplegia (1971-1984). *Clinics in sports medicine* 1987; **6**(1): 61-72.

44. Cantu RC, Mueller FO. Catastrophic spine injuries in American football, 1977-2001. *Neurosurgery* 2003; **53**(2): 358-62; discussion 62-3.

45. Cantu RC, Mueller FO. Brain injury-related fatalities in American football, 1945-1999. *Neurosurgery* 2003; **52**(4): 846-52; discussion 52-3.

46. OECD. Obesity and the Economics of Prevention: Fit not Fat. 2013.

47. Wanless D. Securing our Future Health: Taking a Long-Term View: Final report. London: HM Treasury, 2002.

48. Hamilton MT, Hamilton DG, Zderic TW. Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. *Diabetes* 2007; **56**(11): 2655-67.

49. Li S, Zhao JH, Luan J, et al. Physical activity attenuates the genetic predisposition to obesity in 20,000 men and women from EPIC-Norfolk prospective population study. *PLoS medicine* 2010; **7**(8).

50. Haskell WL, Lee IM, Pate RR, et al. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Medicine and science in sports and exercise* 2007; **39**(8): 1423-34.

51. Hurson C, Browne K, Callender O, et al. Pediatric trampoline injuries. *Journal of pediatric orthopedics* 2007; **27**(7): 729-32.

52. Murphy JC, Gissane C, Blake C. Injury in elite county-level hurling: a prospective study. *British journal of sports medicine* 2012; **46**(2): 138-42.

53. Hayashi T, Tsumura K, Suematsu C, Okada K, Fujii S, Endo G. Walking to work and the risk for hypertension in men: the Osaka Health Survey. *Annals of internal medicine* 1999; **131**(1): 21-6.

54. Hambrecht R, Walther C, Mobius-Winkler S, et al. Percutaneous coronary angioplasty compared with exercise training in patients with stable coronary artery disease: a randomized trial. *Circulation* 2004; **109**(11): 1371-8.

55. Veerman JL, Healy GN, Cobiac LJ, et al. Television viewing time and reduced life expectancy: a life table analysis. *British journal of sports medicine* 2012; **46**(13): 927-30.

56. Blair SN. Physical inactivity: the biggest public health problem of the 21st century. *British journal of sports medicine* 2009; **43**(1): 1-2.

57. GAA. Gaelic Athletic Association Official Guide- Part 2. 2009. http://www.gaa.ie/about-the-gaa/rules-and-regulations/.

58. Emery CA, Hagel B, Morrongiello BA. Injury prevention in child and adolescent sport: whose responsibility is it? *Clin J Sport Med* 2006; **16**(6): 514-21.

59. Hedlund J. Risky business: safety regulations, risks compensation, and individual behavior. *Inj Prev* 2000; **6**(2): 82-90.

60. Committee on Trauma Research, Commision on Life Sciences, National Research Council, Medicine atIo. Injury in America: a continuing public health problem. Washington, D.C.: National Academy Press; 1985.

61. Haddon W, Jr. A logical framework for categorizing highway safety phenomena and activity. *The Journal of trauma* 1972; **12**(3): 193-207.

62. Haddon W, Jr. Editorial: Strategy in preventive medicine: passive vs. active apprroaches to reducing human wastage. *The Journal of trauma* 1974; **14**(4): 353-4.

63. Cameron MH, Vulcan AP, Finch CF, Newstead SV. Mandatory bicycle helmet use following a decade of helmet promotion in Victoria, Australia--an evaluation. *Accident; analysis and prevention* 1994; **26**(3): 325-37.

64. Dennis RJ, Finch CF, Farhart PJ. Is bowling workload a risk factor for injury to Australian junior cricket fast bowlers? *British journal of sports medicine* 2005; **39**(11): 843-6; discussion -6.

65. Orchard JW, James T, Portus M, Kountouris A, Dennis R. Fast bowlers in cricket demonstrate up to 3- to 4-week delay between high workloads and increased risk of injury. *The American journal of sports medicine* 2009; **37**(6): 1186-92.

66. Benjamin HJ, Briner WW, Jr. Little league elbow. *Clin J Sport Med* 2005; **15**(1): 37-40.

67. Lyman S, Fleisig GS, Waterbor JW, et al. Longitudinal study of elbow and shoulder pain in youth baseball pitchers. *Medicine and science in sports and exercise* 2001; **33**(11): 1803-10.

68. Mueller FO. Fatalities from head and cervical spine injuries occurring in tackle football: 50 years' experience. *Clinics in sports medicine* 1998; **17**(1): 169-82.

69. Hagel B, Meeuwisse W. Risk compensation: a "side effect" of sport injury prevention? *Clin J Sport Med* 2004; **14**(4): 193-6.

70. Haddon W, Jr., Suchman EA, Klein D. Accident Research Methods and Approaches. New York: Harper and Row; 1964.

71. Saslow D, Solomon D, Lawson HW, et al. American Cancer Society, American Society for Colposcopy and Cervical Pathology, and American Society for Clinical Pathology screening guidelines for the prevention and early detection of cervical cancer. *American journal of clinical pathology* 2012; **137**(4): 516-42.

72. Linas BP, Wong AY, Freedberg KA, Horsburgh CR, Jr. Priorities for screening and treatment of latent tuberculosis infection in the United States. *American journal of respiratory and critical care medicine* 2011; **184**(5): 590-601.

73. Peto T, Tadros C. Screening for diabetic retinopathy and diabetic macular edema in the United Kingdom. *Current diabetes reports* 2012; **12**(4): 338-45.

74. Nishiyama T, Ozaki N, Iwata N. Practice-based depression screening for psychiatry outpatients: feasibility comparison of two-types of Center for Epidemiologic Studies Depression Scales. *Psychiatry and clinical neurosciences* 2009; **63**(5): 632-8.

75. Lin K, Lipsitz R, Miller T, Janakiraman S, Force USPST. Benefits and harms of prostate-specific antigen screening for prostate cancer: an evidence update for the U.S. Preventive Services Task Force. *Annals of internal medicine* 2008; **149**(3): 192-9.

76. NSC. Criteria for appraising the viability, effectiveness and appropriateness of a screening programme. 07.04.13 2009. http://www.screening.nhs.uk/criteria.

77. Wilson JMG, Jungner G. Principles and practice of screening for disease. Geneva: WHO, 1968.

78. Paterick TE, Jan MF, Paterick ZR, et al. Cardiac evaluation of collegiate student athletes: a medical and legal perspective. *The American journal of medicine* 2012; **125**(8): 742-52.

79. Chang AC. Primary prevention of sudden cardiac death of the young athlete: the controversy about the screening electrocardiogram and its innovative artificial intelligence solution. *Pediatric cardiology* 2012; **33**(3): 428-33.

80. Papadakis M, Sharma S. Electrocardiographic screening in athletes: the time is now for universal screening. *British journal of sports medicine* 2009; **43**(9): 663-8.

81. Corrado D, Basso C, Thiene G. Sudden cardiac death in athletes: what is the role of screening? *Current opinion in cardiology* 2012; **27**(1): 41-8.

82. Madsen NL, Drezner JA, Salerno JC. Sudden cardiac death screening in adolescent athletes: an evaluation of compliance with national guidelines. *British journal of sports medicine* 2013; **47**(3): 172-7.

83. Association GA. Cardiac Screening. 2013. http://www.gaa.ie/medical-and-player-welfare/cardiac-screening/.

84. Waterhouse DF, Ismail TF, Prasad SK, Wilson MG, O'Hanlon R. Imaging focal and interstitial fibrosis with cardiovascular magnetic resonance in athletes with left ventricular hypertrophy: implications for sporting participation. *British journal of sports medicine* 2012; **46 Suppl 1**: i69-77.

85. Hogan BA, Hogan NA, Vos PM, Eustace SJ, Kenny PJ. The cervical spine of professional front-row rugby players: correlation between degenerative changes and symptoms. *Irish journal of medical science* 2010; **179**(2): 259-63.

86. Warrington G, Dolan E, McGoldrick A, et al. Chronic weight control impacts on physiological function and bone health in elite jockeys. *Journal of sports sciences* 2009; **27**(6): 543-50.

87. Moloney ED, Clayton N, Mukherjee DK, Gallagher CG, Egan JJ. The shuttle walk exercise test in idiopathic pulmonary fibrosis. *Respiratory medicine* 2003; **97**(6): 682-7.

88. Smith E, Mahony N, Donne B, O'Brien M. Prevalence of obstructive airflow limitation in Irish collegiate athletes. *Irish journal of medical science* 2002; **171**(4): 202-5.

89. Holzer K, Douglass JA. Exercise induced bronchoconstriction in elite athletes: measuring the fall. *Thorax* 2006; **61**(2): 94-6.

90. Anderson SD. Exercise-induced asthma. In: Kay AB, ed. Allergy and allergic diseases. Oxford: Blackwell Scientific Publications; 1997: 672-711.

91. Hurwitz KM, Argyros GJ, Roach JM, Eliasson AH, Phillips YY. Interpretation of eucapnic voluntary hyperventilation in the diagnosis of asthma. *Chest* 1995; **108**(5): 1240-5.

92. Holzer K, Brukner P. Screening of athletes for exercise-induced bronchoconstriction. *Clin J Sport Med* 2004; **14**(3): 134-8.

93. Beasley R, Crane J, Lai CK, Pearce N. Prevalence and etiology of asthma. *The Journal of allergy and clinical immunology* 2000; **105**(2 Pt 2): S466-72.

94. Weiss KB, Gergen PJ, Hodgson TA. An economic evaluation of asthma in the United States. *The New England journal of medicine* 1992; **326**(13): 862-6.

95. Parsons JP, Mastronarde JG. Exercise-induced bronchoconstriction in athletes. *Chest* 2005; **128**(6): 3966-74.

96. Rundell KW, Jenkinson DM. Exercise-induced bronchospasm in the elite athlete. *Sports medicine (Auckland, NZ* 2002; **32**(9): 583-600.

97. McFadden ER, Jr. Hypothesis: exercise-induced asthma as a vascular phenomenon. *Lancet* 1990; **335**(8694): 880-3.

98. Anderson SD, Daviskas E. The mechanism of exercise-induced asthma is. *The Journal of allergy and clinical immunology* 2000; **106**(3): 453-9.

99. Anderson SD. Is there a unifying hypothesis for exercise-induced asthma? *The Journal of allergy and clinical immunology* 1984; **73**(5 Pt 2): 660-5.

100. Anderson SD, Daviskas E. The airway microvasculature and exercise induced asthma. *Thorax* 1992; **47**(9): 748-52.

101. Barnes PJ. Blunted perception and death from asthma. *The New England journal of medicine* 1994; **330**(19): 1383-4.

102. Rundell KW, Im J, Mayers LB, Wilber RL, Szmedra L, Schmitz HR. Self-reported symptoms and exercise-induced asthma in the elite athlete. *Medicine and science in sports and exercise* 2001; **33**(2): 208-13.

103. Thole RT, Sallis RE, Rubin AL, Smith GN. Exercise-induced bronchospasm prevalence in collegiate cross-country runners. *Medicine and science in sports and exercise* 2001; **33**(10): 1641-6.

104. Carlsen KH, Engh G, Mork M. Exercise-induced bronchoconstriction depends on exercise load. *Respiratory medicine* 2000; **94**(8): 750-5.

105. Amital H, Glikson M, Burstein M, et al. Clinical characteristics of unexpected death among young enlisted military personnel: results of a three-decade retrospective surveillance. *Chest* 2004; **126**(2): 528-33.

106. Lang DM. Asthma deaths and the athlete. *Clinical reviews in allergy & immunology* 2005; **29**(2): 125-9.

107. Becker JM, Rogers J, Rossini G, Mirchandani H, D'Alonzo GE, Jr. Asthma deaths during sports: report of a 7-year experience. *The Journal of allergy and clinical immunology* 2004; **113**(2): 264-7.

108. Corrigan B, Kazlauskas R. Drug testing at the Sydney Olympics. *The Medical journal of Australia* 2000; **173**(6): 312-3.

109. Karjalainen EM, Laitinen A, Sue-Chu M, Altraja A, Bjermer L, Laitinen LA. Evidence of airway inflammation and remodeling in ski athletes with and without bronchial hyperresponsiveness to methacholine. *American journal of respiratory and critical care medicine* 2000; **161**(6): 2086-91.

110. Holzer K, Anderson SD, Douglass J. Exercise in elite summer athletes: Challenges for diagnosis. *The Journal of allergy and clinical immunology* 2002; **110**(3): 374-80.

111. Phillips YY, Jaeger JJ, Laube BL, Rosenthal RR. Eucapnic voluntary hyperventilation of compressed gas mixture. A simple system for bronchial challenge by respiratory heat loss. *The American review of respiratory disease* 1985; **131**(1): 31-5.

112. Anderson SD, Brannan JD. Methods for "indirect" challenge tests including exercise, eucapnic voluntary hyperpnea, and hypertonic aerosols. *Clinical reviews in allergy & immunology* 2003; **24**(1): 27-54.

113. Crapo RO, Casaburi R, Coates AL, et al. Guidelines for methacholine and exercise challenge testing-1999. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, July

1999. *American journal of respiratory and critical care medicine* 2000; **161**(1): 309-29.

114. IOC. Guidelines for Beta 2 Agonist Use in Beijing. 2008. http://multimedia.olympic.org/pdf/en_report_1302.pdf.

115. Anderson SD, Rodwell LT, Du Toit J, Young IH. Duration of protection by inhaled salmeterol in exercise-induced asthma. *Chest* 1991; **100**(5): 1254-60.

116. Brannan JD, Anderson SD, Perry CP, Freed-Martens R, Lassig AR, Charlton B. The safety and efficacy of inhaled dry powder mannitol as a bronchial provocation test for airway hyperresponsiveness: a phase 3 comparison study with hypertonic (4.5%) saline. *Respiratory research* 2005; **6**: 144.

117. Holzer K, Anderson SD, Chan HK, Douglass J. Mannitol as a challenge test to identify exercise-induced bronchoconstriction in elite athletes. *American journal of respiratory and critical care medicine* 2003; **167**(4): 534-7.

118. Freed AN, Anderson SD, Daviskas E. Thermally induced asthma and airway drying. *American journal of respiratory and critical care medicine* 2000; **161**(6): 2112-3.

119. McFadden ER, Jr., Nelson JA, Skowronski ME, Lenner KA. Thermally induced asthma and airway drying. *American journal of respiratory and critical care medicine* 1999; **160**(1): 221-6.

120. Wallace RB, Clark WR. Sports injury research. The numerator, denominator, and the population-at-risk. *The American journal of sports medicine* 1988; **16 Suppl 1**: S55-6.

121. Bernstein J. Evidence-based medicine. *The Journal of the American Academy of Orthopaedic Surgeons* 2004; **12**(2): 80-8.

122. Wright JG. A practical guide to assigning levels of evidence. *J Bone Joint Surg Am* 2007; **89**(5): 1128-30.

123. Tjoumakaris FP, Ganley TJ, Kapur R, Kelly J, Sennett BJ, Bernstein J. Eminence-based medicine versus evidence-based medicine: level V evidence in sports medicine. *The Physician and sportsmedicine* 2011; **39**(4): 124-30.

124. Patterson K. What Doctors Don’t Know (Almost Everything). The New York Times. 2002 May 5.

125. McCrory PR, Bladin PF, Berkovic SF. Retrospective study of concussive convulsions in elite Australian rules and rugby league footballers: phenomenology, aetiology, and outcome. *BMJ (Clinical research ed* 1997; **314**(7075): 171-4.

126. Aubry M, Cantu R, Dvorak J, et al. Summary and agreement statement of the First International Conference on Concussion in Sport, Vienna 2001. Recommendations for the improvement of safety and health of athletes who may suffer concussive injuries. *British journal of sports medicine* 2002; **36**(1): 6-10.

127. McCrory P, Meeuwisse WH, Aubry M, et al. Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. *British journal of sports medicine* 2013; **47**(5): 250-8.

128. Fredericson M, Wolf C. Iliotibial band syndrome in runners: innovations in treatment. *Sports medicine (Auckland, NZ* 2005; **35**(5): 451-9.

129. Gray H. Femoral and Gluteals Muscles. In: Bannister LH, Berry MM, Williams PL, eds. Gray's Anatomy: The Anatomical Basis of Medicine & Surgery. 38 ed: Churchill Livingstone; 1995: 338-79.

130. Khaund R, Flynn SH. Iliotibial band syndrome: a common source of knee pain. *American family physician* 2005; **71**(8): 1545-50.

131. Fredericson M, Weir A. Practical management of iliotibial band friction syndrome in runners. *Clin J Sport Med* 2006; **16**(3): 261-8.

132. Beynnon BD, Johnson RJ, Coughlin KM. Knee. In: DeLee JC, Drez DJ, Miller MD, eds. DeLee and Drez's Orthopaedic Sports Medicine. 2 ed. Philedelphia, Pa: Saunders; 2003: 1871-2.

133. Fairclough J, Hayashi K, Toumi H, et al. The functional anatomy of the iliotibial band during flexion and extension of the knee: implications for understanding iliotibial band syndrome. *Journal of anatomy* 2006; **208**(3): 309-16.

134. Fairclough J, Hayashi K, Toumi H, et al. Is iliotibial band syndrome really a friction syndrome? *Journal of science and medicine in sport / Sports Medicine Australia* 2007; **10**(2): 74-6; discussion 7-8.

135. Conn JM, Annest JL, Gilchrist J. Sports and recreation related injury episodes in the US population, 1997-99. *Inj Prev* 2003; **9**(2): 117-23.

136. Spinks AB, McClure RJ. Quantifying the risk of sports injury: a systematic review of activity-specific rates for children under 16 years of age. *British journal of sports medicine* 2007; **41**(9): 548-57; discussion 57.

137. Purvis JM, Hirsch SA. Playground injury prevention. *Clinical orthopaedics and related research* 2003; (409): 11-9.

138. Knowles SB, Marshall SW, Guskiewicz KM. Issues in estimating risks and rates in sports injury research. *Journal of athletic training* 2006; **41**(2): 207-15.

139. Finch CF. An overview of some definitional issues for sports injury surveillance. *Sports medicine (Auckland, NZ* 1997; **24**(3): 157-63.

140. Emery CA. Injury prevention and future research. *Medicine and sport science* 2005; **48**: 179-200.

141. Boufous S, Williamson A. Reporting of the incidence of hospitalised injuries: numerator issues. *Inj Prev* 2003; **9**(4): 370-5.

142. Boufous S, Finch C. Estimating the incidence of hospitalized injurious falls: impact of varying case definitions. *Inj Prev* 2005; **11**(6): 334-6.

143. Thomson JA, Ampofo-Boateng K, Lee DN, Grieve R, Pitcairn TK, Demetre JD. The effectiveness of parents in promoting the development of road crossing skills in young children. *The British journal of educational psychology* 1998; **68 (Pt 4)**: 475-91.

144. Gotsch K AJ, Holmgreen P et al. Nonfatal sports- and recreation-related injuries in emergency departments, United States, July 2000- June 2001. *JAMA* 2002; **288**: 1977-8.

145. Finch C, Valuri G, Ozanne-Smith J. Sport and active recreation injuries in Australia: evidence from emergency department presentations. *British journal of sports medicine* 1998; **32**(3): 220-5.

146. Cassell EP, Finch CF, Stathakis VZ. Epidemiology of medically treated sport and active recreation injuries in the Latrobe Valley, Victoria, Australia. *British journal of sports medicine* 2003; **37**(5): 405-9.

147. Burt CW, Overpeck MD. Emergency visits for sports-related injuries. *Annals of emergency medicine* 2001; **37**(3): 301-8.

148. Schneider S, Seither B, Tonges S, Schmitt H. Sports injuries: population based representative data on incidence, diagnosis, sequelae,

and high risk groups. *British journal of sports medicine* 2006; **40**(4): 334-9; discussion 9.
149. O'Rourke KP, Quinn F, Mun S, et al. A comparison of paediatric soccer, gaelic football and rugby injuries presenting to an emergency department in Ireland. *Injury* 2007; **38**(1): 104-11.

150. Faude O, Junge A, Kindermann W, Dvorak J. Injuries in female soccer players: a prospective study in the German national league. *The American journal of sports medicine* 2005; **33**(11): 1694-700.

151. Dyson R, Buchanan M, Hale T. Incidence of sports injuries in elite competitive and recreational windsurfers. *British journal of sports medicine* 2006; **40**(4): 346-50.

152. Purvis JM, Burke RG. Recreational injuries in children: incidence and prevention. *The Journal of the American Academy of Orthopaedic Surgeons* 2001; **9**(6): 365-74.

153. Norton C, Nixon J, Sibert JR. Playground injuries to children. *Archives of disease in childhood* 2004; **89**(2): 103-8.

154. Danseco ER, Miller TR, Spicer RS. Incidence and costs of 1987-1994 childhood injuries: demographic breakdowns. *Pediatrics* 2000; **105**(2): E27.

155. Ampofo-Boateng K, Thomson JA. Children's perception of safety and danger on the road. *Br J Psychol* 1991; **82 (Pt 4)**: 487-505.

156. Tinsworth DK MJ. Special Study: Injuries and Deaths Associated with Children's Playground equipment. Washington, DC: US Consumer Safety Commission, 2001.

157. American Academy of Pediatrics, Committee on Accident and Poison Prevention and Committee on Pediatric Aspects of Physical Fitness, Recreation, and Sports. Trampolines II. *Pediatrics* 1981; **67**.

158. Day CS, Yeh AC, Franko O, Ramirez M, Krupat E. Musculoskeletal medicine: an assessment of the attitudes and knowledge of medical students at Harvard Medical School. *Acad Med* 2007; **82**(5): 452-7.

159. Finch C, Cassell E. The public health impact of injury during sport and active recreation. *Journal of science and medicine in sport / Sports Medicine Australia* 2006; **9**(6): 490-7.

160. King S. The Clash of the Ash in Foreign Fields: Hurling Abroad. . Shannon, Co. Clare: Seamus J. King; 1998.

161. Ni Bhrolchain M. Book of Leinster. In: Duffy S, ed. Medieval Ireland An Encyclopedia. New York and Abingdon: Routledge; 2005: 272-4.
162. Hutchinson R. Camanachd!: The Story of Shinty: Birlinn Ltd.; 2004.

163. Davies J, Jenkins N, Baines M, P.I. L. The Welsh Academy Encyclopaedia of Wales. Cardiff: University of Wales Press; 2008.

164. gbrathletics. OLYMPIC GAMES MEDALLISTS - OTHER SPORTS. http://www.gbrathletics.com/olympic/other.htm - DEMO: Athletics Weekly; 2007.

165. NCAA. 2010-12 NCAA MEN'S AND WOMEN'S ICE HOCKEY RULES AND INTERPRETATIONS. 2010 http://www.ncaapublications.com/productdownloads/IH12.pdf.

166. NCAA. 2011-12 NCAA MEN'S LACROSSE RULES. 2010. http://www.ncaapublications.com/productdownloads/LC12.pdf.

167. NCAA. 2012 and 2013 NCAA WOMEN'S LACROSSE RULES AND INTERPRETATIONS. 2012 http://www.ncaapublications.com/productdownloads/WLC13.pdf.

168. Adams J, Hillman M. Bicycle helmets. Risk taking is influenced by people's perception of safety and danger. *BMJ (Clinical research ed* 2001; **322**(7293): 1063-4.

169. Dias JJ, Garcia-Elias M. Hand injury costs. *Injury* 2006; **37**(11): 1071-7.

170. Trybus M, Lorkowski J, Brongel L, Hladki W. Causes and consequences of hand injuries. *American journal of surgery* 2006; **192**(1): 52-7.

171. Watson AW. Sports injuries in the game of hurling. A one-year prospective study. *The American journal of sports medicine* 1996; **24**(3): 323-8.

172. Cardona I, D'Alonzo GE, Jr., Becker J. A pilot survey of beta2-agonist inhaler availability for children with asthma during organized sporting events. *Ann Allergy Asthma Immunol* 2004; **92**(3): 340-3.

173. Brooks JH, Fuller CW, Kemp SP, Reddin DB. Epidemiology of injuries in English professional rugby union: part 1 match injuries. *British journal of sports medicine* 2005; **39**(10): 757-66.

174. Eggleston PA, Rosenthal RR, Anderson SA, et al. Guidelines for the methodology of exercise challenge testing of asthmatics. Study Group on

Exercise Challenge, Bronchoprovocation Committee, American Academy of Allergy. *The Journal of allergy and clinical immunology* 1979; **64**(6 pt 2): 642-5.

175. Godfrey S, Silverman M, Anderson SD. The use of the treadmill for assessing exercise-induced asthma and the effect of varying the severity and duration of exercise. *Pediatrics* 1975; **56**(5 pt-2 suppl): 893-8.

176. Rundell KW, Wilber RL, Szmedra L, Jenkinson DM, Mayers LB, Im J. Exercise-induced asthma screening of elite athletes: field versus laboratory exercise challenge. *Medicine and science in sports and exercise* 2000; **32**(2): 309-16.

177. Grant S, Corbett K, Amjad AM, Wilson J, Aitchison T. A comparison of methods of predicting maximum oxygen uptake. *British journal of sports medicine* 1995; **29**(3): 147-52.

178. Leger LA, Lambert J. A maximal multistage 20-m shuttle run test to predict VO2 max. *European journal of applied physiology and occupational physiology* 1982; **49**(1): 1-12.

179. Helenius I, Haahtela T. Allergy and asthma in elite summer sport athletes. *The Journal of allergy and clinical immunology* 2000; **106**(3): 444-52.

180. IABA. iaba@eircom.net.

181. Borg GA. Psychophysical bases of perceived exertion. *Medicine and science in sports and exercise* 1982; **14**(5): 377-81.

182. Miller MR, Crapo R, Hankinson J, et al. General considerations for lung function testing. *Eur Respir J* 2005; **26**(1): 153-61.

183. MET ÉIREANN. http://www.met.ie.

184. Holzer K, Brukner P, Douglass J. Evidence-based management of exercise-induced asthma. *Current sports medicine reports* 2002; **1**(2): 86-92.

185. Dickinson JW, Whyte GP, McConnell AK, Nevill AM, Harries MG. Mid-expiratory flow versus FEV1 measurements in the diagnosis of exercise induced asthma in elite athletes. *Thorax* 2006; **61**(2): 111-4.

186. WADA. Medical Information to Support the Decisions of TUECs Asthma. 2009. http://www.wada-ama.org/rtecontent/document/Asthma_v1.2_15_01_2009.pdf.

187. Goldberg S, Schwartz S, Izbicki G, Hamami RB, Picard E. Sensitivity of exercise testing for asthma in adolescents is halved in the summer. *Chest* 2005; **128**(4): 2408-11.

188. Akuthota V, Stilp SK, Lento P. Iliotibial band syndrome. In: Frontera WR, Silver JK, eds. Essentials of Physical Medicine and Rehabilitation. Philadelphia, Pa: Hanley & Belfus; 2002: 328-33.

189. Ellis R, Hing W, Reid D. Iliotibial band friction syndrome--a systematic review. *Man Ther* 2007; **12**(3): 200-8.

190. Jordaan G, Schwellnus MP. The incidence of overuse injuries in military recruits during basic military training. *Military medicine* 1994; **159**(6): 421-6.

191. Holmes JC, Pruitt AL, Whalen NJ. Iliotibial band syndrome in cyclists. *The American journal of sports medicine* 1993; **21**(3): 419-24.

192. Messier SP, Edwards DG, Martin DF, et al. Etiology of iliotibial band friction syndrome in distance runners. *Medicine and science in sports and exercise* 1995; **27**(7): 951-60.

193. Orchard JW, Fricker PA, Abud AT, Mason BR. Biomechanics of iliotibial band friction syndrome in runners. *The American journal of sports medicine* 1996; **24**(3): 375-9.

194. Winston P, Awan R, Cassidy JD, Bleakney RK. Clinical examination and ultrasound of self-reported snapping hip syndrome in elite ballet dancers. *The American journal of sports medicine* 2007; **35**(1): 118-26.

195. Segal NA, Felson DT, Torner JC, et al. Greater trochanteric pain syndrome: epidemiology and associated factors. *Archives of Physical Medicine and Rehabilitation* 2007; **88**(8): 988-92.

196. Ekman EF, Pope T, Martin DF, Curl WW. Magnetic resonance imaging of iliotibial band syndrome. *The American journal of sports medicine* 1994; **22**(6): 851-4.

197. Pedowitz RN. Use of osteopathic manipulative treatment for iliotibial band friction syndrome. *The Journal of the American Osteopathic Association* 2005; **105**(12): 563-7.

198. Nishimura G, Yamato M, Tamai K, Takahashi J, Uetani M. MR findings in iliotibial band syndrome. *Skeletal radiology* 1997; **26**(9): 533-7.

199. Noble CA. Iliotibial band friction syndrome in runners. *The American journal of sports medicine* 1980; **8**(4): 232-4.

200. Orava S, Leppilahti J, Karpakka J. Operative treatment of typical overuse injuries in sport. *Annales chirurgiae et gynaecologiae* 1991; **80**(2): 208-11.
201. Muhle C, Ahn JM, Yeh L, et al. Iliotibial band friction syndrome: MR imaging findings in 16 patients and MR arthrographic study of six cadaveric knees. *Radiology* 1999; **212**(1): 103-10.

202. Hamill J, Miller R, Noehren B, Davis I. A prospective study of iliotibial band strain in runners. *Clin Biomech (Bristol, Avon)* 2008; **23**(8): 1018-25.

203. Gunter P, Schwellnus MP. Local corticosteroid injection in iliotibial band friction syndrome in runners: a randomised controlled trial. *British journal of sports medicine* 2004; **38**(3): 269-72; discussion 72.

204. Noehren B, Davis I, Hamill J. ASB clinical biomechanics award winner 2006 prospective study of the biomechanical factors associated with iliotibial band syndrome. *Clin Biomech (Bristol, Avon)* 2007; **22**(9): 951-6.

205. Kirk KL, Kuklo T, Klemme W. Iliotibial band friction syndrome. *Orthopedics* 2000; **23**(11): 1209-14; discussion 14-5; quiz 16-7.

206. Noble CA. The treatment of iliotibial band friction syndrome. *British journal of sports medicine* 1979; **13**(2): 51-4.

207. McKenzie DC, Clement DB, Taunton JE. Running shoes, orthotics, and injuries. *Sports medicine (Auckland, NZ* 1985; **2**(5): 334-47.

208. Schwellnus MP, Theunissen L, Noakes TD, Reinach SG. Anti-inflammatory and combined anti-inflammatory/analgesic medication in the early management of iliotibial band friction syndrome. A clinical trial. *South African medical journal = Suid-Afrikaanse tydskrif vir geneeskunde* 1991; **79**(10): 602-6.

209. Peeler J, Cooper J, Porter MM, Thliveris JA, Anderson JE. Structural parameters of the vastus medialis muscle. *Clinical anatomy (New York, NY* 2005; **18**(4): 281-9.

210. Charnley J. Orthopaedic signs in the diagnosis of disc protrusion. With special reference to the straight-leg-raising test. *Lancet* 1951; **1**(6648): 186-92.

211. Ober FB. The role of the iliotbial band and fascia lata as a factor in the causation of low back disabilities and sciatica. *J Bone Joint Surg* 1936; **18**: 105-10.

212. Barker PJ, Briggs CA, Bogeski G. Tensile transmission across the lumbar fasciae in unembalmed cadavers: effects of tension to various muscular attachments. *Spine* 2004; **29**(2): 129-38.
213. Alshami AM, Babri AS, Souvlis T, Coppieters MW. Biomechanical evaluation of two clinical tests for plantar heel pain: the dorsiflexion-eversion test for tarsal tunnel syndrome and the windlass test for plantar fasciitis. *Foot Ankle Int* 2007; **28**(4): 499-505.

214. Coppieters MW, Alshami AM, Babri AS, Souvlis T, Kippers V, Hodges PW. Strain and excursion of the sciatic, tibial, and plantar nerves during a modified straight leg raising test. *J Orthop Res* 2006; **24**(9): 1883-9.

215. Kogler GF, Veer FB, Solomonidis SE, Paul JP. The influence of medial and lateral placement of orthotic wedges on loading of the plantar aponeurosis. *J Bone Joint Surg Am* 1999; **81**(10): 1403-13.

216. Arampatzis A, Karamanidis K, Morey-Klapsing G, De Monte G, Stafilidis S. Mechanical properties of the triceps surae tendon and aponeurosis in relation to intensity of sport activity. *Journal of Biomechanics* 2007; **40**(9): 1946.

217. Magnusson SP, Hansen P, Aagaard P, et al. Differential strain patterns of the human gastrocnemius aponeurosis and free tendon, in vivo. *Acta Physiologica Scandinavica* 2003; **177**(2): 185.

218. Bryant AL, Clark RA, Bartold S, et al. Effects of estrogen on the mechanical behaviour of the human Achilles tendon in vivo. *J Appl Physiol* 2008.

219. Falvey EC, Clark RA, Franklyn-Miller A, Bryant AL, Briggs C, McCrory PR. Iliotibial band syndrome: an examination of the evidence behind a number of treatment options. *Scandinavian journal of medicine & science in sports* 2010; **20**(4): 580-7.

220. Barber FA, Sutker AN. Iliotibial band syndrome. *Sports medicine (Auckland, NZ* 1992; **14**(2): 144-8.

221. Evans P. The postural function of the iliotibial tract. *Annals of the Royal College of Surgeons of England* 1979; **61**(4): 271-80.

222. McAtee RE, Charland J. Facilitated Stretching. 3rd ed. Champaign, Illinois, USA: Human Kinetics; 2007.

223. Fredericson M, White JJ, Macmahon JM, Andriacchi TP. Quantitative analysis of the relative effectiveness of 3 iliotibial band

stretches. *Archives of Physical Medicine and Rehabilitation* 2002; **83**(5): 589-92.

224. Yinen J. Sports Massage. London: Ebury Press; 1997.

225. Olerud C, Berg P. The variation of the Q angle with different positions of the foot. *Clinical orthopaedics and related research* 1984; (191): 162-5.

226. Perry D. Personal communication on the use of ultrasound in the diagnosis of ITBFS at the London Independent Hospital.; 2013.

Appendices

Appendix 1: Questionnaire of Hurling Related Injuries Presenting to the Emergency Department

Questionnaire of Hurling Related Injuries Presenting to the Emergency Department

Name: ________________________________

Age: ________

Phone Contact: ____________________

Date of Presentation to ED: _________________ **Date of Injury:** _______________

Area Injured: ____

Hand Dominance: ____

Level of Participation:____

Level of Participation at time of Injury: ________________________________

Mechanism of Injury:

Struck by Ball: ☐ Struck By Playing Equipment: ☐ Collision: ☐ Foul Play: ☐

Protective Equipment:

Helmet: ☐ Faceguard: ☐ Hand Protection: ☐ Gumshield: ☐ Shinguard: ☐

Treatment: ____

Time Out of Work: ____

Time Until Back To Play: ____

Have You Previously Tried Using Hand Protection? Yes ☐ No ☐

Why was Use of Hand Protection Discontinued?

Discomfort: ☐ Ineffective Protection: ☐ Limitation in Performance: ☐

Poor Aaesthetics: ☐ Expense: ☐

Do You intend to use other protective equipment in the future? Yes ☐ No ☐

Appendix 2: Exercise Challenge for Exercise Induced Bronchoconstriction Protocol

Whole Body Test- Exercise Challenge for EIB

8-10 Minute High Intensity Exercise

Each Player is given a weighted 'Medicine' ball. These balls weigh 3,4,5 & 6kg. A different ball is used for each 'set'.

1 SET: 4 exercises repeated twice, followed by a sprint.

1. Exercise 1: player standing both feet parallel drives the med ball in explosive throws to his right side in a rugby ball pass motion at the wall for 15 reps
2. Exercise 2: player turns to his left side and repeats the exercise for 15 reps
3. Exercise 3: player holds the med ball at face level and drives the ball in explosives throws/Slams towards the floor for 15 reps
4. Exercise 4: explosive squat jumps with the med ball held out in front of chest 15 reps
5. Steps 1-4 are repeated
6. After this set the player must sprint with the med ball 20 meter out and back all out effort

This circuit has to be repeated four times with no rest in between. After each 20 metre sprint, the player changes ball weight, this can be varied according to preference.

Time of exercise varies according to players ability/strengths. It should take between 8 and 10 mins to complete the full circuit.

Appendix 3: Patient Symptom Questionnaire: Exercise Induced Bronchoconstriction in Rugby

EIB in Rugby Study, Symptom Questionnaire

Department of Rheumatology, Sport & Exercise Medicine, Cork University Hospital

Study Number:__________

Height:____________ **Weight:**____________ **Resting HR:**_____________

Have you had wheezing or whistling in your chest at any time in the last 12 months?
☐ Yes ☐ No

Have you woken with a feeling of chest tightness or shortness of breath in the last 12 months?
☐ Yes ☐ No

Have you had an attack of shortness of breath in the last 12 months?
☐ Yes ☐ No

Have you had an attack of shortness of breath after strenuous exercise in the last 12 months?
☐ Yes ☐ No

Have you ever had pneumonia or other lung problems?
☐ Yes ☐ No

Do you get a cough after strenuous exercise?
☐ Yes ☐ No

Do you suffer from nasal allergies or hay-fever?
☐ Yes ☐ No

Have you ever had asthma? ☐ Yes ☐ No
Was this confirmed by a doctor? ☐ Yes ☐ No

How many asthma attacks have you had in the last 12 months?
☐ 0
☐ 1-20
☐ >20

Medications used:
Inhalers:
1. ____________________________
2. ____________________________
3. ____________________________

Oral medications:
1. ____________________________
2. ____________________________
3. ____________________________

Appendix 4: Patient Information Sheet and Consent Form, Version 2: Exercise Induced Bronchoconstriction.

UNIVERSITY COLLEGE CORK

Clinical Research Ethics Committee Of The Cork Teaching Hospitals

CONSENT BY SUBJECT FOR PARTICIPATION IN RESEARCH PROTOCOL

Section A

Protocol Number: Patient Name:______________________

Title of Protocol: **To evaluate the incidence of exercise induced asthma (EIA) in elite and non-elite Irish Rugby players.**

Doctors Directing Research:

Dr. Michael G Molloy, Dr Eanna Falvey Phone: 021 4922817

You are being asked to participate in a research study. The doctors at University College Cork study the nature of disease and attempt to develop improved methods of diagnosis and treatment. In order to decide whether or not you want to be a part of this research study, you should understand enough about its risks and benefits to make an informed judgment. This process is known as informed consent. This consent form gives detailed information about the research study, which will be discussed with you. Once you understand the study, you will be asked to sign this form if you wish to participate.

Section B

Asthma can occur in everday life but may also be brought on by sport. Exercise induced asthma (EIA) causes symptoms of coughing, wheezing, shortness of breath and chest tightness during or after exercise. If left untreated, this can cause a reduction in ability to exercise at high level.

A number of studies have shown that high intensity exercise performed in cold conditions can increase an athlete's chances of developing this problem. These studies have also shown that the problem is under-treated.
The aim of the study is to identify how many professional players in Ireland are affected by EIA; we will also compare the results with the same number of non-professional players from the All-Ireland League (AIL).

To do this we need to perform a breathing test before and after a contact training session, and to collect information on breathing symptoms you might have.
This study would involve you performing breathing tests before and after training, for this you will blow hard into a machine three times (your best

score being recorded) before a training session. At this time you will fill out a questionnaire about symptoms of asthma. After the training session the breathing test will be repeated in the same manner as before.

There are no risks to this procedure, it will be explained in detail, it is widely used and you will be instructed by a doctor.

Patient confidentiality will be maintained at all times.

This study is for the purposes of diagnosis rather than treatment. The results of the tests will be disclosed only to you, at your request at any time. A contact number for the primary investigator is available on the information sheet provided; please retain this for further use.

Section C

AGREEMENT TO CONSENT

The research project and the treatment procedures associated with it have been fully explained to me. All experimental procedures have been identified and no guarantee has been given about the possible results. I have had the opportunity to ask questions concerning any and all aspects of the project and any procedures involved. I am aware that participation is voluntary and that I may withdraw my consent at any time. I am aware that my decision not to participate or to withdraw will not restrict my access to health care services normally available to me. Confidentiality of records concerning my involvement in this project will be maintained in an appropriate manner. When required by law, the records of this research may be reviewed by government agencies and sponsors of the research.

I understand that the sponsors and investigators have such insurance as is required by law in the event of injury resulting from this research.

I, the undersigned, hereby consent to participate as a subject in the above described project conducted at the Cork Teaching Hospitals. I have received a copy of this consent form for my records. I understand that if I have any questions concerning this research, I can contact the doctor(s) listed above. If I have further queries concerning my rights in connection with the research, I can contact the Clinical Research Ethics Committee of the Cork Teaching Hospitals, Clinical Sciences Building, Cork University Hospital, Wilton, Cork.

After reading the entire consent form, if you have no further questions about giving consent, please sign where indicated.

Doctor: ____________________ ____________________

Signature of Subject or Guardian

Witness:____________________ Date: _______ Time: