U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

Anterior Cruciate Ligament injuries in Portuguese female handball players

A Thesis submitted for the degree of Doctor of Philosophy in Sports Sciences
Sports Faculty – Porto University

Supervisor: Professor Doctor José Leandro da Rocha Massada
Co-supervisor: Professor Doctor João F. Montenegro Andrade Lima Bernardes

Maria Luísa Dias Estriga
Porto, 2008

Estriga, Maria Luísa D. (2008)

Anterior Cruciate Ligament injuries in the Portuguese female handball players
Doctoral dissertation thesis on Sports Sciences, submitted to the Sports Faculty – Porto University

Keywords: sex hormones, muscular strength, epidemiology, knee laxity.

This work was partially financed by the Instituto de Desporto de Portugal – PAFID project

ACKNOWLEDGEMENTS

First of all, I would like to express my deepest gratitude to the many players and coaches who participated in this study. A special thank you *is due to the players who unconditionally participated in the hormonal environment and knee laxity study, which was truly demanding. Without them this project would not have been possible.*

I would like to express my special gratitude to Leandro Massada, MD, PhD, Professor at the Sports Faculty, Porto University, and my adviser, for introducing me to this area and for his guidance during the course of this work. His experience and knowledge of sports medicine is truly impressive.

João Bernardes, MD, PhD, Professor at the Medicine Faculty, Porto University, and my co-adviser, for his guidance and interest.

João Carvalho, assistant at the Physics Department, Faculty of Engineering, Porto University, for his invaluable help in the design and production of this thesis, as well as for his close friendship. Our interesting and stimulating discussions about this study, research and life in general have made this long journey less difficult and more enjoyable.

Elsa Bronze da Rocha, PhD, Professor at the Chemistry Service, Faculty of Pharmacy, Porto University, for invaluable help in the start of this research project.

António Marques, Jorge Bento, José Soares and João Villas-Boas, Professors at the Sports Faculty, Porto University, for listening to me and for the small but sometimes decisive words in several stages of my career.

Sandra Schültz, PhD, University of North Carolina, for the interest and suggestions on all things hormonally related.

Teresa Tomé, Professor at the Oporto Nursing School, and Nuria from RENAFER, for teaching me the secrets of the *basal body temperature* method, used as a co-variable for ovulation detection in some of the otherwise dubious cases.

António Cunha, head of the Handball Department, Sports Faculty, Porto University, for unconditional support and incentive.

Ireneu Moreira, for valuable discussions about handball practice, and unconditional support and incentive.

Eduardo Oliveira, Susana Estriga, Aldina Sofia, Mariana Carvalho, Alda Pena, Sandra Neto and Cecília Hipólito for helping with data gathering.

David Davies, my English teacher, who still hasn't given up on me.

Paula Baptista, for all the caffeine (and more…) shared during this long journey.

Ana Maria, Acácio Couto, Paulo Fernandes, Carla Couto and João Andrade for helping with the practical side of life during my periods of absence.

My parents and sister for unconditional support and love throughout my life and also during the thesis. To them all my sincere gratitude.

My little Afonso, for being the *heart* of this work. His smile, understanding and complicity has been truly inspiring.

Alberto Lemos, MD and Orthopaedics Unit at the Hospital da Prelada, for making the arthrometer available to us.

Dra Graça Salcedo and ENDOCLAB for the helpful response to all my unusual requests.

Abstract

This work studies the æthiology of the most severe injury in the Portuguese female handball population. It follows three main lines of research that interact among themselves.

A first one attempts to characterise the injury incidence and profile of this population. About 1/3 of all the female federated handball players (older than 12 years) were sampled retrospectively and/or prospectively throughout 3 sport seasons. The main conclusion is that ACL ruptures are, among the severe injuries, the most occurring ones.

A second one attempts to compare statistically the muscular force changes and the ACL injury risk. We measured some isokinetic muscular force indicators in the various subgroups (by age, years of practice, playing position, laterality, etc.) and accounted for all previous ACL injuries in these same subgroups. In some cases a few players being followed suffered ACL injuries during the study and constitute themselves as a prospective component of this study. A small correlation (≈60%) could be established between these indicators and the ACL injury risk. A strong predictor is the hamstrings peak torque. Also measured was the jumping performance that revealed as having little correlation with the knee laxity.

A third one consisted in a comparison of the monthly changes in the serum hormonal contents of a few players (with and without usage of oral contraceptives) with both the lower limb muscular strength and the anterior knee laxity. Only a faint correlation (≈50%-60%) could be seen between the hormonal levels and the knee laxity, with no relevance of the jump performance. Improvement in knee laxity in those using OCs could be seen in non-athletes but not in handball players.

These findings are mainly negative results but are strong enough for building the hypothesis of a decisive role of the practice load on the risk and prevention of ACL injuries.

Resumo

Este trabalho estuda a etiologia da lesão mais grave que afecta a população portuguesa de andebol feminino. Percorre três linhas de investigação principais que interagem entre si.

Na primeira caracteriza-se a incidência e o perfil lesional da população em estudo. Cerca de 1/3 das jogadoras de andebol federadas em Portugal (com idade superior a 12 anos) foram estudadas retrospectivamente e/ou prospectivamente em três épocas desportivas. Concluiu-se que a ruptura do Ligamento Cruzado Anterior é, entre as lesões graves, a mais frequente.

Na segunda comparam-se estatisticamente os níveis de força muscular e o risco de lesão do ACL. Foram medidos alguns indicadores de força muscular isocinética em vários subgrupos (em função da idade, anos de prática, posto específico, lateralidade, etc.) bem como de capacidade de salto, tendo sido comparados com lesões prévias e subsequentes. Foi encontrada uma fraca correlação (≈60%) entre os indicadores isocinéticos avaliados e o risco de lesão do ACL. O torque máximo dos isquiotibiais revelou ser um forte preditor de lesão do ACL.

A terceira compara a evolução das concentrações hormonais sanguíneas ao longo do ciclo menstrual (com ou sem a influência de contraceptivos orais), com a força muscular dos membros inferiores e a laxidez anterior do joelho. Uma fraca correlação (≈50%-60%) foi encontrada entre os níveis hormonais e a laxidez anterior do joelho. Não foi observada relevância preditiva na capacidade de salto. O uso de contraceptivos orais diminui a laxidez anterior do joelho em sujeitos não-treinados mas não nas jogadoras de andebol.

Estes resultados são fundamentalmente negativos, mas suficientemente fortes para construir a hipótese da carga desportiva ser um dos principais factores de risco (e de prevenção) de lesão do ACL.

Résumé

Ce travail étudie l'étiologie des blessures les plus graves auprès de la population féminine portugaise de *handball*. Trois lignes principales de recherche en interdépendance ont été retenues.

La première se propose de caractériser l'incidence et le profil des blessures sur cette population. Environ 1/3 des sportives licenciées portugaises (âgées de plus de 12 ans) a fait l'objet d'une observation rétrospective et/ou prospective sur trois saisons sportives. La principale conclusion est que la rupture du ligament croisé antérieur (LCA) est, de toutes les blessures graves, la plus fréquente.

La deuxième a pour objet la comparaison statistique des niveaux de force musculaire et le risque de rupture du LCA. Nous avons mesuré quelques indicateurs de la force musculaire isocinétique sur divers sous-groupes (en fonction de l'âge, années de pratique sportive, position de jeu, latéralité, etc.) ainsi que de la capacité de saut, les indicateurs ayant été mis en relation avec les blessures précédentes et subséquentes. Une faible corrélation (≈60%) entre les indicateurs isocinétiques évalués et le risque de rupture du LCA s'est vérifiée. Le torque maximum des ischio-jambiers s'est avéré un fort prédicteur de la lésion du LCA.

La troisième compare l'évolution du contenu hormonal du sang durant la menstruation (avec ou sans influence de contraceptifs oraux) et la force musculaire des membres inférieurs et la laxité du genou. Une faible corrélation (≈50%-60%) entre ces variables a été démontrée. L'utilisation de contraceptifs oraux diminue la laxité du genou sur des sujets non -entraînés mais pas sur les joueuses de handball.

Les résultats obtenus sont fondamentalement négatifs mais suffisamment consistants pour construire l'hypothèse suivante: l'intensité de la pratique sportive est un des principaux facteurs de risque (et de prévention) de la rupture du LCA.

Table of contents **page**

Figure index **page**

Figure index **page**

Figure index	page

Table index **page**

Table index **page**

Table index	**page**

Acronyms and abbreviations

AAP	Associação de Andebol do Porto (Porto regional handball association)
ACL	anterior cruciate ligament
AIC	Akaike's information criteria
AM	anteromedial ACL bundle
ANOVA	analysis of variance
BBT	body basal temperature
BMD	bone mineral density
BMI	body mass index
CDC	U.S. Centers for Disease Control and Prevention
CI	confidence interval
CMJ	countermovement jump(s)
CRH	corticotrophin-releasing hormones
E_2	oestradiol
ESD	explosive strength deficit
EUR	elastic energy utilization ratio
F	Fisher index
FHA	functional hypothalamic amenorrhea
FPA	Federação Portuguesa de Andebol (Portuguese Handball Federation)
Fr	French, France
FSH	follicle-stimulating hormone
GnRH	gonadotropin-releasing hormone
h	hours / height
H:Q	ratio of peak knee flexor torque to peak knee extensor torque
HPA	hypothalamic-pituitary-adrenal
HSD	Tukey's honestly significant differences test
IGF	insuline-like growth factor
IGFBP	IGF binding protein
IDP	Instituto do Desporto de Portugal (Portuguese Sports Institute)
IHF	International Handball Federation
IQR	interquartile range
LH	luteinizing hormone
LP	luteal phase
LPD	luteal phase deficiency
LSV	least significant value (at 80% power)
MCL	medial collateral ligament

min.	minute(s)
MRI	magnetic resonance imaging
μ	arithmetic mean
NFP	natural family planning
non-OC	Non-user(s) of oral contraceptives
OCP	oral contraceptive pill
OC(s)	oral contraceptive(s) [users]
p	[critical] probability, number of parameters
P_4	progesterone
PCL	posterior cruciate ligament
PL	posterolateral ACL bundle
PMS	premenstrual syndrome
prob	probability
R, r	[Pearson] [Spearman rank] correlation coefficient
ROC	receiver operating characteristics
SHBG	sex hormone binding globulin
SJ	squat jump(s)
T	testosterone
T_3	triiodothyronine
WCh	World Championship

Chapter 1

Introduction

1.1. Introduction

I have played handball since I was 17; when I was 31 I suffered an Anterior Cruciate Ligament (ACL) rupture in a handball game (a very important game for our aspirations). I remember every detail as if it were yesterday. The game was finishing and we were attacking, trying to score a goal. I was in the left back-court position and in order to make a right hand shot or to create numerical superiority, after receiving the ball from the left, while running near the nine metre line, I moved forward to change direction with the ball. When I planted my right foot intending to cut to the left, my foot seemed to lock to the artificial floor. Suddenly my right knee gave way and immediately I fell to the ground, unable to leave without help. My handball career as a player was over! Although I've decided to reconstruct the ACL, six years after being injured and four years after the surgery I still feel knee instability and disability in practicing sports. After this experience I was able to understand what many handball players went through: the fear of getting another ACL injury becoming the motive for many handball players to give up.

As a handball coach I could observe this phenomenon in another perspective. In one season, in my club, we had two ACL injuries from two young promising female players, which brought lots of problems related to insurances, surgeries, treatments, practice reintegration, etc. This made me question: why, on a particular day, the knee strains in a movement done thousands of times before? Could an ACL injury mean that the training process is not well guided? Is this injury inevitable or is there something that can be done by the coach to prevent it or to reduce the risk?

The opportunities for girls and women to play handball have rapidly grown and gained acceptance after the game became Olympic, particularly in Europe. Eventually this phenomenon has extended to Portugal.

The benefits of being physically active are uncontestable, independent of activity type (recreational or competitive sports), although several concerns about female practice have emerged, related to injury susceptibility and triad syndrome.

A focus on the ACL injury problem has been growing, not just in the research field, but also among coaches, athletes, parents, health care personal and even the public at large. This is probably one of the most noticed sports injuries.

The idea of a sex-related pre-disposition to ACL injury has emerged in the last decade or two, supported by studies emphasizing a higher incidence in female, comparative to the male counterparts. The ACL injury has been reported to occur two to nine times more in women compared to men, depending on sport and level of competition.

The attention placed on this injury is connected with the need of reconstructive surgery and intensive rehabilitation, which inevitably results in long intervals on the bench, with significant sporting and economical consequences including, possibly, the end of ones career. Also there are long-term consequences that have been observed whether surgery has been undertaken or not, like premature osteoarthritis of the knee.

Today, the focus has shifted from treatment to prevention. But to design and develop an efficient prophylactic intervention, it is essential to determine the real injury incidence, the underlying mechanisms and the risk factors (internal and external). There are different methodological approaches to describe the *inciting event* of injury but there still aren't any detailed models of the injury mechanism. This is probably due to the multifactorial nature of ACL tears which, in the women's cases, is even more complex. All this suggests the need to include the interaction of the distinct risk factors in our models.

There are lots of contributions and works published trying to explain the sex differences on ACL injury incidence. However after two decades of studies, no more than a few conjectures and common sense observations have become consensual.

Many sex differences have been reported by several authors and identified as *possible* risk factors. They have been classified as

- anatomical: Q-angle, knee morphology, hip angle, pelvic width, ligamentar laxity, etc.
- neuromuscular: coordinative capabilities, muscular strength, skill level, body movement, playing experience, etc.
- hormonal

Women have a specific hormonal environment, so it has been suggested that the hormonal fluctuations during a *normal* menstrual cycle can change the ligamentous structure and elastic properties, and this can possibly alter the risk of ACL injury. So far the experimental studies found contradictory results on the influence of the hormonal environment on either the injury incidence and also in ligament laxity. There is also a lack of consensus on the possible impact of the ligament laxity on ACL injury risk.

Nowadays the use of the oral contraceptive pill (OCP) is expected to be frequent in young females. The main effect of the OCP (containing synthetic derivatives of the female sex hormones) is to promote a stable hormonal environment in order to suppress normal ovarian function, leading some to speculate that they could have a prophylactic effect on the ACL injury risk. Few studies have looked into this hypothesis, and have not yet provided enough evidence to advise female athletes to use OCP in order to reduce ACL injury risk.

1.2. General Purposes

This work tries to contribute to the general effort of understanding the high incidence of ACL injuries in the female sportive population, namely by following the hormonal path and the consequent anterior knee laxity variations.

1.3. Organization of the Thesis

The present study comprises of seven chapters and respective appendices.

Chapter 1 – Introduction

The introduction (this chapter) presents the framework of female ACL injury problem, our personal motivation, emphasizing the complexity of the phenomenon and how much work has still to be done concerning the ætiology, mechanisms and risk factors of the injury, in order to achieve and implement reasonable prevention efforts.

Chapter 2 – Literature Review

In this chapter the established knowledge about women sports participation is reviewed, including the injury profile and menstrual cycle disturbances. The handball game development is analyzed in order to contextualize the injury incidence, severity and pattern in both male and female players. The ætiology of the ACL injuries in female players as well as the published prevention strategies are reviewed. Studies about the female hormonal environment, its possible connection to the ACL injury problem and knee laxity are discussed.

Chapter 3 – Injuries in Female Handball Players and Gynæcological Profile

The purpose of this first survey is to characterize the injury profile of Portuguese handball players, to know the ACL injury context and external mechanisms and its relation with the menstrual cycle characteristics. It’s a prospective and retrospective cohort study, by means of self-completing questionnaire (with pre-tested reliability and validity).

Chapter 4 – Muscle strength and Anterior Knee Laxity

The goal of this study is to analyze and compare the anterior knee laxity and reactive strength (maximal jump height) with previous knee injuries, hand laterality, years of practice, playing position and menstrual cycle characteristics. The players were cross-sectionally evaluated and the knee injury and gynæcological profiles examined using a questionnaire completed by the players themselves.

Chapter 5 – Isokinetic Strength of Hamstrings/Quadriceps and ACL injuries

The aim of this study is to investigate possible isokinetic muscular strength changes or adaptations over the years of handball practice, including the possible effects of specific playing positions and previous ACL injuries. It is also briefly investigated the complementary relation: how isokinetic muscular strength alters the injury risk. The isokinetic strength was cross-sectionally evaluated and the injury incidence was prospectively followed up.

Chapter 6 – Sex Hormones, Muscle Strength and Anterior Knee Laxity

This work explores the possible influence of female sex hormones in the anterior knee laxity in handball players. A prospective study during one menstrual cycle with two groups (with and without usage of OCP) was undertaken.

Chapter 7 – Conclusions

In this chapter the main conclusion of these studies and some considerations about possible lines of research opened by this work are presented.

Appendices

In appendices the main version of the used questionnaire, instructions for self-monitoring the menstrual cycle and informed consent agreements are added.

Chapter 2

Literature Review

2.1 Introduction

Several myths about female's lack of abilities in competitive sports have progressively evaporated over the last half a century (Kanstrup, 2005). Nowadays women have proved to be as capable as men in almost all areas of activity, although there are obvious differences when the performance exclusively depends on physical capacities and morphology.

The onset of puberty is one of the main causes for sex performance differences, but the sociocultural environment can also produce a favourable or unfavourable effect (Lebrun & Rumball, 2001). From a biological perspective, the response mechanism to physical training/detraining is similar in both sexes (Kanstrup, 2005), although males have a different set of characteristics that may make them fitter for most of the sports. These include physical morphology, aerobic and anaerobic capacity and muscular strength (Lebrun & Rumball, 2001).

In addition, the female sex hormonal environment is the main inductor for changes in bone mass, body composition, circulation and metabolism (Holschen, 2004). Comparatively to man, the female shows higher body fat and less lean body mass, which is explained by increased estrogens levels in contrast to increased androgens in males (Ireland & Ott, 2004).

Møller-Nielsen and Hammar (Moller-Nielsen & Hammar, 1989) prospectively investigated traumatic injuries in 108 soccer female players and, for the first time, linked the premenstrual phase and menses to a higher injury susceptibility, emphasizing the role of premenstrual symptoms. They found a decreased rate of traumatic injures in OC users.

2.2 Injuries in Team Handball

Handball is one of the most popular team games in Europe, within both male and female players. Naturally, the injuries are a growing cause of concern as the sport develops, along with an increasing popularity. The risk of injury has been emphasized in relation to the dynamic character of the game, the aggressiveness and the frequent physical contact between players (Ronglan *et al.*, 2006; Myklebust *et al.*, 1997). However, the complete interpretation of the causes and mechanisms of injury may be more complex.

It is quite understandable that the risk of injury is related with the sports structure and function, which explains the variability between different sports. Some sports are considered safer than others: most of the endurance sports compared with sports characterized by high velocity and strong contact (Parkkari *et al.*, 2001). In endurance sports most of the injuries are overuse injuries, and uncommonly they result in permanent disability (Parkkari *et al.*, 2001). Comparatively, the injuries observed in team games, such as handball, soccer, volleyball, basketball, *etc*. are more incapacitating and with potentially severe consequences in sport performance and individual health, among others. Other obvious divisions can be made, like between invasive (*e.g.* handball) *versus* non-invasive (*e.g.* volleyball) sports.

In the last few decades, the structure (and the rules) of the handball game has undertaken considerable changes, along with improvements in players' preparation, material and equipment. Given these changes, the injury risk is more than likely linked to the development of the game. External factors related to culture and political-geographic situation may also influence the risk of injury.

More specifically, using world champions as role models, it appears that the concepts and methods used to play, in attack and defence, as well as the profile of the successful handball player, have changed considerably. These new game trends may produce differences in injury patterns and make the comparison between studies harder. Even though, there is no doubt that today's handball is a possibly traumatic sport (Vlak & Pivalica, 2004).

2.2.1 Handball game development and injuries

In the last years, handball has become clearly faster, with more rhythm, and more effective time of play, along with a higher number of players being involved, especially with the last set of changes introduced in the Rules of the Game (the rules).

Since the 1976 Olympic Games (Montreal) promoting the physical integrity of the players has become a priority of the competent organisms. In the 1980 IHF Congress (Moscow), the disqualification and the exclusion was introduced in the rule of punishments, in an attempt to *moralize* the game. In 1995, more rule changes were carried out for promoting the game spectacularity and a less violent and rough behaviour (Seco, 2006). The more relevant changes are summarized as follows: (1) a faster throw-off from the centre after a goal; (2) a better interpretation of the *advantage rule*; (3) new rules about *passive game*; (4) to allow the registration of 14 players; (5) clarification of the disciplinary sanctions; (6) better interpretations about *faults in attack*; and, more recently, (6) the facilitation of the goalkeeper-throw.

Certainly, the last rule changes and their interpretations by players, coaches and referees have been decisive in the actual trends of the game. Today, the idea of a high-speed game has gained consistency, as recently presented in a technical communication by Dietrich Spatte[1] (see table 2.1).

Table 2.1 Average number of attacks per match and time average by attack (Dietrich Spatte[1]).

	Total attacks/match	Average time/attack
Final World Championship (WCh) 05 – Spain/Croatia	130	27.7’’
Women-WCh 06 (84 analyzed matches)	129,8	27.7’’
Kiel/Magdeburg 2006	160	22.5’’

In addition, Juan de Dios Seco[2] has presented interesting concepts about game tendencies, supporting his ideas in data from the 12 first teams in several international competitions since 2004 (table 2.2).

Table 2.2 Average number of attacks per match and time average by attack, from international competitions (Seco, 2007)

	Total attacks/match	Average time/attack
Athens 04	115	31.1’’
Tunisia 05	120	29.9’’
Switzerland 06	117	31.2’’
Germany 07	117	26.0’’

To be fast in handball means also deciding fast. This high speed decision-making capacity is a defining quality of the best players, as it is emphasized by Lino Cervar, the Croatian national coach, cited by Seco (2007).

Considering this, the modern handball preparation seems guided by a different game concept. The handball preparation based on individual technique capabilities and on a very strict, rigid, game system has been replaced by an emerging tactical-strategic game perspective, supported on sub-group and/or individual tactical decision-making. This gives an increased relevance to the tactical behaviour of the pivot(s) in the *positional game*, and to the tactical decisions of just two or three players instead of six (Seco, 2007). With this, the game has become deeper, played farthest from the goal and as a consequence of the decreased size of the tactical unit, the attacks are shorter, speeding the game.

Exploring further this line of reasoning, the same author used statistics from the World Championship to present data indicating a tendency of an increasing number of goals from 6m in contrast to a slight decrease from the 9m area. The author explains this

[1] VIII Clinic International Asociación de Entrenadores de Balonmano, 2006, León, Spain
[2] Lecturer from the European Handball Federation (EHF) and the International Handball Federation (IHF)

tendency with an increased use of counterattack and/or fast attack, and an increased effort to penetrate the defensive space, which therefore creates more chances of scoring. Additionally the better use of the attacking space, with an increasing number of shots to the goal in more favourable situations, tends to reduce the physical contact.

This new interpretation of the game, with its faster attacks, provoked a symmetrically deeper and faster defence answer, and a more intensive usage of the *big space* (J.I.Moreira private communication, n/d). This deeper/quicker defensive answer is characterized by an immediate defensive organization, probably in the midfield, pressing the player with the ball and interfering with the passing lines by contrast with the traditional strategy of recovering to a classical *team organized defence* near the goal area. These new defence systems tend to be more dynamic and active with a variable geometry and much more real-time options (Seco, 2006).

As mentioned before, a stricter interpretation of the regulations with progressive punishments and even direct disqualifications has been promoted by the competent organisms for increasing the player's safety. However, we were unable to find studies that prove/refute this intended safety increase. Beside all the efforts to have clear rules and ensure its uniform application, there are still difficulties in this area (Seco, 2007). The number of Clarification issues proves this point. Moreover, the *speed game* demands an appropriate physical preparation by the referee in order to adopt an adequate position in the field during the game.

The female handball game seems to follow the same tendencies, yet some particularities can easily be seen, like superior use of counterattack methods and a greater focus in playing near the restrictive area (Bon, 2002), possibly influenced by a weaker shot capacity.

Aagaard[3] (2007) stresses the fact that in the 2006 European female handball Championship the number of attacks didn't increase, contrary to what was expected. She also emphasises her perspective that the game has become faster and with more space, which seems difficult to compatibilize with the attack statistics. Still she enumerates a few possible explanations: (1) defence methods used (an active defence can lead to frequent stops in the game) (2) time expended in substitutions during the positioned offensive phase (3) time required to build-up the positioned attack (this has no time limit). Moreover the official statistics define fast breaks rather restrictively, excluding some fast attacks and not reflecting the true *speed* of the game appropriately. And, in addition to point (1) above, improved defences can make attacks longer without necessarily slowing the game down.

[3] Leader of the Section for Play Development and Coaching in the Norwegian Handball Federation

An additional factor specific to the female handball practice, reported by Dietrich Spatte and based solely on anecdotal evidence, is the possibility that female players show a superior percentage of decision-making mistakes. This obviously needs a rigorous study but, if true, would add to all the physiological differences in explaining the different speed and interpretation of the game.

All these new game tendencies are followed by new training methods, more specific and more tactically orientated, giving less relevance to synthetic exercises. Developing the tactical abilities of small subgroups, their adaptability and response capabilities to real game solicitations is one of these current trends (Czerwinski).

Thus, there is a need to better understand the game's structure and its interaction with the training process, in order to clarify their relation with the prevalence of injuries. We are still unable to quantify the real efficiency of the legal efforts to promote the safety of the players. In any case the picture of an extremely strong, rough and contact intensive game does not have the same meaning now as it had in the past, *e.g.* the Olympic Games (1972, Munich).

2.2.2 How to define the extent of the injury problem

Sports injuries are known to be one of the most common injuries nowadays (2001), which usually results in substantial medical costs, decreased performance and loss of practice and game time (Murphy *et al.*, 2003).

Epidemiology research about sport injuries is essential to understand the extent of the problem, besides supplying information about the injuries that most often occur and their consequences. This is a prerequisite for the development of appropriate preventive interventions (van Mechelen *et al.*, 1992).

Many researchers have studied the injury pattern in several different sports. Because of a lack of common methodology and different definitions, most of the results and conclusions produced are inconsistent and difficult to compare (Brooks & Fuller, 2006; van Mechelen *et al.*, 1992).

To address this lack of comparability, a few *hardcore* researchers have met and produced a Consensus statement, providing a common basis to improve the comparability of studies that adhere to it. An example of such statements, specific to football (soccer) injuries can be found in (Fuller *et al.*, 2006), being mainly focused on injury definitions and data collection procedures. They also suggest that this methodology be extended to other team sports. We eventually followed this suggestion.

The injury definition

When looking for the injury pattern or incidence in a given sport, the injury definition must firstly be clarified. In general *sportive injury* is a term used for a kind of physical damage sustained by practising sports (van Mechelen *et al.*, 1992). The differences in these definitions usually result from the injury recording process (*e.g.*, insurance files, hospitals or other medical sources). The criteria used to identify an injury may differ (Junge *et al.*, 2004):

- all physical complaints in spite of consequences - the *anatomical tissue injury* definition;
- injuries demanding medical intervention, typical of an insurance source;
- injuries leading to restrictions in sports practice, typical of a sporting source.

These differences are well documented in reviews of published studies about the influence of definition and data collection methods on the measured incidence of injuries (Brooks & Fuller, 2006; Junge & Dvorak, 2000; van Mechelen *et al.*, 1992).

Besides all these efforts, several difficulties and limitations are still unsolved. A frequent injury definition is based on time loss: the player missing at least one training or game (Junge & Dvorak, 2000). This definition misses all the injuries that recover before the next game or practice session, very common in athletes who don't have a daily practice routine (Junge & Dvorak, 2000). The authors also emphasise that it is possible to train or continue playing whilst injured, sometimes even supported by medication. In addition, some injuries do not necessary demand an absence from practice in specific sports (*e.g.* a football field player with a hand injury, but not a handball player).

The National Athletic Injury Registration System of the United States uses a limited athletic participation for at least one day as its criterion for injury report (Pelletier *et al.*, 1993). This definition is more accurate, but still does not include all previously cited difficulties.

In 1986 in Papendal, Holland, the European council established the following definition of sports injury: the injury should be sustained during a match or training session, *causing one or more of the following: reduction of activity, the need for treatment or medical advice, and/or social and economic consequences* (Schmidt-Olsen *et al.*, 1991), p. 373. This definition seems appropriate to sports, but it is not often adopted (Junge & Dvorak, 2000).

In the aforementioned Consensus Statement on Injury Definition in Studies of Football an injury is defined as any physical complaint acquired by a player resulting from a football match or training session, irrespective of the need for medical attention or time-loss from football activities. An injury that results in a player receiving medical attention is referred as a *medical-attention* injury and a *time-loss* injury is one that results in a player not being able to fully take part in future football training or match (Fuller *et al.*, 2006). However even with this precise definition there are some inconsistencies: the tolerance to pain and discomfort is different among players, generating different levels of complaints for the same problems (Brooks & Fuller, 2006). This definition may also exclude overuse injuries (Massada, 2001).

Incidence and severity of injury

The most common indicators of sports injury magnitude are injury incidence and severity (van Mechelen *et al.*, 1992). The injury incidence refers to the number of injuries per athlete exposure time (*e.g.* per 1000 hours of sports participation, per match, per tournament, *etc*.) and severity refers to the time of practice lost as a consequence of that injury (van Mechelen *et al.*, 1992). Prevalence is another popular measure and refers to the proportion of athletes injured at a given time (*e.g.* now we x athletes injured in a total of y, giving a prevalence of x/y). Risk and rate are also common words referring to the same concepts.

Note that for computing incidence, the difficulty is in accessing the exposure correctly (practice and game, including substitutions). Several other problems exist, like the incomparability of incidence per tournament with the one per hours of practice or the one per hours of play (Brooks & Fuller, 2006).

2.2.3 Studies about injury patterns in team handball

Several studies on the injury incidence in team handball have been done, mostly in Scandinavian countries and Germany. However, the geographic homogeneity didn't avoid that the studies used different conventions and became difficult to compare. As already stated for the injury incidence, it is well documented that results are influenced by the study design, injury definition used, time of recording and level of competition (Langevoort *et al.*, 2007). In some studies the information was obtained using retrospective questionnaires or interviews (Reckling *et al.*, 2003; Seil *et al.*, 1997; Wedderkopp *et al.*, 1997; Leidinger *et al.*, 1990), others recorded the injuries prospectively during a specific period of time (*e.g.*, tournaments, seasons) (Langevoort *et al.*, ; Seil *et al.*, 1998; Nielsen & Yde, 1988), and a few used video analysis (Oehlert *et al.*, 2004). Although there is a recognised importance of knowing the injury incidence of a given sport for developing specific prevention methods, in handball the approaches to prevention are still rare (Olsen *et al.*, 2005; Wedderkopp *et al.*, 1999).

Due to significant differences between the available studies, it is quite difficult to establish a unified view on the injury pattern in handball, particularly if discrimination by specific age groups, sex, level of competition, playing position (*e.g.*, goalkeeper, pivot) and game characteristics is needed. Table 2.3 summarises the injury incidences from the reviewed studies and table 2.4 presents the injury types and locations. For the articles originally published in German (Oehlert *et al.*, 2004; Reckling *et al.*, 2003; Leidinger *et al.*, 1990) we used only the English abstract.

Table 2.3 Injury incidence in team handball studies

Authors	Sex	Competition Level	Country	No. of players or No. of matches	Methods	Study period	Injury definition	Injuries	Injury incidence		
									Total	Training	Match play
(Nielsen & Yde, 1988)	M/F	NA Youth and senior	Denmark	N= 221 F=112; M=109	Prospectively	1 season (1985-1986)	Time loss[a]	105	4.6	NA	11.4
(Leidinger *et al.*, 1990)	M	Senior	Germany	N= 286	Retrospective (interviews)	After 1981 (5 years)	Medical attention	540	NA	NA	NA
(Yde & Nielsen, 1990)	M/F	NA Youth	Denmark	N= 94 F=54; M=40	Prospectively (questionnaires and interviews)	1 season (1985-1986)	Time loss[a]	36	4.1	1.9	10
(Lindblad *et al.*, 1992)	M/F	NA	Denmark	N: NA	Prospectively (patients from a hospital)	1 year	Medical attention [c]	570	NA	NA	NA
(Loës, 1995)	M/F	NA Youth	Swiss	NA	Prospectively registered by the Military insurance	3 years (1987-1989)	Medical attention [c]	1423	0.76	NA	NA
(Wedderkopp *et al.*, 1997)	F	NA Youth (ages 16-18 years)	Denmark	N= 209 From 22 teams	Retrospective (questionnaire)	1 season (1994-1995)	Time loss [b]	211	3.4	NA	40.7
(Seil *et al.*, 1998)	M	High non-professional Level	Germany	N= 186 From 16 teams	Prospectively (questionnaires and interviews)	1 season	Time loss [a]	91	2.5	0.6	14.3
(Massada, 2001)	M	NA Youth (ages 16-18 years)	Portugal	N= 362	Retrospective (clinic evaluation)	Athlete's career	All injuries	276	2.6	NA	NA
(Reckling *et al.*, 2003)	M/F	NA Youth	Germany	N=100 F=50; M=50	Retrospective (questionnaire)	NA	NA	130	NA	NA	NA
(Oehlert *et al.*, 2004)	M	Olympic	Participating countries	NA	Video analysis	Olympic male handball tournaments	NA	59	NA	NA	NA
(Junge *et al.*, 2006)	M/F	Olympic	Participating countries	NA 72 matches	Injury report system	Olympic Games 2004	Medical attention [c]	114	NA	NA	NA 1.6 /match
(Langevoort *et al.*, 2007)	M/F	Top level	Participating countries	NA 365 matches	Injury report system	World cups (M:2001 and F:2003); Women's EHC 2002, and Olympic Games 2004	Medical attention [c]	478	NA	NA	108 1.5/ match

NA: Not available.
Injury defined by causing the player to miss at least one training session or match [a], or disabling practice without considerable discomfort [b].
Injury defined by any physical complaint occurred in a match that received medical attention despite the consequences [c].
The injury sustained during a match or training session causing: reduction of activity, the need for medical attention, and/or social and economic consequences [d].

Table 2.4 Summary of injury type and location in team handball studies

Authors	(Nielsen & Yde, 1988)	(Yde & Nielsen, 1990)	(Lindblad *et al.*, 1992)	(Loës, 1995)	(Wedderkopp *et al.*, 1997)	(Seil *et al.*, 1998)	(Massada, 2001)	(Junge *et al.*, 2006)	(Langevoort *et al.*, 2007)
Year	1984-1985	1985-1986	NA	1987-1989	1994-1995	1995-1996	Until 2000	2004	2001-2004
Study period	1 season	1 season	1 Year	3 years	1 season	1 season	1 season	Olympic Games	6 International tournaments
Sex	M/F	M/F	M/F	M/F	F	M	M	F/M	F/M
Level/competition	Youth/senior	Youth	NA	Youth	Youth	Senior	Youth	Senior	Senior
No of players/matches	221 players	94 players	NA	NA	209 players	186 players	362 players	77 matches	365 matches
Injuries studied	Time loss [a]	Time loss [a]	Medical attention [c]	Medical attention [c]	Time loss [b]	Time loss [a]	All injuries [d]	Medical attention [c]	Medical attention [c]
No of injuries	105	36	570	1423	211	91	276	114	478
Injury location									
Head & neck	-	-	-	-	-	*4 (4.4%)*	-	*38 (34%)*	*124 (26%)*
Upper limbs	*43 (41%)*	*11 (30%)*	-	-	*42 (32.6%)*	*36 (39.6%)*	*119 (43.3%)*	*17 (15%)*	*85 (18%)*
Trunk	-	-	-	-	-	*2 (2.2%)*	*10 (3.5%)*	*14 (13%)*	*68 (14%)*
Lower limbs	*53 (51%)*	*25 (70%)*	-	-	*87 (67.4%)*	*49 (53.8%)*	*147 (53.2%)*	*43 (38%)*	*197 (42%)*
- Hip/groin (including thigh)	-	-	-	-	-	5 (5.5%)	-	3 (2.6%)	12 (2.5%)
- Thigh	8 (8%)	2 (5%)	-	-	-	-	-	6 (5.3%)	36 (8%)
. Knee	10 (10%)	5 (14%)	-	-	31 (24%)	18 (19.8%)	-	15 (13.2%)	62 (13%)
- Lower leg	-	-	-	-	-	9 (9.9%)	-	6 (5.3%)	26 (5.4%)
- Ankle	35 (33%)	14 (40%)	-	-	56 (43.4%)	14 (15.4%)	-	12 (10.5%)	50 (11%)
- Foot/toe	-	2 (6%)	-	-	-	3 (3.3)	-	1 (0.9%)	11 (2.3%)
Other/not specified	*9 (9%)*	*2 (5%)*	-	-	-	-	-	-	*4 (0.8%)*
Type of injury									
Fractures & bone stress	*4 (4%)*	-	*67 (12%)*	*147 (10.3%)*	*9 (4.3)*	*9 (10%)*	*17 (6.2%)*	*3 (2.6%)*	*5 (1%)*
Joint (non-bone) & ligament	*64 (61%)*	-	*351 (62%)*	*861 (60.5%)*	*134 (63.5%)*	*47 (51.5%)*	*176 (63.8%)*	*15 (13.1%)*	*85 (19.3%)*
- Dislocation/subluxation	-	-	-	43 (3%)	1 (0.5)	5 (5.5%)	-	3 (2.6%)	9 (1.9%)
- Sprain/ligament	64 (61%)	-	351 (62%)	818 (57.5%)	133 (63%)	42 (46%)	-	12 (10.5%)	74 (17%)
- Meniscus & cartilage	-	-	-	-	-	-	-	-	2 (0.4)
Muscle & tendon	-	-	*21 (4%)*	-	-	*24 (26%)*	*6 (2.2%)*	*9 (7.9%)*	*33 (6.2%)*
-Muscle rupture/tear/strain	-	-	-	-	25 (11.9%)	24 (26%)	6 (2.2%)	9 (7.9%)	32 (6%)
- Tendon injury/rupture	-	-	-	-	16 (7.6%)	-	-	-	1 (0.2)
Contusion/bruise	*11 (10%)*	-	*127 (22%)*	*253 (17.8%)*	*22 (10.4%)*	*6 (6.6%)*	*23 (8.3%)*	*58 (50.9%)*	*257 (53.8%)*
Laceration & skin lesion	-	-	-	*58 (3%)*	-	-	-	*3 (2.6%)*	*19 (4%)*
CNS/PNS	-	-	-	-	*5 (2.4%)*	-	-	*2 (1.6%)*	*12 (2.5%)*
- Concussion	-	-	-	-	4 (1.9%)	-	-	2 (1.6%)	12 (2.5%)
- Nerve injury	-	-	-	-	1 (0.5)	-	-	-	-
Other	*26 (25%)*	-	*4 (0.8%)*	*104 (7.3%)*	-	*(5.5%)*	*54 (19.6%)*	*14 (12.3%)*	*42 (8.8%)*
- Other injuries	7 (7%)	-	4 (0.8%)	104 (7.3%)	-	5 (5.5%)	-	10 (8.8%)	-
- Overuse unspecified	19 (18%)	-	-	-	-	-	54 (19.6%)	-	-
- Missing	-	-	-	-	-	-	-	4 (3.5%)	12 (2.5%)

NA: Not available.
Injury defined by causing the player to miss at least one training session or match [a], or disabling practice without considerable discomfort [b].
Injury defined by any physical complaint occurred in a match that received medical attention despite the consequences [c].
The injury sustained during a match or training session causing: reduction of activity, the need for medical attention, and/or social and economic consequences [d]

For the purposes of this approach, we decided to review briefly the studies one by one in their most important aspects.

Nielsen and Yde (1988) observed that ankle sprains were the most frequent injuries (33%) in Danish players. Upper extremity was involved in 41% of the injuries, being 21% finger sprains, which were mostly justified by errors whilst receiving the ball. The overuse injuries represent 18% of all injuries. The physical contact with opponent while running or shooting was the main cause for injury, occurring in 31% of the cases. The risk of a recurrent injury was computed in 32%. In 73% of the injuries the player had to stop handball practice for more than 1 week.

Leidinger *et al.* (1990) in a paper published before the rule changes of 1995, analysed a 5 year data stream of German teams and concluded on the importance of some rule changes to improve physical protection of players by restricting contact.

Yde and Nielsen (1990) compared injuries in adolescent players of soccer, handball and basketball. After analysing all the injuries indistinctly, the results showed that finger sprains (32%) and ankle sprains (25%) were the most prevalent injuries, with smaller records for other pathologies such as tendinitis/apohysitis (12%) and strains in the thigh and leg (10%). The most serious injuries were noticed in soccer.

Lindblad *et al.* (1992) reported all the handball injuries they examined in a hospital facility during one year. They report the incidences relative to 10 000 inhabitants per year, and in those units they found an incidence 61/10 000 inhabitants in women and 31/10 000 inhabitants in men. The most frequent injuries were ligament sprains and tears (62%) and fractures (12%).

We found only one study using a (big) database from an insurance company on acute injuries in young athletes (age 14-20 years) from 32 different sports (Loës, 1995). The handball presented one of the highest injury incidences in male athletes and the highest in female athletes. The injury incidence per 10 000h of practice for male (7.2) and female (7.6) handball players was similar. Female players presented 65% of sprains and male players 55% although, again, the injury incidence for sprains is similar (4 and 5).

The injury pattern of young Danish handball players (16-18 years old) was examined at the end of 1994-95 season (Wedderkopp *et al.*, 1997). In this study the incidence in matches is higher than in any other report we know (40.5 injuries/1000hours of game), possibly as a result of an inclusive injury definition. The results showed that back and line players presented the highest injury incidences (54.8 and 54.4/1000 hours of game, respectively) and the goalkeepers and wings the lowest incidence during matches (30.6 and 23.6/1000 hours of game, respectively). In contrast, the injury incidence during training was similar between all the field positions, whereas the goalkeepers' incidence was twice as high.

The study of Seil *et al* (1998) was conducted around the time of some major rule changes. They found a superior injury incidence in matches (77%) than in training. The lower extremity was involved in 54% of injuries and the upper extremity in 40%. Some specific types of injuries were mainly observed in specific players' positions (or functions), being the case of elbow injuries in goalkeepers and shoulder injuries in throwers. The knee was the most affected joint and in the most severe way, with the highest proportion in high-level players. The finger, ankle and shoulder were also often injured. The external mechanism of injury reveals a high risk in offensive actions with 1/3 of these in counterattacks. In contrast to other studies, the wing players showed the highest rate of severe injuries. The authors also concluded that the high injury incidence in games endorses a need to change the rules of the game.

Massada[4] (2001) compared 1859 young basketball, handball, soccer and volleyball players (ages 12-17 years old) - 362 of these being handball players - and found an average incidence of 2.6 injuries/1000 hours of handball playing. In addition, the highest incidence of injuries in handball was noticed in the group of players with 14 years of age. The most prevalent were joint (63%) and overuse injuries (19.6%).

Another study was developed in Germany by Reckling *et al.* (2003) on juvenile handball players from both sexes. In this study, the injuries were more prevalent in game (69%) than in training and most of them in the offensive phase. The most injured players were backcourt players (29%), line players (27.5%) and goalkeepers (17%). Interestingly, the female goalkeepers were three times more often injured than male keepers of the same age. The injuries occurred at higher proportions in foot and ankle (32%), finger joint (26.9%) and knee (24.6%). The most severe injuries were observed in the knee and, among these, the most often one was the ACL rupture. Attempting to identify the typical game situation when injuries occurred, the authors reported the ball catching (38.5%), contact situations with the opponent (34.5%) and landing from a jump (26%).

In the study of Oehlert *et al.* (2004) 59 injuries were analysed, suffered by male players during the 1992 Olympic Games, based on video analyses by two experts. The anatomic localization of injuries was computed: head (20), lower limb (15), thorax and abdomen (9) and the upper limb (9). The injuries mostly occurred in midfield (20), and in the offensive phase (84%). Also 86% of injuries occurred with contact. In conclusion, for decreasing the

[4] the pioneer in the quantification of injury prevalence in many team and individual sports in Portugal

number of contact injuries the authors advised referees to enforce a closer conformity to the rules of the game.

Junge *et al.* (2006) compared the injury risk between Olympic team sports, using an injury-reporting system. Some questions of the injury report form were adapted to each sport team because of technical differences. The physician or the official medical representative was informed about all the procedures to report injuries after each match. The results confirmed that the risk of injury is different between the sports analysed, with handball and football presenting the highest incidence of injuries. In general, the incidence of injuries, localization and its circumstances were similar between sexes; with a slightly higher incidence in men, as well as a slightly higher time-loss per 1000 matches played. Substantial differences were noticed between the type of injuries with more concussions and sprains in females and a higher rate of fractures and lacerations in males.

More recently, Langevoort *et al.* (2007) analysed the injuries reported by the physicians of participating teams in several international handball tournaments The teams were asked to report injuries after each match on a standardized form. The average injury incidence obtained was the highest of all studies aforementioned. The injuries affected mostly the lower limb (42%), the head (23%), upper extremity (18%) and trunk (14%). The pattern of injury incidence was similar in all tournaments and slightly higher in the second half of match. There was no significant difference between incidence of injuries in male and female (112 *versus* 103 injuries/1000 player-hours), nor the location or type of injuries varied significantly, although women presented more non-contact injuries than men (20% *versus* 12%) did. Concluding that the majority of injuries were mainly related to contact with the opposition, the authors suggested fair play as essential for a safer handball game.

2.3 Anterior Cruciate Ligament

2.3.1 Functional anatomy of the ACL and considerations

The ACL is a central knee ligament (figure 2.1), running from the posterior portion of the femur (internal surface of the lateral condyle) to the anterior portion of the tibia (located laterally to the medial tibial spine) with a slight inwards deviation. Its function is to restrain the anterior displacement of the leg relatively to the thigh and the internal rotation of the leg. Assists also in stabilizing several other degrees-of-freedom of the knee.

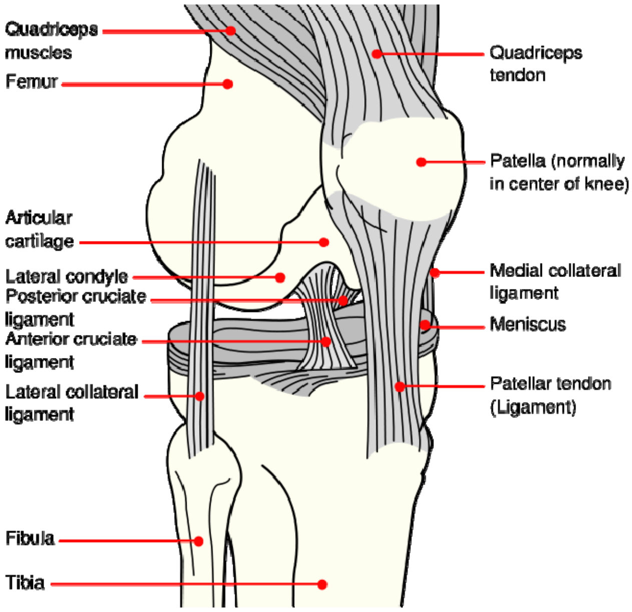


Figure 2.1 Knee joint outline (http://en.wikipedia.org/wiki/Image:Knee_diagram.svg)

The ACL is commonly described as a double-bundle structure, namely the anteromedial (AM) and posterolateral (PL) - see revisions by Petersen & Zantop (2007) or Steckel *et al.* (2007). Its function is complex, as it is challenging to understand the *in situ* forces in the bundles resulting from external loads at *out of plane* actions. The two bundles, that have different and specific bone attachments, perform different tasks: in knee flexion AM is the primary restrain to anterior tibia translation; with the knee near full extension the PL handle the rotational loads (Zantop *et al.*, 2007; Gabriel *et al.*, 2004). Although in this primary functions they also act as stabilizers in all the other degrees of freedom of the knee in a combined (synergistic) way (Zantop *et al.*, 2007). The different function of each bundle enables us to support the idea of a partial ACL or isolated ruptures of one of the bundles. The lack of consensus on this could be motivated by the difficulty in obtaining a precise diagnosis either by conventional magnetic resonance imaging or by arthroscopy (Petersen & Zantop, 2006).

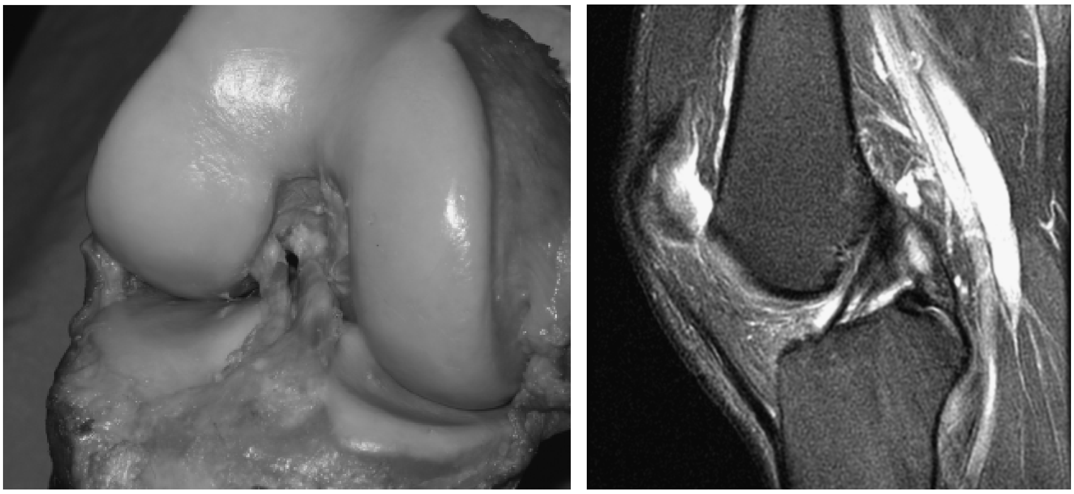


Figure 2.2 Anterior cruciate double structure of a cadaveric knee (left) and MRI focusing on the anteromedial bundle (right) (Steckel *et al.*, 2007), p. 100-101

The role of the muscles

The muscle-tendon complexes acting on the knee joint can work as active stabilizers and offload partially the joint ligaments (Solomonow & Krogsgaard, 2001). The relevant muscular parameters are their speed (usually measured in *time-to-peak-force*) and strength (maximal force or torque).

In the case of the ACL, the main active stabilizers for the anterior displacement of the leg relatively to the thigh are the hamstrings and the *triceps suræ*. The main active stabilizers against the internal rotation of the leg are the pelvitrochanterian, iliopsoas, *gluteus* (the posterior fascicles of all 3 portions), and the *pectinius* muscles (Besier *et al.*, 2001). One usually simplifies this a little and measures the isokinetic knee flexion torques as representative of this muscular stabilizing capacity.

As an indication[5] of the loads these muscles and muscular groups must dissipate, the force of the opposing groups[6] (the knee extensors) is measured and one ends up with a ratio called hamstrings to quadriceps torque ratio – H:Q. Over the years many have published values of *normal* or desirable H:Q ratios, usually in the range 1:2 to 2:3 (see chapter 4).

Are the synthetic muscle strength measurements realistic?

A muscle can work (produce force) while shortening (concentric), lengthening (eccentric) or not changing its length (isometric). The force produced depends of this shortening or lengthening speed (and the recruitment involved).

[5] Note how arbitrary this association is
[6] which have a negative role in anteroposterior ACL protection

For reproducibility, the H:Q is usually measured at an arbitrary constant knee angular velocity, either positive or negative (concentric or eccentric), with an arbitrary fixed hip angle. Several papers have debated if a ratio of pure concentric actions H_{con}:Q_{con} is preferable over one of pure eccentric ones H_{ecc}:Q_{ecc} or the other combinations H_{ecc}:Q_{con} and H_{con}:Q_{exc} (P. Aagaard *et al.*, 1998; Dvir *et al.*, 1989).

A real handball movement however, like a jump and drop, involves the hip, the knee and the ankle joints simultaneously (McLean *et al.*, 2005).

For instance, in a landing from a jump, a change in the knee angle from 0º to 30º is usually accompanied by a change in the hip joint angle from 0º to 45º (slight forward trunk flexion). The result is a lengthening of the *vastus medialis* by about 9% (eccentric) and a slight shortening of the *rectus femoris* by less than 1% (concentric) (Conceição, 2004). One can not say that the *quadriceps* group, which includes both the *vastus medialis* and the *rectus femoris* is working neither concentrically, isometrically or eccentrically. But in an isokinetic machine one can only subject the quadriceps to one of these kinds of movements. Additionally, the hamstrings (that don't suffer from this problem), can work in the same manner as the vastus, or the rectus, or both, or neither. An example (figure 2.3) could be a *bad landing*, with the hip flexing only 15º and the knee flexing 30º resulting in *all* referred muscle groups working eccentrically (Conceição, 2004)

So there is no biomechanical criteria to prefer H_{con}:Q_{con} over H_{ecc}:Q_{con} or H_{con}:Q_{ecc} or H_{ecc}:Q_{ecc} as all of them are biomechanically irrealistic. Additionally, from this perspective one cannot prefer a particular hip angle for the isokinetic tests. They all seem irrealistic, being static.

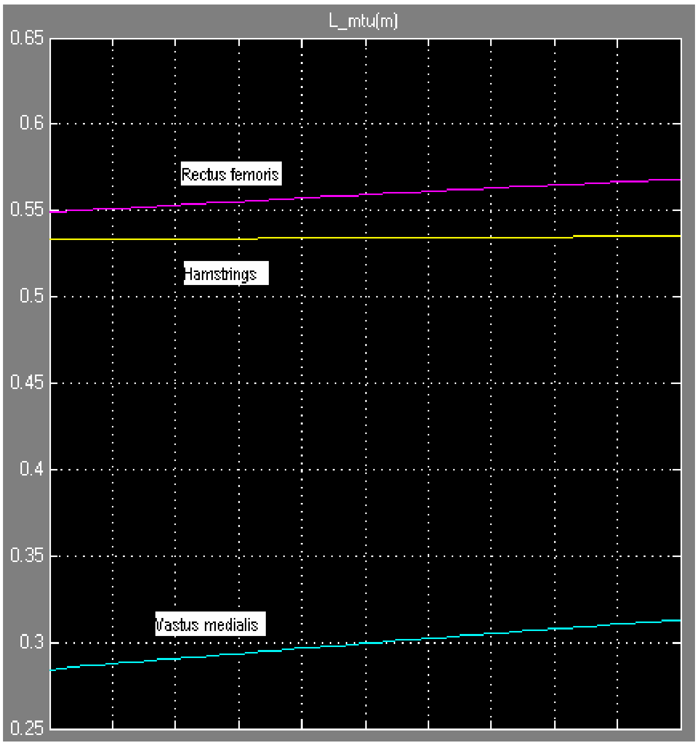


Figure 2.3 Contractile element length *versus* knee angle – parameters from Conceição (2004)

High speed movements

At high articular speeds (high angular velocities), like those found during normal handball practice, there is another important effect: the tendons stretch and shorten significantly and so a particular musculo-tendinous complex can be lengthening while its fascicles could be shortening (or vice-versa) and this can be different for different muscles acting on the same joint at the same time (Sousa *et al.*, 2007). This cannot be reproduced in an isokinetic device, where the dynamics of the tendon is negligible (almost constant force – almost constant length).

So in fact, one cannot even apply the concepts of concentric, isometric or eccentric contractions and much less those of agonists and antagonists in complex movements where biarticular muscles are involved.

2.3.2 The extent of the ACL injury problem

Knee ligament injuries are common in the general population, involving primarily (≅90%) the ACL and MCL (Woo *et al.*, 2006). In sporting activities, the risk of knee injury is increased, with its type linked to the specificity of each sport (Majewski *et al.*, 2006). The true knee injury type incidence is difficult to determine and requires to prospectively follow a large number of athletes over a long period of time. There is a lack of studies concerning this problem (Dallalana *et al.*, 2007).

The MCL injury is thought to be the most frequent acute knee injury, but the ACL injury is often the most severe. The MCL can heal spontaneously and even a complete rupture responds well to conservative treatment (Woo *et al.*, 2006). The ACL has poor healing capacity and a retraction behavior has been found after rupture (Murray *et al.*, 2000). Additionally the rupture occurs mostly at the ligament-to-bone insertion area (Mutsuzaki *et al.*, 2007). Because of its structure, properties and mainly its function, this injury is potentially very debilitating in high demanding sports, involving jumps, landings, pivoting and plan and cut movements. The consequences - high economic costs and extensive time of practice lost - being of particular concern in developed countries.

Surgical reconstruction is often required in athletes mainly to prevent knee instability and premature development of osteoarthritis (Kessler *et al.*, 2008). However some studies have provided evidence that surgery does not prevent increased tibial rotation (Ristanis *et al.*, 2006; Ristanis *et al.*, 2005) and gonarthrosis development (Myklebust *et al.*, 2003b). Repetitive anterior tibial subluxations[7] in ACL-deficient knees seems to induce intra-articular damage, including articular cartilage, meniscus, joint capsule and ligamentous restraints (Marks *et al.*, 2007). To fully evaluate the extent and aetiology of this problem (gonarthrosis), more long-term prospective studies are needed, involving not just athletes with ACL injuries but also with other types of knee pathologies and even with intact knees. We were unable to find studies comparing long-term effects of practice with different knee loads (different sports, different playing positions, and different anatomic characteristics) on the knee, and its relation with sexes.

Another common belief is that not all athletes will be able to fully recover the knee joint function and return to competition at preinjury levels, precipitating the end of a career for some. There is a scarcity of studies investigating the rate of return after injury, the involved treatments and the specific sport training applied.

Myklebust *et al.* (2003b) investigated elite handball players on average 7.8 years after an ACL injury. They found a higher rate of return, at their preinjury levels, in the non-ACL reconstructed group (82%) in comparison to the surgically treated group (58%). This data is difficult to interpret because the reasons for each of the selected treatments are unknown. Twenty two percent of injured players, after returning to play handball, had an ACL reinjury during handball practice. The authors suggested a more cautious attitude towards the returning to sports practice after an ACL injury in high *knee demanding* sports, such as handball. It's also suggested that improvements in ACL reconstructions, may partially explain the increased risk of future osteoarthritis by enabling more players to return to competition with non-intact knees (Myklebust *et al.*, 2003b).

[7] Recurrent giving-away episodes, after an acute ACL injury, during weight bearing activities

A disproportionate rate of ACL injuries between sexes is frequently underlined, however there are few studies comparing the ACL injury risk of male *versus* female in several different sports (handball and other team collision sports, basketball, soccer and skiing). A compilation of these rare results is in Marshall *et al.* (2007). Results are dependent on sport, methodology and sample size but a few common trends are easily identifiable. In all cases, the injury rate for men is no higher than for women. The ratio of female risk to male risk is generally between 2 and 3 (again, sport dependent). There are some studies with results very different than this (as low as 1.0 or as high as 9.5) but they were done with very small sample sizes and the confidence intervals reveal this appropriately (and always including the 2 to 3 times risk ratio from above).

Another critical point of analysis is the age of injury. Female players are mostly affected between 14 and 19 years of age (Renstrom *et al.*, 2008). Figure 2.4 presents the distribution of ACL ligament reconstruction by age for both sexes in Norway.

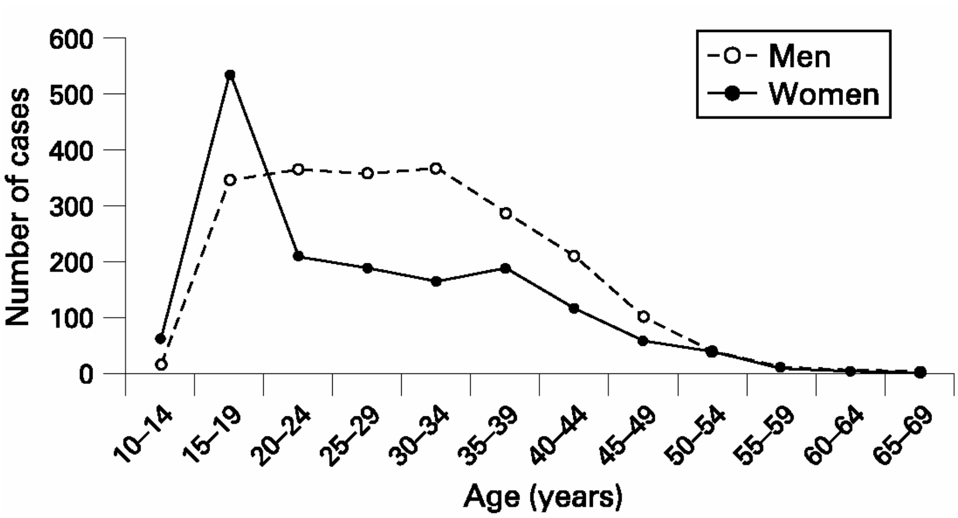


Figure 2.4 Distribution of patients in the Norwegian National Knee Ligament Registry by age and sex (Renstrom *et al.*, 2008), p. 395.

The seriousness of the ACL injury stresses the need to improve the management of its risks and causes - prevention should be the main goal. To design effective preventive programs and strategies, the understanding of its causes and mechanisms is essential. Van Mechelen *et al.* (1992) proposed a sequence of four steps for the design of effective injury prevention measures (figure 2.5). Sometimes it is proposed, as an alternative to the epidemiological step four, a simple randomised controlled trial (Bahr & Krosshaug, 2005).

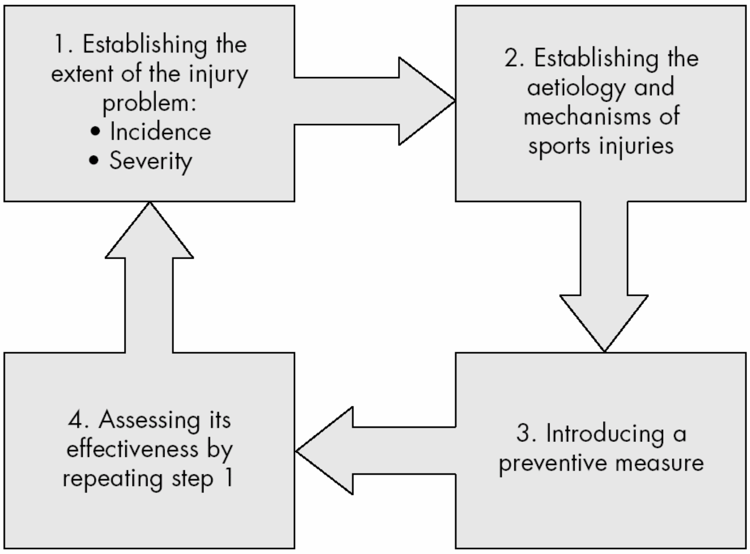


Figure 2.5 Design of an injury prevention strategy (van Mechelen *et al.*, 1992)

2.3.3 Causes of ACL Injury

Several models for the injury causes have been proposed, both from the epidemiological and the biomechanical perspectives (Meeuwisse *et al.*, 2007; Bahr & Krosshaug, 2005; McIntosh, 2005; Meeuwisse, 1994).

In a more biomechanical perspective, a traumatic injury (and several overuse ones) are caused by an energy transfer to some tissue that exceeds its handling capabilities (Baker *et al.*, 1992), cited by (Meeuwisse *et al.*, 2007). This mechanism is always associated with a structural (or histological) damage, although several other processes may also develop concurrently (*e.g.* changes in chemical homeostasis, *etc.*). The mechanical properties of the ACL, such as its stiffness and ultimate load, represent the tissue capacity to handle the load. It has been suggested that female sex hormones may change this capacity, contributing to a higher level of predisposition to injury in female athletes. Additionally the response of the ACL tissue is also dependent on the nature of the load, namely its type (torsion, traction, *etc.*), velocity, *etc.* Note that female long and triple jumpers do not sustain frequent ACL injuries although the loads are high and *fast*.

It would be interesting to know if an ACL injury is the outcome of an accumulation of a large number of very small, undetected, damages, causing the tissue to finally fail at a load that it would, otherwise, handle without problems; or if it is just the natural result of the exposure to an unusual and excessive load.

A more complex, multifactorial, approach to injuries has been proposed by Meeuwisse (1994) in a model including the interaction of several intrinsic and extrinsic risk factors, concurring to make an athlete susceptible to injury. Then, under this model, the final *inciting event* is the one actually causing the injury. This model was criticized because of its excessive simplicity. In it the injury appears as the ending point of a linear sequence of events and concurring risks (Gissane *et al.*, 2001). In real sports practice, the beginning point (if such is possible to determine) is not necessarily a single aspect, characteristic or event. More recently, Meeuwisse *et al.* (2007) presented a revised model (figure 2.6) emphasizing the potential changes in susceptibility to injury as a consequence of exposure to practice, which may lead either to adaptation or to increased susceptibility.

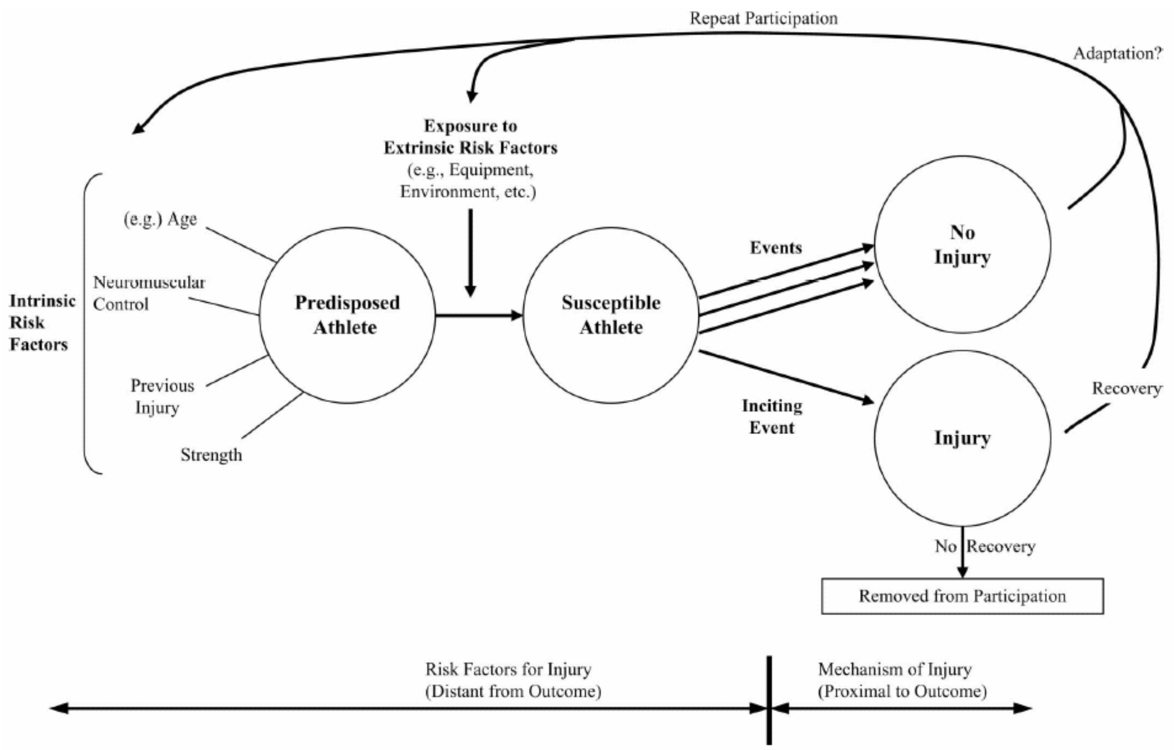


Figure 2.6 A dynamical, recursive model of aetiology of sport injury (Meeuwisse *et al.*, 2007), p. 217

Note that these two approaches (biomechanical and epidemiological) use the term *mechanism of injury* with two very different meanings. For this reason we will use the term *internal mechanism* in the sense of the biomechanics and prefer the expression *external mechanism* or in some cases *context of injury* to describe the sportive situation.

In accordance to the multifactorial approach to the injury causes, the development of preventive efforts based solely on the biomechanical description of an injury mechanism is limited and must be interpreted in the context of a specific sport. An example, in the case of team sports, is the need to describe fully the playing position and situation, to understand the *inciting event*. These considerations lead to another model (figure 2.7) by Bahr and Krosshaug (2005). This one includes the role of external factors in a more team sport oriented way. External factors may include the competitions' structure, the coaching and game model, the playing environment, the referees' interpretation of the rules, and so on.

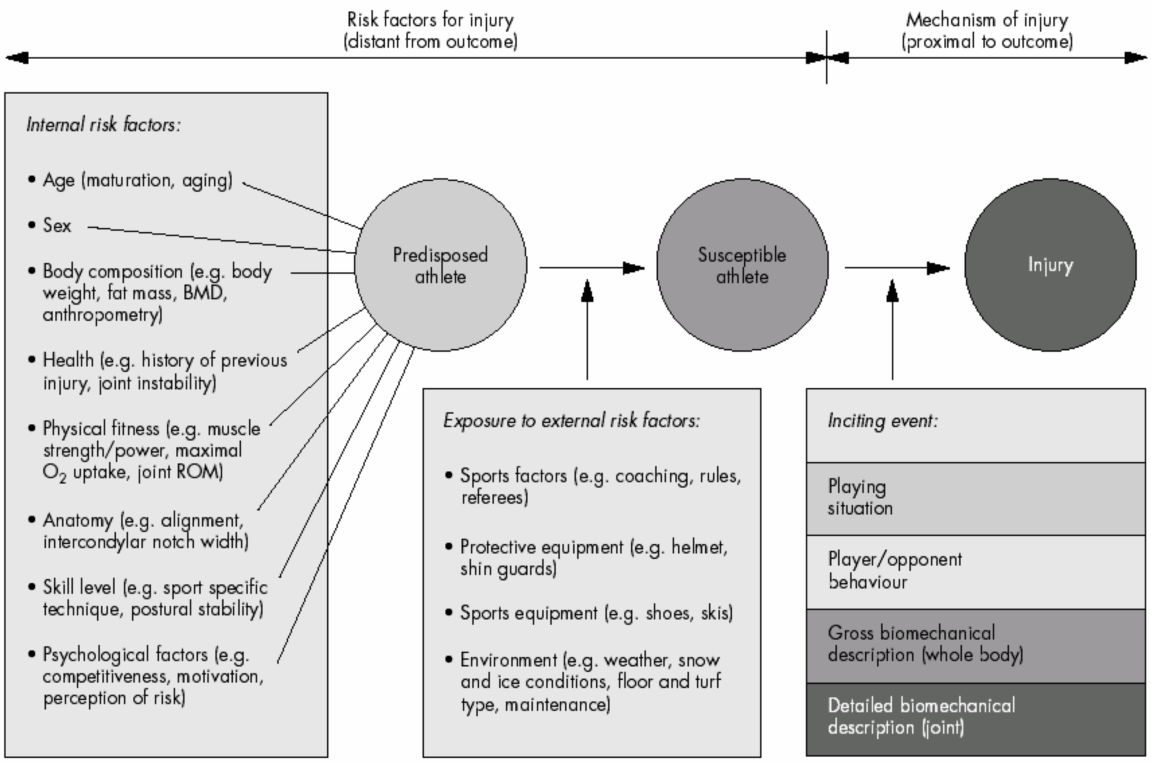


Figure 2.7 Comprehensive model for injury causation (Bahr & Krosshaug, 2005), p. 327

Several different approaches to the study of sport injuries have taken place, ranging from self-reported injury mechanism to clinical studies, including cadaveric studies, laboratory motion analysis, mathematical modelling and simulation of injury situations, *in vivo* studies (*in situ* measurements of strain) and video analysis of real injury situations, all reviewed in (Krosshaug *et al.*, 2005).

There is a need for more detailed information about the sport specific injury situation, because an ACL sustained during landing, for example, is more than likely constrained by the specificity of each sport (*e.g.* with or without contact). From our point of view, this type of description is critical to coaches and for motivating the adoption of more sport specific preventive measures.

Description of ACL Injuries – external mechanism

The injury description in the real sportive context has been limited to self-reports or video motion analyses.

Epidemiological studies have divided ACL injuries in two categories: contact and noncontact. In handball, the proportion of noncontact ACL injuries affecting female players was found to be as high as 89% (Myklebust *et al.*, 1998). This has drawn the researchers attention, as these figures were obtained mainly from invasion sports where contact is a natural part of the game. However, in a recent review (Marshall *et al.*, 2007), only 11 (from 39) studies addressed this issue and only 4 presented data comparing both sexes. In soccer, the proportion of non-contact injuries was 58% in females and 50% in males, and in basketball it ranged between 75%-81% in females and 69%-70% in males. The comparison of these figures is, however, limited by the methodological differences between the studies.

Boden *et al.* (2000) analyzed 100 ACL injuries in both sexes and from several sports, including american football, basketball and soccer. Based on self-reported circumstances of injury (by a questionnaire), the majority were classified as non-contact (72%), occurring at foot strike with knee close to full extension. The noncontact mechanism was associated to a sudden deceleration prior to a change in direction or landing and a contact injury was associated to a valgus collapse of the knee. The authors also reviewed 27 of those injuries in videotapes and confirmed these findings.

Myklebust *et al.* (1997) studied 93 cruciate ligament injuries in handball (87 in the ACL and 6 in the PCL). The female players were the most affected ones, sustaining 59 of the injuries (63%)[8]. Seventy-five percent of the injuries were sustained in a match situation (90% of these in the attack phase). The most affected players were from the back positions (first offensive line) (54/93) and wings (30/93); the line players (5/93) and goalkeepers (11/93) were less affected. There was no contact with another player in 95% of the cases, but in 78% of them the players were in contact with the ball. Cutting and faking movements were associated with 55% of the cases and landing from jump to 30%. Fifty-three percent of the players reported to be moving at high speed when injured, 33% at slow speed and 14% standing still.

Myklebust *et al.* (1998) investigated 28 ACL injuries sustained by elite handball players during three seasons (23 from women and 5 from men). The circumstances were obtained from the players' self-reports. Most of the injuries occurred during competition (24/28), in an attacking situation, while handling the ball (26/28), and at high speed (17/28). There was no contact with another player in most of the cases (23/28) and they occurred while performing faking movements, *e.g.* plant-and-cut (19/23), or landing from a jump (4/23). More detailed and precise information about the knee motion was difficult to obtain, although both internal and external tibial rotation was identified in some cases.

[8] On average, 1.8% of female players were injured compared with 1% of male players.

In a prospective study over three seasons, Myklebust *et al.* (2003a) found 69 ACL injuries in female handball players (13 in the elite division). They analyzed both injuries sustained in the control season (first year) and during the following two seasons of application of an ACL injury[9] prevention program. The authors found that 58/69 of the injuries occurred while attacking, and (10/69) in defensive actions. Thirty-three (33/69) injuries were classified as contact and 35/69 as noncontact. Fifty-one (51/69) injuries were sustained when handling the ball. The most affected players were back players (39/69) and the wing players (19/69) while the line players (4/69) and goalkeepers (5/69) were less affected.

Ebstrup and Bjsen-Møller (2000) analyzed three video-recorded ACL injuries. The methodology is poorly described. The authors reported varus knee loaded by femoral external rotation or valgus loaded knees by femoral internal rotation at the time of injury. The authors suggest that the knee valgus/varus should be avoided while performing side-stepping or sudden changes in speed.

Olsen *et al.* (2004) analyzed 20 videotapes of ACL injuries sustained by female Norwegian handball players (16 from international competitions or elite division). The images were enlarged and digitized prior to examination. The knee positions and playing situations were analyzed by experts (coaches and physicians). Equal numbers of injures by right/left knee were analyzed. All injures occurred while attacking except one (in the defensive phase – a direct blow to the leg by an opponent), with 6/20 during the counter-attack (fast break). Almost all injuries (19/20) occurred actually in the back position (left, middle, or right), even if the usual playing position of the injured player was different (an often forgotten difference). All players were handling the ball (19/20) and focusing directly the opponent or the goal. In 6/20 on the cases, opponents pushed or hold the torso of the player when the injury was sustained. Seven players were classified has being out of balance, and 12/20 showed some kind of motion perturbation. The injuries occurred mostly during plant-and-cut movements (12/20, figure 2.8) or one-leg landing from a jump shot (4/20, figure 2.9). During the plan-and-cut movements, the injuries seem to occur at the push-off knee while accelerating in another direction (towards the medial side of the knee, with one exception), with the foot firmly fixed on the floor, and always out of the vertical projection of the hip. The knee was almost straight, in valgus, with internal or external tibial rotation. During landing, the knee was similarly in a slightly flexed, valgus and with an external rotation of the tibia.

Krosshaug *et al.* (2007) investigated 39 ACL injuries sustained by female (22) and male players (17 male) from different basketball playing levels. Experts to address the playing

[9] Twenty-nine injuries were sustained during the control season (942 followed players), 23 in the first intervention season (855 participating athletes) and 17 in the second intervention season (855 participating athletes).

situation, player's behavior, and joint kinematics analyzed the injury situations. There was contact in 11 of the cases: in males, 4 were direct blows to the knee; in females, which were mainly collisions or pushes by the opponent. The estimated knee flexion angle, on average, was higher in female than in male. The knee valgus collapse was more frequent in female than in male players.

Video analysis proved to be a useful tool to investigate the external ACL injury mechanism in team sports due to its non-invasive and ecological nature. However one must not forget the intrinsic limitations in accuracy (and possibly in reproducibility) of this methodology (Renstrom *et al.*, 2008).

Figure 2.8 The sequence events leading to an ACL injury: plant-and-cut movement (Olsen *et al.*, 2004), p. 1007

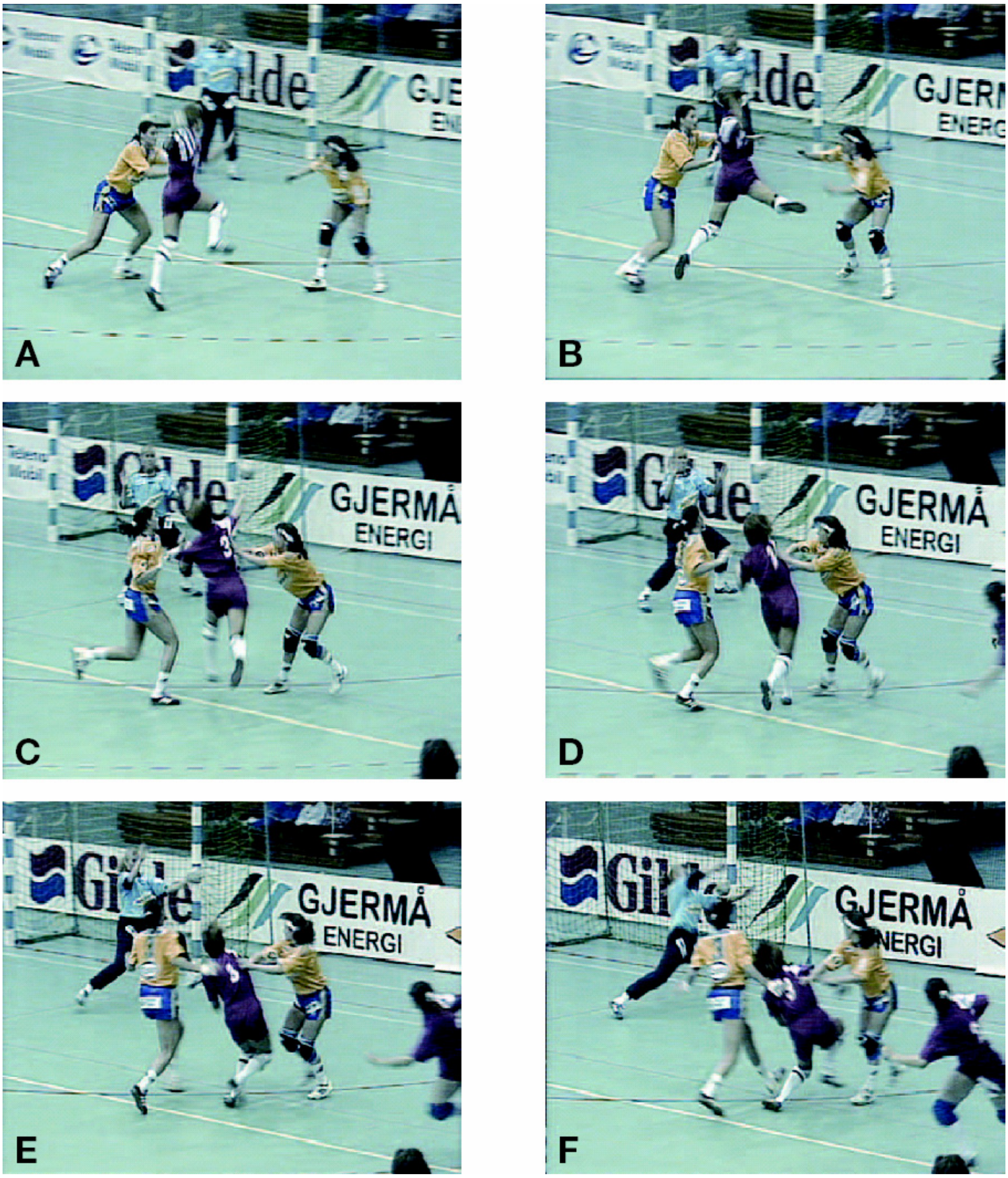


Figure 2.9 The sequence events leading to an ACL injury: landing from a jump (Olsen *et al.*, 2004), p. 108

2.3.4 Non-hormonal risk factors

The risk factors can be divided in internal and external factors. Internal are related to the subject (the athlete), including anatomical, hormonal and neuromuscular factors. External factors are the sport specificities (structural and implementational), including training, competition models, tactics and strategies, opponent skills and behaviour, but also environmental conditions, equipment used (*e.g.* shoes), *etc*. In reality this division is somehow synthetic[10] as the several factors interact continually in a complex way, possibly creating injury circumstances that cannot be attributed to any single factor.

In the case of ACL injuries in female athletes, emphasis has been placed in the internal risk factors as the external conditions are apparently the same for both sexes and the majority of the injuries are contact-free (however one must not forget that the training and game models are still differentiating factors between sexes).

2.3.4.1 Anatomical

Several studies have focused on anatomical risk factors. The subject is very specialised and we will just refer to the main topics based on a few review papers (Renstrom *et al.*, 2008; Griffin *et al.*, 2006; Hewett *et al.*, 2006b; B. Yu *et al.*, 2002):

- static alignment (Q-angle[11]/pelvis width)
- dynamic knee valgus
- decreased femoral notch width
- ACL geometry
- ACL mechanical properties
- posterior tibial slope
- increased general joint laxity
- increased muscle (hamstrings) flexibility
- increased anterior tibial translation and internal-external rotatory knee laxity
- increased foot pronation and navicular drop
- patellar tendon-tibia shaft angle
- body mass index (BMI)
- age and many others

A few studies describe, in detail, the anatomical differences between male and female and these findings may contribute to explain the sex-asymmetry in ACL injury rates (Park *et*

[10] As usual in science, these divisions are always artificial and exist for the benefit of the researcher, to make the reality manageable.
[11] *Quadriceps femoris* angle

al., 2008) or between injured and non injuried subjects (Stijak *et al.*, 2008; Lombardo *et al.*, 2005). Some others used a prospective design to scrutinize the relationship between some anatomic risk factors and the ACL injury risk (Uhorchak *et al.*, 2003). We will take a closer look at some of these possible risk factors.

The *Q-angle*[12] (figure 2.10) is one of the most obvious differences between sexes and is related with the wider pelvis in women, detectable from a young age (Griffin *et al.*, 2006). This misalignment creates a static valgus torque that apparently stresses the knee structures. However the static Q-angle has been found, by some, to be unrelated either to the dynamic valgus (Hewett *et al.*, 2006b; Pantano *et al.*, 2005) or to ACL injury risk (Hertel *et al.*, 2004). Pantano *et al.* (2005) found that the pelvic width to femoral length ratio (the tangent of the Q-angle) seem to be a better predictor of dynamic valgus than the Q-angle. If so, this probably means that this Q-angle effect is real but the data has insufficient statistical quality.

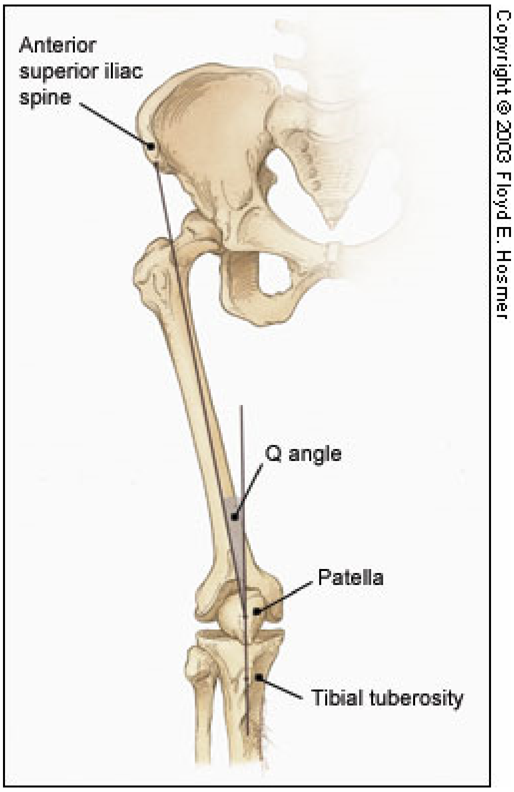


Figure 2.10 Q-angle

Notch size and *ACL geometry* have also been proposed as ACL risk factors (Renstrom *et al.*, 2008; Uhorchak *et al.*, 2003). A possible ACL impingement against the intercondylar notch as been proposed as an alternative mechanism to the rupture by direct loading

[12] angle between the long axis of the femur and the tibia (Myer *et al.*, 2005)

(Fung & Zhang, 2003; Arendt, 2001). In a 3-D mathematical model simulating physical interactions between the ACL and intercondylar notch, the authors stated that the ACL impingement may in fact occur during external rotation/adduction of the tibia relative to femur (Fung & Zhang, 2003). Therefore, this impingement may be induced during landing or cutting manoeuvres, which agrees with several actions already described as possible external mechanisms (Olsen *et al.*, 2004). There have been divergent conclusions on whether notch geometry should be viewed as sex-specific or not. Some authors have found that women with narrow interconylar notch are at a greater risk of ACL rupture than those with larger notch (Uhorchak *et al.*, 2003; Shelbourne *et al.*, 1998; Lund-Hanssen *et al.*, 1994) while others concluded that notch width (or a related undimensional parameter) is positively related to ACL rupture, regardless of sex or notch size (Ireland *et al.*, 2001). Others still (Lombardo *et al.*, 2005) have not found any relation between these two quantities (notch width and ACL injury risk). The ACL geometry has also been implicated in this problem a thinner ACL has a lower *load to failure*. However the different experimental methods used are probably implicated in the lack of quantitative consensus among the studies, as summarized in Griffin *et al.* (2006). Besides smaller ACLs and narrower intercondylar notches, other sex differences including the ACL ultrastructure (Hashemi *et al.*, 2008) and consequently lower tensile properties (Chandrashekar *et al.*, 2006) have also been proposed as risk factors.
Obviously one can always argue that women are, on average, lighter and slower than men, generating smaller loads on the locomotor apparatus. Ultimately, what is needed is the information on the (dis)adaptation of the ACL capabilities to the personal, dynamic, load.

Posterior tibial slope[13] has been implicated in the anterior tibial translation and therefore may play a role on ACL injury (Stijak *et al.*, 2008). These authors reported greater tibial slope of the lateral condoyles in subjects with ACL ruptures, than in the control group. The possible interpretation for such findings is that an increased tibial slope may lead to increased anterior tibial translation, which consequently may induce ligament stretching, particularly during hyperextension with internal rotation of the knee (Stijak *et al.*, 2008). In contrast, other authors compared patients who have sustained noncontact ACL injuries with controls and concluded that increased tibial slopes do not seem to be a risk factor to these injuries (Meister *et al.*, 1998). Anyway, no definite conclusion on the role of the tibial posterior slope in the ACL injury risk (or reinjury) has been obtained so far (Renstrom *et al.*, 2008).

[13] The tibial slope is defined by the angle between the perpendicular line of the tibial axis and the posterior inclination of the tibial plateau (Stijak *et al.*, 2008).

Generalized joint laxity and *hyperextension* have been suggested as other risk factors specific to females (Kramer *et al.*, 2007; Uhorchak *et al.*, 2003). While the later is just a consequence of the former, any misalignments of the knee introduce important torque and force imbalances and are plausible suspects.

The anterior *knee laxity* is frequently quoted as a potential ACL risk factor (Shultz *et al.*, 2007). Several studies have found a relationship between the increased anterior knee laxity and the risk of injury in female athletes (Uhorchak *et al.*, 2003; Trimble *et al.*, 2002; Woodford-Rogers *et al.*, 1994). Others have documented that female have higher anterior knee laxity than men (Shultz *et al.*, 2005; Rozzi *et al.*, 2001; Rosene & Fogarty, 1999) as well as higher *rotatory laxity* (Hsu *et al.*, 2006) which probably has implications in the female knee stability and knee's general health (Schmitz *et al.*, 2008).

As listed above there are many other anatomical risk factors. However, one needs to better understand the dynamical behaviour of these anatomical factors, including their interaction with the neuromuscular system, in order to appropriately quantify their relative importance.

2.3.4.2 Neuromuscular

Many authors have suggested that inadequate neuromuscular control of the lower limb may be a primary contributor to the female ACL injury mechanism (McLean *et al.*, 2004; Hewett, 2000). To explore this hypothesis many investigators have studied movement patterns, proprioception, muscle activation and stiffness, as well as fatigue.

Movement patterns. A productive line of research for the understanding of the neuromuscular mechanisms is the comparison, between sexes, of the external mechanisms of ACL injuries. It has been hypothesised that females may exhibit lower knee flexion angles than males and consequently, increase the potential for ligament failure during some specific movements. However, the studies do not consistently confirm this hypothesis. In controlled laboratory trials, some studies found that women exhibited a decreased knee flexion angle than men during the landing phase of stop-jump tasks (Chappell *et al.*, 2002) while others found an increased knee flexion angle during jump landings in women (Fagenbaum & Darling, 2003). It was also reported by some that, in sidestep cutting, female athletes tended to have less knee flexion angles than male (Malinzak *et al.*, 2001) while others found no such difference (Ford *et al.*, 2005; McLean *et al.*, 1999). In a cohort study design, knee flexion angles were not found to be related with subsequent ACL injury among soccer, basketball, and volleyball players (Hewett *et al.*, 2005). Nevertheless, in this same study an increased peak external hip flexion moment was found in injured players. In fact, Decker *et al.* (2003) compared the energy absorption strategies during landing between female and male recreational athletes, and concluded that male tend to use the hip extensors more than females. Overall, we are far from a consensus about sex-specific high risk movements in the sagittal plane. Contrary to this there is a large consensus about the risk of lower limb movements in the other two planes (frontal and transverse), as already described in the section about ACL injury external mechanisms. There are several papers confirming that females tend to have higher valgus knee motions than men (frontal plane movements), at least at the early stages of landing (Russell *et al.*, 2006; Ford *et al.*, 2005; Ford *et al.*, 2003; Chappell *et al.*, 2002) and that this is a risk factor (Hewett *et al.*, 2005).

An interesting aspect is that a combined or energy transfer mechanism could exist between movements in the sagittal plane and the other planes *i.e.* if women perform landings with less hip flexion this could be associated with an increased hip adduction (*i.e.* knee abduction or a valgus knee) (Pollard *et al.*, 2007; Hewett *et al.*, 2006a).

Curiously enough, in sidestepping (a cutting manoeuvre) women showed larger knee valgus torques associated with larger initial hip flexion (McLean *et al.*, 2005).

It is well known that, on average, women have lower muscular mass. It is also well known that musculature plays a major role in dissipating loads that, otherwise, would end on the passive structures like ligaments, cartilage and bone. In the specific case of the ACL there are a few reports on the protective effect of the posterior musculature and posterior external forces in pure planar movements (Shin *et al.*, 2007; Mesfar & Shirazi-Adl, 2005; Beynnon & Fleming, 1998; Pandy & Shelburne, 1997). So it is important to know if there are different uses of the musculature between sexes. Lephart *et al.* (2002) had already showed that women's *time to peak torque* is higher and the torque per body mass unit is lower than in men. Both these factors contribute for a reduced active stiffness of the hip musculature (Granata *et al.*, 2002) which results in an increased loading of the passive structures of the knee under impulsive loads. Later, Myer *et al.* (2005) suggested a different use of the quadriceps by women, one that increases the chances of dynamic valgus which is a known risk factor. On the issue of quadriceps' dominance, there is no complete agreement with reports of

- no muscle activation differences between the sexes in landing exercises (Fagenbaum & Darling, 2003);
- faster quadriceps recruitment in rotary single leg exercises in females (Carcia *et al.*, 2005);

- reports of higher hamstring preactivation on women in running exercises (DeMont & Lephart, 2004);
- lower hamstring preactivation on women in cutting movements (Hanson *et al.*, 2008) or single leg squats (Zeller *et al.*, 2003).

Finally we would like to emphasize the synthetic nature of these evaluations. The absence of opposition (forcing high-speed movements) and of a ball in hand (the most common situation of ACL injury in handball) could make all these results of poor predictive value (Chaudhari *et al.*, 2005; Cowling & Steele, 2001).

Proprioception. A reduced ability to know one's own physical status in space is probably an important risk factor. Studies have linked the increased knee laxity in women with the diminished knee proprioception (Rozzi *et al.*, 1999). This reduced sensitivity of the ACL is then related to a delayed response (Shultz *et al.*, 2004a) and could prove to be a possible sex-related risk factor of ACL injury. The scarcity of reports in this area is difficult to interpret, as quite a few injury prevention strategies will involve proprioception development.

Fatigue. Fatigue is a neurological condition and as such has implications on the quadriceps activation patterns (Nyland *et al.*, 1997a) as well as other muscles (Nyland *et al.*, 1997b), on kinematics (McLean *et al.*, 2007; Chappell *et al.*, 2005; Madigan & Pidcoe, 2003), on proprioception (Miura *et al.*, 2004) and more importantly in cognition, decision making and motor control (Miura *et al.*, 2004; Lorist *et al.*, 2002). There are few studies on this and the Lorist's reference gives a clue: fatigue effects are mostly visible under complex multi-tasking loads and not in simple laboratory experiments. If there is a link between decision making and fatigue this could mean that there is a dual effect: fatigued, a player could place herself in circumstances that should usually be avoided; and, additionally, she is also less capable of handling that unusual load resulting from the situation (a fall, a late cutting, *etc.*).

2.3.4.3 External

Practice exposure. The training practice and competition is directly implicated in the type of load, and potentially may change the athlete's capacity to sustain it, either positively or negatively – the susceptibility to injury. In fact, this is the subjacent reasoning behind every prevention program – a few changes in training that alter the player's ability to handle the game load. Also implicated are the rules (*e.g.* allowed/not allowed contact), referee's interpretation, *etc*. Very little is known about the positive/negative effect of changes in these parameters, much less its comprehension or quantification. In a dismaying paper written by 20 of the best researches in ACL injuries (Renstrom *et al.*, 2008), no more than a few very abstract lines are written about external risk factors, mentioning only four of them (competition, footwear and floor, protective equipment and meteorological conditions).

Investigations have been limited to the description and analysis of the event itself, ignoring the fact that a long line of accumulating effects could have taken place: duration and intensity of the competition, individual (or collective) stress and commitment, fatigue, *etc*. all of which could concur to the final event. This is the point of the comprehensive models of injury from Bahr & Krosshaug (2005)

The demonstrated increased risk of noncontact ACL injury during competition, at least in handball (Myklebust *et al.*, 2003a; Myklebust *et al.*, 1998; Myklebust *et al.*, 1997), suggest some possible (des)adaptation of the training process to the competition tasks. In addition, a disproportionate number of injuries were found in attack situations, handling the ball, and mainly in the back positions. This has to be further explored.

Footwear and playing surface. An increased friction between sports shoes and floor may produce a favourable effect on performance, through an increased force transmission, resulting in higher accelerations, useful for faking movements, *etc*. However, it may also increase the risk of ACL injury (Renstrom *et al.*, 2008) as, exactly for the same reason (increased force transmission), the forces and torques acting on the lower limbs are higher.

This problem has been studied from an epidemiological perspective, mainly based on injury rates in different types of practice surfaces. However, the final statistical quality of these types of studies is dependent on obtaining a high number of injuries. It is also necessary to study this effect both in practice and in competition, quantifying precisely the exposure times in each case, *etc*.

Strand *et al.* (1990) based on a retrospective study of 144 ACL injuries, sustained while playing handball, concluded that most of the injuries occurred in synthetic surfaces. Myklebust *et al.* (1997), analysing both sexes during two seasons, found similar injury rates between wooden and synthetic floors. However, Olsen *et al.* (2003) analysing data from seven seasons, including the data from Myklebust *et al.* (1997) found an increased risk of injuries in women on synthetic surfaces in comparison to wooden floors.

A prospective study in american football found that sport shoes with a high number of cleats and great torsional resistance were positively associated with ACL injury risk

(Lambson *et al.*, 1996). In the same way, the risk of ACL injury was increased in natural grass *versus* the artificial surfaces (Scranton *et al.*, 1997). Other factors as dry *versus* wet and cold *versus* warm weather have also been studied, being the conclusion that slippery surfaces are associated with lower ACL injury rates (Orchard & Powell, 2003).

2.3.5 Hormonal risk factors

2.3.5.1 Hormonal factors and ACL injuries

It has been suggested that female hormonal fluctuations, during a *normal* menstrual cycle, may change the molecular and mechanical properties of the ACL, and consequently the risk of ACL failure under extreme mechanical load.

Looking for a potential protective effect from hormonal therapy (OCs), Agel *et al.* (2006) compared the rate of noncontact ACL injuries and noncontact ankle strains in a sample of 3150 collegiate basketball and soccer players with/without hormonal therapy, across three seasons. In non-OC users (2050 players) 12 ACL injuries and were sustained in OC users (1024 players) 8 ACL injuries occurred. No differences in injury rates were found between these two groups; although a higher rate was found in basketball in comparison to soccer.

Also following this hypothesis some authors have investigated the periodicity of ACL injury throughout the menstrual cycle. Table 2.5 summarises the reviewed studies. Their findings are not consensual or unequivocal. The majority of the studies found that the risk of suffering an ACL during the menstrual cycle phases does not remain constant, but the time frame in which the risk increased/decreased vary among the studies (see table 2.5). Several methodological differences and practice limitations may partially explain the lack of consensus between the results.

The main methodological assumptions that may have influenced the results are: (a) the menstrual cycle *standard phases* have a distinct, reproducible, hormonal content, and (b) different sports or activities have comparable injury risk.

Menstrual cycle assessment considerations

Not all studies considered the same menstrual phases and correspondent time frame, nor the criteria used for cycle division was uniform. An ovulatory phase was considered in the majority of the studies, which we redesigned *around ovulation*[14]. The criterion used to split the cycle was the estimated day of ovulation, assuming a fixed window of 14 days after

[14] Ovulation is an event, not a phase.

ovulation *until next menses*. The most notable limitations are the hormonal content variability or possible menstrual dysfunctions among the studied subjects which were not prospectively studied. These could have biased the results, particularly in the case of athletes.

Table 2.5 Phases/days of major ACL injury rate according to several authors

Study	Sports	Age (mean ± SD or range) (y)	ACL injuries	Menstrual phase/day at time of injury			
				Determination of phase/days	Follicular (early follicular) (days 1-9)	Around ovulation (late follicular) (days 10-14)	Luteal (day 15 to menses)
(Wojtys *et al.*, 1998)	Basketball, skiing, soccer, and several activities	23 ± 11	28 (5 OCs)	Questionnaire	4 ↓	8 ↑	16
(Myklebust *et al.*, 1998)	Elite handball players	21.9 ± 3.4	17 (9 NOCs; 8 OCs)	Interview	5 (days 1-7)↑*	2 (days 8-14)	1 (days 15-21) 9 (days 22-28)↑*
(Arendt *et al.*, 1999)	Basketball and soccer collegiate players	ND	21 NOCs 7 OCS	Interview type questionnaire	11↑* (NOCs) 4 (OCs)	1 (NOCs) 1 (OCs)	9 (NOCs) 2 (OCs)
(Arendt *et al.*, 2002)	Collegiate athletes from different sports	ND	58 NOCs 25 OCs	Questionnaire	25 ↑ (NOCs)	12 (NOCs)	21 (NOCs)
(Slauterbeck *et al.*, 2002)	ND (athletes)	ND	37 (31 NOCs; 6 OCs)	Questionnaire Salivary sex-hormones (P; E_2)	25 ↑* 5 (OCs)	1	11 1 (OCs)
(Wojtys *et al.*, 2002)	Basketball, skiing, soccer, and several activities	28 ± 3.4 (15-46)	51 NOCs 14 OCs	Questionnaire Urine: P; E_2; LH	13 (NOCs) 2 (OCs) ↓	24 (NOCs) (2.5) ↑* 4 (OCs)↑	14 (NOCs)↓* 8 (OCs)
(Myklebust *et al.*, 2003a)	Elite handball players	ND	18 NOCs 28 OCs	Interview type questionnaire	(days 1-7) 9 (NOCs) 14 (OCs)	(days 8-14) 4 (NOCs) 8 (OCs)	(NOCs): 1 (days 15-21) 4 (days 22-28) (OCs): 4 (days 15-21) 2 (days 22-28)
(Beynnon *et al.*, 2006)	Recreational alpine skiers	39 ± 10 (15-53) Control: 35 ± 9 (14-50)	42 NOCs (45 skiers uninjured – control group)	Questionnaire Serum: P; E_2	Preovulatory phase: 31↑* Control group: 25		Postovulatory:11↓* Control group: 20
(Adachi *et al.*, 2008)	Basketball, volleyball, handball and other activities	16.2 (11-18)	18 NOCs	Questionnaire	2 (NOCs)↓*	13 (NOCs)↑*	3 (NOCs)↓*

ND = not defined; **NOCs** = non-OCS users**;** **OCs** = OCs users; ↑ increased number of injuries**;** ↓ decreased number of injuries**;** *****indicates statistically significant differences (usually on a 5% level).

Maybe to avoid dealing with menstrual disturbances, most of the studies included only subjects that reported regular and/or *normal* cycles (Adachi *et al.*, 2008; Arendt *et al.*, 2002; Wojtys *et al.*, 2002; Myklebust *et al.*, 1998; Wojtys *et al.*, 1998). The state of regularity or normality is not always clear or uniform in surveys. The common used criterion to classify a cycle as *normal* was its length, being *normal* between 23 to 35 days (Wojtys *et al.*, 2002), between 25-30 days (Adachi *et al.*, 2008), or lasting less than 29 days (Arendt *et al.*, 2002). Few authors referred to have adjusted the menstrual cycles length to a standard of 28 days (Myklebust *et al.*, 2003a; Myklebust *et al.*, 1998).

The common procedure to estimate the relative date within menstrual cycle in which the injury occurred was the date of menses onset *before* the sustained injury. This was obtained through retrospective recall of menstrual cycle characteristics and the aforementioned dates. However, it is not detailed to whether full length of this cycle was also tracked, or if the *normal* range reported by the subject was assumed. This particular cycle could be *non-normal* and phases incorrectly assumed.

Few studies (37% of all subjects) confirmed the determined menstrual phases from the hormonal content of serum (Beynnon *et al.*, 2006), saliva (Slauterbeck *et al.*, 2002) or urine (Wojtys *et al.*, 2002). The reliability between the self-reported menstrual cycle time and its determination by urine and saliva was accounted between 80% and 95% - (Wojtys *et al.*, 2002) and (Slauterbeck *et al.*, 2002), respectively. Same bias could have been introduced because only one day per cycle, after the injury (72 hours maximum), was assessed. The accuracy of the decisions made via hormonal levels could have been affected and were not tested. For example, an unovulatory cycle could be confused with the follicular phase from an ovulatory cycle. Undetected luteal phase defects could also make for an inaccurate estimation of ovulation date.

Most of the studies included subjects that were taking OCs when injured. The procedures to investigate the periodicity of ACL injuries throughout their menstrual cycle were the same as for non-OC users. Moreover, the type of OCs used were not available or considered in the studies. Admitting that the used OCs had the same general composition and its main effect was to stabilize the hormonal environment (mainly the estrogen and progesterone) in order to suppress ovulation, the “typical” menstrual phases should not have been used in these cases. For instance, there is no plausible explanation to collectively analyze these two groups on menstrual cycle fluctuations and ACL injury susceptibility, as it was done by some authors (Wojtys *et al.*, 1998).

Other methodological considerations

These types of studies are very constrained by the nature of the variables and the necessary number of ACL injuries to reach powerful analyses. For this reason, the majority of studies considered data from different ages (11 years to 53 years), several different sports and practice levels (recreational to elite), multiple seasons (Myklebust *et al.*, 2003a) up to a decade long (Arendt *et al.*, 2002).

In order to statistically manage co-variables (others risk factors) it’s desirable to include a control group. To our knowledge, just one study used a case-control design (Beynnon *et al.*, 2006). Their study was conducted with recreational alpine skiers, whose control group was similar in age, height and weight, menstrual cycle regularity and mean cycle length. Still the skiers from the control group self-reported a higher expertise, coinciding with a higher average number of practice seasons which needed to be accounted for. After a logistic regression including the expertise as a parameter, it was concluded that the risk of ACL injury in the preovulatory phase was 3 times higher than in the postovulatory phase.

Another important point is how the ACL injury mechanism was recorded and determined. Several studies used a questionnaire, but leaving it unspecified to which type of questions were used and/or which categories were established (Wojtys *et al.*, 1998). It is not always clear if only non-contact ACL injuries were included in the analyses (Arendt *et al.*, 1999; Wojtys *et al.*, 1998).

Methodological differences, such as data reduction, have also created some difficulties when trying to compare the results from the studies. The work developed by *Arendt et al.* (2002) used time series analysis techniques, including *centered moving average smoothing* and Fourier techniques. However, the methods employed are not phase preserving (they shift the peaks forward by 2 or 3 days) and are too sensitive (a single injury omission or inclusion, at the appropriate days, changes the peaks of the adjusted curves by half a cycle). Mainly, its methodology is incomparable to all the other studies.

Despite all the incongruences among studies, there is a common trend of an increased likelihood of sustaining ACL injury during the preovulatory phase (obviously on non OCs users). Attempting to understand the influence mechanism of sex hormones on the structure and metabolism of the ligament, several *in vitro* studies have been done.

2.3.5.2 Effects of sex hormones on ACL collagen structure and synthesis

Several approaches have been made to understand the biological significance of sex hormones, including estrogen, progesterone, relaxin and testosterone, on the cellular metabolism and collagen synthesis of tendons and ligaments. This has been explored because collagen, produced by fibroblasts, is the main load-bearing constituent of ligamentous tissue (Dragoo *et al.*, 2003); therefore, changes in the collagen content and organization may lead to changes in load-bearing capacity. This idea has been further stimulated by the identification of sex hormone receptors in the fibroblasts of the human ACL (Faryniarz *et al.*, 2006; Dragoo *et al.*, 2003; Hamlet *et al.*, 1997; Liu *et al.*, 1996).

To our knowledge, Liu *et al.* (1996) were the first to successfully identify estrogen and progesterone receptors in both male and female ACL. In another study estrogen receptors were identified in 4% to 10% of ACL cells, with no significant differences between sexes (Faryniarz *et al.*, 2006).

The work from Dragoo *et al.* (2003) found specific binding places for relaxin in female ACLs but not in male samples. Faryniarz *et al.* (2006) found a different proportion of relaxin binding sites between sexes: in 4/5 of female ACL cells and 1/5 of male ACL cells.

Androgen receptors were primarily identified in ACL specimens from young male subjects, which was not the case of ACL specimens from women or older men (Liu *et al.*, 1996). More recently, testosterone receptors were also found in female ACL samples (Lovering & Romani, 2005). These findings suggest a possible androgenic effect on female ACL tissue.

Effects on collagen metabolism

Ligament homeostasis is dependent on the balance between cell proliferation, collagen synthesis and collagen degradation (Seneviratne *et al.*, 2004).

Studies on the effect of estrogen on collagen metabolism and structure on ACL tissue include both animal models (Seneviratne *et al.*, 2004; Liu *et al.*, 1997) and humans (W. D. Yu *et al.*, 2001; W. D. Yu *et al.*, 1999).

Liu *et al.* (1997) investigated the effect of 17β-estradiol on cell culture of female rabbit's ACLs. The authors documented a decreased fibroblasts proliferation and collagen synthesis at both physiologic and supraphysiologic levels of estradiol (0.025ng/mL-25.0 ng/mL), in a 2 weeks' prospective examination. An apparent equivoque is that the physiological levels are reported to human's and not to rabbit's (rabbit's physiological levels are 0.005-0.008 ng/mL (J. B. Miller & Keyes, 1978)), meaning that the tissue was always exposed to supraphysiologic levels during a two-week period. Contradicting results were found by Seneviratne *et al.* (2004). These authors found no changes in fibroblast proliferation and collagen synthesis in an *in vitro* ovine cell culture exposed to physiologic or supraphysiologic (for both sheep and humans) concentrations of 17β-estradiol.

W. D. Yu *et al.* (1999) investigated the effects of both physiologic and supraphysiologic levels of 17β-estradiol (range 0.0029 ng/mL-25 ng/mL)[15] using *in vitro* cell cultures from human ACL samples. A short-term (days 1 and 3) decrease in fibroblast proliferation and in type 1 procollagen synthesis was shown as estradiol levels increased progressively. A similar study was conducted afterwards by W. D. Yu *et al.* (2001) to study the combined effect of 17β-estradiol and progesterone. This study showed an increase in fibroblasts

[15] Human serum physiologic estradiol level: ~0.025 ng/mL, but can fluctuate an order of magnitude or more

proliferation and procollagen type 1 synthesis with increasing progesterone concentrations (range 1ng/mL-100ng/mL, physiologic and supraphysiologic), counteracting the estrogen effect.

Further work is needed to fully understand the quantitative effect (dose/exposure and potentiation/inhibition effects) of sex hormones on ACL tissue. We were unable to find any studies about the relaxin and testosterone interactions with the ACL tissue.

Of particular interest is the possible effect of female's *versus* male's hormonal environment on the collagen turnover capacity in a loaded ligament tissue or possibly in microinjury repair.

In a pure *in vitro* study, the group of Lee *et al.* (2004a; 2004b) tested the single effect of physiological levels of estrogen in the mRNA expression of types 1 and 3 of collagen in the pig's ACL. They found a steady increase in collagen expression up to 27pg/mL of estrogen (more than a 3-fold increase for type 3 collagen). The effects of a mechanical load (5% strain at 0.5Hz for 24h) were also studied. *Per si* this load increases type 1 expression 25% and reduces type 3 by 28%. Combined with estrogen there is always a marked reduction of up to 86% of this effect. The hypotheses of an estrogen and load modulated structure (and function) of the ACL is subscribed by this data.

Effects on mechanical properties

Studies addressing the sex hormones effect on the mechanical properties of the ACL have been limited to estrogen. The experimental studies were constrained to animal models, such as rabbits (Komatsuda *et al.*, 2006; Slauterbeck *et al.*, 1999), ovine (Strickland *et al.*, 2003), rats (Warden *et al.*, 2006; Rau *et al.*, 2005), and primates (Wentorf *et al.*, 2006).

Slauterbeck *et al.* (1999) found a reduced (10%) *ultimate load at failure* in ACLs of ovariectomized rabbits treated with estrogen for 30 days, in comparison with the matched control group. These are constrained to high levels of estradiol exposure (52 pg/mL), similar to pregnancy.

In another study, similar results were found also using rabbits as models (Komatsuda *et al.*, 2006). Using a matched-control design, all rabbits were ovariectomized and submitted (by intramuscular injection) to different 17β -estradiol dosages (50, 100, and 500µg/kg), at 1, 2, 3 and 4 weeks. After this treatment period, the animals were euthanized and the mechanical properties of the ACL tested. In both *ultimate tensile stress* and *linear stiffness* statistical differences were found between the group with medium levels of estradiol (mean serum levels of 60 pg/mL) and the group with higher levels of estradiol (mean serum levels of 231 pg/mL). Such findings are, once more, associated to supraphysiological levels.

Such effect was not observed in another animal model (sheep). Six rams and thirty-eight ovariectomized ewes, were randomly divided into five groups: sham-operated, ovariectomized, ovariectomized and estradiol implant, low-dose raloxifene[16] and high-dose raloxifene (Strickland *et al.*, 2003). The implant was used during a period of 6 months, and maintained a serum estradiol level of 2pg/mL, analogous to the luteal phase of the estrus cycle. No significant differences were found between the groups at any of the ACL structural tested properties (maximum force, stiffness, and energy to failure).

Similar findings were obtained when the mechanical properties of the ACL were examined during the natural estrous cycle (Rau *et al.*, 2005). Sixty female rats were euthanized and grouped according to the various stages of the estrous cycle (diestrus, proestrus, estrus, and metestrus). No differences at ACL *load to failure* or *stiffness* were found at any stage of the estrous cycle. Additionally, a high density of α estrogen receptors[17] in the ACL tissue was observed at all stages of the cycle.

Warden *et al.* (2006) investigated the effect of *estrogen* and/or an *estrogen agonist receptor α-specific* in male Sprague-Dawley rats and *estrogen gene mutation on β-receptors*[18] in female BERKO's mice. After being submitted to each treatment daily, for 5 consecutive weeks, the animals were euthanized. The ACL and MCL *load to failure* and the ligament dynamics (response to impulsive loads) was studied. No statistically significant differences in any parameters (ligament viscoelastic and tensile mechanical properties) among the three active groups or the control group (female Wild Mice) were found. Unfortunately, this experiment mixed gender and species in an arguable manner and did not control the serum estrogen levels.

Wentorf *et al.* (2006) looked for a systematic estrogen effect in the mechanical properties of the ACL and patellar tendon. The samples were obtained from two cynomolgus monkey groups - sham-operated with intact ovaries *versus* ovariectomized ones. Significantly different estrogen levels were measured between the two groups at 6, 12, 18, and 24 months after the surgeries. No statistical differences were found at any of the mechanical or material properties evaluated, such as *load to failure*, stiffness, elongation at failure, ultimate stress, ultimate strain or energy to failure at 24 months after surgery.

[16] Raloxifene is an estrogen receptor agonist
[17] *α-receptors* are probably related to cell proliferation
[18] *β-receptors* are probably related to cell differentiation

The reviewed studies in animal models were unsuccessful to detect changes in the ACL mechanical properties under exposure to physiologic levels of estrogen. This usually lead the authors to speculate that female hormonal milieu may not be the key cause for the increased risk of ACL rupture in female athletes compared with male (Warden *et al.*, 2006; Wentorf *et al.*, 2006; Strickland *et al.*, 2003).

Still though, further studies are needed is this area. Studies in animal models are important contributions to the understanding of the effects of estrogen on the mechanical properties of the ligament, even when the used estrogen doses/exposures are very high and not representative of a physiological state. But are not definite.

There is also a need to look for an eventual acute estrogen effect followed by a saturation effect. At supraphysiologic levels, a significant effect on collagen metabolism was detected at day 1, decaying until being almost null by day 7 (W. D. Yu *et al.*, 2001; W. D. Yu *et al.*, 1999). In a standard human menstrual cycle, the estrogen progressively and then sharply rises until a certain point, before ovulation. Usually this phase (follicular) is no more than 20 days long. Several studies missed this point and used fixed exogenous estrogen dosages for long periods: from about 4 weeks (Komatsuda *et al.*, 2006; Slauterbeck *et al.*, 1999) to 5 weeks (Warden *et al.*, 2006), or even 6 months (Strickland *et al.*, 2003). They all could have suffered a saturation effect.

As most of the studies were restricted to estrous cycles, with estrogen fluctuations, phases and periodicity being different from a human menstrual cycle there is a need for more studies using primate models and humans.

We were unable to find any studies about interactions of other sex hormones, like progesterone, relaxin and testosterone, with direct measurements of ACL mechanical properties.

2.3.5.3 Hormonal fluctuations and knee joint laxity

Basic research, indicating possible interaction of sex hormones with ACL fibroblast proliferation and collagen synthesis, have created a framework to investigate the behaviour of the knee under different sex hormonal environments. Several studies have taken place in the last years, mainly focused on “normal” menstrual cycles, under OC treatment or during pregnancy.

Direct measurements of the mechanical properties of the ACL ligament *in vivo* are not easily available. They require the simultaneous assessment of both the strain and the stress (very difficult) of the ACL, ideally in real life situations. Usually one requires only the quantification of the ligament strain under some known external load, abandoning the stress measurements. Invasive techniques have been used (Beynnon & Fleming, 1998; Beynnon *et al.*, 1992). Beynnon *et al.* (1992) measured the strain in the anterior medial portion of the ACL by attaching a transducer arthroscopically.

Other less invasive techniques have also been used to quantify the tibiofemoral joint strain, including radiostereometric analysis (Isberg *et al.*, 2006; Karrholm *et al.*, 1988), plant stress radiography (Staubli *et al.*, 1992) and fluoroscopic imaging (Thompson *et al.*, 2004).

One other strategy is to use only indirect non-invasive measurements. The ACL is commonly described as the primary restrain to anterior tibial translation relative to the femur. The quantification of the passive anterior-posterior knee displacement in the sagittal plane is related (in an unknown way) to the ACL stiffness. Several devices are commercially available, based on this strategy (Carcia *et al.*, 2004; Schuster *et al.*, 2004; Un *et al.*, 2001; Ganko *et al.*, 2000; Strand & Solheim, 1995). Alternatively, one can use another blocking function of the ACL – the internal and external rotation – and perform external rotatory measurements (Tsai *et al.*, 2008; Muaidi *et al.*, 2007; Musahl *et al.*, 2007).

Anterior knee joint laxity versus *stiffness*

Stiffness is a mechanical quantity used, for instance, to indicate the resistance to traction of a material or a system (figure 2.11). It is defined as the derivative of the force-elongation curve and in several materials, notably the human ligaments, varies from point to point (figure 2.12). In the ligament's case, it has a higher stiffness (*resistance*) at higher loads than at lower ones, a fact that can be tracked to the histology of the ligament and the collagen structure. Additionally the ligament has a complex time response, history dependent – it responds differently to short (*fast*) or to long (*slow*) loads (S. M. Gross *et al.*, 2004), differently to successive loads (figure 2.13) and, additionally, after a strong load it takes time to return to its original state. Even more, as a living tissue, it suffers damage and recovery along time (Woo *et al.*, 2006; Woo *et al.*, 2000; Liu *et al.*, 1995). All this conspires to make measures potentially incomparable among researchers, if methods are not clearly detailed.

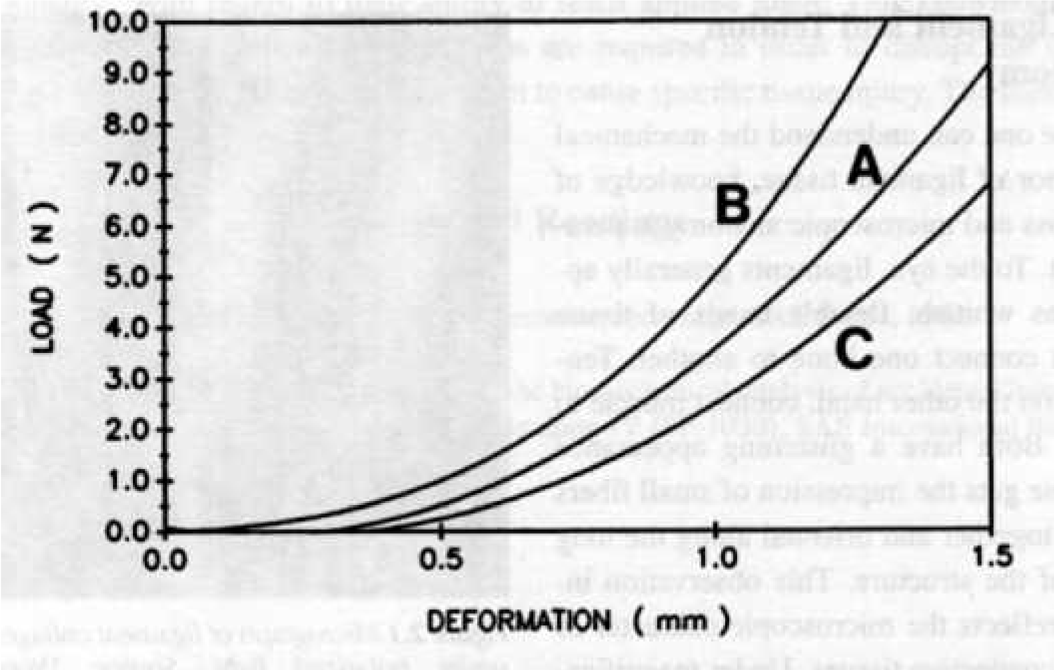


Figure 2.11 Typical human ligament load curves (Gomez, 2001), p. 10

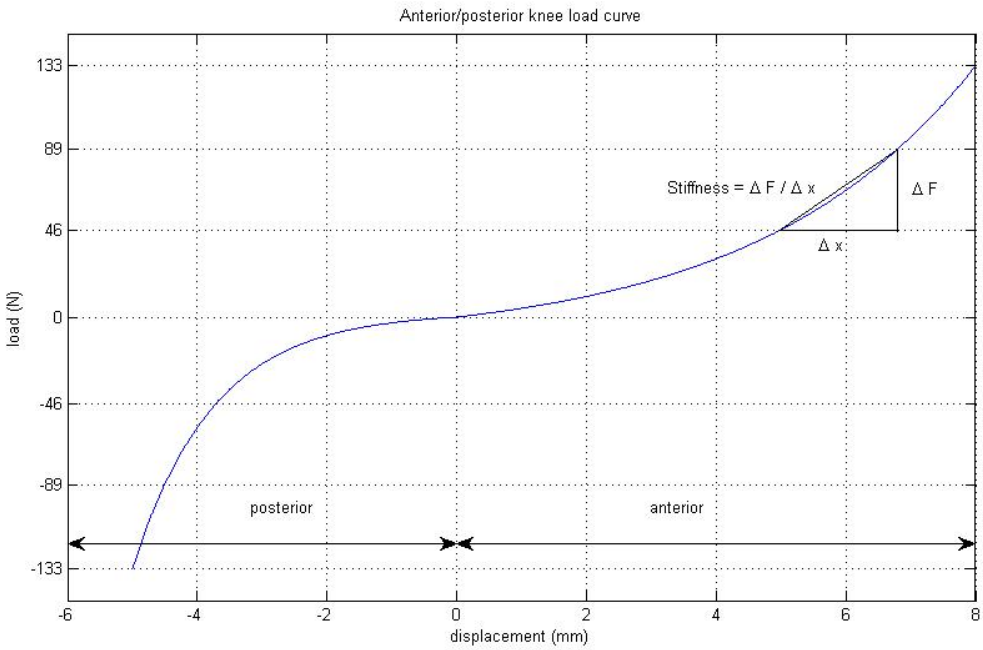


Figure 2.12 Typical knee load curve (spline fit)

A quantity ill defined but loosely related to stiffness is the laxity, which is sometimes used as a synonym of compliance (the inverse of stiffness). In some papers, as the reference forces are fixed, it is also common to use laxity as a synonym of elongation (or relative displacement), as the measured average compliance is just the elongation divided by the reference forces.

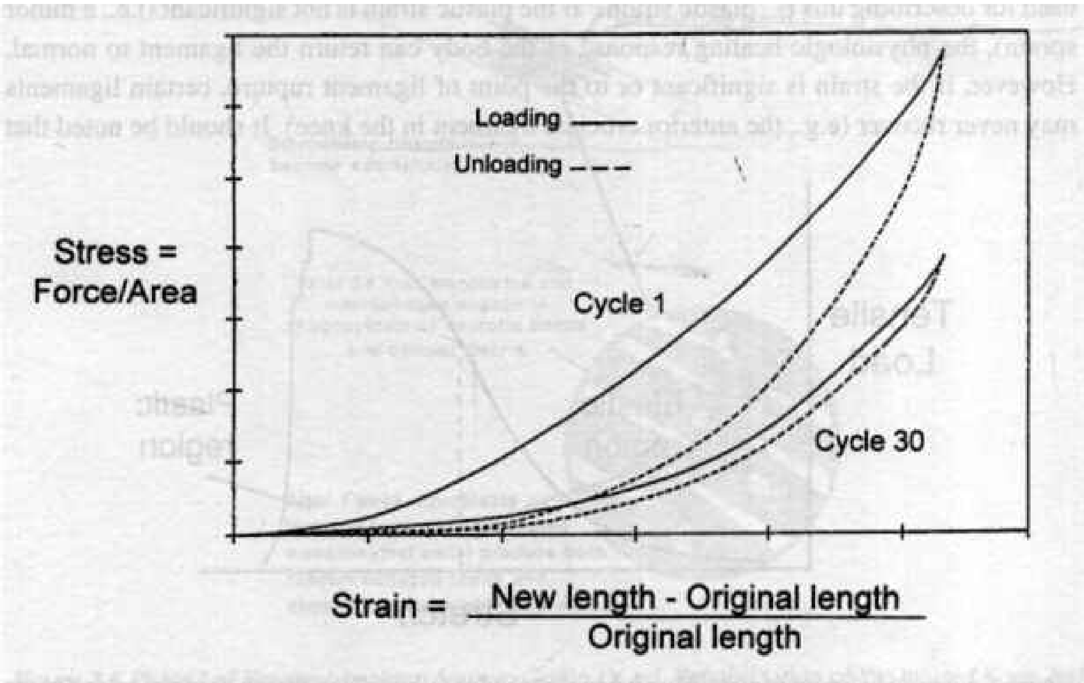


Figure 2.13 Ligament dynamic response curves (Gomez, 2001), p. 11

Studies about menstrual-cycle hormone fluctuations and knee joint laxity

The majority of the studies reviewed (table 2.6) were not successful at identifying menstrual cycle effects on anterior knee laxity, as measured with a knee arthrometer (9 studies in 14). Few studies have documented knee laxity changes between the menstrual cycle phases (Hicks-Little *et al.*, 2007; Shultz *et al.*, 2005; Deie *et al.*, 2002; Heitz *et al.*, 1999). In one of the studies (Hicks-Little *et al.*, 2007) the reported differences were just significant when the data from OC users and non-users were analyzed together. When differences were found they revealed an increased knee laxity in the late follicular phase (so called *ovulatory phase*) and luteal phase in comparison to the early follicular phase (table 2.6).

Apparently, these studies suffer from the same methodological limitations and inconsistencies, which in part restrain the interpretation of the results and the comparisons between studies.

Firstly, the commonly used methods for assessment of the anterior knee displacement are not a direct measure of the ACL strain. Joint capsular tissue and musculotendinous complexes (passive and active) may also be involved (Shultz, 2007). The applied force was not uniform to all studies, ranging between 49N to 156N. Typically the higher test forces provide the most reliable measurements of displacement, because it’s more likely to be working in the *linear region* (out of the *toe region*).

Secondly, the menstrual-cycle hormone fluctuations were not accurately tracked in the majority of the studies, including: (a) non-assessed hormonal content; (b) distinct and few hormones evaluated; (c) low, randomly and distinct time points assessed during the menstrual cycle; (d) lack of control group.

The main equivocal supposition frequently used was that time points in the cycle/phase represents the same hormonal *milieu* for different cycles of each and every woman. Self-reported menstrual cycle regularity and/or a standard cycle length were frequently adopted as an inclusion criteria, maybe in an effort to avoid hormonal variability within each phase or subject. In consequence, several confounding factors may have been introduced and may have masked the results.

Menstrual-cycle monitorization

Three of the reviewed studies did not perform any type of hormonal evaluation (Hicks-Little *et al.*, 2007; Belanger *et al.*, 2004; Karageanes *et al.*, 2000). The phases were divided based on the assumption that ovulation occurs in the middle of the cycle, 14 days prior the next menses (Karageanes *et al.*, 2000) or divided by the estimated day of ovulation based on the temperature nadir (Belanger *et al.*, 2004).

Several studies assessed the hormonal content in three times points, trying to capture a representative hormonal content per phase (Eiling *et al.*, 2006; Carcia *et al.*, 2004; Romani *et al.*, 2003; Van Lunen *et al.*, 2003). In only two of those, daily self-LH tests were used to estimate the day of ovulation and to define the days/phase for blood collection *i.e.* after LH surge and in the luteal phase (Romani *et al.*, 2003; Van Lunen *et al.*, 2003). Between 4 to 8 hormonal assessments per cycle were also taken (Pollard *et al.*, 2006; Beynnon *et al.*, 2005; Deie *et al.*, 2002; Heitz *et al.*, 1999) which then determined the corresponding menstrual phase. Only one used daily self-LH tests to estimate the ovulation day (Beynnon *et al.*, 2005).

In only two studies the serum or urine hormonal content was daily assessed (Hertel *et al.*, 2006; Shultz *et al.*, 2004b).

The studies diverge in the type and number of assessed hormones:

(a) Estrogen and Progesterone (Eiling *et al.*, 2006; Beynnon *et al.*, 2005; Deie *et al.*, 2002; Heitz *et al.*, 1999)
(b) Estrogen, progesterone and testosterone (Lovering & Romani, 2005; Carcia *et al.*, 2004; Shultz *et al.*, 2004b; Van Lunen *et al.*, 2003)
(c) Estrogen (Pollard *et al.*, 2006)
(d) Relaxin (Arnold *et al.*, 2002)

Relation between time points of the cycle and laxity tests

A frequent lapse of a week between consecutive serum sampling was taken (Eiling *et al.*, 2006; Pollard *et al.*, 2006; Beynnon *et al.*, 2005; Carcia *et al.*, 2004; Romani *et al.*, 2003; Van Lunen *et al.*, 2003; Arnold *et al.*, 2002) so the uncertainty to have captured the estrogen peak before ovulation is high. Additionally, if any laxity test was not performed during this time frame, the potential laxity variation could also have been missed. These methodological constrains are somehow supported in a short-time dependence (1 and 3 days) between the main estrogen rise and a decreased fibroblasts cellular activity and collagen synthesis effect (W. D. Yu *et al.*, 2001; W. D. Yu *et al.*, 1999). In these *in vitro* studies, a long continuous estrogen effect on the ACL tissue under a high and constant dosage was not found, being significantly reduced within 4 to 7 days after the beginning of its administration.

In the study of Hertel *et al.* (2006), estrogen and testosterone levels were daily assessed (in urine) but the knee laxity evaluations were limited to a single day per phase.

Only Shultz *et al.* (2004b) determined, daily, the hormonal content of serum and the knee laxity. The authors found that the content of estradiol, progesterone, testosterone and their interactions explained, on average ≈63% of the variance in the knee laxity across the menstrual cycle within *each subject*, when a time delay[19] of 3 to 4 days was considered. Even though, when all menstrual cycles were analyzed together, the explained variance decreased to 8%. Furthermore, the authors noted that cyclic changes in knee laxity were not found in all subjects.

Other methodological considerations

The majority of the studies did not start the testing period on random days or phases of the cycle (Zazulak *et al.*, 2006), what could seem a reasonable methodological critic. Several studies (Pollard *et al.*, 2006; Belanger *et al.*, 2004; Carcia *et al.*, 2004; Shultz *et al.*, 2004b; Romani *et al.*, 2003; Arnold *et al.*, 2002; Deie *et al.*, 2002; Karageanes *et al.*, 2000) started the tests, randomly or not, at any time or at three distinct points of the cycle: on menses, prior to ovulation or somewhere in the luteal phase. Then the phases from different consecutive cycles were added and analyzed as a single menstrual cycle. This procedure could have introduced confounding factors for menstrual phase determination and in consequence also in the knee laxity testing. In some other cases, the adopted procedures aren't even completely described (Hicks-Little *et al.*, 2007; Hertel *et al.*, 2006).

[19] A time lag between the hormonal change and corresponding knee laxity variation.

Few studies used a control group (Pollard *et al.*, 2006; Beynnon *et al.*, 2005; Shultz *et al.*, 2004b; Arnold *et al.*, 2002) constituted by male subjects. Some others also added a group of female OC users, maybe as negative controls (Hicks-Little *et al.*, 2007; Arnold *et al.*, 2002). But both of these two last studies are very confusing regarding the purpose of the control groups, as no laxity variation is presented for this group and there are no additional analyses of its relation with hormonal content.

Table 2.6 Studies about the influence of sex hormones on anterior knee laxity

Study	Subjects	Sports	Age (mean ± SD or range) (y)	Menstrual cycle length	Menstrual cycle monitoring	Assessment protocol (days/phases)	Device used and force applied	Anterior displacement (mm)		
								Follicular phase	Near ovulation	Luteal phase
(Heitz *et al.*, 1999)	7 F non OCs users	Recreational athletes	26.9 ± 4.2	(28-30 d)	Serum: E_2; P_4	Hormones+Laxity: days 1, 10, 11,12,13,20, 21, 22, 23	KT-2000: 133N	5.6 ± 1.34 (days 1)	6.4 ± 1.6 [a] (days 10-13)	7.0 ± 1.7 [a] (days 20-23)
(Karageanes *et al.*, 2000)	26 F non OCs users	High school athletes from 5 sports	15.65 ± 0.98	(26-30 d)	Monthly calendar	8 week period 1 to 7 days between laxity measurements	KT-2000: 89N	Right: 5.0 ± 1.3 Left: 4.5 ± 1.3	Right: 5.2 ± 1.6 Left: 4.4 ± 1.8	Right: 5.1 ± 1.2 Left: 4.6 ± 1.5
(Deie *et al.*, 2002)	16 F 8 M	ND	F/M: (21-23) F: 21.6; M:21.5	(28 ± 4 d)	BBT Serum: E_2 and P_4	8 week period Laxity measurements: 2/3 times a week Serum: 1 time/week	KT-2000: 89N 133N	4.7 ± 0.8 6.4 ± 1.0	5.3 ± 0.7[a]> follicular 6.8 ± 0.9	5.2 ± 0.7[a]> follicular 6.9 ± 1.1[a]> follicular
(Arnold *et al.*, 2002)	49 F non OCs users 11 F OCs users 5 M	Team sport Athletes and non-athletes	19.3 (18-25)	ND	Serum: Relaxin	ND (4 consecutive weeks)	KT-1000: 67N; 89N and maximum force(?)	F (range for the sample average)= 6.3 ± 1.6 to 7.1 ± 2.7		
(Van Lunen *et al.*, 2003)	12 F non OCs users	Mild-moderate active	24.3 ± 4.9	(28-35 d)	Urine LH self test, Serum:Total Estrogens E_2; P_4; T LH; FSH	Hormones+laxity: day 1, near ovulation, day 23	KT-2000: 133N Radiographic measurement	6.0 ± 0.5	6.4 ± 0.4	6.1 ± 0.4
(Lovering & Romani, 2005; Romani *et al.*, 2003)	20 F non OCs users	Recreational athletes	25 ± 5	(28-32 d)	Urine LH self-test Serum:E_2; P_4 ;E_1; SHBG, T	Hormones+laxity: day 1, near ovulation, near day 23	KT-2000 156 N	5.8 ± 1.6[b]	5.7 ± 1.8[b]	6.1 ± 1.7[b]
(Belanger *et al.*, 2004)	18 F non OCs users	Collegiate athletes	20 ± 3	(22-38)	BBT charts	Laxity: almost bi-weekly for 10 weeks, normalised to 28 days	KT-2000 133N	4.6	4.8	4.7
(Carcia *et al.*, 2004)	20F non OCs users	Recreational athletes	20.9 ± 1.6	(28-32 d)	Serum: E_2; P_4; T	Hormones+laxity: day 1, near ovulation, near day 23	KT-2000 133N LigMaster: $\frac{0.18 * BW(N) * tibia(cm)}{tibia(cm) - 10}$	5.3 ± 1.5	5.7 ± 1.9	5.7 ± 1.6
(Shultz *et al.*, 2005; Shultz *et al.*, 2004b)	25; 22; 22 F non OCs users 0/0/20 M	Non-athletes	(18-30) 23±3.5 (F) 23±2 (M)	(28-32)	Serum: E_2; P_4; T	Hormones and laxity: almost daily	KT-2000: 133, 46N and 89N	5.0 to 5.3	5.2 to 5.8	4.2 to 5.3
(Beynnon *et al.*, 2005)	17 F non OCs users, 17 M	ND	F: 21.7 (17-29) M: 24.7	ND	Urine LH self test Serum: E_2; P_4	Hormones+laxity: 5 days: 1-3, 11-13, 20-22, 27-28.	KT-1000 89N	9.1	8.9	Early: 8.7 Late: 8.5

Table 2.6 (cont.) Studies about the influence of sex hormones on anterior knee laxity

Study	Subjects	Sports	Age (mean ± SD or range) (y)	Menstrual cycle length	Menstrual cycle monitoring	Assessment protocol (days/phases)	Device used and force applied	Anterior displacement (mm)		
(Eiling *et al.*, 2006)	11F non OCs users	Netball players	16.3 ± 0.65	ND	Serum: LH; FSH E_2; P_4	Hormones+laxity: day 1, mid-follicular, ovulation, mid-luteal	KT-2000 133N	Day1=4.4 4.6	5.1	4.6
(Hertel *et al.*, 2006)	14 F non OCs users	Collegiate athletes	19.3 ± 1.3	(28-35 d)	Daily urine: E3G; PdG	Laxity: mid-follicular, ovulatory, mid-luteal	KT-1000 133N	4.5 ± 1.7	4.7 ± 1.5	4.2 ± 1.6
(Pollard *et al.*, 2006)	12 F non OCs users 12 M	Moderate active	24.8 ± 7.8(F) 24.3 ± 4.0(M)	(27-31 d)	Serum: Estrogen	Hormones+laxity: days 1, 10, 12; 7 and 9 pos-ovulation	KT-1000 89 N	4.3	4.3	4.3
(Hicks-Little *et al.*, 2007)	28 F non OCs users 25 F OCs users	Female student-athletes	(18-24)	(28-30 d)	NA	Laxity: days 1, 13; 23	KT-1000	No_OCs= 5 ± 2 OCs= 5 ± 2	No_OCs= 6 ± 2 OCs= 6 ± 2	No_OCs= 6 ± 2 OCs= 6 ± 2

[a] Significant increase in the anterior knee laxity ($p<0.05$).
[b] Data adapted from the revision work of Zazulak *et al.* (2006).
F= females; **M**= males (control groups); **ND** = not defined; **NA** = not applicable; E3G=Estrone-3-glucuronide; PdG=Pregnanediol-3-glucoronide.

2.3.5.4 Anterior knee laxity and ACL injury risk

The implications of short-term knee laxity variations on the ACL injury risk remains unclear. Considering the reviewed studies, some congruence seems to exist between the increased knee laxity around ovulation and an increased rate of ACL injury. Although the knee laxity was found to decrease in the early follicular phase contrasting with an increased rate of ACL, and in the luteal phase the knee laxity increased but no increased rate of ACL injuries were found (picture 2.14).

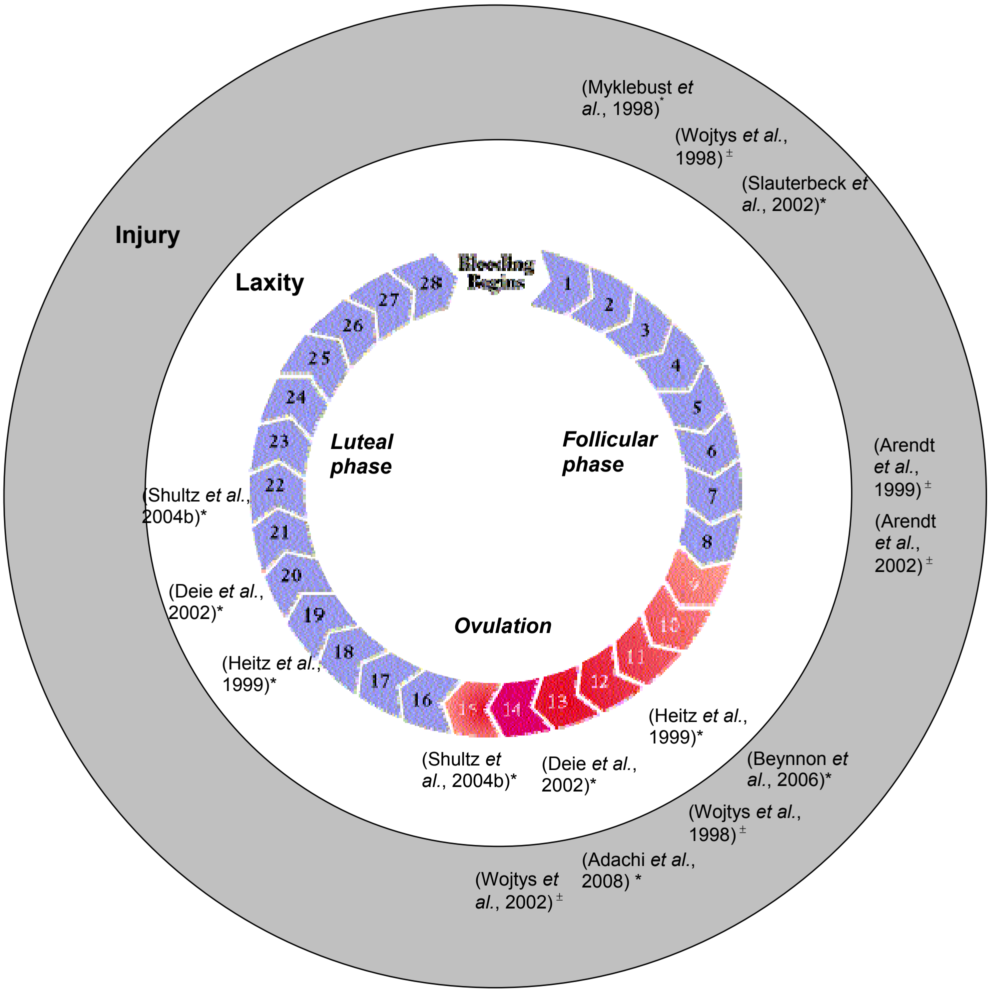


Figure 2.14 Time location of major ACL injury rate and major knee laxity (adapted from Zazulak *et al.*, 2006). *Indicates statistically significant differences (usually at a 5%level); ± indicates trend ($p_c \gtrsim 5\%$).

2.3.5.5 The effect of OCs on knee laxity variation

To our knowledge only one study investigated the long-term effects of OCs on knee laxity (Martineau *et al.*, 2004). Seventy-eight female athletes, from several individual and team sports, were divided into two groups: 42 OC users (average age 20.4 ± 1.4 years) and 36 non OC users (mean age 20.4 ± 1.4 years). They were screened for knee laxity differences. The athletes were excluded if self-reported menstrual irregularities (more than 3 days variation on menstrual cycle duration) or if menstrual dysfunctions existed (amenorrhea and oligomenorrhea). At both 67N and 89N antero-posterior knee loads, significant statistical differences were found between these two groups. At 89N the average difference between the groups was ≈1 mm (displacement for OC users = 3.9 ± 1.1 mm; for non-OC users = 4.8 ± 1.8 mm). In contrast, no mean differences were found in the ligament compliance index[20] between the groups. The authors also documented self-reported menstrual day/phase on which the non-OC users were tested; still they did not take this into account, which could have masked the results.

2.3.5.6 Knee laxity and pregnancy

To further investigate if knee laxity is mediated by sex hormones some authors looked for an estradiol or relaxin effect on knee laxity during pregnancy (Charlton *et al.*, 2001; Schauberger *et al.*, 1996).

Relaxin[21] is the so-called hormone of pregnancy. It was first identified in 1926[22] and its name comes from an observed effect of relaxation and elongation of the interpubic ligament on guinea pigs (Bani, 1997). The administration of relaxin to non-pregnant rats has showed to induce increases in length and weight of the interpubic tissue, along with a great decrease on collagen content (Samuel *et al.*, 1998).

The ability of relaxin to induce collagen turnover in reproductive tissues seems to be extensive to non-reproductive tissues (van der Westhuizen *et al.*, 2008). For instance, it has been implicated in other organs and systems, including the blood vessels, the heart and the brain (Nistri *et al.*, 2007).

During pregnancy, serum levels of relaxin significantly increase. Twenty five days after the LH peak it can reach 800 pg/mL (Stewart *et al.*, 1990). Its main source is known to be the *corpus luteum* of pregnancy (Bryant-Greenwood & Schwabe, 1994). In non conceptive

[20] Compliance index = displacement at 89N – displacement at 67N
[21] Polypeptide hormone, structurally similar to insulin (Bathgate *et al.*, 2003)
[22] Several years had to pass to more deeply investigate its role, because its unavailability in purified forms and difficulties to bioassay relaxin in blood (Bani, 1997).

cycles relaxin levels rise (≈50 pg/mL) in the late luteal phase, beginning 6-9 days after LH peak and declining with the onset of menses (Stewart *et al.*, 1990).

Binding sites for relaxin have been found in female ACL (Faryniarz *et al.*, 2006; Dragoo *et al.*, 2003; Galey *et al.*, 2003) supporting a potential effect on knee joint behavior, during pregnancy and also in the luteal phase of non conceptive cycles.

Schauberger *et al.* (1996) tracked 21 women during pregnancy (on the 1st, 2nd and 3rd trimesters) and post partum (2 and 3 weeks post partum). Within each testing session several joint laxity tests were performed, including the most commonly used methods, and blood was collected for assessment of relaxin levels. Apparently, the laxity progressively increased until the 1st week after partum, although there were no measures during the time period between the 3rd trimester and partum. Looking to relaxin behaviour, it's seemed to have peaked on the 1st trimester and progressively declined (picture 2.15). The authors found no correlation between these two variables during the studied course of time, although no delayed response was investigated and the estrogen levels were not measured.

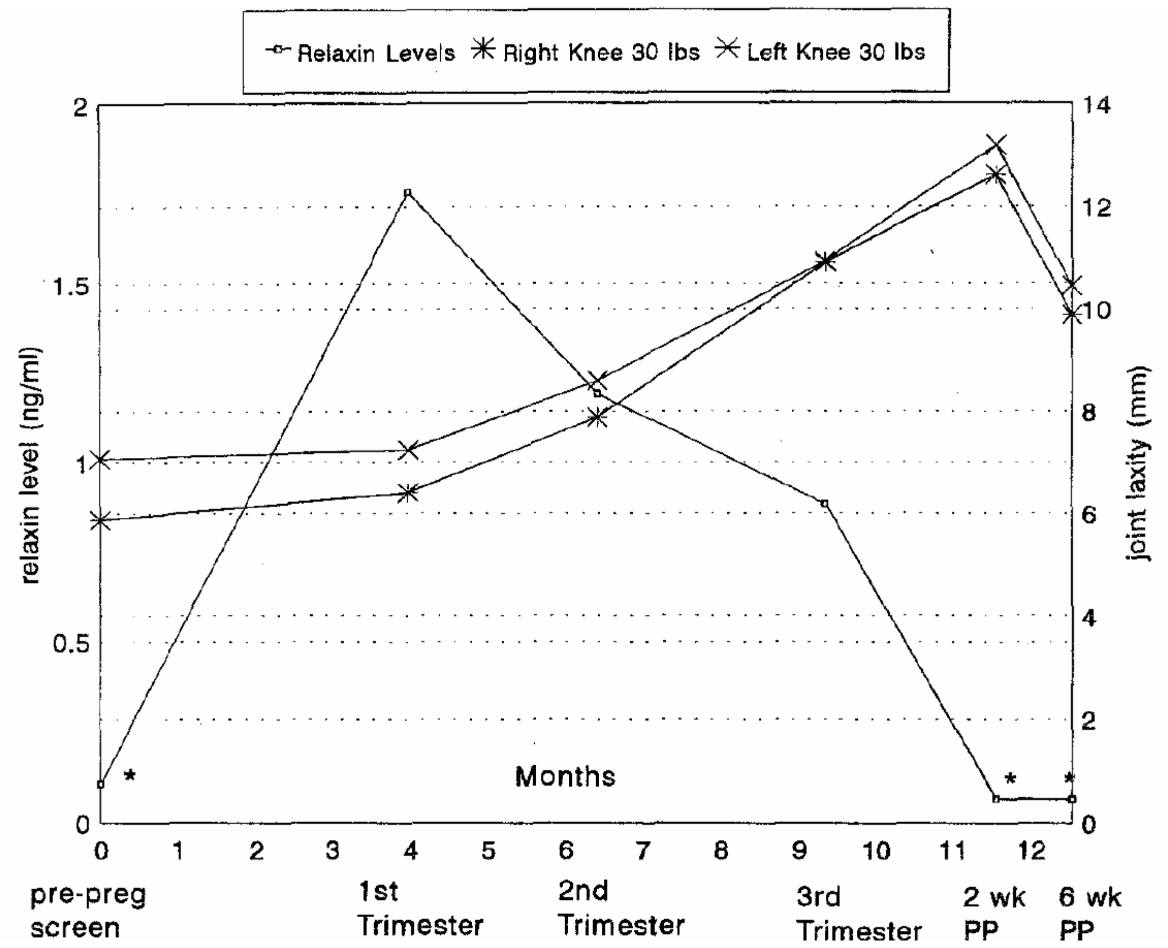


Figure 2.15 Relaxin and joint laxity in pregnant women (Schauberger *et al.*, 1996), p. 669

The study from Charlton *et al.* (2001) compared the knee laxity, from 20 women, between the 3rd trimester of pregnancy and 6 weeks postpartum with estrogen serum levels. The average serum estrogen content decreased by a factor of 200 from the 1st examination to the 2nd trimester (10.755 ng/mL to 50.3pg/mL) and the knee laxity decreased by 2.3mm (range 0 mm-6 mm). Comparing with the previously mentioned study, if just these two time points were assessed, maybe their conclusions would be similar to this study. These two examples stress the danger of making conclusions from scarce data.

Marnach *et al.* (2003) looked for laxity changes just on wrist, using a clinical goniometer, at the 1st, 2nd and 3rd trimesters of pregnancy and around 6 weeks after delivery. Thirty-five pregnant women were screened for laxity and serum levels of cortisol, estradiol, progesterone, and relaxin. An increased laxity was found between the 1st and 3rd trimesters, still not correlated with estradiol, progesterone or relaxin levels. Only with cortisol is there a significant, though faint, correlation (r= 0.18, p=0.03).

2.3.6 ACL injury prevention

In recent years a growing number of studies about ACL injury prevention has been published. In general, there is still a need for more reliable information about ACL injury incidence, injury mechanisms and involved risk factors, in order to develop valuable prevention tools - the 3rd step of van Mechelen's model (van Mechelen *et al.*, 1992). Even though, in the research injury field, several prevention programmes have shown to be effective (Renstrom *et al.*, 2008). The majority of programmes were designed mainly based on the biomechanical and/or external risk factors, attempting to (re)educate the body awareness (proprioception) and to improve neuromuscular control. The main used components include traditional stretching, strengthening, awareness of high-risk positions, technique modifications, aerobic conditioning, sports-specific abilities, proprioceptive and balance training and plyometrics (Renstrom *et al.*, 2008).

Hewett *et al.* (1996) implemented a training program based mainly on lower body plyometric training (incorporating also stretching and weight training) and correction of dynamic movement patterns on landing. The authors demonstrated a positive effect on peak landing forces (decreasing 22%), and on the knee adduction and abduction moments (decreasing ≅ 50%). An increased H:Q ratio was also reported (26% on the nondominant side and 13% on the dominant side), and decreased side-to-side hamstrings strength imbalances (increase hamstring muscle power of 44% on the nondominant side and 21% on dominant side). The vertical jump height increased (≅ 10%). Later the same training protocol was implemented in female players (n= 366) participating in high school soccer, volleyball or basketball teams (Hewett *et al.*, 1999). The incidence of knee injury was compared with a female control group (n= 362) and a male control group (n= 434). The protocol was implemented in a 6-week pre-season period, lasting 60 to 90 minutes, 3 days a week and on alternative days. The results showed a decreased incidence of knee injuries (including noncontact ACL injuries) in the intervened group in comparison to the female control group, and not significantly different from the male control group.

In a prospective controlled study during three soccer seasons, Caraffa *et al.* (1996) examined the effects of progressive balance board training (wobble-boards), 20 min. per day. The authors reported a reduction of ACL injury incidence in the group (300 players) in comparison to the controls (300 players). Also, Wedderkopp *et al.* (1999) investigated the effect of 10 to 15 min. balance training with ankle discs in all training sessions and a not specified functional strength training during warm-up on the injury incidence in young female handball players. The authors reported a decreased number of both traumatic and overuse injuries in the intervention group (111 players) in comparison to the control group (126 players). On the contrary, Soderman *et al.* (2000) examined the effect of a similar balance board training in female soccer players and no injury differences (including ACL) were found between the intervention (62 players) and control groups (78 players).

Myklebust *et al.* (2003a) designed a more comprehensive and dynamic neuromuscular training protocol for ACL injury prevention in female team handball. The authors attempted to improve awareness and body and knee control in standing, jumping, landing and cutting movements. Some preventive technical details were introduced, including knee and hip flexion, two feet-landing, soft landing and lower limb alignment. The intervention protocol comprised of a five-step progression program, three times a week during the first 5-7 weeks of the season, and once a week during the remainder of the season (15min. per session during the warm-up). There were three main types of exercises: on floor, wobble boards and balance mats. In the second season of intervention, the authors introduced same changes in the exercises, to be more handball-specific and challenging, based on players and coaches feedback. The authors didn't find a reduction of ACL injuries (only a trend) in the seasons of intervention (2^{nd} season – 855 players; 3^{rd} 850 players) compared with the control one (1^{st} season, 942 players). Later, Olsen *et al.* (2005) applied a similar protocol, adding some hamstring strength training (*Nordic hamstring lowers),* in young female and male handball players (15-17 years). They reported a decreased number of acute injuries to the knee and ankle in the intervention group (808 female players and 150 male players) compared with the controls (778 female players and 101 male players). On the contrary, Petersen *et al.*. (2005) didn't find a decreased number of acute ankle and knee injuries (including ACL injuries) using a similar prevention protocol. They developed a prospective controlled study with female handball players (intervention group of 134 players and control group of 142 players) and used different balance and jumping exercises combined with some throwing exercises to improve motivation and difficulty.

In these last handball studies it is evident that there is a growing concerning to develop more handball specific motivating exercises (*e.g.* with the ball). Some authors stressed the need to improve the athlete's and coach's compliance to the programs (Myklebust *et al.*, 2003a).

More recently, Pánics *et al.* (2008) found improvements in joint position sensing in elite handball players after an intervention program adapted from Olsen *et al.* (2005). Also Zedis *et al.* (2008) investigated the effects of neuromuscular training, in female soccer (12) and handball (8) players. The authors instructed the players to train 20 min. per day, two times per week, in a six level progression scale, as an adaptation of the program originally proposed by Myklebust *et al.* (2003a). After one season of implementation, the authors found that side-cutting manoeuvres had increased electromyographic activity of the semi-tendinosus during the pre-landing and landing phases, likely decreasing the risk of dynamic valgus.

A few questions remain unanswered.

- Are the prevention programs easy to adopt by the teams? Some take 3 full sessions per week (Hewett *et al.*, 1999) and have very low athlete's compliance (Myklebust *et al.*, 2003a; Soderman *et al.*, 2000).
- Why a few trivial exercises done in the first few weeks have season long effects and others with several seasons long interventions don't? Individual sports knowledge tells us that no conditional parameter will last a season if not trained regularly (*e.g.* reactive force, resistance, velocity, technique, *etc.*).
- Does the team performance change with the introduction of these programs? If performance decreases this could also change the game exposition and the injury risk, besides making adoption improbable.

In all cases, the ACL-preventive efforts are based on adding or correcting something during training. The exercises are too synthetic, performed at very low speed and not sport specific, being very far from real injury game situations, which are decision-making based. They seem much better suited to an early age adoption.

2.4 Menstrual cycle physiology

The main function of the menstrual cycle is reproduction (Pfeifer & Patrizio, 2002). The reproductive function is controlled by a complex system, which mechanism includes feedback interaction between hypothalamus, pituitary and ovaries (Borges *et al.*, 2007; Redman *et al.*, 2003).

Typically, the female reproductive cycle occurs, in healthy women, on a monthly basis, between puberty and menopause. The cycle can be divided into a *follicular* phase (also called *proliferative* or *regenerative)* and a *luteal* phase (also called *progestational* or *secretory*), separated by the ovulation event (figure 2.16). This refers to the rhythmic changes (mainly) in the reproductive organs, under the influences of hormones produced by the endocrine system (Wells, 1991). Women show considerable variation in the lengths of their menstrual cycles, but 28 days ($\pm$7 days) are generally taken as representative of the average ovulatory cycle, thus occurring 10 to 13 times a year. This *standard* is referred to as *eumenorrhea* (Putukian, 1998).

Conventionally, the onset of menstrual flow is used to mark the beginning of the cycle, so the first day of bleeding is called "Cycle Day one"[23]. The normal menstrual flow (or endometrial regression) has a typical length between 3 to 7 days. During this time, the early follicular phase, the estradiol and progesterone are at a low level (Lebrun & Rumball, 2001). The endocrine process leading to follicular grows and development starts earlier than the onset of menses (Miro & Aspinall, 2005). The hypothalamus[24] secretes a *gonadothropin-realising hormone* (GnRH) which stimulates the pituitary to secrete the *follicle stimulating hormone* (FSH) and the *luteinizing hormone* (LH) (Ashley *et al.*, 2000). The first hormonal fluctuation in the blood is the FSH and a small amount of LH, which cause the selection and growth of a new cohort of ovarian follicles (Miro & Aspinall, 2005; Rose *et al.*, 2000). The initial FSH rise occurs 4 days before menses, being related with the fall in the estrogen (E_2) levels, and often defined as luteal-folicular transition (Miro & Aspinall, 2005). This hormonal interaction is a negative feedback system of the hypothalamic-pituitary-ovarian axis (Wells, 1991).

[23] Menstrual cycle is counted from the beginning of menstruation or menses (common usage refers to *period*).
[24] A gland responsible for regulating the body's thirst, hunger, sleep patterns, libido and endocrine functions.

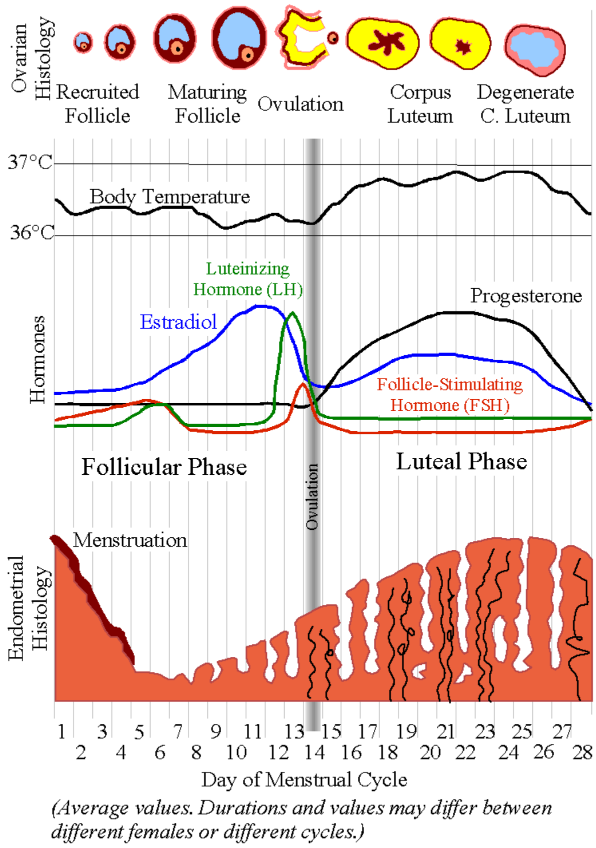


Figure 2.16 Menstrual cycle (http://upload.wikimedia.org/wikipedia/commons/c/cd/MenstrualCycle2.png)

As the follicles grow and mature, over a period of about seven days, the estrogen levels in the bloodstream increase. When the estrogen level reaches a certain point it causes the hypothalamus to release *leutenizing hormone releasing factor* and causing the pituitary to release a large amount of LH. This surge of LH stimulates the single most mature follicle to burst and release an egg. The *follicular* phase gives way to ovulation. After ovulation, the follicle from which the oocyte is expelled becomes the *corpus luteum* (yellow body). As it heals, it produces the hormones estrogen and, in larger amounts, progesterone (P_4). Progesterone is necessary for the maintenance of a pregnancy, through the endometrium preparation for the fertilized ovum to develop (Lebrun & Rumball, 2001). If fertilization and gestation do not occur, the corpus luteum degenerates and the follicle turns white (the corpus albicans). Then declining hormonal levels cause the onset of menstrual flow and the next cycle begins (Lebrun, 1994).

2.4.1 Monitoring the menstrual cycle

Currently, there are several methods available to monitor the hypothalamic-pituitary-ovarian (HPO) axis function. The simplest of these methods (indirect) are usually used to determine the woman fertility status and, the more complex and rigorous, clinically used to diagnose the mechanism of ovulation (Ecochard *et al.*, 2001). The indirect methods are based on dosage of sex hormones in blood, urine or saliva, a log of body basal temperatures (BBT) for nadir determination, analysis of cervical mucus or endometrial histology. Direct methods involve observation of follicle growth and rupture by laparoscopy or high-resolution transvaginal ultrasound examination (Pearlstone & Surrey, 1994; Queenan *et al.*, 1980). These two methods are expensive, with high technical demands, somewhat invasive and, as the others, require daily monitoring (Guermandi *et al.*, 2001).

A simpler, more economic and noninvasive technique is commonly referred to as *natural family planning* (NFP). The main idea is to recognize the "window of fertility", which is the fertile phase of menstrual cycle, in order to schedule intercourse to achieve or avoid a pregnancy. Daily records of natural biomarkers of ovulation, specifically basal body temperature (BBT) and/or cervical mucus are the most commonly used techniques, requiring minimal or no technology.

The *calendar rhythmic method* is based on a mathematical calculation that determines the fertile days considering a certain range of menstrual durations. These methods are based on the assumption that ovulation takes place in the middle of the cycle, and/or occur 14 days prior the subsequent menses (Zinaman, 2006). Firstly, these methods just provide information about the *fertility window*, and are not suitable for estimating the day of ovulation. As demonstrated by some studies, not all women ovulate precisely 14 days before next menses (Wilcox *et al.*, 2000; Baird *et al.*, 1995; Lenton *et al.*, 1984). In a study developed by Wilcox *et al.* (2000), from a sample of 69 cycles with a length of 28 days, only in 10% the ovulation took place exactly 14 days before the onset of menses. In the same study only about 30% of 221 healthy women observed showed their fertile window entirely within the 10 and 17 days guidelines.

The *billings* (or *Ovulation Method*) and *symptothermal* methods are most frequent methods used in the NFP. The *billings method* was termed by the World Health Organization in 1978. Mainly this method is based on the cervical-mucus symptom[25]. The beginning of the fertile period generally corresponds to a significant rise in estrogen levels, which cause a cervical response (Billings, 1991), observed in secretion of estrogenic cervical mucus and characteristics changes in vaginal discharge (Billings *et al.*, 1972).

[25] Fertility is recognized by the development of a particular type of mucus from crypts of the cervix. The Peak day (the last day of lubricative sensation) occurs very close to the time of ovulation. There are several rules to note the changes in sensation at the vulva and the appearance of any mucus discharge seen, which must be noted on a daily chart (Sinai *et al.*, 1999).

Many devices have been developed for the monitoring of the menstrual cycle by the woman herself. Reliability data on most of these methods and systems is still missing[26]. Guida *et al.* (1999) compared different ovulation detection methods used in natural family planning with pelvic ultrasonography diagnosis of ovulation. For this purpose, they selected forty healthy women who were instructed to use the natural family planning procedures correctly and simultaneously motivated. From a total of 148 cycles analyzed, the mucus characteristics and sensation showed high correlation with ultrasonography diagnosis of ovulation.

The *symptothermal* method (also called the *mucothermal* method) is a combination of two methods: the temperature method and cervical-mucus method. The observation of thermogenic shifts, via basal body temperature, is useful in confirming if ovulation may have occurred, and in documentation of luteal phase lengths (Prior *et al.*, 1990), but fails to observe ovarian hormone concentrations, like all the methods referred to above. The rise in progesterone output at ovulation causes a rise in basal body temperature of approximately 0.3 degrees celsius. However, the rise in temperature in relation to the changes in progesterone fluctuation is very variable, so the timing of ovulation by the temperature shift can have a margin of error of -1 to 4 days. Martinez *et al.* (1992) found that the thermal nadir occurred within 1 day of urinary LH surge in 75% of the cases and in 90% of a 2 days window. In contrast, Guida *et al.* (1999) studied 148 cycles and concluded that the body temperature measurements detected ovulation one day before the transvaginal ultrasonography method in 32% of the cases. The information provided by the temperature method is retrospective and is of no value in predicting ovulation. Also, Guermandi *et al.* (2001) concluded that the BBT chart was a poor method for predicting or confirming ovulation, because the nadir may occur between 6 days before and 4 days after ovulation determined by transvaginal ecography.

The salivary ferning test is reported as another method mainly for home ovulation monitoring, particularly developed to aid in assessment of fertile and infertile days (Barbato *et al.*, 1993). This method is based on the observation of salivary ramification, after dried unstained samples of saliva crystallize. For this purpose there are many different optical microscopes with different formats (more or less portable) and different resolutions. Guida *el at.* (1999) concluded that the salivary ferning test is not an accurate method for self-determination of ovulation; because, even if the ferning patterns correlated well with the test results of ultrasonography, there was a high percentage (58.7%) of results considered uninterpretable and therefore excluded from analysis.

[26] Today their diffusion and publicity is enormous, particularly in the internet. There are centres working in advising and teaching women about these methods; so there is an urgent need to clearly establish their effectiveness.

Among the hormonal methods to monitor ovarian function, the daily assessment of LH levels in urine has been extensively used (Guida *et al.*, 1999). In epidemiological studies, daily collection of blood samples is not sustainable for a long period of time as opposed to urine sampling (Li *et al.*, 2002). Furthermore, the changes in the urine hormone metabolites seem to accurately reflect daily fluctuation of hormones circulating in the blood (Munro *et al.*, 1991).

The mid-cycle peak of LH in serum has been frequently used in clinical studies to estimate the day of ovulation, when daily samples are collected (Li *et al.*, 2002). Garcia *et al.* (1981) estimated ovulation based on clinical parameters, steroid hormone values and follicle development evaluation by ultrasonography. The authors observed that ovulation occurs approximately 5 to 15 hours after the LH peak, and the variability in this peak level seems to indicate that there is a relatively low specific LH value necessary for ovulation.

The LH levels can be easily determined by the women themselves, using a stick system. P. B. Miller and Soules (1996) conducted a study to assess the predictive value of in-home urine LH monitoring kits on the time of ovulation. The authors concluded that a positive urine LH testing, if performed every evening to increase reliability, predicts that ovulation will occur within 24 to 48 hours. Guida *el at.* (1999) evaluated the efficacy of different ovulation detection methods in comparison with pelvic ultrasonography. They found no differences between ovulation timings ascertained by urinary LH self tests and by ultrasonography.

Luciano *et al.* (1990) found a high correlation between self LH testing, serum LH peak and ovulation in all the cycles. The mid-cycle serum LH surge starts in the morning between 5 and 9 AM (Seibel *et al.*, 1982), but the LH testing in evening urine is more accurate for this purpose in comparison with morning urine (Luciano *et al.*, 1990). Although, the possibility for false-negative results can not be excluded (Guermandi *et al.*, 2001).

Other biomarkers have been proposed and used to estimate the day of ovulation: FSH peak, estrogen peak and the ratio of estrogen and progesterone (day of luteal transition).

Li *el al*. (2002) measured the daily hormonal changes of LH, FSH and E_2 in blood and/or urine and used transvaginal ultrasonography to detect follicular collapse. The authors observed that the urinary FSH peak occurred within 1 day of follicular collapse in 97% of the cycles, still not as good as the methods of urinary LH, serum FSH or the method of Waller *et al.* (1998).

2.4.2 Menstrual cycle disturbances in Athletes

Prolonged, vigorous exercise training may result in several favourable physiological adaptations (Williams *et al.*, 2001). However, the female reproductive system seems to be sensitive to the physiological stress (Warren & Perlroth, 2001), as several exercise-associated perturbations demonstrate. This subject was only investigated intensively when it was reported that progressive skeletal demineralization can occur in hypoestrogenic amenorrheic athletes (Drinkwater *et al.*, 1984; Malina *et al.*, 1978). Research into the effect of exercise on menstrual function has been done in order to determine the incidence, clinical consequences and mechanisms of these disorders (Redman & Loucks, 2005).

Exercise induced changes in the menstrual cycle can manifest as physiological adaptations or endocrine dysfunctions. They can range from subtle perturbations resulting from transitory endocrine response to exercise to the most severe abnormalities, including disruption of follicular development, luteal phase deficiency (LPD), anovulation and amenorrhea. LPD and anovulation are probably the most frequent menstrual abnormalities associated with exercise, although frequently unreported due to their asymptomatic nature. LPD can occur without any perceived change in menstrual cycle length, because follicular phase is lengthened and the luteal phase is shortened (De Souza *et al.*, 1998). LPD is related with abnormal *corpus luteal* function, which includes reduced luteal phase length (≤ 10 days), with or without inadequate progesterone production (De Souza, 2003; Ginsburg, 1992). Women with LPD continue to ovulate, but frequently this event occurs later than the conventional mid-cycle days 12-14 (De Souza & Williams, 2004). In addition, a longer follicular phase is connected with decreased E_2 exposure between days 2-12 and in the luteal phase (De Souza *et al.*, 1998; Winters *et al.*, 1996).

Studies have documented a much higher prevalence of LPD and anovulation in athletes than in non-active women (De Souza *et al.*, 1998). De Souza *et al.* (1998) examined the characteristics of three consecutive menstrual cycles in sedentary and moderately exercising women, regularly menstruating, and reported significant menstrual cycle differences between the groups. The group of non-exercising women showed consistent menstrual cycles, with 91% ovulatory and 9% exhibiting LPD. The exercising group exhibits inconsistent menstrual cycle characteristics in 46% of women, with LPD (short and/or inadequate luteal phase) in 42% and anovulation in 16% of women.

The amenorrhea is the most severe abnormality observed in exercising women (De Souza *et al.*, 2003). The prevalence of amenorrhea in this athletic population has been reported to occur in the range of 1%-46% (Loucks, 1990). However, the studies are mainly focused in endurance type sports (such as running) and sports in which leanness and/or low body weight are favourable traits to performance.

Typically, the athlete develops functional hypothalamic amenorrhea (FHA) with no evidence of organic cause (Marcus *et al.*, 2001). Amenorrhea is divided into two subcategories. Primary amenorrhea means absence of menstruation by the age of 16 in a girl with secondary sex characteristics (Ireland & Ott, 2004) and therefore the menarche is considered to be delayed. Secondary amenorrhea is used to define the cessation of menstruation for three or more consecutive menstrual cycles, after menarche occurrence (Waldrop, 2005). Oligomenorrhea refers to irregular and inconsistent menstrual cycles lasting 36 to 90 days (Loucks & Horvath, 1985). Because in oligomenorrhoeic cycles the ovulation may either occur or be absent (De Souza & Williams, 2004) the hormonal profile is difficult to predict, though a low E_2 pattern is expected to occur. There is also a lack of reports and detailed descriptions on oligomenorrhoeic cycles in the literature (De Souza & Williams, 2004).

2.4.2.1 Aetiology of exercise-associated disturbances in athletes

The aetiology of exercise-associated menstrual disturbances has not been clearly established. Several stressors of the neuroendocrine control of the reproduction system have been identified, particularly in the case of FHA (Genazzani *et al.*, 2006). The related stress factors have been mostly associated to conditions such as dieting, exercise or intense emotional events (Genazzani *et al.*, 2006).

The FHA is mainly thought to be an insufficient hypothalamic GnRH generation, leading to a decreased pituitary secretion of gonadotropins (LH and FSH) which consequently compromises ovulation (Marcus *et al.*, 2001). The mechanism of disruption in GnRH release from the hypothalamus remains to be elucidated (Perkins *et al.*, 1999). Evidence supports the idea that the stress induces change in the central neural functions that result in increased hypothalamic-pituitary-adrenal (HPA) activity and therefore GnRH release is inhibited (Berga *et al.*, 1997). This mechanism has been linked to hypercortisolemia because corticotrophin-releasing hormones (CRH) increase cortisol production and both suppress GnRH release, suggesting that FHA is stress-induced (Berga *et al.*, 2000; Berga *et al.*, 1997). Moreover, several studies have documented high cortisol production in hypothalamic amenorrhea (Berga *et al.*, 1997; Biller *et al.*, 1990) suggesting that an increased adrenocorticotropic hormone (ACTH) secretion and/or increased adrenal sensitivity to ACTH are a possible precursors (Genazzani *et al.*, 2001). An increased adrenal steroid secretion was also found in response to CHR factor administration, as showed in blunted cortisol and ACTH concentrations (Genazzani *et al.*, 2001; Meczekalski *et al.*, 2000). However, CRH levels in cerebrospinal fluid were reported to be similar between women with FHA and with eumenorrhea and therefore not directly connected with increased cortisol and decreased GnRH (Berga *et al.*, 2000).

Many studies are being done to support the idea of negative energy balance or deficiency in energy intake, as a likely cause of exercise induced menstrual disturbances, including FHA (Loucks *et al.*, 1998). Several experiments by Loucks, conveniently reviewed in a single paper (De Souza & Williams, 2004), manipulated energy intake and energy expenditure and provided evidence of a straight relation between energy availability and GnRH pulsatility. This hypothesis supposes that the pulsating release of GnRH is disrupted when energy availability is not adequate for needs of both reproduction and exercise-demands (Loucks *et al.*, 1998). However, the critical signal or signals that inform the neural circuits of the available metabolic fuel remain unclear.

This hypothesis grew stronger with the positive results of prevention or reversion of menstrual disorders in response to an increase in dietary energy intake without changing the exercise demands (Williams *et al.*, 2001; Williams *et al.*, 1995). Moreover, Williams *et al.* (2001) emphasizes the energy modulation of the induction and reversing of amenorrhea in a primate model. Specifically, the authors demonstrate that the induction of amenorrhea was a product of the volume of calories used during training, which decreased the energy available for reproduction and other necessary metabolic functions. Additional evidence was provided from changes in circulating total triiodothyronine (T_3) (a marker of energy balance) which correlated well with either induction or reversal of amenorrhoea. The energy deficit is also associated to less severe menstrual disruptions such as LPD and anovulation (De Souza, 2003).

Initially, a critical link was proposed between body weight and/or body fat and the reproductive function (Frisch & McArthur, 1974; Frisch & Revelle, 1970). This hypothesis was later argued; because short-term studies in humans and other primates showed that the slowing of pulsatile LH secretion was unrelated to loss of body weight, in an experiment food-deprivation (Cameron & Nosbisch, 1991; Cameron *et al.*, 1991). In addition, no significant differences in cortisol concentration were noticed in a state of feeding or fasting in male subjects suggesting that the activation of the adrenal axis is an unlikely cause of reproductive axis disruption (Cameron *et al.*, 1991). Also a lengthy study using female monkeys as models documented an abrupt transition from normal menstrual cyclicity to an amenorrheic state after increased strenuous exercise, with no significant changes in body weight, although food intake remained invariable (Williams *et al.*, 2001).

While the studies have provided evidence emphasizing the role of low energy availability in the development of exercise-associated disturbances, the mechanism by which a hypometabolic state regulates GnRH secretion remains to be understood. Several different indicators (metabolic hormones and substrates) are advanced as potential key signals to the brain of changes in energy balance such as Grelin, T_3, Leptin, Insulin, growth hormone, IGF-1/IGFBP-1, Cortisol and Glucose (see Wade *et al.* (1996) for review). The evidence that neural mechanisms control pulsatile LH release is strong. The fast LH pulse responses to fuel deprivation or refeeding emphasizes that, whatever the metabolic cues are, they must be very fast.

The relation between exercise and menstrual disturbances defy any simple model. In sports training and competition the three factors linked to menstrual disruption, metabolic, physical and psychological, may be simultaneously present. The ever-changing demands of competition differ significantly between different sports and even within the same sport. So do the training processes. The inter-individual variability response to sports practices and other conditions is an additional factor. Bouchard and Tremblay (1997), on studies with twins, concluded that there are individual genetic differences in susceptibility to overfeeding and/or in response to negative energy balance. Intrapair similar responses were observed relative to body mass, body composition and body fat distribution. Also Williams *et al.* (2001) reported a high interindividual variability among monkeys in the development of exercise–induced reproductive dysfunctions.

Other studies (Williams *et al.*, 2001; Schneider & Wade, 1989) have looked for the relation between the initial energy stores and the susceptibility to reproductive function suppression in response to food deprivation. The data from these studies is not convergent. For instance, lean hamsters revealed to be more sensitive to starvation-induced disturbances than fat hamsters (Schneider & Wade, 1989). However, Williams *et al.* (2001) observed that the time course between the training changes and the development of amenorrhoea wasn't dependent on the initial body weight.

In our understanding most of these results must be viewed with care because athletes are not the normal sedentary female population. Most of them were submitted to years of training and were particularly selected because of evident favourable traits for a specific sport. Forsberg and Lock (2006) underlined the need to understand the relation between traits of perfectionism and the development of eating disorders in athletes, and therefore we may suspect that associations between menstrual dysfunctions and the level of commitment with the sport could exist.

2.4.2.2. Detection of exercise-associated menstrual disturbances

The majority of the studies have used questionnaires and self-reporting data to evaluate the menstrual cycle status, length and regularity in female athletes (De Souza & Williams, 2004). Significant suspicions are attributed to this recall and self-reporting process. The use of other techniques, including the dosage of hormonal contents in blood, urine and saliva are important in objectively characterize the menstrual cycle profile of each athlete.

Inconsistent menstrual cycle characteristics were reported between consecutive cycles in exercising women leading to conclude that the menstrual cycle length is not an accurate marker of ovarian function in athletes (De Souza *et al.*, 1998). As monitoring just an isolated menstrual cycle in this population may underestimate the prevalence of menstrual disturbances, some have recommended the use of at least three consecutive cycles for a more accurate assessment of menstrual cycle profile (McNeely & Soules, 1988).

Self reported menstrual cycle duration is usually used as indication for assessing regularity or irregularity in athletes, in comparison to commonly accepted ranges (De Souza & Williams, 2004). There is no agreement in the range of days for a *normal* menstrual cycle length (De Souza & Williams, 2004), although in studies on exercising women the range of 26-32 days is frequently adopted (De Souza *et al.*, 1998; Loucks *et al.*, 1989).

To clearly perceive the menstrual cycle status in athletes and associated hormonal disturbances, daily hormonal measurements should be pursued, such as E_2, progesterone and LH (De Souza & Williams, 2004). For this purpose, daily urine samples are less invasive than daily blood sampling.

The luteal phase is classified as short by comparison with a vague *standard*, ranging from 9 to 11 days. As the luteal phase length is the number of days between ovulation and next menses, the difficulty in establishing the ovulation day makes this a usually inaccurate measure. The methods proposed for assessing LP length are not different from the ones reported before. For a sensitive diagnostic of LPD in exercising women, it has been recommended to use progesterone dosage as a primary reference (Jordan *et al.*, 1994). These authors compared the sensitivity and/or specificity of different tests to assess LPD and concluded that BBT charts, standard luteal phase length estimation and preovulatory follicle tests are inappropriate in detecting LPD. They concluded that appropriate methods based in the serum P_4 concentration are, for a single midluteal sample, values below 10ng/mL or, for the sum of three midluteal samples, values below 30ng/mL.

2.4.2.3. The effects of exercise-associated menstrual disturbances on bone health

Benefits of exercise for bone structure and strength

Skeletal tissue responds well to mechanical stress (Birch, 2005). Physical loading potentially has a favourable effect in the process of bone modelling and remodelling (Heinonen *et al.*, 1995). The bone has the capacity of restoring micro-damages resulting from fatigue and shock (Lemaire *et al.*, 2004). Besides, mechanical loading is considered to be essential for maintenance of bone homoeostasis (T. S. Gross & Srinivasan, 2006; Tromp *et al.*, 2006). It is well documented that bone mass is negatively affected in conditions of diminished mechanical loading like space flight (R. T. Turner, 2000), inactivity or immobility (Inoue *et al.*, 2000) and overweight (Hinton *et al.*, 2006; Ensrud *et al.*, 2003).

Bone is a living dynamic tissue, whose structure has the capacity to adapt to change continually during life (Lemaire *et al.*, 2004). Indeed, when the linear growth period of the bones is terminated, this tissue maintains the ability to modify its internal structure through the remodelling process. This process occurs under a complex interaction of specific factors leading to the resorption of old bone by osteoclasts and the replacement with new bone formed by esteoblasts, in a spatially localized process (*op. cit.*).

Mechanical loading associated to sport exerts a potent osteogenic stimulus to bone density, size and shape resulting in increased mechanical bone strength (C. H. Turner & Robling, 2005). Previous studies on asymmetric sports (*e.g.* racquet sports and handball), provided evidences of significant augmented BMD rates in the dominant arm (Vicente-Rodriguez *et al.*, 2004; Kontulainen *et al.*, 1999; Kannus *et al.*, 1995). In another study, Haapasalo *et al.* (2000) documented bone length increase in a male tennis player's dominant playing arm, without changes in volumetric bone density. Moreover, the increase of bone mass by regular tennis practice seems likely to be preserved even when lapses in training occur (Kontulainen *et al.*, 1999).

Certain type, intensity, frequency, and duration of exercises can affect the bone homoeostasis, inducing site-specific adaptations (Bemben *et al.*, 2004). An oestrogenic advantage has been suggested in high-impact sports or weight-bearing physical activities (Torstveit & Sundgot-Borgen, 2005; Bemben *et al.*, 2004). This idea has gained significance because of evident increased bone formation in sports with high strain rates and peak forces (*i.e.* handball, racquet sports and gymnastic) in contrast with sports with systematic low-force repetition (*i.e.*, long distance running) (Vicente-Rodriguez *et al.*, 2004; Heinonen *et al.*, 1995).

Most of the studies on bone mass accrual in girls have been done in gymnastics (Proctor *et al.*, 2002; Nickols-Richardson *et al.*, 2000) where the ground force reactions may rise to peak magnitudes of 10 times the body weight of a young athlete (Daly *et al.*, 1999). Additionally, it seems to be positive to initiate weight-bearing activities at an early age, like in gymnastics, because child gymnasts demonstrated greater BMD gains in comparison with a controlled group of non-gymnasts. Moreover, a higher BMD was found in premenarcheal gymnasts than in controlled subjects of matching weight (Nickols-Richardson *et al.*, 2000). The studies have also reported an increased rate in BMD around puberty in girls (11-15 years) (Bonjour *et al.*, 1991), representing the most favourable time to improve peak bone mass (Bonjour *et al.*, 1994).

The importance of achieved peak bone mass is frequently underlined for later bone strength and hence a lifelong advantage in respect to osteoporoses, particularly in women. Even though, during the skeletal growth period the variance in BMD and body mass content between subjects is largely dependent on genetic factors and environmental factors, such as nutrition and characteristics of exercise. Further details are in the reviews of (Heaney *et al.*, 2000; Soyka *et al.*, 2000).

Menstrual disturbances to bone integrity

Despite the positive effect of exercise on bone strength, a critical bone mass reduction has been found in athletes with exercise-induced FHA (Rencken *et al.*, 1996; Drinkwater *et al.*, 1984). Decreased BMD in female athletes is thought to result from a chronic hypoestrogenemia state, characteristic of amenorrhea. Evidence that low levels of oestrogen exposure are implicated in the pathogenesis of osteoporosis includes premature accelerated bone loss and fragility fracture observed in exercising-amenorrheic athletes (Drinkwater *et al.*, 1984). The effects of oestrogen deficiency are characterized by an increase of bone turnover with disproportionate acceleration of bone resorption comparative to bone formation leading to metabolic bone diseases (Kanno *et al.*, 2004). The mechanisms underlying this phenomenon are still unclear. Besides, synergistic effects of estrogen deficiency, body mass, nutritional factors, mechanical loading characteristics and skeleton localization may occur. For instance, competitive female artistic gymnastics have average high BMD, despite reported absence of menses and inadequate calcium intakes (Kirchner *et al.*, 1995). Also, in the study of Robinson *et al.* (1995), higher bone mass and lumbar spine BMD was found in competitive gymnasts than in runners with similar prevalence of amenorrhea and oligomenorrhea and similar dietary calcium intake, suggesting a sport specific loading-pattern effect. In the same study, BMD assessment on the femoral neck, the lumbar spine and whole body did not differ among the women with amenorrhea and oligomenorrhea or eumenorrhea.

The relative contributions of sex hormones, nutritional deficiency and mechanical loading in bone homeostasis is difficult to establish. Other sex hormones have been also implicated in bone health, mostly progesterone (De Souza *et al.*, 1997; Prior, 1990) and FSH (Iqbal *et al.*, 2006). Both hormones are directly involved in menstrual disruptions in exercising women.

Progesterone has been suggested to promote bone formation and/or increase bone turnover - reviewed in (Prior, 1990). However, in the case of subtle menstrual disorders such as short or inadequate luteal phases, there is no data supporting the idea that decreased progesterone and estrogen levels and/or time-duration affects bone strength in exercising women (De Souza *et al.*, 1997; Waller *et al.*, 1996; Hetland *et al.*, 1993).

It is important to clearly identify the long-term consequences of menstrual disturbances in athletes, particularly if associated with irreversible bone loss. Keen and Drinkwater (1997) followed a group of athletes for 8 years and concluded that, despite several years of consistent menstrual cycles or use of oral contraceptives, the BMD in the vertebral bones remained reduced.

Studies suggest that there are skeletal sites that are more sensitive to oestrogen deficiency than others. The vertebral spine seems to be very susceptive to estrogen deficits because of its greater trabecular component (Keen & Drinkwater, 1997). Data presented by Pettersson *et al.* (1999) revealed that weight was the best predictor for BMD in the spine for both groups of amenorrheic runners and eumenorrheic soccer players. Likewise, in the same study, the authors concluded that the effect of estrogen deprivation is also extended to cortical bone, considering that decreased BMD in tibia diaphysis and femoral diaphysis was found in amenorrheic runners (Pettersson *et al.*, 1999).

Despite all methodological constrains in studying the relation between sex hormonal changes in athletes and bone mass evolution throughout life, low BMD is two to three times more prevalent in non-exercising premenopausal women than in elite athletes (Torstveit & Sundgot-Borgen, 2005).

The effects of oral contraceptive use and bone loss prevention

Substantial interest has emerged regarding the effect of using OCs in reversing menstrual disorders in exercising women and therefore preserving and restoring BMD (De Cree, 1998; Brukner & Bennell, 1997).

The most important effect from the use of OC is to inhibit ovulation, and consequently women do not experience high estrogen levels typical of pre-ovulation and the luteal phase. The OC's content on synthetic estrogen and progesterone have been significantly reduced since OCs were first introduced (1960s), maintaining their effectiveness.

The available data on the effects of prolonged OC usage in girls before archiving skeletal maturity and in the acquisition of peak bone mass is controversial. In a longitudinal study, Lloyd *et al.* (2000) found no effect on peak bone mass in teenage women related to OC use.

In a retrospective and cross-sectional study, Hartard *et al.* (2004) examined the effect of low-dose monophasic OC in BMD in long-distance runners. They observed that OCs were associated with decreased BMD in the spine and the femoral neck; but the major determinant of spine BMD in these athletes was the starting age of OC usage. In another study with long-distance runners, with appropriate dietary intakes, the oligo-amenorrheic displayed a significant lower lumbar spine BMD than eumenorrheic and OC users, and no differences were found in proximal and midshaft femur (Gremion *et al.*, 2001). In a random controlled trial with fifty-five young dancers, where nearly half were amenorrheic, there were no changes in BMD on the amenorrheic dancers' group after two years of hormone therapy (Warren *et al.*, 2003).

Finally, research has not yet produced consistent evidence that hormonal therapy or OC usage increases the bone mass in young and adult women with exercise-related menstrual disturbances.

2.5 References

Aagaard, K. (2007). 7th European Women's Championship Sweden 2006 - Qualitative Trend Analysis [Electronic Version]. *EHF Web Periodical*.

Aagaard, P., Simonsen, E. B., Magnusson, S. P., Larsson, B. & Dyhre-Poulsen, P. (1998). A new concept for isokinetic hamstring: quadriceps muscle strength ratio. *Am J Sports Med, 26*(2), 231-237.

Adachi, N., Nawata, K., Maeta, M. & Kurozawa, Y. (2008). Relationship of the menstrual cycle phase to anterior cruciate ligament injuries in teenaged female athletes. *Arch Orthop Trauma Surg, 128*(5), 473-478.

Agel, J., Bershadsky, B. & Arendt, E. A. (2006). Hormonal therapy: ACL and ankle injury. *Med Sci Sports Exerc, 38*(1), 7-12.

Arendt, E. A. (2001). Anterior cruciate ligament injuries. *Curr Womens Health Rep, 1*(3), 211-217.

Arendt, E. A., Agel, J. & Dick, R. (1999). Anterior Cruciate Ligament Injury Patterns Among Collegiate Men and Women. *J Athl Train, 34*(2), 86-92.

Arendt, E. A., Bershadsky, B. & Agel, J. (2002). Periodicity of noncontact anterior cruciate ligament injuries during the menstrual cycle. *J Gend Specif Med, 5*(2), 19-26.

Arnold, C., Van Bell, C., Rogers, V. & Cooney, T. (2002). The relationship between serum relaxin and knee joint laxity in female athletes. *Orthopedics, 25*(6), 669-673.

Ashley, C. D., Kramer, M. L. & Bishop, P. (2000). Estrogen and substrate metabolism: a review of contradictory research. *Sports Med, 29*(4), 221-227.

Bahr, R. & Krosshaug, T. (2005). Understanding injury mechanisms: a key component of preventing injuries in sport. *Br J Sports Med, 39*(6), 324-329.

Baird, D. D., McConnaughey, D. R., Weinberg, C. R., Musey, P. I., Collins, D. C., Kesner, J. S., et al. (1995). Application of a method for estimating day of ovulation using urinary estrogen and progesterone metabolites. *Epidemiology, 6*(5), 547-550.

Baker, S., O'Niel, B. & Karpf, R. (1992). *The Injury Fact Book*. New York, NY: Oxford University Press.

Bani, D. (1997). Relaxin: a pleiotropic hormone. *Gen Pharmacol, 28*(1), 13-22.

Barbato, M., Pandolfi, A. & Guida, M. (1993). A new diagnostic aid for natural family planning. *Adv Contracept, 9*(4), 335-340.

Bathgate, R. A., Samuel, C. S., Burazin, T. C., Gundlach, A. L. & Tregear, G. W. (2003). Relaxin: new peptides, receptors and novel actions. *Trends Endocrinol Metab, 14*(5), 207-213.

Belanger, M. J., Moore, D. C., Crisco, J. J., 3rd, Fadale, P. D., Hulstyn, M. J. & Ehrlich, M. G. (2004). Knee laxity does not vary with the menstrual cycle, before or after exercise. *Am J Sports Med, 32*(5), 1150-1157.

Bemben, D. A., Buchanan, T. D., Bemben, M. G. & Knehans, A. W. (2004). Influence of type of mechanical loading, menstrual status, and training season on bone density in young women athletes. *J Strength Cond Res, 18*(2), 220-226.

Berga, S. L., Daniels, T. L. & Giles, D. E. (1997). Women with functional hypothalamic amenorrhea but not other forms of anovulation display amplified cortisol concentrations. *Fertil Steril, 67*(6), 1024-1030.

Berga, S. L., Loucks-Daniels, T. L., Adler, L. J., Chrousos, G. P., Cameron, J. L., Matthews, K. A., et al. (2000). Cerebrospinal fluid levels of corticotropin-releasing hormone in women with functional hypothalamic amenorrhea. *Am J Obstet Gynecol, 182*(4), 776-781; discussion 781-774.

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

Beynnon, B. D., Bernstein, I. M., Belisle, A., Brattbakk, B., Devanny, P., Risinger, R., et al. (2005). The effect of estradiol and progesterone on knee and ankle joint laxity. *Am J Sports Med, 33*(9), 1298-1304.
Beynnon, B. D. & Fleming, B. C. (1998). Anterior cruciate ligament strain in-vivo: a review of previous work. *J Biomech, 31*(6), 519-525.
Beynnon, B. D., Howe, J. G., Pope, M. H., Johnson, R. J. & Fleming, B. C. (1992). The measurement of anterior cruciate ligament strain in vivo. *Int Orthop, 16*(1), 1-12.
Beynnon, B. D., Johnson, R. J., Braun, S., Sargent, M., Bernstein, I. M., Skelly, J. M., et al. (2006). The relationship between menstrual cycle phase and anterior cruciate ligament injury: a case-control study of recreational alpine skiers. *Am J Sports Med, 34*(5), 757-764.
Biller, B. M., Federoff, H. J., Koenig, J. I. & Klibanski, A. (1990). Abnormal cortisol secretion and responses to corticotropin-releasing hormone in women with hypothalamic amenorrhea. *J Clin Endocrinol Metab, 70*(2), 311-317.
Billings, E. L. (1991). The simplicity of the Ovulation Method and its application in various circumstances. *Acta Eur Fertil, 22*(1), 33-36.
Billings, E. L., Brown, J. B., Billings, J. J. & Burger, H. G. (1972). Symptoms and hormonal changes accompanying ovulation. *Lancet, 1*(7745), 282-284.
Birch, K. (2005). Female athlete triad. *Bmj, 330*(7485), 244-246.
Boden, B. P., Dean, G. S., Feagin, J. A., Jr. & Garrett, W. E., Jr. (2000). Mechanisms of anterior cruciate ligament injury. *Orthopedics, 23*(6), 573-578.
Bonjour, J. P., Theintz, G., Buchs, B., Slosman, D. & Rizzoli, R. (1991). Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. *J Clin Endocrinol Metab, 73*(3), 555-563.
Bonjour, J. P., Theintz, G., Law, F., Slosman, D. & Rizzoli, R. (1994). Peak bone mass. *Osteoporos Int, 4 Suppl 1*, 7-13.
Borges, L. E., Morgante, G., Musacchio, M. C., Petraglia, F. & De Leo, V. (2007). New protocol of clomiphene citrate treatment in women with hypothalamic amenorrhea. *Gynecol Endocrinol, 23*(6), 343-346.
Bouchard, C. & Tremblay, A. (1997). Genetic influences on the response of body fat and fat distribution to positive and negative energy balances in human identical twins. *J Nutr, 127*(5 Suppl), 943S-947S.
Brooks, J. H. & Fuller, C. W. (2006). The influence of methodological issues on the results and conclusions from epidemiological studies of sports injuries: illustrative examples. *Sports Med, 36*(6), 459-472.
Brukner, P. & Bennell, K. (1997). Stress fractures in female athletes. Diagnosis, management and rehabilitation. *Sports Med, 24*(6), 419-429.
Bryant-Greenwood, G. D. & Schwabe, C. (1994). Human relaxins: chemistry and biology. *Endocr Rev, 15*(1), 5-26.
Cameron, J. L. & Nosbisch, C. (1991). Suppression of pulsatile luteinizing hormone and testosterone secretion during short term food restriction in the adult male rhesus monkey (Macaca mulatta). *Endocrinology, 128*(3), 1532-1540.
Cameron, J. L., Weltzin, T. E., McConaha, C., Helmreich, D. L. & Kaye, W. H. (1991). Slowing of pulsatile luteinizing hormone secretion in men after forty-eight hours of fasting. *J Clin Endocrinol Metab, 73*(1), 35-41.
Caraffa, A., Cerulli, G., Projetti, M., Aisa, G. & Rizzo, A. (1996). Prevention of anterior cruciate ligament injuries in soccer. A prospective controlled study of proprioceptive training. *Knee Surg Sports Traumatol Arthrosc, 4*(1), 19-21.
Carcia, C. R., Shultz, S. J., Granata, K. P., Gansneder, B. M. & Perrin, D. H. (2004). Knee ligament behavior following a controlled loading protocol does not differ by menstrual cycle day. *Clin Biomech (Bristol, Avon), 19*(10), 1048-1054.
Carcia, C. R., Shultz, S. J., Granata, K. P., Perrin, D. H. & Martin, R. L. (2005). Females recruit quadriceps faster than males at multiple knee flexion angles following a weight-bearing rotary perturbation. *Clin J Sport Med, 15*(3), 167-171.

Chandrashekar, N., Mansouri, H., Slauterbeck, J. & Hashemi, J. (2006). Sex-based differences in the tensile properties of the human anterior cruciate ligament. *J Biomech, 39*(16), 2943-2950.

Chappell, J. D., Herman, D. C., Knight, B. S., Kirkendall, D. T., Garrett, W. E. & Yu, B. (2005). Effect of fatigue on knee kinetics and kinematics in stop-jump tasks. *Am J Sports Med, 33*(7), 1022-1029.

Chappell, J. D., Yu, B., Kirkendall, D. T. & Garrett, W. E. (2002). A comparison of knee kinetics between male and female recreational athletes in stop-jump tasks. *Am J Sports Med, 30*(2), 261-267.

Charlton, W. P., Coslett-Charlton, L. M. & Ciccotti, M. G. (2001). Correlation of estradiol in pregnancy and anterior cruciate ligament laxity. *Clin Orthop*(387), 165-170.

Chaudhari, A. M., Hearn, B. K. & Andriacchi, T. P. (2005). Sport-dependent variations in arm position during single-limb landing influence knee loading: implications for anterior cruciate ligament injury. *Am J Sports Med, 33*(6), 824-830.

Conceição, F. A. V. d. (2004). *Estudo biomecânico do salto em comprimento - modelação, simulação e optimização da chamada.* U.P., Porto.

Cowling, E. J. & Steele, J. R. (2001). The effect of upper-limb motion on lower-limb muscle synchrony. Implications for anterior cruciate ligament injury. *J Bone Joint Surg Am, 83-A*(1), 35-41.

Czerwinski, J. (2006). The intensity of the training work of a pivot and wing players in the extra-class Polish men's handball teams [Electronic Version]. *EHF Web Periodical*, 10.

Dallalana, R. J., Brooks, J. H., Kemp, S. P. & Williams, A. M. (2007). The epidemiology of knee injuries in English professional rugby union. *Am J Sports Med, 35*(5), 818-830.

Daly, R. M., Rich, P. A., Klein, R. & Bass, S. (1999). Effects of high-impact exercise on ultrasonic and biochemical indices of skeletal status: A prospective study in young male gymnasts. *J Bone Miner Res, 14*(7), 1222-1230.

De Cree, C. (1998). Sex steroid metabolism and menstrual irregularities in the exercising female. A review. *Sports Med, 25*(6), 369-406.

De Souza, M. J. (2003). Menstrual disturbances in athletes: a focus on luteal phase defects. *Med Sci Sports Exerc, 35*(9), 1553-1563.

De Souza, M. J., Miller, B. E., Loucks, A. B., Luciano, A. A., Pescatello, L. S., Campbell, C. G., et al. (1998). High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *J Clin Endocrinol Metab, 83*(12), 4220-4232.

De Souza, M. J., Miller, B. E., Sequenzia, L. C., Luciano, A. A., Ulreich, S., Stier, S., et al. (1997). Bone health is not affected by luteal phase abnormalities and decreased ovarian progesterone production in female runners. *J Clin Endocrinol Metab, 82*(9), 2867-2876.

De Souza, M. J., Van Heest, J., Demers, L. M. & Lasley, B. L. (2003). Luteal phase deficiency in recreational runners: evidence for a hypometabolic state. *J Clin Endocrinol Metab, 88*(1), 337-346.

De Souza, M. J. & Williams, N. I. (2004). Physiological aspects and clinical sequelae of energy deficiency and hypoestrogenism in exercising women. *Hum Reprod Update, 10*(5), 433-448.

Decker, M. J., Torry, M. R., Wyland, D. J., Sterett, W. I. & Richard Steadman, J. (2003). Gender differences in lower extremity kinematics, kinetics and energy absorption during landing. *Clin Biomech (Bristol, Avon), 18*(7), 662-669.

Deie, M., Sakamaki, Y., Sumen, Y., Urabe, Y. & Ikuta, Y. (2002). Anterior knee laxity in young women varies with their menstrual cycle. *Int Orthop, 26*(3), 154-156.

DeMont, R. G. & Lephart, S. M. (2004). Effect of sex on preactivation of the gastrocnemius and hamstring muscles. *Br J Sports Med, 38*(2), 120-124.

Dragoo, J. L., Lee, R. S., Benhaim, P., Finerman, G. A. & Hame, S. L. (2003). Relaxin receptors in the human female anterior cruciate ligament. *Am J Sports Med, 31*(4), 577-584.
Drinkwater, B. L., Nilson, K., Chesnut, C. H., 3rd, Bremner, W. J., Shainholtz, S. & Southworth, M. B. (1984). Bone mineral content of amenorrheic and eumenorrheic athletes. *N Engl J Med, 311*(5), 277-281.
Dvir, Z., Eger, G., Halperin, N. & Shklar, A. (1989). Thigh muscle activity and anterior cruciate ligament insufficiency *Clinical Biomechanics, 4*(2), 87-91.
Ebstrup, J. F. & Bojsen-Møller, F. (2000). Anterior cruciate ligament injury in indoor ball games. *Scand J Med Sci Sports, 10*(2), 114-116.
Ecochard, R., Boehringer, H., Rabilloud, M. & Marret, H. (2001). Chronological aspects of ultrasonic, hormonal, and other indirect indices of ovulation. *Bjog, 108*(8), 822-829.
Eiling, E., Bryant, A. L., Petersen, W., Murphy, A. & Hohmann, E. (2006). Effects of menstrual-cycle hormone fluctuations on musculotendinous stiffness and knee joint laxity. *Knee Surg Sports Traumatol Arthrosc*.
Ensrud, K. E., Ewing, S. K., Stone, K. L., Cauley, J. A., Bowman, P. J. & Cummings, S. R. (2003). Intentional and unintentional weight loss increase bone loss and hip fracture risk in older women. *J Am Geriatr Soc, 51*(12), 1740-1747.
Fagenbaum, R. & Darling, W. G. (2003). Jump landing strategies in male and female college athletes and the implications of such strategies for anterior cruciate ligament injury. *Am J Sports Med, 31*(2), 233-240.
Faryniarz, D. A., Bhargava, M., Lajam, C., Attia, E. T. & Hannafin, J. A. (2006). Quantitation of estrogen receptors and relaxin binding in human anterior cruciate ligament fibroblasts. *In Vitro Cell Dev Biol Anim, 42*(7), 176-181.
Ford, K. R., Myer, G. D. & Hewett, T. E. (2003). Valgus knee motion during landing in high school female and male basketball players. *Med Sci Sports Exerc, 35*(10), 1745-1750.
Ford, K. R., Myer, G. D., Toms, H. E. & Hewett, T. E. (2005). Gender differences in the kinematics of unanticipated cutting in young athletes. *Med Sci Sports Exerc, 37*(1), 124-129.
Forsberg, S. & Lock, J. (2006). The relationship between perfectionism, eating disorders and athletes: a review. *Minerva Pediatr, 58*(6), 525-536.
Frisch, R. E. & McArthur, J. W. (1974). Menstrual cycles: fatness as a determinant of minimum weight for height necessary for their maintenance or onset. *Science, 185*(4155), 949-951.
Frisch, R. E. & Revelle, R. (1970). Height and weight at menarche and a hypothesis of critical body weights and adolescent events. *Science, 169*(943), 397-399.
Fuller, C. W., Ekstrand, J., Junge, A., Andersen, T. E., Bahr, R., Dvorak, J., et al. (2006). Consensus statement on injury definitions and data collection procedures in studies of football (soccer) injuries. *Br J Sports Med, 40*(3), 193-201.
Fung, D. T. & Zhang, L. Q. (2003). Modeling of ACL impingement against the intercondylar notch. *Clin Biomech (Bristol, Avon), 18*(10), 933-941.
Gabriel, M. T., Wong, E. K., Woo, S. L., Yagi, M. & Debski, R. E. (2004). Distribution of in situ forces in the anterior cruciate ligament in response to rotatory loads. *J Orthop Res, 22*(1), 85-89.
Galey, S., Konieczko, E. M., Arnold, C. A. & Cooney, T. E. (2003). Immunohistological detection of relaxin binding to anterior cruciate ligaments. *Orthopedics, 26*(12), 1201-1204.
Ganko, A., Engebretsen, L. & Ozer, H. (2000). The rolimeter: a new arthrometer compared with the KT-1000. *Knee Surg Sports Traumatol Arthrosc, 8*(1), 36-39.
Garcia, J. E., Jones, G. S. & Wright, G. L., Jr. (1981). Prediction of the time of ovulation. *Fertil Steril, 36*(3), 308-315.
Genazzani, A. D., Bersi, C., Luisi, S., Fruzzetti, F., Malavasi, B., Luisi, M., et al. (2001). Increased adrenal steroid secretion in response to CRF in women with hypothalamic amenorrhea. *J Steroid Biochem Mol Biol, 78*(3), 247-252.

Genazzani, A. D., Ricchieri, F., Lanzoni, C., Strucchi, C. & Jasonni, V. M. (2006). Diagnostic and therapeutic approach to hypothalamic amenorrhea. *Ann N Y Acad Sci, 1092*, 103-113.

Ginsburg, K. A. (1992). Luteal phase defect. Etiology, diagnosis, and management. *Endocrinol Metab Clin North Am, 21*(1), 85-104.

Gissane, C., White, J., Kerr, K. & Jennings, D. (2001). An operational model to investigate contact sports injuries. *Med Sci Sports Exerc, 33*(12), 1999-2003.

Gomez, M. A. (2001). *Biomechanics of soft tissue injury*. Tucson, AZ: Lawyers & Judges Pub.

Granata, K. P., Wilson, S. E. & Padua, D. A. (2002). Gender differences in active musculoskeletal stiffness. Part I. Quantification in controlled measurements of knee joint dynamics. *J Electromyogr Kinesiol, 12*(2), 119-126.

Gremion, G., Rizzoli, R., Slosman, D., Theintz, G. & Bonjour, J. P. (2001). Oligo-amenorrheic long-distance runners may lose more bone in spine than in femur. *Med Sci Sports Exerc, 33*(1), 15-21.

Griffin, L. Y., Albohm, M. J., Arendt, E. A., Bahr, R., Beynnon, B. D., Demaio, M., et al. (2006). Understanding and preventing noncontact anterior cruciate ligament injuries: a review of the Hunt Valley II meeting, January 2005. *Am J Sports Med, 34*(9), 1512-1532.

Gross, S. M., Carcia, C. R., Gansneder, B. M. & Shultz, S. J. (2004). Rate of force application during knee arthrometer testing affects stiffness but not displacement measurement. *J Orthop Sports Phys Ther, 34* (3), 132-139.

Gross, T. S. & Srinivasan, S. (2006). Building bone mass through exercise: could less be more? *Br J Sports Med, 40*(1), 2-3; discussion 2-3.

Guermandi, E., Vegetti, W., Bianchi, M. M., Uglietti, A., Ragni, G. & Crosignani, P. (2001). Reliability of ovulation tests in infertile women. *Obstet Gynecol, 97*(1), 92-96.

Guida, M., Tommaselli, G. A., Palomba, S., Pellicano, M., Moccia, G., Di Carlo, C., et al. (1999). Efficacy of methods for determining ovulation in a natural family planning program. *Fertil Steril, 72*(5), 900-904.

Haapasalo, H., Kontulainen, S., Sievanen, H., Kannus, P., Jarvinen, M. & Vuori, I. (2000). Exercise-induced bone gain is due to enlargement in bone size without a change in volumetric bone density: a peripheral quantitative computed tomography study of the upper arms of male tennis players. *Bone, 27*(3), 351-357.

Hamlet, W. P., Liu, S. H., Panossian, V. & Finerman, G. A. (1997). Primary immunolocalization of androgen target cells in the human anterior cruciate ligament. *J Orthop Res, 15*(5), 657-663.

Hanson, A. M., Padua, D. A., Troy Blackburn, J., Prentice, W. E. & Hirth, C. J. (2008). Muscle activation during side-step cutting maneuvers in male and female soccer athletes. *J Athl Train, 43*(2), 133-143.

Hartard, M., Kleinmond, C., Kirchbichler, A., Jeschke, D., Wiseman, M., Weissenbacher, E. R., et al. (2004). Age at first oral contraceptive use as a major determinant of vertebral bone mass in female endurance athletes. *Bone, 35*(4), 836-841.

Hashemi, J., Chandrashekar, N., Mansouri, H., Slauterbeck, J. R. & Hardy, D. M. (2008). The human anterior cruciate ligament: sex differences in ultrastructure and correlation with biomechanical properties. *J Orthop Res, 26*(7), 945-950.

Heaney, R. P., Abrams, S., Dawson-Hughes, B., Looker, A., Marcus, R., Matkovic, V., et al. (2000). Peak bone mass. *Osteoporos Int, 11*(12), 985-1009.

Heinonen, A., Oja, P., Kannus, P., Sievanen, H., Haapasalo, H., Manttari, A., et al. (1995). Bone mineral density in female athletes representing sports with different loading characteristics of the skeleton. *Bone, 17*(3), 197-203.

Heitz, N. A., Eisenman, P. A., Beck, C. L. & Walker, J. A. (1999). Hormonal Changes Throughout the Menstrual Cycle and Increased Anterior Cruciate Ligament Laxity in Females. *J Athl Train, 34*(2), 144-149.

Hertel, J., Jennifer, H. & Rebbeca, A. (2004). Lower extremity malalignments and anterior cruciate ligament injury history. *Journal of Sports Science and Medicine, 3*, 220-225.

Hertel, J., Williams, N. I., Olmsted-Kramer, L. C., Leidy, H. J. & Putukian, M. (2006). Neuromuscular performance and knee laxity do not change across the menstrual cycle in female athletes. *Knee Surg Sports Traumatol Arthrosc*, 1-6.

Hetland, M. L., Haarbo, J., Christiansen, C. & Larsen, T. (1993). Running induces menstrual disturbances but bone mass is unaffected, except in amenorrheic women. *Am J Med, 95*(1), 53-60.

Hewett, T. E. (2000). Neuromuscular and hormonal factors associated with knee injuries in female athletes. Strategies for intervention. *Sports Med, 29*(5), 313-327.

Hewett, T. E., Ford, K. R., Myer, G. D., Wanstrath, K. & Scheper, M. (2006a). Gender differences in hip adduction motion and torque during a single-leg agility maneuver. *J Orthop Res, 24*(3), 416-421.

Hewett, T. E., Lindenfeld, T. N., Riccobene, J. V. & Noyes, F. R. (1999). The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. *Am J Sports Med, 27*(6), 699-706.

Hewett, T. E., Myer, G. D. & Ford, K. R. (2006b). Anterior cruciate ligament injuries in female athletes: Part 1, mechanisms and risk factors. *Am J Sports Med, 34*(2), 299-311.

Hewett, T. E., Myer, G. D., Ford, K. R., Heidt, R. S., Jr., Colosimo, A. J., McLean, S. G., et al. (2005). Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med, 33*(4), 492-501.

Hewett, T. E., Stroupe, A. L., Nance, T. A. & Noyes, F. R. (1996). Plyometric training in female athletes. Decreased impact forces and increased hamstring torques. *Am J Sports Med, 24*(6), 765-773.

Hicks-Little, C. A., Thatcher, J. R., Hauth, J. M., Goldfuss, A. J. & Cordova, M. L. (2007). Menstrual cycle stage and oral contraceptive effects on anterior tibial displacement in collegiate female athletes. *J Sports Med Phys Fitness, 47*(2), 255-260.

Hinton, P. S., Rector, R. S. & Thomas, T. R. (2006). Weight-bearing, aerobic exercise increases markers of bone formation during short-term weight loss in overweight and obese men and women. *Metabolism, 55*(12), 1616-1618.

Holschen, J. C. (2004). The female athlete. *South Med J, 97*(9), 852-858.

Hsu, W. H., Fisk, J. A., Yamamoto, Y., Debski, R. E. & Woo, S. L. (2006). Differences in torsional joint stiffness of the knee between genders: a human cadaveric study. *Am J Sports Med, 34*(5), 765-770.

Inoue, M., Tanaka, H., Moriwake, T., Oka, M., Sekiguchi, C. & Seino, Y. (2000). Altered biochemical markers of bone turnover in humans during 120 days of bed rest. *Bone, 26*(3), 281-286.

Iqbal, J., Sun, L., Kumar, T. R., Blair, H. C. & Zaidi, M. (2006). Follicle-stimulating hormone stimulates TNF production from immune cells to enhance osteoblast and osteoclast formation. *Proc Natl Acad Sci U S A, 103*(40), 14925-14930.

Ireland, M. L., Ballantyne, B. T., Little, K. & McClay, I. S. (2001). A radiographic analysis of the relationship between the size and shape of the intercondylar notch and anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc, 9*(4), 200-205.

Ireland, M. L. & Ott, S. M. (2004). Special concerns of the female athlete. *Clin Sports Med, 23*(2), 281-298, vii.

Isberg, J., Faxen, E., Brandsson, S., Eriksson, B. I., Karrholm, J. & Karlsson, J. (2006). KT-1000 records smaller side-to-side differences than radiostereometric analysis before and after an ACL reconstruction. *Knee Surg Sports Traumatol Arthrosc, 14*(6), 529-535.

Jordan, J., Craig, K., Clifton, D. K. & Soules, M. R. (1994). Luteal phase defect: the sensitivity and specificity of diagnostic methods in common clinical use. *Fertil Steril, 62*(1), 54-62.

Junge, A. & Dvorak, J. (2000). Influence of definition and data collection on the incidence of injuries in football. *Am J Sports Med, 28*(5 Suppl), S40-46.

Junge, A., Dvorak, J., Graf-Baumann, T. & Peterson, L. (2004). Football injuries during FIFA tournaments and the Olympic Games, 1998-2001: development and implementation of an injury-reporting system. *Am J Sports Med, 32*(1 Suppl), 80S-89S.

Junge, A., Langevoort, G., Pipe, A., Peytavin, A., Wong, F., Mountjoy, M., et al. (2006). Injuries in team sport tournaments during the 2004 Olympic Games. *Am J Sports Med, 34*(4), 565-576.

Kanno, S., Hirano, S. & Kayama, F. (2004). Effects of phytoestrogens and environmental estrogens on osteoblastic differentiation in MC3T3-E1 cells. *Toxicology, 196*(1-2), 137-145.

Kannus, P., Haapasalo, H., Sankelo, M., Sievanen, H., Pasanen, M., Heinonen, A., et al. (1995). Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. *Ann Intern Med, 123*(1), 27-31.

Kanstrup, I. L. (2005). Women and sports--still fewer restrictions and something to be learned for both sexes? *Scand J Med Sci Sports, 15*(2), 67-68.

Karageanes, S. J., Blackburn, K. & Vangelos, Z. A. (2000). The association of the menstrual cycle with the laxity of the anterior cruciate ligament in adolescent female athletes. *Clin J Sport Med, 10*(3), 162-168.

Karrholm, J., Selvik, G., Elmqvist, L. G., Hansson, L. I. & Jonsson, H. (1988). Three-dimensional instability of the anterior cruciate deficient knee. *J Bone Joint Surg Br, 70*(5), 777-783.

Keen, A. D. & Drinkwater, B. L. (1997). Irreversible bone loss in former amenorrheic athletes. *Osteoporos Int, 7*(4), 311-315.

Kessler, M. A., Behrend, H., Henz, S., Stutz, G., Rukavina, A. & Kuster, M. S. (2008). Function, osteoarthritis and activity after ACL-rupture: 11 years follow-up results of conservative versus reconstructive treatment. *Knee Surg Sports Traumatol Arthrosc*.

Kirchner, E. M., Lewis, R. D. & O'Connor, P. J. (1995). Bone mineral density and dietary intake of female college gymnasts. *Med Sci Sports Exerc, 27*(4), 543-549.

Komatsuda, T., Sugita, T., Sano, H., Kusakabe, T., Watanuki, M., Yoshizumi, Y., et al. (2006). Does estrogen alter the mechanical properties of the anterior cruciate ligament? An experimental study in rabbits. *Acta Orthop, 77*(6), 973-980.

Kontulainen, S., Kannus, P., Haapasalo, H., Heinonen, A., Sievanen, H., Oja, P., et al. (1999). Changes in bone mineral content with decreased training in competitive young adult tennis players and controls: a prospective 4-yr follow-up. *Med Sci Sports Exerc, 31*(5), 646-652.

Kramer, L. C., Denegar, C. R., Buckley, W. E. & Hertel, J. (2007). Factors associated with anterior cruciate ligament injury: history in female athletes. *J Sports Med Phys Fitness, 47*(4), 446-454.

Krosshaug, T., Andersen, T. E., Olsen, O. E., Myklebust, G. & Bahr, R. (2005). Research approaches to describe the mechanisms of injuries in sport: limitations and possibilities. *Br J Sports Med, 39*(6), 330-339.

Krosshaug, T., Nakamae, A., Boden, B. P., Engebretsen, L., Smith, G., Slauterbeck, J. R., et al. (2007). Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. *Am J Sports Med, 35*(3), 359-367.

Lambson, R. B., Barnhill, B. S. & Higgins, R. W. (1996). Football cleat design and its effect on anterior cruciate ligament injuries. A three-year prospective study. *Am J Sports Med, 24*(2), 155-159.

Langevoort, G., Myklebust, G., Dvorak, J. & Junge, A. (2007). Handball injuries during major international tournaments. *Scand J Med Sci Sports, 17*(4), 400-407.

Lebrun, C. M. (1994). The effect of the phase of the menstrual cycle and the birth control pill on athletic performance. *Clin Sports Med, 13*(2), 419-441.

Lebrun, C. M. & Rumball, J. S. (2001). Relationship between athletic performance and menstrual cycle. *Curr Womens Health Rep, 1*(3), 232-240.

Lee, C. Y., Liu, X., Smith, C. L., Zhang, X., Hsu, H. C., Wang, D. Y., et al. (2004a). The combined regulation of estrogen and cyclic tension on fibroblast biosynthesis derived from anterior cruciate ligament. *Matrix Biol, 23*(5), 323-329.

Lee, C. Y., Smith, C. L., Zhang, X., Hsu, H. C., Wang, D. Y. & Luo, Z. P. (2004b). Tensile forces attenuate estrogen-stimulated collagen synthesis in the ACL. *Biochem Biophys Res Commun, 317*(4), 1221-1225.

Leidinger, A., Gast, W. & Pforringer, W. (1990). [Traumatology in indoor handball sports. A sports medicine analysis of the incidence of injuries and accident epidemiology of indoor handball sports in senior players in the Federal Republic of Germany after 1981]. *Sportverletz Sportschaden, 4*(2), 65-68.

Lemaire, V., Tobin, F. L., Greller, L. D., Cho, C. R. & Suva, L. J. (2004). Modeling the interactions between osteoblast and osteoclast activities in bone remodeling. *J Theor Biol, 229*(3), 293-309.

Lenton, E. A., Landgren, B. M. & Sexton, L. (1984). Normal variation in the length of the luteal phase of the menstrual cycle: identification of the short luteal phase. *Br J Obstet Gynaecol, 91*(7), 685-689.

Lephart, S. M., Ferris, C. M., Riemann, B. L., Myers, J. B. & Fu, F. H. (2002). Gender differences in strength and lower extremity kinematics during landing. *Clin Orthop Relat Res*(401), 162-169.

Li, H., Chen, J., Overstreet, J. W., Nakajima, S. T. & Lasley, B. L. (2002). Urinary follicle-stimulating hormone peak as a biomarker for estimating the day of ovulation. *Fertil Steril, 77*(5), 961-966.

Lindblad, B. E., Hoy, K., Terkelsen, C. J. & Helleland, H. E. (1992). Handball injuries. An epidemiologic and socioeconomic study. *Am J Sports Med, 20*(4), 441-444.

Liu, S. H., al-Shaikh, R., Panossian, V., Yang, R. S., Nelson, S. D., Soleiman, N., et al. (1996). Primary immunolocalization of estrogen and progesterone target cells in the human anterior cruciate ligament. *J Orthop Res, 14*(4), 526-533.

Liu, S. H., Al-Shaikh, R. A., Panossian, V., Finerman, G. A. & Lane, J. M. (1997). Estrogen affects the cellular metabolism of the anterior cruciate ligament. A potential explanation for female athletic injury. *Am J Sports Med, 25*(5), 704-709.

Liu, S. H., Yang, R. S., al-Shaikh, R. & Lane, J. M. (1995). Collagen in tendon, ligament, and bone healing. A current review. *Clin Orthop Relat Res*(318), 265-278.

Lloyd, T., Taylor, D. S., Lin, H. M., Matthews, A. E., Eggli, D. F. & Legro, R. S. (2000). Oral contraceptive use by teenage women does not affect peak bone mass: a longitudinal study. *Fertil Steril, 74*(4), 734-738.

Loës, M. (1995). Epidemiology of sports injuries in the swiss organization "youth and sports" 1987-1989. *Int J Sports Med, 16*, 134-138.

Lombardo, S., Sethi, P. M. & Starkey, C. (2005). Intercondylar notch stenosis is not a risk factor for anterior cruciate ligament tears in professional male basketball players: an 11-year prospective study. *Am J Sports Med, 33*(1), 29-34.

Lorist, M. M., Kernell, D., Meijman, T. F. & Zijdewind, I. (2002). Motor fatigue and cognitive task performance in humans. *J Physiol, 545*(Pt 1), 313-319.

Loucks, A. B. (1990). Effects of exercise training on the menstrual cycle: existence and mechanisms. *Med Sci Sports Exerc, 22*(3), 275-280.

Loucks, A. B. & Horvath, S. M. (1985). Athletic amenorrhea: a review. *Med Sci Sports Exerc, 17*(1), 56-72.

Loucks, A. B., Mortola, J. F., Girton, L. & Yen, S. S. (1989). Alterations in the hypothalamic-pituitary-ovarian and the hypothalamic-pituitary-adrenal axes in athletic women. *J Clin Endocrinol Metab, 68*(2), 402-411.

Loucks, A. B., Verdun, M. & Heath, E. M. (1998). Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *J Appl Physiol, 84*(1), 37-46.

Lovering, R. M. & Romani, W. A. (2005). Effect of testosterone on the female anterior cruciate ligament. *Am J Physiol Regul Integr Comp Physiol, 289*(1), R15-22.
Luciano, A. A., Peluso, J., Koch, E. I., Maier, D., Kuslis, S. & Davison, E. (1990). Temporal relationship and reliability of the clinical, hormonal, and ultrasonographic indices of ovulation in infertile women. *Obstet Gynecol, 75*(3 Pt 1), 412-416.
Lund-Hanssen, H., Gannon, J., Engebretsen, L., Holen, K. J., Anda, S. & Vatten, L. (1994). Intercondylar notch width and the risk for anterior cruciate ligament rupture. A case-control study in 46 female handball players. *Acta Orthop Scand, 65*(5), 529-532.
Madigan, M. L. & Pidcoe, P. E. (2003). Changes in landing biomechanics during a fatiguing landing activity. *J Electromyogr Kinesiol, 13*(5), 491-498.
Majewski, M., Susanne, H. & Klaus, S. (2006). Epidemiology of athletic knee injuries: A 10-year study. *Knee, 13*(3), 184-188.
Malina, R. M., Spirduso, W. W., Tate, C. & Baylor, A. M. (1978). Age at menarche and selected menstrual characteristics in athletes at different competitive levels and in different sports. *Med Sci Sports, 10*(3), 218-222.
Malinzak, R. A., Colby, S. M., Kirkendall, D. T., Yu, B. & Garrett, W. E. (2001). A comparison of knee joint motion patterns between men and women in selected athletic tasks. *Clin Biomech (Bristol, Avon), 16*(5), 438-445.
Marcus, M. D., Loucks, T. L. & Berga, S. L. (2001). Psychological correlates of functional hypothalamic amenorrhea. *Fertil Steril, 76*(2), 310-316.
Marks, P. H., Droll, K. P. & Cameron-Donaldson, M. (2007). Does ACL reconstruction prevent articular degeneration? In T. E. Hewett, S. J. Shultz & L. Y. Griffin (Eds.), *Understanding and Preventing Noncontact ACL injuries* (pp. 31-45). Champaign, IL: American Orthopeadic Society for Sports Medicine
Marnach, M. L., Ramin, K. D., Ramsey, P. S., Song, S. W., Stensland, J. J. & An, K. N. (2003). Characterization of the relationship between joint laxity and maternal hormones in pregnancy. *Obstet Gynecol, 101*(2), 331-335.
Marshall, S. W., Padua, D. & McGrath, M. (2007). Incidence of ACL Injury. In T. E. Hewett, S. J. Shultz & L. Y. Griffin (Eds.), *Undertanding and Preventiing Noncontact ACL injuries* (pp. 5-29). Champaign, IL: American Orthopeadic Society for Sports Medicine
Martineau, P. A., Al-Jassir, F., Lenczner, E. & Burman, M. L. (2004). Effect of the Oral Contraceptive Pill on Ligamentous Laxity. *Clin J Sport Med 14*(5), 281-286.
Martinez, A. R., Bernardus, R. E., Vermeiden, J. P. & Schoemaker, J. (1992). Reliability of home urinary LH tests for timing of insemination: a consumer's study. *Hum Reprod, 7*(6), 751-753.
Massada, J. L. (2001). *A Lateralidade Anatómica e Biomecânica. Sua repercussão na Assimetria Morfológica e na patologia Traumática do Esqueleto Axial e Apendicular do Atleta.* Unpublished PhD, Universidade do Porto, Porto.
McIntosh, A. S. (2005). Risk compensation, motivation, injuries, and biomechanics in competitive sport. *Br J Sports Med, 39*(1), 2-3.
McLean, S. G., Fellin, R. E., Suedekum, N., Calabrese, G., Passerallo, A. & Joy, S. (2007). Impact of fatigue on gender-based high-risk landing strategies. *Med Sci Sports Exerc, 39*(3), 502-514.
McLean, S. G., Huang, X. & van den Bogert, A. J. (2005). Association between lower extremity posture at contact and peak knee valgus moment during sidestepping: implications for ACL injury. *Clin Biomech (Bristol, Avon), 20*(8), 863-870.
McLean, S. G., Lipfert, S. W. & van den Bogert, A. J. (2004). Effect of gender and defensive opponent on the biomechanics of sidestep cutting. *Med Sci Sports Exerc, 36*(6), 1008-1016.
McLean, S. G., Neal, R. J., Myers, P. T. & Walters, M. R. (1999). Knee joint kinematics during the sidestep cutting maneuver: potential for injury in women. *Med Sci Sports Exerc, 31*(7), 959-968.

McNeely, M. J. & Soules, M. R. (1988). The diagnosis of luteal phase deficiency: a critical review. *Fertil Steril, 50*(1), 1-15.
Meczekalski, B., Tonetti, A., Monteleone, P., Bernardi, F., Luisi, S., Stomati, M., et al. (2000). Hypothalamic amenorrhea with normal body weight: ACTH, allopregnanolone and cortisol responses to corticotropin-releasing hormone test. *Eur J Endocrinol, 142*(3), 280-285.
Meeuwisse, W. H. (1994). Assessing causation in sport injury: a multifactorial model. *Clin J Sport Med, 14*, 166-170.
Meeuwisse, W. H., Tyreman, H., Hagel, B. & Emery, C. (2007). A dynamic model of etiology in sport injury: the recursive nature of risk and causation. *Clin J Sport Med, 17*(3), 215-219.
Meister, K., Talley, M. C., Horodyski, M. B., Indelicato, P. A., Hartzel, J. S. & Batts, J. (1998). Caudal slope of the tibia and its relationship to noncontact injuries to the ACL. *Am J Knee Surg, 11*(4), 217-219.
Mesfar, W. & Shirazi-Adl, A. (2005). Biomechanics of the knee joint in flexion under various quadriceps forces. *Knee, 12*(6), 424-434.
Miller, J. B. & Keyes, P. L. (1978). Transition of the rabbit corpus luteum to estrogen dependence during early luteal development. *Endocrinology, 102*(1), 31-38.
Miller, P. B. & Soules, M. R. (1996). The usefulness of a urinary LH kit for ovulation prediction during menstrual cycles of normal women. *Obstet Gynecol, 87*(1), 13-17.
Miro, F. & Aspinall, L. J. (2005). The onset of the initial rise in follicle-stimulating hormone during the human menstrual cycle. *Hum Reprod, 20*(1), 96-100.
Miura, K., Ishibashi, Y., Tsuda, E., Okamura, Y., Otsuka, H. & Toh, S. (2004). The Effect of Local and General Fatigue on Knee Proprioception. *The Journal of Arthroscopic and Related Surgery, 20*(4), 414-418.
Moller-Nielsen, J. & Hammar, M. (1989). Women's soccer injuries in relation to the menstrual cycle and oral contraceptive use. *Med Sci Sports Exerc, 21*(2), 126-129.
Muaidi, Q. I., Nicholson, L. L., Refshauge, K. M. & Eisenhuth, J. P. (2007). Design of a knee rotatory kinaesthetic device. *Med Eng Phys, 29*(9), 1035-1042.
Munro, C. J., Stabenfeldt, G. H., Cragun, J. R., Addiego, L. A., Overstreet, J. W. & Lasley, B. L. (1991). Relationship of serum estradiol and progesterone concentrations to the excretion profiles of their major urinary metabolites as measured by enzyme immunoassay and radioimmunoassay. *Clin Chem, 37*(6), 838-844.
Murphy, D. F., Connolly, D. A. & Beynnon, B. D. (2003). Risk factors for lower extremity injury: a review of the literature. *Br J Sports Med, 37*(1), 13-29.
Murray, M. M., Martin, S. D., Martin, T. L. & Spector, M. (2000). Histological changes in the human anterior cruciate ligament after rupture. *J Bone Joint Surg Am, 82-A*(10), 1387-1397.
Musahl, V., Bell, K. M., Tsai, A. G., Costic, R. S., Allaire, R., Zantop, T., et al. (2007). Development of a simple device for measurement of rotational knee laxity. *Knee Surg Sports Traumatol Arthrosc, 15*(8), 1009-1012.
Mutsuzaki, H., Sakane, M., Ikeda, K., Ishii, T., Hattori, S., Tanaka, J., et al. (2007). Histological changes and apoptosis of cartilage layer in human anterior cruciate ligament tibial insertion after rupture. *Knee Surg Sports Traumatol Arthrosc, 15*(5), 602-609.
Myer, G. D., Ford, K. R. & Hewett, T. E. (2005). The effects of gender on quadriceps muscle activation strategies during a maneuver that mimics a high ACL injury risk position. *J Electromyogr Kinesiol, 15*(2), 181-189.
Myklebust, G., Engebretsen, L., Braekken, I. H., Skjolberg, A., Olsen, O. E. & Bahr, R. (2003a). Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. *Clin J Sport Med, 13*(2), 71-78.
Myklebust, G., Holm, I., Maehlum, S., Engebretsen, L. & Bahr, R. (2003b). Clinical, functional, and radiologic outcome in team handball players 6 to 11 years after

anterior cruciate ligament injury: a follow-up study. *Am J Sports Med, 31*(6), 981-989.

Myklebust, G., Maehlum, S., Engebretsen, L., Strand, T. & Solheim, E. (1997). Registration of cruciate ligament injuries in Norwegian top level team handball. A prospective study covering two seasons. *Scand J Med Sci Sports, 7*(5), 289-292.

Myklebust, G., Maehlum, S., Holm, I. & Bahr, R. (1998). A prospective cohort study of anterior cruciate ligament injuries in elite Norwegian team handball. *Scand J Med Sci Sports, 8*(3), 149-153.

Nickols-Richardson, S. M., Modlesky, C. M., O'Connor, P. J. & Lewis, R. D. (2000). Premenarcheal gymnasts possess higher bone mineral density than controls. *Med Sci Sports Exerc, 32*(1), 63-69.

Nielsen, A. B. & Yde, J. (1988). An epidemiologic and traumatologic study of injuries in handball. *Int J Sports Med, 9*(5), 341-344.

Nistri, S., Bigazzi, M. & Bani, D. (2007). Relaxin as a cardiovascular hormone: physiology, pathophysiology and therapeutic promises. *Cardiovasc Hematol Agents Med Chem, 5*(2), 101-108.

Nyland, J. A., Caborn, D. N., Shapiro, R. & Johnson, D. L. (1997a). Fatigue after eccentric quadriceps femoris work produces earlier gastrocnemius and delayed quadriceps femoris activation during crossover cutting among normal athletic women. *Knee Surg Sports Traumatol Arthrosc, 5*(3), 162-167.

Nyland, J. A., Shapiro, R., Caborn, D. N., Nitz, A. J. & Malone, T. R. (1997b). The effect of quadriceps femoris, hamstring, and placebo eccentric fatigue on knee and ankle dynamics during crossover cutting. *J Orthop Sports Phys Ther, 25*(3), 171-184.

Oehlert, K., Drescher, W., Petersen, W., Zantop, T., Gross, V. & Hassenpflug, J. (2004). [Injuries in Olympic handball tournaments: a video analysis]. *Sportverletz Sportschaden, 18*(2), 80-84.

Olsen, O. E., Myklebust, G., Engebretsen, L. & Bahr, R. (2004). Injury mechanisms for anterior cruciate ligament injuries in team handball: a systematic video analysis. *Am J Sports Med, 32*(4), 1002-1012.

Olsen, O. E., Myklebust, G., Engebretsen, L., Holme, I. & Bahr, R. (2003). Relationship between floor type and risk of ACL injury in team handball. *Scand J Med Sci Sports, 13*(5), 299-304.

Olsen, O. E., Myklebust, G., Engebretsen, L., Holme, I. & Bahr, R. (2005). Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. *Bmj, 330*(7489), 449.

Orchard, J. W. & Powell, J. W. (2003). Risk of knee and ankle sprains under various weather conditions in American football. *Med Sci Sports Exerc, 35*(7), 1118-1123.

Pandy, M. G. & Shelburne, K. B. (1997). Dependence of cruciate-ligament loading on muscle forces and external load. *J Biomech, 30*(10), 1015-1024.

Panics, G., Tallay, A., Pavlik, A. & Berkes, I. (2008). Effect of proprioception training on knee joint position sense in female team handball players. *Br J Sports Med, 42*(6), 472-476.

Pantano, K. J., White, S. C., Gilchrist, L. A. & Leddy, J. (2005). Differences in peak knee valgus angles between individuals with high and low Q-angles during a single limb squat. *Clin Biomech (Bristol, Avon), 20*(9), 966-972.

Park, H. S., Wilson, N. A. & Zhang, L. Q. (2008). Gender differences in passive knee biomechanical properties in tibial rotation. *J Orthop Res, 26*(7), 937-944.

Parkkari, J., Kujala, U. M. & Kannus, P. (2001). Is it possible to prevent sports injuries? Review of controlled clinical trials and recommendations for future work. *Sports Med, 31*(14), 985-995.

Pearlstone, A. C. & Surrey, E. S. (1994). The temporal relation between the urine LH surge and sonographic evidence of ovulation: determinants and clinical significance. *Obstet Gynecol, 83*(2), 184-188.

Pelletier, R. L., Montelpare, W. J. & Stark, R. M. (1993). Intercollegiate ice hockey injuries. A case for uniform definitions and reports. *Am J Sports Med, 21*(1), 78-81.

Perkins, R. B., Hall, J. E. & Martin, K. A. (1999). Neuroendocrine abnormalities in hypothalamic amenorrhea: spectrum, stability, and response to neurotransmitter modulation. *J Clin Endocrinol Metab, 84*(6), 1905-1911.
Petersen, W., Braun, C., Bock, W., Schmidt, K., Weimann, A., Drescher, W., et al. (2005). A controlled prospective case control study of a prevention training program in female team handball players: the German experience. *Arch Orthop Trauma Surg, 125*(9), 614-621.
Petersen, W. & Zantop, T. (2006). Partial rupture of the anterior cruciate ligament. *Arthroscopy, 22*(11), 1143-1145.
Petersen, W. & Zantop, T. (2007). Anatomy of the anterior cruciate ligament with regard to its two bundles. *Clin Orthop Relat Res, 454*, 35-47.
Pettersson, U., Stalnacke, B., Ahlenius, G., Henriksson-Larsen, K. & Lorentzon, R. (1999). Low bone mass density at multiple skeletal sites, including the appendicular skeleton in amenorrheic runners. *Calcif Tissue Int, 64*(2), 117-125.
Pfeifer, S. & Patrizio, P. (2002). The female athlete: some gynecologic considerations. *Sports Medicine & Arthroscopy Review, 10*, 2-9.
Pollard, C. D., Braun, B. & Hamill, J. (2006). Influence of gender, estrogen and exercise on anterior knee laxity. *Clin Biomech (Bristol, Avon), 21*(10), 1060-1066.
Pollard, C. D., Sigward, S. M. & Powers, C. M. (2007). Gender differences in hip joint kinematics and kinetics during side-step cutting maneuver. *Clin J Sport Med, 17*(1), 38-42.
Prior, J. C. (1990). Progesterone as a bone-trophic hormone. *Endocr Rev, 11*(2), 386-398.
Prior, J. C., Vigna, Y. M., Schulzer, M., Hall, J. E. & Bonen, A. (1990). Determination of luteal phase length by quantitative basal temperature methods: validation against the midcycle LH peak. *Clin Invest Med, 13*(3), 123-131.
Proctor, K. L., Adams, W. C., Shaffrath, J. D. & Van Loan, M. D. (2002). Upper-limb bone mineral density of female collegiate gymnasts versus controls. *Med Sci Sports Exerc, 34*(11), 1830-1835.
Putukian, M. (1998). The female athlete triad. *Clin Sports Med, 17*(4), 675-696, v.
Queenan, J. T., O'Brien, G. D., Bains, L. M., Simpson, J., Collins, W. P. & Campbell, S. (1980). Ultrasound scanning of ovaries to detect ovulation in women. *Fertil Steril, 34*(2), 99-105.
Rau, M. D., Renouf, D., Benfield, D., Otto, D. D., Thornton, G. M., Raso, V. J., et al. (2005). Examination of the failure properties of the anterior cruciate ligament during the estrous cycle. *Knee, 12*(1), 37-40.
Reckling, C., Zantop, T. & Petersen, W. (2003). [Epidemiology of injuries in juvenile handball players]. *Sportverletz Sportschaden, 17*(3), 112-117.
Redman, L. M. & Loucks, A. B. (2005). Menstrual disorders in athletes. *Sports Med, 35*(9), 747-755.
Redman, L. M., Scroop, G. C. & Norman, R. J. (2003). Impact of menstrual cycle phase on the exercise status of young, sedentary women. *Eur J Appl Physiol, 90*, 505-513.
Rencken, M. L., Chesnut, C. H., 3rd & Drinkwater, B. L. (1996). Bone density at multiple skeletal sites in amenorrheic athletes. *Jama, 276*(3), 238-240.
Renstrom, P., Ljungqvist, A., Arendt, E., Beynnon, B., Fukubayashi, T., Garrett, W., et al. (2008). Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. *Br J Sports Med, 42*(6), 394-412.
Ristanis, S., Stergiou, N., Patras, K., Tsepis, E., Moraiti, C. & Georgoulis, A. D. (2006). Follow-up evaluation 2 years after ACL reconstruction with bone-patellar tendon-bone graft shows that excessive tibial rotation persists. *Clin J Sport Med, 16*(2), 111-116.
Ristanis, S., Stergiou, N., Patras, K., Vasiliadis, H. S., Giakas, G. & Georgoulis, A. D. (2005). Excessive tibial rotation during high-demand activities is not restored by anterior cruciate ligament reconstruction. *Arthroscopy, 21*(11), 1323-1329.

Robinson, T. L., Snow-Harter, C., Taaffe, D. R., Gillis, D., Shaw, J. & Marcus, R. (1995). Gymnasts exhibit higher bone mass than runners despite similar prevalence of amenorrhea and oligomenorrhea. *J Bone Miner Res, 10*(1), 26-35.
Romani, W., Patrie, J., Curl, L. A. & Flaws, J. A. (2003). The correlations between estradiol, estrone, estriol, progesterone, and sex hormone-binding globulin and anterior cruciate ligament stiffness in healthy, active females. *J Womens Health (Larchmt), 12*(3), 287-298.
Ronglan, L. T., Raastad, T. & Borgesen, A. (2006). Neuromuscular fatigue and recovery in elite female handball players. *Scand J Med Sci Sports, 16*(4), 267-273.
Rose, M. P., Gaines Das, R. E. & Balen, A. H. (2000). Definition and measurement of follicle stimulating hormone. *Endocr Rev, 21*(1), 5-22.
Rosene, J. M. & Fogarty, T. D. (1999). Anterior Tibial Translation in Collegiate Athletes with Normal Anterior Cruciate Ligament Integrity. *J Athl Train, 34*(2), 93-98.
Rozzi, S. L., Lephart, S. M., Gear, W. S. & Fu, F. H. (1999). Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. *Am J Sports Med, 27*(3), 312-319.
Rozzi, S. L., Yuktanandana, P., Pincivero, D. & Lephart, S. M. (2001). Role of fatigue on proprioception and neuromuscular control. In S. M. Lephart & F. H. FU (Eds.), *Proprioception and neuromuscular control in joint stability* (pp. 375-383): Human kinetics-copyright.
Russell, K. A., Palmieri, R. M., Zinder, S. M. & Ingersoll, C. D. (2006). Sex differences in valgus knee angle during a single-leg drop jump. *J Athl Train, 41*(2), 166-171.
Samuel, C. S., Coghlan, J. P. & Bateman, J. F. (1998). Effects of relaxin, pregnancy and parturition on collagen metabolism in the rat pubic symphysis. *J Endocrinol, 159*(1), 117-125.
Schauberger, C. W., Rooney, B. L., Goldsmith, L., Shenton, D., Silva, P. D. & Schaper, A. (1996). Peripheral joint laxity increases in pregnancy but does not correlate with serum relaxin levels. *Am J Obstet Gynecol, 174*(2), 667-671.
Schmidt-Olsen, S., Jorgensen, U., Kaalund, S. & Sorensen, J. (1991). Injuries among young soccer players. *Am J Sports Med, 19*(3), 273-275.
Schmitz, R. J., Ficklin, T. K., Shimokochi, Y., Nguyen, A. D., Beynnon, B. D., Perrin, D. H., et al. (2008). Varus/valgus and internal/external torsional knee joint stiffness differs between sexes. *Am J Sports Med, 36*(7), 1380-1388.
Schneider, J. E. & Wade, G. N. (1989). Availability of metabolic fuels controls estrous cyclicity of Syrian hamsters. *Science, 244*(4910), 1326-1328.
Schuster, A. J., McNicholas, M. J., Wachtl, S. W., McGurty, D. W. & Jakob, R. P. (2004). A new mechanical testing device for measuring anteroposterior knee laxity. *Am J Sports Med, 32*(7), 1731-1735.
Scranton, P. E., Jr., Whitesel, J. P., Powell, J. W., Dormer, S. G., Heidt, R. S., Jr., Losse, G., et al. (1997). A review of selected noncontact anterior cruciate ligament injuries in the National Football League. *Foot Ankle Int, 18*(12), 772-776.
Seco, J. D. (2006). LOS INICIOS DEL SIGLO XXI: EVOLUCION Y TENDENCIAS DEL JUEGO [Electronic Version]. *e-balonmano.com: Revista Digital Deportiva*, *2,* 3-29.
Seco, J. D. (2007). Handball Development In The 2004-2008 Olympic Cycle [Electronic Version]. *EHF Web Periodical*, 5.
Seibel, M. M., Shine, W., Smith, D. M. & Taymor, M. L. (1982). Biological rhythm of the luteinizing hormone surge in women. *Fertil Steril, 37*(5), 709-711.
Seil, R., Rupp, S., Tempelhof, S. & Kohn, D. (1997). [Injuries during handball. A comparative, retrospective study between regional and upper league teams]. *Sportverletz Sportschaden, 11*(2), 58-62.
Seil, R., Rupp, S., Tempelhof, S. & Kohn, D. (1998). Sports injuries in team handball. A one-year prospective study of sixteen men's senior teams of a superior nonprofessional level. *Am J Sports Med, 26*(5), 681-687.

Seneviratne, A., Attia, E., Williams, R. J., Rodeo, S. A. & Hannafin, J. A. (2004). The effect of estrogen on ovine anterior cruciate ligament fibroblasts: cell proliferation and collagen synthesis. *Am J Sports Med, 32*(7), 1613-1618.
Shelbourne, K. D., Davis, T. J. & Klootwyk, T. E. (1998). The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears. A prospective study. *Am J Sports Med, 26*(3), 402-408.
Shin, C. S., Chaudhari, A. M. & Andriacchi, T. P. (2007). The influence of deceleration forces on ACL strain during single-leg landing: a simulation study. *J Biomech, 40*(5), 1145-1152.
Shultz, S. J. (2007). Hormonal influences on ligament biology In T. E. Hewett, S. J. Shultz & L. Y. Griffin (Eds.), *Undertanding and Preventiing Noncontact ACL injuries* (pp. 219-238). Champaign, IL: American Orthopeadic Society for Sports Medicine
Shultz, S. J., Carcia, C. R. & Perrin, D. H. (2004a). Knee joint laxity affects muscle activation patterns in the healthy knee. *J Electromyogr Kinesiol, 14*(4), 475-483.
Shultz, S. J., Kirk, S. E., Johnson, M. L., Sander, T. C. & Perrin, D. H. (2004b). Relationship between sex hormones and anterior knee laxity across the menstrual cycle. *Med Sci Sports Exerc, 36*(7), 1165-1174.
Shultz, S. J., Nguyen, A. & Beynnon, B. (2007). Anatomical factors in ACL injury risk. In T. E. Hewett, S. J. Shultz & L. Y. Griffin (Eds.), *Undertanding and Preventiing Noncontact ACL injuries* (pp. 239-258). Champaign, IL: American Orthopeadic Society for Sports Medicine
Shultz, S. J., Sander, T. C., Kirk, S. E. & Perrin, D. H. (2005). Sex differences in knee joint laxity change across the female menstrual cycle. *J Sports Med Phys Fitness, 45*(4), 594-603.
Sinai, I., Jennings, V. & Arevalo, M. (1999). The TwoDay Algorithm: a new algorithm to identify the fertile time of the menstrual cycle. *Contraception, 60*(2), 65-70.
Slauterbeck, J. R., Clevenger, C., Lundberg, W. & Burchfield, D. M. (1999). Estrogen level alters the failure load of the rabbit anterior cruciate ligament. *J Orthop Res, 17*(3), 405-408.
Slauterbeck, J. R., Fuzie, S. F., Smith, M. P., Clark, R. J., Xu, K., Starch, D. W., et al. (2002). The Menstrual Cycle, Sex Hormones, and Anterior Cruciate Ligament Injury. *J Athl Train, 37*(3), 275-278.
Soderman, K., Werner, S., Pietila, T., Engstrom, B. & Alfredson, H. (2000). Balance board training: prevention of traumatic injuries of the lower extremities in female soccer players? A prospective randomized intervention study. *Knee Surg Sports Traumatol Arthrosc, 8*(6), 356-363.
Solomonow, M. & Krogsgaard, M. (2001). Sensorimotor control of knee stability. A review. *Scand J Med Sci Sports, 11*(2), 64-80.
Sousa, F., Ishikawa, M., Vilas-Boas, J. P. & Komi, P. V. (2007). Intensity- and muscle-specific fascicle behavior during human drop jumps. *J Appl Physiol, 102*(1), 382-389.
Soyka, L. A., Fairfield, W. P. & Klibanski, A. (2000). Clinical review 117: Hormonal determinants and disorders of peak bone mass in children. *J Clin Endocrinol Metab, 85*(11), 3951-3963.
Staubli, H. U., Noesberger, B. & Jakob, R. P. (1992). Stressradiography of the knee. Cruciate ligament function studied in 138 patients. *Acta Orthop Scand Suppl, 249*, 1-27.
Steckel, H., Starman, J. S., Baums, M. H., Klinger, H. M., Schultz, W. & Fu, F. H. (2007). The double-bundle technique for anterior cruciate ligament reconstruction: a systematic overview. *Scand J Med Sci Sports, 17*(2), 99-108.
Stewart, D. R., Celniker, A. C., Taylor, C. A., Jr., Cragun, J. R., Overstreet, J. W. & Lasley, B. L. (1990). Relaxin in the peri-implantation period. *J Clin Endocrinol Metab, 70*(6), 1771-1773.

Stijak, L., Herzog, R. F. & Schai, P. (2008). Is there an influence of the tibial slope of the lateral condyle on the ACL lesion? A case-control study. *Knee Surg Sports Traumatol Arthrosc, 16*(2), 112-117.
Strand, T. & Solheim, E. (1995). Clinical tests versus KT-1000 instrumented laxity test in acute anterior cruciate ligament tears. *Int J Sports Med, 16*(1), 51-53.
Strand, T., Tvedte, R., Engebretsen, L. & Tegnander, A. (1990). [Anterior cruciate ligament injuries in handball playing. Mechanisms and incidence of injuries]. *Tidsskr Nor Laegeforen, 110*(17), 2222-2225.
Strickland, S. M., Belknap, T. W., Turner, S. A., Wright, T. M. & Hannafin, J. A. (2003). Lack of hormonal influences on mechanical properties of sheep knee ligaments. *Am J Sports Med, 31*(2), 210-215.
Thompson, M. T., Conditt, M. A., Ismaily, S. K., Agarwal, A. & Noble, P. C. (2004). Brief report: validation of a system for automated measurement of knee laxity. *Clin Biomech (Bristol, Avon), 19*(3), 308-312.
Torstveit, M. K. & Sundgot-Borgen, J. (2005). Low bone mineral density is two to three times more prevalent in non-athletic premenopausal women than in elite athletes: a comprehensive controlled study. *Br J Sports Med, 39*(5), 282-287; discussion 282-287.
Trimble, M. H., Bishop, M. D., Buckley, B. D., Fields, L. C. & Rozea, G. D. (2002). The relationship between clinical measurements of lower extremity posture and tibial translation. *Clin Biomech (Bristol, Avon), 17*(4), 286-290.
Tromp, A. M., Bravenboer, N., Tanck, E., Oostlander, A., Holzmann, P. J., Kostense, P. J., et al. (2006). Additional weight bearing during exercise and estrogen in the rat: the effect on bone mass, turnover, and structure. *Calcif Tissue Int, 79*(6), 404-415.
Tsai, A. G., Musahl, V., Steckel, H., Bell, K. M., Zantop, T., Irrgang, J. J., et al. (2008). Rotational knee laxity: reliability of a simple measurement device in vivo. *BMC Musculoskelet Disord, 9*, 35.
Turner, C. H. & Robling, A. G. (2005). Exercises for improving bone strength. *Br J Sports Med, 39*(4), 188-189.
Turner, R. T. (2000). Invited review: what do we know about the effects of spaceflight on bone? *J Appl Physiol, 89*(2), 840-847.
Uhorchak, J. M., Scoville, C. R., Williams, G. N., Arciero, R. A., Pierre, P. S. & Taylor, D. C. (2003). Risk factors associated with noncontact injury of the anterior cruciate ligament: a propective four-year evaluation of 859 West Point Cadets. *American Journal of Sports Medicine, 31*(6), 831-842.
Un, B. S., Beynnon, B. D., Churchill, D. L., Haugh, L. D., Risberg, M. A. & Fleming, B. C. (2001). A new device to measure knee laxity during weightbearing and non-weightbearing conditions. *J Orthop Res, 19*(6), 1185-1191.
van der Westhuizen, E. T., Halls, M. L., Samuel, C. S., Bathgate, R. A., Unemori, E. N., Sutton, S. W., et al. (2008). Relaxin family peptide receptors - from orphans to therapeutic targets. *Drug Discov Today, 13*(15-16), 640-651.
Van Lunen, B. L., Roberts, J., Branch, J. D. & Dowling, E. A. (2003). Association of Menstrual-Cycle Hormone Changes with Anterior Cruciate Ligament Laxity Measurements. *J Athl Train, 38*(4), 298-303.
van Mechelen, W., Hlobil, H. & Kemper, H. C. (1992). Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. *Sports Med, 14*(2), 82-99.
Vicente-Rodriguez, G., Dorado, C., Perez-Gomez, J., Gonzalez-Henriquez, J. J. & Calbet, J. A. (2004). Enhanced bone mass and physical fitness in young female handball players. *Bone, 35*(5), 1208-1215.
Vlak, T. & Pivalica, D. (2004). Handball: The Beauty or the Beast. *Croatian Medical Journal, 45*(5), 526-530.
Wade, G. N., Schneider, J. E. & Li, H. Y. (1996). Control of fertility by metabolic cues. *Am J Physiol, 270*(1 Pt 1), E1-19.
Waldrop, J. (2005). Early identification and interventions for female athlete triad. *J Pediatr Health Care, 19*(4), 213-220.

Waller, K., Reim, J., Fenster, L., Swan, S. H., Brumback, B., Windham, G. C., et al. (1996). Bone mass and subtle abnormalities in ovulatory function in healthy women. *J Clin Endocrinol Metab, 81*(2), 663-668.
Waller, K., Swan, S. H., Windham, G. C., Fenster, L., Elkin, E. P. & Lasley, B. L. (1998). Use of urine biomarkers to evaluate menstrual function in healthy premenopausal women. *Am J Epidemiol, 147*(11), 1071-1080.
Warden, S. J., Saxon, L. K., Castillo, A. B. & Turner, C. H. (2006). Knee ligament mechanical properties are not influenced by estrogen or its receptors. *Am J Physiol Endocrinol Metab, 290*(5), E1034-1040.
Warren, M. P., Brooks-Gunn, J., Fox, R. P., Holderness, C. C., Hyle, E. P., Hamilton, W. G., et al. (2003). Persistent osteopenia in ballet dancers with amenorrhea and delayed menarche despite hormone therapy: a longitudinal study. *Fertil Steril, 80*(2), 398-404.
Warren, M. P. & Perlroth, N. E. (2001). The effects of intense exercise on the female reproductive system. *J Endocrinol, 170*(1), 3-11.
Wedderkopp, N., Kaltoft, M., Lundgaard, B., Rosendahl, M. & Froberg, K. (1997). Injuries in young female players in European team handball. *Scand J Med Sci Sports, 7*(6), 342-347.
Wedderkopp, N., Kaltoft, M., Lundgaard, B., Rosendahl, M. & Froberg, K. (1999). Prevention of injuries in young female players in European team handball. A prospective intervention study. *Scand J Med Sci Sports, 9*(1), 41-47.
Wells, C. L. (1991). *Women, sports, and performance* (Second Edition ed.). Champaign (IL): Human Kinetics
Wentorf, F. A., Sudoh, K., Moses, C., Arendt, E. A. & Carlson, C. S. (2006). The effects of estrogen on material and mechanical properties of the intra- and extra-articular knee structures. *Am J Sports Med, 34*(12), 1948-1952.
Wilcox, A. J., Dunson, D. & Baird, D. D. (2000). The timing of the "fertile window" in the menstrual cycle: day specific estimates from a prospective study. *Bmj, 321*(7271), 1259-1262.
Williams, N. I., Caston-Balderrama, A. L., Helmreich, D. L., Parfitt, D. B., Nosbisch, C. & Cameron, J. L. (2001). Longitudinal changes in reproductive hormones and menstrual cyclicity in cynomolgus monkeys during strenuous exercise training: abrupt transition to exercise-induced amenorrhea. *Endocrinology, 142*(6), 2381-2389.
Williams, N. I., Young, J. C., McArthur, J. W., Bullen, B., Skrinar, G. S. & Turnbull, B. (1995). Strenuous exercise with caloric restriction: effect on luteinizing hormone secretion. *Med Sci Sports Exerc, 27*(10), 1390-1398.
Winters, K. M., Adams, W. C., Meredith, C. N., Loan, M. D. & Lasley, B. L. (1996). Bone density and cyclic ovarian function in trained runners and active controls. *Med Sci Sports Exerc, 28*(7), 776-785.
Wojtys, E. M., Huston, L. J., Boynton, M. D., Spindler, K. P. & Lindenfeld, T. N. (2002). The effect of the menstrual cycle on anterior cruciate ligament injuries in women as determined by hormone levels. *Am J Sports Med, 30*(2), 182-188.
Wojtys, E. M., Huston, L. J., Lindenfeld, T. N., Hewett, T. E. & Greenfield, M. L. (1998). Association between the menstrual cycle and anterior cruciate ligament injuries in female athletes. *Am J Sports Med, 26*(5), 614-619.
Woo, S. L., Abramowitch, S. D., Kilger, R. & Liang, R. (2006). Biomechanics of knee ligaments: injury, healing, and repair. *J Biomech, 39*(1), 1-20.
Woo, S. L., Debski, R. E., Zeminski, J., Abramowitch, S. D., Saw, S. S. & Fenwick, J. A. (2000). Injury and repair of ligaments and tendons. *Annu Rev Biomed Eng, 2*, 83-118.
Woodford-Rogers, B., Cyphert, L. & Denegar, C. R. (1994). Risk Factors for Anterior Cruciate Ligament Injury in High School and College Athletes. *J Athl Train, 29*(4), 343-346.

Yde, J. & Nielsen, A. B. (1990). Sports injuries in adolescents' ball games: soccer, handball and basketball. *Br J Sports Med, 24*(1), 51-54.
Yu, B., Kirkendall, D. & Garrett, W. (2002). Anterior cruciate ligament injuries in female athletes: anatomy, physiology, and motor control. *Sports Medicine & Arthroscopy Review, 10*, 58-68.
Yu, W. D., Liu, S. H., Hatch, J. D., Panossian, V. & Finerman, G. A. (1999). Effect of estrogen on cellular metabolism of the human anterior cruciate ligament. *Clin Orthop Relat Res*(366), 229-238.
Yu, W. D., Panossian, V., Hatch, J. D., Liu, S. H. & Finerman, G. A. (2001). Combined effects of estrogen and progesterone on the anterior cruciate ligament. *Clin Orthop Relat Res*(383), 268-281.
Zantop, T., Herbort, M., Raschke, M. J., Fu, F. H. & Petersen, W. (2007). The role of the anteromedial and posterolateral bundles of the anterior cruciate ligament in anterior tibial translation and internal rotation. *Am J Sports Med, 35*(2), 223-227.
Zazulak, B. T., Paterno, M., Myer, G. D., Romani, W. A. & Hewett, T. E. (2006). The effects of the menstrual cycle on anterior knee laxity: a systematic review. *Sports Med, 36*(10), 847-862.
Zebis, M. K., Bencke, J., Andersen, L. L., Dossing, S., Alkjaer, T., Magnusson, S. P., et al. (2008). The effects of neuromuscular training on knee joint motor control during sidecutting in female elite soccer and handball players. *Clin J Sport Med, 18*(4), 329-337.
Zeller, B. L., McCrory, J. L., Kibler, W. B. & Uhl, T. L. (2003). Differences in kinematics and electromyographic activity between men and women during the single-legged squat. *Am J Sports Med, 31*(3), 449-456.
Zinaman, M. J. (2006). Using cervical mucus and other easily observed biomarkers to identify ovulation in prospective pregnancy trials. *Paediatr Perinat Epidemiol, 20 Suppl 1*, 26-29.

Chapter 3

Injury and Gynæcological Profiles of Female Handball Players

3.1 Introduction

In Portugal, female handball started only in 1975, one year before it has officially entered the Olympic Games, and today it is one of the most practiced sports (10 759 female players/5 200 000 inhabitants). Even so, the Portuguese male teams have international careers more expressive in comparison to female teams, which may be partially explained by a higher number of males playing handball, as well as their opportunity to play at higher level of competition, along with a more organized and (semi)professional handball team (*e.g.* Portuguese Professional League).

The reviewed studies, about injury incidence and prevalence in handball practice, have revealed an injury pattern linked to the specific traits of this sport. The upper and lower joint extremities are the most affected structures, both by overstress and by physical contact. Among the most frequent injuries in handball, the knee injuries have been cause for an increasing concern and an in depth study of their causes, which is reasonable given their nature, severity, and consequences. To our understanding, this type of information is important also for coaches, who are responsible and interested in promoting the safety and physical integrity of the players. Handball coaches, well informed about the injury problems, are in a better position to adopt efforts for preventing injuries, and to make educated decisions about reintegration - in the training process and competition - of clinically recovered players, and therefore decreasing the likelihood of reinjury in players.

Study purposes: (a) to investigate the injury profile of Portuguese handball players and its external mechanisms; (b) to evaluate in detail the circumstances of ACL injury and consequences; and (c) to characterize the gynæcological profile of female handball players.

3.2 The Portuguese handball population

The number of federated handball players has increased over the last few years being, between 1996 and 2003, the most practised sport after soccer (2005). However basic statistical information is not published timely by both the Portuguese Sports Institute (IDP) and the Portuguese Handball Federation (FPA). Comparing the total amount of handball players by sex (table 3.1) there are twice as many male players. The asymmetry between sexes increases in the most competitive categories, such as juvenile, juniors and seniors, and is less pronounced in the other categories. Possibly these numbers do not precisely reflect the reality in the youngest categories, as in many regional associations the young players initiating handball practice are reported to schools and not to organized handball clubs. This means that probably the official numbers of handball players in Portugal are artificially inflated.

Table 3.1 The categorised number of players in the 2006-07 season

Players	Seniors Adults	Juniors Age 16 to18	Juvenile Age14 to16	Starters Age 12 to14	Infants Age 10 to12	Bambis and Minis < age 10	Sum
Male	1 730	1 002	1 959	2 786	3 199	7 477	19 765
Female	463	360	804	1 274	3 135	4 723	10 759
Sum	2 193	1 362	2 763	4 060	6 334	13 812	30 524
% female	21.1	26.4	29.1	31.4	49.5	34.2	35.2

Table 3.2 shows the demographics of Portuguese handball. The Porto regional handball association has the highest number of female handball players in the most competitive categories, such as seniors, juniors and juveniles. Moreover many of the female players registered in the Braga regional handball association are actually from a club in Porto, which was created via political interests. If the correction regarding the number of players from this club into Porto's association is introduced then, the proportion of players from this association in relation to all Portuguese female players is as follows: seniors 28%; juniors 24.5%; and juveniles 24.4%.

Table 3.2 Number of female players by regional associations in the 2006-07 season

	CATEGORIES						
Regional Association	*Seniors* Adults	*Juniors* Age 16 to18	*Juvenile* Age14 to16	*Starters* Age 12 to14	*Infants* Age 10 to12	*Bambis and Minis* > age 10	Sum
Algarve	13	17	16	35	101	135	317
Aveiro	56	73	103	159	161	909	1 461
Beja	0	0	21	1	6	9	37
Braga	11	11	22	22	17	20	103
Bragança					1	4	5
Castelo Branco	15		1	16	5	34	71
Coimbra	12			5	15	103	135
Evora				2	64	21	87
Guarda				21	88	182	291
Açores			70	51	69	24	214
Leiria	57	29	76	113	361	381	1 017
Lisboa	64	73	104	369	1038	519	2 167
Madeira	75	47	80	92	184	131	609
Portalegre			14	36	64	154	268
Porto	114	78	174	170	205	101	842
Santarém	16	1	24	35	67	36	179
Setúbal	13	19	54	43	190	363	682
Viana do Castelo			9	23	89	855	976
Vila Real			2	31	304	254	591
Viseu	17	12	34	50	106	488	707
Sum	463	360	804	1274	3135	4723	10 759

3.3 Subsidiary studies

3.3.1 Preliminary study

As a preparation for the main studies, a few small scale tests were done, both for developing the tools and for obtaining rough estimates of the population profile (for devising a sample methodology and size). Much of this was done in a monographic study conducted by another researcher (Seabra, 2003), under the orientation of the advisor of this thesis and our co-orientation. It focused on a small sample from the Portuguese premiere league: 112 players who answered and returned a custom made retrospective questionnaire (from a total of 153 given). This was applied during the 2001-02 and 2002-03 seasons. The main conclusions of this study were published (Seabra *et al.*, 2004) and presented in the *1st World Congress on Sports Injury Prevention* (Seabra *et al.*, 2005). As this is not part of our thesis and has been fully published, we will just mention it briefly. Essentially, this study revealed that the lower limb accounted for most of the reported injuries (59%) and that 10% of the players had already suffered an ACL injury.

Then, a full-blown project was designed (table 3.3). Some of the field work was done with the help of other researchers and those parts have been presented in a few monographic studies (Fernandes, 2007; Carvalho, 2004) and in the *1st World Congress on Sports Injury Prevention* (Carvalho *et al.*, 2005). The custom-made questionnaire was, later on, adapted and used for other injury studies in the Sports Faculty of Porto, namely in Football and Volleyball.

Table 3.3 Project design regarding seasons involved, samples and methods

Season	Criteria for selecting the players for participate	Methods
2003-04	Teams participating in the 1st National Division Top teams of the regional competition from AAP (2nd Division) Young players from the clubs of the adult teams reported earlier	Injuries before 2003-04 were documented retrospectively Injuries during 2003-04 were documented prospectively
2004-05	*idem*	Injuries before 2004-05 were documented retrospectively Injuries during 2004-05 were documented prospectively Quality control by a clinical evaluation of a sub-sample
2006-07	Teams participating in the 1st National Division Teams with better level competing in the regional competition from AAP (2nd division) Young Players from National Teams (Juniors A/B) Young players from clubs competing in the AAP Young players selected for Regional Selections (Porto, Lisboa, Madeira, Leiria, Aveiro, Algarve).	Injuries until 2007 were documented in a retrospective, cross-sectional study

3.3.2 Clinical evaluation

3.3.2.1 Methods

A group of handball players were clinically examined in the Sport Faculty of Porto by an experienced orthopedic surgeon. During this evaluation, the players were asked to recall all injuries sustained and health related complaints during their handball career, which were documented in a proper form. The surgeon examined any complaints in detail and investigated further (if necessary by using complementary means of diagnostic) the injuries reported by the players or any others he found.

Additional clinical information was assessed regarding leg-length discrepancies, spine deformities and knee alignment angles.

For clinical assessment of spine deformities the differences between the axial back surface rotation amplitude in standing versus forward bending posture (*bending test*) was considered. The level of deformity on the axial back surface rotation of the lumbar and thoracic spine (back shape) was evaluated by visual observation, as well as the lateral vertebral translation in standing posture. This test is well described for the routine screening of the idiopathic scoliosis (Massada, 2001).The examiner was aware of factors that have been described as affecting the sensitivity and specificity of the test, such as leg length inequality (Hackenberg *et al.*, 2006). Whenever necessary the players were advised for complementary diagnostic exams (*e.g.* x-Ray).

For estimating the extent of the asymmetry between legs, the examiner used a method based on palpation of the iliac crests in bipedal standing (Massada, 2001). The examiner crouched in front of the player and visually compared the side level of the iliac crests. Next, the length side differences were visually assessed comparing the distance between the anterior inferior iliac spine and the internal malleolus with the player lying on the back (supine).

Clinically the knee alignment angle is defined as the angle formed by the axis from the anterior superior iliac spine to the middle point of the patella and by the axis from that point to the centre of the ankle joint[1] (Massada, 2001). The angle was measured by a transparent universal goniometer aligned against the mentioned anatomic landmarks. Each player was in a standard position, lying on the back, so that the lower extremities were fully extended. The hip and ankle joints visually align with minimal distortion.

[1] This is very similar, but not the same, as the Q-angle.

3.3.2.2 Results

Sixty-two subjects were clinically evaluated. The sample can be briefly characterized by figures 3.1 to 3.4 and tables 3.3 and 3.4, below.

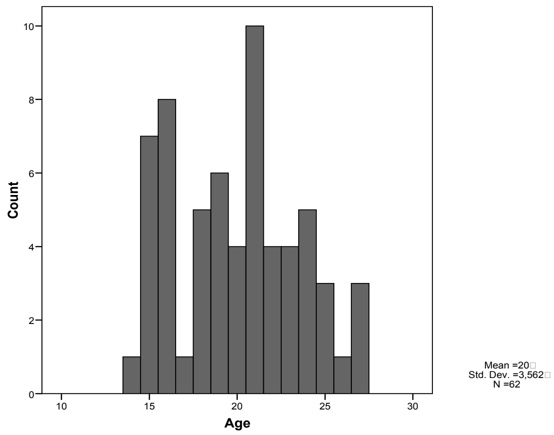


Figure 3.1 Age in years

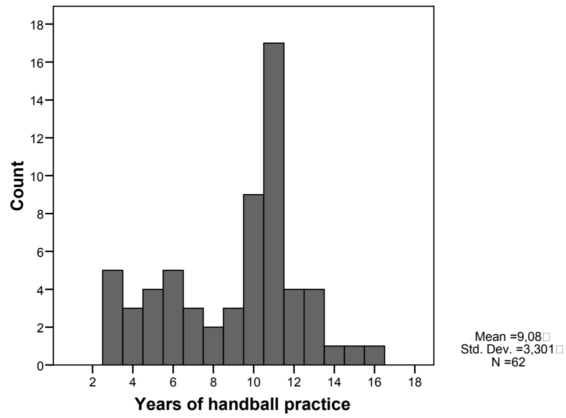


Figure 3.2 Years of handball practice

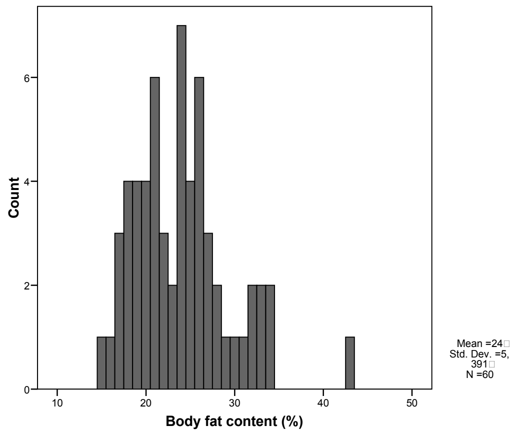


Figure 3.3 Body fat content (w/w)

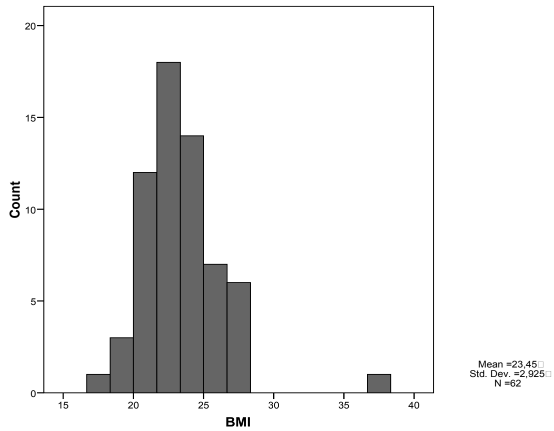


Figure 3.4 Body mass index (kg/m^2)

Table 3.4 Playing position distribution

Positions	N	%
Goalkeeper	8	13
Field player	47	76
Pivot	3	5
Missing/undefined	4	6
Sum	62	100

Table 3.5 Hand dominance

Dominance	N	%
Left dominance	4	6
Right dominance	55	89
missing/undefined	3	5
Sum	62	100

A brief summary of the clinical evaluation results are presented in tables 3.6 and 3.7.

Leg length discrepancies were recorded whenever noticeably paired limbs were unequal (Gurney, 2002). Structurally this can result from changes in length of any of the lower limb segments (Walsh *et al.*, 2000). Based on the clinic assessment, 73% of the players presented lower limb dissymmetry (table 3.6). Massada (2001) examined 362 young male handball players and found a smaller prevalence (39.5%) of lower limb asymmetries. This structural condition is also common in the general population, with a prevalence of 40% to 70%, as reviewed by Gurney (2002). We did not investigate any dynamical or functional implications of these leg asymmetries although significant leg length discrepancies may induce flexion of the knee on the longer leg (Walsh *et al.*, 2000). We were unable to find any studies about any possible dynamic compensatory mechanism during landing or plant-and-cut movements, but it seems obvious that there could exist implications at the pelvis, knee, ankle and/or column.

The small sample size and the small incidence of *severe knee injuries* (including ACL injuries) makes it pointless to perform any sort of statistical inference with this data.

Table 3.6 Leg length comparisons

Leg-length	N	%
Right Leg = Left Leg	16	26
Right Leg > Left Leg	24	39
Right Leg < Left Leg	21	34
Unspecified dissymmetry	1	2
Sum	62	100

Table 3.7 Upper body status

Conditions	N	%
Scoliosis	16	26
Kyphosis / hyperlordosis	3	5
Back pain	26	42
Unilateral muscular hypertrophy (*king kong arm*)	13	21
Dead arm	2	3
Normal	27	44
missing	8	13
Sum	95	100

The upper body status was also clinically examined (in standing position and with the forward bending test), as summarized in table 3.7. In 40% of the players axial anomalies were clinically detected. Back pain (particularly the low back pain) was reported by 42% of the players. The female athlete is at considerable risk of back injury and back pain, which is also quite common in the general female population (Omey *et al.*, 2000). We found idiopathic scoliosis in 26% of the players, a much higher account than in young male handball players (8.6%) (Massada, 2001). In the general population, scoliosis tends to be more common in girls and to progress more than in boys, as reviewed by Roach (Roach, 1999).

Twenty-one percent of the players presented unilateral muscular hypertrophy, which is likely to be related with hand-dominance in handball throwing techniques, which may also be indicative of a lack of specific strength training compensation. Massada (2001) found a much higher percentage of young male handball players without axial anomalies (73.2%) than we did, which is probably due to age, experience and sex differences.

The expectable dependence between the back pain condition and the playing position or BMI could not be verified in our sample (tables 3.8 to 3.10, figure 3.5)[2].

Table 3.8 Playing position *vs.* back pain condition

Back pain	No	Yes	sum
Goalkeeper	3	5	8
Field player	29	18	47
Pivot	2	1	3
sum	34	24	58

Table 3.9 χ^2 test for the association *playing position – back pain condition*

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.74	56	0.42
Likelihood Ratio	1.71	56	0.43
Linear-by-Linear Association	1.43	1	0.23
N of Valid Cases	58		

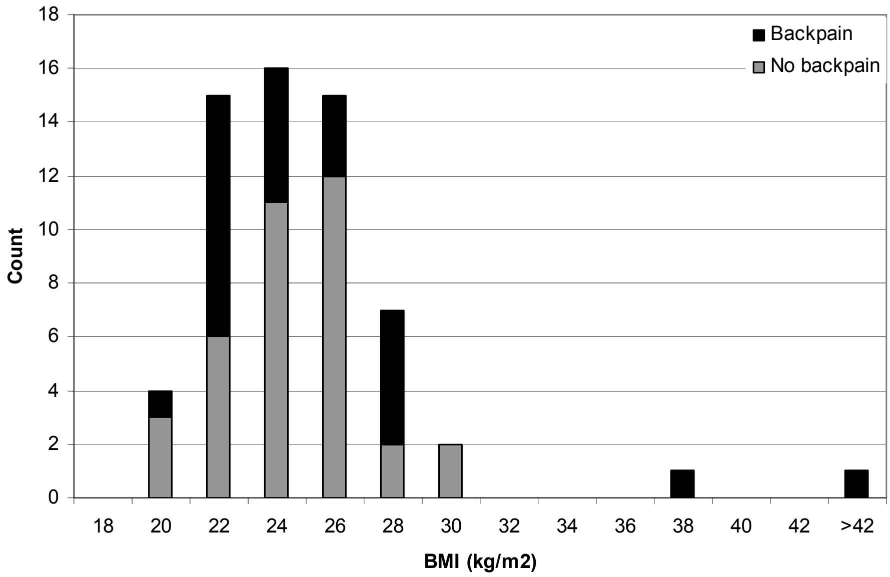


Figure 3.5 Backpain condition distribution over BMI

Table 3.10 Association tests for BMI - *backpain condition*

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	62	60	0.40
Likelihood Ratio	84.3	60	0.02
Linear-by-Linear Association	0.05	1	0.82
N of Valid Cases	62		

[2] Interestingly enough the backpain-BMI association is very probable under a G test (maximum likelihood) but very unlikely under the standard χ^2 and Mantel-Haenszel tests. This has to do with the long tail distribution.

An association study between lower limb dissymmetry and column status is presented in tables 3.11 and 3.12 and figure 3.6. There is no reason to support the thesis of a mutual association between these two variables with our data although the small sample size makes for a low power test.

Table 3.11 Cross tabulation: column axial health *vs* lower limb dissymmetry

Lower limb length	equal	unequal	Sum
Healthy column	6	21	27
Non healthy column	10	25	35
Sum	16	46	62

Table 3.12 χ^2 association test for column health *vs.* lower limb dissymmetry

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	0.32	60	0.57
Likelihood Ratio	0.32	60	0.57
Linear-by-Linear Association	0.32	1	0.57
N of Valid Cases	62		

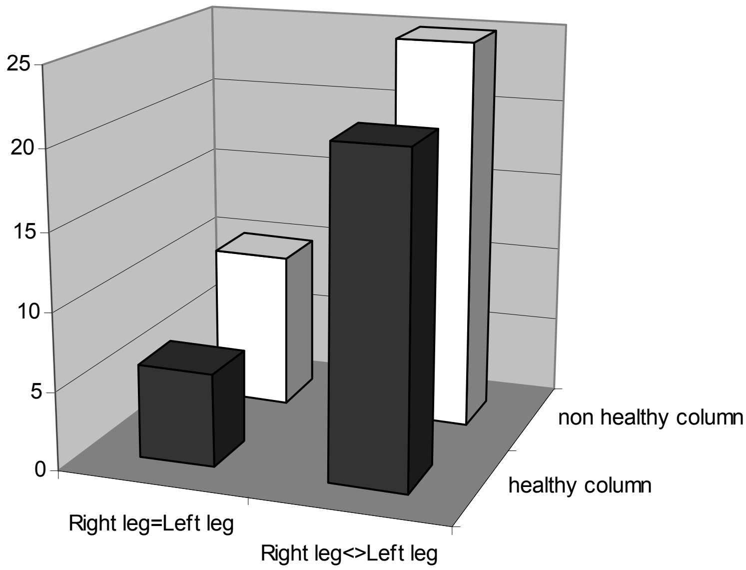


Figure 3.6 Column axial health *versus* lower limb dissymmetry

An analysis of the anatomical knee angle (figure 3.7) shows that there is a significant difference between the two legs with the right leg exhibiting a higher angle (3.8º±4.1º) than the left one (3.1º±4.9º) for a p_c<0.06%. As the values for the general population are unknown and the difference is much smaller than the inter-subject variation, this is probably irrelevant. Also Massada (2001) found a right leg knee angle (5.0º±3.6º) higher than the left one (4.5º±3.5º) in young male handball players.

Sex differences in lower extremity anatomical and postural characteristics have been intensively investigated (Shultz *et al.*, 2008; Nguyen & Shultz, 2007; Shultz & Nguyen, 2007). Females have greater pelvic tilt, internal thigh rotation, knee *valgus*, and gene *recurvatum* (Nguyen & Shultz, 2007; Shultz & Nguyen, 2007). While the literature is consensual about sex-differences on static lower extremity alignment, less agreement is seen about its possible influence on dynamic movements and knee injury risk.

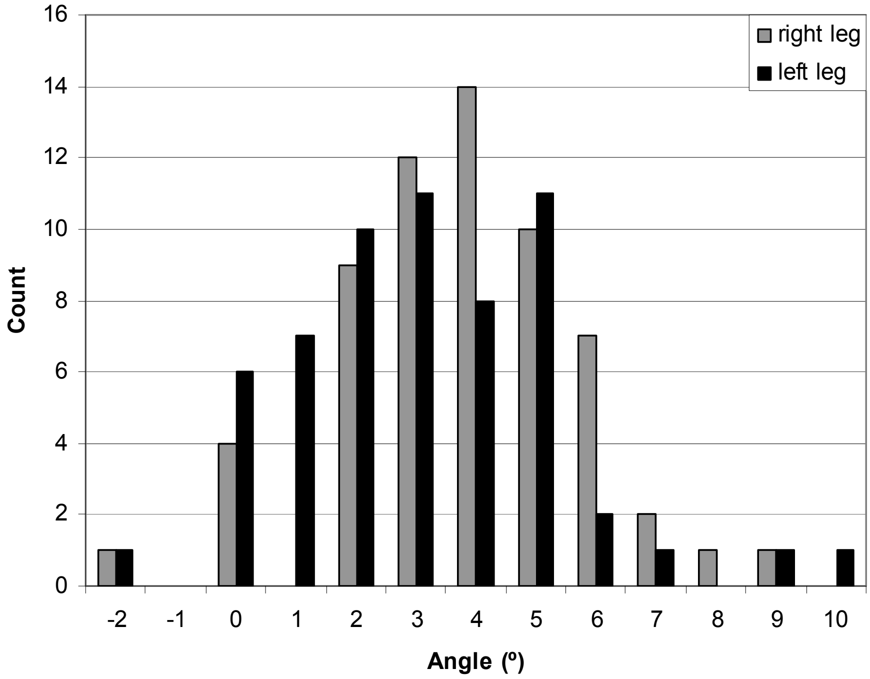
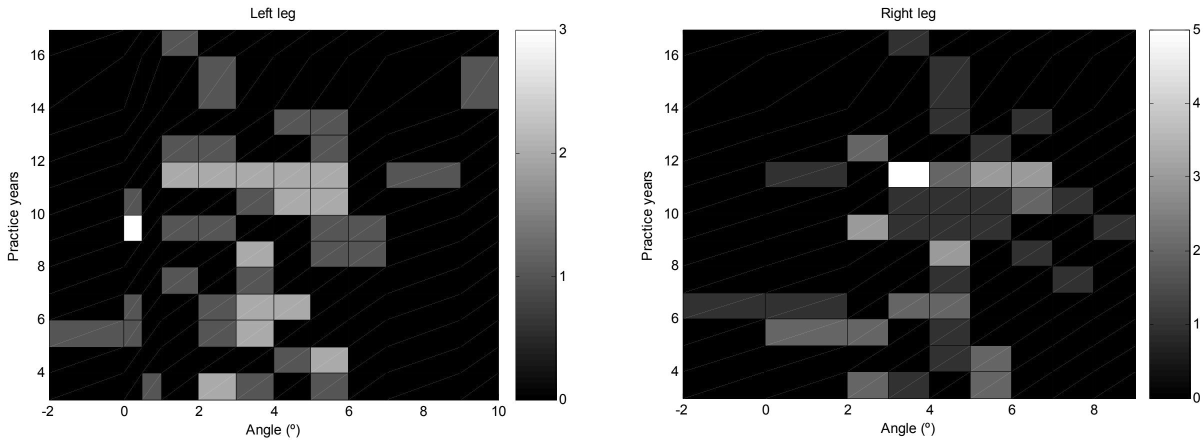


Figure 3.7 Anatomical knee angle distribution

The evolutionary comparison of the anatomical knee angle with the time of handball practice (less sensitive to the normal subjects' growth) shows no obvious pattern and no statistically significant association (figures 3.8 and 3.9, tables 3.13 and 3.14).



Figures 3.8 & 3.9: Color coded *knee alignment angle* vs *handball practice years* (left & right legs)

Tables 3.13 & 3.14 χ^2 association tests:*knee alignment* vs *handball practice years* (left & right legs)

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	134.9	143	0.67
Likelihood Ratio	98.59	143	1.00
Linear-by-Linear Association	1.5	1	0.23
N of Valid Cases	59		

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	141.3	117	0.06
Likelihood Ratio	102.6	117	0.83
Linear-by-Linear Association	3.4	1	0.06
N of Valid Cases	61		

3.4 Methods

Experimental design

The detailed thesis project was submitted to (and approved by) the Scientific Council of the Sports Faculty. It was not submitted to the Porto University ethical committee as it was just being founded.

Confidentiality of all information gathered was always ensured, but the players were asked to give their names and contacts in the questionnaires in case of additional information being needed or to follow the players in the next season. To each questionnaire/player a code number was attributed, and the data was anonymously introduced in the database. After the conclusion of these studies the key (code ↔ identity) was destroyed.

At the start of the first studied season (2003-04) all the selected clubs were invited to collaborate in the study. Specific meetings (face-to-face) were done in order to explain the study aims and proceedings to club directors, coaches, doctors and/or physiotherapists. Guide notes on how to complete the questionnaires were made available to the medical and technical staff of each club/team. Personal commitment was asked of coaches and physiotherapists to prospectively document the injuries which occurred during the season.

We noticed that just a small number of teams had a doctor or physiotherapist (almost only those in the senior's category and competing in the 1st national league) and their presence in the matches/training sessions was not always regular. All players who consented to participate were personally informed of the study purposes and procedures (*e.g.* how to report the injuries) and signed an informed consent agreement. Players who were injured at the beginning of the study were not excluded from the sample, but the sustained injury was reported as a previous injury. Players joining or leaving the teams during the season were included in the cohort whenever the individual time exposure was available.

Around August/September 2003, the players were asked to complete the part of the questionnaire on individual information, including sports participation, previous injuries and menstrual cycle characteristics (*follow up 1* – table 3.15).

In case of injury the coaches/physiotherapists were instructed to provide the injured player with an injury registration form, which should have been completed as soon as possible (the provided guide notes asked for a maximum of one week), and attached to the main questionnaire. The players were instructed to record the injury diagnosis made by the doctor or physiotherapist who examined them (usually from the team's club, hospital or insurance company). In the case of a light or minor injury, sometimes the player was examined just by the coach or no examination was done at all.

The proceedings adopted for returning the questionnaires were established considering the structure and organization of each club/team. In most of the cases the clubs' collaborators proposed to deliver the questionnaires at the end of the season (June/July, 2004). The investigator kept regular contact with all teams by telephone and/or email to ensure that the injuries were properly reported.

At the end of the following season, all players were asked to complete *follow up 2* (table 3.15), reporting the individual training and game exposition, all extended periods of injury recovering (more than a week absent from practice) and all surgeries and/or other major treatments undertaken.

The exposure time was also asked to coaches regarding both the team and each player, because we could anticipate that some players had weekly training sessions with other clubs (teams) and/or additional preparation with team selections (regional and/or national).

In the next season (2004-05, *follow up 3*) the procedures adopted were similar, but special commitment was asked from coaches and players. Even though almost all teams invited accepted to participate (except one) in the previous season, there was a considerable number of non-returned questionnaires from some teams (about half of all questionnaires were not returned). In general the compliance of people intervening in the injury registration improved in the season 2004-05.

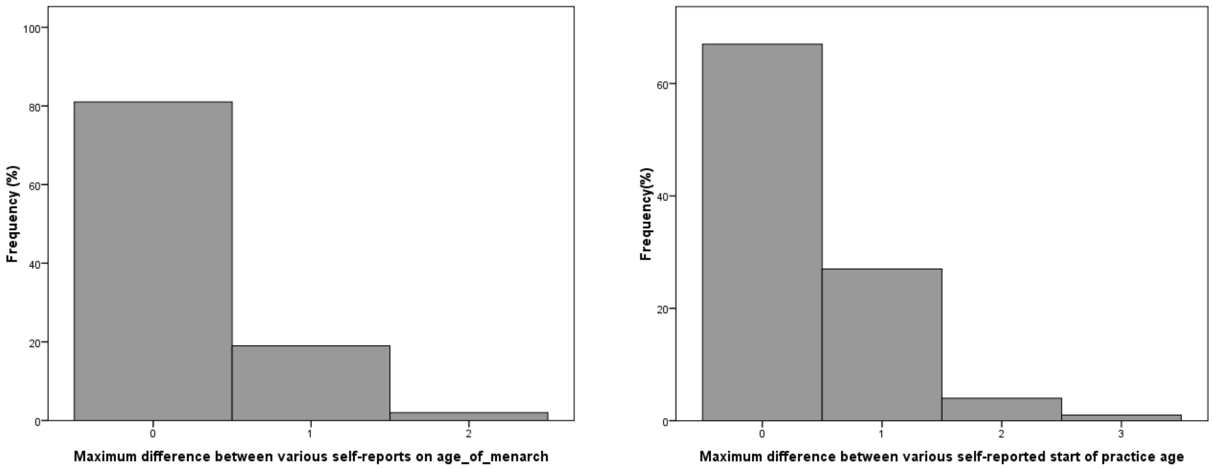
During this 2004-05 season, all athletes already being followed and registered in the Porto regional association, were asked to participate in a clinical examination and anthropometric-functional evaluation at the Sports Faculty (*follow up 4*). Players from other regional associations were also included. They were mainly adult players from the 1st division. This clinical examination was described and analysed above (§3.3.2).

The study was interrupted in the next season and resumed later, in 2006-07, with a new (adapted/modified) questionnaire. The new objective was to do a retrospective, cross-sectional study, on a different, much larger, sample. It covered the handball sports participation and menstrual cycle characteristics and was accompanied by a new set of field measures unrelated to this subject (to be presented in a following chapter).

Injury assessment

For the purpose of this study a questionnaire was developed with formal methodological rules (Hill & Hill, 2002). The starting point was a set of studies about injuries in team sports (Hawkins *et al.*, 2001; Lindblad *et al.*, 1992; Nielsen & Yde, 1988), ACL injuries (Myklebust *et al.*, 1998) and the menstrual cycle characteristics (Haywood *et al.*, 2002; Wells, 1991). The developed questionnaire was then analysed: first the content validation and structural validation by experts in Handball (n=3), Traumatology and Sports Medicine (n=3) and Gynaecology (n=3). Then a pre-test was implemented to evaluate the understanding of questions and to confirm its accuracy for capturing the intended information. For this purpose a group of young handball players (15 juvenile female players) completed the preliminary version of the questionnaire. An individual oral reflection was done considering their impressions about the questionnaire presentation, issues and type of questions, and the requested information about injuries and menstrual cycle. The same group of players completed an identical questionnaire 30 days later to determine the repeatability of the answers in each section of the questionnaire. On average, it took 15 min to complete the questionnaire, but in some cases it took as long as 40 min.

Later on, to access the quality of the subject’s history as self-reported in the questionnaires, a sub-sample of 102 players were asked to refill selected parts of the questionnaires, several times (2 to 4), with time intervals of 1 to 3 years. Two objective questions were studied: age of beginning handball practice and age of menarche. The results are summarized in figures 3.10 and 3.11.



Figures 3.10 & 3.11 Homogeneity of answers to ‘Age of menarche’ & ‘Age at beginning of handball practice’, in years

Although collectively the correlations between the various answers are above 90%, we want to emphasize that, at least, 1/5 to 1/3 of all the objective numerical (integer) answers are wrong, sometimes by about 30% (3 years in around 10).

We have not performed any study on the more subjective questions.

The questionnaires included different type of questions. Closed questions (dichotomous and multiple choice) were developed whenever possible, but some open-ended questions were also introduced, and some contingency questions were also used to avoid asking questions that did not apply to all players or injury types/circumstances.

The final questionnaire structure and the information collected in each section is presented in tables 3.15 and 3.16.

Table 3.15 Main data collected by questionnaire (items)

Follow up 1
Player identification and other information
Date of answering the questionnaire
Name (facultative)
Date of birth
Phone number and email
Dominant arm (right, left or bilateral)
Body mass (kg) and stature (cm)
Sports history
Age of starting to play handball/ years of handball practice
Other sports practiced
Clubs/teams of handball practice
Regional or national selections participation
Details of previous injuries
Season/age of injury
Injury diagnosis (supplied by the doctor or physiotherapist)
Surgery (yes/no)
Time loss
Menstrual cycle characteristics
Age of menarche
Menstrual cycle regularity (always/some times/never)
Average menstrual cycle length (days)
Average of menstrual period (days)
Number of menstrual periods in the last 6 months
Oral contraceptive use: use of oral contraceptives in the last 6 months (yes/no); Age at first oral contraceptive use
Oral contraceptive type (commercial name)
Duration of Oral Contraceptives use (years/months)
Motive(s) for oral contraceptive use and change
Regular medication (yes/no) and their commercial name
Smoking or drinking behaviour (yes/no, regularity and quantity)
Subjective impression during menses in the training/playing context (normal, limited, well, not well)
Menstrual cycle symptoms relative to the last 6 months (presence/absence): irritability, sleep disturbances (poor sleep/sleepy), acne, tension, depression, fatigue and tiredness, increased appetite, decreased appetite, anxiety, headache, joint pain, weight gain, low back pain, decreased concentration, mastalgia, abdominal pain and bloating, dizziness, oily skin, leg pain or fullness, and others
Perception of performance changes relatively to premenstrual phase, menstrual phase and after menstrual phase (no differences, increased performance or decreased performance)
Enable for training or playing by the motive of menstrual cycle issues (yes/no); if answered yes, reasons were asked to be explained
Appendix. Calendar for reporting menstrual cycle days during the season

Table 3.15 (continuation) Main data collected in the developed questionnaire (items)

Follow up 2
Season (2004-03, 2004-05 or 2006-07)
Club/team
Playing categories
Playing position(s) in attack and defense
Number of matches played in the club and national/regional selections
Average match time played in the club and national/regional selections
Sessions/hours of training
Type of floor used for training (artificial, wood with and without air sub-floor system, concrete or other)
Season' injuries (table)
Number of injuries suffered during the season
Injury diagnosis (supplied by the doctor, physiotherapist or others)
Number of episodes by injury
Surgery (yes/no)
Time loss
Function restrictions to play handball normally, at the date of the reported questionnaire (yes/no)

Table 3.16 Main data collected in the injury report form

Injury form
Injury recordable: pathology occurred during handball practice resulting in function limitations to fully train or play
Player's name/identification number
Injury data and time of day
Name of sports location where the injury occurred
A brief written description on the injury context
Injured body part: right, left or not applicable
Localization of injury: head/face, column, thorax, abdomen, pelvis/sacrum, shoulder, upper arm, elbow, forearm, wrist, hand, fingers, thumb, hip/groin, thigh, knee, lower leg/Achilles tendon, ankle, foot/toe
Diagnosis: contusions; injury joint (sprain with or without ligament injury or meniscal, subluxation, luxation); tendinosis injuries (tendinitis and tendon rupture); muscle injuries (contusion and muscle rupture); fractures (acute and stress fracture); injuries in the vertebral column (acute or chronic and located at cervical spine and upper or lower back); skin lesion (laceration); other injuries
Severity of injury: absence from practice in days/months
Recovering from another injury
Recurrent injury (same location and symptoms)
After sustained injury: stopped immediately or continued (with or without physical limitation)
Immediate or postponed medical attention required
Restrictions to full practice
Circumstances: in the club or in a team selection; during match or training; type of match and competition; match importance; match time of injury occurrence; match phase (offensive/defensive); field position when the injury was sustained
With ball, contact with another players or object, repetitive movements, sudden (abrupt) movement, turning, jump (during impulsion or landing), another and not specified
Foul judgment of the player or aggressive behaviour
Referee's sanction of the foul that caused the injury
Type of external support worn when the injury occurred and its specific localisation
Treatment by a doctor or physiotherapist (during training/match or post training/match)

Main definitions

An ***injury*** was defined according to the definition adopted by the European Council and as used by Massada (2001). Players, coaches and physiotherapists were asked to report any physical complaint (injury) resulting from training or matches irrespective of any functional limitation to handball practice.

For reporting an injury sustained during the season, an independent single page (injury report form) was developed, which is described as follows. In the case of an ACL injury sustained before the studied season, the player was asked to recall as much information as possible using an adapted injury form (some questions were excluded; see appendix).

Regarding the mechanism, the injuries were diagnosed as being either ***acute*** - caused by a sudden traumatic event while practising handball - or ***overuse*** - caused by repeated microtrauma without a sudden, single event as it's onset, during the continued handball practice (Fuller *et al.*, 2006; Verhagen *et al.*, 2004).

Regarding the documented absence time from normal handball practice, the injury ***severity*** was classified as slight (0 days), minimal (1 to 3 days), mild (4 to 7 days), moderate (8 to 28 days), severe (> 28 days) and career-ending (Fuller *et al.*, 2006).

An injury was classified as a ***recurrence*** (reinjury) according to the judgment of an orthopaedic surgeon (the supervisor of this study). Each reported injury was categorized by this examiner, according to Fuller *et al.* (2006) and Massada (2001).

Training ***exposure*** was determined regarding the average weekly number of training hours reported by each player multiplied by the number of weeks per season. For collecting individual training exposure we did not take into account the precise amount of sessions lost weekly or time lost during each session. Competition exposure for each team was determined multiplying the number of matches by the official time for each match (which presents some differences regarding the player's categories or tournaments) by 7 players. For this purpose we did not considered the players' time of exclusion during matches.

3.5 Results

3.5.1 Participants

A total of 804 handball players were included in the study. Between the first and the second studied seasons there was an increased numbers of returned questionnaires. In the 2003-04 season the studied players were from the Porto association (11 clubs), Madeira (3), Aveiro (3), Algarve (1) and Lisboa (1). In the 2004-05 season the clubs involved remained the same, with few exceptions: another senior team club was involved (from the Castelo Branco association) and some teams were excluded or included, because their handball practice was ceased or started, most of the times with the same players and coaches (an integral team shift between clubs). In the 2006-07 season the number of players/clubs involved was further extended (37 clubs), mainly because the injuries were documented retrospectively and young players, from the category of *infants*, were added to the sample.

The distribution of players by each playing category is presented in table 3.17, considered the corresponding date of birth; although there is a significant number of players playing/training in more than one category simultaneously. For example, in the 2003-04 season the *starters* were included in the study because they were playing regularly in the superior category (*juveniles*). During the first studied seasons about 14% of the players played and/or trained regularly in more than one category. In the 2006-07 season the number of young players was extended and the fraction of multi-category players increased to 28.4%.

Table 3.17 Players studied by season and playing categories

	2003-04 Season N (%)	2004-05 Season N (%)	2006-07 Season N (%)	Total N
Seniors (Adults)	55 (42.6)	93 (30.2)	97 (19.4)	245
Juniors (age 16 to 18)	30 (23.3)	33 (10.7)	73 (14.6)	136
Juvenile (age 14 to 16)	36 (27.9)	104 (33.8)	109 (21.8)	249
Starters (age 12 to 14)	8 (6.2)	55 (17.9)	131 (26.2)	194
Infants (age 10 to 12)		23 (7.5)	90 (18.0)	113
Sum (%)	129 (100.0)	308 (100.1)	500 (100.0)	937

The following chart (figure 3.12) presents, for each season, the distribution of players studied regarding their age.

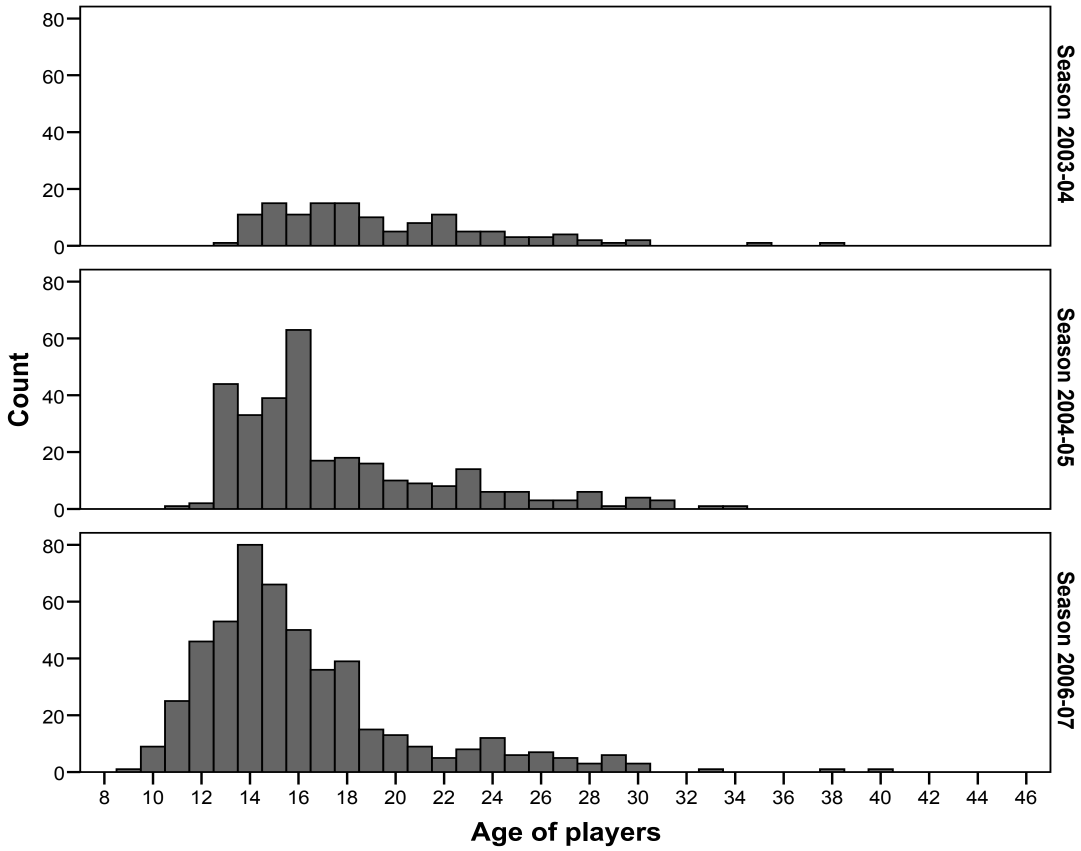


Figure 3.12 Number of players per each category of age relative to studied seasons

The table 3.18 presents the average age of starting to practice handball and the duration of the practice (years) until the beginning of each studied season.

On the senior category, within all analysed groups, the majority were from the first national league (about 65%).

Table 3.18 Demographics

Season	N	Age of the players μ ± SD	Age of start to practice μ ± SD	Years of practice μ ± SD
2003-04	129	19.4 ± 4.6	11.1 ± 2.0	8.0 ± 4.0
2004-05	308	17.5 ± 4.5	11.2 ± 2.5	6.0 ± 4.5
2006-07	500	16.2 ± 4.5	11.7 ± 2.5	5.6 ± 4.5

The number of players participating in some kind of team selection (youth or adult and national and/or regional) during the period of examination is reported in table 3.19.

Table 3.19 Number of players studied per team selection

	2003-04 Season N (%)	2004-05 Season N (%)	2006-07 Season N (%)
National A	4 (3.1)	11 (3.6)	11 (2.2)
National Juniors A	4 (3.1)	6 (1.9)	18 (3.6)
National Juniors B	9 (7.0)	12 (3.9)	19 (3.8)
Regional	8 (6.2)	29 (9.4)	80 (16.0)
Sum (%)	24 (18.6)	58 (18.8)	128 (25.6)

In the first two sample groups about 7% of the players were left-handed. In the third group (2006-07) the fraction was 9%. Overall about 1% to 2% of players were bilateral-handed.

Regarding the playing position, about 40% of the players regularly played in more than one position (we obtain 38 different combinations for the question: *playing position*). Whenever the player reported more than one playing position the answers were set into (re)categorised playing positions, as presented in the following table (3.20).

Table 3.20 Playing position distribution at the beginning of the study

	2003-04 Season N (%)	2004-05 Season N (%)	2006-07 Season N (%)
Goalkeeper	18 (14.0)	42 (13.6)	61 (12.2)
Left wing	7 (5.4)	27 (8.8)	46 (9.2)
Left back	8 (6.2)	26 (8.4)	24 (4.8)
Middle back	11 (8.5)	19 (6.2)	36 (7.2)
Right back	5 (3.9)	14 (4.5)	25 (5.0)
Right wing	12 (9.3)	20 (6.5)	39 (7.8)
Pivot	14 (10.9)	26 (8.4)	41 (8.2)
Back (three playing positions)	17 (13.2)	24 (7.8)	55 (11.0)
Wings (left and right)	7 (5.4)	26 (8.4)	35 (7.0)
2nd line (wings and pivot)	2 (1.6)	8 (2.6)	5 (1.0)
Left side (back and wing)	7 (5.4)	10 (3.2)	17 (3.4)
Right side (back and wings)	2 (1.6)	5 (1.6)	21 (4.2)
1st and 2nd lines (back, wings and pivot)	18 (14.0)	34 (11.0)	71 (14.2)
Non-defined	1 (0.8)	27 (8.8)	24 (4.8)
Sum (%)	129 (100.2)	308 (99.8)	500 (100.0)

Figure 3.13 summarizes the overall amount of playing practice over a season by age and playing category.

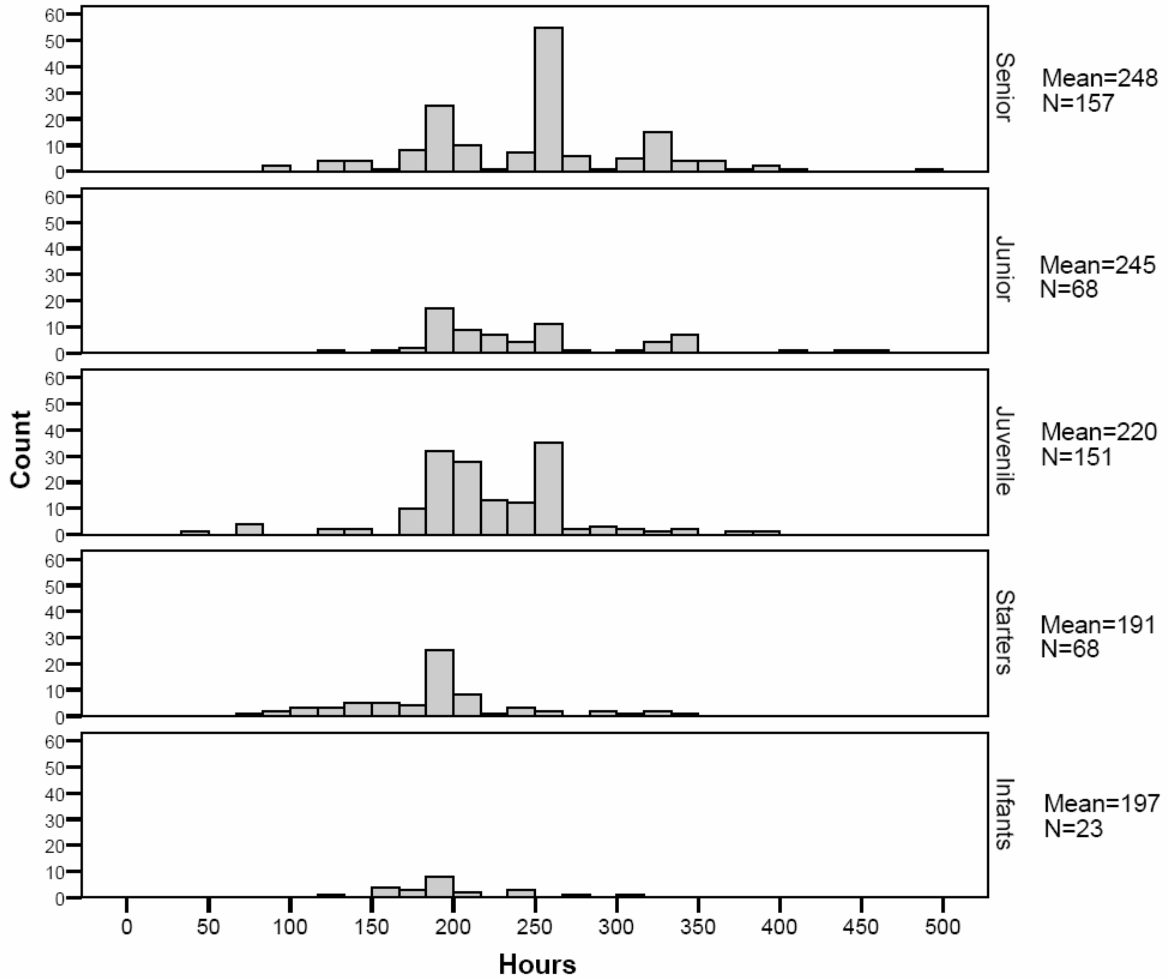


Figure 3.13 Hours of handball practice by age category (one season)

The following table (3.21) shows the ratio of training time to time in matches during the seasons 2003-04 and 2004-05 considering the different age of playing categories.

Table 3.21 Ratio of training to match time

Category	N	Minimum	Maximum	Mean	Std. Deviation
Senior	157	5.5	41.1	15.6	6.3
Junior	68	4.2	82.3	13.4	10.5
Juvenile	150	3.6	82.6	16.3	11.3
Starters	68	1.9	82.6	16.2	11.6
Infants	23	8.8	64.0	25.9	11.1
all	466	1.9	82.6	16.1	9.8

The artificial floor is the most used type of training surface (overall 43% to 51%), followed by wood (32% to 40%) as presented in the table 3.22.

Table 3.22 Type of floor used during the handball training

	2003-04 Season N (%)	2004-05 Season N (%)	2006-07 Season N (%)
Artificial	47 (36.4)	107 (34.7)	246 (49.2)
Wood with air sub-floor system	16 (12.4)	90 (29.2)	116 (23.2)
Wood without air sub-floor system	23 (17.8)	12 (3.9)	39 (7.8)
Concrete	1 (0.8)	4 (1.3)	49 (9.8)
Artificial and wood with air sub-floor system	0 (0.0)	1 (0.3)	5 (1.0)
Wood and artificial	11 (8.5)	24 (7.8)	39 (7.8)
Wood with/without air sub-floor system	0	0	2 (0.4)
Not reported	31 (24.0)	70 (22.7)	4 (0.8)
Sum (%)	129 (99.9)	308 (99.9)	500 (100.0)

3.5.2 Injury measures

A total of 286 injuries were reported from the two first seasons (103 injuries in the 2003-04 season and 183 injuries in the 2004-05 season).

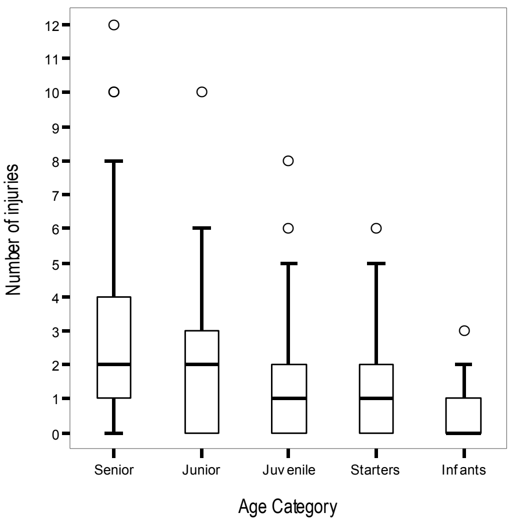
We computed an overall incidence of 3.0 injuries per 1000 player-hour of handball practice, which corresponds to an incidence of 3.3 injuries per 1000 hours of handball practice (95% CI: 2.7- 3.9) in the first season and an incidence of 2.6 injuries per 1000 hours of handball practice in the second season (95% CI: 2.2- 3.1) (see table 3.23). The injury incidence between these two groups was significantly different (according to Mann-Whitney *U* Test; $p \leq 1.2\%$). When comparing the central tendency between consecutive seasons, only for *juniors* and *starters* were there significant differences in the injury incidence (see table 3.23). However, the *starters* groups are very dissimilar in size (8 *vs.* 55) and in the 2003-04 season all the *starters* were playing in two categories: *starters* and *juveniles*. Using the Kruskal-Wallis test, the differences between categories were marginally non significant for the 2003-04 season (p>7%) but were marginally significant in the 2004-05 season (p<5%). Considering the two seasons collectively, a Kruskall-Wallis analysis and corresponding *post-hoc* analysis revealed that the injury incidence follows the increasing age categories exactly, with 3 homogeneous groups with overlap: from age 10 to 16 years (1.25 injuries per 1000 player-hour), from 12 to 18 years and from 16 to adults (3.67 injuries per 1000 player-hour).

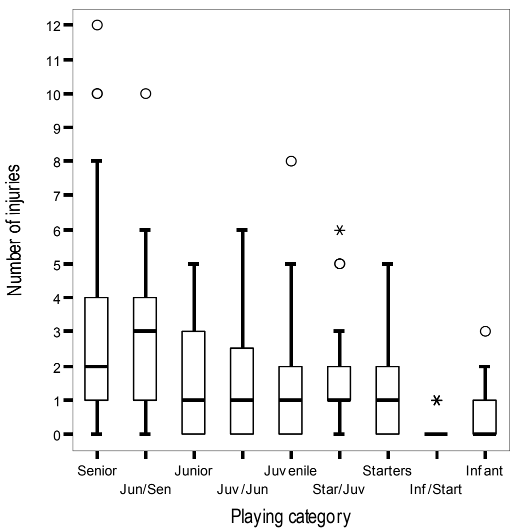
In the 2006-07 season, a total of 759 injuries were registered regarding all cumulative years of handball practice (data collected between January and June of 2007), giving an average injury rate of 1.5 injuries per player. Statistically significant differences were found between the injury incidences of the various categories defined by both *age category* and *playing categories* (not necessarily the same) according to a Kruskal-Wallis test (p<0.1%). The following pictures (3.14 and 3.15) present the distribution of injuries (frequencies) regarding the two criteria for classifying the categories. The injury number is homogeneous from starters to juniors in the playing category criteria (Kruskal Wallis p>7.5%) but is different onwards (p<2.3%). In the junior age category the number of injuries is higher (p<2.3%) for those playing also at the senior level than those playing only at the junior level.

Table 3.23 Incidence and prevalence of injuries by season and playing category

	Incidence (numbers of injuries/1000 hours practice)			Numbers of previous injuries per player
	2003-04 Season µ± SD	2004-05 Season µ± SD	Mann-Whitney U (p-value)	Until 2006-07 Season µ± SD
Seniors (Adults)	3.7 ± 4.0	3.7 ± 3.6	0.70	2.7 ± 2.4
Juniors (age 16 to 18)	3.2 ± 2.9	2.0 ± 3.4	0.04*	2.2 ± 2.0
Juvenile (age 14 to 16)	2.4 ± 3.3	2.9 ± 4.8	0.80	1.4 ± 1.6
Starters (age 12 to 14)	4.8 ± 8.0	1.4 ± 2.8	0.00*	1.2 ± 1.3
Infants (age 10 to 12)		1.3 ± 2.8		0.3 ± 0.6
Total	3.3 ± 3.5	2.6 ± 4.0	0.01*	1.5 ± 1.8

*Significantly different (*p* ≤ .05).





Figures 3.14 & 3.15 Injuries previous to the season 2006-07, by category

Regarding the playing positions reported at the beginning of the study, no significant differences were found related to injury incidence or injury rate (*p* >0 .05), for all seasons.

3.5.3 Injury type, localization and severity

A total of 1227 injuries were computed comprising of all seasons evaluated and methods used (prospective study, retrospective study and complementary clinical examination).

An overview of the overall number of injuries according to anatomic localization, and within each season, is shown in table 3.24. The location of injuries were recorded using the categories proposed by Fuller *et al.* (2006), with adjustments proposed by the supervisor of this study and some others from Massada (2001).

In the column identified as "overall" all injuries documented prospectively and retrospectively are accounted, including data from the clinical examination as well as injuries that occurred during the season 2005-06 (in the group of players participating in the hormonal study). In the columns labelled *season 2003-04* and *2004-05* only the injuries prospectively documented in these seasons were considered.

3.24 Injuries according to anatomic localization and within studies

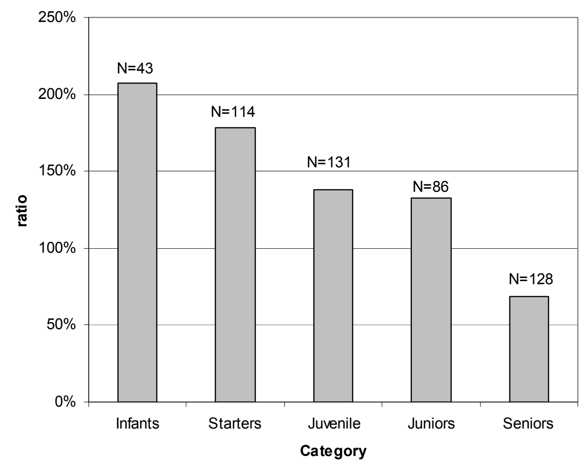
	Overall	SEASONS	
Injured body part	N (%)	2003-04	2004-05
Head/face	*24 (2.0)*	*1 (1.0)*	*8 (4.4)*
Trunk (including neck)	*110 (9.0)*	*4 (3.9)*	*15 (8.2)*
Back/Column	97	4	13
Sternum/ribs	6	0	0
Abdomen	2	0	0
Pelvis/sacrum	5	0	2
Upper extremity	*403 (32.8)*	*32 (31.1)*	*58 (31.7)*
Shoulder/clavicle	125	13	11
Upper arm	5	0	2
Elbow	65	6	9
Forearm	4	1	1
Wrist	38	1	3
Hand	13	1	2
Finger	107	7	23
Thumb	46	3	7
Lower extremity	*686 (55.9)*	*65 (63.1)*	*102 (55.7)*
Hip/groin	10	2	3
Thigh	43	2	11
Knee	250	23	33
Lower Leg	26	4	5
Ankle/Achilles' tendon	345	34	48
Foot/toe	12	0	2
Missing / not specified	*4 (0.3)*	*1 (1.0)*	*0 (0.0)*
Sum (%)	*1227 (100.0)*	*103 (100.1)*	*183 (100.0)*

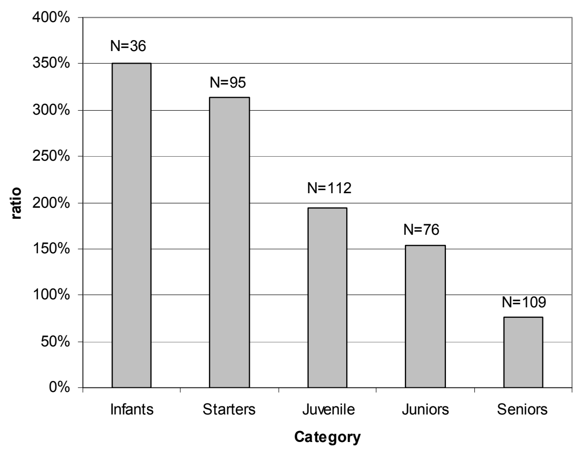
Considering all documented injuries (1227), the lower extremity was the most commonly injured body region, with 686 injuries (55.9% of all injuries), followed by the upper extremity, 403 injuries (32.8% of all injuries) (for detail, see Table 3.24). About the same proportion is observed regarding the prospective two-season cohort study (2003-04 and 2004-05).

Regarding the injury mechanism about 72% (879/1227) were classified as being acute, and 26% (315/1227) as overuse (in 2.7% of the cases their nature was not specified) (table 3.25).

The most injured areas were the ankle (28%, of which 94% were acute), knee (20%, of which 68% were acute), shoulder (10%, of which 66% were from overuse), finger (9%, of which 99% were acute), back (8%, of which 79% are from overuse) and elbow (8%, of which 78% were from overuse).

Figure 3.16 presents the ratio of ankle to knee injuries by age category, accounting all reported injuries while figure 3.17 presents the same but considering only acute injuries.





Figures 3.16 & 3.17 Ratio of ankle to knee injuries by age category, overall & just acute

Table 3.25 displays an overview of the number and proportion of injures occurred in several anatomic areas in consequence of a sudden traumatic event (acute) or caused by repeated microtrauma (overuse).

3.25 Overview of all computed injuries regarding their mechanism

	Overall	Injury type		
Location and type	N (%)	Acute N (%)	Overuse N (%)	*Not specified* N (%)
Head and face	*24 (2.0)*	*24 (100.0)*		
Trunk (including neck)	*110 (9.0)*	*38 (34.5)*	*59 (53.6)*	*13 (11.8)*
Back/Column	97	26	59	12
Sternum/ribs	6	6		
Abdomen	2	2		
Pelvis/sacrum	5	4		1
Upper extremity	*403 (32.8)*	*248 (61.5)*	*146 (36.2)*	*9 (2.2)*
Shoulder/clavicle	125	42	80	3
Upper arm	5	3	2	
Elbow	65	11	50	4
Forearm	4	3	1	
Wrist	38	27	9	2
Hand	13	12	1	
Finger	107	106	1	
Thumb	46	44	2	
Lower extremity	*686 (55.9)*	*566 (82.5)*	*110 (16.0)*	*10 (0.1)*
Hip/groin	10	8	1	1
Thigh	43	40	3	
Knee	250	171	71	8
Lower Leg	26	15	11	
Ankle/Achilles' tendon	345	325	19	1
Foot/toe	12	7	5	
Missing / not specified	*4 (0.3)*	*3*		*1*
Sum (%)	*1227 (100.0)*	*879 (71.6)*	*315 (25.7)*	*33 (2.7)*

Table 3.26 resumes the distribution of injury types in our studies. About half of the injuries were joint sprains (n= 698; 49.6%), followed by tendon injuries (n= 200; 16.3%), fractures (n= 74; 6.2%) and muscles injuries (n=64; 5.2%).

Table 3.26 Overview of the number of injuries regarding their type

	TOTAL	SEASONS	
Type of injury	N (%)	2003-04 N (%)	2004-05 N (%)
Fractures and bone stress	*74 (6.2)*	*7 (6.8)*	*10 (5.5)*
Fracture	71	7	10
Stress fractures	3		
Joint (nonbone) and ligament	*703 (57.3)*	*66 (64.1)*	*103 (56.3)*
Dislocation/subluxation	40	3	5
Sprain/ligament injury/meniscus lesion	608	62	90
Lesion of cartilage	25		3
Other/Not specified	30	1	5
Muscle and tendon	*280 (22.8)*	*20 (19.4)*	*38 (20.8)*
Muscle rupture/tear/strain/contusion/cramps	64	6	20
Tendon injury/rupture/tendinosis/bursitis	200	13	18
Other/Not specified	16	1	
Contusions	*50 (4.1)*	*3 (2.9)*	*12 (6.6)*
Hematoma/contusion/bruise	48	3	10
Laceration	2		2
Central/peripheral nervous system	*11 (0.9)*		*1 (0.5)*
Concussion	2		
Nerve injury	9		1
Other injuries (including column)	*90 (7.3)*	*6 (5.8)*	*13 (7.1)*
Missing / not specified	*15 (1.2)*	*1 (1.0)*	*6 (3.3)*
Sum (%)	*1227 (100.0)*	*103 (100.0)*	*183 (100.0)*

Table 3.27 provides the number and proportion of injuries distributed by the main body regions regarding the main types of injuries. As already stated, the upper and the lower extremities were the most affected regions. In both, the most frequent types of injuries were joint/ligamentar and muscle-tendinous. The lower extremity presented a higher proportion of joint/ligamentar injuries in comparison to the upper extremity (74.6% vs 49.9%). In the case of the muscle-tendinous, the upper extremity computed a superior proportion of these types of injuries than the lower extremity (37.2% vs 17.1%).

Table 3.27 Diagnosis of injuries

	Overall	SEASONS	
Location and type	N (%)	2003-04	2004-05
Head and face	*24 (2.0)*	*1 (1.0)*	*8 (4.4)*
Fracture/bone stress	9	1	4
Joint (non-bone) and ligaments	1		
Contusion	10		2
Central/peripheral nervous system	2		
Laceration	2		2
Trunk (including neck)	*110 (9.0)*	*4 (3.9)*	*15 (8.2)*
Fracture	2		0
Contusion	11		1
Muscle and tendon	10		1
Central/peripheral nervous system	8		1
Others (including Column)	76	4	13
Not specified	3		
Upper extremity	*403 (32.8)*	*32 (31.1)*	*58 (31.7)*
Fracture	44	5	5
Joint (non bone) and ligaments	189	12	32
Muscle and tendon	150	12	16
Contusion	13	2	4
Central/peripheral nervous system	1		
Not specified	6	1	1
Lower extremity	*686 (55.9)*	*65 (63.1)*	*102 (55.7)*
Fracture	21	2	1
Joint (non bone) and ligaments	512	53	71
Muscle and tendon	117	7	22
Contusion	16	1	3
Others	14	2	1
Not specified	6		4
Missing / not specified	*4 (0.3)*	*1 (1.0)*	*0 (0.0)*
Sum (%)	*1227 (100.0)*	*103 (100.0)*	*183 (100.0)*

Table 3.28 displays a more detailed overview of the types of injuries according to location. The majority of the injuries (n= 672; 54.8%) were classified (Massada, 2001) as being articular, which included joint and ligament damage. The most affected areas were the ankle (n= 313; 46.6%), the knee (n=170; 25.2%), the finger (n= 86; 12.8%) and the shoulder (n=35; 5.2%). The tendon injuries also accounted for an important number of injuries (16.2%); and were more frequent in the shoulder (38.7%), followed by the elbow (24.1%), the knee (14.1%) and the ankle (including the Achilles' tendon) (10.1%). The category named "other" presented an expressive amount of injuries (9.3%; n=113) because it also included the spinal column/back pain. Analysing only the injuries associated to back pain, chronic back pain was observed in 62.8% of the cases (n= 59)

and acute back pain was reported in 23.4% of the cases (n=22). Back pain affected mostly the lumbar region (n=67; 71.3%), followed by the cervical spine (n=9; 9.6%), thoracic and lumbar regions (n=5; 5.3%) and coccygeal and sacral regions (n=3; 3.2%). The sciatic nerve pain was documented in 7.4% of the cases (n=7) and cervicobrachial in one of the cases (1.1%).

The fractures represented 6.2% of the injuries and were mostly consequence of an acute mechanism. The fractures on the wrist, hand, fingers and the thumb accounted for 38% of the occurrences, revealing a higher number on phalange V (little finger – 11.8%) and the thumb (5.3%). The nasal bone represents 11.8% of fractures and the fibula 9.2% (with medial and distal fractures). Other bones were fractured in smaller proportions, including the metatarsals (5.2%), scaphoid (4%), tibia (4%), clavicle (2.6%), radius (2.6), ulna (1.3%), pisiform (1.3%) and patella (1.3%).

3.28 Cross-tabulation of injury locations with the injury type

	Type of injury (count/percentage)								
Injured body part	Contusion	Articular	Tendinosis	Muscular	Fracture	Cartilage	Other	Not specified	Sum (%)
Head/face	*12 (50.0)*	*1 (4.2)*			*9 (37.5)*		*2 (8.3)*		*24 (100.0)*
Trunk (including neck)	*6 (5.5)*			*8 (7.3)*	*2 (1.8)*		*94 (85.5)*		*110 (100.0)*
Back/Column	2			6			89		97
Sternum/ribs	4				2				6
Abdomen				2					2
Pelvis/sacrum							5		5
Upper extremity	*12 (3.0)*	*185 (45.9)*	*141 (35.0)*	*8 (2.0)*	*44 (10.9)*		*3 (0.7)*	*10 (2.5)*	*403 (100.0)*
Shoulder/clavicle	3	35	77	5	2			3	125
Upper arm			2	2				1	5
Elbow	1	9	48		1		2	4	65
Forearm				1	3				4
Wrist	3	14	12	0	8			1	38
Hand	3	1			8			1	13
Finger	2	86	1		18				107
Thumb		40	2		4				46
Lower extremity	*14 (2.0)*	*485 (70.7)*	*58 (8.5)*	*56 (8.2)*	*21 (3.1)*	*25 (3.6)*	*14 (2.0)*	*10 (1.5)*	*686 (100.0)*
Hip/groin	1	1	3	3	1			1	10
Thigh	1		2	40					43
Knee	11	170	28		1	25	9	6	250
Lower Leg			3	13	4		5	1	26
Ankle/Achilles' tendon		313	20		10			2	345
Foot/toe	1	1	2		5			3	12
Missing / not specified		*1*		*3*					*3*
Sum (%)	*44 (3.6)*	*672 (54.8)*	*199 (16.2)*	*75 (6.1)*	*76 (6.2)*	*25 (2.0)*	*113 (9.3)*	*23 (1.8)*	*1227 (100.0)*

Table 3.29 provides the distribution of injuries according to their severity and injury localization. About 24.7% of the injuries (excluding the unspecified cases) demanded an absence from practice of more than 4 weeks (82% of these were acute). Of the severe injuries, 50% were in the knee and 25% were in the ankle. A total of more than 28% of all knee injuries demanded a surgical intervention. In the ankle the surgical intervention rate was only 6%.

3.29 Cross-tabulation of injury locations with injury severity

	Injury severity (absence time) Number of cases (%)						
Injured body part	Slight 0 days	Minimal 1 to 3 days	Mild 4 to 7 days	Moderate 8 to 28 days	Severe > 28 days	Not specified	Sum (%)
Head/face	*5 (20.8)*		*6 (25.0)*	*5 (20.8)*	*2 (8.3)*	*6 (25.0)*	*24 (99.9)*
Trunk (including neck)	*18 (16.4)*	*8 (7.3)*	*18 (16.4)*	*11 (10.0)*	*8 (4.5)*	*50 (45.5)*	*110 (100.0)*
Back/Column	16	8	12	6	8	48	97
Sternum/ribs	1		1	2		2	6
Abdomen			1	1			2
Pelvis/sacrum	1		2	2			5
Upper extremity	*117 (29.0)*	*12 (3.0)*	*70 (17.4)*	*62 (15.4)*	*32 (7.9)*	*110 (27.3)*	*403 (100.0)*
Shoulder/clavicle	36	4	22	19	12	32	125
Upper arm	1		3		1		5
Elbow	27		9	5	4	20	65
Forearm	1				3		4
Wrist	8	1	9	11	1	8	38
Hand	2		3	3	2	3	13
Finger	30	7	13	19	6	32	107
Thumb	12		11	5	3	15	46
Lower extremity	*89 (13.0)*	*31 (4.5)*	*124 (18.1)*	*118 (17.2)*	*186 (27.1)*	*138 (20.1)*	*686 (100.0)*
Hip/groin	1	1	6	1	1		10
Thigh	7	2	14	7	5	8	43
Knee	33	4	24	28	114	47	250
Lower Leg	8		4	3	7	4	26
Ankle/Achilles' tendon	37	24	75	75	58	76	345
Foot/toe	3		1	4	1	3	12
Not applicable					*3*	*1*	*3*
Sum (%)	*229 (18.7)*	*51 (4.2)*	*218 (17.8)*	*196 (16.0)*	*228 (18.6)*	*305 (24.9)*	*1227 (100.0)*

Figure 3.18 represents the average absence time per acute knee and ankle injury for each age category (in days).

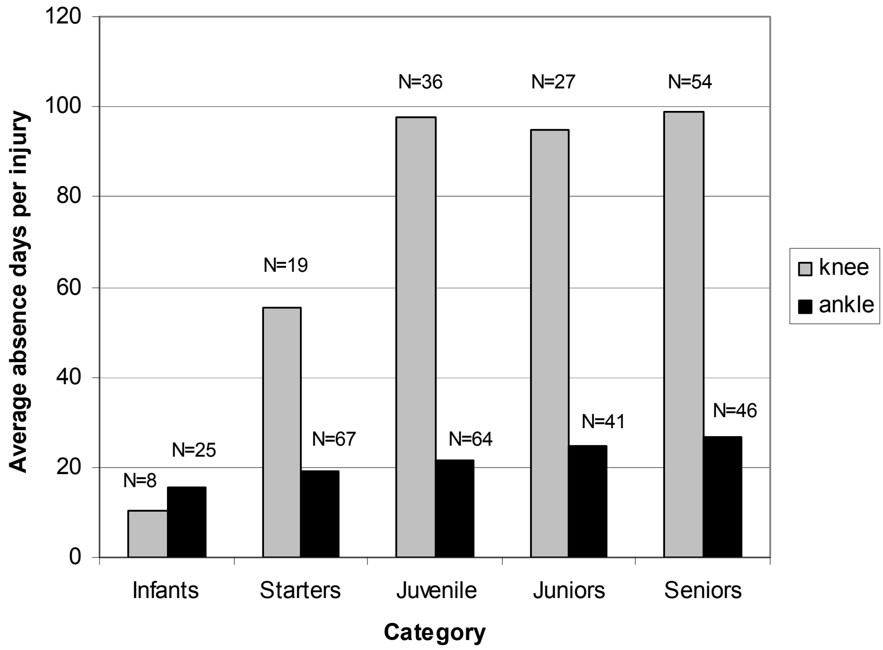


Figure 3.18 Average absence time per acute knee or ankle injury for each age category (in days)

Of the severe injuries, the majority (57%) were sprains with ligament ruptures and/or meniscal tears. The main structures affected in these severe injuries were the anterior cruciate ligament (28%), the meniscus (22%) and the collateral ligaments (11%).

A total of 105 surgeries were reported. From those 71 were in the knee (67.7%), 21 in the ankle (20%), 5 in the face (4.8%), 3 in the finger (2.9%), 2 in the elbow (1.9%), 1 in the shoulder (0.9%) and 1 in the sternum/ribs (0.9%).

From all injuries (valid cases), 19.6% were reported as recurrent (n=196) and from those 67.4% were sprains and about 18.9% were tendinosis. In the 2003-04 season there were 22.6% of reinjuries (n=16) and in the 2004-05 season there was 26.7% (n=31).

3.5.4 Injury context and external mechanisms

A total of 448 injury forms were completed regarding the injuries occurred between the 2003-04 and 2006-07 seasons.

The majority of injuries occurred in the club context (92.6% vs 5.1% in a team selection). Analysing only the acute injuries (n=369), 50.3% of the cases occurred in a training session (28.6% in a training match) and 47.4% in a match (about half in a regional competition, 39% in a national competition and 8% in an international competition).

The distribution of injuries during matches is presented in table 3.30. The proportion of injuries regarding the first and second halves was 31.7% and 40.4%, respectively (some were in the warm-up or were not specified). In the first half the proportion of injuries increased towards the middle (minutes 11-20) and in the second half it peaks at the beginning and decreases steadily towards the end. The same table also presents the players self-perception of match importance when injuries occurred. For this purpose a five-level Likert scale was used. There was a tendency to evaluate the matches where injuries occurred as important (44.2% of the valid cases) and highly important or decisive (25.8% of the valid cases).

3.30 Cross-tabulation of time of injury with player's self-perception of match importance

Minute of match	Perception of match importance						Sum (%)
	Decisive	High important	Important	Low important	Not important	*Not reported*	
Warm up			4	2	1	5	12 (5.2)
First half							
1-10	0	3	4	3	1	1	12 (5.2)
11-20	6	7	13	6	2	3	37 (16.1)
21-30	2	4	11	6	0	1	24 (10.4)
Second half							
31-40	7	6	18	7	2	1	41 (17.8)
41-50	1	7	19	5	1	1	34 (14.8)
51-60	3	0	10	4	0	1	18 (7.8)
Not reported	1	2	5	11	6	27	52 (22.6)
Sum (%)	20 (8.7)	29 (12.6)	84 (36.5)	44 (19.1)	13 (5.7)	40 (17.4)	230 (100.0)

More than half of the injuries reported in a match occurred with opponents' contact (66.1%, n=152) or contact with an object like the goal or the ball (7.4%). Whenever there was some kind of physical contact, in about 41% of the cases aggressive behaviour from the opposition was reported, but in 42.9% of the situations the referees did not judge the behaviour as foul play. In a small portion (20.6%) of the injury cases with opponents' foul play, the fouls were severe and were accompanied by additional sanctions (*e.g.* 2' suspension*, etc.*).

The injuries occurred most often in the offensive phase (71.6%, n=141) (table 3.31): 48.2% in the positional attack and 23.4% in the counter or fast attack. In the defensive phase the injuries occurred predominantly in the positional defense (18.8%) and few (7.6%) during the transition from attack to defence.

Table 3.31 Cross-tabulation of injury occurrence *vs* match type and phase of the game

	Match type					
	Training N (%)	Regional N (%)	National N (%)	International N (%)	Not specified N (%)	Sum (%)
Offensive phase	29 (14.7)	58 (29.4)	45 (22.8)	8 (4.1)	1 (0.5)	141 (71.6)
Positional attack	20	39	30	5	1	95
Speed attack	9	19	15	3		46
Defensive phase	10 (5.1)	23 (11.7)	17 (8.6)	4 (2.0)	2 (1.0)	56 (28.4)
Positional defence	7	15	11	4		37
Defense recuperation	3	6	6			15
Goalkeeper defence		1				1
Others	2	1				
Not specified/missing cases	11	10	7	2		30
Sum (%)	52 (22.9)	92 (40.5)	69 (30.4)	14 (6.2)		227

Table 3.32 lists the distribution of traumatic injuries by playing position and phase of the game. The proportion of injuries is significantly higher in back playing positions (70.3% of the valid cases) and slightly increased in the right back position. Regarding its proximity to the goal, about 70% occurred around the 9 metre line, followed by the 6 metre line (27.7%) and, lastly, somewhere in between like around 7 metres from the goal (12.8%).

In almost half of the traumatic injuries the players were carrying the ball, although the ball interception or its dispute was reported in 8.9% of the cases (n=18). During the positional attack, 64% of the injuries occurred in ball possession and 32.5% without ball, but in the counter or fast attack the proportion of injuries when handling the ball increased to 69.2%. In the defence, 10% of the injuries occurred while trying to intercept the ball (from a pass or shot).

Table 3.32 Cross-tabulation of injury occurrence *vs* game phase and playing position

	Offensive phase		Defensive phase		*Not specified*	Sum
Playing position	Positional N (%)	Speed attack N (%)	Positional N (%)	Recuperation N (%)	*N*	(%)
Left wing	1	1	1		1	4 (2.1)
Left back	24	8	5	5	4	46 (24.5)
Middle back	17	6	5	1	5	34 (18.1)
Right back	27	11	9	2	3	52 (27.7)
Right wing	4	5	2	3	1	15 (8.0)
Pivot	4	1				5 (2.7)
Goalkeeper	1	2	8	1	5	17 (9.0)
Advanced player				1	1	2 (1.1)
Middle field		1		1	4	6 (3.6)
Irrelevant		2				2 (1.1)
Not specified	4	4	3	1		
Sum (%)	82 (44.8)	41 (22.4)	33 (18.0)	15 (8.2)	12 (6.6)	183 (100.0)

3.5.5 Knee and ACL injuries

A total of 56 knee injuries were assessed in the 2003-04 and 2004-05 (27 and 35, respectively), from which 17 were ACL injuries (10 and 7, respectively). The difference between knee injury incidence in these two seasons was significant (according to the Mann-Whitney *U* test; $p < 0.8\%$). Although, discriminating the playing categories, the differences in knee injury incidence were significant only for seniors, as presented in table 3.33.

A total of 162 previous knee injuries were reported in the 2006-07 study, giving an average injury rate of 0.3 knee injuries per player. From the reported knee injuries 23 were ACL injuries.

Table 3.33 Incidence and number of knee injuries by season and playing category

	Knee Injury incidence (numbers of injuries/1000h practice)			Number of previous knee injuries per player
	2003-04 Season µ± SD	2004-05 Season µ± SD	Mann-Whitney U (p-value)	Until 2006-07 Season µ ± SD
Seniors (Adults)	1.5 ± 2.5	0.5 ± 1.4	0.005*	0.6 ± 0.8
Juniors (age 16 to 18)	0.8 ± 1.8	0.3 ± 0.8	0.30	0.5 ± 0.8
Juvenile (age 14 to 16)	0.5 ± 1.8	0.7 ± 1.9	0.50	0.3 ± 0.6
Starters (age 12 to 14)	0.7 ± 1.9	0.3 ± 1.2	0.50	0.3 ± 0.6
Infants (age 10 to 12)		0.5 ± 2.3		0.1 ± 0.3
Total	1.0 ± 2.2	0.5±1.6	0.008*	0.3 ± 0.7

*Significantly different ($p \leq .05$).

The ACL injury incidence difference between the seasons 2003-04 and 2004-05 was significant (according to Mann-Whitney *U* test; $p<0.7\%$). Moreover, comparing the playing categories between the two seasons, for senior and starters the differences for ACL injury incidence revealed to be significant, as presented in the table 3.34. In the group of players from 2006-07 study, the differences in the reported numbers of knee injuries and ACL injuries were significant when the different playing categories were considered ($p \leq 0.05$).

Table 3.34. Incidence and number of ACL injuries by season and playing category

	ACL Injury incidence (numbers of injuries/1000h practice)			Number of ACL injuries per player
	2003-04 Season μ ± SD	2004-05 Season μ ± SD	Mann-Whitney U (p-value)	Until 2006-07 Season μ ± SD
Seniors (Adults)	0.5 ± 1.6	0.09± 0.6	0.02*	0.2 ± 0.5
Juniors (age 16 to 18)	0.5 ± 1.4	0	0.07	0.1 ± 0.3
Juvenile (age 14 to 16)	0	0.2 ± 1	0.2	0.01 ± 0.1
Starters (age 12 to 14)	0.7 ± 1.9	0	0.009*	0
Infants (age 10 to 12)	0	0	0	0
Total	0.4 ± 1.3	0.1 ± 0.7	0.007*	0.1 ± 0.3

*Significant difference ($p \leq .05$).

3.5.5.1 Injury measures in senior players

Because the seniors are the most representative category of a competitive sport, an analysis of this category by the two top divisions of playing was made. As shown in the following table (3.35), in the first season, there are significant different incidences on the three injury categories between the two leagues (p<0.03), which was not the case in the second season (p≥0.3). Analysing the data from the two divisions collectively the incidences were significantly different between the two seasons on the knee injury incidence (p<0.02) and the ACL injury incidence (p<0.03) while the differences in overall injury incidence were insignificant (p>0.7). When grouping the two seasons we estimated an ACL injury rate of 0.1 and 0.5 injuries per 1000 player-hours of practice for the 1st and 2nd divisions, respectively. It is a five-fold ratio with strong statistical significance (p<0.017).

Table 3.35 Incidence and number of injury by season and playing category

Season		N (numbers of players)	Injury incidence μ± SD	Mann-Whitney *U* (p-value)	Knee injury incidence μ± SD	Mann-Whitney *U* (p-value)	ACL incidence μ± SD	Mann-Whitney *U* (p-value)
2003-04	*1st division*	*39*	*2.3 ± 2.3*	*0.019**	*0.7 ± 1.6*	*0.023**	*0.2 ± 1.0*	*0.002**
	2nd division	26	*5.5 ± 4.7*		*2.2 ± 3.1*		*0.9 ± 1.9*	
	Total	65	3.6 ± 3.8		1.3 ± 2.4		0.5±1.5	
2004-05	*1st division*	*71*	*3.8 ± 3.7*	*0.3*	*0.6 ± 1.4*	*0.6*	*0.04 ± 0.3*	*0.3*
	2nd division	*36*	*2.5 ± 3.3*		*0.3 ± 1.2*		*0.2 ± 0.9*	
	Total	107	3.4 ± 3.7		0.5 ± 1.4		0.07±0.6	

*Significant difference ($p \leq 0.05$).

3.5.6 Knee injury type and severity

Focusing on all knee injuries accounted (n=250), around 68% were related to direct trauma and 28% were from chronic microtrauma (3% were not specified). The most frequent acute injuries reported were joint sprains (n=146), grouped as followed (Massada, 2001): without damaging any specific structure (n=33); with ligament injury (n=71); with meniscal injury (n=28); and with ligament and meniscal injury (n=14). Moreover, the knee sprains affecting soft tissues had the highest impact in time of absence from full handball practice (table 3.36).

The majority of overuse injuries affecting the knee were associated to patellofemoral disorders (table 3.36). In 77.4% of all reported patellofemoral joint pain cases (n=31), the cartilage (chondral compartment) was also compromised.

The recurrent knee injuries were reported as being joint sprains (n=14), patellar subluxation (n=1) and patellar tendinopathy (n=1). The reported recurrent knee joint sprains affected mostly the ACL (n=6), followed by the other ligaments and meniscus (n=6).

3.36 Cross-tabulation of type of knee injury with the severity

	Injury severity (absence time) Number of cases (%)							
Type of knee injury	Slight 0 days	Minimal 1 to 3 days	Mild 4 to 7 days	Moderate 8 to 28 days	Severe > 28 days	Ending Career	*Not specified*	**Sum**
Sprain	9	4	6	9	2		3	33
Sprain with ligament injury	2		4	7	51	1	6	71
Sprain with meniscal injury			1	4	19		4	28
Sprain with meniscal and ligament injury			1		12		1	14
Lesion of meniscus and patellar tendinopathy					1			1
Subluxation of patella			1				1	2
Dislocated patella					5			5
Fractured patella					1			1
Knee haematoma (extra-articular)				1				1
Contusion (incl. patella contusion)	2				1			3
Knee joint swelling			1		4		1	6
Popliteus tendonitis/strain	1			1			1	3
Chondral injury not specified					1			1
Patellofemoral joint pain	7		3		7		15	32
Patellar tendinopathy	4		2	5	6		5	22
Tibial tuberosity pathology	3						2	5
Iliotibial band syndrome	2							2
Prepatellar bursitis			1		1			2
Knee pain undiagnosed					1			1
Other							2	2
Not specified	3		4	1	1		6	14
Sum (%)	33 (13.2)	4 (1.6)	25 (9.6)	28 (11.2)	113 (45.2)	1 (0.4)	47 (18.8)	250 (100.0)

The knee injuries were (re)categorized according to the categories used by Dallalana *et al.* (2007), although some adaptations were introduced because of a lack of information regarding the affected structures of the joint (table 3.37). The categories used were: ACL, MCL, PCL/posterior corner, chondral/meniscal, patellofemoral/extensor mechanism, or other injuries (including minor injuries and cases with lack of information).

Table 3.37 Frequency of knee injury type

Injury category	Type of knee injury	TOTAL	SEASONS	
		N (%)	2003-04	2004-05
ACL injury	ACL rupture	35	7	2
	ACL rupture plus meniscal injury	6		
	ACL rupture plus MCL tear and meniscal injury	3		1
	ACL rupture plus LCL tear	1		
	Total	45 (19.1)	7	3
MCL injury (including collateral ligaments not specified)	MCL injury	11		2
	MCL injury plus meniscal injury	3		1
	Collateral ligaments injury (not specified)	3	2	
	Total	18 (7.6)	2	3
PCL/ posterolateral corner	PCL partial rupture	1		
	Popliteus tendonitis/strain	3		
	Lateral collateral ligament strain	3	1	
	Lateral collateral ligament strain and meniscal injury	1		
	Total	8 (3.4)	1	
Meniscal injury/ chondral	Medial meniscal tear	5		1
	Lateral meniscal tear	6		
	Meniscal tear, unspecified	17	1	5
	Chondral injury, unspecified	1		
	Total	29 (12.3)	1	6
Patellofemoral/ extensor mechanism	Patellofemoral joint pain	32	2	3
	Patellar tendinopathy	22	1	2
	Dislocated and subluxation of patella	7	2	1
	Fractured patella	1		
	Tibial tuberosity pathology	5		
	Total	67 (28.4)	5	6
Other injury	Knee sprain	31		3
	Ligament/meniscal injury not specified	17	3	3
	Knee hematoma (extra-articular)	3	1	
	Iliotibial band syndrome	2		
	Knee joint swelling	6	1	2
	Prepatellar bursitis	2		
	Knee pain undiagnosed	1		1
	Baker's cyst	1		
	Other (including ACL injuries not confirmed)	7	2	6
	Total	70 (29.7)	7	15
Sum		236 (100.5)		
Not specified		*14*		
Sum	Total	250	23	33

Table (3.38) displays a distribution of the practice time lost according to the surgery demanding type of knee injury. The excluded cases were career-ending cases consequence of an ACL injury (n=1), still in recuperation of an ACL rupture when questioned (n=1), ending/interruption of career because postponed surgery (that is, when reached 18 years of age; n=1), and missing information cases (n=5).

Regarding which knee suffered these surgery demanding injuries, the left side accounted for 59.7% of the interventions, the right side for 35.8% and 4.5% (n=3) where bilateral interventions.

3.38 Cross-tabulation the time lost according to the injury type submitted to surgery

	Absence time (days)						
Injury	Number of injuries	Excluded cases	μ	Minimal	Maximal	Median	Sum (days)
ACL rupture	29	3	173.4	28	336	172	3976
ACL rupture plus MCL tear	1		280.0			280	280
ACL rupture plus meniscal injury	5	1	189.0	196	336	182	756
ACL rupture plus MCL tear and meniscal injury	3		252.0	168	252	252	252
ACL rupture plus LCL	1		280.0			280	280
Medial meniscal tear	3		112.0	56	168	112	168
Lateral meniscal tear	5	1	49.0	28	84	35	84
Meniscal tear, unspecified	8	1	84.0	56	168	67	448
Ligament and/or meniscal injury, unspecified	3		102.7	56	168	84	252
Meniscal injury plus MCL	1		84.0			84	84
Patellar tendinopathy	3		46.7	84	168	168	168
Medial Collateral ligament	3	1	70.0	56	84	56	140
Patellofemoral joint pain	4	1	130.7	112	168	119	392
Dislocated patella	3		196.0	84	336	168	84
Sum	72	8	146.4	28	336	159	7364

3.5.7 Acute knee injury context and external mechanisms

Analysing the 115 reports of acute knee injuries, 58.3% of those occurred in a match competition, and 40% occurred during a training session, including 15.7% injuries reported in training matches.

Table 3.39 shows the computed number on injuries occurred by time of match and player’s self-perception of match importance.

The acute knee injuries occurred predominantly in the attack phase (n=39, 72.2%), with 25 during a positional attack and 14 during a fast/counter attack. When defending, there were 10 knee injuries during positional defence, 5 in the attack to defence transition and 11 suffered by the goalkeepers.

The majority of the affected players were playing in the back position when injured in the knee, that is, if we exclude the cases where there wasn't a defined playing position (in the big space), we conclude that 97.1% of the injuries occurred in the back position (67 of 69 valid records). Within this playing position, there were different percentages relative to the left (18.8%), middle (25.9%) and right back (33.3%) positions.

3.39 Acute knee injury *vs* time of match and self-perception of match importance

Minute of match	Perception of match importance						Sum (%)
	Decisive	High important	Important	Low important	Not important	*Not reported*	
Warm-up			1			1	2 (4.3)
First half							
1-10			1	2			3 (6.5)
11-20	2	2	4		1	3	12 (26.1)
21-30	1	1	2	2			6 (13.0)
Second half							
31-40	1	2	3	2			8 (17.4)
41-50		1	2	2			5 (10.9)
51-60			2			1	3 (6.5)
Not reported	1	2	1	2	1		7 (15.2)
Sum (%)	5 (10.9)	8 (17.4)	16 (34.8)	10 (21.7)	2 (4.3)	5 (10.9)	46

The proportion of acute knee injuries sustained with contact or without contact with an opponent was similar (49.1% and 50.9%, respectively, N=108 valid cases). From the contact situations 6.4% were with an object (the ball), and 15.8% of the players reported aggressive behaviour from the opponent, being sanctioned (in a match context) by the referees in 10.2% of the cases. Table 3.40 displays a more detailed overview of the number of injuries sustained when combining two types of criteria to understand the context and the mechanism of the knee injury in handball (playing position, with or without contact). When attacking, there was a relation of about 4:1 injuries with the ball versus without the ball.

Table 3.40 Cross-tabulation of injuries regarding the playing position and the occurrence of contact

Playing position	Without contact (N)	With contact (N)				
		Ball	Contact with another player	Aggressive behaviour of the opponent	Fault playing sanctioned	Sum (%)
Left wing	1					1 (1.1)
Left back	7		4	2	4	17 (19.1)
Middle back	5		4	1	1	11 (12.4)
Right back	9		7	2	5	23 (25.8)
Right wing						0
Pivot	1					1 (1.1)
Goalkeeper	5	5				10 (11.2)
Advanced player			1			1 (1.1)
Middle field	1		1			2 (2.2)
Irrelevant	3	1				4 (4.5)
Not specified	12	1	6			19 (21.3)
Sum (%)	44 (49.4)	7 (7.9)	23 (25.8)	5 (5.6)	10 (11.2)	89 (100.0)

Forty-six percent (46%) of acute knee injuries occurred when planting and cutting, and 22.4% while landing from a jump (table 3.41).

Table 3.41 Injury occurrence regarding playing position and player action

Movement/situation	Playing position								
	Left wing	Left back	Middle back	Right back	Right wing	Pivot	Goal- -keeper	Other/not specified	Sum (%)
Plant and cut	1	9	4	8		1	2	14	39 (44.8)
Jump take off phase		1		2				1	4 (4.6)
Landing from a jump		5	4	2			3	5	19 (21.8)
Fall		3		1			2	1	7 (8.0)
Collision			1					2	3 (3.5)
Defensive movement		2		3			3		8 (9.2)
Others		1	2	1				1	5 (5.7)
Not specified				1			1		2 (2.3)
Sum (%)	1 (1.1)	21 (24.1)	11 (12.6)	18 (20.7)	0	1 (1.1)	11 (12.6)	24 (27.6)	87 (100.0)

Figure 3.19 shows the age distribution of knee strain injures. About half of them occurred before the age of seventeen and more than 61% occurred before the senior level (age of 19).

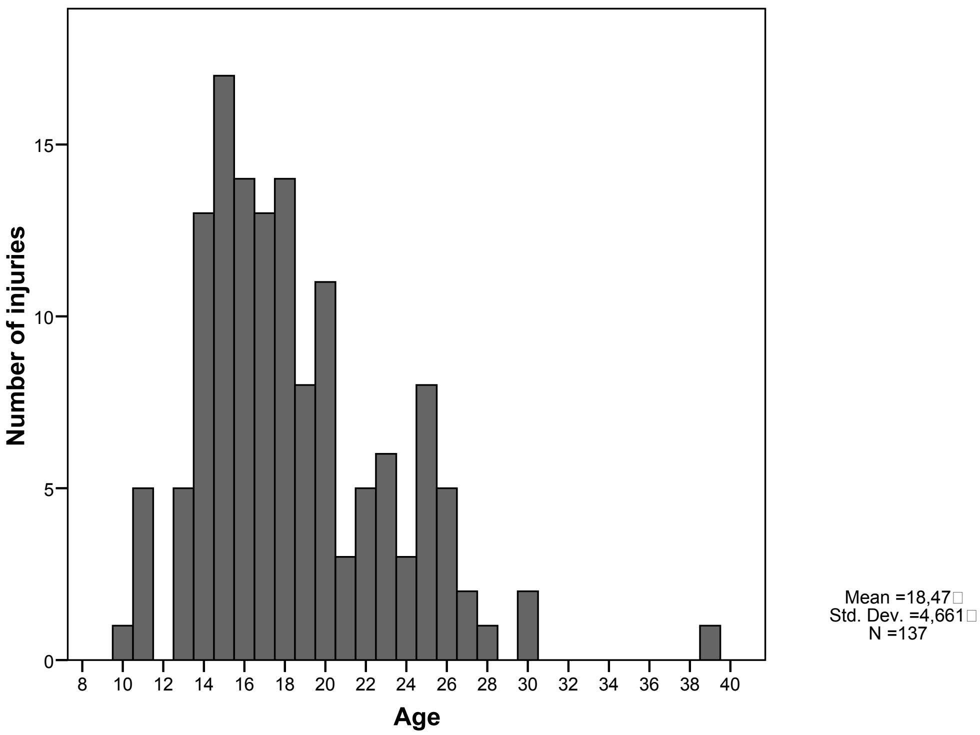
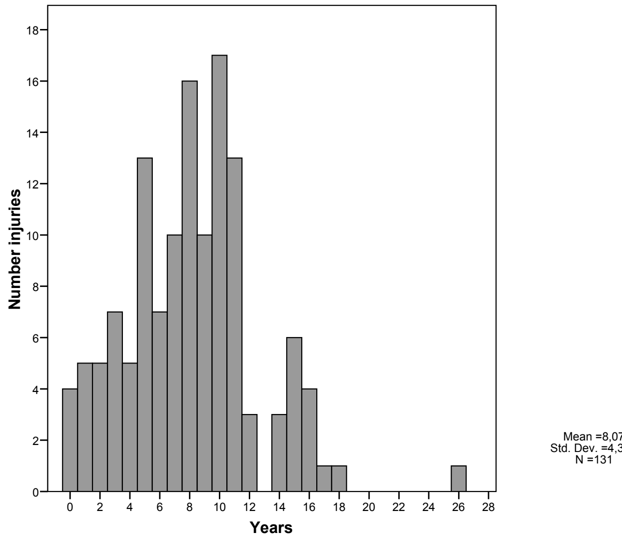


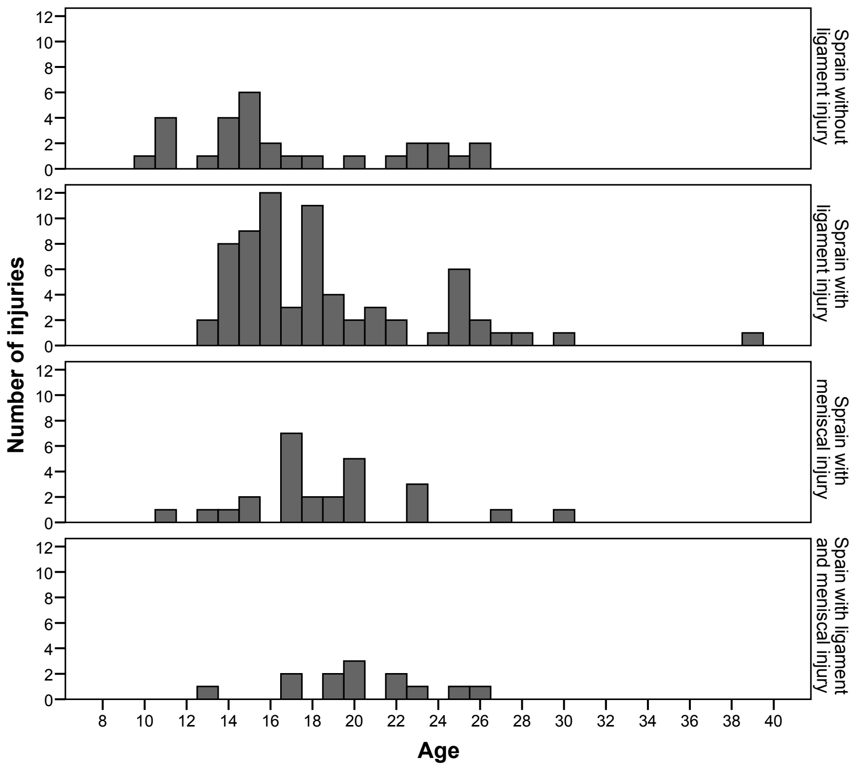
Figure 3.19 Age at knee strain

In figure 3.20 the cumulative years of handball practice until the reported knee strain are displayed. About half of the knee strains occurred before completing 8 years of exposure.



3.20 Years of handball practice before the documented knee strain

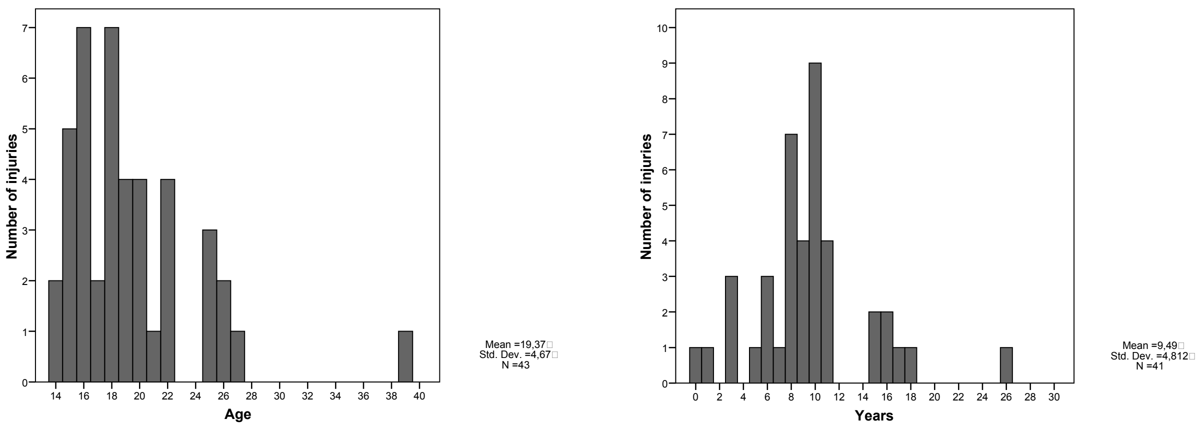
Figure 3.21 presents the age distribution of injuries distributed by the general type of knee strain.



3.21 Age of knee strain with or without affecting other joint structures

3.5.8 ACL rupture context and external mechanisms

The age at ACL rupture varied from 14 to 39 years, and more than half occurred before the age of 19 (53.5%) (figure 3.22) and with less than 10 years of handball practice (figure 3.23).



Figures 3.22 & 3.23 Age at ACL injury & years of exposure before ACL rupture

From the accounted ACL ruptures, a recurrence was identified in 5/28 cases, and in two cases the players reported to be recovering from another knee injury (a knee strain or a medial collateral ligament injury) at the time of the reinjury.

About the same number of ACL injuries occurred in training and in match (19 vs 18). Having noticed that half of the ACL injuries sustained in training were in a training match, we analysed the injury mechanism by grouping all the matches, irrespectively of being official or practice ones. During matches, all ACL ruptures were reported to attack situations except in the case of goalkeepers (n=3). Eleven injuries occurred in the positional attack and 8 in counter/fast attack. The majority of the players were playing in the back position when injured (82%), with a uniform distribution between the distinct positions. More than sixty percent of the injuries involved ball possession and about the same rate (61.1%) were without contact. Most of the injuries occurred when landing from a jump (14 cases, being 7 of them after a shot) or at a plant and cut movement (n=14). Six other ACL injuries were distributed by five other categories.

The rate of ACL injuries between artificial and wooden floors was 19/17.

All players had already reached menarche when injured (n=38), and the majority of the players were not OC users at the time of injury (70.3%).

A factorial analysis on the variables, *time absent from practice*, *age*, *years of practice*, *years of OC intake* and *years passed since menarche,* was performed on the set of all injuries and several subsets (knee injuries, acute knee injuries, ACL injuries). Excluding the case of all injuries, the results are very similar to those of the ACL injuries, as in table 3.42 and figure 3.24. The first component explained an overall variance (after varimax rotation) of 55.8% and the second one of 22.9%.

Table 3.42 Explained variance (Rotated Component Matrix)

	Component	
	1	2
Absence from practice	0.077	0.923
Years of practice	0.923	-0.027
Years since menarche	0.921	-0.050
Years of OC usage	0.426	-0.514
Age	0.952	-0.167

Extraction Method: Principal Component Analysis.
Rotation Method: Varimax with Kaiser Normalization.

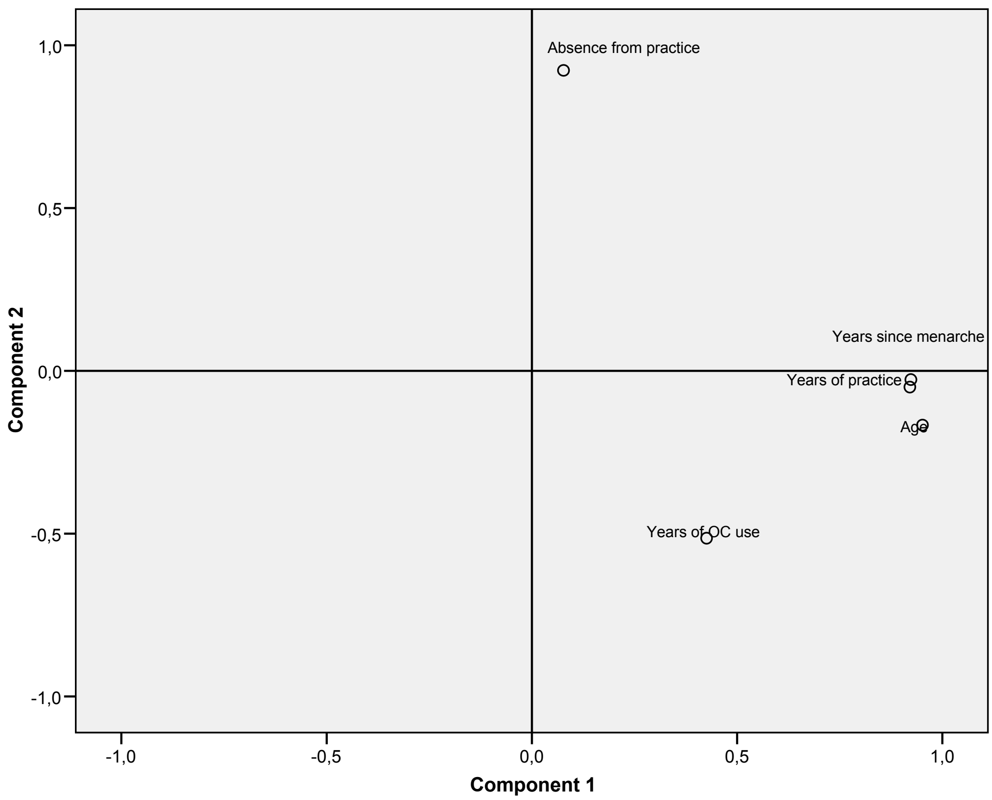


Figure 3.24 Factor analyses with varimax rotation

3.5.9 Menstrual cycle characteristics

At the beginning of the first studied season the players completed the questionnaire about their menstrual cycle characteristics. In the next season, a menstrual cycle calendar was introduced in the questionnaire, because even after explaining the meaning and differences between menstrual cycle length and flow, the answers about bleeding history remained frequently vague or inaccurate. The players were asked to chart their menstrual menses and pain (*i.e.*, dysmenorrhoea) during the season (from September 2004 to July 2005). Whenever the menstrual cycle calendar wasn't delivered at the end of the season, the reported average of menstrual length and flow in the beginning of the study was considered for analyses. In 2006-07 the menstrual cycle calendar was excluded from the questionnaire.

All the players from the 2003-04 season had already reached menarche; but from the other groups/seasons there were players who reported not having reached menarche: 16 players (5.2%) in 2004-05 and 62 players (12.4%) in 2006-07. The next picture (3.25) provides a graphical summary of the data distribution of age at menarche (excluding the cases of players without menarche).

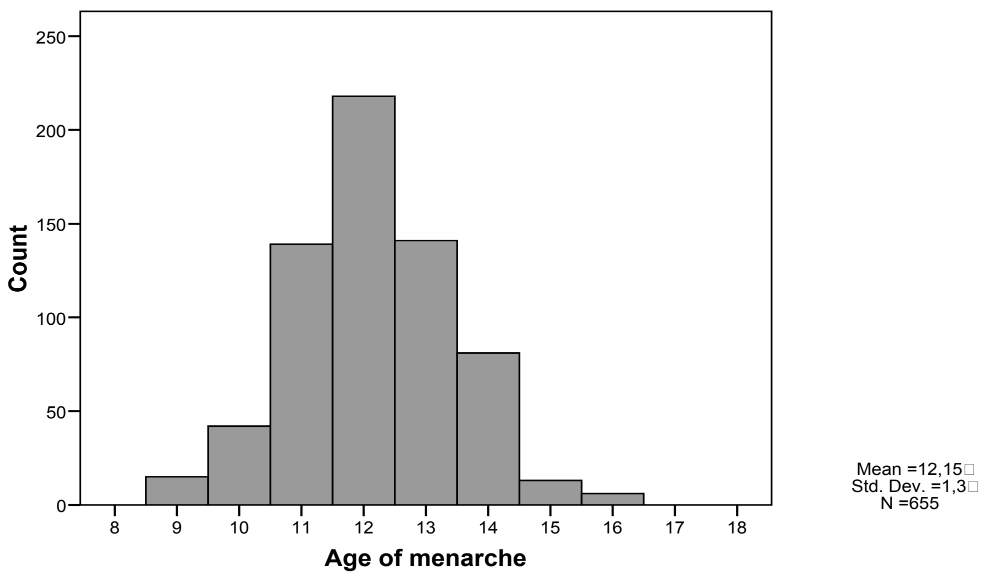


Figure 3.25 Age at menarche

Attempting to investigate the relation between "age of menarche" and "exposure to handball practice", we considered only players which reported the start of handball practice before menarche occurrence (n=351; 51.5%).

Using bivariate (orthogonal) regression methods a significant association between the age of menarche and the years of handball practice was found (p<0.0001), but only 10% (R=0.31) of the *age of menarche* variance is explained in a linear model by the number of *previous years of handball practice* until the menarche event (figure 3.26).

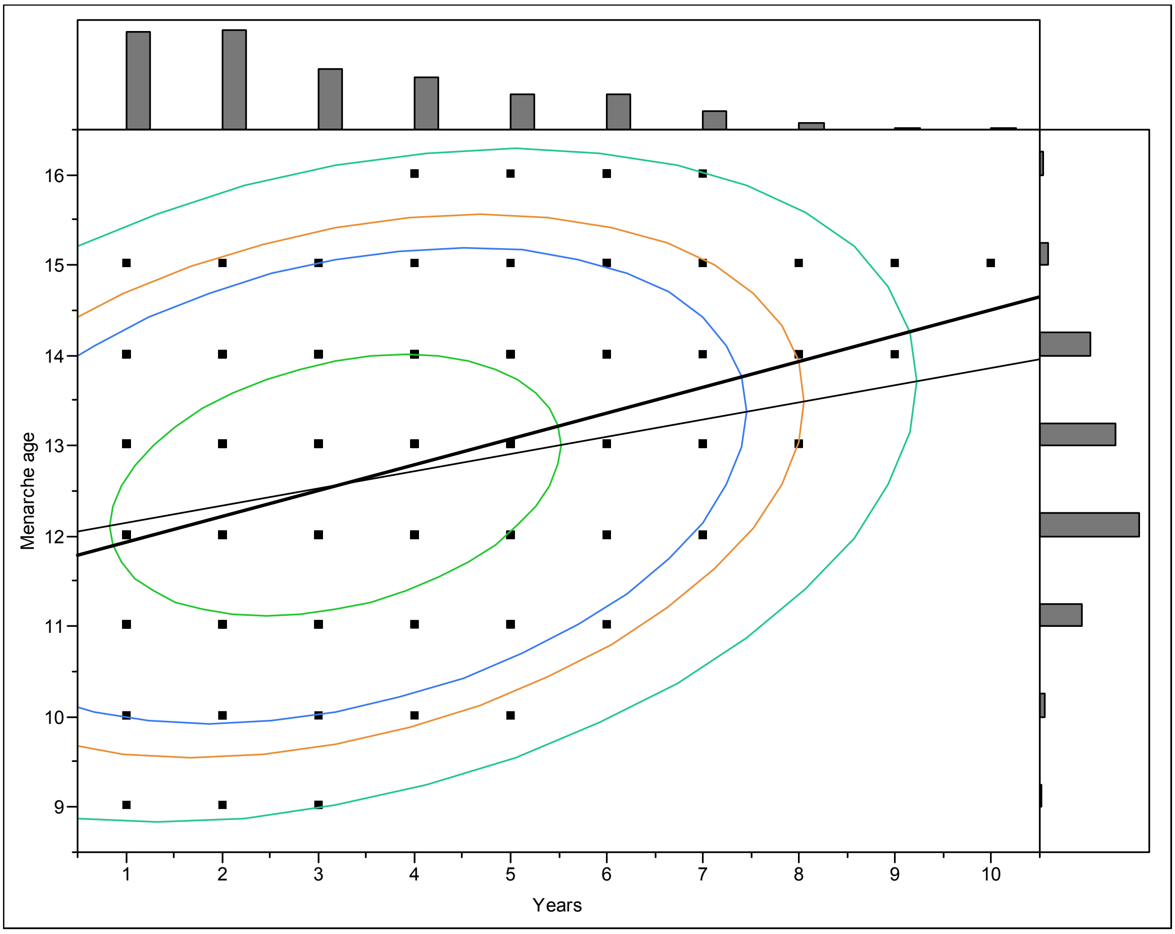


Figure 3.26 Bivariate normal ellipses at 50%, 90%, 95% and 99%. Orthogonal regression line: age of menarche *vs.* previous handball experience (y=11.66+x*(0.28±0.08))

In the studied groups the number of players not using OCs was higher than those using OCs, as it is presented in the table 3.43.

Table 3.43 Oral contraceptive use

Use of oral contraceptives	SEASONS 2003-04 N (%)	2004-05 N (%)	2006-07 N (%)	Sum
Yes	49 (38)	71 (23.1)	98 (19.6)	218
No	74 (57.4)	217 (70.5)	340 (68)	631
Missing / not specified	6 (4.7)	20 (6.5)	62 (12.4)	88
Sum	129	308	500	937

When asked about cycle regularity, more than half of the non-OC users reported to be irregular (51.2%; n= 330). To further understand this question the players were asked to characterise/explain their menstrual cycle irregularity in an open question (without being constrained by a fixed set of possible responses). The answers were analysed on the basis of associations between words on a map, in order to establish categories. The main ideas identified were: irregular; variance in the cycle length (occurrence of shorter and/or longer cycles than expected), occurrence of cycles of 15 days, 60 days or 90 days; and absence of period for more than 90 days. From this analysis we excluded the players who had reported the occurrence of menarche recently (all cases of just one previous menstrual flow).

The groups of OC and non-OC users were analysed separately regarding their menstrual cycle length. Even after explaining the meaning of menstrual cycle length, a significant number of players didn’t answered properly to this question or didn’t know their menstrual cycle length (about 50% of all the non-OC players).

From all groups analysed the average length of the menstrual cycle was about 28 days for OC users and about 30 days for non-OC users. The next figure (3.27) shows the distribution of non-OC users over the menstrual cycle length.

The average duration of the menstrual period for OC users was 4.5 days (±1.0) and for non-users was 5.0 days (±1.3) (figure 3.28).

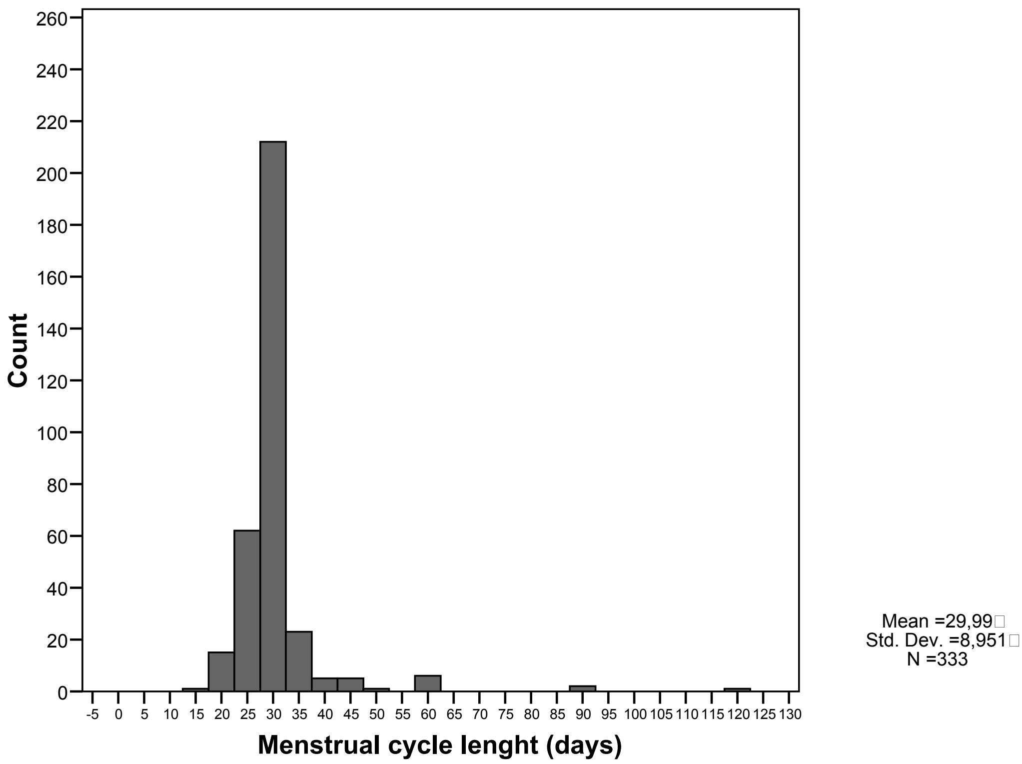


Figure 3.27 Menstrual cycle length (non-OC)

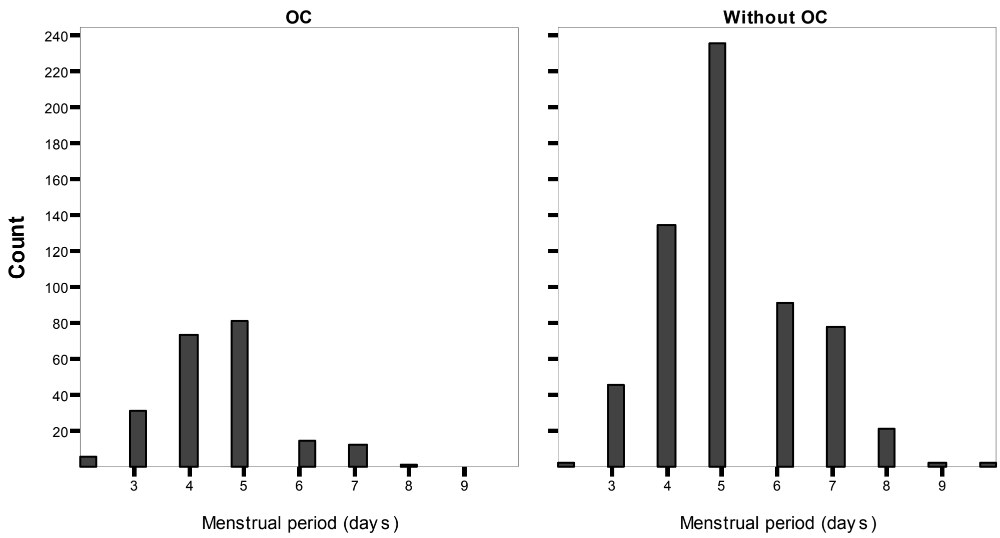


Figure 3.28 Average mens duration for OC users and for non-OC users

To better understand the menstrual cycle characteristics of female handball players, in the 2004-05 and 2006-07 seasons the players were also questioned about the age of first OC usage and the reason for its use. The questions were included in an open answer format.

For this analysis we just considered the last questionnaire completed by the players. Figure 3.29 presents the age distribution for the first OC usage.

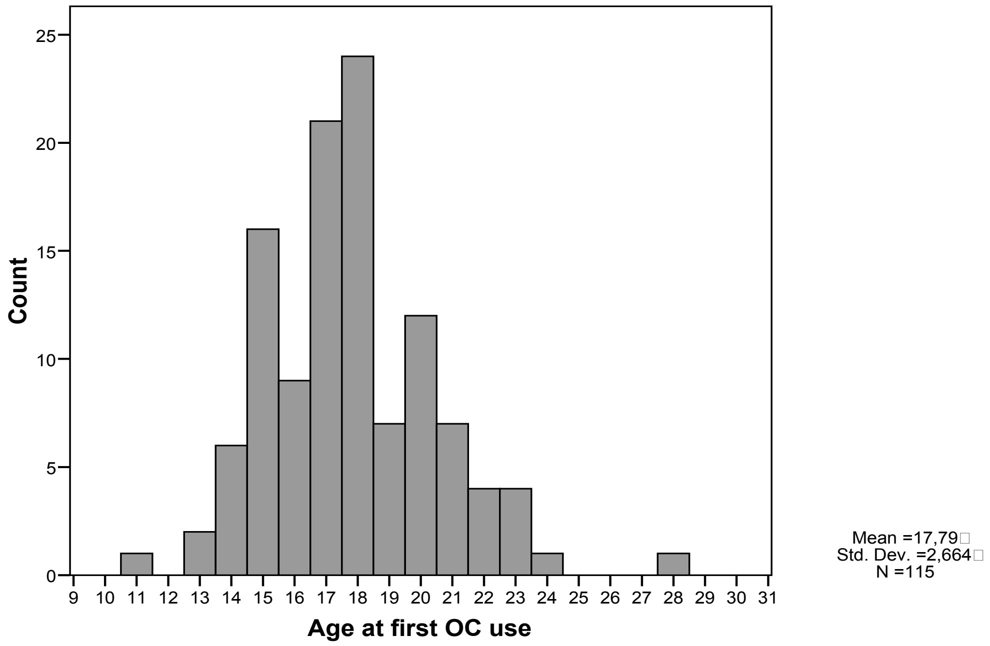


Figure 3.29 Age distribution at first OC usage

To determine the duration of OC usage we considered the reported age of first OC use and the periods of OC usage interruption, until the last completed questionnaire. The results are displayed in the following figure (3.30).

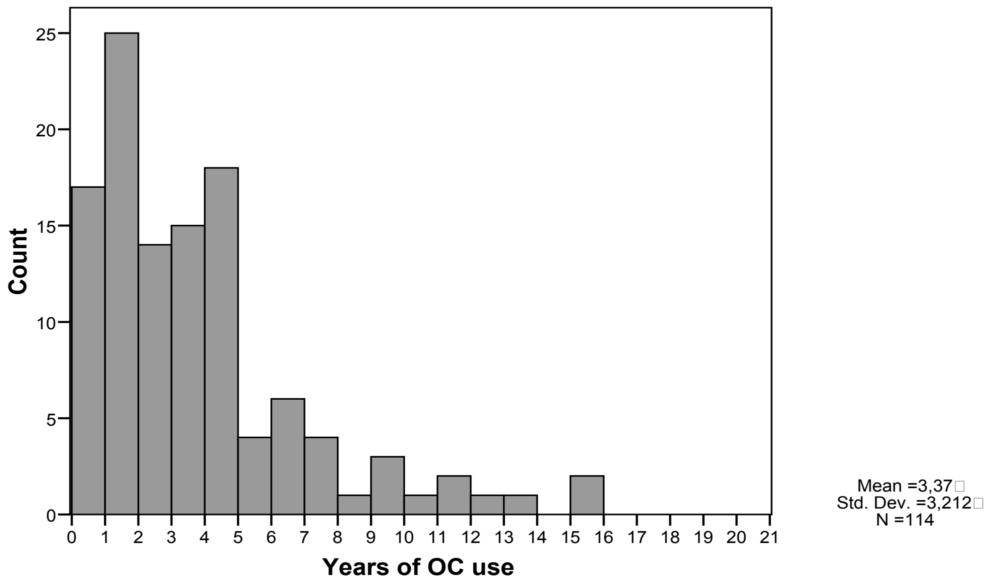


Figure 3.30 History of OC usage (years)

Figure 3.31 represents the time distribution between menarche and first OC usage. The average time was about 5 years, although in 29% of the cases this occurred just after 3 or less years.

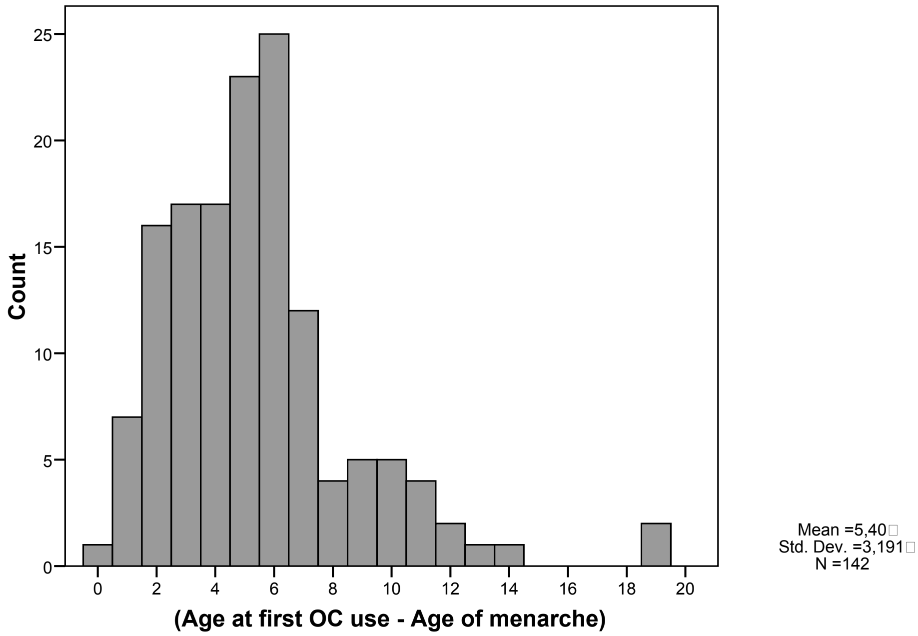


Figure 3.31 Time since the age of menarche to the beginning of OC usage

The reasons for starting OC usage were not known in 30% of the cases (n=58), and in the other cases a large spectrum of causes were identified, sometimes in a combined way. The most commonly reported motives were: menstrual cycle irregularity (33.6%), contraception (18.0%), menstrual pain (12.4%), acne (11.8%) and ovarian cysts (1.0%). Because this question was formulated without a fixed set of possible responses, in the cases of menstrual cycle irregularity it is not possible to accurately determine which menstrual cycle disturbances justified the use of OC. Only one player reported to be in a state of amenorrhea.

Some players (n=23; 12%) reported to have changed their OC drug, predominantly because of unwanted secondary effects and/or medical advice (*e.g.* weight gain, abnormal vaginal bleeding or missing periods).

The menstrual cycle symptoms experienced by the players not using OCs were analysed. These symptoms are usually referred to as premenstrual syndrome (PMS) but we also searched to see if these symptoms lasted through out their menstrual period or not (table 3.44).

Table 3.44 Presence of *premenstrual symptoms* during the menstrual cycle in non-OC users

Menstrual symptoms	Presence of symptoms N (%)			Absence of symptoms N (%)	*Missing*
	Premenstrual phase	Period phase	In both phases		
Irritability	109 (16.7)	89 (13.7)	24 (3.7)	403 (61.9)	26 (4)
Insomnia (sleeplessness)	21 (3.2)	20 (3.1)	2 (0.3)	580 (89.1)	28 (4.4)
Hypersomnia (sleepy)	30 (4.6)	55 (8.4)	15 (2.3)	525 (80.6)	26 (4)
Acne	157 (24.1)	61 (9.4)	41 (6.3)	364 (55.9)	28 (4.4)
Tension	66 (10.1)	62 (9.5)	18 (2.2)	478 (73.4)	27 (4.2)
Depression	18 (2.8)	20 (3.1)	2 (0.3)	584 (89.7)	27 (4.2)
Fatigue and tiredness	39 (6)	121 (18.6)	16 (2.5)	448 (68.8)	27 (4.2)
Increased appetite	54 (8.3)	77 (11.8)	22 (3.4)	471 (72.4)	27 (4.2)
Decreased appetite	39 (6)	77 (11.8)	10 (1.5)	498 (76.5)	27 (4.2)
Anxiety	51 (7.8)	21 (3.8)	8 (1.2)	539 (82.8)	14 (2.2)
Headache	48 (7.4)	65 (10)	18 (2.8)	494 (75.9)	26 (4)
Joint pain	16 (2.5)	31 (4.8)	7 (1.1)	571 (87.7)	26 (4)
Weight gain	45 (6.9)	74 (11.4)	14 (2.2)	491 (75.4)	27 (4.2)
Low back pain	27 (4.1)	45 (6.9)	26 (4)	438 (67.3)	21 (3.2)
Decreased concentration	21 (3.2)	60 (9.2)	8 (1.2)	535 (82.2)	27 (4.2)
Mastalgia	110 (16.9)	52 (8)	41 (6.3)	420 (64.5)	28 (4.4)
Abdominal pain and bloating	90 (13.8)	158 (24.3)	78 (12)	299 (45.9)	26 (4)
Dizziness	43 (6.6)	20 (3.1)	3 (0.5)	539 (82.8)	28 (4.4)
Oily skin	32 (4.9)	43 (6.6)	21 (3.2)	528 (81.1)	27 (4.2)
Leg pain or swelling	17 (2.6)	39 (6)	3 (0.5)	566 (86.9)	26 (4)
Others (constipation, diarrhea or vomits)	3 (0.5)	6 (1)	0		

The severity of the symptoms was not asked, although some questions were introduced regarding the menstrual cycle interference on daily sports participation.

When menstruating (during menstrual period) the majority of the players feel normal and/or well (72% of the OC users and 61% of the non-OC users) in the sports context (playing/training), but a noticeable number of players reported to feel indisposed (11.5% OC users and 20% non-OC users), and others reported some kind of limitation (6% OC users and 8.7% non-OC users) or inhibition (6% OC users and 10.8% non-OC users).

Measuring the interference on handball practice of the menstrual symptoms experienced by the players, independently of any kind of hormonal therapy, about 11 % reported to be at least once unable to train and 1.5% were also unable to play. While the reasons were mostly related to abdominal pain (56%), others reasons were also reported, such as:

lower back pain, headache, indisposition, leg pain, dizziness, abnormal vaginal bleeding, fatigue and weakness.

Relative to changes in sports performance during the menstrual phase, the majority of players did not perceive any changes, independently of OC usage or not, as shown in table 3.45. In any case, their perception of changes in performance is higher during the menstrual period, which seems to be independent of OC usage (about 19% of OC users and about 30% of the non-OC users).

Table 3.45 Perception of changes in performance

	Sports performance							
Menstrual phase	No differences N (%)		Increased N (%)		Decreased N (%)		*Missing* N (%)	
	OC	No OC	OC	No OC	OC	No OC	OC	No OC
Pre-menstrual phase	138 (83.6)	389 (76.6)	7 (4.2)	62 (12.2)	3 (1.8)	9 (1.8)	17 (10.3)	48 (9.5)
Menstrual phase	109 (66.1)	297 (58.5)	10 (6.1)	21 (4.1)	31 (18.8)	149 (29.3)	15 (9.1)	41 (8.1)
After menstrual phase	128 (77.6)	384 (75.6)	12 (7.3)	19 (3.7)	11 (6.7)	62 (12.2)	14 (8.1)	43 (8.5)

In relation to smoking habits, about 5% of the players reported to smoke regularly.

3.6 Discussion

3.6.1 Methodological considerations

There is a lack of studies about the sports injury problem in Portugal. As far as we know this study is the first to attempt to characterize the injury incidence in Portuguese female handball players using a prospective methodology. But to fully characterise the spectrum of injuries mostly affecting a player’s career, we decided to combine a prospective and a retrospective approach and, whenever possible, a clinical evaluation to confirm or to complement the obtained information.

In developed countries and in the most important competitions (Olympic Games, World Competitions, FIFA or UEFA competitions) the use of injury surveillance systems for collecting information regarding the occurrence of injuries and exposure time are often used. Constrains and limitations to our study are not independent from the reality of female handball practice in Portugal. Even if handball is the most practised sport by females in Portugal, several problems for the development of this sport can be easily identified (*e.g.* structural and organizational).

Our main problems were related to the absence of physiotherapists and doctors in the majority of the female handball teams/clubs and the usual task overload of coaches. Our best efforts were done to remain in close contact with coaches/physiotherapists during the prospective period of the study, for checking and encouraging them to follow the injury report procedures. The objective was to make available, to any injured player, an *injury report form* as soon as possible and to support its completion. However, we were unable to avoid some incomplete reporting and to guarantee that all the injured players were properly followed-up. Even when there was a physiotherapist regularly present in the training sessions/matches, his main role was treating injuries. In only one club we observed, already implemented, a systematic tracking routine for injuries/complaints including the procedures adopted for each player/case.

Another difficulty was the need to change the investigator responsible to establish and maintain the link with each club when entering the second prospective study. We also noticed that at a non-professional team level and when the research resources are scarce (human and financial) there is a critical necessity of committing the players and coaches/physiotherapists with the study.

The collected data about injuries was predominantly based on the players' self-reports using the provided *injury report form*. In addition, detailed and accurate information on the injury circumstances is limited by the player's ability for recall and interpret what really happened (Krosshaug *et al.*, 2005); which can also have been influenced by the coaches' interpretation regarding the injury situation. Nevertheless, we believe that whenever an injury became a landmark in a player's career, like when an ACL injury is sustained, it's unlikely that the player would have difficulties in recalling the circumstances of injury (the sporting situation). Olsen *et al.* (2004) supports this belief when by confirming that players accurately reported the external mechanisms of their ACL injuries when compared with video analysis of the events.

It's possible that the reports could have missed some injuries or its type been imprecisely described, particularly in the case of slight and minor injuries or overuse injuries. The registration and classification of overuse injuries was a difficult task. When justified and whenever the clinical evaluation was not performed, the player was contacted to clarify the reported symptoms. In comparison, injuries with a higher severity were probably better diagnosed, because they usually demanded some sort of medical examination, and in some cases complementary diagnostic exams. A fair amount of the injured players were medically followed-up by the supervisor of this study and, in some cases, also surgically assisted by him, making the injury characterization easier to obtain.

In other cases, the players' difficulty to accurately describe the diagnosis made by medical professionals, mainly in the case when very technical terms had been used, could be a possible source of data contamination. To minimise this problem, we asked assistance to coaches in the completion of the questionnaires, particularly in the case of young players. However, the remaining inaccuracies are extremely difficult to detect and correct.

It is also possible that some injury report forms were lost/not returned, deflating the real injury incidence. For instance, two clubs lost all data recorded in that season. This was easily detected and properly considered in the statistics but the less severe cases of under-reporting are much harder to detect.

The information on exposure time (number and duration of training sessions and matches) was provided by coaches on a collective basis (the team), because it becomes very difficult to obtain accurate exposure time of each individual player.

Despite our best efforts, the above described procedure limitations on injury data recording could not be completely overcome and so the results of this study should be viewed with some moderation.

3.6.2 Injury measures

A methodological difficulty emerged when we aimed to compare the injury incidence with other studies, because of differences in injury definitions, procedures for collecting data and characteristics of the population/samples (*i.e.*, age of players, sex, handball development phase, type of competition). Moreover, our study combines different sub-groups, regarding its number, playing level and age.

The senior level presented the highest injury incidence rate in both prospective studied seasons. In the 2003-04 season there were significant differences between the two senior divisions, with a superior rate in the second division; this tendency was not observed in the 2004-05 season. Several circumstances explain these differences; one of the most important is the change in sample size (54 in 2003-04 and 96 in 2004-05).

At the senior level, most of the published studies were done with male handball players or during main competitions (both sexes), as summarized in the table 2.3 (chapter 2). However, in international handball tournaments, no difference at all (Headey *et al.*, 2007) or only a slightly higher injury incidence in male (Junge *et al.*, 2006), was found. The incidence values that we obtained for the female senior players (3.7 injuries/1000 practice hours) are similar to those previously reported for male senior players. In two prospective studies with male senior players by Nielsen and Yde (1988) and Seil *et al.* (1998) the incidence per 1000 practice hours was 4.6 injuries and 2.5 injuries, respectively.

The injury incidence increased with the increasing age of playing categories (from age 10 to adults). Globally, in young players with less than 18 years of age (n=289) we found an incidence of 2.4±3.8 injuries per 1000 playing hours. In comparison to other studies, this value is not much different, having been computed 3.4 injuries per 1000 practice hours in young female handball players (age 16 to 18) (Wedderkopp *et al.*, 1997) and 2.6 injuries in young male handball players (age 12 to 17) (Massada, 2001). Also Olsen *et al.* (Olsen *et al.*, 2006) found no injury rate differences between young female and male handball players (1.3/1000 player hours vs 2.1 injuries/1000 player hours).

To our understanding it's quite reasonable to find an increased injury incidence in the superior playing categories, which seems to follow the increasing demands of the handball practice as well. The playing categories represent different phases of a continuum process of sportive preparation until the highest level. During this process, the juvenile and junior categories configure the most important phases for a player affirmation, combined with a stage of playing specialization, being relatively frequent to find the best players of a certain age category playing also in a superior category. In 2006-07 we found that 28.4% of all players were in this situation. If we compare figures 3.14 and 3.15 which represent the number of previous injuries relative to age category and playing categories, respectively, there are significant differences when a junior player plays only in her category or plays also in the senior category. This can be explained by the evident differences in the game at junior and senior level, mainly expressed in the type of opposition and the efforts to succeed. We also know that the best young players are those who usually are selected to play at senior level before the established age; which may suggest that these players are more exposed to matches and are key elements within their junior teams and maybe also in the senior teams which, by itself, represents a different type of exposure to injury risk. But, we don't know if there are differences regarding the years of practice between these two types of junior players which can contribute to the findings.

Considering the reported *usual playing position*, which is linked with some kind of function specialization, we were unable to find differences in the injury incidence or number of previous injuries. These results can have several possible explanations, but this is most probably related with the fact that many players frequently played in more than one position, a desirable fact in young categories.

3.6.3 Injury type, localization and severity

The lower extremity accounted for the highest number of injuries (about 60%) independently of the studied season and methodology used. These findings are consistent with the published ones, from young to adult players of different levels and sexes (Langevoort *et al.*, 2007; Junge *et al.*, 2006; Olsen *et al.*, 2006; Seil *et al.*, 1998; Wedderkopp *et al.*, 1997; Nielsen & Yde, 1988 1224).

The ankle and knee injuries are the most prevalent, but the ankle was 1.5 times more affected than the knee. These results are consistent with several published results (Petersen *et al.*, 2005; Wedderkopp *et al.*, 1997; Yde & Nielsen, 1990; Nielsen & Yde, 1988). However, Olsen *et al.* (2006) found a similar proportion of injuries in the knee and the ankle in both young female and male players.

At the senior level and in major international handball tournaments a close proportion of injuries between the ankle and knee was also found when applying consistently an injury-reporting system (Langevoort *et al.*, 2007; Junge *et al.*, 2006). We found that the rate of ankle to knee injuries (including the Achilles' tendon) (figure 3.16) decreased with the increase in age category, being below one (0.75) at the senior level. We do not know the reason for this contrast with the published results, but admitting that the overuse knee injuries could have somehow affected the ratio we analysed it taking into account only the acute injuries. The tendency remained about the same (figure 3.17).

Ignoring categories, we found that the ankle injuries accounted for 37% of all the acute injuries and the knee for 19.5%, which collectively represents more than half of all sustained acute injuries.

Another important finding was that from the *severe* injuries, 81.6% of the cases affected the lower extremity, mainly the knee (50%) and the ankle (25%). The average time of absence from knee injury increases sharply until the juvenile category, stabilising afterwards (figure 7). In the juvenile, junior and senior categories the average absence time is 3 times more for recovering from a knee injury than from an ankle one.

The differences in the consequences of these two types of injuries are even more expressive when analysing the associated number of surgeries. The rate of surgeries per knee injury was almost 68% of all interventions versus 20% per ankle injury. We did not account for financial consequences but they are unequivocally serious.

These findings stress the need to focus on the knee injury causes, mechanisms and its possible prevention.

Possibly as a consequence of our methodology, where we requested a full report of all complaints from each player (later submitted to a clinical evaluation – and in some cases further complementary exams) we found a superior ratio of overuse injuries (26%) compared with other reports – 7% to 21% in young players (Wedderkopp *et al.*, 1997), (Olsen *et al.*, 2006) and 17% in senior female players (Myklebust, 2004). However if we analyse only the overuse injuries reported prospectively the rate decreases to values more in line with these studies (19.4% in the season 2003-04 and 12.6% in the season 2004-05).

The main regions of the body affected by overuse injuries were in the spinal column/back (54%), shoulder (24%), knee (23%) and elbow (16%). Further investigation on an association of these injuries with the playing position, revealed that only in the case of the elbow there was a clear association with the playing position. This association was with the goalkeepers who suffered 64% of all the overuse elbow injuries, mostly tendinosis (87.5%). It's important to underline that the definition of injury used was not restricted to time-loss injuries. Typical elbow overuse pathologies of the handball goalkeeper clearly illustrate the possibility of physical complaints occurring, usually expressed by pain and associated functional limitation, without time-loss.

Recent studies on major international tournaments (Langevoort *et al.*, 2007; Oehlert *et al.*, 2004) have revealed high rates of head injuries. In our study we found an overall percentage of head injuries of only 2%. In fact, these differences are not surprising and can be attributed to several factors: first and foremost the context of the studies is different – a full season, including practice sessions versus a sequence of very important matches with a high psychological load. Secondly, we can not compare young players with elite ones – the attack concepts and skills (individual and collective) are very different and so are the demands on the defence. The speed and power of the game and of the players has, of course, a role in this too. So, generally we find it hard to compare the game played by young national level players with elite senior ones.

3.6.4 Injury context and external mechanisms

The majority of injuries occurred when practicing handball at the club level. The number of participating payers which were involved in any type of team selection was quite significant (ranging from 19% to 26%) although only 5% of injuries occurred in a selection context. This should be viewed with caution because we do not know which type of load was involved and it's relation with the injury incidence should be further investigated.

In the present study, 76% of injuries occurred in a match situation (47.4% was official and 28.6% in training). These values are quite relevant if we take into account that, on average, a player was exposed to training 16 times more than to matches, during the two prospective seasons studied. Other authors also found similar results and the conclusion is no different: the risk of injury in matches is much higher than in trainings. For instance, in male senior handball players, 77% of injuries were sustained in matches (Seil *et al.*, 1998). Stated in another way, the ratio between the injury incidence in matches versus training, in young female handball players, revealed rates of 10.4 (Olsen *et al.*, 2006) and 11.9 (Wedderkopp *et al.*, 1997).

In the course of the matches, the number of injuries slightly increased in the second half (figure 3.32). A similar pattern was described by Langevoort *et al.* (2007) for major international tournaments. Also converging with their findings, there was a decreasing number of injuries towards the end of the matches, which is surprising. Admitting that there is a relation between the injury risk and the match's importance or even with a specific match moment, further investigations should focus on the critical moments of matches, which may not be coincidently with the end of matches. We only tested the players subjective perception on the match's importance and, in most cases (70%), the injuries occurred when players perceived the match as important, very important or decisive. This suggests a commitment based risk, which is not counter intuitive.

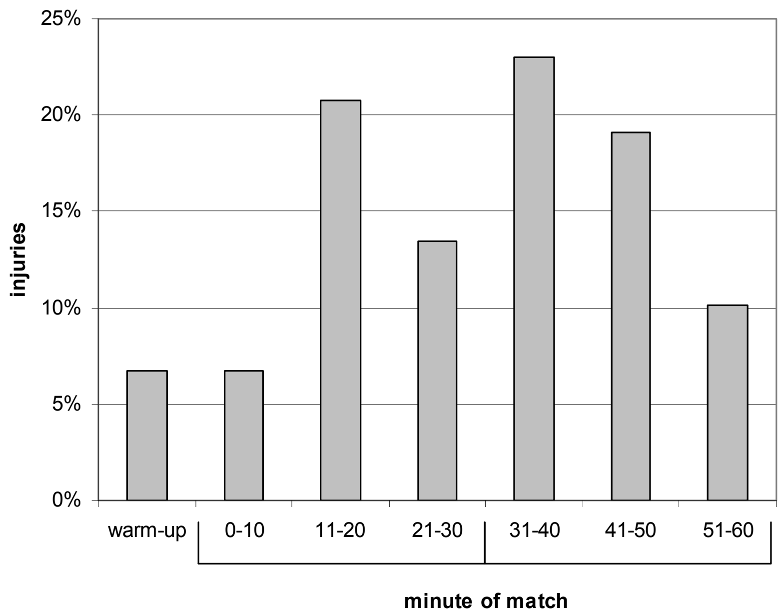


Figure 3.32 Injury distribution by match phase

In a match, we found a high number of contact injuries (66%, including the cases of contact with an object), with more than 40% of those related to aggressive opponent behaviour. In recent major tournaments, there is an obvious tendency towards a high rate of injuries in contact circumstances (75% to 92%) and in foul play situations (50% or more) (Langevoort *et al.*, 2007; Junge *et al.*, 2006). In whole season's analysis, 51% of all injuries in young female players were contact injuries (Wedderkopp *et al.*, 1997) while for male the values ranged from 53% to 61% (Seil *et al.*, 1998; Nielsen & Yde, 1988). This high incidence of contact injuries explains partially why the injury rates are so much higher in matches than in training. On the other hand it is difficult to devise strategies to prevent or reduce the impact of these contact induced injuries.

We have found a three-fold increase in injury rates when comparing the defensive with the offensive phases. This is a little higher than the two-fold increase published by Olsen *et al.* (2006) and Seil *et al.* (1998) and than the 2.5-fold increase published by Wedderkopp *et al.* (1997). Of the injuries sustained in the defensive phase we found that about 20% are suffered by the goalkeepers. When we detailed the sub-phases of the game we found that 2/3 of the attack injuries occurred in the positional attack while 1/3 occurred in the fast/counter attack. About 5/7 of the defence injuries occurred in the positional defence while 2/7 occurred in the transition attack-defence.

This may allow us to speculate that the game model (the attack, defense and transitions) of a team may have some consequences in the frequency and mechanism of injuries. In a similar way the recent rule changes, promoting a faster game, may also have similar consequences.

We also analysed the playing situation when an injury was sustained. The majority (70%) of the injured players were in a back position (left, middle and right) followed by the goalkeepers (10%). This is in contrast with the results from Seil *et al.* (1998) who found no differences between any field positions, from Olsen *et al.* (2006) who found similar rates for wing and back players or from Nielsen and Yde (1988) who found no difference in incidence between line and back players. We may suppose that these works attribute the injuries to the usual playing position of the player and not to the actual playing situation when injured.

While the minority of the injuries were sustained when being in possession or in dispute of the ball, in the counter/fast attack this rate increases to about 70%, as there is a need to attack the ball carrier immediately. These results have no equivalent in the published literature as analysing the ball position is not standard.

3.6.5 Knee injury and ACL rupture measures

During the two prospective studies the ACL injuries represented 30% of the reported knee injuries. The global knee injury incidence was 0.7± 1.9 per 1000 hours of practice, but the knee injury incidence was more than two-fold in the first studied season in comparison with the second (1.3 vs. 0.5 injuries; $p \leq 0.05$). We do not have a good explanation for these findings, except the possible random fluctuations in a small population. Behind a possible size effect of the sub-groups analysed, there are sportive factors that can be related with these results. For example, we can not exclude that from one season to the next, there were important changes in the training process and also in the competition demands of the participating teams that could have produced changes in the knee injury incidence and in its risk.

In the first national division there are still only 4 or 5 teams with a stable organizational structure that are always competing for the top places, while all the others are involved in promotion or demotion to or from the 1st division. This produces cyclic changes in the type of training and match exposure. Perhaps these specific circumstances are connected with knee incidence injury changes when comparing the 1st and 2nd division between the two seasons.

On average the ACL injury incidence, during the two studied seasons, was evaluated at 0.2 ± 1.0 injuries per 1000 hours of practice, including the juniors playing in the senior category. Our results are similar to other studies (Petersen *et al.*, 2005; Myklebust *et al.*, 1998). Myklebust *et al.* (1998) found an incidence of 0.31±0.06 ACL injuries per 1000 player hours for the elite female Norwegian handball team (about 5 times more than the male players). Peterson *et al.* (2005) found and incidence of 0.21 ACL injuries per 1000 hours in female German handball players.

In the season 2003-04, and among the group of players competing in the second division, we estimated a rate of 0.9 ACL injuries per 1000 hours of practice, which was quite high. As explained for the incidence of knee injuries, the estimated values could be misleading because of the small sample size. Even though, the differences found between the computed incidences of ACL injury regarding the two divisions were significant, being superior in the 2nd division. The results presented by Myklebust *et al.* (1997), using a sample of 3392 Norwegian handball players from both sexes, revealed a superior ACL injury rate in the upper divisions. In this study it was also found that 1.8% of the female players suffered an ACL injury by season, which was almost twice the value found for male handball players (1.0%). In comparison, and considering not only seniors but all players at or above the juvenile category, we found a higher percentage of players who had sustained an ACL by season: 8.3% players were injured in the 2003-04 season, 3% of the players were injured in the 2004-05 season and a global rate of 4.8% ACL injuries by season.

In the season 2006-2007, on average, from a team of 14 senior players, 2.5 had suffered an ACL injury in their handball career, which is about 17% of the players. When considering all categories except only the infants and initiated players, we found that 8.2% of the players had reported a past ACL injury.

In the prospective cohort study (two seasons), it became quite difficult for us to control the response rate, particularly in the juvenile category and in the second division teams, because of the high frequency of players joining and leaving the teams or even interrupting the practice during the season. In the senior category the response rate was about 49% in the 2003-04 season, and in the next season it increased significantly (75%). In the retrospective study the return rate was almost 100%.

Having considered the number of non-returned questionnaires, and admitting that all players which had sustained an ACL returned their questionnaire, maybe the proportion of ACL injured players and non-injured was over estimated for this reason. We also have to notice that it is possible that some ACL injuries remained undetected, particularly in players from young categories and from the second division. For instance, when questioning a group of players from the second division, one of them reported not having had previous injuries, and informally described some kind of knee instability, with frequent knee sprains followed by knee swelling but, in her opinion, *nothing special*. After advising the player and the coach for medical attention, an ACL injury was diagnosed and later surgery was performed. In another case, a 14 year old player sustained an ACL rupture but the Club decided not to participate the incident to the insurance company. This player was clinically examined by the supervisor of this study and reconstructive surgery was advised. To our knowledge this player dropped out of handball.

3.6.6 Knee injury type, severity and external context

Our results confirm that knee injuries are of great concern for players, coaches and researchers, among others. This is due to the alarming number of knee injuries sustained (20%, n=250) and the highest impact in terms of days absent from practice (50% were severe injuries), need of surgery (28%, n=71) and unavoidable damages in the performance and well-being of the players. However, we were unable to find published detailed information on knee injuries in handball players, except for the ACL injuries.

Langevoort *et al.* (2007), listing the prevalence of knee injuries in major championships between men and women reveal no substantial differences, with values ranging from 10% to 16%. We found little published data related to women's knee injury incidences to compare with our results. Wedderkopp *et al.* (1997) found 24% of knee injuries in young female players in a season's long retrospective study while Olsen *et al.* (2006), in a 7 month prospective study on young players of both sexes, found 26% of knee injuries.

When paying attention to the most affected knee side (right or left), we concluded that the left knee was the most affected (about 48%, plus 12% on both knees), a rate that increases when just those injuries requiring surgery are considered (60%). The players affected by knee injuries were mostly right-handed (89.6%) with just 9.6% left-handed, which is not much different from the overall group laterality. The high left knee incidence is however independent of hand laterality and injury nature (acute vs overuse). One could expect a superior injury rate between left/right knees considering that, with exception of the goalkeepers, common load limb is quite asymmetric - if one usually plays the ball with the right hand, usually the most loaded limb and also most exposed to physical contact is the left one. However one can also argue that the right limb is not so well trained to support both the typical and the atypical loads (*e.g.*, some high risk game situations), as this applies both to overuse and to acute injuries. Concluding, the knee injury mechanism reveals as more complex than expected.

The acute knee injuries are of great concern because of their high impact in terms of absence from practice, which is quite evident from the juvenile category onwards (figure 3.18). The majority of these injuries occurred in a match (74%, official or training matches) even though the players spend a lot more time exposed to practice than to matches (about 16 times more) *i.e.* the game incidence is much higher than the practice incidence (about 46 times more). This may have to do with a particular motivation or engagement with the competition, which is corroborated by the players' self-perceived report that most of the injuries occurred in important, highly important or decisive matches (70.5%).

This strong link between players' commitment with real match opposition and knee injury incidence, combined with the fact that about half of the injuries occurred without physical contact (only in 15% of cases did the player report an aggressive behaviour from the opponent), may lead us to think that there is a need to improve the player's ability to withstand these higher dynamic opposition levels. As 68% of the knee injuries occurred when landing from a jump or in a plant-and-cut situation, these movements should receive special attention.

The majority of the knee injuries follow a pattern similar to all other injuries: occurring during the positional attack or fast/counter attack phases (72%) and affected mainly the back players (97%).

The fact that the majority of injuries occurred early on in a player's career (50% before 8 years of practice and before 17 years old) is a possible sign of this misadaptation between skills (physical and collective) and match load.

Considering the severity distribution of the knee injuries, the majority of severe injuries are the knee sprains (74%) but the single category is the *knee sprain with ligament rupture* which accounts, on its own, to 56% of these. In the ligament ruptures the majority (67%) and more severe on average (142 days of absence) are ACL ruptures. They accounted for 52% of all surgeries and more than twice the average absence time of any other injury.

Curiously the average age of first knee sprain (18 years) is one year less than the average age of first ACL rupture. This may suggest a connection between injury severity and increasing match demands.

3.6.7 ACL rupture context and external mechanisms

To understand the injury mechanism and to develop specific intervention methods and strategies to prevent injuries, it is essential to precisely describe and detail the playing situation involved. In the case of ACL injuries we believe that its causes are strongly linked to the specific game structure and function as a couple of studies have found. For instance, in the English Professional Rugby Union, the ACL and MCL injuries were found to result more often from contact than from noncontact situations (about 2:1) (Dallalana *et al.*, 2007) contrasting with handball where 95% of all ACL injuries result from non-contact situations (Myklebust *et al.*, 1997). Another perspective is that the way male and female adapt and perform is somehow different in the different sports. Agel *et al.* (2005) found that in basketball the ratio of ACL non-contact vs contact is similar in men and women (from 2:1 to 3:1). However in soccer they found that this ratio was different between men (1:1) and women (3:5). In any case the consensus is that there is a superior overall ACL injury rate in female relatively to male, in jumping and cutting sports (Agel *et al.*, 2005; Bonci, 1999).

In our study the ACL injuries followed the same general pattern of all the knee injuries found, namely they occurred mainly during matches (75%), in the attack phase (all cases except goalkeepers), affecting mostly the players in the back position (82%), without contact (61%) and when landing from a jump (41%) or in a plant-and-cut movement (also 41%).

Two published reports on the subject are from Myklebust *et al.* (1998; 1997). The 1997 one is about a sample of handball players from the three Norwegian top divisions, referring both sexes and including both cruciate ligaments. They state that the ACL ruptures account for 94% of all cruciate ligament ruptures and that the female/male incidence ratio is 180%. The 1998 one is exclusively about ACL injuries in elite Norwegian team handball players. They found a similar or superior rate of ACL injury occurrence during games (86% in the 1998 paper *vs.* 75% in the 1997 one) and in the attack phase (93% *vs.* 90%). In the 1997 study slightly different results were also found in other variables, with 84% of injuries distributed among back and wing players and with a superior rate of non-contact ACL injuries (88% in the 1998 study *vs.* 95% in the 1997 one). The major differences, however, are about the relative importance of the plant-and-cut movement (83% in 1998 and 55% in 1997) and landing from a jump (17% in 1998 and 30% in 1997).

The data for the two above mentioned studies was obtained through questionnaires. A classification of contact injury was attributed when the situation somehow involved player-to-player contact regardless of real cause or contact interference in the injury occurrence (similarly to our study). Olsen *et al.* (2004) used a different contact ACL injury classification, considering *contact* exclusively when a direct blow to the knee was suffered (and confirming the player's interpretation with video analysis performed by medical doctors and national team coaches).

During our data collecting process, besides the reported injury mechanism (questionnaire), we also informally interviewed the players, what clarified some issues (and, in some cases we even witnessed injuries first hand). For instance, one player reported to have been injured during physical contact with a defending player, although she attributed the injury occurrence to friction produced between her new sport shoes with the artificial floor while performing a rotational faking movement. In another case the player described a lateral side impact or collision of the lower limb by another player in consequence of falling. In this last case we have no doubt in attributing or to classify this as a contact ACL injury.

Looking at the main differences between our results and the results from Norwegian female players we came up with a couple of possible reasons. Norway is one of the most contributing countries for the development of handball and they include the world's handball elite, which means that their handball level (*i.e.*, game model, conditioning, experience and player skills) is not comparable to ours. With respect to our experience, there are specific characteristics in our way of playing that can contribute to explain some diverging findings. For example, it is possible that the back players have higher injury rates because of the incipient game organization, with the collective playing decisions dependent mostly on these player's abilities, which perform most of the faking actions (and at a higher initial speed). Also the higher proportion of contact ACL injuries is possibly related with the not so highly sophisticated individual abilities that can contribute to avoid the defence contact, independently of the real contribution of the contact mechanism to the injury occurrence. Moreover, even if the results are unequivocal to underlining an increased risk of ACL injury when playing matches than in training, being the playing positions affected distinctively, there is no available data on the load differences between match and training and therefore all advanced explanations are just conjectural.

Attempting to explain the ACL proportion of injuries between sexes in handball, and looking exclusively to the elite levels, where one expects similar training demands and conditions (Myklebust *et al.*, 1997), we do think that there is a lack of studies trying to understand the game differences between sexes. For example, we can argue that a superior percentage of mistakes in decision-making by females in comparison to males (Spatte, D. - personal communication, 2006) can somehow condition the aetiology of these injuries to a not yet investigated field. Other possible characteristics, such as a higher proportion of counter/fast attack and a superior number of playing situations near the restrictive areas in the handball played by girls (Bon, 2002), concurs to create sex specific match exposures. All of this makes a gender specific game interpretation and consequently a sex specific biomechanical load which, together with a different anatomy and physiology may explain some of the differences in injury incidence. Still there is a need to understand why the ACL injury rate is so high in matches: what is so fundamentally different between matches and trainings? We may ask if the training fails to prepare the players well enough for the match adversities, or if the match represents a load non reproducible in training, with the possible exception of match trainings. This reasoning refers to non-contact ACL injuries, because collision injuries or accidents are quite difficult to prevent and are much less prevalent.

On a prevention basis, Olsen *et al.* (2004) noted that some ACL injuries occurred when landing on just one leg after a jump shot, with the players' motion, perturbed or not, by an opponent. Myklebust, during the 1st World Congress on Sports Injury Prevention (personal communication, 2005), suggested that landing with both legs is an important strategy for preventing this type of ACL injury. However, this does not necessarily address all those situations when the player is off balance.

The high number of non-contact ACL injuries, particularly in the move-and-cut actions, suggests the need to address the possible contribution of the ground-shoe interaction. We did not found any specific correlation between the floor material and the number or severity of knee and ankle strains (figure 3.33), although there were a large number of unanswered reports (missing data) on this matter. There are not many studies on this subject, but Olsen *et al.* (2003) reported a possible increase in ACL injury risk when synthetic floors are used. This question must be further investigated. Moreover, in Portugal there are different types of synthetic floors and the majority of halls are not acclimatised, which allows for radical changes in the playing surface conditions, with air moisture condensing and turning the surface quite slippery (one player attributed the sustained ACL injury to this floor condition).

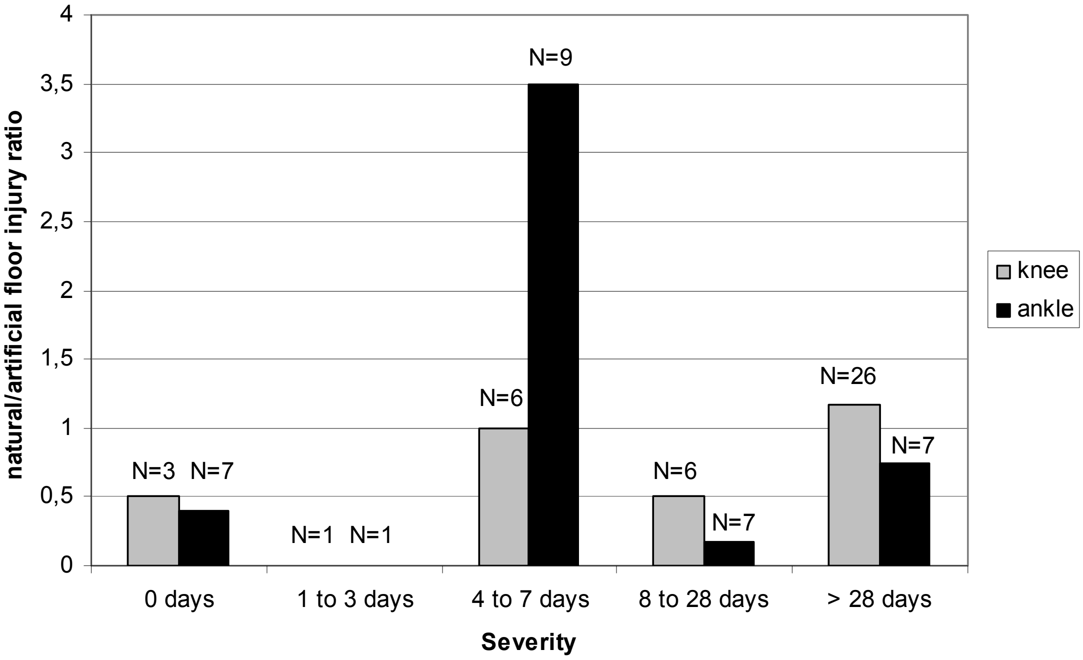


Figure 3.33 Noncontact strain distribution

In an attempt to better understand the contributing factors to the high female ACL injury incidence, we looked for possible relations between the hormonal environment and the ACL injuries, using indirect indicators.

A factorial analysis on *age*, *years since menarche*, *years of practice*, *years of OC intake* and *time absent from practice* (figure 3.24) reveals a strong identification of the first three with one of the synthetic orthogonal factors and the last one with the other orthogonal synthetic factor. The parameter *years of OC intake* is explained about half by the first factor and about half (negatively) by the second one. This suggested a negative association between *years of OC intake* and *time absent from practice* (OCs as ACL injury severity moderator) what needs further investigation. Maybe the female hormonal environment modulates the biomechanical and physiological characteristics and affects the ACL injury risk.

3.6.8 Menstrual cycle characteristics

Strenuous exercise has been linked to menstrual disturbances since the seventies (Malina *et al.*, 1978) including delayed menarche or secondary amenorrhea. On the subject of exercise induced primary amenorrhea, we have to consider several factors: menarche age and its dispersion in the general population, the type of sport and exercise loads, the age of practice start and the duration of the training period prior to menarche.

In our sample (N=804) we have found a reported mean age of menarche of 12±1 years, which compares well with some reported studies about the general Portuguese population (Vasconcelos, 1995), where an average of 12.4±1.1 years was found. Even the highest menarche age reported in our study (16 years) is not a clinical problem by some standards (*e.g.*, Ireland & Ott, 2004). Analyzing just those players having started the handball practice prior to menarche (N=351), *i.e.* those in which the sport practice could have had an influence in the menarche (figure 3.26), we have found a very low association between *years of handball practice* and *age of menarche*. The weekly number of training sessions (2 to 3, usually), its typical duration (less than 2 hours) and intensity does not seem high enough to induce delayed menarche. Some authors (Malina, 1983) have suggested a kind of sport selection based on some physical traces enhanced by a late menarche. So, this effect could indirectly induce a higher average menarche age in the sporting population. We did not find any evidence of this effect, possibly because the number of players is still low in the youngest categories and no structured form of selection is needed. The average age of starting to play handball was between 11 and 12 years (infants category), which is the advised age to start in this sport (Estriga, 2000).

There are several studies describing a possible association between a female hormonal environment and ligament fragility (Shultz *et al.*, 2004; Deie *et al.*, 2002; Heitz *et al.*, 1999). As OCs somewhat stabilize the sex hormones fluctuations (particularly the estrogen and progesterone) we asked for information about OC usage and checked for an association with ACL injuries. We found no significant difference (p>0.28) between the prevalence of ACL injuries in these two subpopulations (OC and non-OC users), although the raw prevalence is twice for OC players than for non-OC players in the two prospective studied seasons (22 injuries in 437 players). However, when the sample size is increased, considering all the data available (38 injuries in 804 players) we found that more than 70%

where not using OC at the time of injury. This number should be compared with the 66% of players who do not use OCs. Overall we can not say that there is a definitive conclusion from this analysis as we have to consider, at least, the characteristics of the actual cycle in those not using OCs – anovulation and other menstrual cycle disturbances.

Data gathering on this subject was compounded by the players' generalized difficulty in recalling the menarche age, start of OC usage, typical menstrual cycle length and its variations. Based on the self reports we found that more than half (53%) refered some kind of menstrual irregularity, many of an unspecified type. About one third of the OC users started their use precisely to overcome menstrual cycle irregularities including one case of amenorrhea. As we have no knowledge of the magnitude of this problem in the Portuguese general population we can not establish a connection of this with handball practice. We had also 3 reported cases (1%) of amenorrhea in the non-OC users and 20 cases of oligoamenorrheia (6%). This implies that a division of the players by the categories OC and non-OC users is not a homogeneous division as the non-OC users are probably a very heterogeneous group with respect to hormonal level exposure. From a self-perception point of view (table 3.45) we have conflicting results about the performance advantages of OC usage.

About one third of the non-OC users report some kind of pre-menstrual syndrome and about 12% of the OC users refer menstrual pain as the cause for OC usage. Again, we have no evidence that these groups differ from the general Portuguese population.

3.6.9 Self consistency - reliability

As described above, 62 players were doubly evaluated, both by the standard method (questionnaires) and by an exhaustive clinical evaluation.

Overall, 54 subjects (87% of those subjected to the clinical evaluation) did not report or were unaware of medical conditions, relevant for or consequence of handball practice. These 54 subjects, in the retrospective questionnaire, reported 116 injuries and the surgeon added another 154 (57% of the total). They range from simple sprains to full (unknown) ACL ruptures.

This is a rough indicator of the quality of the questionnaire's approach to a career long retrospective study – obtain only about half of the total injuries.

Back pain is another example of this volunteer under-report: the subjects are obviously aware of it but self-reported only 4 cases while the surgeon recorded another 25.

Considering the distribution of the clinically evaluated injuries, as reference, and the overall data gathered, the under-reported injuries were mainly above the waist (figure 3.34).

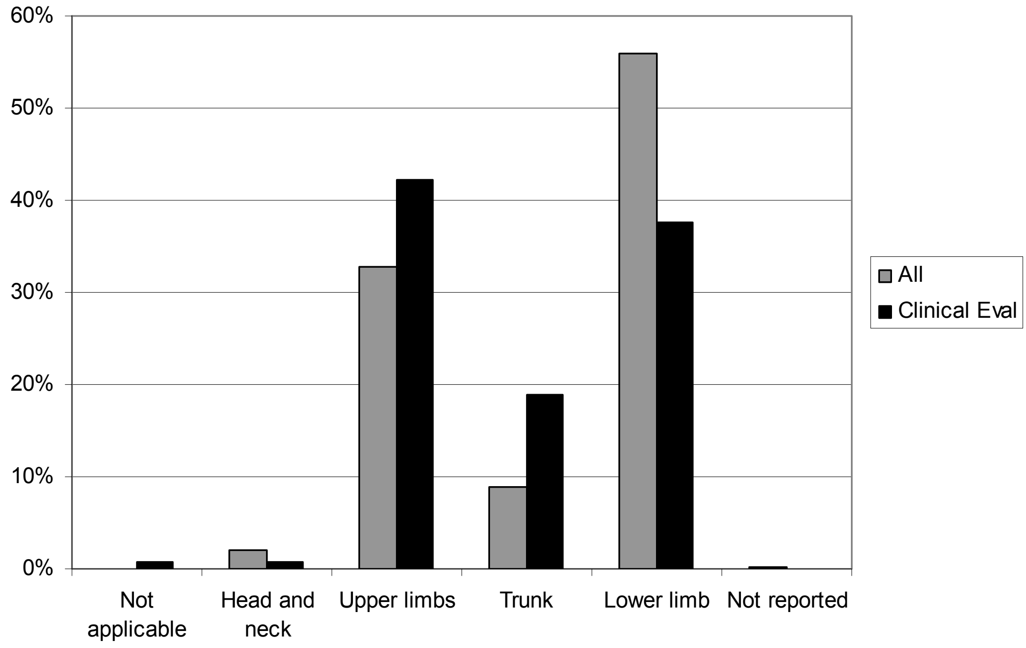


Figure 3.34 Injured body part

Subjects seem to report (remember?) the traumatic injuries more often than the overuse ones (figure 3.35)

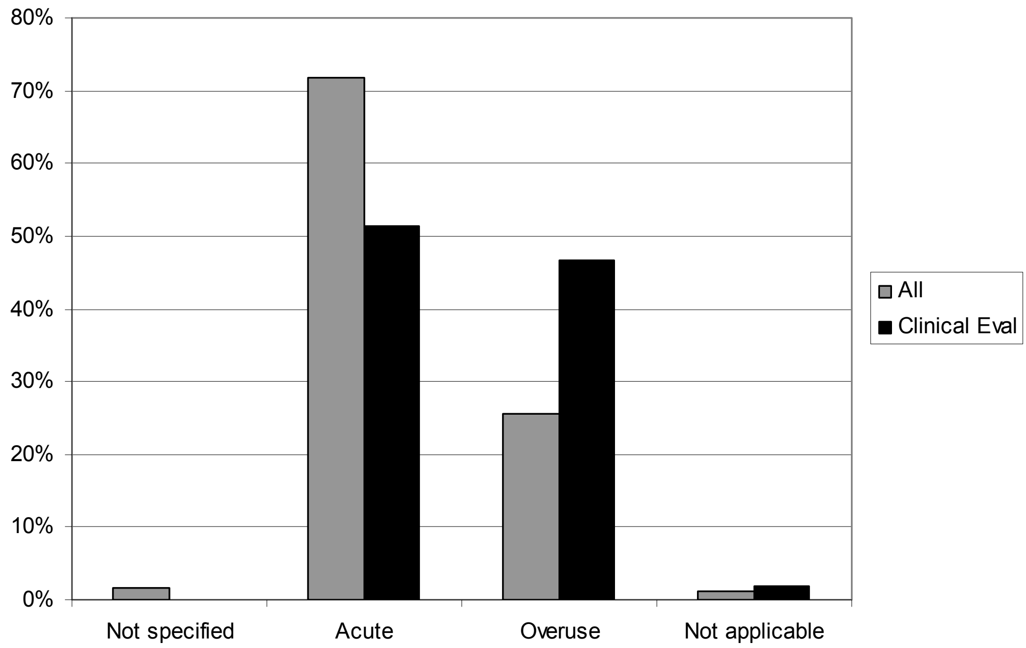


Figure 3.35 Nature of the reported injuries

Finally the subjects tend to report less the more *silent* injuries like those of the spinal column and tendons (figure 3.36).

We think this sketches, briefly, the quality of the questionnaire method for this purpose – around 10% to 20% error.

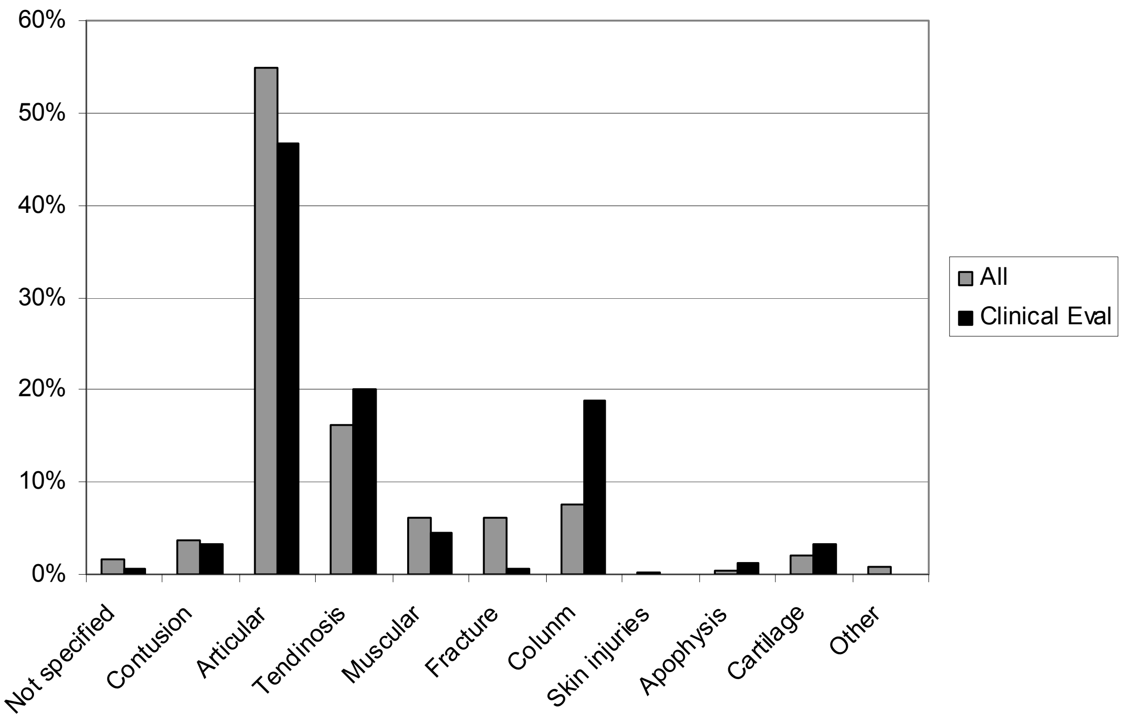


Figure 3.36 Injury diagnostics

3.6 Conclusions

The lower limbs were the most frequently injured body region, accounting for more than 80% of all the acute injuries, somehow contrasting with the focus of the proximal defense, which is mainly to restrain the opponent's upper body and arm handling the ball. Collectively ankle and knee injuries represented more than half of all acute injuries. These structures were most often injured in a match and in a playing offensive situation. Still the players spend 16 times more time in training than in matches. Maybe the training fails to stimulate the neuromuscular control of the lower limb well enough to withstand match loads and adversities.

The knee is the structure with the highest risk of severe injury in the female handball practice, accounting for almost 70% of all reported surgeries. The left knee was the most affected one (60% of the knee surgeries), most likely because for most of the technical abilities on right-handed players the left leg is the standing leg, frequently in closer proximity to the opponent.

The ACL rupture is, among the severe injuries, the most occurring one, and with the highest impact in terms of absence from practice (on average of 142 days per injury). These injuries were the motive of more than half of all the surgeries, which relates to high economical costs.

The majority of ACL injuries were sustained in official or practice matches, without contact. These injuries occurred predominantly in the attack phase, while landing from a jump or in a plan-and-cut movement, handling the ball, and in the back playing positions.

We found that 17% of the senior players have had an ACL injury during their handball career, which on average represents 2.5 ACL injuries for a team of 14 senior players. Still, this finding misses all the players who did not return to handball practice after an ACL injury (which rate we do not know).

More than half of the ACL ligament injuries occurred before the age of 19, which seem to show some mis-adaptations to the increased game demands, mainly from juveniles until seniors, along with physical and physiological puberty associated changes. Perhaps the hormonal factors may underline this increased ACL risk after growth spurt. Also ours results underline the need to improve the player’s ability to withstand the jump and plan-and-cut loadings at dynamic opposition levels, such as those observed in matches.

3.7 Next Goals

At the current development state of the Portuguese female handball teams, even the most competitive, the medical supporting structures are incipient, at best. This causes an unexpected load on the research teams.

A closer and almost daily contact with the players and (very important) with the existing health professionals is arguably the only way to improve the quality of the injury and exposure data.

Longer longitudinal studies may be important in controlling the inter-individual variations and in better understanding the true injury consequences, presently mitigated by the missing data in all the cases of practice drop out.

Additionally, a broader set of independent clinical evaluations may be decisive to assess the complete range on silent injuries. The mandatory annual examinations do not screen (nor are expected to) all these details.

3.8 References

Agel, J., Arendt, E. A. & Bershadsky, B. (2005). Anterior cruciate ligament injury in national collegiate athletic association basketball and soccer: a 13-year review. *Am J Sports Med, 33*(4), 524-530.

Bon, M. (2002). 4th Women's Junior European Handball Championship. *Handball Periodical, 10*(2), 30-41.

Bonci, C. M. (1999). Assessment and Evaluation of Predisposing Factors to Anterior Cruciate Ligament Injury. *J Athl Train, 34*(2), 155-164.

Carvalho, M. (2004). *Caracterização do perfil lesional das andebolistas seniores: Um estudo centrado na problemática das lesões ao nível da articulação do joelho.* Unpublished Monografia, Universidade do Porto, Porto.

Carvalho, M., Estriga, M. & Massada, L. (2005). *Epidemiology of injuries in female senior handball players - A knee joint centered study.* Paper presented at the 1st World Congress on Sports Injury Prevention Oslo.

Dallalana, R. J., Brooks, J. H., Kemp, S. P. & Williams, A. M. (2007). The epidemiology of knee injuries in English professional rugby union. *Am J Sports Med, 35*(5), 818-830.

Deie, M., Sakamaki, Y., Sumen, Y., Urabe, Y. & Ikuta, Y. (2002). Anterior knee laxity in young women varies with their menstrual cycle. *Int Orthop, 26*(3), 154-156.

Estriga, M. L. (2000). *Orientação desportiva : em crianças e jovens nas modalidades de andebol, basquetebol e voleibol.* Unpublished MSc, Porto University: Sports Faculty.

Fernandes, G. (2007). *Estudo do perfil lesional das atletas portuguesas de Andebol (duas épocas consecutivas).* Unpublished Monografia, Universidade do Porto, Porto.

Fuller, C. W., Ekstrand, J., Junge, A., Andersen, T. E., Bahr, R., Dvorak, J.*, et al.* (2006). Consensus statement on injury definitions and data collection procedures in studies of football (soccer) injuries. *Br J Sports Med, 40*(3), 193-201.

Gurney, B. (2002). Leg length discrepancy. *Gait Posture, 15*(2), 195-206.

Hackenberg, L., Hierholzer, E., Bullmann, V., Liljenqvist, U. & Gotze, C. (2006). Rasterstereographic analysis of axial back surface rotation in standing versus forward bending posture in idiopathic scoliosis. *Eur Spine J, 15*(7), 1144-1149.

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

Haywood, A., Slade, P. & King, H. (2002). Assessing the assessment measures for menstrual cycle symptoms: a guide for researchers and clinicians. *J Psychosom Res, 52*(4), 223-237.

Headey, J., Brooks, J. H. & Kemp, S. P. (2007). The epidemiology of shoulder injuries in English professional rugby union. *Am J Sports Med, 35*(9), 1537-1543.

Heitz, N. A., Eisenman, P. A., Beck, C. L. & Walker, J. A. (1999). Hormonal Changes Throughout the Menstrual Cycle and Increased Anterior Cruciate Ligament Laxity in Females. *J Athl Train, 34*(2), 144-149.

Hill, M. & Hill, A. (2002). *Investigação por questionário*. Lisboa: Edições Sílabo, Lda.

IDP. (2005). *Estatísticas do Associativismo Desportivo, 1996-2003*. Lisboa: Instituto de Desporto de Portugal.

Ireland, M. L. & Ott, S. M. (2004). Special concerns of the female athlete. *Clin Sports Med, 23*(2), 281-298, vii.

Junge, A., Langevoort, G., Pipe, A., Peytavin, A., Wong, F., Mountjoy, M.*, et al.* (2006). Injuries in team sport tournaments during the 2004 Olympic Games. *Am J Sports Med, 34*(4), 565-576.

Krosshaug, T., Andersen, T. E., Olsen, O. E., Myklebust, G. & Bahr, R. (2005). Research approaches to describe the mechanisms of injuries in sport: limitations and possibilities. *Br J Sports Med, 39*(6), 330-339.

Langevoort, G., Myklebust, G., Dvorak, J. & Junge, A. (2007). Handball injuries during major international tournaments. *Scand J Med Sci Sports, 17*(4), 400-407.

Lindblad, B. E., Hoy, K., Terkelsen, C. J. & Helleland, H. E. (1992). Handball injuries. An epidemiologic and socioeconomic study. *Am J Sports Med, 20*(4), 441-444.

Malina, R. M. (1983). Menarche in athletes: a synthesis and hypothesis. *Ann Hum Biol, 10*(1), 1-24.

Malina, R. M., Spirduso, W. W., Tate, C. & Baylor, A. M. (1978). Age at menarche and selected menstrual characteristics in athletes at different competitive levels and in different sports. *Med Sci Sports, 10*(3), 218-222.

Massada, J. L. (2001). *A Lateralidade Anatómica e Biomecânica. Sua repercussão na Assimetria Morfológica e na patologia Traumática do Esqueleto Axial e Apendicular do Atleta.* Unpublished PhD, Universidade do Porto, Porto.

Myklebust, G. (2004). personal communication - cited by. *Scandinavian Journal of Medical Science in Sports, 16*, 7.

Myklebust, G., Maehlum, S., Engebretsen, L., Strand, T. & Solheim, E. (1997). Registration of cruciate ligament injuries in Norwegian top level team handball. A prospective study covering two seasons. *Scand J Med Sci Sports, 7*(5), 289-292.

Myklebust, G., Maehlum, S., Holm, I. & Bahr, R. (1998). A prospective cohort study of anterior cruciate ligament injuries in elite Norwegian team handball. *Scand J Med Sci Sports, 8*(3), 149-153.

Nguyen, A. D. & Shultz, S. J. (2007). Sex differences in clinical measures of lower extremity alignment. *J Orthop Sports Phys Ther, 37*(7), 389-398.

Nielsen, A. B. & Yde, J. (1988). An epidemiologic and traumatologic study of injuries in handball. *Int J Sports Med, 9*(5), 341-344.

Oehlert, K., Drescher, W., Petersen, W., Zantop, T., Gross, V. & Hassenpflug, J. (2004). [Injuries in Olympic handball tournaments: a video analysis]. *Sportverletz Sportschaden, 18*(2), 80-84.

Olsen, O. E., Myklebust, G., Engebretsen, L. & Bahr, R. (2004). Injury mechanisms for anterior cruciate ligament injuries in team handball: a systematic video analysis. *Am J Sports Med, 32*(4), 1002-1012.

Olsen, O. E., Myklebust, G., Engebretsen, L. & Bahr, R. (2006). Injury pattern in youth team handball: a comparison of two prospective registration methods. *Scand J Med Sci Sports, 16*(6), 426-432.

Olsen, O. E., Myklebust, G., Engebretsen, L., Holme, I. & Bahr, R. (2003). Relationship between floor type and risk of ACL injury in team handball. *Scand J Med Sci Sports, 13*(5), 299-304.

Omey, M. L., Micheli, L. J. & Gerbino, P. G., 2nd. (2000). Idiopathic scoliosis and spondylolysis in the female athlete. Tips for treatment. *Clin Orthop Relat Res*(372), 74-84.

Petersen, W., Braun, C., Bock, W., Schmidt, K., Weimann, A., Drescher, W.*, et al.* (2005). A controlled prospective case control study of a prevention training program in female team handball players: the German experience. *Arch Orthop Trauma Surg, 125*(9), 614-621.

Roach, J. W. (1999). Adolescent idiopathic scoliosis. *Orthop Clin North Am, 30*(3), 353-365, vii-viii.

Seabra, A. (2003). *Lesões no andebol feminino português. Um estudo no Campeonato Nacional da Primeira Divisão - seniores femininos.* Unpublished Monografia, Universidade do Porto.

Seabra, A., Estriga, M., Cunha, A. & Massada, L. (2005). *Injuries in portuguese female handball players - a prospective study covering two seasons.* Paper presented at the 1st World Congress on Sports Injury Prevention, Oslo.

Seabra, A., Estriga, M. & Massada, L. (2004). Lesões no andebol feminino português. Um estudo no Campeonato Nacional da Primeira Divisão - seniores femininos. *Treino Desportivo, 25*(Dez), 46-51.

Seil, R., Rupp, S., Tempelhof, S. & Kohn, D. (1998). Sports injuries in team handball. A one-year prospective study of sixteen men's senior teams of a superior nonprofessional level. *Am J Sports Med, 26*(5), 681-687.

Shultz, S. J., Kirk, S. E., Johnson, M. L., Sander, T. C. & Perrin, D. H. (2004). Relationship between sex hormones and anterior knee laxity across the menstrual cycle. *Med Sci Sports Exerc, 36*(7), 1165-1174.

Shultz, S. J. & Nguyen, A. D. (2007). Bilateral asymmetries in clinical measures of lower-extremity anatomic characteristics. *Clin J Sport Med, 17*(5), 357-361.

Shultz, S. J., Nguyen, A. D. & Schmitz, R. J. (2008). Differences in lower extremity anatomical and postural characteristics in males and females between maturation groups. *J Orthop Sports Phys Ther, 38*(3), 137-149.

Vasconcelos, O. F. (1995). *A imagem corporal no período peripubertário: comparação de três grupos étnicos numa perspectiva biocultural.* Unpublished PhD, Porto University: Sports Faculty.

Verhagen, E. A., Van der Beek, A. J., Bouter, L. M., Bahr, R. M. & Van Mechelen, W. (2004). A one season prospective cohort study of volleyball injuries. *Br J Sports Med, 38*(4), 477-481.

Walsh, M., Connolly, P., Jenkinson, A. & O'Brien, T. (2000). Leg length discrepancy--an experimental study of compensatory changes in three dimensions using gait analysis. *Gait Posture, 12*(2), 156-161.

Wedderkopp, N., Kaltoft, M., Lundgaard, B., Rosendahl, M. & Froberg, K. (1997). Injuries in young female players in European team handball. *Scand J Med Sci Sports, 7*(6), 342-347.

Wells, C. (1991). *Women, Sport & Performance - A Physiological Perspective* (Second Edition ed.). Champaign, Illinois: Human Kinetics Books.

Yde, J. & Nielsen, A. B. (1990). Sports injuries in adolescents' ball games: soccer, handball and basketball. *Br J Sports Med, 24*(1), 51-54.

Chapter 4

Muscle Strength and Anterior Knee Laxity

4.1 Introduction

The risk of joint sprains, patellofemoral disorders, meniscal tears, cartilage damage and ACL ruptures seems to be higher in female athletes (adolescents and young adults) in comparison to male athletes, as reviewed by several authors (Quatman *et al.*, 2008; Hass *et al.*, 2005; Adirim & Cheng, 2003). Although we did not study the male population, in the Portuguese female handball population we found an increased number of knee strains and ACL ruptures between the ages of 14 and 19 (chapter 3).

It has been suggested that the anatomic and physiological changes occurring during female puberty increase the functional joint instability (Quatman *et al.*, 2006), which would be related to an increased risk of ACL ruptures. Curiously, this is the critical period for the development of the player's technical abilities and tactical decision-making. These improved competences (physical, technical and tactical) by themselves, make better protected players. Unfortunately the opposition is also becoming stronger and more dynamic, placing even greater demands on the players. These demands can even be greater in the case of the best young players who, frequently, play in the above age category (as well as in their own).

Throughout maturation, males experience a neuromuscular spurt whereas females show little changes (Quatman *et al.*, 2006; Hewett *et al.*, 2004; Buchanan & Vardaxis, 2003). It has also been reported that females experience greater generalized joint laxity after the onset of puberty (Quatman *et al.*, 2008). There are some evidences that suggest that these two factors are linked to an increased risk of ACL injury among young female athletes (Kramer *et al.*, 2007; Hewett *et al.*, 2004). However, limited research has examined the effect of maturation and female hormonal *milieu* on the knee laxity and neuromuscular system, particularly in young female athletes.

As reviewed, anterior knee laxity is often proposed as a risk factor for ACL injury, with females exhibiting greater knee laxity than males (Shultz *et al.*, 2005; Rozzi *et al.*, 2001; Rosene & Fogarty, 1999), before and after exercise (Pollard *et al.*, 2006). Some authors have suggested that increased knee laxity may induce changes in muscle activation patterns under loading to compensate for reduced passive joint stability (active stabilization). The increased laxity may also account for a decreased joint sensitivity to movement which makes for delayed reflex responses and a harder active stabilization (Shultz *et al.*, 2004; Rozzi *et al.*, 1999). However, the implications of increased knee laxity and neuromuscular changes on the knee functional stability are not well understood.

As the musculature can have a protective affect on the knee (Solomonow & Krogsgaard, 2001) through the active knee stabilization mechanism, it is interesting to know the strength profile of our handball population, from an early age until the senior category.

In handball, the lower limb muscle strength (particularly fast and explosive strength) is generally accepted as essential to perform effective motor tasks while attacking or defending. Typical movements require significant amounts of muscle power, such as changes in direction, accelerations and jumps. In addition, these movements are also the most frequently implicated in the ACL injuries and therefore the main focus of the preventive programs. Some studies demonstrated that plyometric training and/or strength training has a potential preventive effect of ACL injuries in female athletes, because of knee neuromuscular control improvements (Grindstaff *et al.*, 2006; Hewett *et al.*, 1999).

Study purposes: to examine the influence of maturation, anthropometric characteristics, OC status, previous knee injuries and handball exposure in jump performance and knee laxity.

4.2 Methods

Experimental design

A cross-sectional experimental design was employed in the present study, which was conducted during the season 2006-07 (between January and June of 2007).The participating clubs and team selections were described in chapter 3.

Participants underwent a sequence of anthropometric, anterior knee laxity and jumps measurements as well as filling a questionnaire about injuries and menstrual cycle characteristics (described in chapter 3). Data from the screening questionnaire was used to identify players with previous knee injuries and to assess some vital and sports related information (*e.g.* age, years of practice, menarcheal status, OC status, years of handball practice, playing categories and playing positions). All measurements were done during a training session exclusively dedicated to this purpose. The knee laxity measurements were performed before the jump tests.

Prior to being studied the players were informed about the study goals and testing procedure, and all players (or parents, if the player was underage) gave informed written consent to participate in the screening tests.

The study was approved by the Scientific Council of the Sports Faculty.

4.2.1 Participants

Four hundred and ninety-six female handball players from 38 clubs, from the category of infants until seniors, were included in this study. Figure 4.1 shows the number of federated handball players by age category during the studied season, and the proportion of evaluated players.

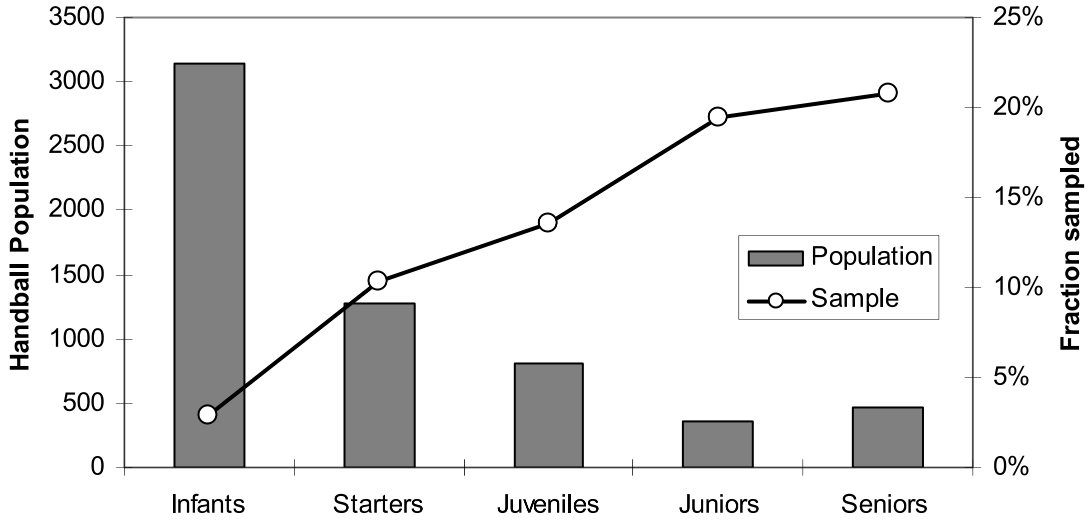


Figure 4.1 Number of federated handball players by age playing category in the season 2006-07 and the fraction sampled

Initial anthropometric measurements, including standing body height, body mass and percentage of fat body mass (by bioelectrical impedance) were always carried out by the same investigator. In the case of players at menses or those that were evaluated shortly after meals, the bioelectric data was discarded, as usual.

On average the players had 16.1 ± 4.4 years of age, were practicing handball for 5.5 ± 4.5 years, had a mean body mass of 60 ± 11 kg, a mean height of 162 ± 8 cm and a mean body fat content of 27.1 ± 5.7 % (table 4.1).

Table 4.1 Characteristics of sample categories (average ± standard deviation, where applicable)

Category	N	Age	Height (m)	Body mass (kg)	BMI[1] (kg/m^2)	% fat mass	# number of players affected by previous ACL/knee injuries
Infants	90	11.7	1.53 ± 0.09	48.4 ± 10.5	20.6 ± 3.5	25.4 ± 6.5	0/6
Starters	131	13.8	1.63 ± 0.06	59.4 ± 10.1	22.4 ± 3.6	27.1 ± 5.7	0/25
Juveniles	109	15.5	1.63 ± 0.06	61.9 ± 9.0	23.2 ± 2.7	27.6 ± 5.0	1/18
Juniors	70	17.6	1.65 ± 0.07	63.2 ± 10.2	23.1 ± 3.1	26.5 ± 6.5	6/18
Seniors	96	23.2	1.66 ± 0.07	65.0 ± 9.2	23.6 ± 2.7	28.8 ± 4.6	12/39
Total	496	16.1	1.62 ± 0.08	59.5 ± 11.2	22.7 ± 3.7	27.1 ± 5.7	19/106

Sixty-three players, who had not already reached menarche when evaluated, represent 11.7% of the samples. The majority of these players were from the infants category (53 subjects – 84%), and a few others from starters (9 subjects – 14%) or juveniles (1 subject). On average these sub-groups of players have been practicing handball for 2.5 ± 1.5 years.

Ninety-five players (19%) were OC users and had a mean age of 20.6 ± 3.6 years, with an average history of OC usage of 3 ± 3 years. All players reported to be using low-dose combination pills (containing synthetic estrogens and progestins). The large majority were using monophasic OCs (with 21 or 24-day active pills), with only three players using combiphasic OCs. The OC users were mostly from the senior's category (61%) (figure 4.2). On average this group of players had 10 ± 5 years of handball practice, contrasting with 5 ± 4 years of handball practice from the non OC users group.

Table 4.2 Hormonal status groups (average ± standard deviation, where applicable)

Category		N	Age	Height (m)	Body mass (kg)	BMI (kg/m^2)	% fat mass	# number of players affected by previous ACL/knee injuries
Without menarche		63	11.7	1.52 ± 0.08	44.9 ± 9.3	19.5 ± 3.3	23.0 ± 6.5	0/7
OCs status	Non users	332	15.7	1.63 ± 0.07	61.4 ± 11.5	23.4 ± 2.6	27.6 ± 5.4	8/66
	Users	95	20.6	1.65 ± 0.07	63.9 ± 9.0	23.1 ± 3.8	28.2 ± 5.1	10/33
	Unknown	6	15.8	1.63 ± 4.8	62.3 ± 5.6	23.3 ± 2.3	27.4 ± 3.6	1/2
Total		496	16.1	1.62 ± 0.08	59.7 ± 12	22.7 ± 3.7	27.1 ± 5.7	19/106

[1] Body Mass Index is defined as the ratio of body weight by the subject's height squared.

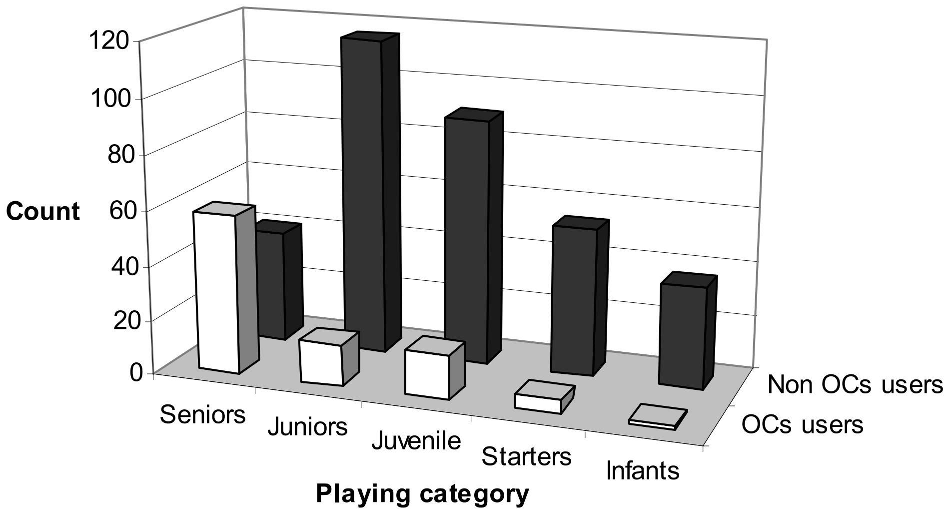


Figure 4.2 Number OC users *vs* non OC users per playing category

4.2.2 Vertical jumps

The vertical jump performance is a measure of the lower limb muscle strength (Rousanoglou *et al.*, 2008; Vanezis & Lees, 2005). The use of vertical jump tests to assess muscle power is a common field method. The tests were performed with the techniques of Bosco (1983b) that, although certainly not immune to imprecision and critics (Sands *et al.*, 2004) are a standard in the area. The most relevant force to the motor tasks specific to handball seems to be the reactive force (Bobbert *et al.*, 1996) and a simple field test for this is the counter-movement jump. Additionally we opted to measure also the performance in the squat jump, both as a means to accelerate the subjects' familiarity with the jumping procedures and to evaluate the populations' *explosive strength deficit* (Zatsiorsky, 1995).

A contact platform was used to assess the height jumped (h). This was calculated from flight time (t_f) and gravity acceleration (g) as $h= g{\cdot}t_f^2/8$ (m) (Bosco *et al.*, 1996).

The players were instructed to warm-up (5-10 min.) prior to the vertical jumps assessment, both by means of low-intensity running and by a few submaximal jumps. Before the experiments, the players completed a few submaximal vertical jumps in the mat under supervision, to become familiarized with the testing procedures.The testing session included a sequence of three attempts of vertical squat jumps (SJ) and three attempts of vertical counter movement jumps (CMJ). After the first set of jumps the players were given 1-2 min. of rest before the second set. The players performed at least three executions of each type of jumps, although if technical incorrections were visually detected or jump heights were inconsistent, the players were asked to rest for 10 minutes before additional jumps were performed. All players performed the tests in identical conditions, *i.e.* in their usual training place.

The players were instructed to jump with their hands on hips, to avoid the influence of the upper limbs on jump height; to keep their feet positioned under their body with weight equally distributed; to take-off and land in the same position on the mat. The SJ were completed starting from a standing position, with the knees flexed ≅ 90º (semisquatting position), and after that they were encouraged to jump as high as possible trying to avoid any knee or trunk counter-movement (Bosco *et al.*, 1983b). The CMJ were performed starting from an upright standing position on the mat, using a preparatory counter-movement to jump as high as possible. The players were instructed to squat down (eccentric action) until the starting position of the SJ (opus citæ), and then to extend the lower limbs, consisting in a fast flexion-extension movement. Three to five measurements were taken for each jump test.

In order to check the reproducibility of these measures, the median of each jump type was determined for each day and the trials of the first 9 days of the menstrual cycle were tested for homogeneity.

These jump tests do not depend on the operator, so these preliminary *quality control* calculations were not a measure of reliability but rather an assessment of the intra-individual daily variations in jumping performance (in force, indirectly). The results are presented in tables 4.3 and 4.4.

Table 4.3 Squat jump consistency tests

N	33
Averaged half-amplitude	23mm
Maximum amplitude	50mm
Mean difference	2mm
95% CI	[-7,11] mm
Bilateral t-Test: prob>|t|	0.68
Correlation	0.78
Power	0.22
Wilcoxon sign-rank: prob>|z|	0.48

Table 4.4 Counter Movement jump consistency tests

N	33
Averaged half-amplitude	22mm
Maximum amplitude	76mm
Mean difference	2mm
95% CI	[-9,13] mm
Bilateral t-Test: prob>|t|	0.69
Correlation	0.73
Power	0.20
Wilcoxon sign-rank: prob>|z|	0.43

Note: a proper power (like 0.8) would demand at least 180 subjects, which, although feasible, seemed a waste of resources on a marginal issue.

4.2.3 Anterior knee laxity

In this work, we defined knee laxity as the amount of anterior tibial displacement at standard loads (30lbf ≈ 133N), as measured by an arthrometer[2]. According to the

[2] KT-1000™ knee arthrometer (MEDmetric® Corp; San Diego, CA, USA)

manufacturer's instructions, the knee was positioned in a 30º flexion angle using a thigh support placed proximal to the popliteal fossa, as the subject lay supine. Heels were placed in a foot support while a velcro strap around the thighs ensured the alignment of the lower limb, to control external rotation. With the device properly positioned on the leg, aligned with the joint line reference and with the patella engaged in the femoral trochlea, the subjects were instructed to relax the lower limb muscles while the examiner executed manual pressure to stabilize the patella sensor (figure 4.3). After following the standard guidelines for device placement and with the initial testing reference position established, anterior directed force was applied in the handle by the examiner at 67N, 89N and 133N.

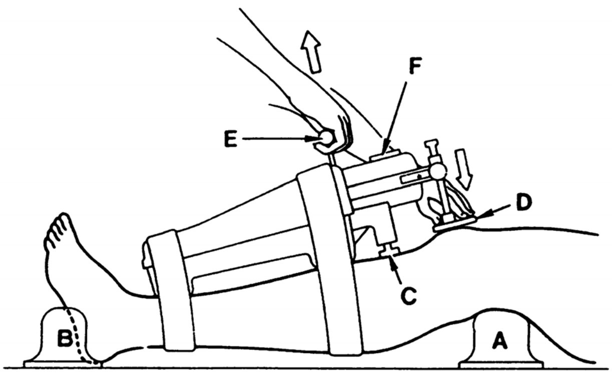


Figure 4.3 Schematic drawing of the KT-1000 device, from Daniel *et. al* (1985b): A, thigh support; B, foot rest; C, tibial sensor pad; D, patellar sensor pad; E, force handle; F, displacement dial.

In each knee, measurements were repeated a few times (up to 7, at each of the three loads), in a round robin fashion, until the researcher was subjectively convinced of the stability of the measures. The same examiner performed all measurements.

The median of the displacements obtain at the higher load (30lbf ≈ 133N) was then used as the measured laxity. The standard in physiology is to use the maximum of all the measures (*e.g.* isokinetic torques, anterior knee laxity). Here we opted to use a more robust measure (the median) on the basis that the subjects' lack of familiarity with the apparatus and techniques most certainly contaminated the first few days of measurements.

As the ligament laxity can vary by an order of magnitude between subjects and in each subject may vary along the menstrual cycle[3], a reliability test was done considering the set of repeated measurements performed on each subject during the first 9 days of the menstrual cycle, after its onset. This data was binned in a low category and a high category and a repeated measures t-test was performed on these two bins (table 4.5).

[3] It is possibly dependent on the hormonal environment. See chapter 6.

The observed variations have, possibly, a contribution from the natural daily subject's performance variation. Overall, no significant variation was observed, so the laxity measurements are, at least, self-consistent.

Table 4.5 Laxity measurements' reliability tests

N	35
Averaged half-amplitude	0.67mm
Maximum amplitude	2.25mm
Mean difference	0.11mm
95% CI	[-0.20,0.42] mm
Bilateral t-Test: prob>|t|	0.47
Correlation	0.88
Power	0.55
Wilcoxon sign-rank: prob>|z|	0.44

4.3 Results and discussion

4.3.1 Jump performance

The squat jump (SJ) and counter movement jump (CMJ) tests assessed with a chronometer and a mat, operating as a contact switch, have been described as reliable and valid field tests to estimate the explosive muscle power of the locomotor apparatus (Markovic *et al.*, 2004).

On average, there was an 8% (14 mm) increase on the CMJ result in comparison with the SJ. The two jump tests (figure 4.4) were highly correlated (r^2=0.75).

The SJ is essentially a measure of the concentric function whereas the CMJ assesses mainly the elastic energy usage during the stretch-shortening cycle (Kalapotharakos *et al.*, 2005; Bobbert *et al.*, 1996). The stretch-shortening cycle is the paradigm of force-enhancement mechanisms. During the *eccentric* phase, the muscle-tendon complexes are loaded in such a way that in the consecutive *concentric* phase they produce more work than they could if started from rest. Several mechanisms contribute to this. The elastic energy storage and reutilization, in tendons and other connective tissue (Bobbert *et al.*, 1996; Komi & Bosco, 1978; Cavagna *et al.*, 1965). The potentiation mechanisms, caused by the sarcomer asymmetries (Herzog *et al.*, 2003), the charge transfer mechanism (Hatze, 1990) and other short range stiffness mechanisms. The neural mechanisms, mainly the *muscle spindle – short reflex loop* mechanisms (Dietz *et al.*, 1979; Jones & Watt, 1971). Although these mechanisms are well known, there is still no agreement to the relative importance of each of these mechanisms to the final outcome.

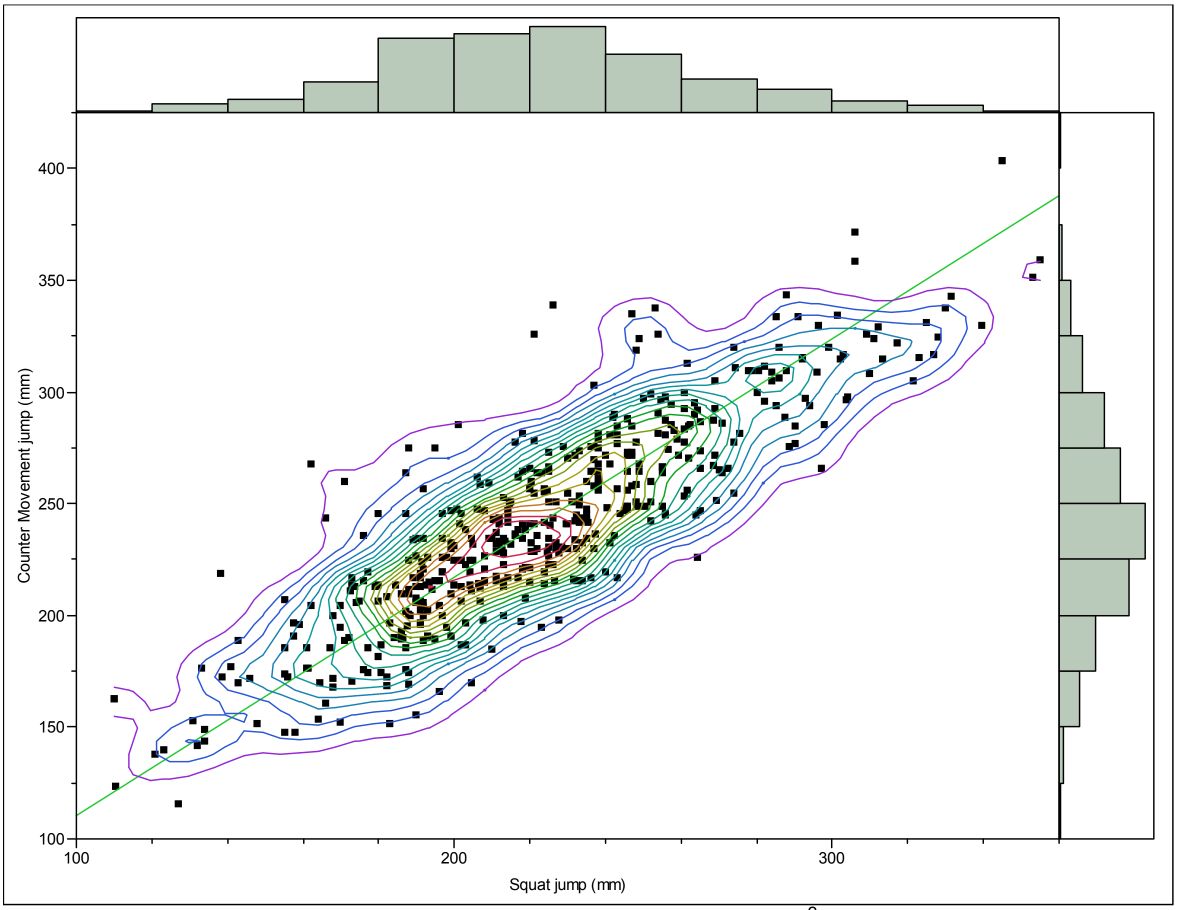


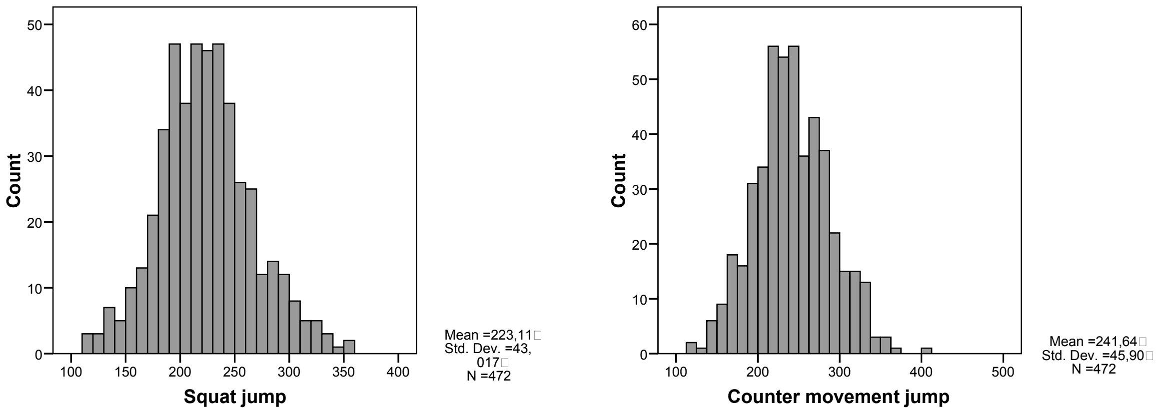
Figure 4.4 Orthogonal regression between CMJ and SJ ($p<0.0001$, $r^2>0.75$, CMJ=3.58+1.07*SJ)

The *explosive strength deficit* (ESD) was defined by Zatsiorsky (1995) as the relative difference between the maximum force demonstrated under unloaded (ideal) and loaded conditions (real) $ESD = \frac{F_{\max}^{ideal} - F_{\max}^{real}}{F_{\max}^{real}}$. This was born in a very specific context and its determination would demand the use of force plates, strain gauges, *etc.* In field applications, it is frequently approximated by tests like those we used, which can be trivially related with the *average* force produced (through the impulse). An adapted quantity, still vaguely related to the (inverse) original ESD concept, is the *eccentric utilization ratio* (EUR), defined prosaically as EUR=CMJ/SJ (McGuigan *et al.*, 2006). We will report a modified EUR, mEUR=EUR-1, usually as a percentage of increase.

Table 4.6 shows the average results for each jump test regarding the age category and playing category and also the mEUR figure. In figures 4.5 and 4.6 we present the distribution of jump height for SJ and CMJ.

Table 4.6 Jump height by playing categories

Age category	Playing category	N	SJ (mm) Average (mm)	SJ (mm) Standard deviation	CMJ (mm) Average (mm)	CMJ (mm) Standard deviation	mEUR
Infants		85	205.6	35.9	219.7	41.4	7%
	Infants	*68*	*198.6*	*34.0*	*212.6*	*40.9*	*7%*
	Infants/starters	*17*	*233.5*	*29.8*	*247.9*	*30.5*	*6%*
Starters		128	230.5	46.6	248.3	44.3	8%
	Starters	*83*	*220.1*	*45.6*	*238.1*	*43.8*	*8%*
	Starters/juveniles	*45*	*249.6*	*42.6*	*267.2*	*39.2*	*7%*
Juvenile		107	217.1	38.4	234.1	43.3	8%
	Juveniles	*63*	*209.5*	*40.3*	*227.3*	*46.7*	*8%*
	Juveniles/Juniors	*44*	*227.9*	*33.0*	*243.7*	*36.4*	*7%*
Juniors		60	219.6	42.7	242.5	40.6	10%
	Juniors	*34*	*207.8*	*37.4*	*232.3*	*34.8*	*12%*
	Juniors/Seniors	*26*	*235.0*	*45.0*	*255.9*	*44.4*	*9%*
Seniors		92	238.4	42.5	260.8	48.6	9%
Total		472	223.1	43.0	241.6	45.9	8%



Figures 4.5 & 4.6 Jump height results for SJ and CMJ

An ANOVA (p<0.0005) and a *post-hoc* Tukey HSD test[4] revealed, for the SJ, the following homogeneous groups (in increasing performance):

- infants, juveniles and juniors (p>0.16)
- juvenile, juniors and starters (p>0.20)
- starters and seniors (p>0.70)

For the CMJ, the homogeneity groups were (in increasing performance):

- infants and juveniles (p>0.19)
- juveniles, juniors and starters (p>0.19)
- starters and juniors (p>0.32)

Even though jump height tests are quite popular among handball coaches, we could only find a couple of published studies on this subject. In a small sample of young Spanish

[4] A Dunnett T3 test was also performed for dealing with some small homoscedasticy effects. The results were exactly the same for the CMJ and almost the same for the SJ (an additional difference was detected, between Juniors and Infants) – under T3 the SJ and CMJ grouping is identical.

female handball players (N=24; age=14.2 ± 0.4 years) on average the results measured with a force plate were 20.2 ± 0.01 cm for the SJ and 22.0 ± 0.01 cm for the CMJ, representing an increase of 8.2% (Vicente-Rodriguez *et al.*, 2004). Their results are quite similar to ours (figure 4.7), just slightly lower in the categories of starters and juveniles, in both jumps.

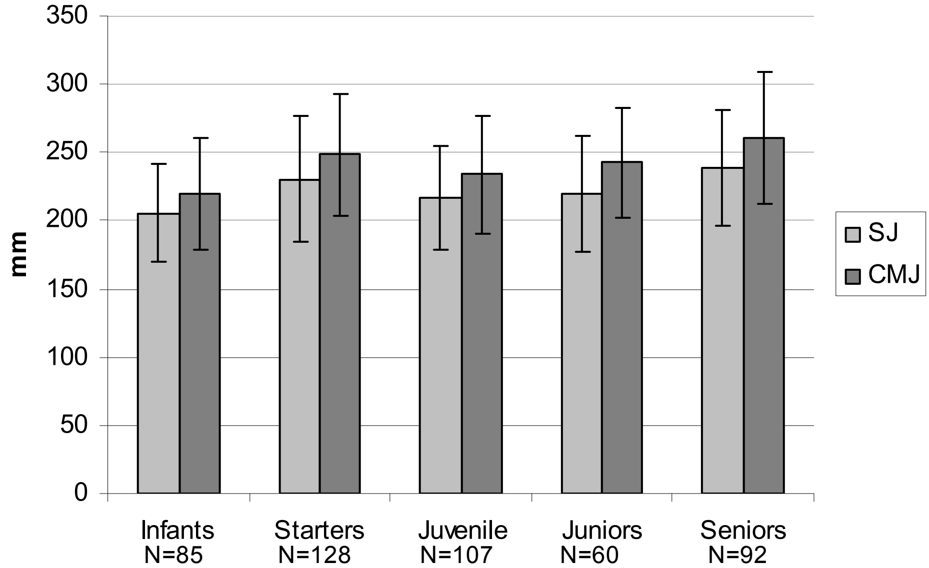


Figure 4.7 Jump height results per age category

However we felt that an age distribution did not properly reflect the personal development stages (athletically, at least) and we opted for an additional analysis, based on the playing categories. The same statistical methods revealed differences ($p<0.0005$) between groups (figure 4.8). The *post-hoc* tests revealed four homogeneous groups relative to SJ performance (table 4.7). Two extreme groups, performance wise, are easily identifiable: all those playing exclusively in their age category except seniors (lowest performance, Tukey HSD, $p>0.3$), and seniors together with those playing in two categories simultaneously (highest performance, Tukey HSD, $p>0.3$).

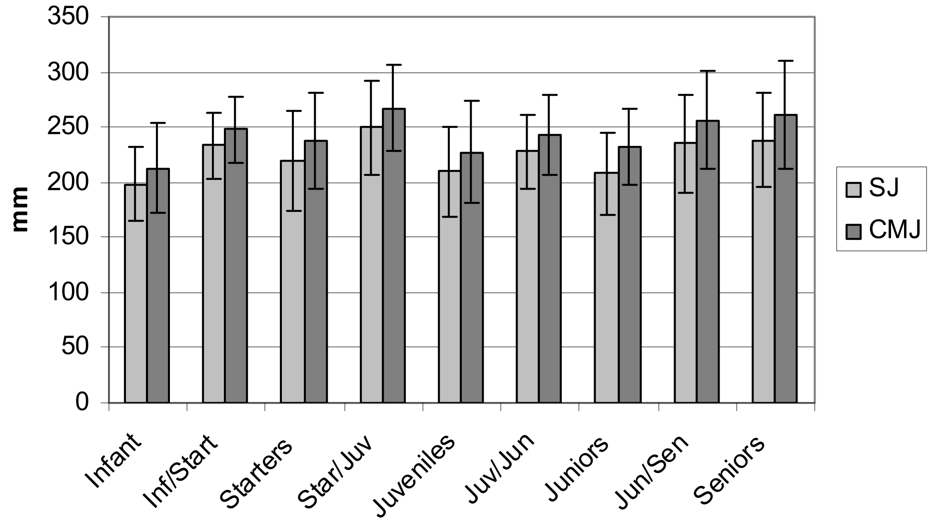


Figure 4.8 Jump height results per playing categories

Table 4.7 Homogeneous groups for SJ under HSD *post hoc* test

		Subset for alpha = .05			
Playing category	N	1	2	3	4
Infants	68	198			
Juniors	34	207	207		
Juveniles	63	209	209		
starters	83	220	220	220	
Juveniles/Juniors	44		227	227	227
Infants/starters	17		233	233	233
Juniors/Seniors	26		235	235	235
Seniors	92			238	238
Starters/juveniles	45				249
Sig.		0.293	0.065	0.516	0.278

For the counter movement jump, the same methods reveal almost the same grouping, with only a significant difference: starters are now included in all groups, including the one with highest performance (table 4.8).

Table 4.8 Homogeneous groups for CMJ under HSD *post hoc* test

Playing category	N	Subset for alpha = .05			
		1	2	3	4
Infants	68	212			
Juveniles	63	227	227		
Juniors	34	232	232	232	
Starters	83	238	238	238	238
Juveniles/Juniors	44		243	243	243
Infants/starters	17		247	247	247
Juniors/Seniors	26		255	255	255
Seniors	92			260	260
Starters/juveniles	45				267
Sig.		0.165	0.072	0.072	0.062

The jumping performance seems to mimic the playing performance. All those playing in two categories are usually the best players. These tests have also revealed that they are the best jumpers, maybe the strongest athletes. The reason for this is not clear. Are they fitter because of the higher loads or are they the best players because (also) of better conditional capacities?

An analysis on the body weight (figure 4.9) does not reveal anything unusual. On the body mass index (figure 4.10) we can see a (statistically insignificant, $p>0.55$) curiosity: the best players (playing in two categories) have an average BMI lower than the players of the same age playing only at their age category (excluding infants, where 59% have not reached menarche).

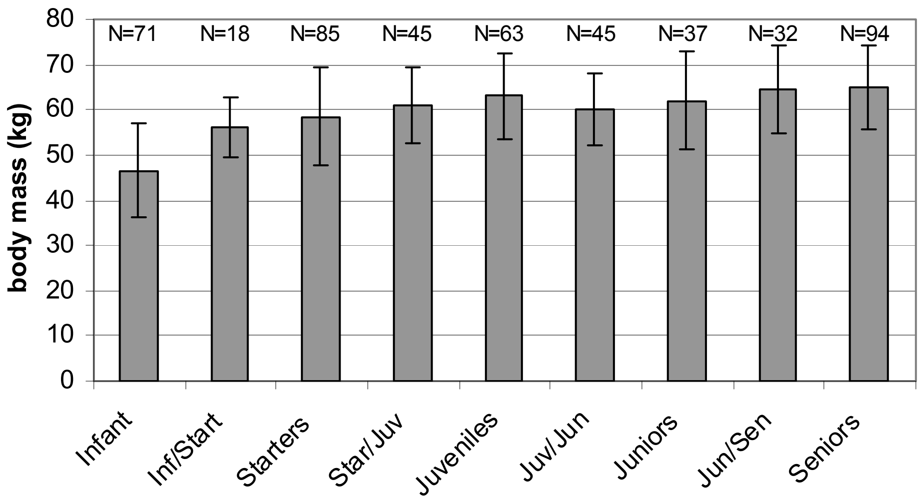


Figure 4.9 Body mass per playing category

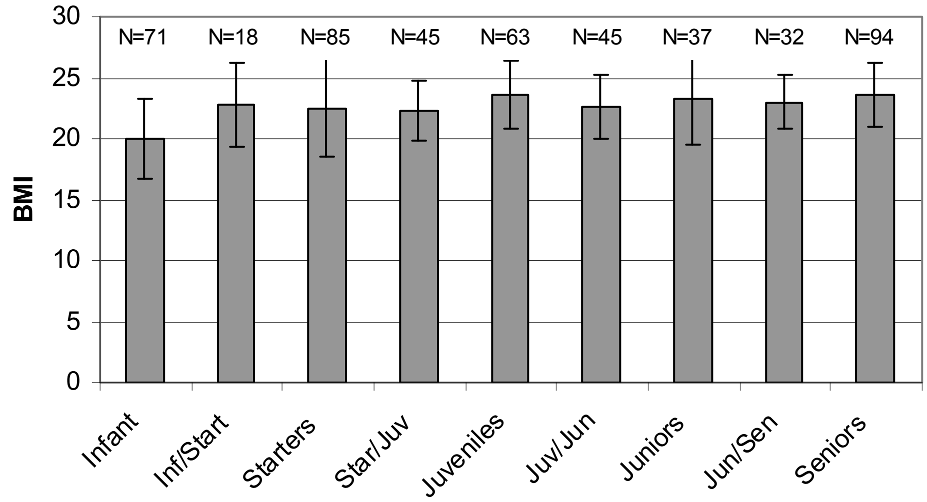


Figure 4.10 BMI per playing category

Is there an overweight problem among our players? Based on the growth tables published by the CDC (Kuczmarski *et al.*, 2000), we built table 4.9. One must not forget that these tables are for the general north-american population (non-athletes), so any conclusions based on them should be taken cautiously. We suspect of a possible overweight problem among Portuguese young female handball players, which should be further investigated. However, we did not pursue this lead nor did we try any comparisons with the Portuguese population at large. We were just concerned with the injury risks of handball practice.

Table 4.9 BMI distribution and female growth indicators

Category	BMI	Age	Percentil
Infant	20.04	11.7	75.6
Infants/starters	22.79	11.7	90.4
Starters	22.53	13.8	81.8
Starters/juveniles	22.29	13.8	80.3
Juveniles	23.56	15.5	81.0
Juveniles/Juniors	22.60	15.5	74.8
Juniors	23.23	17.6	71.9
Juniors/Seniors	22.98	17.6	69.8
Seniors	23.63	23.2	69.1

A complementary approach is the study of the jump performance by playing position. Significant differences were found for the CMJ among the different field positions (ANOVA, $p < 0.005$). Figure 4.11 presents the SJ and CMJ results by playing position. Whenever the player reported to be currently playing in more than one attacking position, the answers were re-grouped by field sides. For the CMJ, the pair wise comparisons revealed statistical differences between the pivots and those playing on the left side of the attack (*left wings*, *left back*, and *left side;* p<0.05, Tukey HSD and Dunnett T3). These differences are probably related to the body mass, as the pivots are the weightiest players (figure 4.12), with specific training processes and game functions.

Based on anecdotal evidences, in Portugal, the left back players and left wings players are usually the best jumpers, although it is difficult to know if the training process induces those differences or if it is just the result of sports selection.

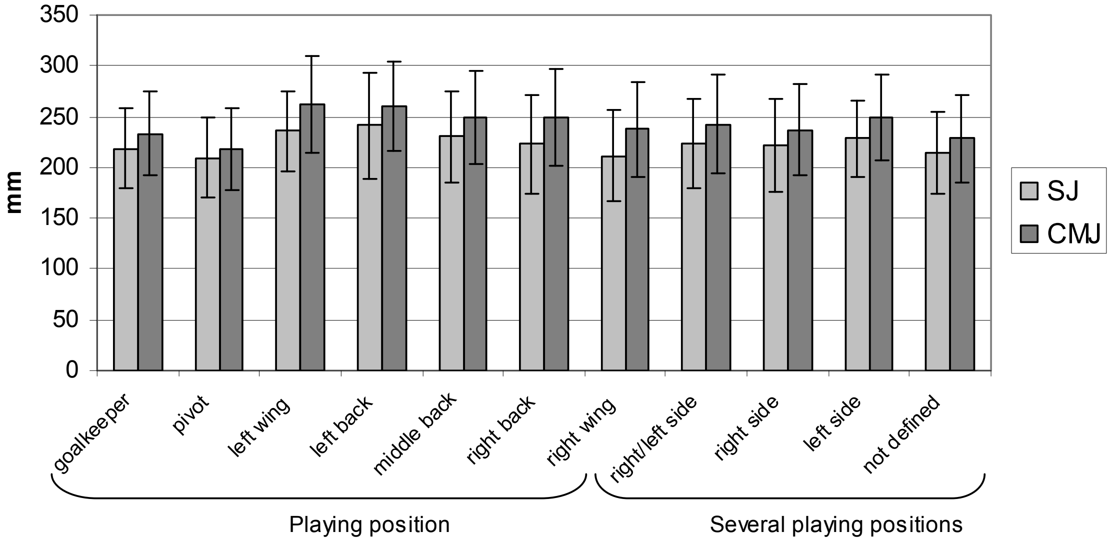


Figure 4.11 Jump height results per playing position

Significant differences were found between the playing positions considered (ANOVA, p<0.005). As expected *wings* and *middle back players* presented a lower body mass than other playing positions such as *pivots* and *goalkeepers* (Tukey HSD *post-hoc* test, p<0.006). By a marginal amount, no statistical differences were found between *goalkeepers*, *pivots*, *left*/*right back* and *left side players* (Tukey HSD *post-hoc* test, p>0.07). Interestingly, the body mass and BMI have been suggested as indicators of ACL injury risk in females (Hewett *et al.*, 2006; Uhorchak *et al.*, 2003). Still, the heaviest Portuguese handball players are not the ones most frequently injured (chapter 3).

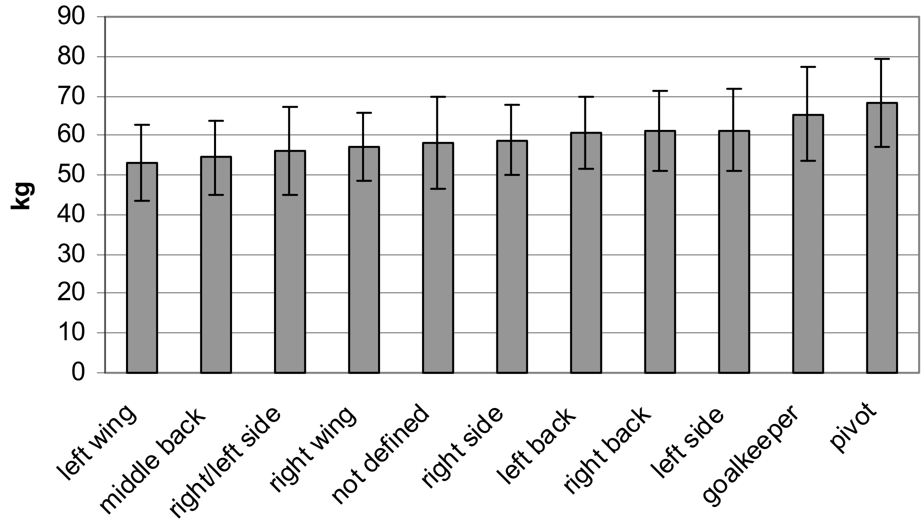


Figure 4.12 Average body mass per playing position

The menarche status is a well-established index of female physical maturation, which occurs during puberty, and triggers the beginning of the female hormonal secretion cycle. The average results for both jump tests were higher in players already menstruating[5]: higher 9.6 mm in the SJ ($p>0.1$) and higher 16.5 mm in the CMJ ($p<0.008$) (figure 4.13).

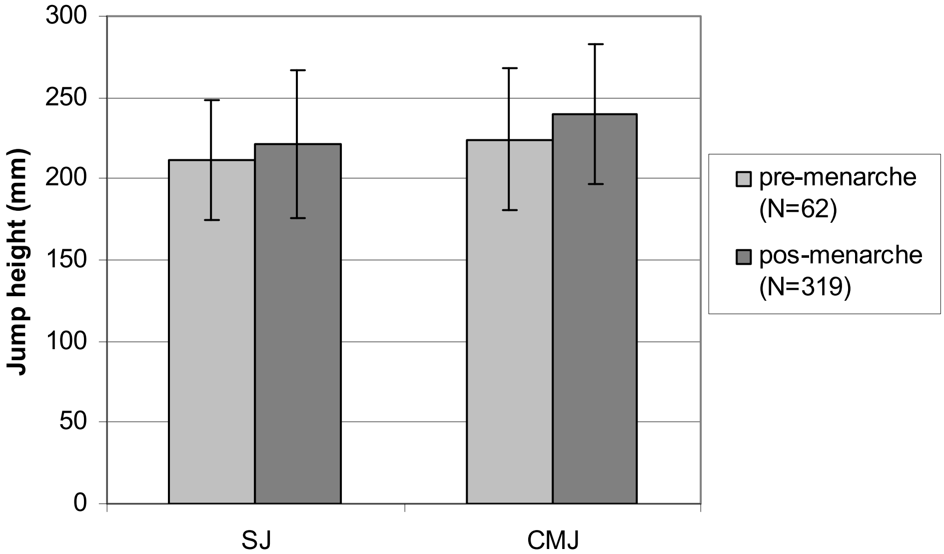


Figure 4.13 Jump height results by menarche status

Obviously, an age effect can't be ignored. Figure 4.14 is a plot of the jump performance over age. What we can see is that even if there are changes coinciding specifically with

[5] For this analysis we excluded all senior players (see first paragraph of page 176)

menarche (average 12 years) they are no bigger than other changes occurring along the full range of ages. There are strong reasons to suspect of a significant performance change during puberty. It is just that there is not a dramatic change in this indicator in our population.

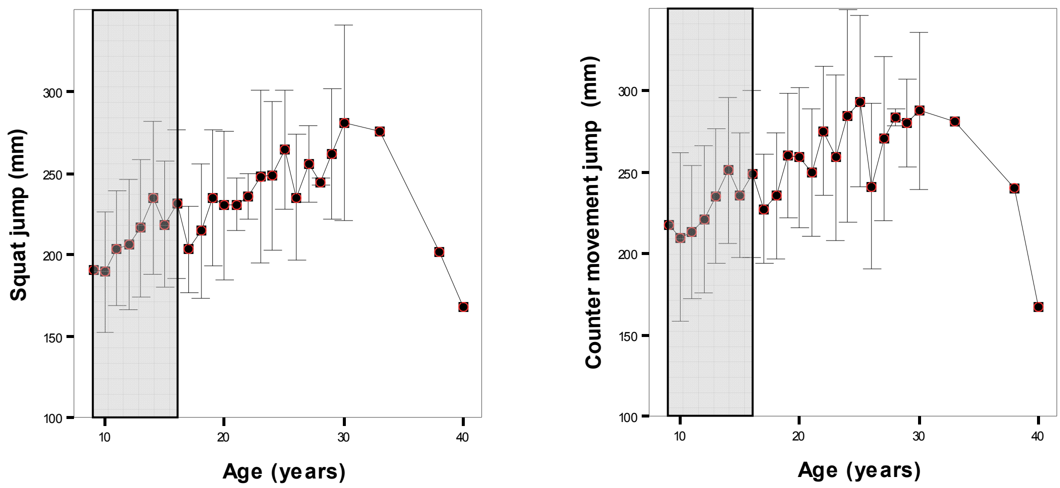


Figure 4.14 Age distribution of jump performance (with window of menarche occurrence)

There are similar studies on these subjects, with dissimilar conclusions. Quatman *et.al.* (2006) found that male athletes showed increased vertical jump height with maturation along with reduced landing forces, while no similar adaptations were found in female athletes. However, both sexes showed a reduction in lower limb loading rates with maturation. Similarly, other authors have reported sex differences in jump height during maturation, with an increased jump height performance in boys and unchanged jump height performance in girls (Temfemo *et al.*, 2008). These authors concluded that the observed differences were due to a larger increase in leg length and lean body mass in boys than in girls, particularly from 14 years onwards.

Analysing the dependence on OC usage, we observed an increase of ≅8% in the CMJ height in comparison with the SJ irrespective of OC usage (figure 4.15). The mean differences between the OC users and non-OC users (2.0 mm in the SJ and 2.5 mm in the CMJ) were not statistically significant ($p>0.7$). However, OC users (μ = 64 ± 9 kg) were heavier than the non-OC users (μ = 61 ± 10 kg) ($p=0.01$). No significant differences were found in BMI ($p>0.1$) or % fat mass ($p>0.3$) between the OCs and non-OCs groups.

Several questions have been raised about OCs-induced weight gain, metabolic changes and their potential negative effect on physical performance. Rickenlund *et al.* (2004) found significant increases in weight and body fat content only in oligo-/amenorrheic by contrast to eumenorrheic athletes after 10 months of OCs treatment, with an insignificant impact on several physical performance indicators (including endurance and strength tests).

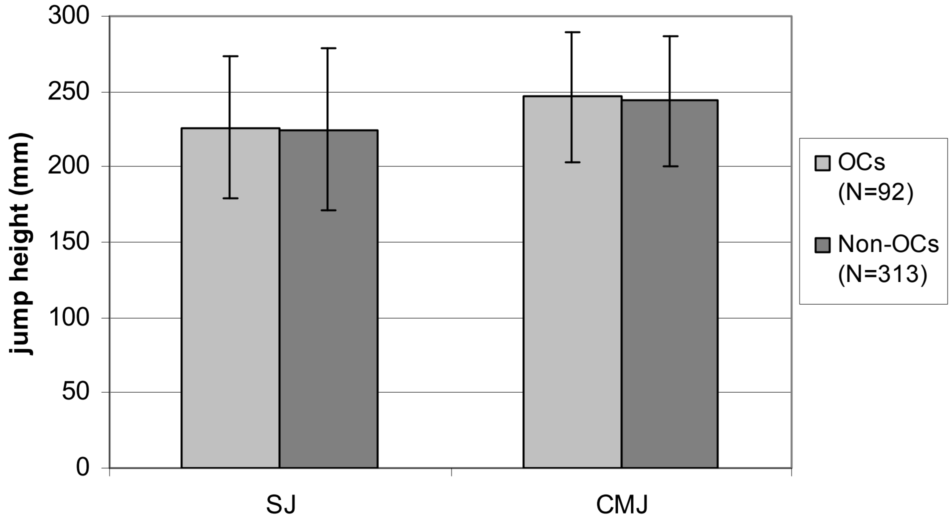


Figure 4.15 Jump height results by OC status

Comparing the mean jump heights between players with previous knee injuries and those without knee injuries, respecting laterality (figure 4.16), there were significant differences in the SJ between players with healthy knees (221mm) and players with previous injuries in the non-dominant knee (243mm) – ANOVA: $p<0.04$; HSD: $p<0.022$. These differences are not statistically significant in the case of the CMJ (marginally, $p>0.057$).

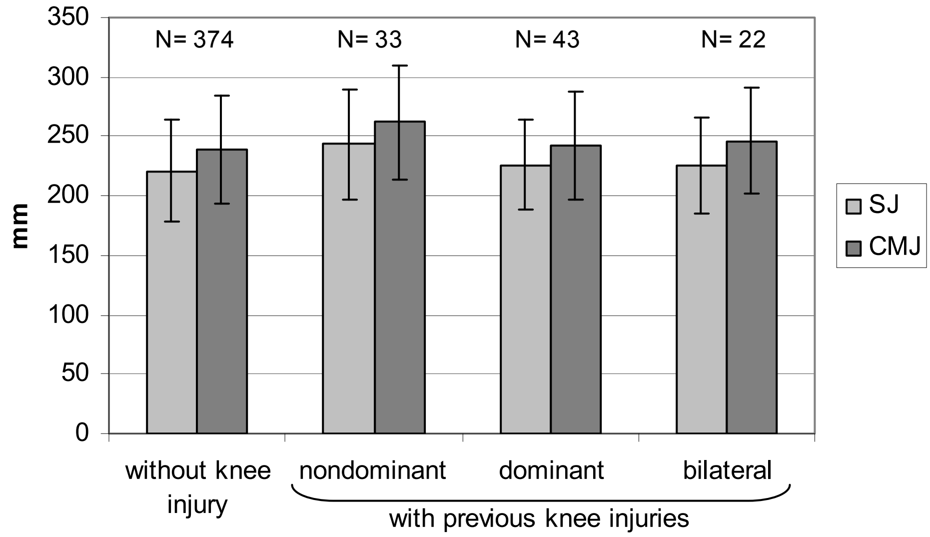


Figure 4.16 Jump height results by previous knee injury

Figure 4.17 presents the jump values by different injury categories. We excluded those with less than 6 players. No differences could be found for both jump types (ANOVA, p> 0.09).

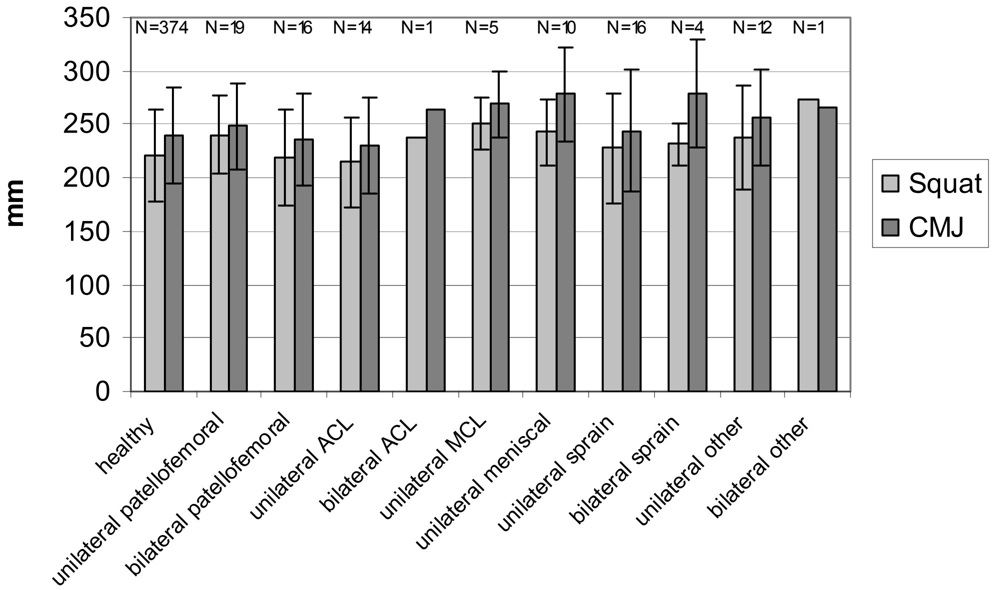


Figure 4.17 Jump height results by previous knee injury type

A stepwise maximum-likelihood regression analysis was performed on the dependent variable *counter movement jump* and on the independent variables *age, years of practice, playing category, body fat content, duration of OC usage, BMI, gynaecological age, field position* and *weight* (forward only, p_{in}=0.25). With these rules, the variables *body fat content, duration of OC usage, gynaecological age* and *weight* were not included in the model. A mixed model (p_{in}=p_{out}=0.25) would exclude *years of practice* and a backward model (p_{out}=0.25) would substitute *age* by *gynaecological age*. As *gynaecological age* only applies to players who had reached menarche, we opted for the use of the *age* variable. *Years of practice* is the first variable to be included in the forward model and the last to be removed in the mixed model. It is a borderline case and we opted for its inclusion. Together, this is the forward model, which is also the one with a higher estimated regression coefficient (R=0.64). Akaike's criteria[6] was technically even among all models while Mallow's[7] C_p suggests something like 23 variables.

[6] Akaike's information criteria (AIC) is a model selection criteria based on a entropy measure. One should opt for the model with lowest AIC value (Akaike, 1974).
[7] Mallow's C_p is a parameter selection aid based on the rescaling of the sum of squared errors. Each new parameter addition changes C_p. When C_p is similar to the number of parameters (p), the model has no gain in additional inclusions (Mallows, 1973).

The results of such a model are presented in tables 4.10 and 4.11 and figure 4.18. As expected, not much predictive power (R^2=18%).

Table 4.10 Maximum likelihood regression model on Counter movement jump

Term	Estimate	Std Error	Prob>|t|	AdjPower.05
Intercept	281.33956	19.31717	<0.0001	1.0000
Age	3.018802	0.989219	0.0024	0.8185
Years of practice	0.9871866	0.821616	0.2302	0.1012
Playing category (1&3&4&5&6&7&8-9)	1.7846369	4.291552	0.6777	0.0500
BMI	-4.272832	0.649942	<0.0001	1.0000
Field position (1&10&8&9&4-0&6&3&7&5&2)	-1.149051	2.090953	0.5829	0.0500
Field position (0&6-3&7&5&2)	-6.344211	2.883961	0.0283	0.4959
Field position (0-6)	-2.490408	4.614329	0.5897	0.0500
RSquare	0.180571			
ANOVA: Prob > F	<0.0001			
Observations	449			

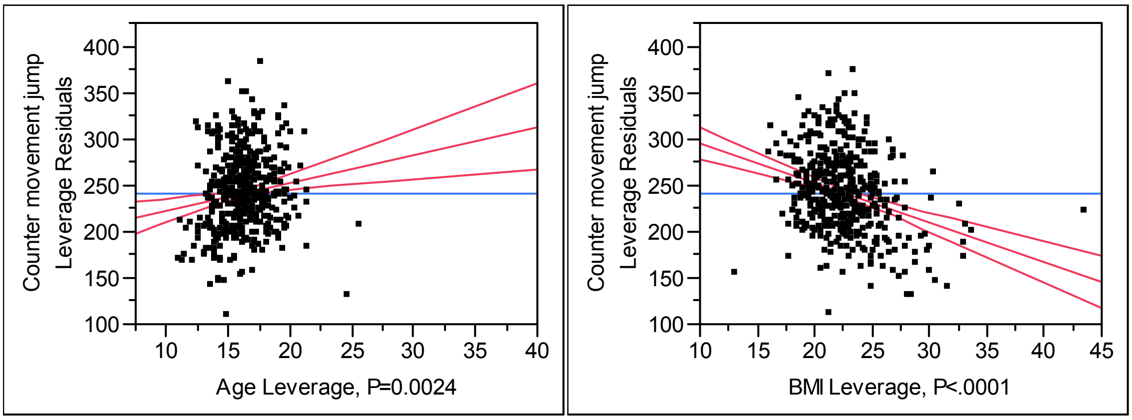


Figure 4.18 Leverage plots for *counter movement jump* on *age* and *BMI*

Table 4.11 Playing position effect on CMJ – linear fitting model

Playing position	Estimate
Pivot	-7.68557
Goalkeeper	-1.14905
Left wing	7.493262
Left back	7.493262
Middle back	-1.14905
Right back	7.493262
Right wing	-2.70475
Right/left side	7.493262
Right side	-1.14905
Left side	-1.14905
Not defined	-1.14905

The same stepwise regression techniques were used to analyse the squat jump. The model with more information per parameter is the forward model that include only the *age,* *playing category* and *BMI*. Again, Mallow's C_p is not applicable. The estimated regression coefficient is R=0.63. The resulting model parameters, after fitting to our data, are summarized in table 4.12 and figure 4.19. Again, not much predictive power (R^2=15%).

Table 4.12 Maximum likelihood regression model on Squat jump

Term	Estimate	Std Error	t Ratio	Prob>|t|	AdjPower.05
Intercept	271.59676	16.9128	16.06	<0.0001	1.0000
Age	2.8956329	0.699916	4.14	<0.0001	0.9791
Playing category (1&3&4&5&6&7-8&9)	-1.076951	3.490317	-0.31	0.7578	0.0500
BMI	-4.226533	0.606571	-6.97	<0.0001	1.0000
RSquare	0.148244				
ANOVA: Prob > F	<0.0001				
Observations	449				

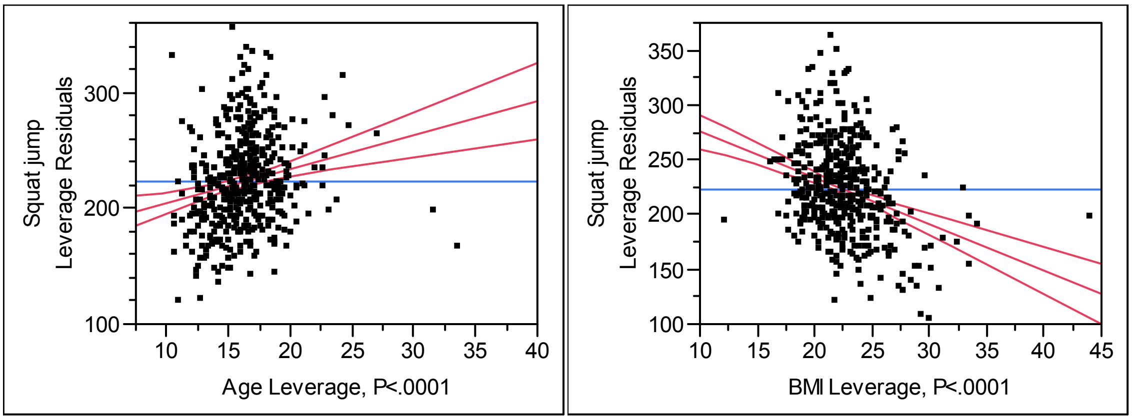


Figure 4.19 Leverage plots for *squat jump* on *age* and *BMI*

Globally, on the subject of jumps, we could summarize this study in just a few words:

- Age and BMI are the most reliable and general predictors of jump performance
- There is a positive dependence on *age* and a negative one on *BMI*
- These models have unexpectedly low predicting power
- The average BMI values need further investigation but seem high
- The absolute jump results and the ratio of eccentric energy reutilization seems low.

These seem to describe a population where the conditional aspects are not of great importance, which could concur to an added risk of injury. A comparison with a sample of 240 male and 240 female French students (Temfemo *et al.*, 2008) is interesting (figures 4.20, 4.21 and 4.22). The mEUR differences are dramatic but disappear with aging, the jump capacity starts the same but ends radically different (although the French girls are non athletes) and the anthropometric indicators reveal a fatter but not shorter Portuguese population.

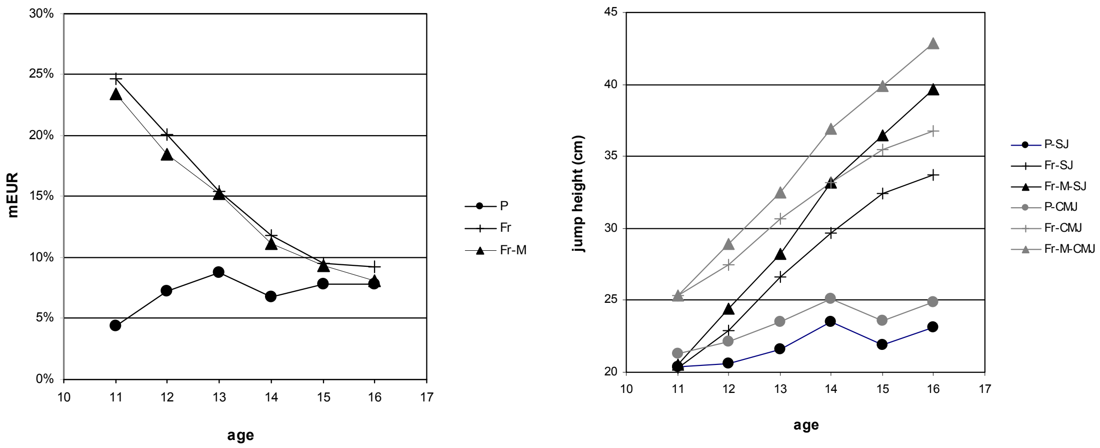


Figure 4.20 & 4.21 EUR (left) and jump performance (right) age distribution for our sample (P) and a sample of French non-athlete girls (Fr) and boys (Fr-M)

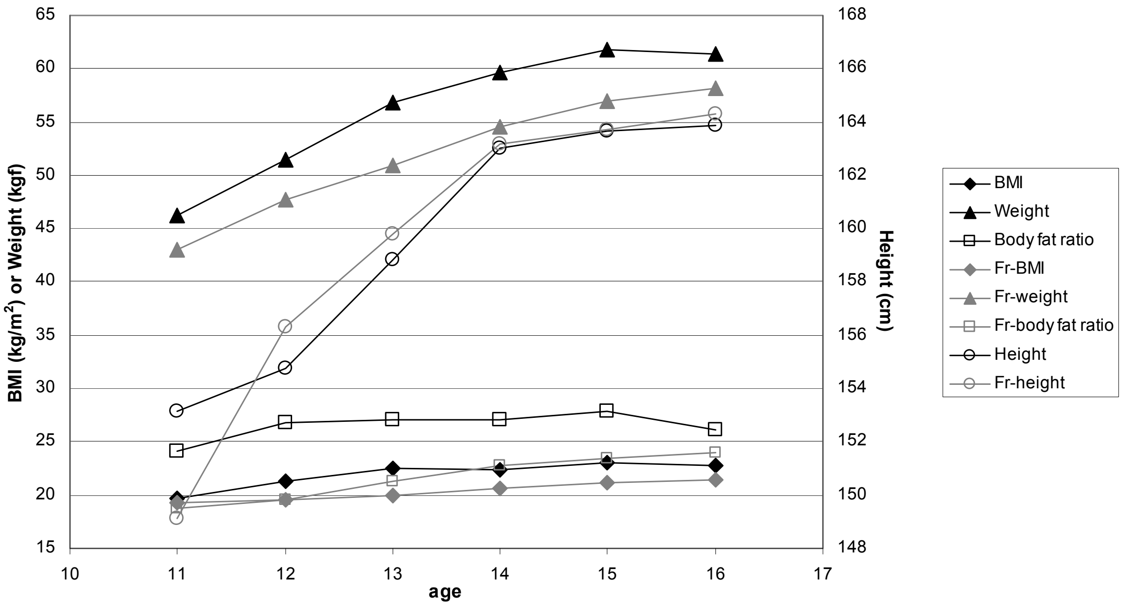


Figure 4.22 Anthropometric data comparison: Portuguese female handball players *vs.* French school girls (Fr)

Methodological considerations on force measurements

There is a trivial relation between maximum height (h) and take-off velocity (v_0) for a particle under a gravitational field

$$\begin{cases} h = v_0 \cdot t_{up} - \frac{1}{2} g \cdot t_{up}^2 \\ v_0 - g \cdot t_{up} = 0 \end{cases} \Rightarrow h = \frac{v_0^2}{2g}$$

where t_{up} is the time from take-off till maximum height and g is the gravitational acceleration. There is also a (less-trivial) relation between take-off velocity and the force exerted on the particle (*F*)

$$v_0 = \int_{pushoff} \frac{F}{m} \cdot dt$$

This is the impulse relation where *m* is the object's mass and the integral is evaluated during the whole pushoff duration. Geometrically the integral is the area under the force curve and very different curves can enclose the same area.

Mathematically this makes the maximum height a function[8] of the impulse force, but a non-invertible one *i.e.* knowing the force gives us the jumped height but knowing the height tells us little about the force. See figure 4.23 as an extreme synthetic example and figure 4.24 as an example of the forces we are pursuing.

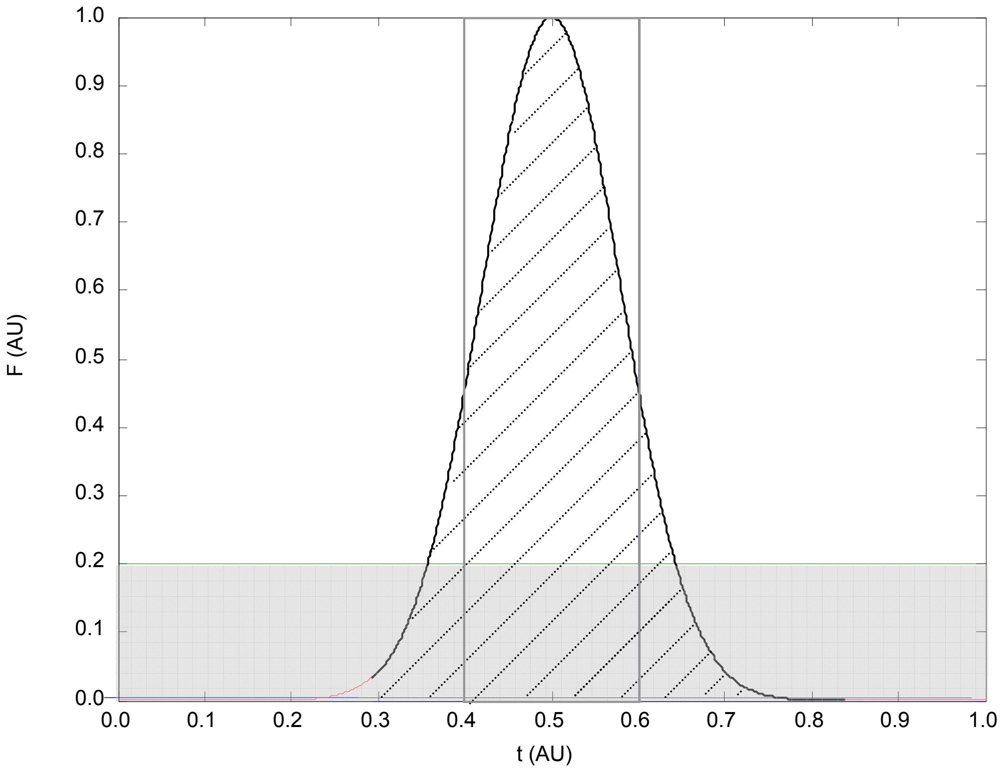


Figure 4.23 Three arbitrary functions (tall rectangle, large rectangle, gaussian) with the same integral (0.2 AU)

[8] Or a functional

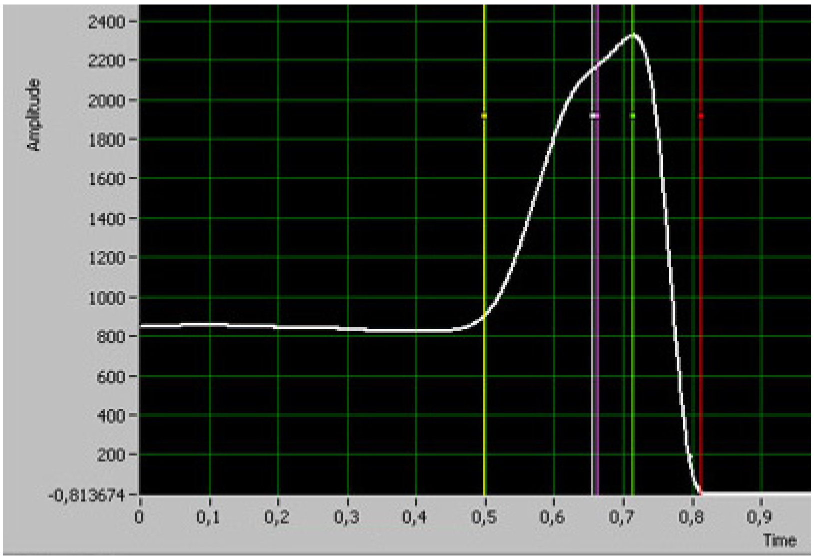


Figure 4.24 Squat jump ground reaction force (http://www.wise-coach.com)

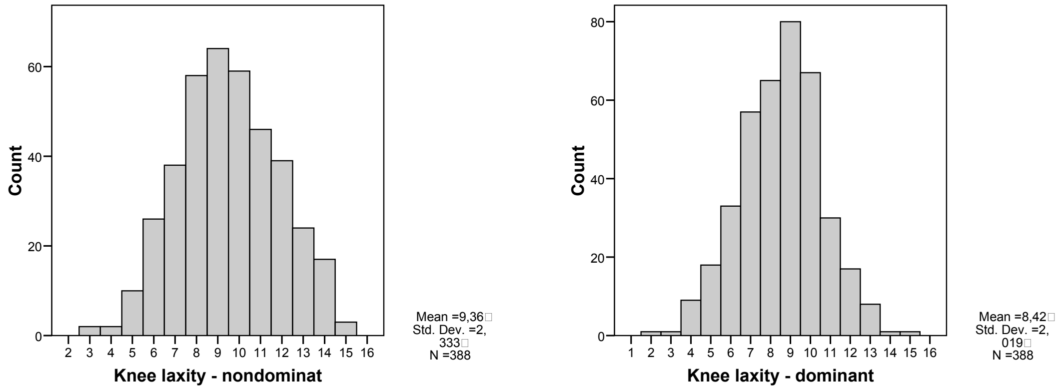
This theory is exactly applicable to the centre of mass of any particle system such as a human body. But the determination of the human centre of mass is tedious and it changes constantly when the limbs (and head, body fluids, *etc.*) change their relative positions. The Bosco method uses this model applied to the feet(!), with a set of rules trying to maintain a more or less constant distance from the feet to the body's centre of mass. The method is very simple, moderately accurate (Bosco *et al.*, 1983a) and widely used in the field, but probably not accurate enough for every application. An average error of about 10% is frequently quoted (Leard *et al.*, 2007).

4.3.2 Anterior knee laxity

Three hundred and eighty-eight players without previous knee pathologies were evaluated. The non dominant knee (μ= 9.4 ± 2mm) presented higher values of laxity than the dominant one[9] (μ= 8.4 ± 2mm) ($p<0.001$), see figures 4.25 and 4.26. Ninety percent showed an absolute difference of 3 mm or less between the dominant and non-dominant knees, with a minimum difference of -5 mm and a maximum of 4 mm. Significant side-to-side knee laxity differences were also found in others studies (Sernert *et al.*, 2007; Sernert *et al.*, 2004; Rosene & Fogarty, 1999; Riederman *et al.*, 1991), while others did not find such difference (Skinner *et al.*, 1986). Several studies presented 2 mm (measured at 20lbf) as a common side-to-side knee laxity differences in healthy knees (Daniel *et al.*,

[9] We assumed a side dominance based on hand dominance. Even if this is not the case, we must notice that when a player is right-handed the standing leg for most of the handball technical abilities is the left one (to shoot, to pass, to plant-and-cut to the dominant side). In this sample, 91% were right-handed, 7% were left-handed and the remaining were ambidextrous (1%).

1985a; Daniel *et al.*, 1985b). Our results were obtained in a younger population and at 30lbf which makes them in reasonable agreement with the published ones.



4.25 & 4.26 Knee laxity distribution (non-dominant & dominant side)

The dominant and non-dominant knee laxities were significantly correlated (r^2=0.73; p=<0.0001). There is, however, a large dispersion around the trend line (figure 4.27).

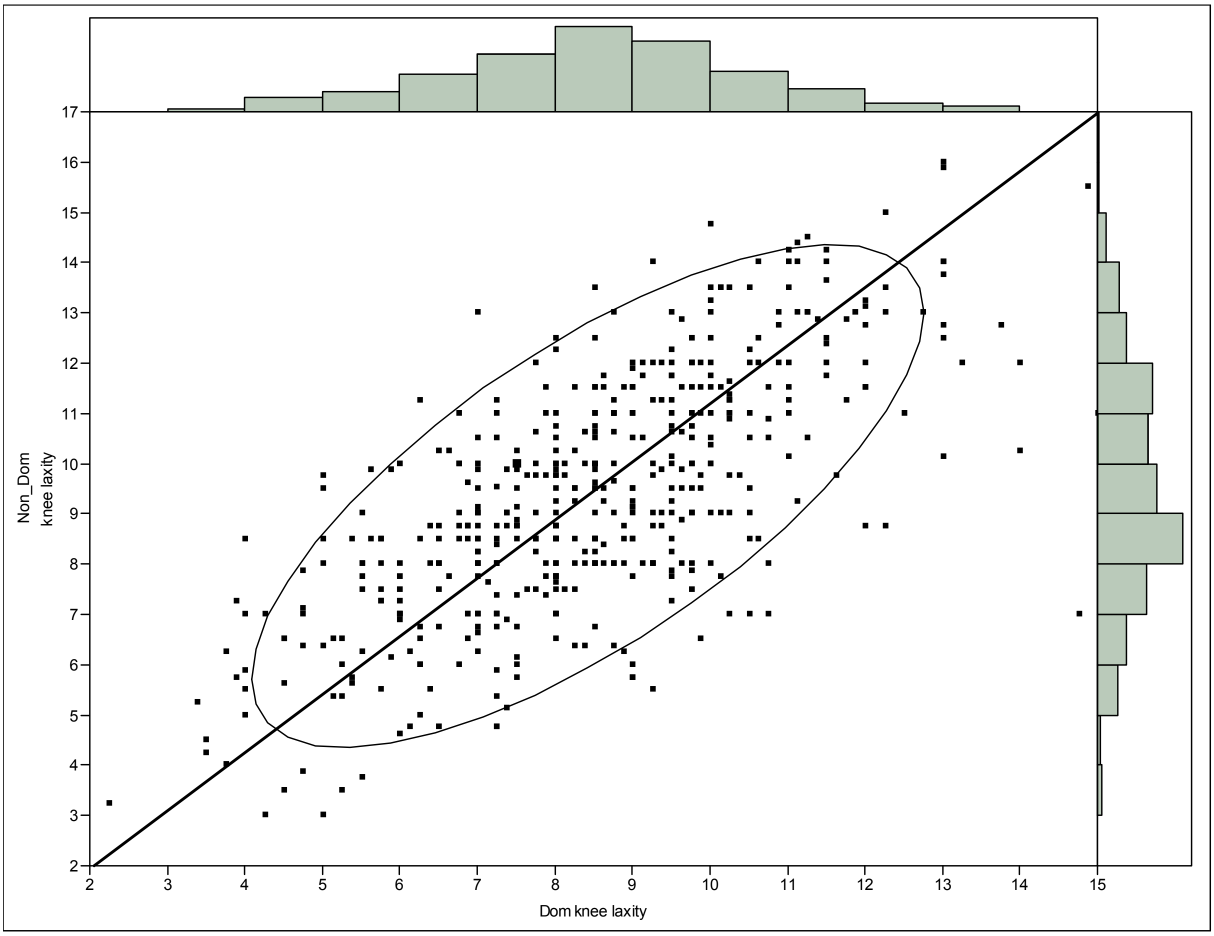


Figure 4.27 Orthogonal regression between *dominant* and *non-dominant* knee laxity (p<0.0001, r^2>0.73, Non_Dom_knee_laxity=-0.37+1.16*Dom_knee_laxity). The *best fit ellipse* encloses 90% of the trials.

The knee laxity tends to decrease as the *age category* increases (table 4.13 and figure 4.28). The different age categories have statistically different knee laxities, considering only healthy knees (ANOVA, p< 0.008). The *Tukey HSD post hoc test* reveals differences between seniors and infants (p<0.004) in the dominant knee and between the infants and seniors, juniors and juveniles (p<0.05) in the non-dominant knee. The side-to-side knee laxity differences were constant along all categories (p>0.09).

Table 4.13 Knee laxity (mm) by playing categories (average ± standard deviation)

Knee laxity	Infants	Starters	Juvenile	Juniors	Seniors
Dominant knee	9.0 ± 2.2	8.5 ± 1.9	8.4 ± 1.9	8.1 ± 2.1	7.8 ± 1.9
Nondominant knee	10.3 ± 2.3	9.5 ± 2.3	9.3 ± 2.1	9 ± 2.4	8.3 ± 2.1
Side-to-side differences	1.3	1.0	0.9	0.8	0.5

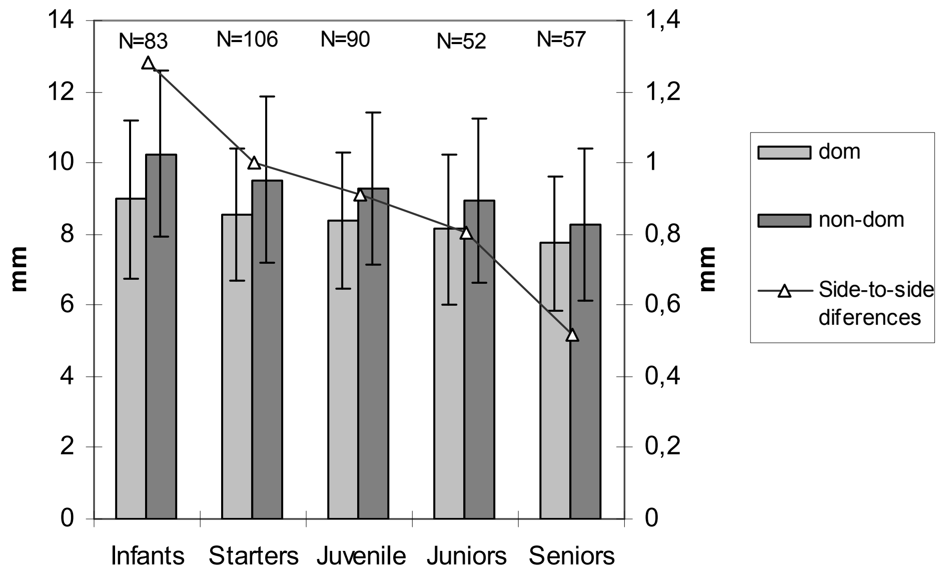


Figure 4.28 Knee laxity evolution by category

Beyond the usual binning, an analysis on the almost continuous variable *age* reveals that the knee laxity seems to decrease with increasing age (p<0.001), but the estimated regression coefficient is very low for both knees (figures 4.29 & 4.30). Some other studies found a decreased anterior knee laxity with increasing age in children and adolescents from both sexes (Hinton *et al.*, 2008; Flynn *et al.*, 2000).

About the sex-dependence, Hilton *et al*. (2008) found higher knee laxity, at both 20 lbf and 30lbf, in girls than in matched boys, contrary to Flynn *et al.* (2000) who didn’t found any knee laxity sex differences during childhood and puberty.

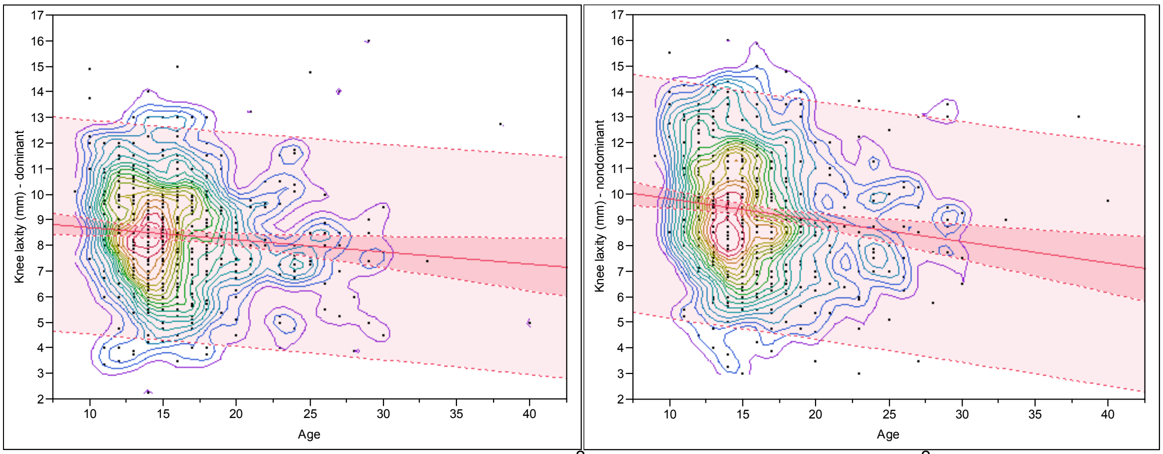


Figure 4.29 & 4.30 Knee laxity aging (dominant r^2=0.01 & non-dominant limb r^2=0.02)

The players which were not yet menstruated when evaluated, showed higher knee laxity than the players already menstruating (p<0.0005) (figure 4.31). The group of players *postmenarche* had on average 4.0 ± 3.7 years of gynaecological age[10]. The onset of the female cyclic hormonal fluctuations that occurs with menarche does not seem to have an increasing effect on knee laxity. Our results do not agree with others which found an increased generalized joint laxity after puberty (Quatman *et al.*, 2008).

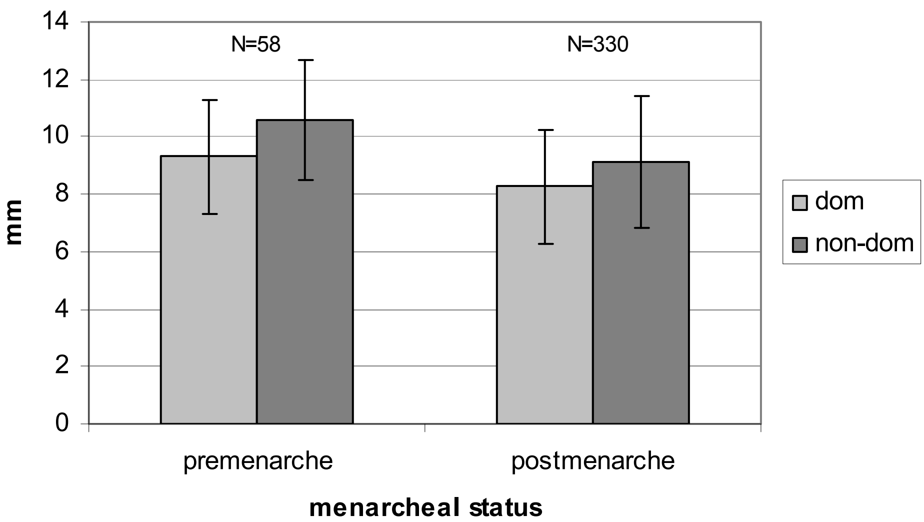


Figure 4.31 Knee laxity results by dominant and non-dominant limb before and after menarche

The knee laxity of OC and non-OC users was similar (dominant limb, p>0.8; non-dominant limb, p>0.1), at least among players without knee pathologies (figure 4.32). We must

[10] Calculated as the player's chronological age minus the age at menarche

notice that the non-OC users (≅16 ± 4 years of age) were 5 years younger than the OC-users (≅21 ± 4 years of age), and this could have contributed to masquerade some differences. We must also take into account the differences in their gynaecological age and handball experience. On average, the non-OC users were menstruating for ≅4 ± 3 years, and playing handball for about 5 years while the OC users were menstruating for ≅8± 4 years, and playing handball for about 8 years. So our two groups have several differences between them but they are big samples (table 4.2) that represent well the true populations under study. Other studies may have different results as they sampled other populations (Cammarata & Dhaher, 2008; Martineau *et al.*, 2004; Pokorny *et al.*, 2000).

Similarly to our findings, Pokorny *et al.* (2000) did not find any significant knee laxity differences between OC users (N=30) and non-OC users (N=25). Cammarata *et al.* (2008) also investigated the knee laxity differences between non-OC users (N=11), monophasic OC users (N=11) and triphasic OC users (N=11), finding no statistical differences between them. On the other hand, Martineau *et al.* (2004) found decreased knee laxity in athletes using OCs (with an average OC usage of ≅36 months, N= 42 athletes) than in non users (N=36 athletes). These results were obtained at lower loads (67N and 89N) and so are difficult to compare.

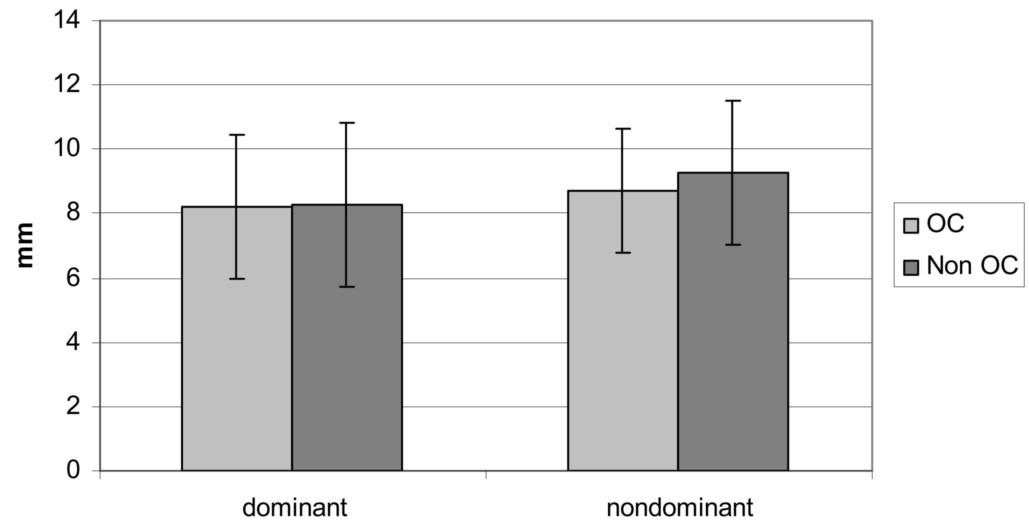


Figure 4.32 Knee laxity results by dominant and non-dominant limb, with and without OC treatment

A stepwise maximum-likelihood regression analysis (forward only, p_{in}=0.25) was performed on the dependent variable *anterior knee laxity* (laterally discriminated) with the independent variables *age, years of practice, playing category, type of practice floor, field position*, *SJ, CMJ, body mass, body fat content, duration of OC usage, BMI* and *gynaecological age*. With these rules applied to the dominant limb, the variables *years of practice, playing category, field position*, *CMJ* and *body fat content* were included in the model. This model presented the lowest AIC value (51.36) and a maximum explanation power of R^2=0.61. Mallow's C_p criteria suggests a model of 2 to 3 variables: *playing category, field position* and possibly *body fat content*. The results of this model are presented in figure 4.33 and tables 4.14, 4.15 and 4.16. The model is valid (P<0.0001) but the explained variance is just R^2=11%.

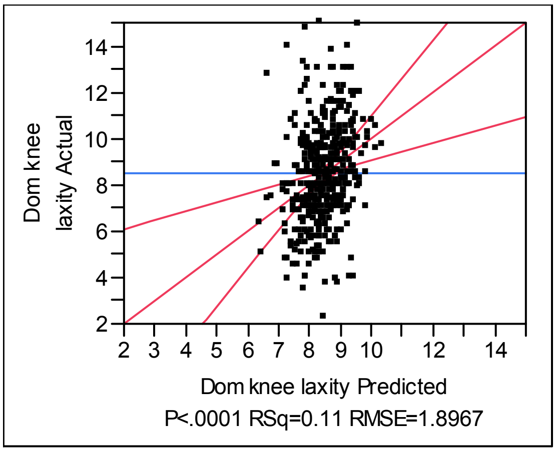


Figure 4.33 Actual by predicted plot for *dominant limb knee laxity*

Table 4.14 Maximum likelihood regression model on *dominant limb knee laxity*

Term	Estimate	Std Error	t Ratio	Prob>|t|	AdjPower.05
Intercept	5.7603342	0.945671	6.09	<.0001	1.0000
Years of practice	-0.063629	0.033012	-1.93	0.0548	0.3732
Playing category(1&3-4&5&6&7&8&9)	0.3499856	0.118622	2.95	0.0034	0.7877
Playing category(1-3)	0.3607991	0.172029	2.10	0.0367	0.4488
Playing category(4&5-6&7&8&9)	0.0627453	0.153249	0.41	0.6825	0.0500
Field position(1&2&0&9&6&10-4&5&7&8&3)	-0.370706	0.128742	-2.88	0.0042	0.7652
Field position(1-2&0&9&6&10)	-0.382133	0.200179	-1.91	0.0571	0.3651
Counter movement jump	0.0082732	0.002589	3.20	0.0015	0.8547
Fat content (%)	0.0396539	0.019986	1.98	0.0481	0.3982
RSquare	0.106138				
ANOVA: Prob > F	<0.0001				
Observations	341				

Table 4.15 Playing category effect on dominant knee laxity – linear fitting model

Playing category	Estimate
infant	0.710785
infant/starter	0
starter	-0.732412
starter/juvenile	-0.28724
juvenile	-0.28724
juvenile/junior	-0.412731
junior	-0.412731
junior/senior	-0.412731
senior	-0.412731

Table 4.16 Playing position effect on dominant knee laxity – linear fitting model

Playing position	Estimate
Goalkeeper	0.011427
Pivot	-0.752839
Left wing	0.011427
Left back	0.370706
Middle back	0.370706
Right back	0.370706
Right wing	0.011427
Right/left side	0.370706
Right side	0.370706
Left side	0.011427
Not defined	0.011427

In the non-dominant limb, the best model (lowest AIC) was obtain with a backward model (p_{out}=0.25). It included the variables *playing category, type of practice floor, field position*, *SJ*, *CMJ* and *body fat content*, of 50.36 and a maximum explanation power of R^2=0.77. Mallow's C_p criteria is not applicable. The results of this model are presented in figure 4.34 and tables 4.17, 4.18 and 4.19. The model is valid (P<0.0007) but the explained variance is just R^2=11%.

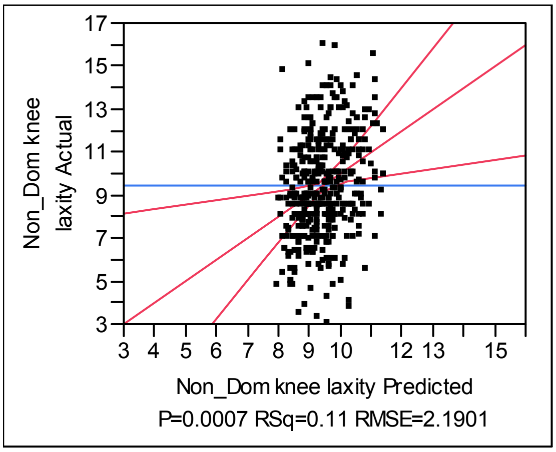


Figure 4.34 Actual by predicted plot for *non-dominant limb knee laxity*

Table 4.17 Maximum likelihood regression model on *non-dominant limb knee laxity*

Term	Estimate	Std Error	t Ratio	Prob>|t|	AdjPower.05
Intercept	7.5387247	1.177632	6.40	<.0001	1.0000
Playing category(1&3-4&5&6&7&8&9)	0.7259835	0.146104	4.97	<.0001	0.9980
Playing category(1-3)	0.5253663	0.199065	2.64	0.0087	0.6795
Playing category(4&5&6&7-8&9)	-0.002497	0.194693	-0.01	0.9898	0.0500
Playing category(4&5&6-7)	0.2777312	0.24053	1.15	0.2491	0.0878
Playing category(4&5-6)	0.2589681	0.221477	1.17	0.2432	0.0918
Usual practice floor type(4&3&1-6&2&5)	-0.365556	0.185592	-1.97	0.0497	0.3917
Usual practice floor type(4-3&1)	-0.252901	0.239928	-1.05	0.2926	0.0619
Usual practice floor type(3-1)	0.0918648	0.158441	0.58	0.5624	0.0500
Field position(2&6&1&9-10&0&4&5&7&8&3)	-0.220966	0.137661	-1.61	0.1094	0.2381
Field position(10&0&4&5&7-8&3)	-0.072676	0.187984	-0.39	0.6993	0.0500
Field position(8-3)	-0.235561	0.332983	-0.71	0.4798	0.0500
Squat jump	-0.002035	0.005785	-0.35	0.7253	0.0500
Counter movement jump	0.0079674	0.005402	1.47	0.1412	0.1892
Fat content (%)	0.0232467	0.023175	1.00	0.3166	0.0500
RSquare	0.107124				
ANOVA: Prob > F	<0.0007				
Observations	338				

Table 4.18 Playing category effect on non-dominant knee laxity – linear fitting model

Playing category	Estimate
infant	1.25135
infant/starter	0
starter	0.200617
starter/juvenile	-0.191781
juvenile	-0.191781
juvenile/junior	-0.709717
junior	-1.006212
junior/senior	-0.723487
senior	-0.723487

Table 4.19 Playing position effect on non-dominant knee laxity – linear fitting model

Playing position	Estimate
Goalkeeper	0.14829
Pivot	-0.220966
Left wing	-0.220966
Left back	0.529203
Middle back	0.14829
Right back	0.14829
Right wing	-0.220966
Right/left side	0.14829
Right side	0.058081
Left side	-0.220966
Not defined	0.14829

The same analysis was performed on the knee laxity asymmetry (dominant knee laxity – non-dominant knee laxity), with the same base variables. The chosen model was obtained with a forward technique (p_{in}=0.25). It included the variables *playing category, type of practice floor, field position*, which presented an AIC value of 20 and a maximum explanation power of R^2=0.49. Mallow's C_p criteria is not applicable. The results of this model are presented in figure 4.35 and tables 4.20, 4.21 and 4.22. The model is valid (P<0.0001) but the explained variance is just R^2=7%.

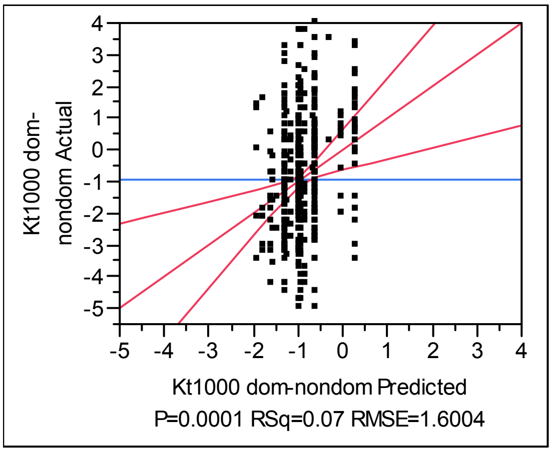


Figure 4.35 Actual by predicted plot for *knee laxity asymmetry*

Table 4.20 Maximum likelihood regression model on *knee laxity asymmetry*

Term	Estimate	Std Error	t Ratio	Prob>|t|	AdjPower.05
Intercept	-1.195556	0.141308	-8.46	<.0001	1.0000
Playing category(1&3&4-5&6&7&8&9)	-0.159171	0.083954	-1.90	0.0588	0.3598
Usual practice floor type(2&5-6&3&1&4)	-0.311608	0.139151	-2.24	0.0257	0.5126
Field position(3&0&8&1&10-4&9&5&7&6&2)	-0.257791	0.088942	-2.90	0.0040	0.7718
Field position(4&9&5&7-6&2)	-0.182859	0.122135	-1.50	0.1352	0.1976
Field position(6-2)	0.5651218	0.209399	2.70	0.0073	0.7027
RSquare	0.068194				
ANOVA: Prob > F	<0.0001				
Observations	369				

Table 4.21 Playing category effect on knee laxity asymmetry – linear fitting model

Playing category	Estimate
infant	-0.159171
infant/starter	0
starter	-0.159171
starter/juvenile	-0.159171
juvenile	0.159171
juvenile/junior	0.159171
junior	0.159171
junior/senior	0.159171
senior	0.159171

Table 4.22 Playing position effect on knee laxity asymmetry – linear fitting model

Playing position	Estimate
Goalkeeper	-0.257791
Pivot	-0.257791
Left wing	-0.124472
Left back	-0.257791
Middle back	0.074932
Right back	0.074932
Right wing	1.005772
Right/left side	0.074932
Right side	-0.257791
Left side	0.074932
Not defined	-0.257791

The net result of this exercise (a few faint regressions) is the identification of the two most important contributing factors for the laxity: age, through playing category, and load, both through playing category and through field position. This is the thesis of a load modulated (damage/adaptation) laxity. The infant category seems to be the only one where the probability of finding a high laxity measure is high.

The field position effect is most notorious in the case of the left back players, mostly in the non-dominant leg (both the take-off and landing leg). Among us they are usually the best shooters and so face strong and constant opposition. They jump heavily during a game many times with contact and falling out of balance. They carry the responsibility of generating positional superiority for their teams with constant plant-and-cut actions, most frequently to the right. They are also one of the heaviest players in a team. All this adds to the hypothesis of a load-induced laxity.

The amount of anterior-posterior knee displacement is sometimes used clinically, either to detect an ACL rupture or to evaluate a treatment's success. The clinical guides propose a difference of 3 mm or higher for discriminating between a normal knee and an ACL injury, again for 20lbf (89N) force (Rangger *et al.*, 1993; Daniel *et al.*, 1985b).

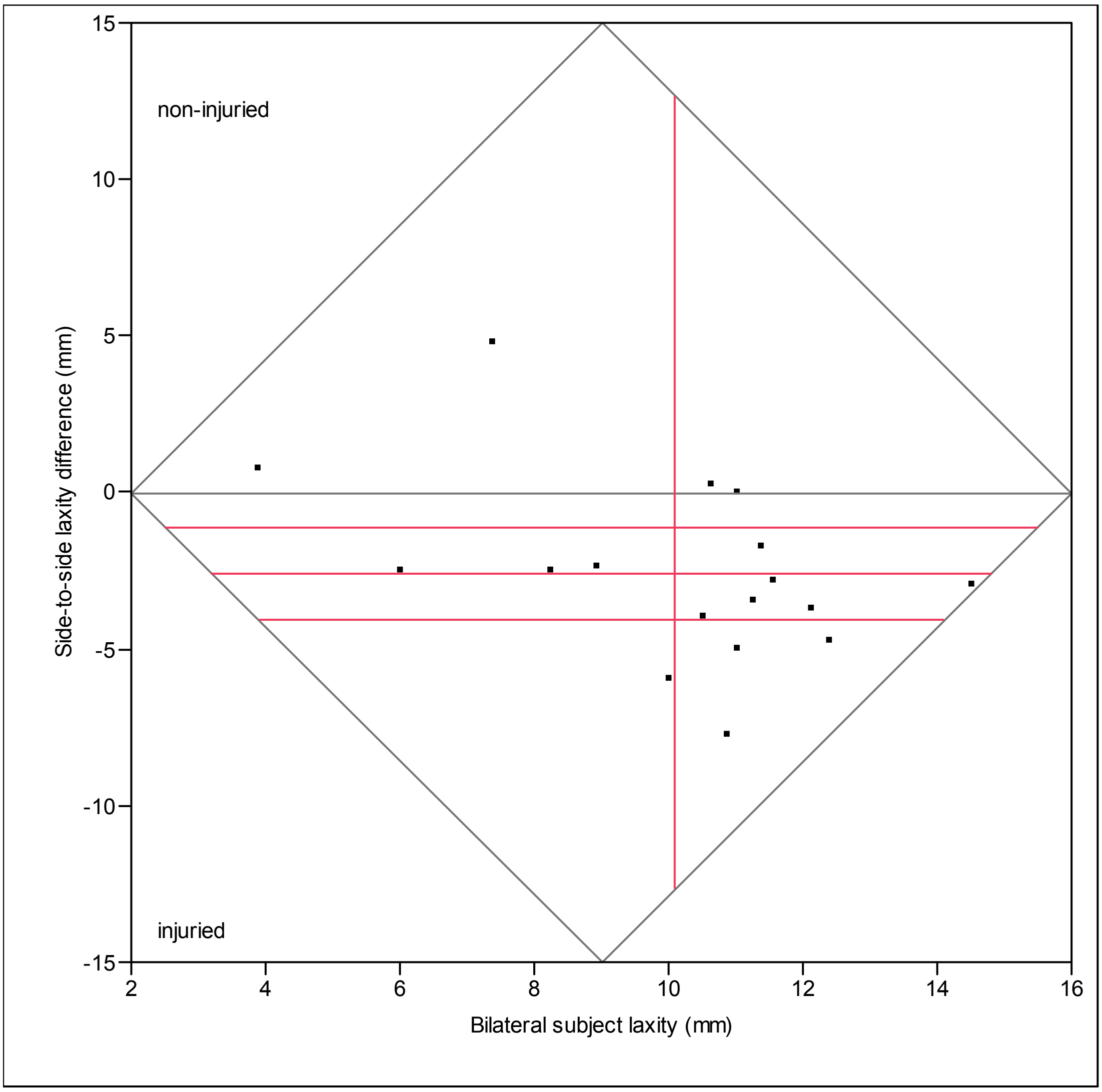


Figure 4.36 t-test on bilateral laxity in subjects with one ACL rupture ($p<0.0020$, N=17)

We compared the bilateral anterior knee laxity of all those players who suffered *one* ACL rupture and no other relevant knee injuries. The results are in figures 4.36 and 4.37. We can see here 3 trends: an extreme subject where the reconstructed ACL was too short and the resulting laxity (<5mm) was much smaller than that of the non-injured knee (9.7mm); a majority of subjects (9) where the injured knee had a laxity more than 3mm higher than the non-injured one (90% of the general population has asymmetries not exceeding 3mm); a third group of a little less than half of the cases where the surgery resulted in a correct anterior knee laxity, similar to the uninjured knee. Additional tests on the rotational laxity and, fundamentally, on the active stabilization of the injured knee would be highly desirable.

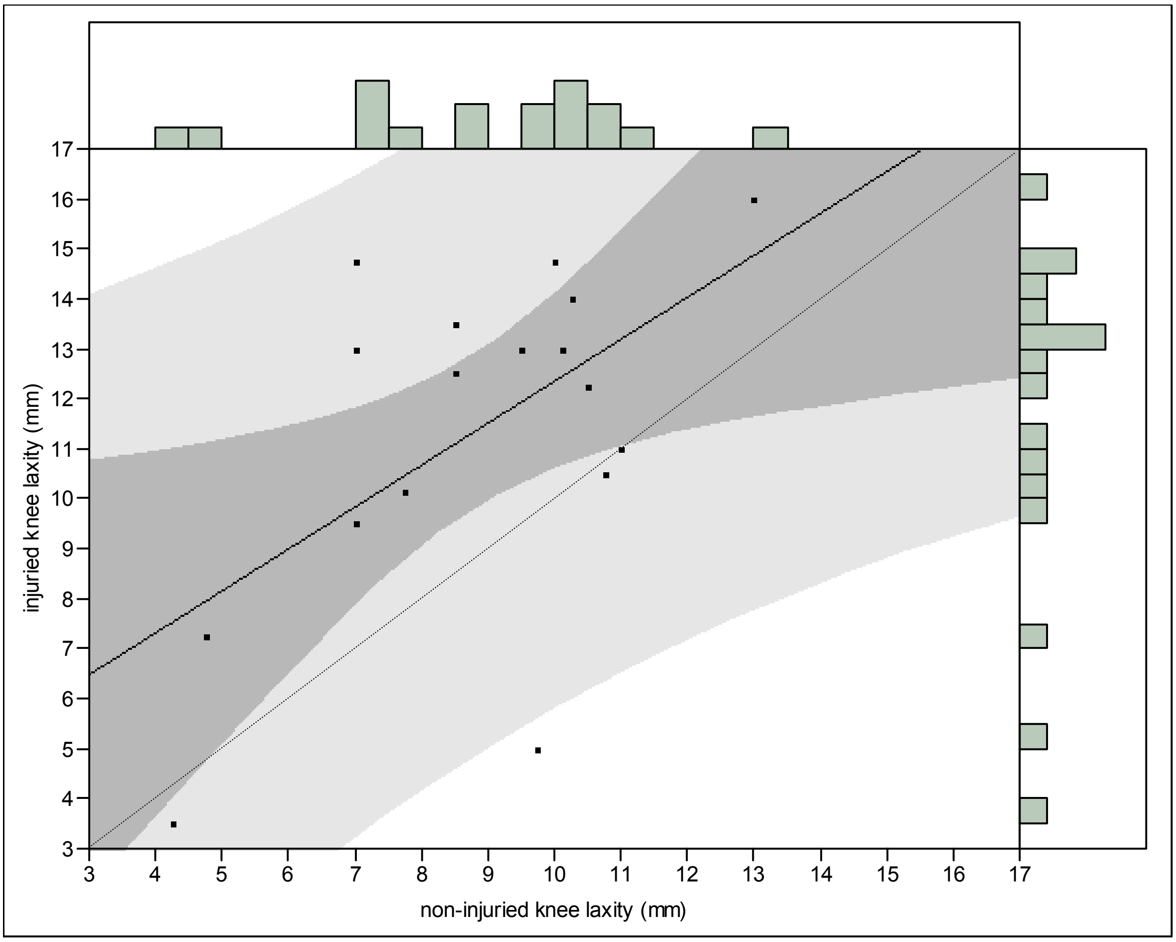


Figure 4.37 Orthogonal regression between injured and non-injured knee laxity ($p<0.0199$, $R^2=0.31$, injuried_knee_laxity=3.98+0.84*non-injured_knee_laxity)

Other factors like the type of injury (partial or integral), type of treatment (surgical or *conservative*), *etc.* may also have an impact on the final knee laxity and function. We were unable to collect this data reliably, and also to include all those who have abandoned the handball practice as a consequence of an ACL injury.

We should also stress two other aspects. Firstly, this population had an anterior knee laxity on the non-injured knee well above that of the other players of the same age category (8±2 mm; table 4.13). This[11] is consistent with the hypothesis of an excessive laxity risk factor. Secondly, even after an ACL reconstruction, the increased knee laxity (on average) may itself be a risk of premature gonarthrosis and/or other knee pathologies (Marks *et al.*, 2007; Myklebust *et al.*, 2003).

A little less than 90 subjects were excluded from this study on anterior knee laxity on the basis of previous (non ACL) knee injuries. *A posteriori*, we confirmed that they have knee laxities similar to the non-injured population (dominant limb $p>0.62$, non-dominant limb $p>0.31$). Our sample size was large enough to cope with this removal of 90 subjects without compromising the analysis.

Methodological limitations

As already stated (p. 51-54) ACL laxity measures are usually obtained indirectly. We used a popular measuring technique (anterior knee laxity)[12] although no one knows the quantitative relation between the anterior knee laxity and the ACL laxity.

This is a rather simple and rough method, developed for clinical purposes, that easily raises concerns about its reliability and reproducibility (Wiertsema *et al.*, 2008; Isberg *et al.*, 2006; Thompson *et al.*, 2004; Fleming *et al.*, 2002). These concerns have its roots in several areas, well detailed in the literature (Fung, 1993; Viidik, 1973):

- The behaviour of the ACL when loaded (stress-strain functional relation) is not linear but sigmoidal (chapter 2, pp 52-53). Therefore, there's not one stiffness but a stiffness function (of the load)
- The ligament response to load (and so it's stiffness) depends on the dynamics of the load *e.g.* the lengthening velocity or the loading rate – the viscoelastic characteristics of the ligament
- The ligament changes its structure, and consequently its elastic properties, after previous loads. This is apparent both in the stress relaxation under constant load or in the decreasing stiffness under cyclic loading. The *relaxed status* is only partially recoverable in the minutes scale (Gomez, 2001; Viidik, 1973).
- The load-elongation dependence of the ACL on the preconditioning state eventually reaches a steady state where ligament deformation becomes constant for the same type of loads (Fung, 1993; Viidik, 1973).

[11] If these measures are representative of the laxity at the time of injury
[12] The measurement of the anterior tibial translation relative to femur under calibrated forces

- Hormones and temperature may also influence the load-elongation of the ACL (Viidik, 1973).
- The ACL has bundles[13] that are not stressed by knee translation methods (Zantop *et al.*, 2007; Gabriel *et al.*, 2004).
- The muscular relaxation status interferes with the anterior knee laxity measurements (Pedowitz & Popejoy, 2004; Feller *et al.*, 2000) but (obviously) not with the ACL laxity.
- The technique is sensitive to the operator's hand dominance, experience and technique, alignment with the limb, *etc.* (Sernert *et al.*, 2007; Fleming *et al.*, 2002; Ballantyne *et al.*, 1995).

We believe that the translation and rotation methods combined, if applied shortly *after* a training session by an experienced operator on experienced subjects, produce indicators, accurate enough, of the ACL's health. They may even be of some quantitative value for our purposes if several velocities and loads are considered and extrapolated. However, in the interest of simplicity[14] and comparability with the mass of published results we decided to use the standard measurement protocols, namely to perform the anterior knee laxity tests exclusively, applied prior to any intense physical activity (ACL relaxed status), at an arbitrarily low velocity and at low loads.

4.4 Conclusions

In this population we showed that lower limb force, measured by the jump capacity, have insignificant progresses from 11 to 16 years old (between 13% and 17%, tables 4.7 & 4.8, figure 4.21), where the *general* French female population shows a 66% evolution. There is also a weight problem (table 4.9) but no asymptotic height difference to French females (figure 4.22). This all points to a deficit in sportive selection and conditional training.

These players are subjected to the competitive loads (even international) but have to carry the added weight and body fat content while showing lower conditional capacity *e.g.* to avoid risk situations, to dynamically stabilize the knee, *etc.* They are exposed to a high dynamic risk of knee injury.

There are strong critics to the ACL laxity measurement procedures based on the anterior knee laxity.

[13] Namely the posterolateral bundle, in a classic two-bundle classification
[14] To avoid the development and construction of a new kind of measuring machines

The anterior knee laxity decreases with age (table 4.13, figures 4.28, 4.29 & 4.30). It does not depend on OC usage (figure 4.32), but seems to depend on the specificity of the load (tables 4.16 & 4.19). It is higher in the left back and lower in the pivots (tables 4.16 & 4.19).

ACL injuries could be related to high anterior knee laxity and, on average, do not seem to recover completely after surgery. This increased knee laxity and the return to practice (that surgery allows), leave the subject at a high risk of premature gonarthrosis and/or other knee pathologies.

4.5 Perspectives

It would be interesting to return to these athletes a year later and study all those who, in this period, suffered an ACL rupture. We plan on doing this.

Is there a (micro) damage induced laxity *i.e.* is the (excessive) load an important contributive factor for an increased laxity? A histological study *e.g.* in an animal model, it would be very interesting in helping to clarify this issue.

The application of more precise passive knee laxity measurements (rotatory, after load, *etc.*) and the addition of some active stabilization measurements (*e.g.* hop jumps) might help in building a better understanding of this problem and in the evaluation of the ACL injury and recovery after reconstruction.

4.6 References

Adirim, T. A. & Cheng, T. L. (2003). Overview of injuries in the young athlete. *Sports Med, 33*(1), 75-81.

Akaike, H. (1974). A new look at the statistical model identification. *IEEE Transactions on Automatic Control, 19*(6), 7.

Ballantyne, B. T., French, A. K., Heimsoth, S. L., Kachingwe, A. F., Lee, J. B. & Soderberg, G. L. (1995). Influence of examiner experience and gender on interrater reliability of KT-1000 arthrometer measurements. *Phys Ther, 75*(10), 898-906.

Bobbert, M. F., Gerritsen, K. G., Litjens, M. C. & Van Soest, A. J. (1996). Why is countermovement jump height greater than squat jump height? *Med Sci Sports Exerc, 28*(11), 1402-1412.

Bosco, C., Luhtanen, P. & Komi, P. V. (1983a). A simple method for measurement of mechanical power in jumping. *Eur J Appl Physiol Occup Physiol, 50*(2), 273-282.

Bosco, C., Mognoni, P. & Luhtanen, P. (1983b). Relationship between isokinetic performance and ballistic movement. *Eur J Appl Physiol Occup Physiol, 51*(3), 357-364.

Bosco, C., Tihanyi, J. & Viru, A. (1996). Relationships between field fitness test and basal serum testosterone and cortisol levels in soccer players. *Clin Physiol, 16*(3), 317-322.

Buchanan, P. A. & Vardaxis, V. G. (2003). Sex-Related and Age-Related Differences in Knee Strength of Basketball Players Ages 11-17 Years. *J Athl Train, 38*(3), 231-237.

Cammarata, M. L. & Dhaher, Y. Y. (2008). The differential effects of gender, anthropometry, and prior hormonal state on frontal plane knee joint stiffness. *Clin Biomech (Bristol, Avon), 23*(7), 937-945.

Cavagna, G. A., Saibene, F. P. & Margaria, R. (1965). Effect of Negative Work on the Amount of Positive Work Performed by an Isolated Muscle. *J Appl Physiol, 20*, 157-158.

Daniel, D. M., Malcom, L. L., Losse, G., Stone, M. L., Sachs, R. & Burks, R. (1985a). Instrumented measurement of anterior laxity of the knee. *J Bone Joint Surg Am, 67*(5), 720-726.

Daniel, D. M., Stone, M. L., Sachs, R. & Malcom, L. (1985b). Instrumented measurement of anterior knee laxity in patients with acute anterior cruciate ligament disruption. *Am J Sports Med, 13*(6), 401-407.

Dietz, V., Schmidtbleicher, D. & Noth, J. (1979). Neuronal mechanisms of human locomotion. *J Neurophysiol, 42*(5), 1212-1222.

Feller, J., Hoser, C. & Webster, K. (2000). EMG biofeedback assisted KT-1000 evaluation of anterior tibial displacement. *Knee Surg Sports Traumatol Arthrosc, 8*(3), 132-136.

Fleming, B. C., Brattbakk, B., Peura, G. D., Badger, G. J. & Beynnon, B. D. (2002). Measurement of anterior-posterior knee laxity: a comparison of three techniques. *J Orthop Res, 20*(3), 421-426.

Flynn, J. M., Mackenzie, W., Kolstad, K., Sandifer, E., Jawad, A. F. & Galinat, B. (2000). Objective evaluation of knee laxity in children. *J Pediatr Orthop, 20*(2), 259-263.

Fung, Y. C. (1993). *Biomechanics : mechanical properties of living tissues* (2nd ed.). New York: Springer-Verlag.

Gabriel, M. T., Wong, E. K., Woo, S. L., Yagi, M. & Debski, R. E. (2004). Distribution of in situ forces in the anterior cruciate ligament in response to rotatory loads. *J Orthop Res, 22*(1), 85-89.

Gomez, M. A. (2001). *Biomechanics of soft tissue injury*. Tucson, AZ: Lawyers & Judges Pub.

Grindstaff, T. L., Hammill, R. R., Tuzson, A. E. & Hertel, J. (2006). Neuromuscular control training programs and noncontact anterior cruciate ligament injury rates in female athletes: a numbers-needed-to-treat analysis. *J Athl Train, 41*(4), 450-456.

Hass, C. J., Schick, E. A., Tillman, M. D., Chow, J. W., Brunt, D. & Cauraugh, J. H. (2005). Knee biomechanics during landings: comparison of pre- and postpubescent females. *Med Sci Sports Exerc, 37*(1), 100-107.

Hatze, H. (1990). The charge transfer model of myofilamentary interaction - prediction of force enhancement and related myodynamic phenomena. In J. M. Winters & S. L. Y. Woo (Eds.), *Multiple muscle systems : biomechanics and movement organization* (pp. 24 - 45). New York: Springer-Verlag.

Herzog, W., Schachar, R. & Leonard, T. R. (2003). Characterization of the passive component of force enhancement following active stretching of skeletal muscle. *J Exp Biol, 206*(Pt 20), 3635-3643.

Hewett, T. E., Lindenfeld, T. N., Riccobene, J. V. & Noyes, F. R. (1999). The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. *Am J Sports Med, 27*(6), 699-706.

Hewett, T. E., Myer, G. D. & Ford, K. R. (2004). Decrease in neuromuscular control about the knee with maturation in female athletes. *J Bone Joint Surg Am, 86-A*(8), 1601-1608.

Hewett, T. E., Myer, G. D. & Ford, K. R. (2006). Anterior cruciate ligament injuries in female athletes: Part 1, mechanisms and risk factors. *Am J Sports Med, 34*(2), 299-311.

Hinton, R. Y., Rivera, V. R., Pautz, M. J. & Sponseller, P. D. (2008). Ligamentous laxity of the knee during childhood and adolescence. *J Pediatr Orthop, 28*(2), 184-187.

Isberg, J., Faxen, E., Brandsson, S., Eriksson, B. I., Karrholm, J. & Karlsson, J. (2006). KT-1000 records smaller side-to-side differences than radiostereometric analysis before and after an ACL reconstruction. *Knee Surg Sports Traumatol Arthrosc, 14*(6), 529-535.

Jones, G. M. & Watt, D. G. (1971). Observations on the control of stepping and hopping movements in man. *J Physiol, 219*(3), 709-727.

Kalapotharakos, V. I., Tokmakidis, S. P., Smilios, I., Michalopoulos, M., Gliatis, J. & Godolias, G. (2005). Resistance training in older women: effect on vertical jump and functional performance. *J Sports Med Phys Fitness, 45*(4), 570-575.

Komi, P. V. & Bosco, C. (1978). Utilization of stored elastic energy in leg extensor muscles by men and women. *Med Sci Sports, 10*(4), 261-265.

Kramer, L. C., Denegar, C. R., Buckley, W. E. & Hertel, J. (2007). Factors associated with anterior cruciate ligament injury: history in female athletes. *J Sports Med Phys Fitness, 47*(4), 446-454.

Kuczmarski, R. J., Ogden, C. L., Grummer-Strawn, L. M., Flegal, K. M., Guo, S. S., Wei, R., et al. (2000). CDC growth charts: United States. *Adv Data*(314), 1-27.

Leard, J. S., Cirillo, M. A., Katsnelson, E., Kimiatek, D. A., Miller, T. W., Trebincevic, K., et al. (2007). Validity of two alternative systems for measuring vertical jump height. *J Strength Cond Res, 21*(4), 1296-1299.

Mallows, C. L. (1973). Some Coments on Cp. *Technometrics, 15*, 14.

Markovic, G., Dizdar, D., Jukic, I. & Cardinale, M. (2004). Reliability and factorial validity of squat and countermovement jump tests. *J Strength Cond Res, 18*(3), 551-555.

Marks, P. H., Droll, K. P. & Cameron-Donaldson, M. (2007). Does ACL reconstruction prevent articular degeneration? In T. E. Hewett, S. J. Shultz & L. Y. Griffin (Eds.), *Understanding and Preventing Noncontact ACL injuries* (pp. 31-45). Champaign, IL: American Orthopeadic Society for Sports Medicine

Martineau, P. A., Al-Jassir, F., Lenczner, E. & Burman, M. L. (2004). Effect of the Oral Contraceptive Pill on Ligamentous Laxity. *Clin J Sport Med 14*(5), 281-286.

McGuigan, M. R., Doyle, T. L., Newton, M., Edwards, D. J., Nimphius, S. & Newton, R. U. (2006). Eccentric utilization ratio: effect of sport and phase of training. *J Strength Cond Res, 20*(4), 992-995.

Myklebust, G., Holm, I., Maehlum, S., Engebretsen, L. & Bahr, R. (2003). Clinical, functional, and radiologic outcome in team handball players 6 to 11 years after anterior cruciate ligament injury: a follow-up study. *Am J Sports Med, 31*(6), 981-989.

Pedowitz, R. A. & Popejoy, D. J. (2004). Initial laxity does not correlate with subsequent laxity after ACL reconstruction in humans: a prospective evaluation. *Am J Orthop, 33*(11), 560-564.

Pokorny, M. J., Smith, T. D., Calus, S. A. & Dennison, E. A. (2000). Self-reported oral contraceptive use and peripheral joint laxity. *J Orthop Sports Phys Ther, 30*(11), 683-692.

Pollard, C. D., Braun, B. & Hamill, J. (2006). Influence of gender, estrogen and exercise on anterior knee laxity. *Clin Biomech (Bristol, Avon), 21*(10), 1060-1066.

Quatman, C. E., Ford, K. R., Myer, G. D. & Hewett, T. E. (2006). Maturation leads to gender differences in landing force and vertical jump performance: a longitudinal study. *Am J Sports Med, 34*(5), 806-813.

Quatman, C. E., Ford, K. R., Myer, G. D., Paterno, M. V. & Hewett, T. E. (2008). The effects of gender and pubertal status on generalized joint laxity in young athletes. *J Sci Med Sport, 11*(3), 257-263.

Rangger, C., Daniel, D. M., Stone, M. L. & Kaufman, K. (1993). Diagnosis of an ACL disruption with KT-1000 arthrometer measurements. *Knee Surg Sports Traumatol Arthrosc, 1*(1), 60-66.

Rickenlund, A., Carlstrom, K., Ekblom, B., Brismar, T. B., Von Schoultz, B. & Hirschberg, A. L. (2004). Effects of oral contraceptives on body composition and physical performance in female athletes. *J Clin Endocrinol Metab, 89*(9), 4364-4370.

Riederman, R., Wroble, R. R., Grood, E. S., VanGinkel, L. & Shaffer, B. L. (1991). Reproducibility of the knee signature system. *Am J Sports Med, 19*(6), 660-664.

Rosene, J. M. & Fogarty, T. D. (1999). Anterior Tibial Translation in Collegiate Athletes with Normal Anterior Cruciate Ligament Integrity. *J Athl Train, 34*(2), 93-98.

Rousanoglou, E. N., Georgiadis, G. V. & Boudolos, K. D. (2008). Muscular strength and jumping performance relationships in young women athletes. *J Strength Cond Res, 22*(4), 1375-1378.
Rozzi, S. L., Lephart, S. M., Gear, W. S. & Fu, F. H. (1999). Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. *Am J Sports Med, 27*(3), 312-319.
Rozzi, S. L., Yuktanandana, P., Pincivero, D. & Lephart, S. M. (2001). Role of fatigue on proprioception and neuromuscular control. In S. M. Lephart & F. H. FU (Eds.), *Proprioception and neuromuscular control in joint stability* (pp. 375-383): Human kinetics-copyright.
Sands, W. A., McNeal, J. R., Ochi, M. T., Urbanek, T. L., Jemni, M. & Stone, M. H. (2004). Comparison of the Wingate and Bosco anaerobic tests. *J Strength Cond Res, 18*(4), 810-815.
Sernert, N., Helmers, J., Kartus, C., Ejerhed, L. & Kartus, J. (2007). Knee-laxity measurements examined by a left-hand- and a right-hand-dominant physiotherapist, in patients with anterior cruciate ligament injuries and healthy controls. *Knee Surg Sports Traumatol Arthrosc, 15*(10), 1181-1186.
Sernert, N., Kartus, J. T., Ejerhed, L. & Karlsson, J. (2004). Right and Left Knee Laxity Measurements: A Prospective Study of Patients With Anterior Cruciate Ligament Injuries and Normal Control Subjects. The Journal of Arthroscopic and Related Surgery, 20(6), 564-571.
Shultz, S. J., Carcia, C. R. & Perrin, D. H. (2004). Knee joint laxity affects muscle activation patterns in the healthy knee. *J Electromyogr Kinesiol, 14*(4), 475-483.
Shultz, S. J., Sander, T. C., Kirk, S. E. & Perrin, D. H. (2005). Sex differences in knee joint laxity change across the female menstrual cycle. *J Sports Med Phys Fitness, 45*(4), 594-603.
Skinner, H. B., Wyatt, M. P., Hodgdon, J. A., Conard, D. W. & Barrack, R. L. (1986). Effect of fatigue on joint position sense of the knee. *J Orthop Res, 4*(1), 112-118.
Solomonow, M. & Krogsgaard, M. (2001). Sensorimotor control of knee stability. A review. *Scand J Med Sci Sports, 11*(2), 64-80.
Temfemo, A., Hugues, J., Chardon, K., Mandengue, S. H. & Ahmaidi, S. (2008). Relationship between vertical jumping performance and anthropometric characteristics during growth in boys and girls. *Eur J Pediatr*.
Thompson, M. T., Conditt, M. A., Ismaily, S. K., Agarwal, A. & Noble, P. C. (2004). Brief report: validation of a system for automated measurement of knee laxity. *Clin Biomech (Bristol, Avon), 19*(3), 308-312.
Uhorchak, J. M., Scoville, C. R., Williams, G. N., Arciero, R. A., Pierre, P. S. & Taylor, D. C. (2003). Risk factors associated with noncontact injury of the anterior cruciate ligament: a propective four-year evaluation of 859 West Point Cadets. *American Journal of Sports Medicine, 31*(6), 831-842.
Vanezis, A. & Lees, A. (2005). A biomechanical analysis of good and poor performers of the vertical jump. *Ergonomics, 48*(11-14), 1594-1603.
Vicente-Rodriguez, G., Dorado, C., Perez-Gomez, J., Gonzalez-Henriquez, J. J. & Calbet, J. A. (2004). Enhanced bone mass and physical fitness in young female handball players. *Bone, 35*(5), 1208-1215.
Viidik, A. (1973). Functional properties of collagenous tissues. *Int Rev Connect Tissue Res, 6*, 127-215.
Wiertsema, S. H., van Hooff, H. J., Migchelsen, L. A. & Steultjens, M. P. (2008). Reliability of the KT1000 arthrometer and the Lachman test in patients with an ACL rupture. *Knee, 15*(2), 107-110.
Zantop, T., Herbort, M., Raschke, M. J., Fu, F. H. & Petersen, W. (2007). The role of the anteromedial and posterolateral bundles of the anterior cruciate ligament in anterior tibial translation and internal rotation. *Am J Sports Med, 35*(2), 223-227.
Zatsiorsky, V. M. (1995). *Science and practice of strength training*. Champaign, IL: Human Kinetics.

Chapter 5

Isokinetic Strength of Hamstrings/Quadriceps and ACL Injuries

5.1 Introduction

ACL injuries have been usually implicated in an early progression of knee osteoarthritis and the main objective of any posterior intervention would be to stabilize the knee and avoid its decay, more than recover function. Unfortunately there are reports that suggest that even the surgical reconstruction of the ACL is unable to completely avoid both the malfunction and the osteoarthritis (Kessler *et al.*, 2008; Fleming, 2003). Consequently every effort has to be done to prevent an ACL rupture and to detect and diagnose those at higher risk.

The ACL retrains (among other) the anterior translation of the tibia over the femur, a movement that is promoted by the quadriceps and demoted by the hamstrings (among other muscle groups; see section 2.3). In a *naïf* description, quadriceps are *bad* to the ACL and hamstrings are *good*. The reality is a little more complex however, as the highest loads are external, not internal (see below).

Some particular movements and their mechanical loads over the lower limb, as well as the minimum neuromuscular capabilities and strategies to handle them, have been investigated in order to detect possible risk situations. Some cadaveric, simulation (Shin *et al.*, 2007; Mesfar & Shirazi-Adl, 2005; Pandy & Shelburne, 1997) and in-vivo (Beynnon & Fleming, 1998) studies about pure sagital movements have found a protective effect in the posterior force by counteracting the quadriceps action. These same studies have also emphasised the improbability of any kind of injury from the quadriceps action alone in these kinds of movements (pure anterior-posterior displacements). In planar jumps, the main ACL loading (2%-3% strain) occurs in the beginning of the landing phase (30ms) i.e. at low knee flexion angles (less than 30º) but far from the ACL rupture limit (*opus citæ*).

The conclusions of these mechanical studies, and also from others on a more epidemiological point of view (Hewett *et al.*, 2006; Boden *et al.*, 2000), are that the *out of plane* actions (valgus-varus torques, lateral displacements, rotations) are more suspicious from an ACL injury perspective than the purely planar movements.

However many authors have, since 1955, pursued the pure agonist/antagonist perspective and opted to consider the ratio of maximum voluntary knee flexion-extension torques, reviving the thesis that quadriceps are a source of ACL risk. This has started with a mere statistical description of the average human knee torque ratio (2:3 according to Steindler (1955)) and evolved to the surprising extrapolation that a H:Q (hamstrings:quadriceps) torque ratio lower than 2/3 is an indicator of ACL injury risk. Variations were introduced along the way, with the H:Q determined both concentrically

(Heiser *et al.*, 1984) or in combinations of eccentric/concentric contractions (Aagaard *et al.*, 1998; Dvir *et al.*, 1989).

Even though the H:Q concept has never been proved right (as an ACL risk factor) and despite having long time opponents (Ekstrand & Gillquist, 1983), quite a lot of researchers still use these techniques. They usually perform these measurements in isokinetic devices at concentric speeds chosen between 60º/s and 360º/s, although sports actions can occur at very different speeds. The H:Q ratio is known to present an angular velocity (reviewed by Nosse (1982)) and sex dependence (reviewed by Hewett *et a*l. (2006)) that is usually ignored.

One common criticism is related to the choice of the maximum torque over all the range of action for each muscle, irrespectively of the angle at which it occurred. The H:Q ratio is the ratio of maximum torques and can be the ratio of the hamstrings' torque at 35º over the torque of quadriceps at 60º (figure 5.1), a ratio with poor significance in a real action at both angles (or at any other angle) (Ekstrand *et al.*, 2003). Some determine the continuous torque ratio (Aagaard *et al.*, 1998) and select the maximum, what is probably a little more realistic but incomparable with the results of the more common methodology.

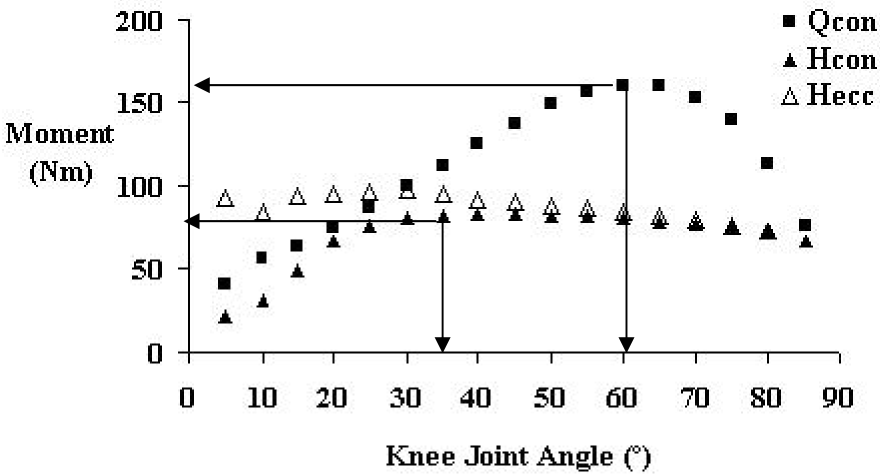


Figure 5.1 Isokinetic knee torques (Coombs & Garbutt, 2002, p57)

Another problem is the fact that fatigue changes all these measures and ratios (figure 5.2), in a manner that is subject (and load) dependent and independent of the maximum force – those with more resistance to fatigue are not necessarily the strongest ones. Interestingly, in our sample the majority of injuries did not occur at the beginning of the games.

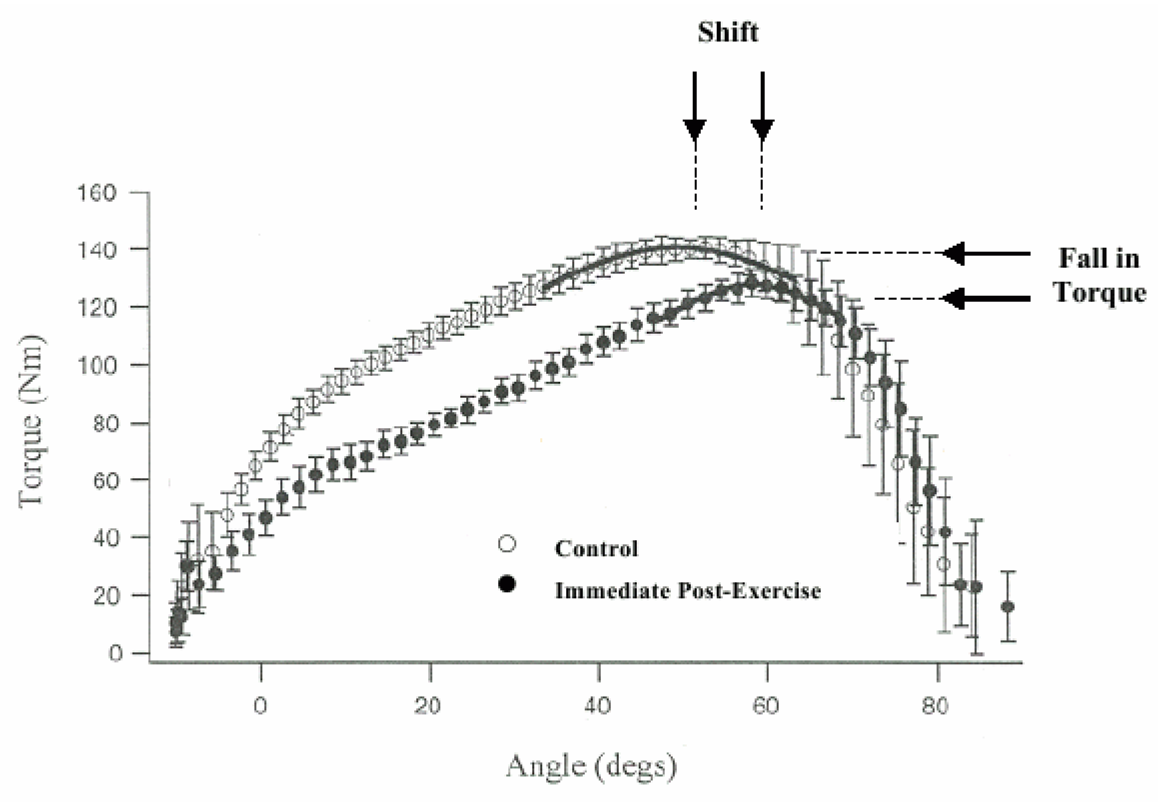


Figure 5.2 Isokinetic hamstrings torques (Proske *et al.*, 2004, p27)

Despite all these critics, a more radical extrapolation (also not validated) has been done and the H:Q started to be used as a global ACL risk indicator for general movements in different sports[1] (Soderman *et al.*, 2001; Ostenberg & Roos, 2000; Knapik *et al.*, 1991).

As the torque-angle curve can deviate significantly from the classical parabolic shape (Hill derived models), some authors prefer to include also an analysis of the total flexors' or extensors' work[2] (Rosene *et al.*, 2001) as a more realistic indicator of muscular competence and knee function than just peak torque measurements.

Others have preferred to determine the ACL loads in real sports' movements (Sell *et al.*, 2006; Hewett *et al.*, 2005), either by instrumenting the ACL (Beynnon & Fleming, 1998), by inverse dynamics or by direct simulation (Shin *et al.*, 2007; Mesfar & Shirazi-Adl, 2005; Pandy & Shelburne, 1997). In some cases the muscle recruitment patterns of the lower limb were also investigated (Simonsen *et al.*, 2000), with the use of electromyographic techniques.

Lower limb force is important in the active stabilization of the knee (Solomonow & Krogsgaard, 2001). The H:Q indicator, with it's limitations, is just a crude attempt to

[1] obviously including movements out-of-the-plane
[2] the area beneath the torque-angle curve

quantify this. However, from a biomechanics perspective, the determination of a singular ACL risk parameter seems to be a yet unfinished task.

Study purposes: (a) to characterize any muscular force adaptations over the players' handball career, including asymmetries and consequent risk of ACL injuries; and (b) to investigate a possible epidemiological correlation between lower limb isokinetic strength and previous or subsequent ACL injuries.

5.2. Methods

5.2.1 Experimental design

This work was conducted during the 2004-05 season, concurrently with the second prospective injury study (follow up 4). The players, all volunteers, were from Porto's Regional Association (7 Clubs). The players' evaluation started 3 months after the beginning of the 2004-05 season. The players were asked to come to the Sports Faculty for both an anthropometric-functional evaluation and a clinical examination performed by an orthopaedic surgeon.

5.2.2 Isokinetic Strength Measurements

The isokinetic torque-angle curves were obtained with a standard dynamometer[3] at an angular speed of 90º s^{-1}. Prior to the tests, subjects performed a 5 minute warm-up in a cycloergometer with a resistance corresponding to 2% of body weight. The standard practice and procedures where observed, including leg positioning and placement, alignment and sub maximal trials for final warm-up and familiarization. The protocol consisted of a set of three flexion-extension cycles, from full leg extension to 90º flexion. The $(H:Q)_{conc}$ ratio refers to the ratio of maximal concentric isokinetic flexors torque relative to maximal concentric isokinetic extensors torque.

The measurements were always performed by the same trained operator.

5.2.3 Preliminary study

This study assumes that one can attribute a value (or a distribution) to the isokinetic characteristics of each player. In fact, the human performance varies in time and the

[3] Biodex - System 2, NY, USA

instruments and procedures used to measure that performance also introduce their own sources of error. To quantify both those parameters, a pair of homozygotic twins was invited to perform a set of five repeated isokinetic measurements in five different days.

The twins lived together and studied together (in the same class, both sport students), so their physical and psychological loads were similar. However, their menstrual cycles were out of phase (not synchronized).

In figure 5.3 (left) we can see a waving pattern that can be due to instrumental causes or to a true fluctuation of ones physical performance. It applies to both legs, both twins and all measured quantities.

If we plot this by menstrual cycle day (figure 5.3, right) the waving pattern becomes fuzzier and the parallelism between the twins is harder to identify, indicating a stronger influence of daily loads than from cycle environment.

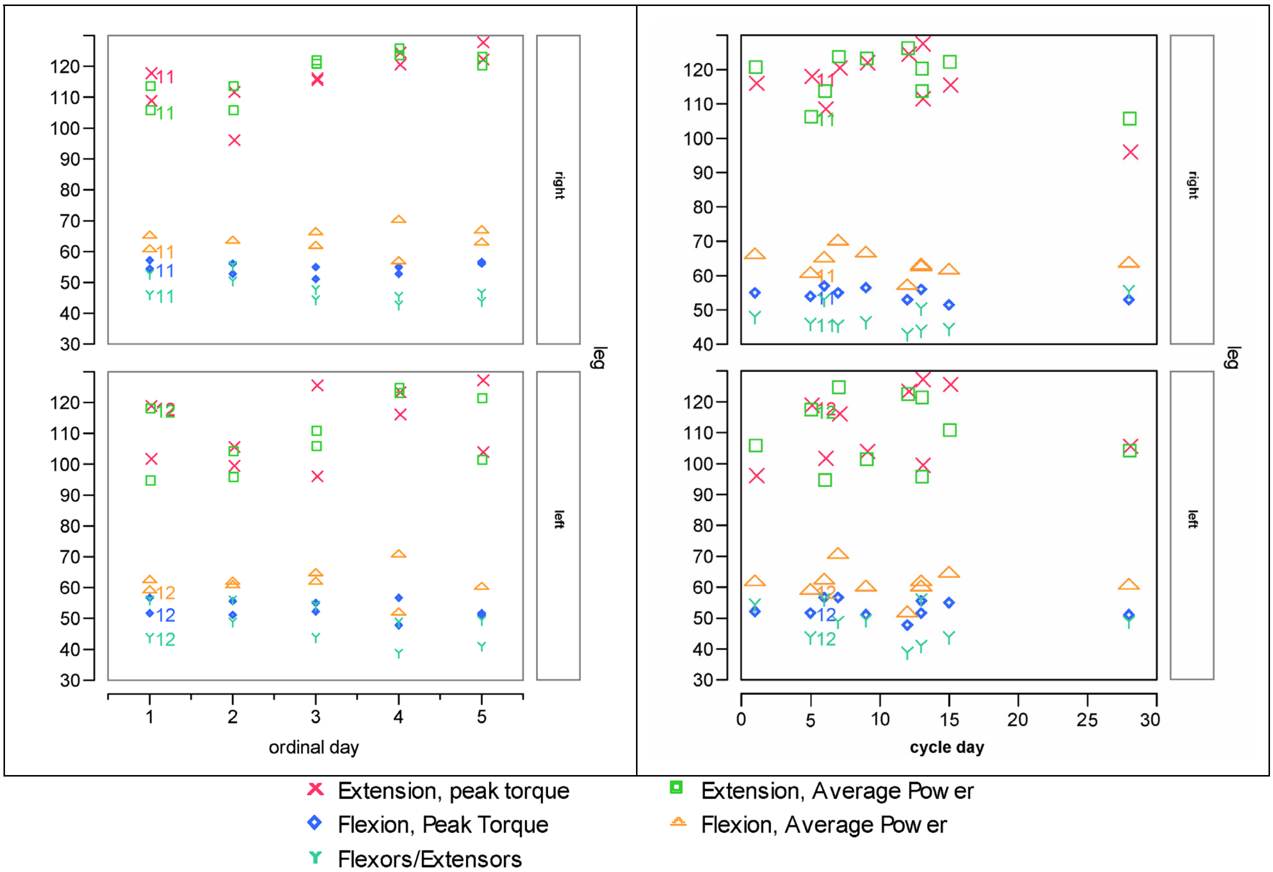


Figure 5.3 Preliminary study: twin's isokinetic repeated measures

Analyzing globally the set of measures, one obtains table 5.1 that is further reduced in table 5.2. The first two lines of table 5.2 indicate the minimal differences one wants to be able to detect and the typical dispersions on expects to find. The bottom two lines indicate the expected power (in tests assuming normality) for a fixed sample size or, conversely, the sample size for a target test power. Material limitations forced us to no more than 100 isokinetic essays so we expected to have no more than 1-47%=53% false negatives (type II errors) in tests involving the extension peak torque, etc. These are just estimates but indicate us that the H:Q ratio will be more coarsely tested than the other parameters.

Table 5.1 Preliminary study: twins isokinetic repeated measures

	Extension peak torque		Extension average power		Flexion peak torque		Flexion average power		H:Q	
	Mean	Stdev	Mean	Stdev	Mean	Stdev	Mean	Stdev	Mean	Stdev
global	113.4	10.4	113.4	10.0	53.8	2.5	62.4	4.3	0.48	0.05
Subject 11, right leg	117.1	8.2	118.9	5.4	54.7	2.4	61.8	3.1	0.47	0.04
Subject 11, left leg	114.8	13.7	108.79	13.5	53.3	3.6	59.9	5.0	0.47	0.08
Subject 12, right leg	113.9	10.6	115.69	9.2	54.7	1.4	65.3	3.6	0.48	0.04
Subject 12, left leg	107.7	9.5	110.5	9.9	52.6	2.24	62.4	4.7	0.49	0.04

Table 5.2 Preliminary study: twins isokinetic repeated measures & power prediction

	Extension peak torque	Extension average power	Flexion peak torque	Flexion average power	H:Q
Mean interclass diference	4.0	5.1	1.1	2.3	0.01
Mean intraclass dispertion	10.5	9.6	2.4	4.1	0.05
Test power (N=100)	47%	75%	62%	79%	19%
N (test power=80%)	219	114	152	102	669

This study was approved by the Scientific Council of the Sports Faculty, and all players gave informed written consent to participate in the screening tests.

5.2.4 Participants

The sample was comprised of 100 female players 19±4 years old, practicing handball at 7±4 years, having a mean body mass of 65±13 kg, a mean height of 164±13 cm and a mean body fat content of 23±5 %, all of which had already reached menarche when evaluated (table 5.3). The sample, in this chapter, will always be grouped by age of playing category and its distribution is presented in figure 5.4. They were further subdivided into 3 groups: those with no previous history of ACL injuries (N=91), those with previous or chronic ACL injury (N=9) and those that in the following year suffered an ACL rupture (N=4).

Table 5.3 Sample characteristics

Category	N	Age	Height (m)	Body mass (kg)	BMI (kg/m^2)	% fat mass	# previous ACL injuries	# following ACL injuries
Starters	8	13.4	1.64	60	22.0	20.4	0	0
Juveniles	25	15.5	1.65	62	22.8	23.4	0	2
Juniors	19	17.6	1.65	63	23.3	23.5	1	0
Seniors	48	22.0	1.66	66	23.7	23.6	8	2

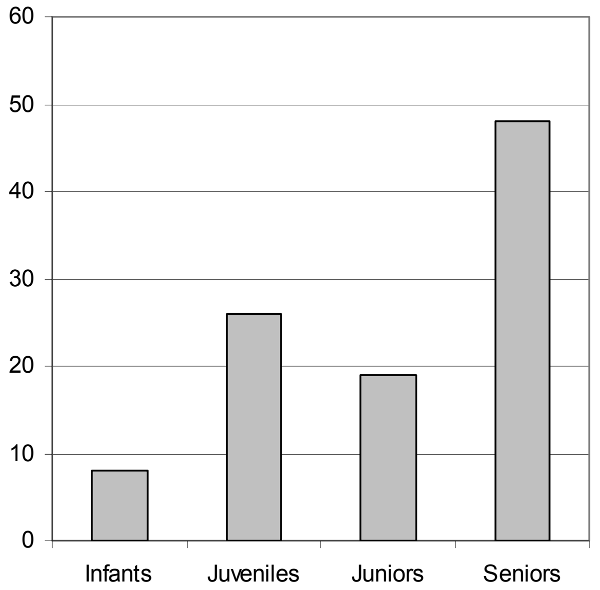


Figure 5.4 Sample distribution by age categories (percentage)

The Infants and Juveniles had a routine of two or three practice sessions a week and the Juniors and Seniors between three and five sessions a week. The typical Juniors and Seniors training sessions lasted between 90 min and 120 min, while for the other categories, in some cases, they lasted only 60 min. The participants were a sample of players from the two national leagues (Seniors), from the national teams (Juniors and Seniors), regional teams (young players) and from the regional championships.

All evaluations were done on Tuesdays or Fridays because the games are at weekends and Wednesdays. This choice assured a period of at least 48 hours between a match and the evaluation.

5.3 Results and discussion

We found just a few published female handball data to compare with our results. Therefore the following discussion includes also comparisons with data from other sports and populations. These should be seen with some reserve.

5.3.1 Players with no previous history of ACL injury

After normalization to body weight the isokinetic evaluation at 90º/s (table 5.4) revealed an $(H:Q)_{conc}$ ratio of 45%±7% what is regarded by some as a low value and an ACL risk factor (Hewett *et al.*, 2001). This ratio is statistically different ($p<3.3\%$, N=82) between nondominant and dominant limbs (46% *vs* 45%) but this asymmetry is insignificant from an epidemiological and biomechanical viewpoint (Hewett *et al.*, 2001). Other nondominant-dominant asymmetries statistically significant (p<5%) but also biomechanically inconsequent (Hewett *et al.*, 2001) regard the extension peak torque and total flexion work.

Table 5.4 Lower limb isokinetic measures at 90º/s for players with intact ACLs

Lower limb	Measure	Movement type	Average	Standard deviation
Dominant (n=82)		$(H:Q)_{conc}$	44.5%	7.2%
	Peak Torque/BW (mm)	Flexion	102	21
		Extension	228	34
	Total Work /BW (mm)	Flexion	327	98
		Extension	627	123
Nondominant (n=82)		$(H:Q)_{conc}$	45.8%	6.1%
	Peak Torque/BW (mm)	Flexion	103	21
		Extension	222	32
	Total Work /BW (mm)	Flexion	338	91
		Extension	610	107

Table 5.5 presents the isokinetic values for each category.

The $(H:Q)_{conc}$ ratio measured for each category relative to the dominant and nondominant lower limb is presented in figure 5.5. For both dominant and nondominant limbs the $(H:Q)_{conc}$ ratio seem to increase along with the playing categories, particularly until the Juniors category, but in any of the cases the differences were not statistically significant (p>5%). The Juniors presented the highest $(H:Q)_{conc}$ ratio (48%±6%), particularly at the nondominant lower limb. Curiously the majority of the ACL ruptures studied in our work (see chapter 3) occurred in the nondominant limb and in players up to 19 years old (Juniors or below).

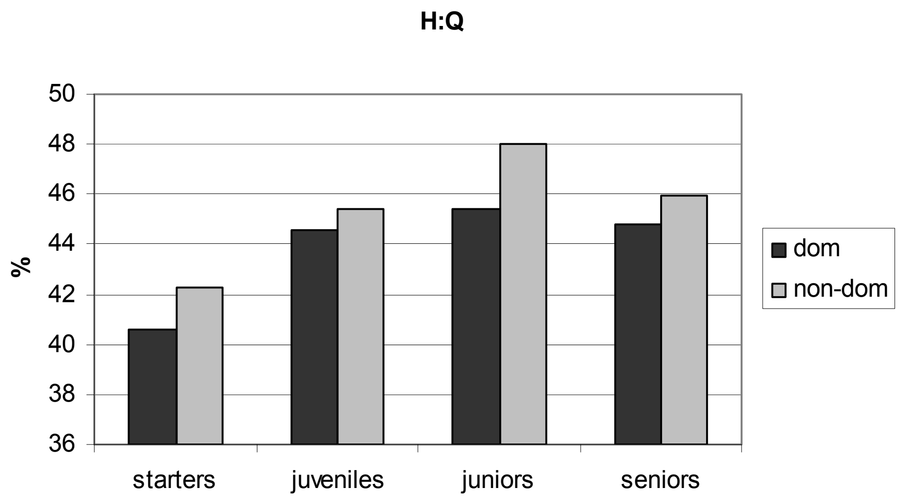


Figure 5.5 $(H:Q)_{conc}$ ratio by dominant (black filled bars) - nondominant (grey filled bars) lower limb and by category.

Table 5.5 Lower limb isokinetic measures at 90º/s for players with intact ACLs, by categories

Age Category	Lower Limb	Measure	Movement type	Average	Standard deviation
Starters (n=7)	Dominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	41%	6 %
			Flexion	85	28
			Extension	204	51
		Total Work /BW (mm)	Flexion	247	115
			Extension	561	155
	Nondominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	42%	8%
			Flexion	83	24
			Extension	194	34
		Total Work /BW (mm)	Flexion	245	99
			Extension	544	107
Juveniles (n=23)	Dominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	45 %	9 %
			Flexion	95	18
			Extension	215	33
		Total Work /BW (mm)	Flexion	292	92
			Extension	567	117
	Nondominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	45 %	6 %
			Flexion	95	15
			Extension	211	29
		Total Work /BW (mm)	Flexion	306	78
			Extension	542	88
Juniors (n=15)	Dominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	45 %	7 %
			Flexion	109	16
			Extension	240	23
		Total Work /BW (mm)	Flexion	361	73
			Extension	672	102
	Nondominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	48 %	6 %
			Flexion	113	15
			Extension	236	25
		Total Work /BW (mm)	Flexion	386	68
			Extension	663	96
Seniors (n=37)	Dominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	45 %	6 %
			Flexion	107	22
			Extension	236	30
		Total Work /BW (mm)	Flexion	349	95
			Extension	658	112
	Nondominant	Peak Torque/BW (mm)	$(H:Q)_{conc}$	46 %	5 %
			Flexion	107	22
			Extension	229	32
		Total Work /BW (mm)	Flexion	357	88
			Extension	643	97

The following figure (figure 5.6) presents the other measured quantities. In no category is there a significant (p>5%) dominant – nondominant limb difference. With regard to category evolution of these quantities they increase with the increasing category, but there are no significant differences (p>21%) between Juniors and Seniors in any case. In all cases there are significant differences between Juveniles and both Juniors (p<1.6%) and Seniors (p<3.1%). This can be interpreted as marking an important increase in these conditional capacities around the transition from Juvenile to Junior that stabilizes onwards.

These findings differ from those from Hewett *et al*. (2004) who, in a sample of female soccer and basketball players from around 11 to 17 years old found no age evolution in the relative peak torque of both hamstrings and quadriceps (it is interesting to notice that they reported an evolution in the male players). Their values, obtained at 300º/s, are not comparable to ours: relative hamstrings peak torque ranges from 30 mm to 33 mm and relative quadriceps peak torque ranges from 59 mm to 66 mm.

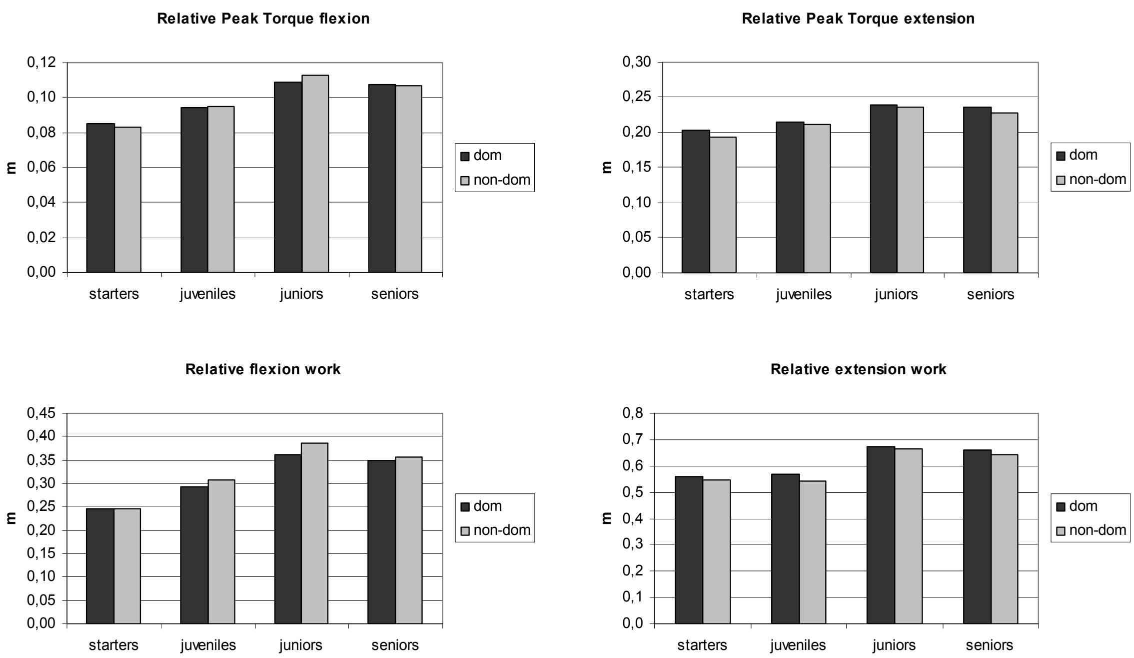


Figure 5.6 a) Relative extension peak torque b) relative flexion peak torque c) relative flexion work d) relative extension work by dominant (black filled bars) *vs.* nondominant (grey filled bars) lower limb and by category.

Analysing the quantities by playing position for the specialization categories (Juniors and Seniors) one can see a remarkable difference between the goalkeepers and the field players (figures 5.7 and 5.8). However the sub-sample is very inhomogeneous with just 4 goalkeepers (of the 11 tested), 8 pivots and 39 field players. We have found no statistically significant differences between any groups, but possibly the very small sample size for the goalkeepers affected the conclusions.

However, a laterality analysis (paired samples) found asymmetric H:Q values for the field players ($p<4.8\%$) and asymmetric extensors peak torque for the pivots ($p<3.5\%$). This only makes us hypothesize the existence of a connection between the specific load of playing and the muscular adaptation. In any case, further work on this should be done with larger sample sizes and with more specific tests.

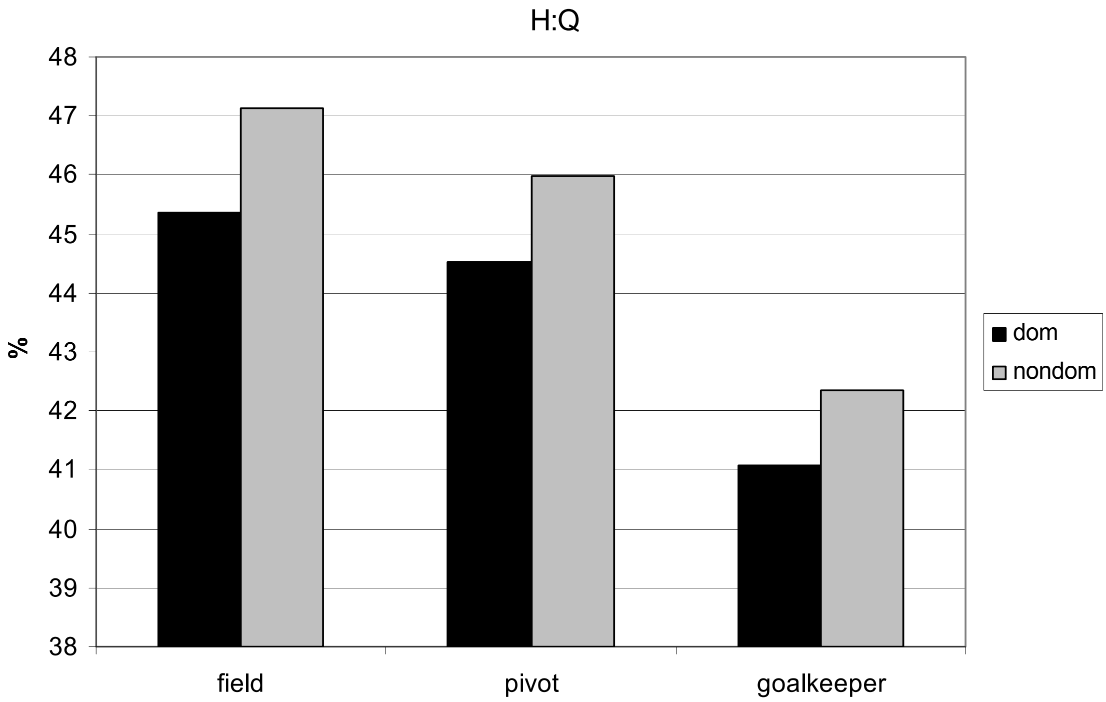


Figure 5.7 $(H:Q)_{conc}$ ratio by dominant (black filled bars) - nondominant (grey filled bars) lower limb and by playing position.

Plotting the measured quantities against the years of practice above 10 years of age (figures 5.9 and 5.10) one can see a steady tendency for an increase in all cases, what is generally not surprising. The possible increase of the H:Q ratio during a players' career (field players in this case) is the most relevant aspect as low H:Q values are considered by some as an ACL risk factor. Although the regressions are statistically significant, the linear correlation coefficients are very poor (large intra-class spreads) so one should be cautious about inferences based on these plots.

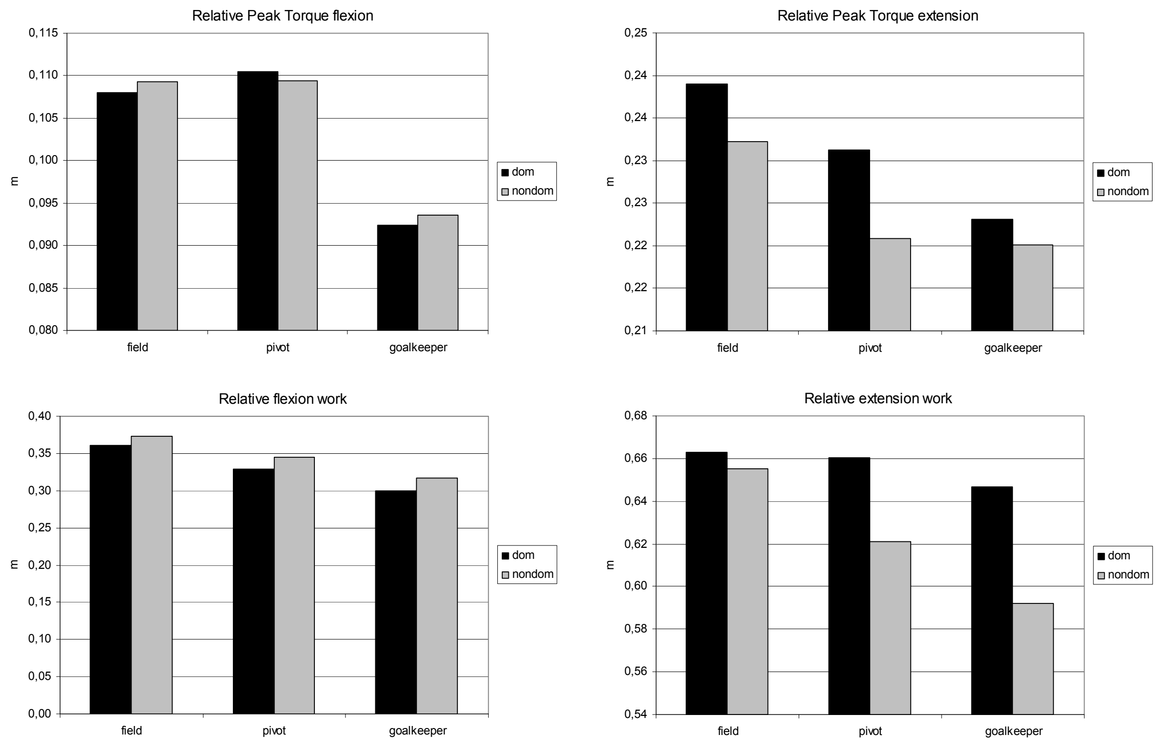


Figure 5.8 a) Relative extension peak torque b) relative flexion peak torque c) relative flexion work d) relative extension work by dominant (black filled bars) vs. nondominant (grey filled bars) lower limb and by playing position.

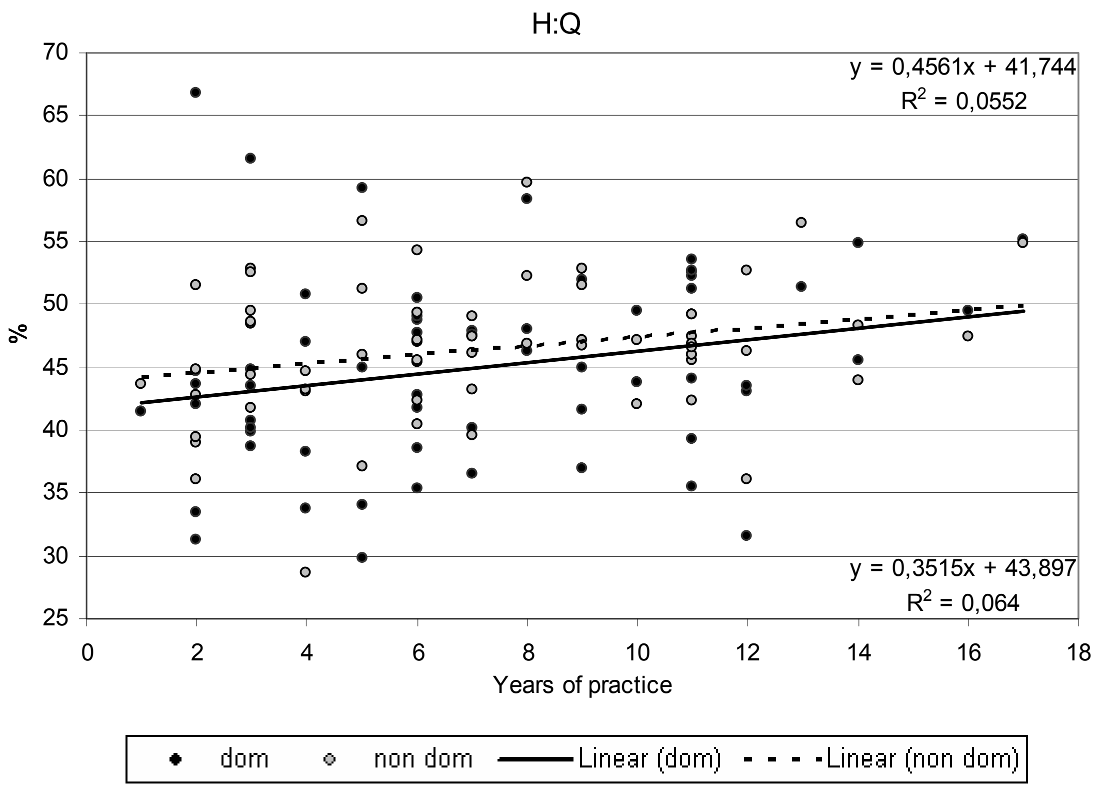


Figure 5.9 $(H:Q)_{conc}$ ratio by dominant (black filled circles) - nondominant (grey filled circles) lower limb of field players vs years of practice.

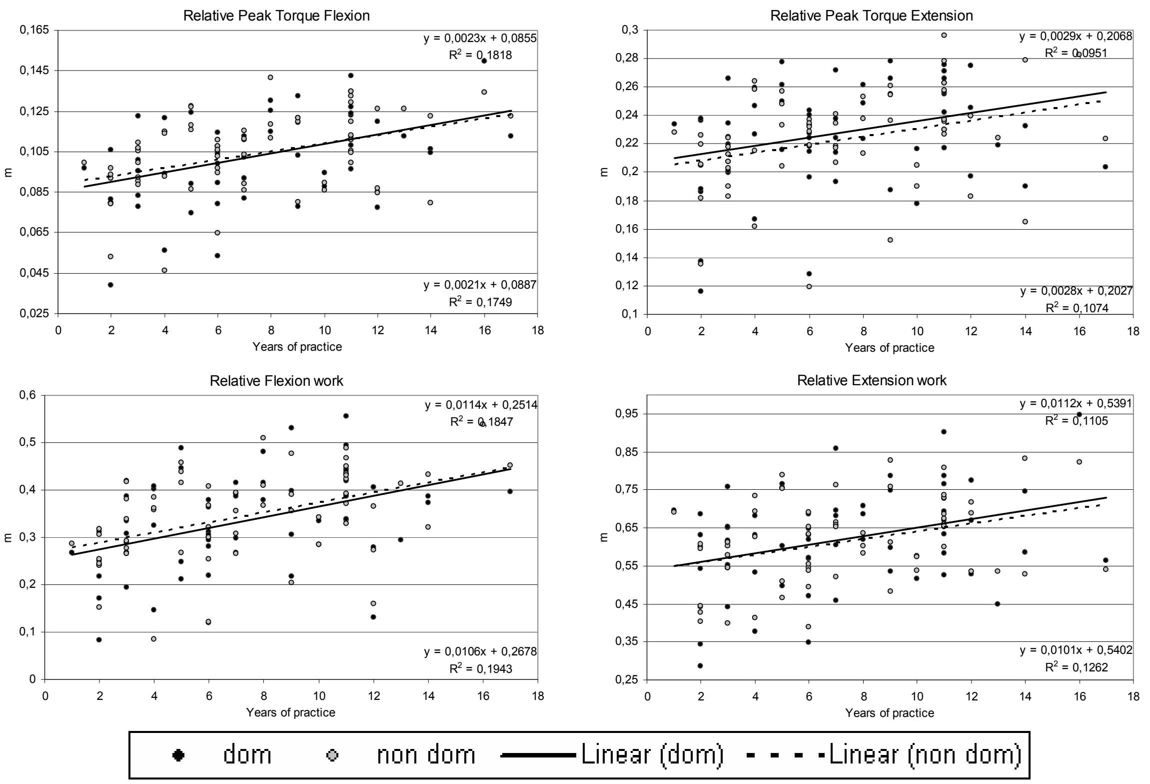


Figure 5.10 a) Relative extension peak torque b) relative flexion peak torque c) relative flexion work d) relative extension work by dominant (black filled circles) vs. nondominant (grey filled circles) lower limb of field players *vs* years of practice.

5.3.2 Players with previous history of ACL injury reconstruction

This small (N=9) group was composed of 8 Senior players (1 goalkeeper and 7 field players) and one Junior player (field player). They had sustained an ACL rupture and subsequent reconstructive surgery, on average, 4 years ago (1 to 7 years). After this time the isokinetic evaluation is summarized in table 5.6.

Table 5.6 Lower limb isokinetic measures at 90º/s for players with previous ACL ruptures

Lower limb	Measure	Movement type	Average	Standard deviation
Healthy limb (n=9)	Peak Torque/BW (mm)	$(H:Q)_{conc}$	49.0%	7.1%
		Flexion	108	15
		Extension	222	27
	Total Work /BW (mm)	Flexion	375	62
		Extension	655	78
Injured limb (n=9)	Peak Torque/BW (mm)	$(H:Q)_{conc}$	52.9%	13.3%
		Flexion	105	17
		Extension	203	27
	Total Work /BW (mm)	Flexion	379	68
		Extension	618	68

There is no significant injured – non-injured leg asymmetry in this population in any of the isokinetically measured parameters. However there is a significant difference (p<1.7%) in the extension peak torque between the injured leg of this group (203 mm) and the nondominant leg of the general population (group with no previous ACL injuries) in the same age category (231 mm). The knee where reconstructive surgery was performed shows, 4 years later on average, about 10% reduction of the extension peak torque, but no reduction in the extension work (there is even a non significant increase). This may indicate a change in the torque-angle curve of the injured limb. The current practice of using segments of the patellar ligament for the ACL reconstruction may, or may not, have some role on this. In any case it seems fair to say that, in our population, players who underwent an ACL reconstruction and returned to handball practice, did not recover completely even after an average of 4 years post surgery. This is in agreement with other published reports (Jarvela *et al.*, 2002; Osteras *et al.*, 1998; Lund-Hanssen *et al.*, 1996).

5.3.3 Players with an ACL injury in the following year

This very small group (N=4) refers to subjects that had been evaluated with their ACLs intact and had suffered an injury a few months later. The very small sample size makes us avoid any kind of global or statistical description of this subgroup. The details of these 4 subjects are summarized in table 5.7.

Table 5.7 Data on *players with an ACL injury in the following year*

	Subject			
	# 63	**# 77**	**# 162**	**# 173**
Category	***Juvenil***	***Senior***	***Senior***	***Juvenil***
Playing categories	Juvenil and Junior	Senior	Senior	Juvenil and Junior
Years of handball practice	11	12	12	3
Age of injury	16	24	21	16
Hand laterally	Right	Right	Right	Right
Injured limb	Nondom (left)	Nondom (left)	Dom (right)	Dom (right)
$H:Q_{Dom}$(%)	48.7	40.3	**46.7**	**61.5**
$H:Q_{Nondom}$(%)	**49.3**	**37.9**	45.9	52.8
Height (cm)	158.5	179.2	158.7	167.9
Body mass (kg)	50.4	89.2	58.2	76
BMI (kg/m^2)	19.9	27.8	23.1	26.9
% fat mass	22	25	29.8	33
Usual playing position	Middle back	Pivot	Middle back and right wing	Pivot and left back
Injured in a match or training	Match	Match	Match	Match
Game phase	Offensive phase (positional attack)	Offensive phase (counter attack)	Defensive recuperation	Offensive phase (counter attack)
Ball possession	Yes	Yes	No	Yes
Contact *vs* noncontact	Contact	Noncontact	Noncontact	Noncontact
Movement	Jump landing (one leg)	Cutting maneuver (one leg)	Rapid deceleration	Cutting maneuver (both legs)
Playing position when injured	Middle back	Left back	Middle field	Left back
Type of floor	Wood	Artificial	Artificial	Artificial
OC Usage	Non	Non	Yes	Non
Menstrual cycle	Irregular	Regular/irregular	Regular	Irregular
Leg length discrepancy	No	NA	No	**Dom**t>Nondom
Clinical evaluation	**Left** metatarsalgia	NA – previous ACL tear suspicion on **left** knee	**Right** ankle overpronation	**Right** patellofemoral syndrome
Knee alignment angle	Dom=6º; Nondom=5º	NA	NA	Dom=2º=Nondom

NA: not applicable

There is no apparent common trend in these 4 subjects except that all suffered the injuries during a match. We have injuries in both the knee with the highest $H:Q_{conc}$ ratio (61.5%) and the knee with the lowest $H:Q_{conc}$ ratio (37.9%). Subject 162 does not present any of the usual non-clinical risk factors. Three of the subjects had eleven or more years of handball practice prior to this event.

The clinical evaluation reveals that three of the subjects had some prior knee or ankle related problems. The excessive foot pronation, as clinically detected in subject 162, has been implicated in the ACL injury risk (Trimble *et al.*, 2002; Allen & Glasoe, 2000; Loudon *et al.*, 1996), causing an increased tibial translation and internal rotation as compensation and also a valgus angulation (Shultz *et al.*, 2007; Trimble *et al.*, 2002). In the fourth case (subject 77) the suspicion of a previous (undetected) ACL injury arose during the arthroscopy. In this case it is known that a previous history of major injuries and/or surgeries to the knee correlates highly with an increased risk of future injuries (Walden *et al.*, 2006).

5.3.4 ACL injury risk factors

Aggregating data from all subgroups enabled a logistic regression, to identify the most promising isokinetic risk factors. In the database we have 4 ACL injuries in the right knee and 7 in the left one among 97 valid cases (a 4% to 6% incidence in about a year). This difference, although not significant (Wilcoxon p>0.7) makes analysis to the right leg data statistically harder.

The logistic regression is the classical tool for accessing risk. Unfortunately its application to our data, on the predictive power of isokinetic measures for the ACL injury risk, is insignificant for both the right (p>0.72) and left limbs (p>0.32).

However a simple odds ratio analysis (table 5.8) shows that an improvement of a single N.m of knee flexor torque improves the chances of *not having* an ACL injury by 1.27, on average. Unfortunately, the 95% confidence interval still contains unity.

Table 5.8 Unit odds ratio non-injury/injury for the left limb (from logistic models)

Term	Odds Ratio	Lower CL	Upper CL
H left	1.271879	0.875739	1.758175
Q left	0.980668	0	1.155192
H:Q left	0.831422	0.63042	0
H left (work)	0.980069	0.937362	1.013653
Q left (work)	0.982679	0.958753	1.001407

A more flexible tool is the neural network (figure 5.11 and table 5.9). We are aware of the low statistical quality of the subjacent data, however the introduction of other statistical tools is of great importance when data is valuable (hard to obtain) and refractive to the *classical* methods. A simple one layer, two node, linear network[4] explains r^2=62.6% of all variation.

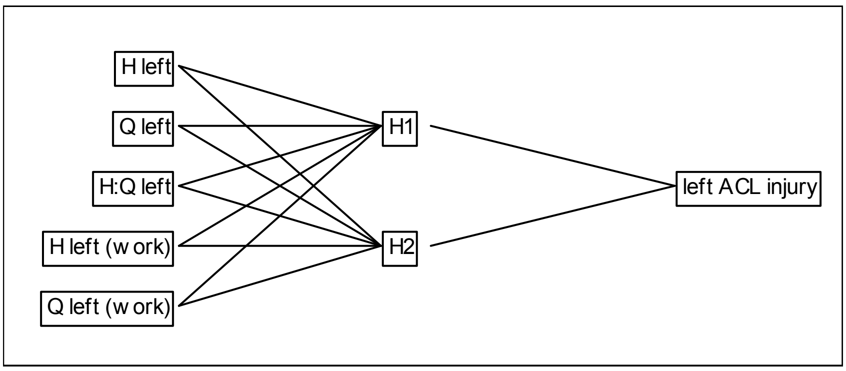


Figure 5.11 Neural network

[4] Overfit penalty: 0.001; 50 tours; 10^{-5} convergence criterium

In a laboratory language, the resulting model is very sensitive[5] and very specific[6], as can be seen in figure 5.12. With this model we predict every ACL injury in our database and obtain only 4 *false-positives*, in 97 cases, with a cut-off point of 0.218 (insert in table 9).

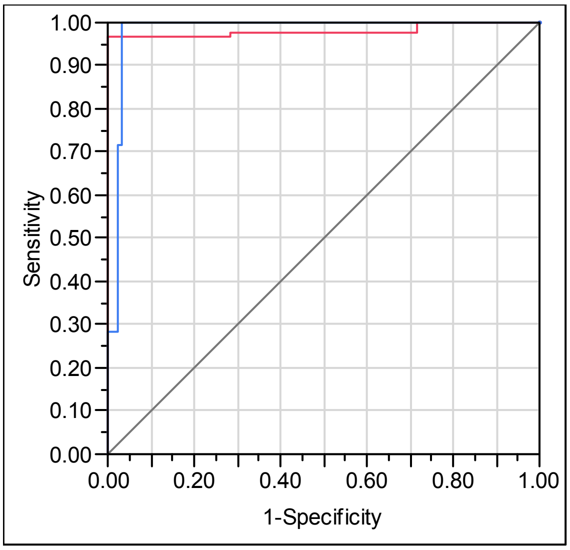
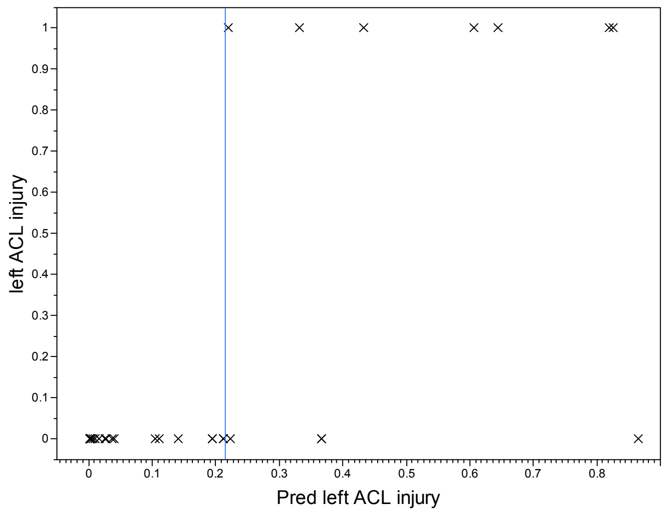


Figure 5.12 ROC diagram for the neuronal network description of ACL injury risk (area=0.981)

Table 5.9 Neuronal network adjusted model

L-R ChiSquare	DF	Prob > ChiSquare	Missclassification Rate	RSquare
31.48164	14	0.00474	0.041	0.62606

	Objective
-LogLikelihood	9.4019029678
Penalty	1.3643924785
Total	10.766295446
N	97
Parameter	**Estimate**
H1:Intercept	-4.955660456
H2:Intercept	0.2413960782
H1:H left	-0.685322629
H1:Q left	-5.162378267
H1:H:Q left	5.8522957347
H1:H left (work)	-16.97258463
H1:Q left (work)	10.2054228
H2:H left	7.2026579015
H2:Q left	0.3991551212
H2:H:Q left	-9.761767752
H2:H left (work)	14.873801231
H2:Q left (work)	-10.55945559
left ACL injury:Intercept	-3.103755409
left ACL injury:H1	14.502469956
left ACL injury:H2	13.992989495



This is a maximum-likelihood fit of a model to data. To really test the potential of the tool we also did a fit to only half the available data and compared its predictions with the other half of the data. We obtained very similar results: 4 false negatives and no (0) false positives, a good result based only on a few isokinetic measures.

[5] low type I error rate – false positives
[6] low type II error rate – false negatives

The main information we can extract from this model is the importance of the hamstrings. Above a certain level of extensor's torque (figure 5.13) the ACL injury risk varies symmetrically to the flexors peak torque. At the lower quadrant, an increase of the peak torques, even maintaining constant the H:Q relation, is an increase on the risk.

If we look at the H:Q ratio and the H peak torque (figure 5.14), we don't observe the iso-risk lines as being simply horizontal. Our model and it's results probably are not as easy to interpret as the classical H:Q torque model but the two couldn't be more distinct.

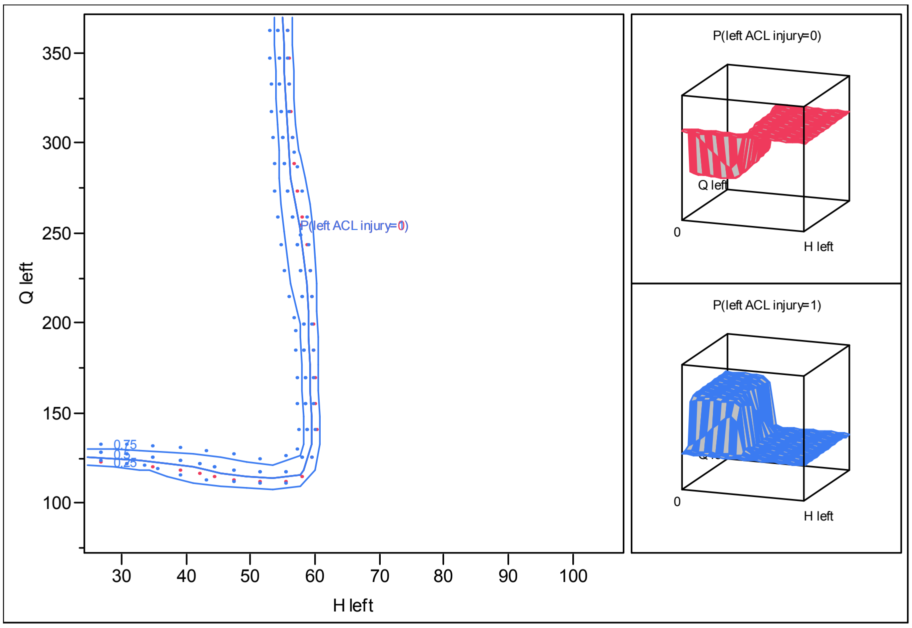


Figure 5.13 Three iso-risk contours (25%, 50%, 75%) according to the neural network model

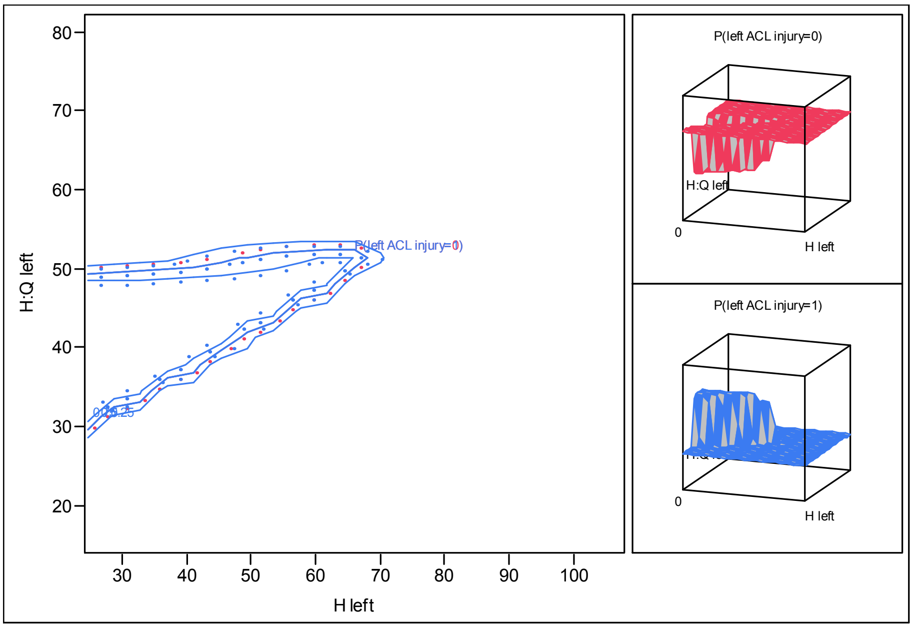


Figure 5.14 Three iso-risk contours (25%, 50%, 75%) according to the neural network model

5.4 Conclusions

We have characterized the Portuguese female handball population on some of its isokinetic parameters. We have established some slight associations between the handball career evolution, some isokinetic quantities and the ACL injury risk.

Our results, although based on weak data, contradict the H:Q risk hypothesis. According to our model, the hamstrings peak torque alone is a better indicator of ACL injury risk.

We concluded that the ACL injury (even with surgical reconstruction) may take a long time to recover, at least the isokinetic muscle function parameters.

5.5 Perspectives

It is very difficult to do a prospective study to identify ACL injury risk factor due to its low incidence. The need to continuously measure all the putative risk factors of a sample large enough and for a long enough period to generate a few dozen ACL injuries has avoided any definitive study on the subject.

Isokinetic measurements in general and the $H{:}Q_{conc}$ ratio in particular are used by some and questioned by others as knee injury risk indicators (Ostenberg & Roos, 2000). What seems undisputed is that the surgical technique used for ACL reconstruction affects primarily the extensors (Q in the case of a BTB) or the flexors (H in the case of ST or STG) and so alters the H.Q ratio in a specific way (Moisala *et al.*, 2007). So it is natural that these isokinetic measures are used as indicators of injury recovery (Li *et al.*, 1996) although this is also disputed (Lephart *et al.*, 1992). Another recovery indicator, probably more sport specific, is the hop test (Jarvela *et al.*, 2002; Rudolph *et al.*, 2000).

However, on the whole, the studies on the injury mechanism using simulation models (Shin *et al.*, 2007; Frey *et al.*, 2006) or direct measurements (Fleming & Beynnon, 2004) may be more promising than those epidemiologically based.

5.6 References

Aagaard, P., Simonsen, E. B., Magnusson, S. P., Larsson, B. & Dyhre-Poulsen, P. (1998). A new concept for isokinetic hamstring: quadriceps muscle strength ratio. *Am J Sports Med, 26*(2), 231-237.

Allen, M. K. & Glasoe, W. M. (2000). Metrecom Measurement of Navicular Drop in Subjects with Anterior Cruciate Ligament Injury. *J Athl Train, 35*(4), 403-406.

Beynnon, B. D. & Fleming, B. C. (1998). Anterior cruciate ligament strain in-vivo: a review of previous work. *J Biomech, 31*(6), 519-525.

Boden, B. P., Dean, G. S., Feagin, J. A., Jr. & Garrett, W. E., Jr. (2000). Mechanisms of anterior cruciate ligament injury. *Orthopedics, 23*(6), 573-578.

Coombs, R. & Garbutt, G. (2002). Developments in the use of the Hamstring/Quadriceps ratio for the assessment of muscle balance. *Journal of Sports Science and Medicine, 1*, 7.

Dvir, Z., Eger, G., Halperin, N. & Shklar, A. (1989). Thigh muscle activity and anterior cruciate ligament insufficiency *Clinical Biomechanics, 4*(2), 87-91.

Ekstrand, J. & Gillquist, J. (1983). Soccer injuries and their mechanisms: a prospective study. *Med Sci Sports Exerc, 15*(3), 267-270.

Ekstrand, J., Karlsson, J. & Hodson, A. (Eds.). (2003). *Football Medicine* (1st ed.): Martin Dunitz.

Fleming, B. C. (2003). Biomechanics of the anterior cruciate ligament. *J Orthop Sports Phys Ther, 33*(8), A13-15.

Fleming, B. C. & Beynnon, B. D. (2004). In vivo measurement of ligament/tendon strains and forces: a review. *Ann Biomed Eng, 32*(3), 318-328.

Frey, M., Riener, R., Michas, C., Regenfelder, F. & Burgkart, R. (2006). Elastic properties of an intact and ACL-ruptured knee joint: measurement, mathematical modelling, and haptic rendering. *J Biomech, 39*(8), 1371-1382.

Heiser, T. M., Weber, J., Sullivan, G., Clare, P. & Jacobs, R. R. (1984). Prophylaxis and management of hamstring muscle injuries in intercollegiate football players. *Am J Sports Med, 12*(5), 368-370.

Hewett, T. E., Myer, G. D. & Ford, K. R. (2001). Prevention of anterior cruciate ligament injuries. *Curr Womens Health Rep, 1*(3), 218-224.

Hewett, T. E., Myer, G. D. & Ford, K. R. (2004). Decrease in neuromuscular control about the knee with maturation in female athletes. *J Bone Joint Surg Am, 86-A*(8), 1601-1608.

Hewett, T. E., Myer, G. D. & Ford, K. R. (2006). Anterior cruciate ligament injuries in female athletes: Part 1, mechanisms and risk factors. *Am J Sports Med, 34*(2), 299-311.

Hewett, T. E., Myer, G. D., Ford, K. R., Heidt, R. S., Jr., Colosimo, A. J., McLean, S. G., *et al.* (2005). Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med, 33*(4), 492-501.

Jarvela, T., Kannus, P., Latvala, K. & Jarvinen, M. (2002). Simple measurements in assessing muscle performance after an ACL reconstruction. *Int J Sports Med, 23*(3), 196-201.

Kessler, M. A., Behrend, H., Henz, S., Stutz, G., Rukavina, A. & Kuster, M. S. (2008). Function, osteoarthritis and activity after ACL-rupture: 11 years follow-up results of conservative versus reconstructive treatment. *Knee Surg Sports Traumatol Arthrosc*.

Knapik, J. J., Bauman, C. L., Jones, B. H., Harris, J. M. & Vaughan, L. (1991). Preseason strength and flexibility imbalances associated with athletic injuries in female collegiate athletes. *Am J Sports Med, 19*(1), 76-81.

Lephart, S. M., Perrin, D. H., Fu, F. H., Gieck, J. H., McCue, F. C. & Irrgang, J. J. (1992). Relationship between Selected Physical Characteristics and Functional Capacity

in the Anterior Cruciate Ligament-Insufficient Athlete. *J Orthop Sports Phys Ther, 16*(4), 174-181.
Li, R. C., Maffulli, N., Hsu, Y. C. & Chan, K. M. (1996). Isokinetic strength of the quadriceps and hamstrings and functional ability of anterior cruciate deficient knees in recreational athletes. *Br J Sports Med, 30*(2), 161-164.
Loudon, J. K., Jenkins, W. & Loudon, K. L. (1996). The relationship between static posture and ACL injury in female athletes. *J Orthop Sports Phys Ther, 24*(2), 91-97.
Lund-Hanssen, H., Gannon, J., Engebretsen, L., Holen, K. & Hammer, S. (1996). Isokinetic muscle performance in healthy female handball players and players with a unilateral anterior cruciate ligament reconstruction. *Scand J Med Sci Sports, 6*(3), 172-175.
Mesfar, W. & Shirazi-Adl, A. (2005). Biomechanics of the knee joint in flexion under various quadriceps forces. *Knee, 12*(6), 424-434.
Moisala, A. S., Jarvela, T., Kannus, P. & Jarvinen, M. (2007). Muscle strength evaluations after ACL reconstruction. *Int J Sports Med, 28*(10), 868-872.
Nosse, L. (1982). Assessement of selected reports on the strengths relationship of the knee musculature. *Journal of Orthopaedics and Sports Physical Therapy, 4*, 78-85.
Ostenberg, A. & Roos, H. (2000). Injury risk factors in female European football. A prospective study of 123 players during one season. *Scand J Med Sci Sports, 10*(5), 279-285.
Osteras, H., Augestad, L. B. & Tondel, S. (1998). Isokinetic muscle strength after anterior cruciate ligament reconstruction. *Scand J Med Sci Sports, 8*(5 Pt 1), 279-282.
Pandy, M. G. & Shelburne, K. B. (1997). Dependence of cruciate-ligament loading on muscle forces and external load. *J Biomech, 30*(10), 1015-1024.
Proske, U., Morgan, D. L., Brockett, C. L. & Percival, P. (2004). Identifying athletes at risk of hamstring strains and how to protect them. *Clin Exp Pharmacol Physiol, 31*(8), 546-550.
Rosene, J. M., Fogarty, T. D. & Mahaffey, B. L. (2001). Isokinetic Hamstrings:Quadriceps Ratios in Intercollegiate Athletes. *J Athl Train, 36*(4), 378-383.
Rudolph, K. S., Axe, M. J. & Snyder-Mackler, L. (2000). Dynamic stability after ACL injury: who can hop? *Knee Surg Sports Traumatol Arthrosc, 8*(5), 262-269.
Sell, T. C., Ferris, C. M., Abt, J. P., Tsai, Y. S., Myers, J. B., Fu, F. H.*, et al.* (2006). The effect of direction and reaction on the neuromuscular and biomechanical characteristics of the knee during tasks that simulate the noncontact anterior cruciate ligament injury mechanism. *Am J Sports Med, 34*(1), 43-54.
Shin, C. S., Chaudhari, A. M. & Andriacchi, T. P. (2007). The influence of deceleration forces on ACL strain during single-leg landing: a simulation study. *J Biomech, 40*(5), 1145-1152.
Shultz, S. J., Nguyen, A. & Beynnon, B. (2007). Anatomical factors in ACL injury risk. In T. E. Hewett, S. J. Shultz & L. Y. Griffin (Eds.), *Undertanding and Preventiing Noncontact ACL injuries* (pp. 239-258). Champaign, IL: American Orthopeadic Society for Sports Medicine
Simonsen, E. B., Magnusson, S. P., Bencke, J., Naesborg, H., Havkrog, M., Ebstrup, J. F.*, et al.* (2000). Can the hamstring muscles protect the anterior cruciate ligament during a side-cutting maneuver? *Scand J Med Sci Sports, 10*(2), 78-84.
Soderman, K., Alfredson, H., Pietila, T. & Werner, S. (2001). Risk factors for leg injuries in female soccer players: a prospective investigation during one out-door season. *Knee Surg Sports Traumatol Arthrosc, 9*(5), 313-321.
Solomonow, M. & Krogsgaard, M. (2001). Sensorimotor control of knee stability. A review. *Scand J Med Sci Sports, 11*(2), 64-80.
Steindler, A. (1955). *Kinesiology of the Human Body under Normal and Pathological Conditions*. Springfield, Illinois: Charles C. Thomas Publiser.
Trimble, M. H., Bishop, M. D., Buckley, B. D., Fields, L. C. & Rozea, G. D. (2002). The relationship between clinical measurements of lower extremity posture and tibial translation. *Clin Biomech (Bristol, Avon), 17*(4), 286-290.

Walden, M., Hagglund, M. & Ekstrand, J. (2006). High risk of new knee injury in elite footballers with previous anterior cruciate ligament injury. *Br J Sports Med, 40*(2), 158-162; discussion 158-162.

Chapter 6

Sex Hormones, Muscle Strength and Anterior Knee Laxity

6.1 Introduction

The ACL rupture is the most worrying injury in the Portuguese female handball population, affecting mostly young players, as shown in chapter 3. We did not study the ACL incidence among Portuguese male handball players, although the risk of ACL rupture was found, in other populations, to be 5 times higher in female handball players than in male players (Myklebust *et al.*, 1998). As reviewed in chapter 2 researchers have conjectured that, the female hormonal environment is one of the possible contributing factors for the sex rate disparity of these injuries.

While receptors specific to estrogen, progesterone, relaxin and testosterone have been isolated on the female ACL, *in vitro* studies have only investigated the effect of estradiol and progesterone in ACL fibroblasts proliferation and collagen synthesis. The influence of estrogen in mechanical properties of the ACL have also been investigated. The results have been conflicting, and further studies are required to known how much of the collagen metabolism and therefore of mechanic properties of the ACL are hormone-dependent or hormone-mediated.

In recent years, several *in vivo* experiments have been employed to explore the influence of female hormonal factors in knee stability, whether affecting the ACL mechanical properties or neuromuscular control. However, contradictory results have emerged and no clear consensus has been reached (see chapter 2). While only a few studies showed knee laxity (Shultz *et al.*, 2004; Deie *et al.*, 2002; Heitz *et al.*, 1999) and neuromuscular control changes (Friden *et al.*, 2006) throughout the menstrual cycle phases, there is much we still do not know about the potential hormonal effect on knee joint stability and therefore in the ACL injury risk.

Given that the main effect of the OCs (containing synthetic derivatives of the female sex hormones) is to promote a stable hormonal environment in order to ovarian suppression (figure 6.1), some speculation arose that they could have a prophylactic effect on the ACL injury risk. The research into OCs, knee laxity and neuromuscular control is sparse and the findings are inconsistent (Cammarata & Dhaher, 2008; Burrows & Peters, 2007; Martineau *et al.*, 2004; Pokorny *et al.*, 2000). In addition, a significant increase in the number of athletes using OCs is expected irrespectively of the sport type, whether it is for menstrual cycle control, dysmenorrheal or contraception motives. In the Portuguese handball population, we found that 20 to 40% of handball players self-reported to be using OCs, which was dependent on the player’s age and studied season (chapter 3).

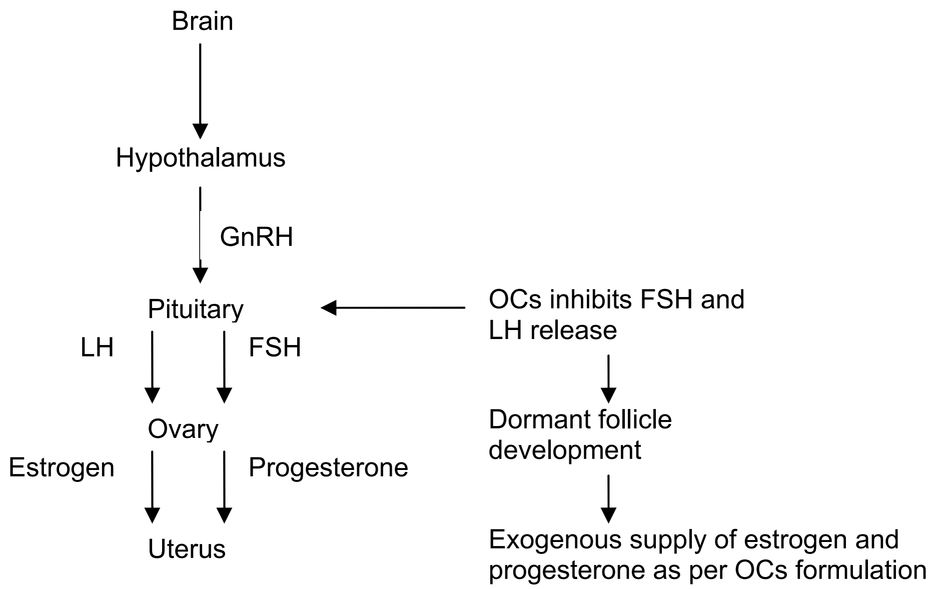


Figure 6.1 The OCP and control of endogenous sex hormones, from Burrows and Peters (2007) p558.

Studies investigating the impact of high intensity exercise on endocrine equilibrium, which regulates the female reproductive function, have identified several menstrual associated disturbances (including disruption of follicular development, luteal phase deficiency [LPD], anovulation and oligo/amenorrhea) (chapter 2). Consequently, impaired production of involved hormones is a common finding, which would expose the body and therefore ligament to a different hormonal environment/menstrual phase from *normal* cycles in *non-athletic* females. Thus, it is also important to explore the hormonal clue as a possible ACL risk factor in subjects exposed to typical sport related stress factors (physical or psychological). How mechanical properties of the ACL are hormone-dependent in a systematic loaded joint, as it occurs in handball practice, has not yet been explored. Also, young female athletes should be included as are those with a higher risk of ACL rupture.

All these evidences on the possible effects of sex hormones on muscular strength and ligamentar laxity have been demonstrated with high hormonal doses and with long interaction delays and half-lifes. Here we will try to see the effects of very small variations, both on a long term scale (through the OC usage) and on a *very* short scale, mainly the result of a resonance[1] effect with the monthly hormonal variations in non-OC users.

Study purposes: to investigate the relation between sex hormonal levels, jump height performance and knee laxity with both usage and non-usage of OCs in female handball players.

[1] Any existing recurrent effect must manifest and recess in exactly the cycle length (28 days).

6.2. Preliminary study

A preliminary study was employed to determine the sample size and for testing some methodological procedures for menstrual cycle monitoring, such as body basal temperature (BBT) method. This study had a prospective experimental design, and was undertaken between January and June 2005.

For this purpose, students from the Sports Faculty completed a questionnaire about injuries and menstrual cycle characteristics (described in chapter 3). Data from the screening questionnaire was then used to identify students without previous knee injuries and to assess some vital and sports related information (e.g. age, menstrual cycle irregularity, menarcheal status, OC status, sports participation). All measurements were done in our faculty whenever the students schedule allowed it, between practical (physical) or theoretical lessons.

Prior to the study, the participants were informed about the study goals and testing procedures, and gave informed written consent to participate in the screening tests.

This study was approved by the Scientific Council of the Sports Faculty.

Participants. Forty young females sport students from our faculty, were enrolled in this study with, according to the inclusion criteria (e.g. no previous history of knee injuries). Two subgroups were constituted: using OCs (n=20) uninterruptedly for 6 months prior to the testing sessions, and not subjected to hormonal therapy (n=20) and being eumenorrheic for the past 6 months. During the testing period the student had at least 10 hours a week of practice lessons in different sports (including handball).

The students were asked to perform daily anterior knee laxity measurements' (excluding weekends). During the evaluation period, one student (Olympic swimming athlete) got an ACL injury in a practical lesson of athletics, being therefore excluded from the analysis.

Procedures. The students were instructed to immediately start the measurements whenever they reported consistent information about the onset of previous menses or if they were OC users. The non-OC users were instructed to start recording the body basal temperature immediately on a daily basis, independent of the day of starting the knee laxity measurements'. For this purpose they were given a Digital Basal Thermometer[2] for oral use, a BBT chart and written instructions about all the procedures (see appendixes V and VI). Ovulation was mainly calculated from the thermal shift rather than from the onset

[2] Fairhaven Health, LLC

of the next menses. Anterior knee laxity was measured at 15 lbf, 20 lbf and 30lbf with a Knee arthrometer[3], as many times as possible during one menstrual cycle. The adopted procedures to evaluate the anterior knee laxity were described in chapter 5.

The BBT charts were analysed by a Gynaecologist (co-supervisor of this study), qualified monitors/instructors of natural family planning methods[4] and ourselves. Because of academic time interruption, some students were followed during two menstrual cycles.

The knee laxity measurements were all performed by the same examiner. The intratester reliability for the examiner was established and presented in chapter 5.

Results. Thirty-five BBT charts were obtained, corresponding to 37 cycles, being classified as followed: 32% not ovulating cycles, 27% ovulate cycles and 40% inaccurate and therefore excluded from analyses. The BBT tracking demands a great commitment and to be meticulous with the procedures. Several students forgot to take their temperature on quite a few days.

Temporal series diverge in their extension and amplitude. As each basic temporal unit is the menstrual cycle, all time series were rescaled to 1, with it beginning in day 1 of the menstrual cycle and the last day in the day before the onset of next menses. Based on the estimated day of ovulation, which was arbitrarily positioned in the middle of the cycle. Two other temporal rescaling data was taken: from day 1 until ovulation and from ovulation until the last day of the cycle. This procedure allowed us to deal with shortened luteal phases (LPD; ≤ 10 days) as well as with long menstrual cycles (oligomenorrheic). Still we must be cautious with the analyses because the estimated ovulation day based on temperature nadir could have a margin of -1 to 4 days (as reviewed in chapter 2, p 68).This synchronization of measurements is essential for the analyses of time series and temporal aggregation. The laxity variation was also normalized to permit the series aggregated treatment.

A total of 3545 anterior knee laxity tests on 24 students (initially 40 students), revealed the complexity of the task: dealing with the huge amounts of missing or incomplete data and find a pattern in figure 6.2. Even discarding the 519 cases (15%) where the players provided incomplete or doubtful data, the plots do not change much.

[3] KT-1000 ™ Knee arthrometer (MEDmetric® Corp; San Diego, CA, USA)
[4] Asociación Española de Profesores de Planificación Familiar Natural

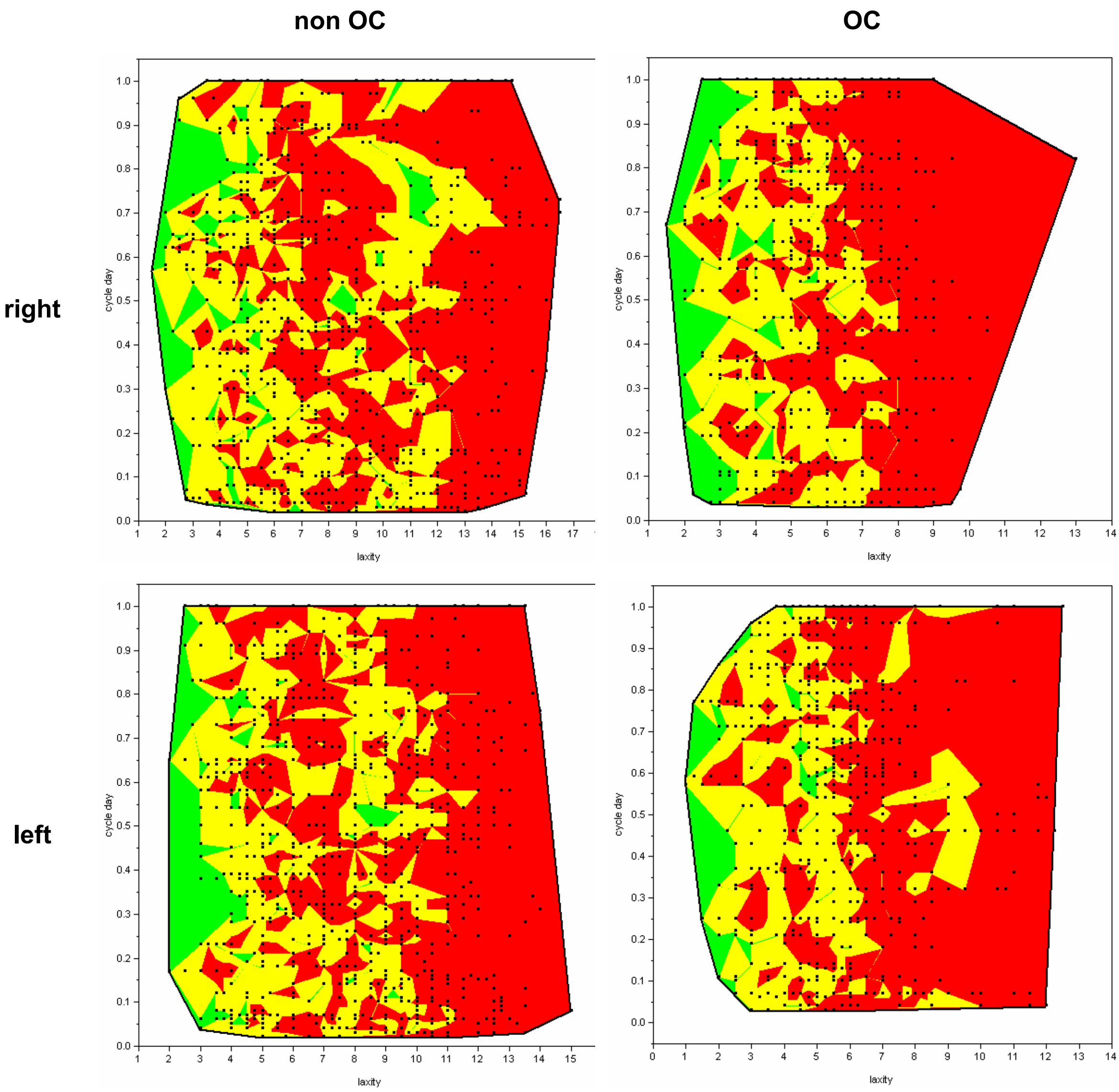


Figure 6.2 Cycle day (normalized), load (middle gray=15lbf, light gray=20lbf, dark gray=30lbf) and anterior knee laxity (mm)

At this stage we just want to design and dimension an experiment. Just plotting the highest load (30lbf) cyclic variations (figure 6.3) it is clear that the red curve (right knee, non-OC) is always the highest, followed by the blue (left, non-OC), the green (right, OC) and finally the orange (left, OC), almost always the lowest one.

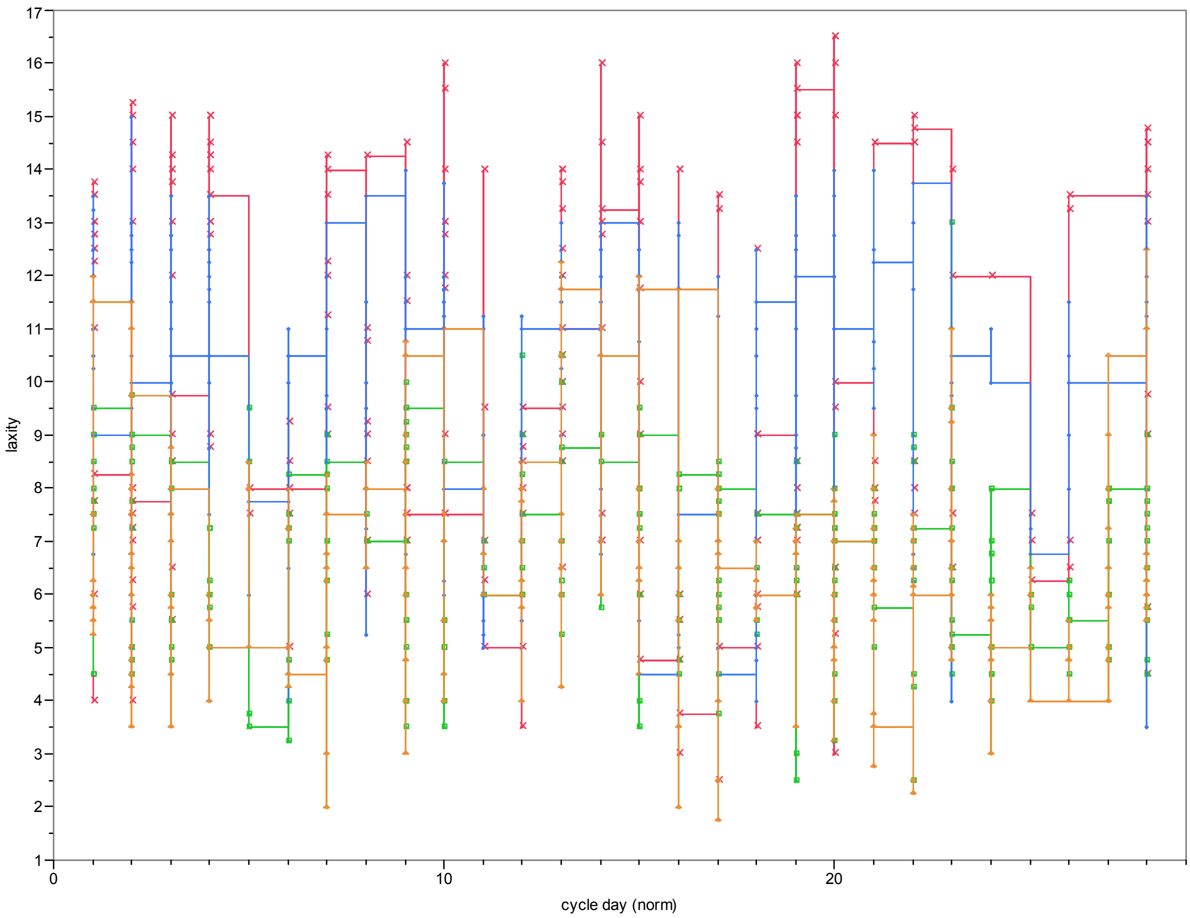


Figure 6.3 Anterior knee laxity (mm) evolution along the menstrual cycle (red: right knee, non-OC; blue: left, non-OC; green: right, OC; orange: left, OC)

The vital univariate statistics are in table 6.1. It is interesting to note that the laxity distributions for the OC group are identical and normal or almost normal, while for the non-OC group they are far from normality (figure 6.4). Curiously using a much larger sample with handball players (chapter 4) we were unable to find knee laxity differences between OC users and non OC users. We must note that these two groups of students are more similar in age and practice exposure.

Table 6.1 Basic univariate statistics – preliminary study

Knee	non OC	OC
right	μ_1=10.2±1.7 (N=248) median=9.5;IQR=1.0	μ_3=6.7±3.5 (N=246) median=7.0;IQR=4.0
left	μ_2=9.5±2.1 (N=257) median=10.0;IQR=1.0	μ_4=6.5±2.7 (N=253) median=6.5;IQR=1.8
	ANOVA p<0.0001	HSD $\mu_1>\mu_2>\mu_3\approx\mu_4$

A simple sample size calculation, based on a desired power of 80%, a desired resolution of 0.2 mm and a typical dispersion of 4mm, indicates the need to perform 12540 knee laxity tests.

Non-OC right knee

OC right knee

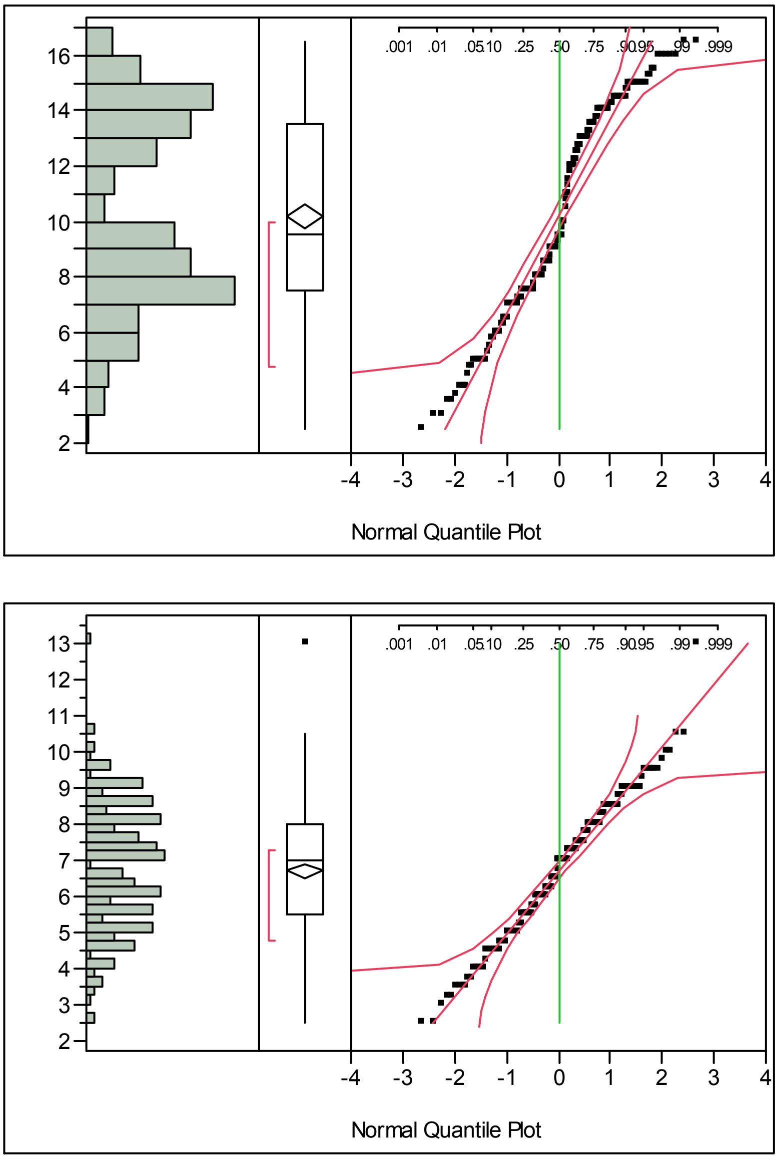


Figure 6.4 Anterior knee laxity (mm) distribution – preliminary study

As many authors, we were unable to find any appropriate model for the laxity, dependent on the cycle day, OC usage and laterality. Even a complex, 10 node – 81 degrees of freedom, neural network can not do any better than the results plotted in figure 6.5 (r^2=0.38). Clearly if there is any association between the hormonal levels and laxity, as suggested by figure 6.3 and table 6.1, the hormonal levels cannot be deduced by the day of cycle and OC status. We need to sample the real serum hormonal content.

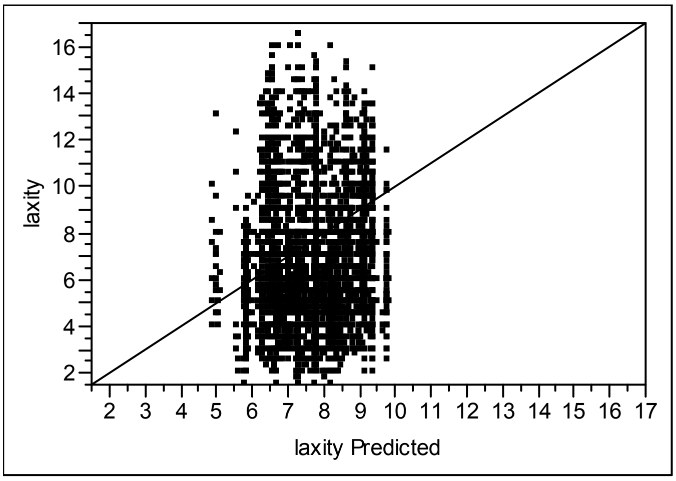


Figure 6.5 Anterior knee laxity (mm) actual *vs*. predicted – neural network, preliminary study

6.3 Methods

Experimental design

A prospective experimental design was employed. This study was conducted during the 2005-06 and 2006-07 sport seasons.

The participating female handball players were all from the Porto's Regional Association (6 clubs). Female handball players' eumennorrheic and currently ingesting OCs were included in the study, being tracked on one complete menstrual cycle during the competitive period.

Personal information from each player, about their handball career, actual handball training process and competition, previous injuries, menstrual cycle characteristics and smoking behaviour, was obtained using the same questionnaire as previously presented in chapter 3. Players with previous knee injuries were not included in the study. Still, when subjective knee joint complaints were self-reported, the player was examined by an orthopaedic surgeon in order to verify the integrity of their knees. Some anthropometric measures were performed (including height, body mass, BMI and fat mass content) by the same examiner, using standard equipment, at the beginning of each tracked period.

All players were familiarized with the instruments, material and procedures of testing sessions (knee laxity measurement, jump tests and menstrual cycle tracking) prior to the starting of data collection.

During each menstrual cycle, the knee laxity and jump measurements were performed with the same periodicity as training sessions and matches[5] (usually 15 to 50 min. before starting). For this purpose, the examiner routinely transported the necessary equipment for each field place. The serum hormonal content was monitored by two types of proceedings, daily in a small sample of players (n=5) and important sampling in 37 players. For collecting blood, the players went voluntary to a clinic analysis laboratory[6].

Prior to being studied the players were informed about the study goals and testing procedure, and all players (or parents, if the player was underage) gave informed written consent to participate in the screening tests. The directors of the enrolled clubs and coaches were also informed of the study goals and procedures, and their collaboration was very important to create logistic conditions to perform the measurements.

This study was approved by the Scientific Council of the Sports Faculty, although the study was not submitted to the Porto University Ethical Committee because of its recent creation.

6.3.1 Participants

Forty-two national level female handball players, from juvenile (n=7), juniors (n=13) and senior (n=22) categories, were accompanied during one menstrual cycle, being divided into two groups. One group of players (N=17) was submitted to OC treatment uninterruptedly for 6 months prior to the testing sessions, and the other group of players (N=25) was not submitted to any hormonal therapy for the past 6 months or had a recent amenorrhea history.

The general inclusion criteria was: to have reached menarche for more than 2 years; no history of pregnancy; to be a non smoker; no prior history of serious knee joint injury or surgery; and no medical condition affecting the connective tissue.

Three players were excluded from analysis (not accounted in the size of the groups) because of inaccuracy in self-monitoring the menstrual cycle and several absences to training sessions because of academic demands.

The average age of all players was 20 ± 3 years old (range = 15-27 years). On average the participants had played handball for about 9 years (± 3.4). Data regarding average age, age of menarche, height, body mass and BMI and body fat content for each group are found in table 6.2. On average the players started to use OCs at 18 ± 2.5 years

[5] Whenever the participants volunteered, more testing sessions were performed.

[6] Endoclab.

(range = 14-23) and for an average period of 3 ± 2.2 years. Among the players not using OCs, only one player reported to have been subjected to hormonal therapy in the past because of painful menstruation (dysmenorrhea).

Table 6.2 Sample characteristics'

Groups	N	Age μ±SD	Age of Menarche μ±SD	Years of handball practice μ±SD	Height (m)	Body mass (kg)	BMI (kg/m^2)	% fat mass
OC users	17	21 ± 3.0	12.3 ± 0.8	10.3 ± 2.2	1.67 ± 0.1	65.8 ± 10.0	79 ± 8.4	26.9 ± 5.5
Non-OC users	25	19 ± 3.5	12.0 ± 1.3	7.8 ± 3.8	1.66 ± 0.7	64.6 ± 11.4	78 ± 12	26.6 ± 5.2
Total	42	20 ± 3.0	12.1 ± 1.1	8.8 ± 3.4	1.66 ± 0.7	65.1 ± 10.8	78 ± 11	26.7 ± 5.2

Almost all players were using monophasic OCs (n= 15), being submitted daily to a similar dose of combined synthetic estrogens and progestins (21 days followed by 7-days hormone-free period to allow for withdrawal bleeding) as described in table 6.3. Just one player was subjected to a 24-day oral contraceptive regime, containing the lowest dose of ethinyl estradiol (15 µg) combined with gestoden (75 µg), followed by 4 days of placebo pills (without active substances). A combiphasic OC was used only by one player, with an estrogen-dominant phase (7 days: 25 µg desogestrel/40 µg ethinyl estradiol) followed by a progestogen-dominant phase (15 days: 125 µg desogestrel/30 µg ethinyl estradiol).

Table 6.3 Type of OC pills used by the OC group

Type of Pill	N	estrogens	progestins	Dosage and administration
Monophasic (e.g., Yasmim, Harmonet, Diane 35, Mercilon, Minulet, gynera, Microgest)	4	30 µg ethinyl estradiol	3 mg drospirenone	21 active pills
	4	20 µg ethinyl estradiol	75 µg gestoden	21 active pills
	2	35 µg ethinyl estradiol	2mg cyproterone acetate	21 active pills
	1	20 µg ethinyl estradiol	150 µg desogestrel	21 active pills
	4	30 µg ethinyl estradiol	75 µg gestoden	21 active pills
	1	15 µg ethinyl estradiol	60 µg gestoden	24 active pills, plus 4 inert pills
Combiphasic (Gracial)	1	(blue pill) 40 µg ethinyl estradiol (white pill) 30 µg ethinyl estradiol	(blue pill) 25 µg desogestrel (white pill) 125 µg desogestrel	7 (blue) active pills, plus 15 (white) active pills

None of the OC users reported to have missed one or more pills during the evaluated menstrual cycle. Only one player reported breakthrough bleeding (*spotting*) during the tracked menstrual cycle, without an apparent cause, because she was taking the same OCs for more than 6 months on a regular basis.

The players reported several motives for starting OC use, such as menstrual irregularity (including amenorrhea), contraception, dysmenorrhea, acne and ovarian cysts.

All participants had been playing handball for a minimum of 5 years and/or integrated regional or national main teams and were training/competing for at least four times a week at the time of testing, in all of the cases physical activity was higher than 6 hours a week. Some players reported an increased amount of physical activity because of regular, weekly sports lessons (7 players were students at our Faculty) or cumulative training sessions and matches in regional and/or national handball teams (n=4). Also some players (n=9) reported to be playing in theirs age category and above.

Because the evaluation was performed during a competition period, the risk of being injured during the testing time was high. Four players had an injury during the evaluation period; two had a unilateral knee contusion and two others had a unilateral ankle sprain. In both cases, the jump tests were immediately interrupted, as well as the knee laxity measurements in the injured knee.

6.3.2 Menstrual cycle monitoring

Given the economical constrains and logistic demands for measuring the daily hormonal levels in all players, across one complete menstrual cycle, we decided to divide the study into two phases. Firstly, two non-OC users and three OC users were subjected to daily blood sampling (excluding the weekends). The non-OC users were instructed to begin on day 5 using a commercially available ovulation kit[7] (sensitivity 20-25 mIU/ml LH, accuracy 99%) and to record each day's body basal temperature to confirm and to help point out when ovulation may have occurred. A specific digital basal thermometer was given for this purpose, accurate to 0.1° F. We also used the method based on the observation of salivary ramification (as described in p68) which would be helpful for predicting the estrogen peak. However, this method revealed to be very unpractical, because it was very difficult to obtain appropriate daily salivary specimens. Given the daily blood analyses from this first phase, we defined a different protocol to monitor the player's menstrual cycle (on specific and important times), based on a minimal number of blood samples, between two and four per subject.

In the second phase, all the players were informed to go for the 1st blood test until the 6th day of menses. The OC users were asked to go for more blood tests on the 14th and the 20th days. Non-contraceptive users were asked to go for a blood test immediately after the positive LH test, and 6 to 8 days after the positive LH event. Whenever the positive LH

[7] Clear Plan and ovulation test strips

test did not occur before the 20th day, the players were instructed to go for blood samples for a second time to confirm that the ovulation did not occur.

The players were instructed to inform the investigator when the first day of menses occurred and to start testing sessions as soon as possible, limited to the 6th day of the menstrual cycle. The intention was to study a complete menstrual cycle and not to randomly start in any of the other menstrual cycle phases, because we could be in presence of two different cycle characteristics, especially in the case of non-contraceptive users. The consequences were that the examiner knew the participant's day in their menstrual cycle, but still was not knowledgeable to other menstrual cycle indicators.

An anovulatory cycle was declared if the player was using OCs or if there was no strong evidence that it actualy occurred. The ovulation was confirmed using some of the indicators: positive test in the ovulation kit, progesterone levels rising to $3ng.mL^{-1}$ and the thermal nadir using BBT charts. BBT charts were analysed as mentioned in the preliminary study, while the investigator was ignorant of the hormonal levels and LH tests.

6.3.3 Blood Samples

For hormonal determination, 184 blood samples were collected for both OC users and non-users.

The participants were instructed to undergo, in the same general time of day, blood sampling. The blood samples were taken from the antecubital vein of the forearm[8] by medical practitioners in a clinical analysis laboratory[9]. The blood samples were properly coded and centrifuged at 3000 rpm for 15 minutes to extract the plasma. For relaxin and testosterone the extracted plasma was then immediately frozen and stored at -20ºC for post-processing. Progesterone (ng/mL), estradiol (pg/mL) and LH (mUI/mL) were analysed using chemiluminescence assays[10]. Average coefficient of variance (CV) for progesterone was 12% for intra-assay and 28% for interassay comparisons. For estradiol the mean CV was 7.4% for intra-assay and 13.9% for interassay comparisons. Testostenone concentration was analysed using RIA assays[11]; mean CV for interassay comparisons was 11% for intra-assay and 30.2% for interassay comparisons. Assay sensitivities for estradiol, progesterone and testosterone were 15 pg/mL, 0.1 ng/mL and 4

[8] Vacutainer System
[9] Endoclab
[10] Immulite 2000 System; SIEMENS
[11] Coat-A-Count Total Testosterone; SIEMENS

ng/mL, respectively. Relaxin (ng/dL) was analysed by ELISA[12] mean CV was 8.2% for intra-assay comparisons.

6.3.4 Anterior Knee laxity

Knee laxity was considered to be the amount of anterior tibial displacement measured, as previously described in chapters 2 and 4. The same knee arthrometer during all the study was used. The same techniques and procedures were also used. The laxity was measured at 15lbf, 20lbf and 30lbf. In each test occasion, the laxity assessment was repeat two to five times for each knee.

All knee laxity measurements were performed by a single investigator throughout the study. The intratester reliability was established and considered appropriate, as previously reported (chapter 4).

6.3.5 Vertical jumps

The same protocol used in chapter 4 was used to asses jump height. The same investigator conducted all jump tests throughout the study.

6.4 Results and discussion

6.4.1 Menstrual cycle Characteristics'

Daily hormonal fluctuations

Two non-OC users were tracked for hormonal content on a daily basis during a complete menstrual cycle, which results are presented in figures 6.6 and 6.7. The same procedure was adopted for two OC-users, which results are shown in figures 6.8 and 6.9.

As we can see by the graphs from the non-OC users, their cycles were very different, despite the fact that they were from the same team and were about the same age. Both cycles were ovulatory, but largely different in length of phases and hormonal content. The menstrual cycle from player 1 was characterised by a lengthened follicular phase, showing a low estradiol production between days 1-25, and a shortened luteal phase, revealing menstrual cycle disturbances most likely associated to the exercise (De Souza *et al.*, 1998). This cycle was classified as oligomenorrhoeic, because it lasted more than

[12] Immundiagnostik AG

36 days (Loucks & Horvath, 1985). In addition, there is strong evidence of luteal phase deficiency, as the luteal phase lasted about 8 days. We did not measure the hormonal content in the last couple of days, as it was weekend. At just two or three days of the end of the cycle (were we know that the progesterone levels are around a few ng/cL), the subject still was well above 100 ng/cL. This is possibly related with abnormal *corpus luteum* function (De Souza, 2003). In this cycle, we also found a slight increased relaxin content before the estimated day of ovulation and decreased after ovulation. The increased relaxin content before ovulation was possibly related with the follicular burst. Different relaxin hormonal behaviour was found in the other player, as the relaxin secretion slightly increased during the luteal phase (figure 6.7). In comparison, the daily relaxin secretion in OC users showed no clear cyclicity throughout the menstrual cycle (figure 6.8 and 6.9). There are reports of increased relaxin in serum in response to OC treatment, indicating that the *corpus luteum* is not the only source of relaxin (Wreje *et al.*, 1995). The authors found two peaks of relaxin content (on days 14 and 20). Based on their findings we marked these days for blood sampling in the OC users.

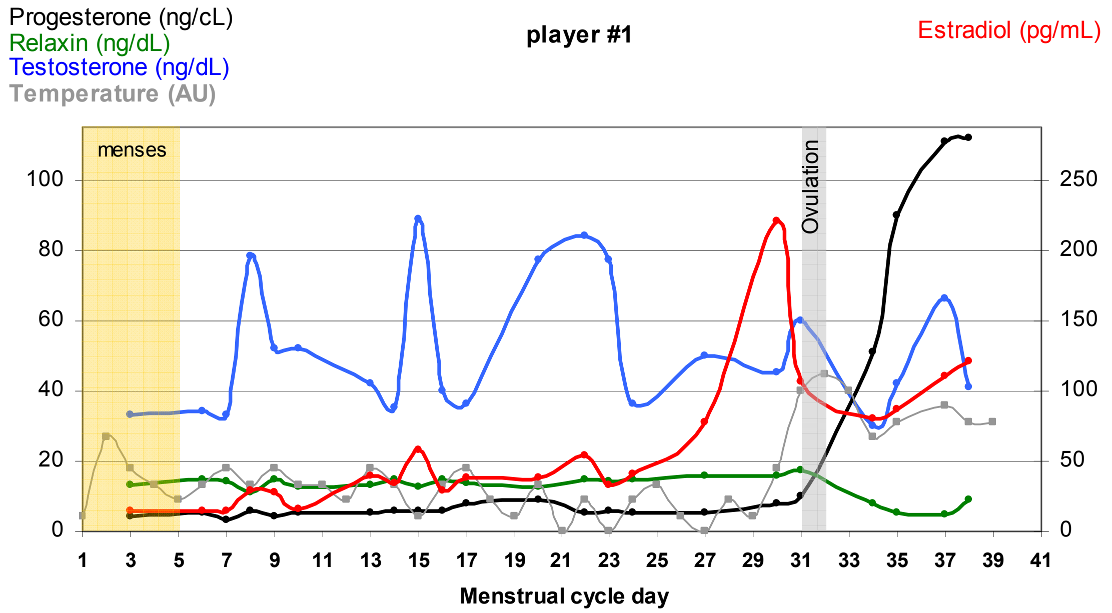


Figure 6.6 Daily hormonal fluctuation from the player #1, non-OC user

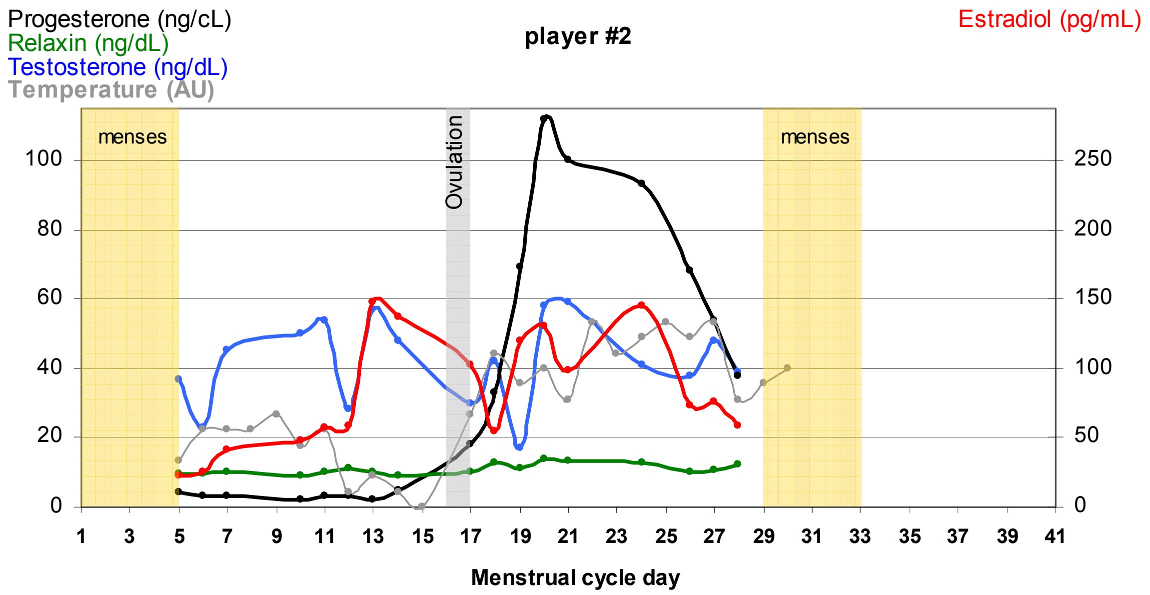


Figure 6.7 Daily hormonal fluctuation from the player #2, non-OC user

We also examined the endogenous testosterone levels daily. In both OC-users and non-OC users significant fluctuations were found across the menstrual cycle, but we were unable to find a clear pattern. It is well known that females have significantly lower testosterone levels than males (10-fold less), and that this hormone fluctuates during the menstrual cycle (Shultz *et al.*, 2005; Shultz *et al.*, 2004; Wajchenberg *et al.*, 1989). Research has suggested that elevated testosterone levels are associated with enlarged follicular and shortened luteal phases (Smith *et al.*, 1979).

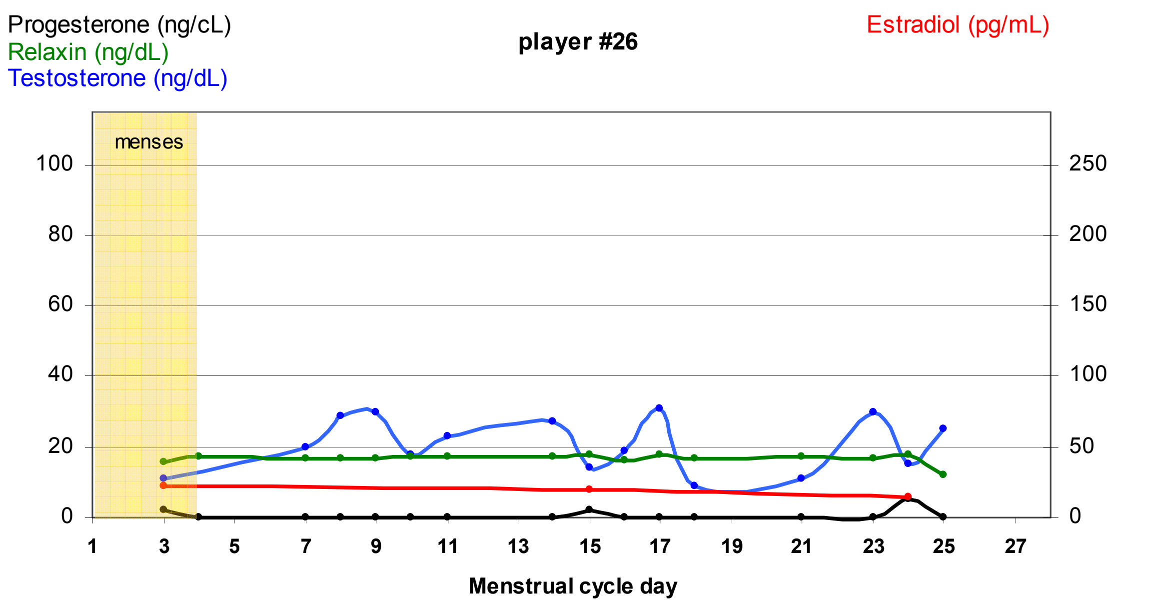


Figure 6.8 Daily hormonal fluctuation from the player #26, OCs user

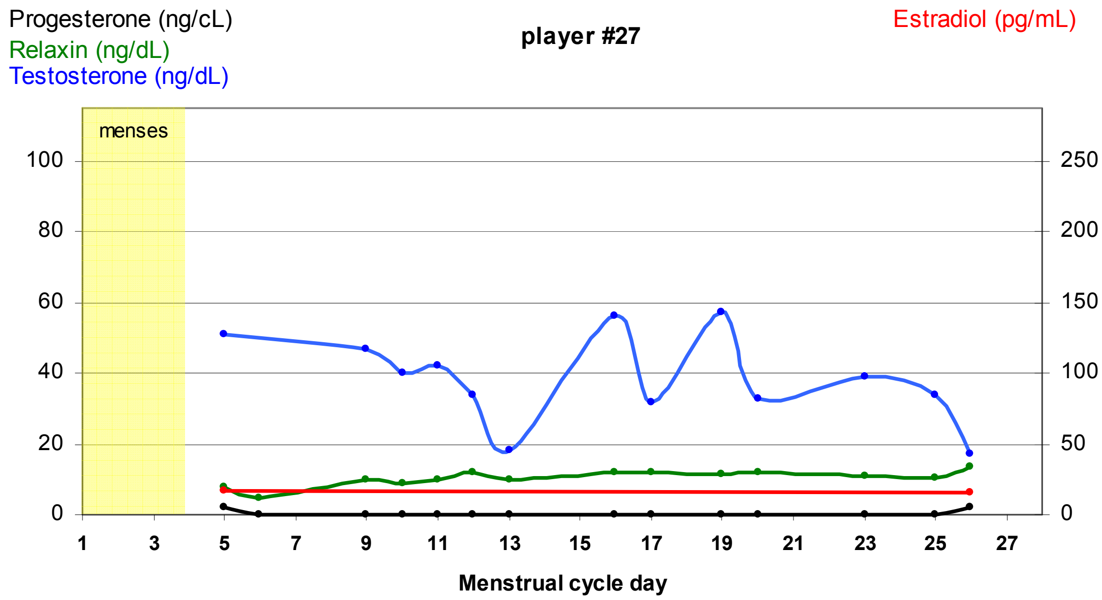


Figure 6.9 Daily hormonal fuctuation from the player #27, OCs user

Hormonal fluctuation pattern

The non-OC users presented different lengths of cycle phases (table 6.4) as well as different hormonal content, within the same phase.

According to standard guidelines, ovulation takes place 24 to 48 hours after positive urine LH test, but only when the progesterone secretion raised to 3ng.mL^{-1} the ovulation was confirmed. We do not really know when the ovulation occurred, as all methods used are indirect methods. Consequently, some bias may have been introduced in the estimation of the follicular and luteal phase's length.

The players exhibited menstrual cycles ranging between 23 to 41 days. One was unovulatory, which was not analysed in this work. Three (subjects 1, 8 and 10) had a long cycle (oligomenorrhoeic). The most frequent menstrual cycle disturbance observed was the luteal phase deficiency (length ≤10 days), representing 32% of the cycles (8 players). Only two players showed a precise luteal length of 14 days. The majority of the players showed a lengthened follicular phase, which may be possibly related with a decreased estrogen production. It is difficult to compare the documented menstrual cycle disturbance with other studies, because of the specificity of the samples. In a recreational runners sample, the LPD was reported in 46% of the cycles and the absence of ovulation in 16% (De Souza *et al.*, 1998). To better understand the observed menstrual disturbances it would be necessary to monitor, on a daily basis, the serum hormonal levels, dieting, exercise and other possible stress factors.

Table 6.4 Menstrual cycle characteristics' from the non-OC users

Subject	Cycle length (days)	Cycle type	Follicular phase length	LH surge	Estimated day of ovulation	Luteal phase lenght
#1	41	Ovulatory	32	31	33	8
#2	29	Ovulatory	17	15	18	11
#3	32	Ovulatory	20	19	21	11
#4	32	Ovulatory	21	17	20	12
#5	34	Ovulatory	23	20	24	10
#6	28	Ovulatory	14	13	15	13
#7	27	Ovulatory	17	17	18	9
#8	38	Ovulatory	23	23	24	14
#9	33	Anovulatory				
#10	37	Ovulatory	25	24	26	11
#11	32	Ovulatory	20	19	21	11
#12	27	Ovulatory	14	12	15	12
#13	29	Ovulatory	15	14	16	13
#14	33	Ovulatory	18	17	19	14
#15	28	Ovulatory	16	15	17	11
#16	34	Ovulatory	21	20	22	12
#17	31	Ovulatory	20	17	21	10
#18	23	Ovulatory	18	17	19	4
#19	28	Ovulatory	15	14	16	12
#20	31	Ovulatory	17	16	18	13
#21	35	Ovulatory	25	24	26	9
#22	32	Ovulatory	20	19	21	11
#23	25	Ovulatory	15	14	16	9
#24	31	Ovulatory	20	19	21	10
#25	27	Ovulatory	15	14	16	11
Average	31		19	18	20	11

Based on the serum levels we can confirm the obvious: the usage of OC alters significantly the serum hormonal content of all the tested hormones (tables 6.5 and 6.6).

Table 6.5 Serum hormonal levels: means comparison

	OC	Non-OCs	$p_c\,(\chi^2)$
Relaxin (ng/dL)	11.68 (N=82)	10.31 (N=91)	$2.5*10^{-2}$
Estradiol (pg/mL)	26.24 (N=43)	74.46 (N=94)	$2*10^{-7}$
Progesterone (ng/mL)	0.2655 (N=43)	3.362 (N=94)	$5*10^{-5}$
Free testosterone (pg/mL)	0.3668 (N=6)	1.471 (N=18)	$6*10^{-7}$
Total testostorene (ng/mL)	28.88 (N=79)	42.81 (N=93)	$7*10^{-7}$

Table 6.6 Serum hormonal levels: instrumental considerations

	OC	Non-OCs	Difference	Lab uncertainty[13]	Sensitivity
Relaxin (ng/dL)	11.68	10.31	12%	8.2%	0.4 ng/dL
Estradiol (pg/mL)	26.24	74.46	65%	13.9%	15 pg/mL
Progesterone (ng/mL)	0.2655	3.362	92%	28%	0.1 ng/mL
Free testosterone (pg/mL)	0.3668	1.471	75%	5%-17%	0.15 pg/mL
Total testosterone (ng/mL)	28.88	42.81	33%	11%	4 ng/mL

[13] Intermediate precision (BIPM *et al.*, 2008) or inter-assay precision: Progesterone, Estradiol. Repeatability (BIPM *et al.*, 2008) or intra-assay precision: Relaxin and total Testosterone.

The players showed different cycle lengths and different hormonal levels, and a simple average is highly uncorrelated with a possible general pattern. A rescaling and synchronization method was applied to enable the recovery of the main shape of hormonal variation. These techniques were applied to all measurements in our work and enabled us to study groups. The results from these analyses (estradiol, progesterone, testosterone and relaxin) between OC users and non-OC users are displayed in figures 6.10 and 6.11.

The non-OC users had, on average, significantly greater estradiol levels than OC users (table 6.5), which were most likely dependent on the day of menstrual cycle. In both groups, the estradiol levels remained constant during the first five days. In the ovulatory cycle, estradiol raises sharply just before ovulation (late follicular phase). Then, about five days after ovulation, there is a second estradiol rise that maintains itself constant until the last 3 days before the next menses, falling thereafter to the initial levels.

As expected, the estradiol and progesterone levels were highly mitigated in the OC group in comparison with the non-OC users (figures 6.12 and 6.13).

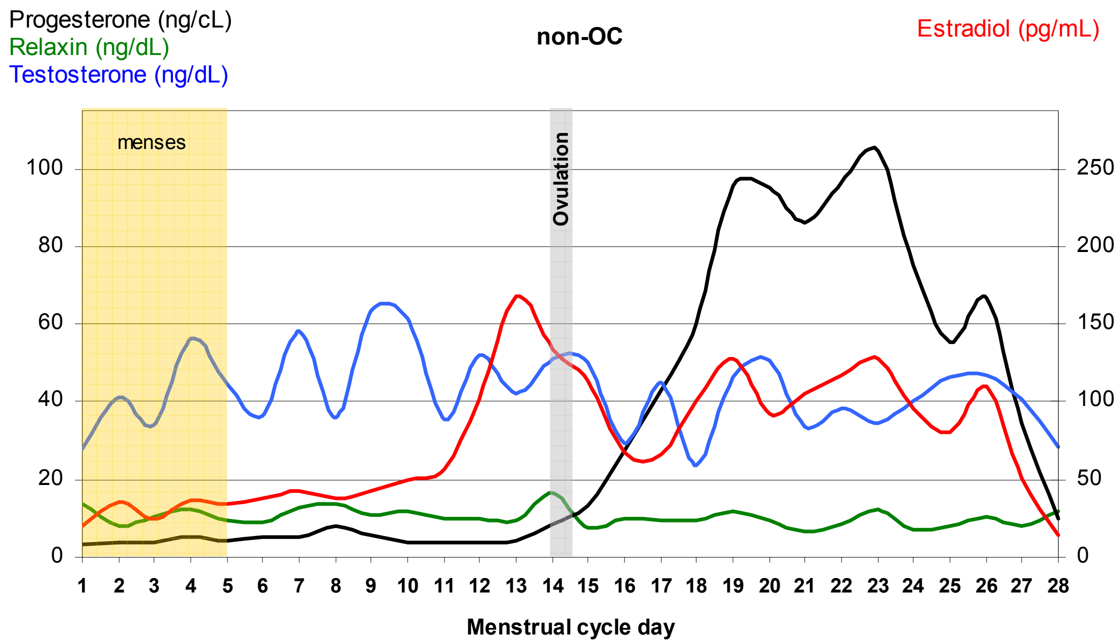


Figure 6.10 Daily hormonal in non-OC users

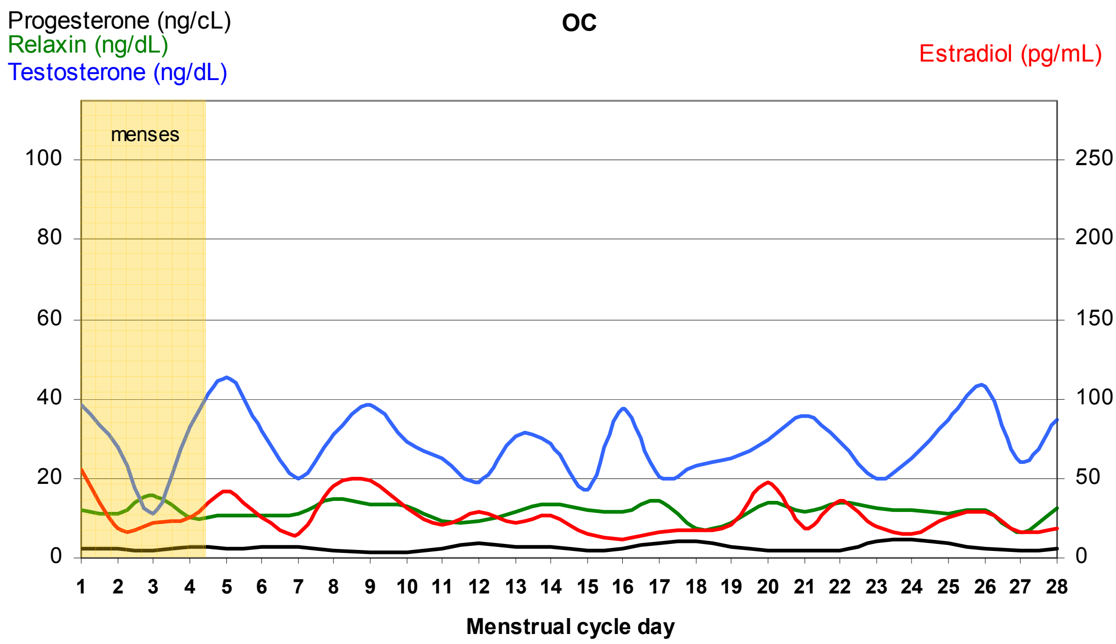


Figure 6.11 Daily hormonal in OC users

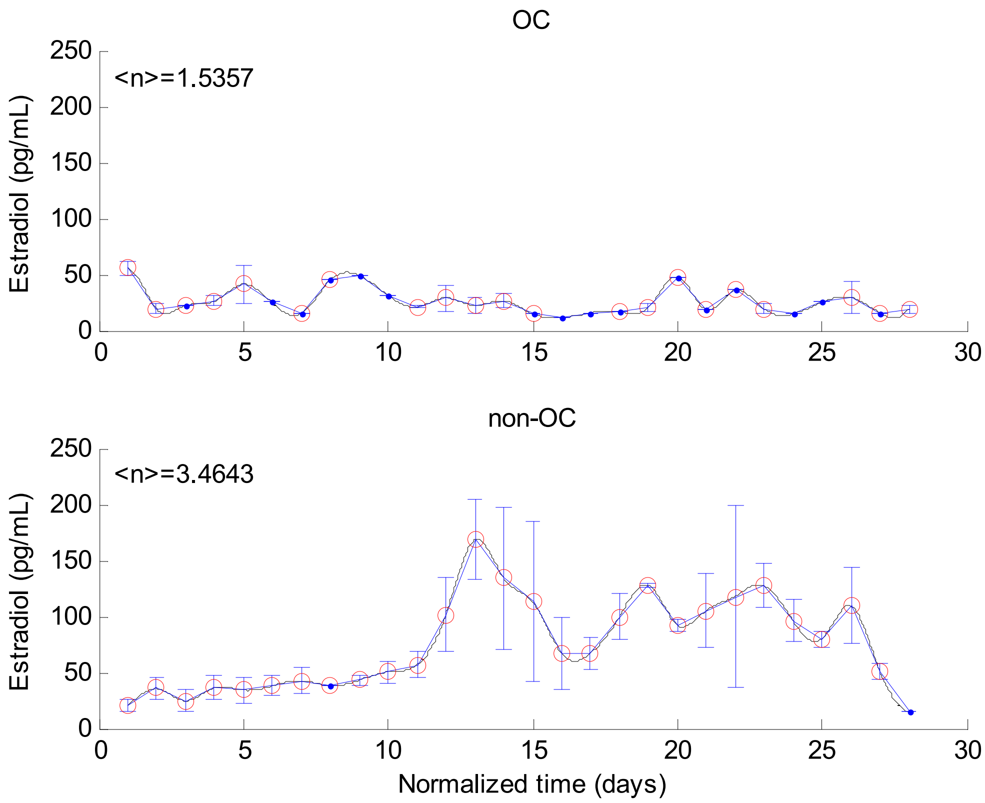


Figure 6.12 Estradiol fluctuation during the menstrual cycle with and without the use of OCs

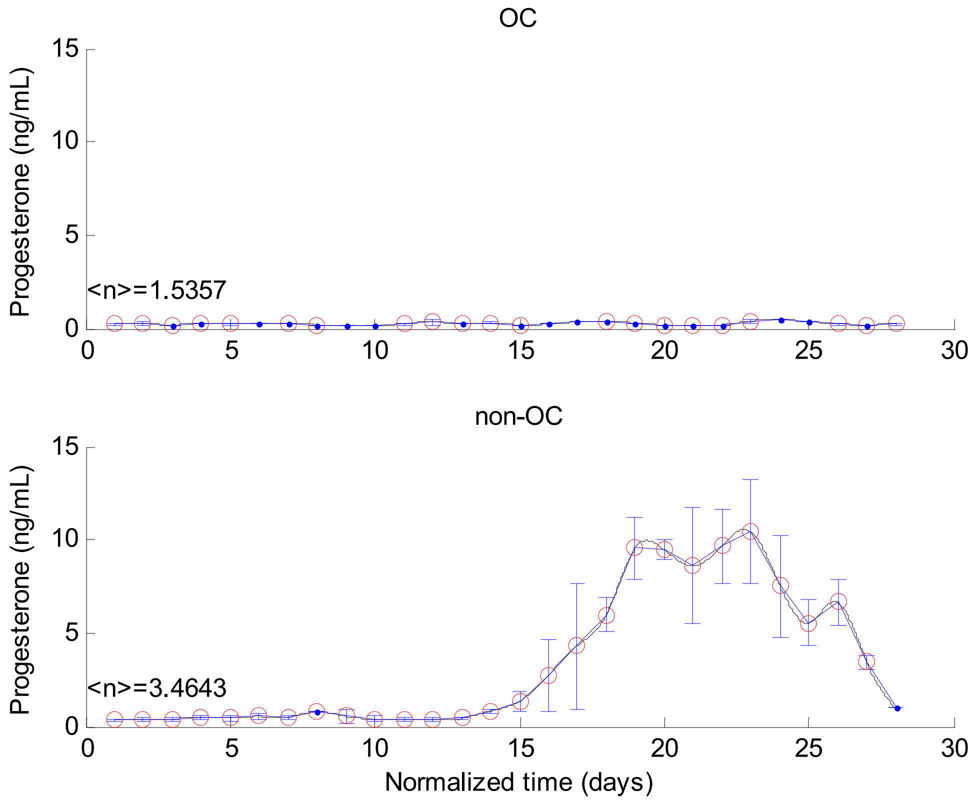


Figure 6.13 Progesterone fluctuation during the menstrual cycle with and without the use of OCs

On average, the OC users (11.7 ng/dL) presented a higher relaxin level per day than the non-OC users (10.3 ng/dL) (t-test p<2.7%; normal distributions: Jarque-Bera test, 5%). Relaxin was detectable in serum of all players, with no obvious cyclicity, independently of using OCs or not. In the ovulatory cycles, there was a slight increase of relaxin around ovulation, which may be related with the follicular burst, as already mentioned. There is little information on the literature about relaxin levels during the menstrual cycle, but the available reports found an increased relaxin content approximately 6 days after LH peak (Wreje *et al.*, 1995; Stewart *et al.*, 1990). Its main source is thought to be the *corpus luteum* (Stewart *et al.*, 1990).

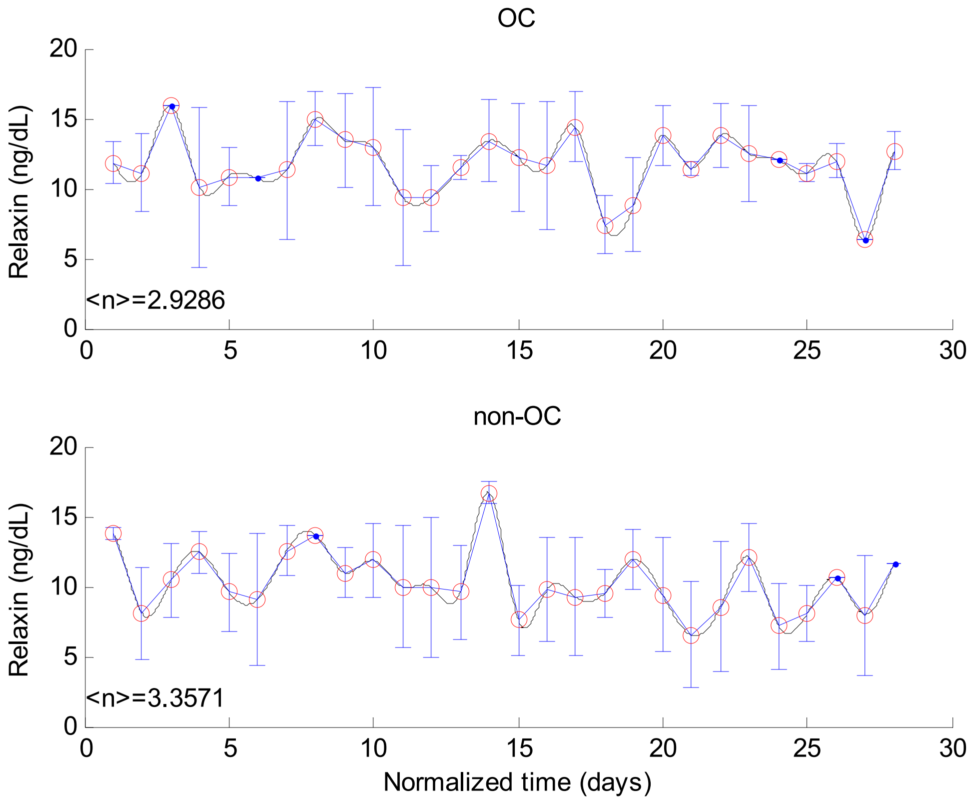


Figure 6.14 Relaxin fluctuation during the menstrual cycle with and without the use of OCs

Total testosterone concentrations (figure 6.15) showed higher levels per day in non-OC users (μ=79 ng/dL) than in OC users (μ=96 ng/dL) (t-test p<0.0001%; normal distributions: Jarque-Bera test, 5%). As in the non-OC users, the testosterone content presented a very different pattern among the players, with no cyclicity. In the work of Shultz *et al*. (2005) daily serum testosterone levels also changes during the menstrual cycle, still peaking in the early luteal phase.

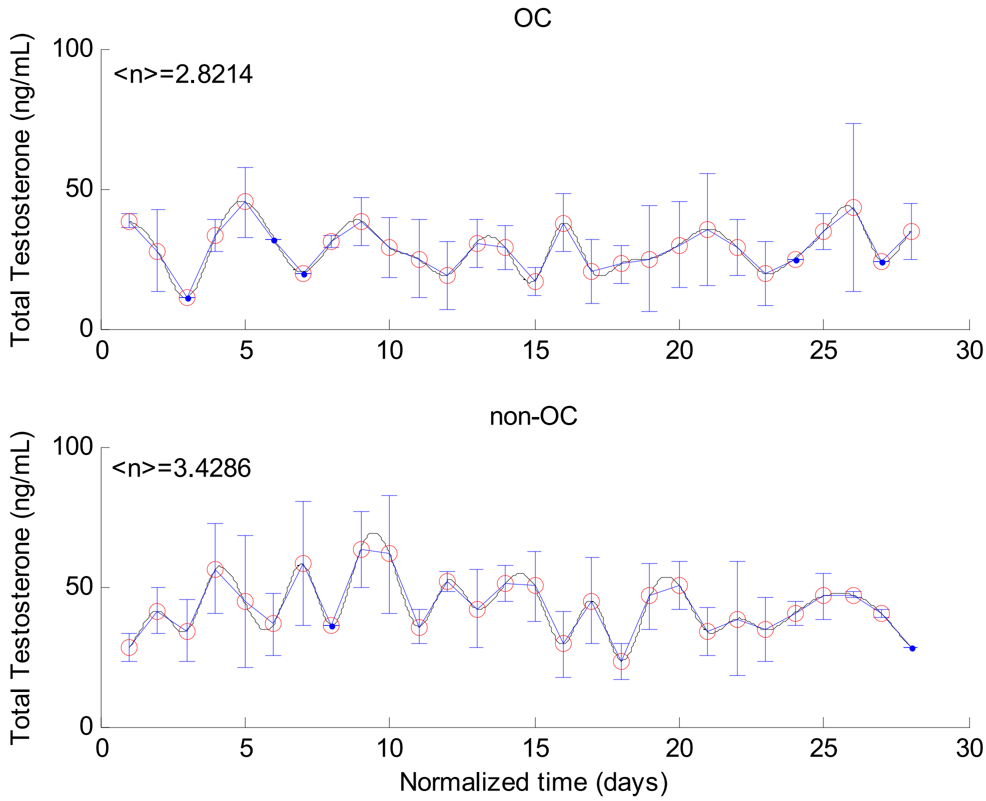


Figure 6.15 Testosterone fluctuation during the menstrual cycle with and without the use of OCs

6.4.2 Relation between hormonal content and anterior knee laxity

The knee laxity was measured at 30lbf with a KT-1000 arthrometer; 3271 measures were done on each leg. On average, there were no significant differences on knee laxity between OC users and non-OC users (table 6.7). These findings are consistent with previous studies (Hicks-Little *et al.*, 2007; Arnold *et al.*, 2002).

Figure 6.16 shows the distribution of anterior knee laxity during the menstrual cycle with and without the use of OCs. Each data point is the average of about 12 subjects, and for each subject we used the median of all unilateral (right/left knees) or bilateral measurements (about 20 per subject each day). Injured limbs were excluded.

Table 6.7 Anterior knee laxity

OCs mean	Non-OCs mean	p_c (χ^2)	Power	Difference	Measurement uncertainty	Sensitivity	LSV
7.52 (N=638)	7.60 (N=710)	0.93	12%	1.0%	0.11mm	0.25 mm	0.29 mm

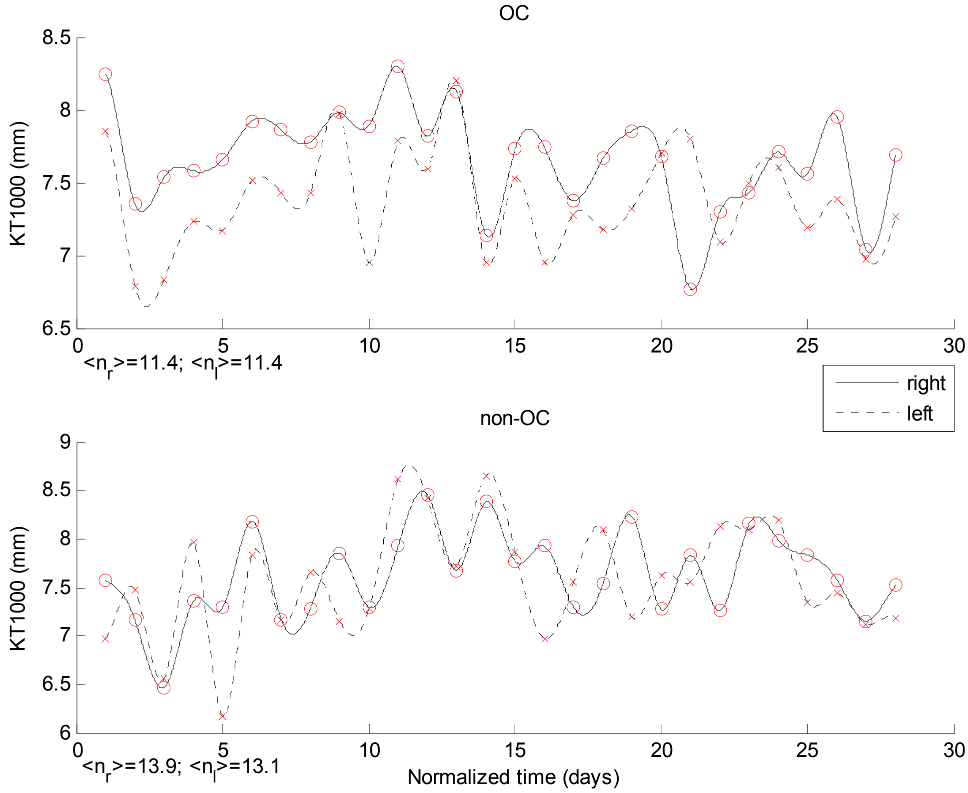


Figure 6.16 Anterior knee laxity at 30lbf (right/left knees) during the menstrual cycle

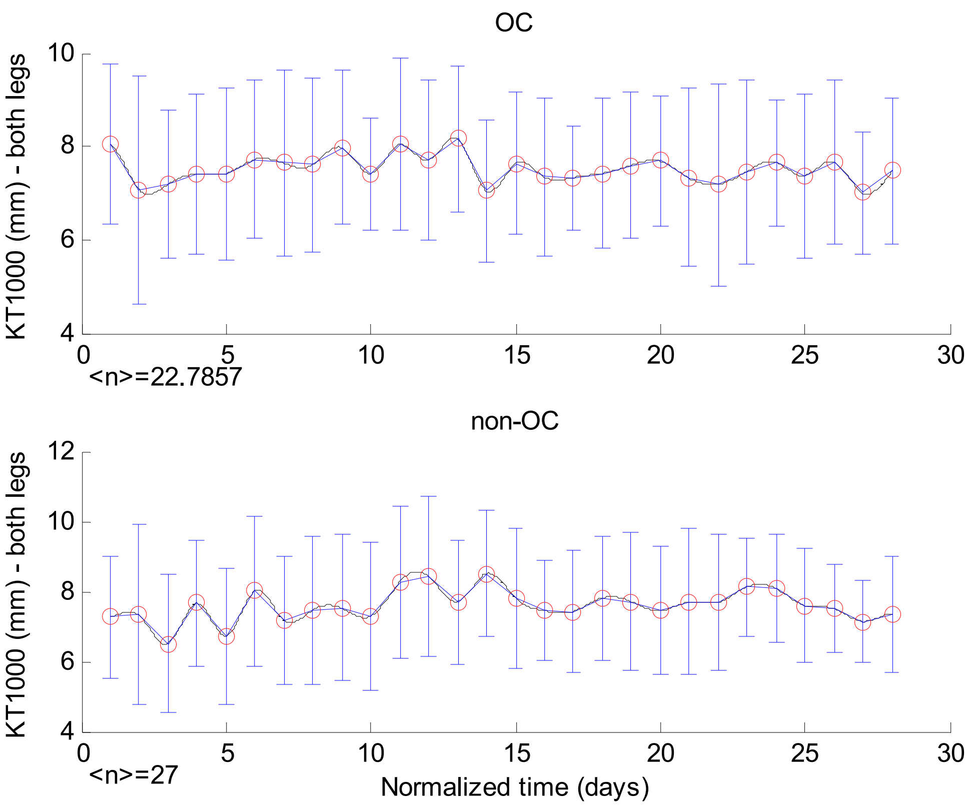


Figure 6.17 Anterior knee laxity at 30lbf (both knees) during the menstrual cycle

While most of the studies looked for knee laxity differences between the menstrual cycle phases, we were aware of whether knee laxity variance is hormonal variance dependent. As some of authors found a delayed interaction between hormonal levels and knee ligament laxity (Shultz *et al.*, 2004), using cross correlation techniques for each of the hormonal time series we calculated the “optimal” time delay (lag) between hormonal variations and knee laxity variations. We also measured the strength of that interaction (table 6.8).

Table 6.8 Cross correlation (and time delay) between hormonal variation and knee laxity

	Laxity					
	Both knee		Right knee		Left knee	
	Non-OC	OC	Non-OC	OC	Non-OC	OC
Estradiol	**0.553 (0)**	0.344 (0)	**0.481 (0)**	0.333 (6)	**0.496 (0)**	0.291 (0)
Progesterone	-0.383 (8)	-0.400(4)	-0.461 (4)	-0.318 (8)	-0.356 (8)	-0.312 (4)
Relaxin	0.288 (6)	0.324 (4)	0.507 (5)	0.420 (4)	-0.315 (2)	-0.312 (2)
Testosterone	**0.466 (2)**	0.269 (8)	**0.501 (2)**	-0.209 (2)	0.387 (4)	-0.329 (6)

The correlation between the estradiol levels with anterior knee laxity (both knees) was low in both groups (explained variance of 31% non-OC and 12% OC) with no delay (figure 6.18). *In vitro* studies support a short ACL tissue response (day 1 and 3) to increased estradiol levels, namely on its collagen structure and metabolism. Only in the group where daily hormones were performed the initial estrogen rise was possibly known, because the remaining players were instructed to collect blood when the LH test was positive, than most probably just after estrogen peak. Shultz *et al.* (2004) also found increased knee laxity following increased estradiol levels, but with a higher time shift, about 3 days. They monitored serum hormones and knee laxity measurements in a sample of nonathletic female subjects (n=25) with *normal* cycles on a daily basis. Also Heitz *et al.* (1999) found an increased knee laxity throughout the menstrual cycle, assessing serum estrogen and progesterone levels and knee laxity on days 1 (phase 1), 10-13 (phase 2), 20-23 (phase 3) of 7 normal menstruating females. Several other authors (Eiling *et al.*, 2006; Pollard *et al.*, 2006; Beynnon *et al.*, 2005 1346; Carcia *et al.*, 2004; Van Lunen *et al.*, 2003) reported an absence of estrogen influence on knee laxity during the menstrual cycle. However, as most of the studies used three/four distinctive points in the cycle, assuming their representative for each phase, direct comparisons are difficult to establish (see revision, p 55).

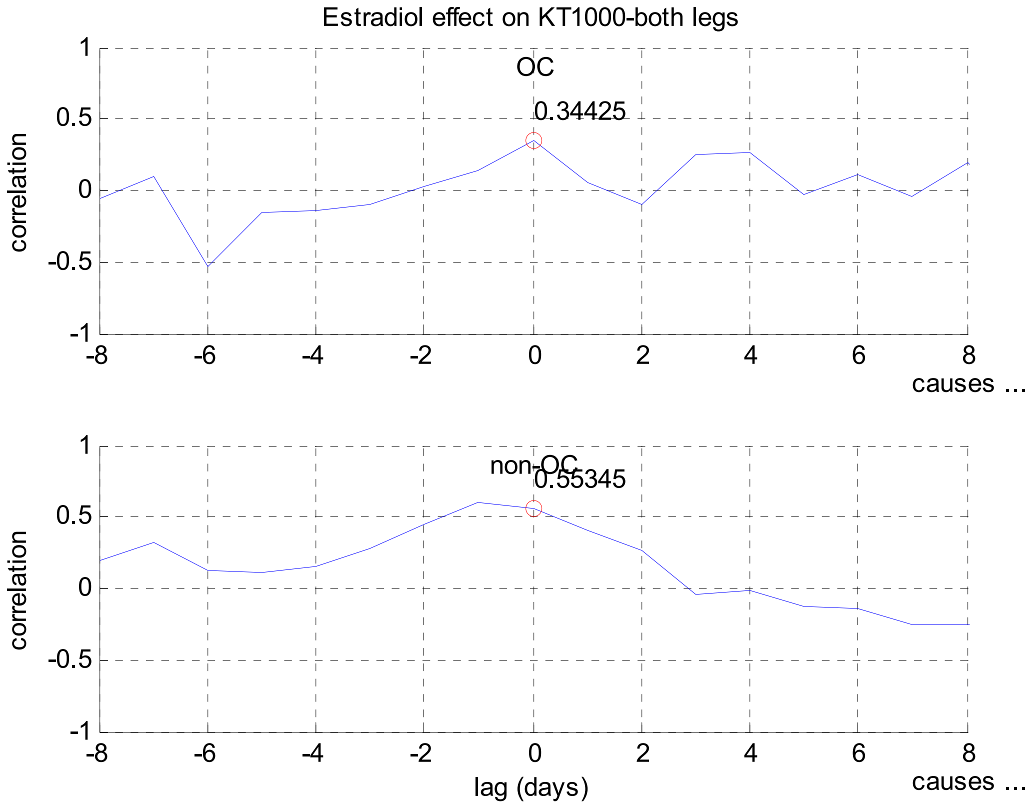


Figure 6.18 Correlation between estradiol fluctuation and anterior knee laxity

The correlation between the progesterone levels and anterior knee laxity was negative (figure 6.19). The explained variance was 16 and 15% respectively, with an optimum delay of 4 (OC) to 8 (non-OC) days. This is physiologically unrealistic and is probably just a numerical artifact. Even though, *in vitro* studies have suggested that progesterone stimulates the fibroblasts proliferation and collagen synthesis, counteracting the estrogen effect (Yu *et al.*, 2001). In contrast, Shultz *et al*. (2004) found an increased knee laxity approximately 4 days after progesterone changes. While some other studies also reported an increased knee laxity in the luteal phase that they could be related with increased estrogen and progesterone levels (Heitz *et al.*, 1999), others have failed to verify such association (Beynnon *et al.*, 2005; Van Lunen *et al.*, 2003).

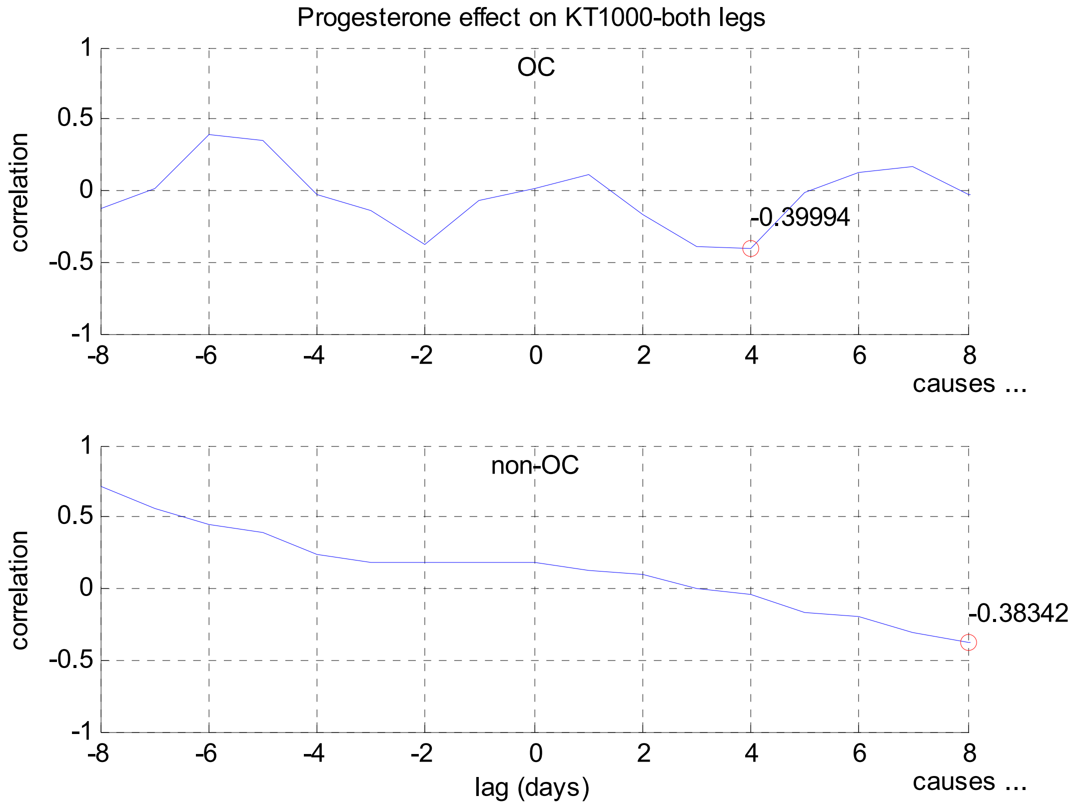


Figure 6.19 Correlation between progesterone fluctuation and anterior knee laxity

Relaxin levels were not decreased in the OC group (figure 6.14). The knee laxity variance was explained in 10 (OC) and 8% (non-OC), by this hormone, with an optimal delay of 3 and 5 days (respectively). To our knowledge the relaxin effect on knee laxity changes, in non-contraceptive cycles, is limited to one published study (Arnold *et al.*, 2002). In this study, only relaxin serum content and anterior knee laxity were quantified. Measures were performed only once a week and no significant differences were found in laxity. The potential effect of relaxin on ACL tissue is currently undetermined. However, there are some evidences of a potential effect of relaxin in fibroblast function and collagen production in other tissues, mainly inhibiting collagen synthesis and increasing collagen degradation (van der Westhuizen *et al.*, 2008; Mookerjee *et al.*, 2005; Samuel *et al.*, 1998; Samuel *et al.*, 1996; Unemori *et al.*, 1993; Unemori & Amento, 1990).

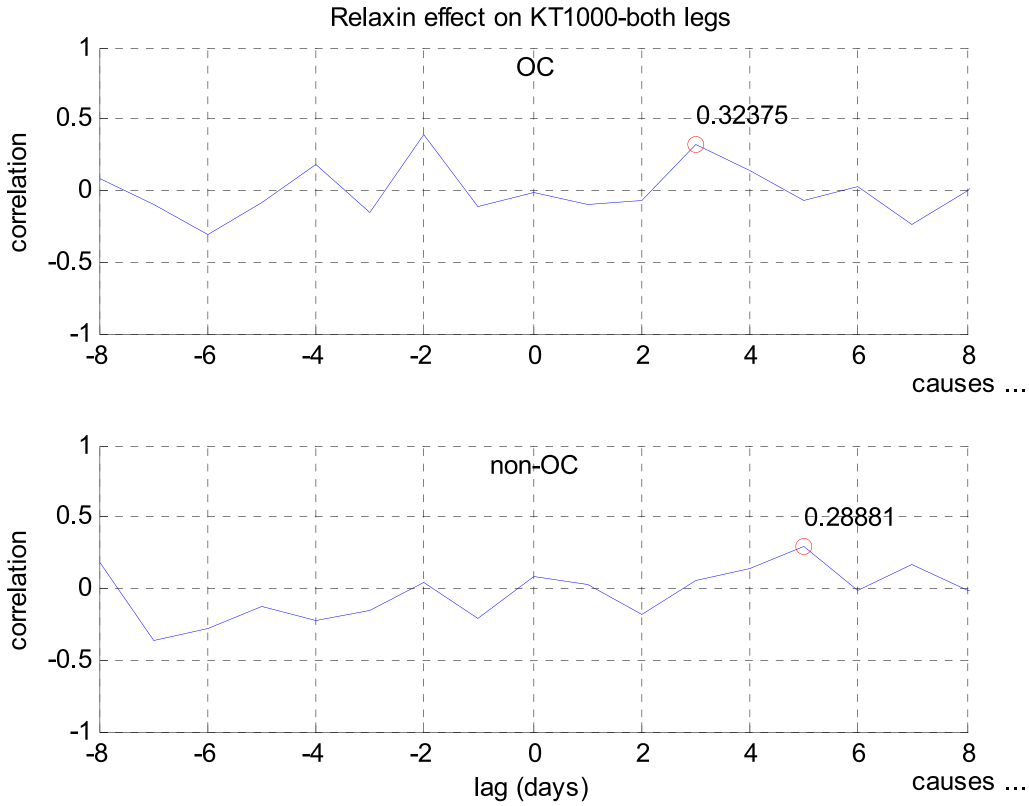


Figure 6.20 Correlation between relaxin fluctuation and anterior knee laxity

Testosterone levels were only slightly reduced in the OC group (figure 6.15). The knee laxity variance was explained in 7 (OC) and 22% (non-OC) by this hormone, with an optimum delay of 2 days in the case of non-OC users (figure 6.21). Also Shultz *et al.* (2004) found a positive effect of testosterone on knee laxity changes, with a time shift of 4.5 days. In contrast, Lovering and Romani (2005) found a significant correlation between ACL stiffness[14] and testosterone near ovulation. Still, their findings were based on three data collection points, guessed to be one in each cycle phase (menses, follicular, luteal). Using a similar design study, this relation was contradicted (Carcia *et al.*, 2004; Van Lunen *et al.*, 2003). While a few studies have suggested that testosterone may play a role on collagen remodelling and ligamentous tensile strength (Tipton *et al.*, 1971), it is yet not clear if female ACL is androgen-responsive tissue.

[14] Change between 89N and 134N divided by the displacement (mm) between 89N and 134N (Lovering & Romani, 2005).

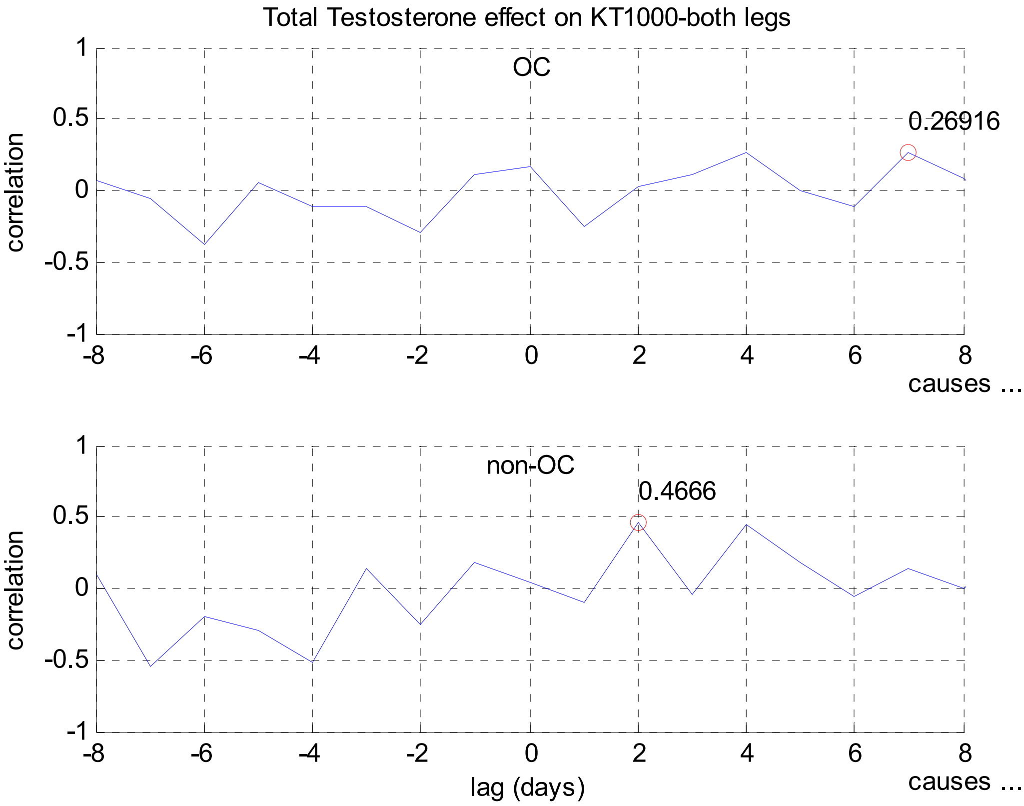


Figure 6.21 Correlation between testosterone fluctuation and anterior knee laxity

6.4.3 Relation between hormonal content and jump performance

The effect of reproductive hormones on muscle function in females is a topic of much interest in the strength training process (Reis *et al.*, 1995). Previous studies examining the potential effect of hormones in muscular strength during the *menstrual cycle* has produced equivocal findings (Friden *et al.*, 2006; Elliott *et al.*, 2003; Fridén *et al.*, 2003; Sarwar *et al.*, 1996). The lack of consensus is possible explained by different study design, including distinct menstrual cycle days sampled, distinct muscle strength parameters assessed, examination of different muscle groups, undetermined hormonal levels and different sampled populations (athletes and non-athletes).

The possibly effect of OCs on muscle strength has been less investigated than the relation of OCs and aerobic or anaerobic performance (for review see Lebrun (1994) and Burrows and Peters (2007).

The purpose of this study was to examine the effect of hormonal variation on simple field tests of lower limb muscle strength: SJ and CMJ. For both jumps, on average, there were no significant differences between the OC users and non-OC users (table 6.9). With 80% certainty, OC usage does not change the jumping performance of both SJ and CMJ by more than 8 mm (3%).

Table 6.9 Jump height

	OCs mean	Non-OCs mean	p_c (χ^2)	Power	Difference	Measurements uncertainty	Sensitivity	LSV
SJ (mm)	228.4 (N=305)	230.3 (N=332)	0.59	10%	0.8%	2 mm	1 mm	8 mm
CMJ (mm)	240.5 (N=305)	241.8 (N=332)	0.88	7%	0.6%	2 mm	1 mm	8 mm

The same methodology used to analyse the relation between hormonal variation and knee laxity throughout the menstrual cycle was adopted to analyse the jump height changes during the menstrual cycle. To our knowledge, this methodology is somehow peculiar so direct comparisons with other studies are difficult.

Figures 6.22 and 6.23 shows height variance in SJ and CMJ, respectively, during the menstrual cycle with and without the use of OCs. Each data point is the average of about 12 subjects, and for each subject we used the median of jump height. Injured limbs were excluded.

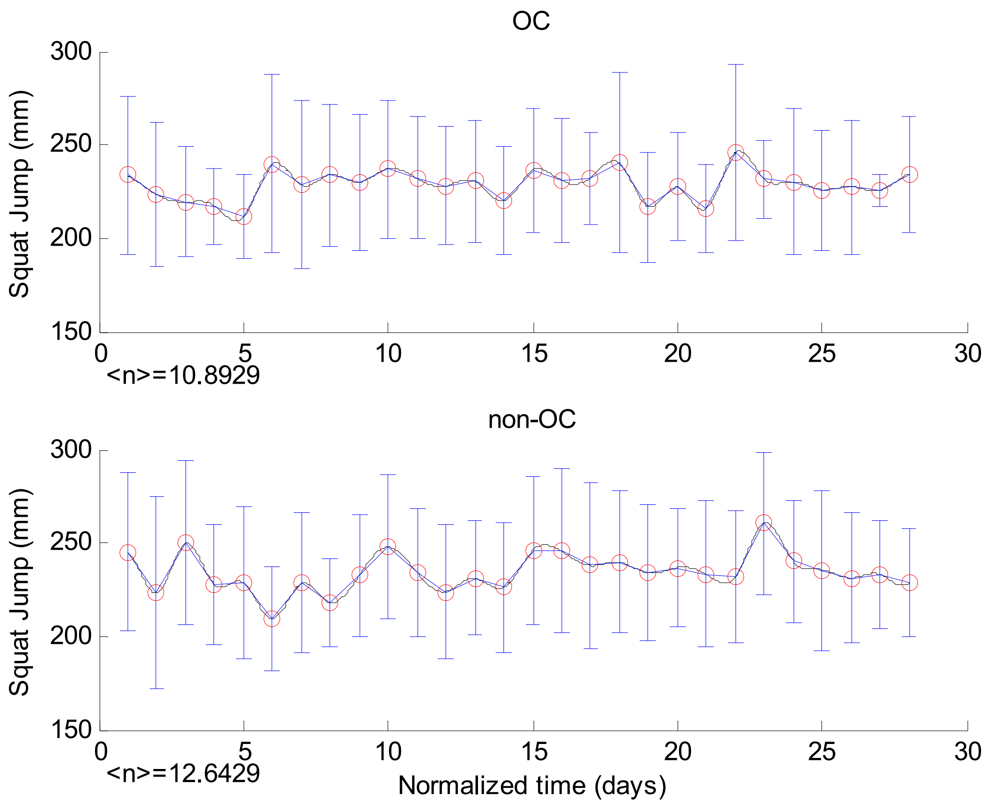


Figure 6.22 Squat jump height during the menstrual cycle

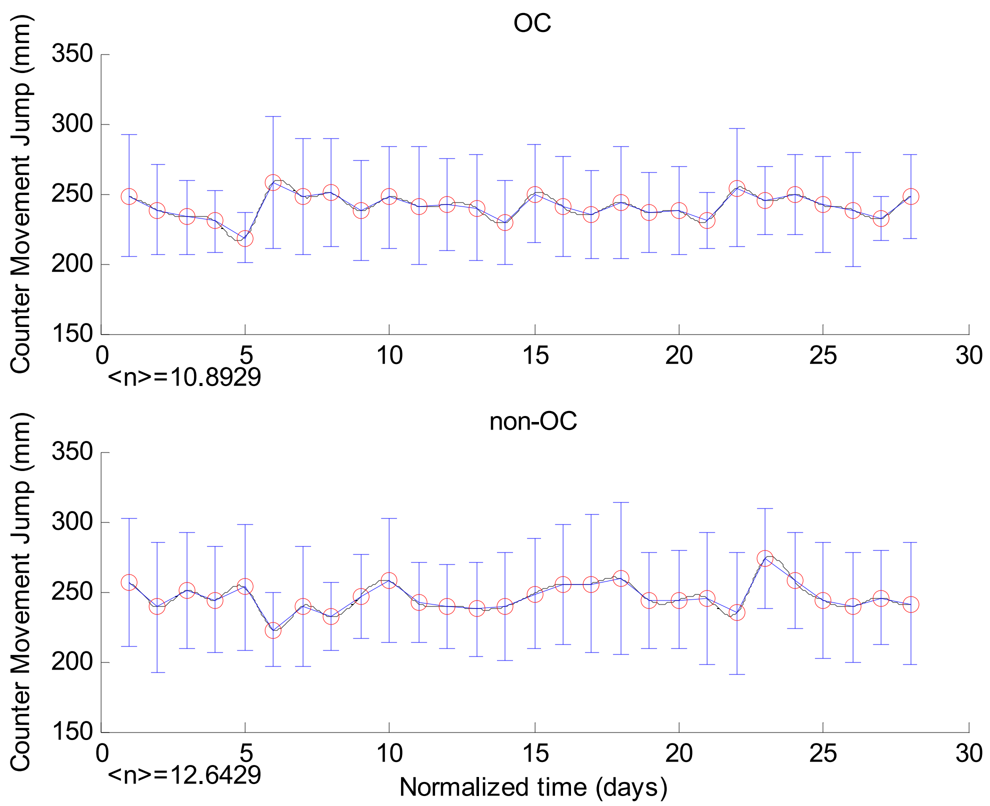


Figure 6.23 Counter movement jump height during the menstrual cycle

Table 6.10 presents the correlation between hormonal levels and jump height, considering an “optimal” time delay (lag).

Table 6.10 Cross correlation (and time delay) between hormonal variation and jump height

	Jump height			
	Squat Jump		Counter Movement Jump	
	Non-OC	OC	Non-OC	OC
Estradiol	**0.468 (4)**	-0246 (4)	**0.467 (6)**	-0.311 (4)
Progesterone	0.328 (2)	-0.386 (8)	0.290 (2)	-0.227 (2)
Relaxin	-0.246 (6)	-0.398 (4)	0.260 (4)	-0.265 (4)
Testosterone	-0397 (2)	**0.451 (2)**	-0.372 (2)	**0.560 (2)**

The correlation between the estradiol levels with jump height was higher in non-OC users than in OC users, with an explained variance in the CMJ of 23% in non-OC users and 10% in the OC users (figure 6.24). In both jumps there was a positive effect of estradiol in jump height in non OC users. Studies concerning the estradiol effect on muscle strength have found either a significant correlation (Reis *et al.*, 1995) or an insignificant correlation (Friden *et al.*, 2006; Elliott *et al.*, 2003). Most of the studies looked for muscle strength differences among the menstrual cycle phases, assuming typical and unchangeable hormonal environment in each phase. Fridén *et al.* (2006) compared the results in the square hop test[15] between the first few days (3-5), around ovulation (24 to 48 hours after detecting the LH-surge) and in the luteal phase (7 days after the estimated ovulation). An increased number of jumps in the *ovulatory phase* in comparison with other phases ($p < 0.001$) was found. However, no significant relation was achieved between the number of jumps and the hormonal levels (E_2, P_4, T, SHBG, and free T). Also, Sarwar *et al*. (1996) found a 10% increase in quadriceps and handgrip strength around ovulation, compared to follicular and luteal phases. As the subjects were tested weekly and the hormonal content was not determined, their results are not easily comparable with ours. Others studies found no significant variation in muscle strength (including handgrip strength, 1-leg hop test, isokinetic muscle strength, maximum voluntary isometric strenght) between menstrual cycle phases (Elliott *et al.*, 2003; Fridén *et al.*, 2003). Previous research examining the effect of OC use on muscle strength has been limited. However, unchanged muscle strength have been found throughout the OC cycle (Elliott *et al.*, 2005; Sarwar *et al.*, 1996).

The correlation between progesterone levels and CMJ height was low in both groups, being either positive or negative. It has been suggested that progesterone has a catabolic effect on muscle, contracting an anabolic effect of estrogen (Reis *et al.*, 1995). Because the progesterone increased after ovulation as well as estrogen, it is quite difficult to isolate their effects.

[15] Functional multi-directional test, requiring repeated changes of direction of the lower limb (Ostenberg *et al.*, 1998)

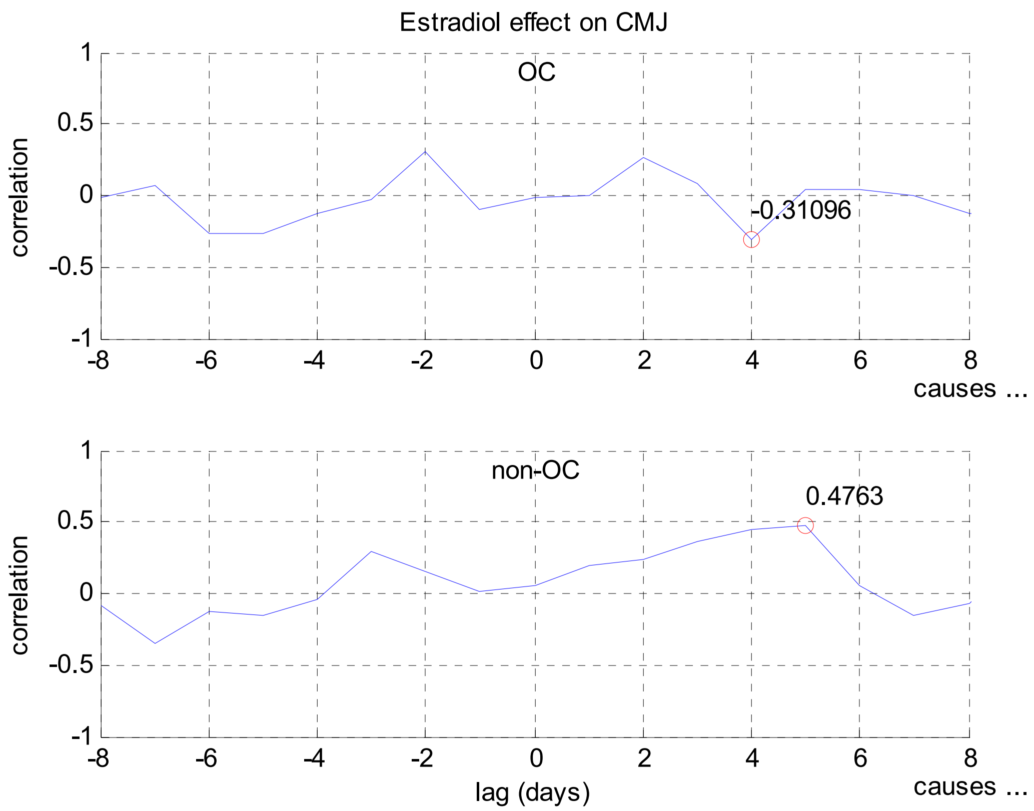


Figure 6.24 Correlation between estradiol fluctuation and CMJ

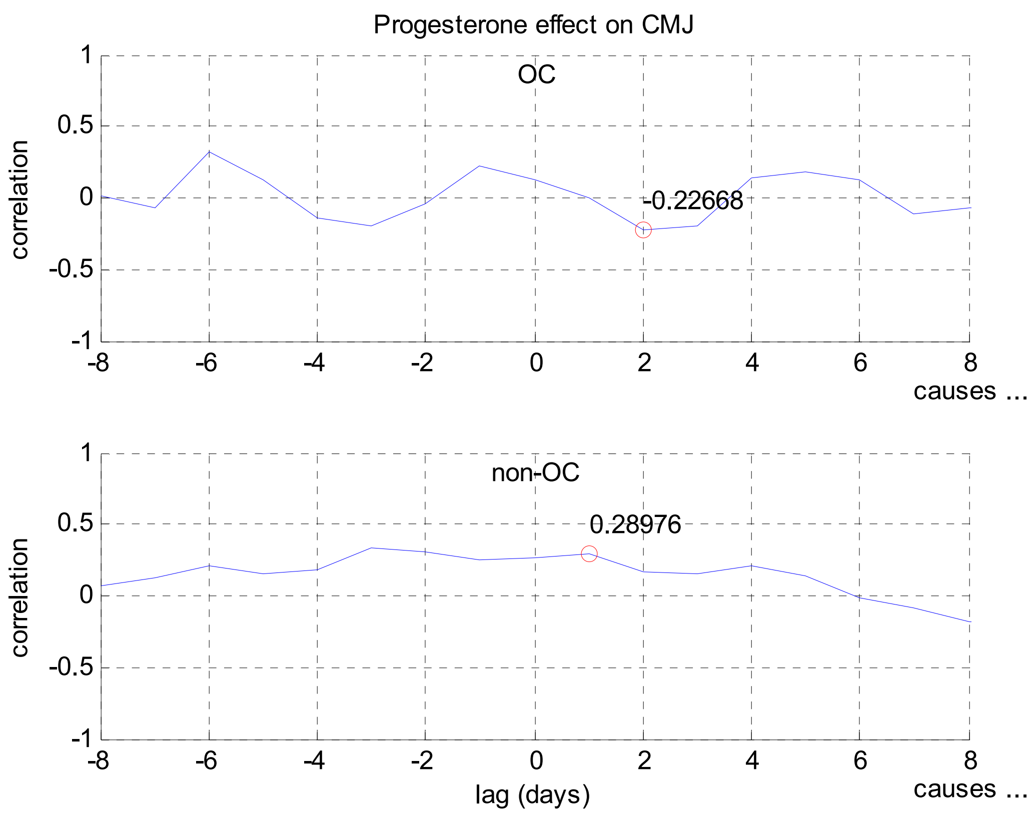


Figure 6.25 Correlation between progesterone fluctuation and CMJ

The correlation between relaxin levels and jump tests height was low in both groups, being either positive or negative. We were unable to find any published information about the possible effect of relaxin in muscle strength.

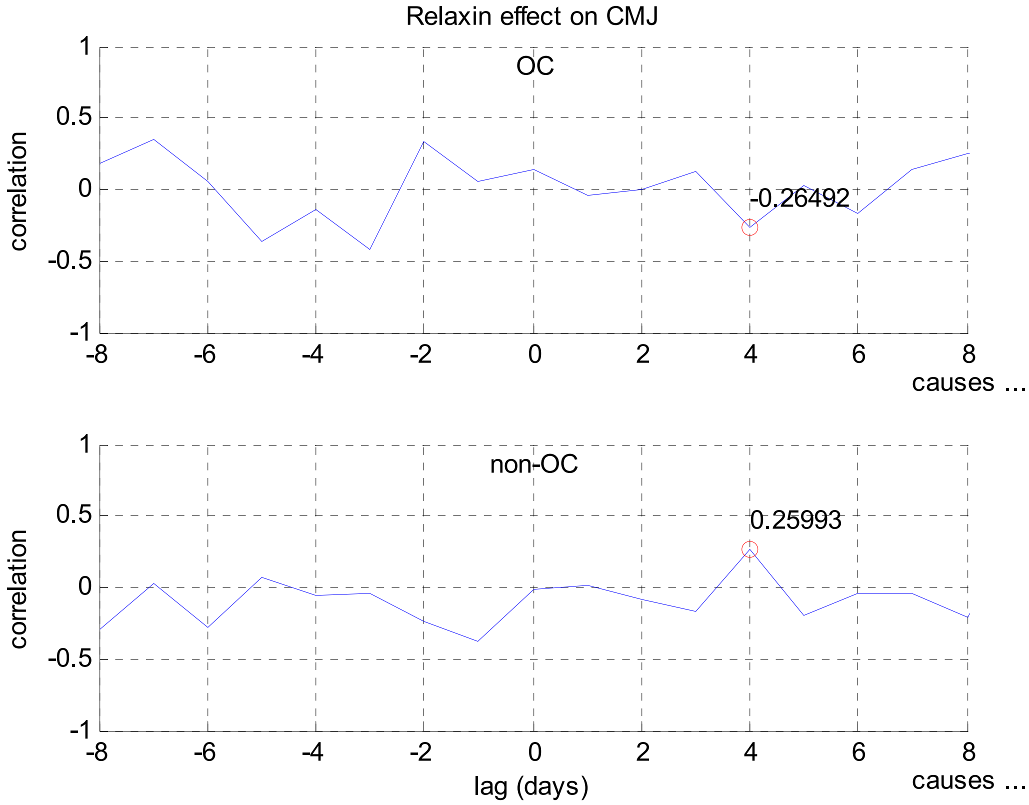


Figure 6.26 Correlation between relaxin fluctuation and CMJ

The correlation between testosterone levels and jump tests height was low in both groups (table 6.10). The correlation between the testosterone levels with CMJ height was higher in OC users than in non-OC users, being either positive or negative, with an explained variance in of 14% in non-OC users and 31% in the OC users (figure 6.27). We suspect that such a diverse results are a consequence of the unsynchronicity of the testosterone variations. As they don't follow any pattern along the cycle or between subjects, the averaging scheme used does *not* represent appropriately the population values and so any attempted statistics with this variable is just spurious. Extrapolation from the known effects of anabolic steroids might suggest that this should be an important variable, at least in higher doses and with much longer delays. We were unable to test this.

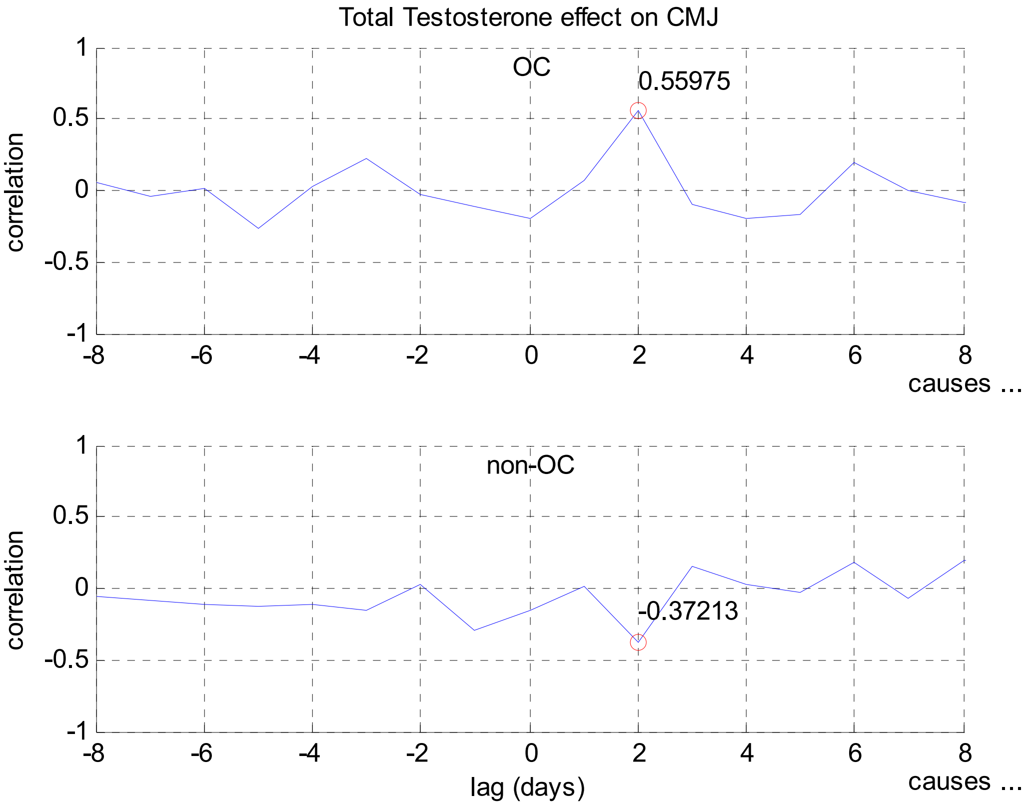


Figure 6.27 Correlation between testosterone fluctuation and CMJ

6.4.4 Hormones, knee laxity and jump performance

Contradicting the findings of the preliminary study, the OC and the non-OC groups do not differ in anterior knee laxity by more than 0.3mm (least significant value at 80% power, already similar to the test sensitivity). It is possible that the reason for such a contrast between the findings of the preliminary study (with non-athletes) and the conclusions of the current study (with athletes) is the physical load. A specific test would need to be carried on to fully demonstrated or negate this hypothesis but the possibility of a load modulated laxity being significantly more important than an hormonally driving laxity is an important one to enlighten the injury mechanisms.

Our data shares a common trend on the possible existence of a delayed interaction between these hormones and some biomechanical parameters, namely anterior knee laxity. This is easy to study at least with respect to estradiol and progesterone, on a sample of athletes, but hard to study with respect to relaxin and testosterone.

The first hormones have very large and predictable changes along the ovulatory cycle. An averaging scheme, using time rescaling and synchronization procedures, will describe adequately the bulk data even in the cases of some individually incomplete data - hormonal quantification and biomechanical evaluations performed in different days, skipped days, etc. Having the *group* daily measures, it is a trivial operation to model the statistical interactions including delays.

In the case of relaxin and testosterone, the hormones don't seem to have any definite variation pattern along the cycle. They may respond to other variables, as physical or psychological stress, food ingested, etc. This makes useless all daily averaging procedures intra or inter-individually. And, unless we have *all* days of a subject, we are unable to do a full model with delays even for that single subject (tables 6.11 and 6.12). We think this individuality and delayed interaction are the main difficulties with all studies about this subject.

Anyhow, from a prevention perspective, even if relaxin and testosterone affect the ACL laxity and/or the jumping capacity, as they are not controlled by the cycle day (or even by OC usage), we have no simple means of attributing a risk to each cycle day (or any preventive value to the usage of OC).

Table 6.11 Typical measurement routine – 7/15 days fully available

day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	…	27	28
Relaxin	☑	X	X	☑	☑	☑	X	☑	X	X	☑	☑	X	☑	X	…	a	b
Testosterone	☑	X	X	☑	☑	☑	X	☑	X	X	☑	☑	X	☑	X	…	c	d
Biomechanics	☑	X	X	☑	☑	☑	X	☑	X	X	☑	☑	X	X	☑	…	e	f

Table 6.12 Including 1 and 2 days delays for relaxin and testosterone – 1/15 days available

day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	…	28
Relaxin	b	☑	X	X	☑	☑	☑	X	☑	X	X	☑	☑	X	☑	…	a
Testosterone	a	b	☑	X	X	☑	☑	☑	X	☑	X	X	☑	☑	X	…	…
Biomechanics	☑	X	X	☑	☑	☑	X	☑	X	X	☑	☑	X	X	☑	…	f

A model of the interaction of both *well behaved* hormones (estradiol and progesterone) with the bilateral anterior knee laxity (averaging both knees), was built by a stepwise regression scheme. Both hormones with all possible integer delays not exceeding 8 days were included and a bidirectional strategy ($p<0.25$) was used. The result is in table 6.13 and figures 6.28 and 6.29. Estradiol does not reveal any delayed interaction but progesterone does: 8 (or 7) days. It is a statistically significant model ($p=0.08\%$) with a low explanatory power ($r^2=50\%$).

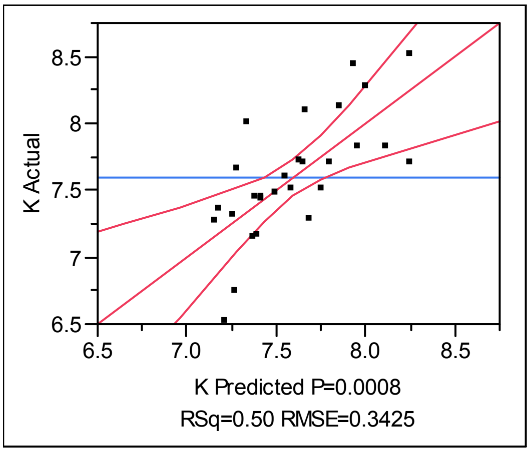


Figure 6.28 Anterior knee laxity as a function of estradiol and progesterone

Table 6.13 Parameter estimates

Term	Estimate	Std Error	t Ratio	Prob>|t|
Intercept	7.0361696	0.139839	50.32	<.0001
Progesterone (delay =8 days)	0.1255552	0.048645	2.58	0.0164
Estradiol (no delay)	0.0063221	0.001873	3.38	0.0025
Progesterone (delay =7 days)	*-0.098982*	*0.051315*	*-1.93*	*0.0657*

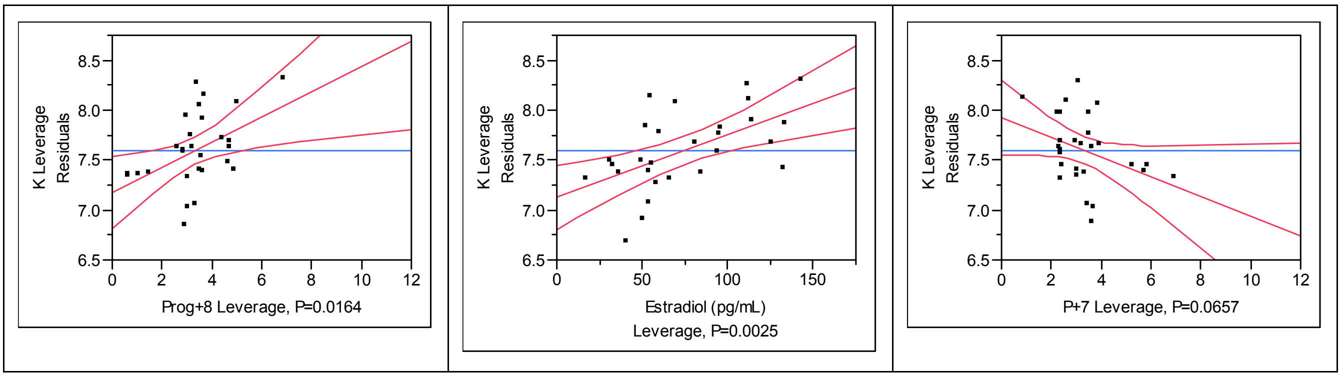


Figure 6.29 Leverage plots for progesterone (delay=8days), estradiol and progesterone (delay=7days)

Another model, of the interaction of both *badly behaved* hormones (relaxin and testosterone) with the bilateral anterior knee laxity (averaging both knees), was built by a stepwise regression scheme identical to the above described. The result is in table 6.14 and figures 6.30 and 6.31. Relaxin with a 2 days' delay and testosterone with both 2 and 4 days' delay are the significant constituents. The model is statistically significant (p=0.38%) with low explanatory power (r^2=57%).

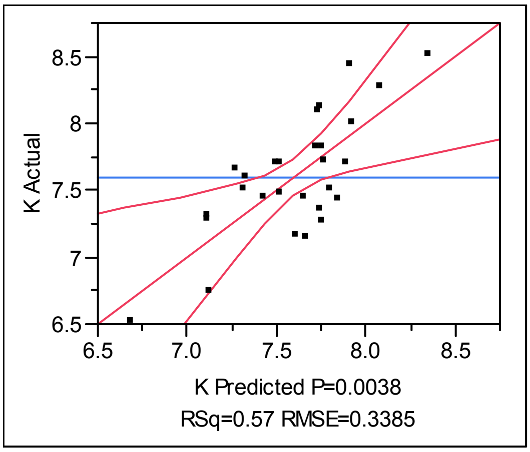


Figure 6.30 Anterior knee laxity as a function of relaxin and testosterone

Table 6.14 Parameter estimates

Term	Estimate	Std Error	t Ratio	Prob>|t|
Intercept	5.7837011	0.687672	8.41	<.0001
Relaxin (delay = 2 days)	-0.061364	0.029856	-2.06	0.0525
Relaxin (delay = 3 days)	*-0.047922*	*0.031878*	*-1.50*	*0.1477*
Relaxin (delay = 6 days)	*0.0579428*	*0.03015*	*1.92*	*0.0683*
Testosterone (delays = 2 days)	0.0259004	0.006947	3.73	0.0012
Testosterone (delays = 4 days)	0.0181454	0.006797	2.67	0.0143
Testosterone (delays = 8 days)	*0.0108539*	*0.006778*	*1.60*	*0.1243*

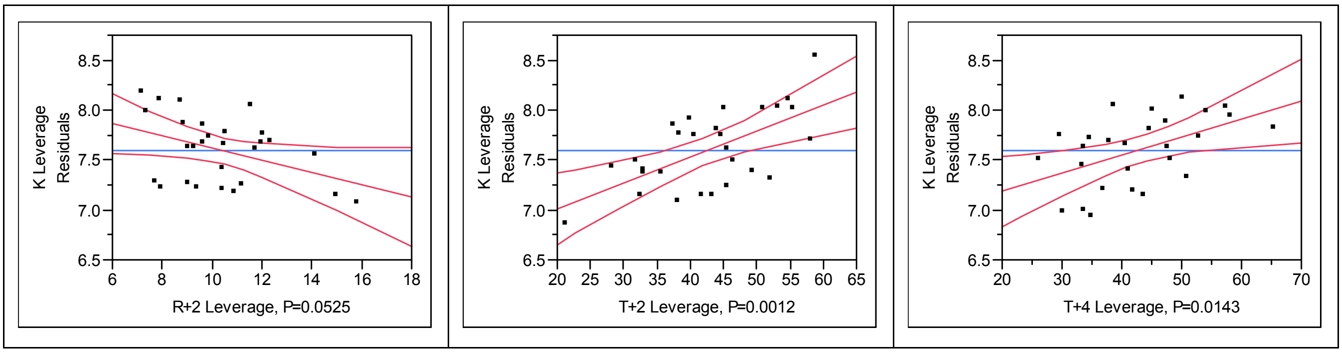


Figure 6.31 Leverage plots for relaxin (delay=2days) and testosterone (delays=2 and 4 days)

Finally, a full model was built, including the effects of all hormones, with all possible delays and also the effects of the jumps (SJ and CMJ). The same methodology as before resulted in the model described in table 6.15 and figures 6.32 and 6.33. Estradiol, relaxin with a four days' delay and testosterone with a two days' delay are the final model variables. The complete model is also statistically significant model (p=0.01%) but again has a very low explanatory power (r^2=60%).

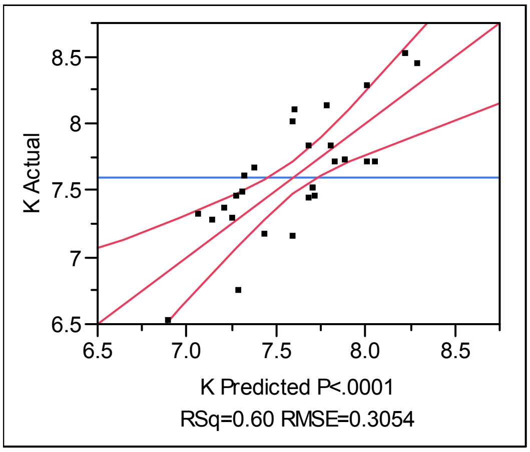


Figure 6.32 Anterior knee laxity as a function of estradiol, relaxin and testosterone

Table 6.15 Parameter estimates

Term	Estimate	Std Error	t Ratio	Prob>|t|
Intercept	5.8618599	0.37353	15.69	<.0001
Estradiol	0.0061875	0.001446	4.28	0.0003
Relaxin (delay= 4 days)	0.0341707	0.026094	1.31	0.2028
Testosterone (delay=2days)	0.0215518	0.005695	3.78	0.0009

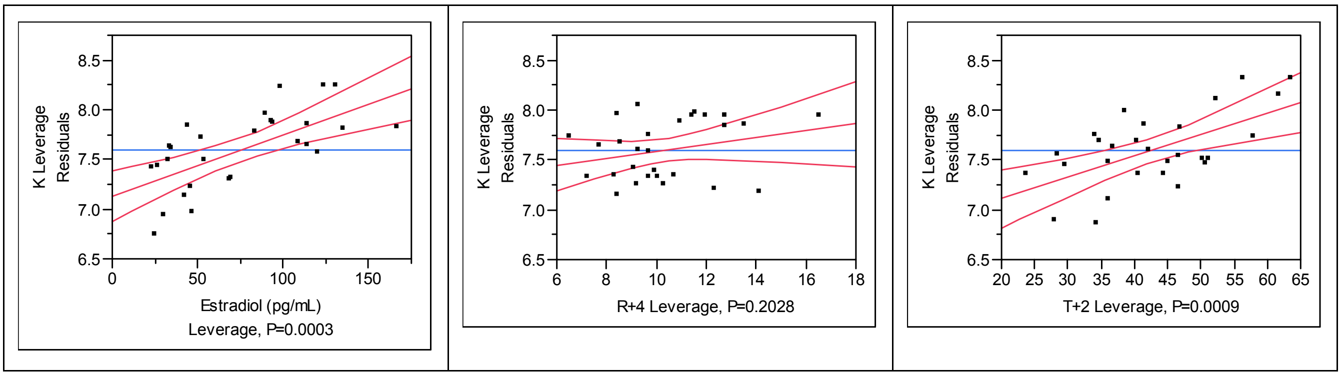


Figure 6.33 Leverage plots for estradiol, relaxin (delay=4days) and testosterone (delay=2 days)

With all the risks described above, we have found very faint correlations (r^2=0.50-0.60) between the serum hormonal content and the anterior knee laxity (figures and tables above), with no significant effects of the jump performance. A full-blown model reveals that testosterone have the most important effect on anterior knee laxity (around 1 mm full scale), followed by estradiol and relaxin. However, the very small explanatory power, the methodological objections on testosterone and relaxin data reduction, the incapability of intervening on the hormonal levels and, ultimately, the incapability of obtaining this data routinely on site, makes us conclude that this hormonal lead is not promising from an injury prevention perspective.

6.5 Conclusions

We found an obvious interaction between OC usage and the serum hormonal levels: estradiol, progesterone and testosterone are reduced by a factor of 3 to 12 while relaxin increases slightly (10%) on OC users.

Untrained non-OC users had 50% higher anterior knee laxity than untrained OC users. However, in trained users there was no visible difference (none above 0.25 mm, or 1%) between OC users and non-users. The hypothesis of a load modulated knee laxity needs further investigation.

There were no differences in jump capacity (none bigger than 8 mm) between OC users and non-users.

Relaxin and testosterone do not show a monthly pattern and so are refractive to data reduction methods. The introduction of delayed interactions reduces drastically the available data.

We developed a few linear and quadratic models, with delayed interactions, between the hormonal time series and the anterior knee laxity (and the jump performance). The models have low explanatory power, in the range of 50% to 60%.

Jump capacity is unrelated to anterior knee laxity.

As OCs do not change the athletes’ knee laxity or jumping performance (hypothesised indicator of ACL active protection capacity), OCs cannot be recommended on a pure ACL injury prevention perspective.

The hormonal lead reveals itself too complex and with very few explanatory or predicting power.

These are good news for coaches: maybe everything is back in their hands. From motor development at early ages to better conditional, technical and tactical training, there could lay the simplest solution to the puzzle – what can be done to avoid the ACL injury?

6.6 References

Arnold, C., Van Bell, C., Rogers, V. & Cooney, T. (2002). The relationship between serum relaxin and knee joint laxity in female athletes. *Orthopedics, 25*(6), 669-673.

Beynnon, B. D., Bernstein, I. M., Belisle, A., Brattbakk, B., Devanny, P., Risinger, R., et al. (2005). The effect of estradiol and progesterone on knee and ankle joint laxity. *Am J Sports Med, 33*(9), 1298-1304.

VIM - Vocabulaire international de métrologie, JCGM 200:2008 C.F.R. (2008).

Burrows, M. & Peters, C. E. (2007). The influence of oral contraceptives on athletic performance in female athletes. *Sports Med, 37*(7), 557-574.

Cammarata, M. L. & Dhaher, Y. Y. (2008). The differential effects of gender, anthropometry, and prior hormonal state on frontal plane knee joint stiffness. *Clin Biomech (Bristol, Avon), 23*(7), 937-945.

Carcia, C. R., Shultz, S. J., Granata, K. P., Gansneder, B. M. & Perrin, D. H. (2004). Knee ligament behavior following a controlled loading protocol does not differ by menstrual cycle day. *Clin Biomech (Bristol, Avon), 19*(10), 1048-1054.

De Souza, M. J. (2003). Menstrual disturbances in athletes: a focus on luteal phase defects. *Med Sci Sports Exerc, 35*(9), 1553-1563.

De Souza, M. J., Miller, B. E., Loucks, A. B., Luciano, A. A., Pescatello, L. S., Campbell, C. G., et al. (1998). High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *J Clin Endocrinol Metab, 83*(12), 4220-4232.

Deie, M., Sakamaki, Y., Sumen, Y., Urabe, Y. & Ikuta, Y. (2002). Anterior knee laxity in young women varies with their menstrual cycle. *Int Orthop, 26*(3), 154-156.

Eiling, E., Bryant, A. L., Petersen, W., Murphy, A. & Hohmann, E. (2006). Effects of menstrual-cycle hormone fluctuations on musculotendinous stiffness and knee joint laxity. *Knee Surg Sports Traumatol Arthrosc*.

Elliott, K. J., Cable, N. T. & Reilly, T. (2005). Does oral contraceptive use affect maximum force production in women? *Br J Sports Med, 39*, 15-19.

Elliott, K. J., Cable, N. T., Reilly, T. & Diver, M. J. (2003). Effect of menstrual cycle phase on the concentration of bioavailable 17-beta oestradiol and testosterone and muscle strength. *Clin Sci (Lond), 105*(6), 663-669.

Fridén, C., Hirschberg, A. L. & Saartok, T. (2003). Muscle strenght and endurance do not significantly vary across 3 phases of menstrual cycle in moderately active premenopausal women. *Clin J Sport Med, 13*, 238-241.

Friden, C., Hirschberg, A. L., Saartok, T. & Renstrom, P. (2006). Knee joint kinaesthesia and neuromuscular coordination during three phases of the menstrual cycle in moderately active women. *Knee Surg Sports Traumatol Arthrosc, 14*(4), 383-389.

Heitz, N. A., Eisenman, P. A., Beck, C. L. & Walker, J. A. (1999). Hormonal Changes Throughout the Menstrual Cycle and Increased Anterior Cruciate Ligament Laxity in Females. *J Athl Train, 34*(2), 144-149.

Hicks-Little, C. A., Thatcher, J. R., Hauth, J. M., Goldfuss, A. J. & Cordova, M. L. (2007). Menstrual cycle stage and oral contraceptive effects on anterior tibial displacement in collegiate female athletes. *J Sports Med Phys Fitness, 47*(2), 255-260.

Lebrun, C. M. (1994). The effect of the phase of the menstrual cycle and the birth control pill on athletic performance. *Clin Sports Med, 13*(2), 419-441.

Loucks, A. B. & Horvath, S. M. (1985). Athletic amenorrhea: a review. *Med Sci Sports Exerc, 17*(1), 56-72.

Lovering, R. M. & Romani, W. A. (2005). Effect of testosterone on the female anterior cruciate ligament. *Am J Physiol Regul Integr Comp Physiol, 289*(1), R15-22.

Martineau, P. A., Al-Jassir, F., Lenczner, E. & Burman, M. L. (2004). Effect of the Oral Contraceptive Pill on Ligamentous Laxity. *Clin J Sport Med 14*(5), 281-286.

Mookerjee, I., Unemori, E. N., Du, X. J., Tregear, G. W. & Samuel, C. S. (2005). Relaxin modulates fibroblast function, collagen production, and matrix metalloproteinase-2 expression by cardiac fibroblasts. *Ann N Y Acad Sci, 1041*, 190-193.

Myklebust, G., Maehlum, S., Holm, I. & Bahr, R. (1998). A prospective cohort study of anterior cruciate ligament injuries in elite Norwegian team handball. *Scand J Med Sci Sports, 8*(3), 149-153.

Ostenberg, A., Roos, E., Ekdahl, C. & Roos, H. (1998). Isokinetic knee extensor strength and functional performance in healthy female soccer players. *Scand J Med Sci Sports, 8*(5 Pt 1), 257-264.

Pokorny, M. J., Smith, T. D., Calus, S. A. & Dennison, E. A. (2000). Self-reported oral contraceptive use and peripheral joint laxity. *J Orthop Sports Phys Ther, 30*(11), 683-692.

Pollard, C. D., Braun, B. & Hamill, J. (2006). Influence of gender, estrogen and exercise on anterior knee laxity. *Clin Biomech (Bristol, Avon), 21*(10), 1060-1066.

Reis, E., Frick, U. & Schmidtbleicher, D. (1995). Frequency variations of strength training sessions triggered by the phases of the menstrual cycle. *Int J Sports Med, 16*(8), 545-550.

Samuel, C. S., Butkus, A., Coghlan, J. P. & Bateman, J. F. (1996). The effect of relaxin on collagen metabolism in the nonpregnant rat pubic symphysis: the influence of estrogen and progesterone in regulating relaxin activity. *Endocrinology, 137*(9), 3884-3890.

Samuel, C. S., Coghlan, J. P. & Bateman, J. F. (1998). Effects of relaxin, pregnancy and parturition on collagen metabolism in the rat pubic symphysis. *J Endocrinol, 159*(1), 117-125.

Sarwar, R., Niclos, B. B. & Rutherford, O. M. (1996). Changes in muscle strength, relaxation rate and fatiguability during the human menstrual cycle. *J Physiol, 493 (Pt 1)*, 267-272.

Shultz, S. J., Kirk, S. E., Johnson, M. L., Sander, T. C. & Perrin, D. H. (2004). Relationship between sex hormones and anterior knee laxity across the menstrual cycle. *Med Sci Sports Exerc, 36*(7), 1165-1174.

Shultz, S. J., Sander, T. C., Kirk, S. E. & Perrin, D. H. (2005). Sex differences in knee joint laxity change across the female menstrual cycle. *J Sports Med Phys Fitness, 45*(4), 594-603.

Smith, K. D., Rodriguez-Rigau, L. J., Tcholakian, R. K. & Steinberger, E. (1979). The relation between plasma testosterone levels and the lengths of phases of the menstrual cycle. *Fertil Steril, 32*(4), 403-407.

Stewart, D. R., Celniker, A. C., Taylor, C. A., Jr., Cragun, J. R., Overstreet, J. W. & Lasley, B. L. (1990). Relaxin in the peri-implantation period. *J Clin Endocrinol Metab, 70*(6), 1771-1773.

Tipton, C. M., Tcheng, T. K. & Mergner, W. (1971). Ligamentous strength measurements from hypophysectomized rats. *Am J Physiol, 221*(4), 1144-1150.

Unemori, E. N. & Amento, E. P. (1990). Relaxin modulates synthesis and secretion of procollagenase and collagen by human dermal fibroblasts. *J Biol Chem, 265*(18), 10681-10685.

Unemori, E. N., Beck, L. S., Lee, W. P., Xu, Y., Siegel, M., Keller, G., et al. (1993). Human relaxin decreases collagen accumulation in vivo in two rodent models of fibrosis. *J Invest Dermatol, 101*(3), 280-285.

van der Westhuizen, E. T., Halls, M. L., Samuel, C. S., Bathgate, R. A., Unemori, E. N., Sutton, S. W., et al. (2008). Relaxin family peptide receptors - from orphans to therapeutic targets. *Drug Discov Today, 13*(15-16), 640-651.

Van Lunen, B. L., Roberts, J., Branch, J. D. & Dowling, E. A. (2003). Association of Menstrual-Cycle Hormone Changes with Anterior Cruciate Ligament Laxity Measurements. *J Athl Train, 38*(4), 298-303.

Wajchenberg, B. L., Marcondes, J. A., Mathor, M. B., Achando, S. S., Germak, O. A. & Kirschner, M. A. (1989). Free testosterone levels during the menstrual cycle in obese versus normal women. *Fertil Steril, 51*(3), 535-537.
Wreje, U., Kristiansson, P., Aberg, H., Bystrom, B. & von Schoultz, B. (1995). Serum levels of relaxin during the menstrual cycle and oral contraceptive use. *Gynecol Obstet Invest, 39*(3), 197-200.
Yu, W. D., Panossian, V., Hatch, J. D., Liu, S. H. & Finerman, G. A. (2001). Combined effects of estrogen and progesterone on the anterior cruciate ligament. *Clin Orthop Relat Res*(383), 268-281.

Chapter 7

Conclusions

7.1 Conclusions

We showed that the ACL rupture is the injury with the highest impact in the Portuguese female handball population, affecting mostly young players.

ACL ruptures occur mostly at the age of specialization and entry at the highest competing level.

They affect 17% off all senior players. Even years after reconstructive surgery, the affected knee has not yet fully recovered its isokinetic strength and, in the majority of cases, it still has a higher laxity than the contralateral knee. The possibility of premature osteoarthritis is high, according to the literature.

The most promising aspect is that the injuries occur mostly during games (75%) although the players, on average, are exposed to 16 times more practice time than game time. The high BMI and body fat content of the Portuguese players, combined with a low jumping capability indicate that there is room for improvement in conditional (and technical/tactical) capabilities. These improvements could lead to a decrease in the injury incidence at large and to the ACL's in particular.

The isokinetic measures reinforce the importance of lower limb strength in reducing the ACL injury risk. The isokinetic methodology is probably not the best suited to evaluate this in healthy players but is a common accompanying tool in the recovery of athletes after knee surgery. Although the H:Q concept is fragile and the H measures are of more value by themselves, the importance of the dynamic knee stabilization mechanism in ACL protection is reinforced in this study.

High anterior knee laxity is very likely related to a high knee injury risk, as it is a direct measure of passive knee instability. We have tried to establish links of this laxity with several other factors, with mixed results.

There is probably a role played by the hormonal environment in the high female ACL injury risk. Probably the practice exposure, anatomical, social, psychological and a few more factors contribute to the general risk. Unfortunately, it is very difficult to isolate any of these and attribute them a single contribution figure. We were unable to solve this problem satisfactorily, having found contradicting results in the role of sex hormones or OC usage in anterior knee laxity.

7.2 Perspectives

We think that there are three main lines of intervention for ACL injury risk reduction.

The usage of the Nordic injury prevention philosophy, at a very early age, is a tool for postural and movement education that should be pursued. It has showed results that we feel would be much greater if applied at the proper age.

The information of athletes is another big injury prevention factor. Players should know more about the injury, the risks, consequences and predisposing factors. This is also key to obtain their commitment in any *boring* prevention scheme.

Finally, as the load/exposition is probably the most important factor of all, it is advisable that the selection criteria, the training and the long-term career management should consider injury risk. The training changes, both from a conditional and a tactical point of view, are the fastest and more promising routes for intervention future research.

Appendices

Handouts for participants

U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

QUESTIONÁRIO NÚMERO (não preencher) ☐☐☐

QUESTIONÁRIO

Estimada Atleta. O presente questionário é um instrumento de trabalho elaborado para o estudo das **LESÕES** no **Andebol Feminino em Portugal** e sua posterior prevenção. Para o que a sua ajuda é imprescindível.

Investigadores: Mestre Maria Luísa Estriga, docente da Faculdade de Desporto-UP;
Prof. Doutor Leandro Massada, médico especialista em Ortopedia e Medicina Desportiva e docente da FADE-UP;
Prof. Doutor João Bernardes, médico especialista em Ginecologia e Obstetrícia (Faculdade de Medicina do Porto).

Contacto em caso de dúvidas: Luísa Estriga (93******* ou 225074770 ou lestriga@fade.up.pt)

1. IDENTIFICAÇÃO

DATA DE PREENCHIMENTO: ☐☐ / ☐☐ / ☐☐☐☐

1. Nome:

2. Data de nascimento: ☐☐ / ☐☐ / ☐☐☐☐

3. TLF/TLM:

4. E-mail: ______

5. Peso (Kg): ☐☐☐ Altura (cm): ☐☐☐

6. Mão dominante: Direita ☐ Esquerda ☐ Ambas ☐

2. CURRICULUM DESPORTIVO

ANDEBOL

1. Prática de Andebol:

	Idade de iniciação	Número de anos
Desporto escolar		
Federado		

2. Clubes que representou:

Clube ______ com idade: ______
Clube ______ com idade: ______
Clube ______ com idade: ______
Clube ______ com idade: ______
Clube ______ com idade: ______
Clube ______ com idade: ______

3. Selecção: Regional ☐ Nacional ☐

Regional em que idade? ______

Nacional em que idade? ______

Na actualidade integra alguma selecção: Sim ☐ Não ☐

Se sim, qual(ais)? ______

OUTRAS MODALIDADES

4. Prática de outras modalidades a nível federado; quais e em que idade?

Modalidade ______ com idade: ______
Modalidade ______ com idade: ______

5. Prática de outras modalidades a nível recreativo / escolar; quais e em que idade?

Modalidade ______ com idade: ______
Modalidade ______ com idade: ______
Modalidade ______ com idade: ______

3. ÉPOCA DESPORTIVA 2006/2007

1. Clube actual:

2. Escalão (ões) onde habitualmente joga: (no caso de jogar em dois escalões: marque com **1** o escalão em que joga com mais frequência e com **2** o outro escalão)

Infantil ☐ Iniciadas ☐ Juvenil ☐ Júnior ☐ Sénior ☐

3. Em que fase(s) de jogo joga frequentemente:

Clube	Ataque ☐	Defesa ☐	Ambas ☐
Selecção	Ataque ☐	Defesa ☐	Ambas ☐

4. Posição (ões) que geralmente ocupa no Ataque e na Defesa: Se joga apenas num escalão marque nas colunas identificadas com **1**; se joga em dois escalões utilize a coluna **1** para o escalão em que joga com mais frequência e a **2** para o outro escalão.

Ataque	1	2	Selecção	Defesa	1	2	Selecção
Ponta esquerdo				Ponta esquerdo			
Lateral esquerdo				Lateral esquerdo			
Central				Defesa central			
Lateral direito				Lateral direito			
Ponta direito				Ponta direito			
Pivot				Defesa avançado			

5. Total de tempo que, em média, joga por jogo (Clube – escalão 1 e escalão 2 / Selecção):

Tempo	1	2	Selecção
45 a 60 minutos			
30 a 44 minutos			
15 a 29 minutos			
Menos de 15 minutos			

6. Treina habitualmente com a equipa do clube que representa? Sim ☐ Não ☐
Se não, Porquê? ______

7. Carga de treino semanal (em média) no Clube:

		1	2
Pavilhão/Ringue	Número de treinos		
	Total de horas		
Musculação (trabalho com máquinas e/ou pesos livres)	Número de treinos		
	Total de horas		

8. Realiza trabalho de flexibilidade / alongamentos:

Clube	Sempre ☐	Algumas vezes ☐	Nunca ☐
Selecção	Sempre ☐	Algumas vezes ☐	Nunca ☐

9. Se respondeu sempre ou algumas vezes na questão anterior, fá-lo no (Clube / Selecção):

	Clube	Selecção
Início do treino		
Durante o treino		
Final do treino		
Extra treino		

10. Em que tipo de piso habitualmente treina:

Sintético ☐ Madeira – *comcaixa-de-ar* ☐
Cimento ☐ Madeira – *sem caixa-de-ar* ☐
Outro: ☐ Qual? ______

U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

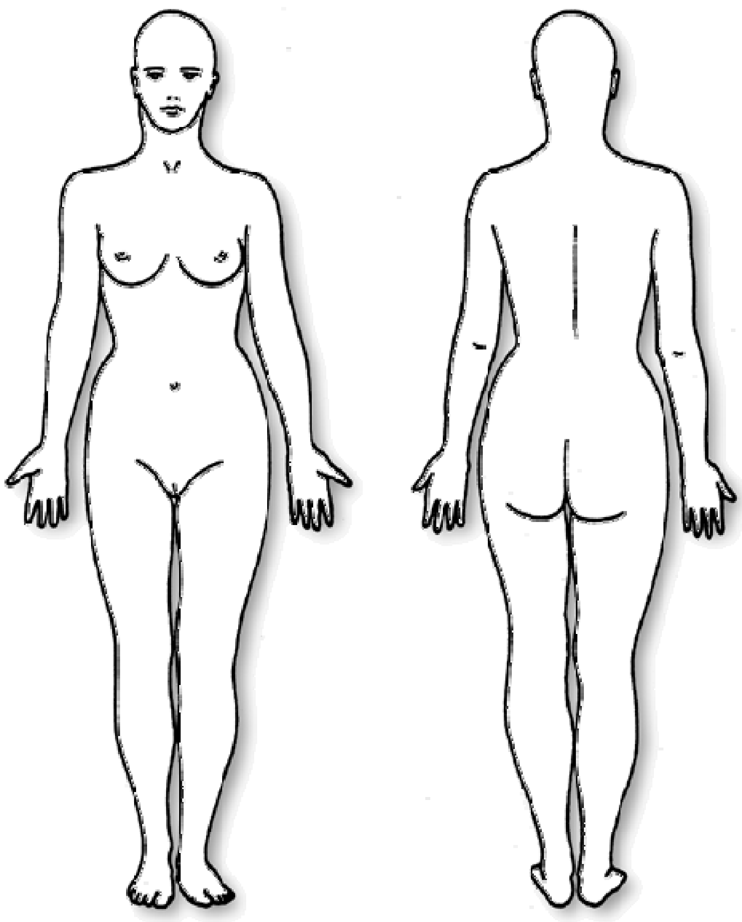
QUESTIONÁRIO NÚMERO (não preencher) ☐☐☐

4. LESÕES DESPORTIVAS DURANTE A CARREIRA DE ANDEBOLISTA

Por LESÃO entenda-se todo e qualquer traumatismo adquirido durante a prática do andebol que tenha condicionado posteriormente a continuação dessa prática.

Refira TODAS as LESÕES que se recorda ter sofrido até ao presente momento e aponte, com uma seta (→), na figura abaixo o local exacto da lesão identificando-a por escrito (*exemplo: entorse no tornozelo*) e com uma cruz (X) o local da dor, no caso de dor localizada, e com um sombreado no caso da dor ser generalizada.

Idade	**Lesão: tipo de lesão** (ex. entorse, rotura, tendinite, luxação) e **órgão afectado** (ex.tornozelo, tendão rotuliano, menisco externo)	Direito /Esquerdo	Nº de episódios de lesão	Operação **(Sim / Não)**	Tempo sem treinar / jogar	Na actualidade ainda lhe condiciona a prática do andebol **(Sim/Não)**



U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

QUESTIONÁRIO NÚMERO (não preencher) ☐☐☐

Nome: ______________________________

6. LESÕES DESPORTIVAS

LESÃO: ______________________________

1. Mês de ocorrência da lesão: ____________________

2. Hora do dia (escala de 1 a 24 horas): ☐☐ Hora(s)

3. Local (pavilhão) onde ocorreu a lesão? ____________________

4. Faça uma descrição detalhada das circunstâncias em que se lesionou: _______

____

____

CLASSIFICAÇÃO/DIAGNÓSTICO DA LESÃO

5. Lado: **Direito** ☐ **Esquerdo** ☐

6. Natureza: **Aguda (traumática, num momento)** ☐ **Crónica (progressiva)** ☐

7. Local:

Cabeça / Face	☐	Mão	☐
Coluna	☐	Dedos	☐
Esterno / Costelas	☐	Polegar	☐
Zona abdominal	☐	Anca	☐
Pélvis / Sacro	☐	Coxa	☐
Ombro	☐	Joelho	☐
Braço	☐	Perna	☐
Cotovelo	☐	Tornozelo	☐
Antebraço	☐	Pé	☐
Punho	☐	Outro: Qual? ____________	☐

8. Nome específico do órgão (osso, músculo, tendão ou ligamento):

____

9. Diagnóstico:

Contusão (pancada ou queda)			☐
Lesões articulares	Entorse sem lesão ligamentar		☐
	Entorse com lesão ligamentar		☐
	Entorse com lesão meniscal		☐
	Sub - luxação		☐
	Luxação		☐
Lesões tendinosas	Tendinites		☐
	Roturas tendinosas		☐
Lesões musculares	Contusão muscular		☐
	Rotura muscular		☐
Fracturas	Agudas		☐
	De fadiga		☐
Lesões da coluna vertebral	Agudas	Cervical	☐
		Dorsal	☐
		Lombar	☐
	Crónicas	Cervicalgia	☐
		Dorsalgia	☐
		Lombalgia	☐
Lesões da pele	Lacerações cutâneas		☐
Outra lesão: qual?			☐

GRAVIDADE DA LESÃO

10. Imediatamente à ocorrência da lesão continuou a actividade desportiva? **Sim** ☐ **Não** ☐

Se continuou, fê-lo: **Com limitações** ☐ **Sem limitações** ☐

11. Teve necessidade de intervenção médica? **Sim** ☐ **Não** ☐

Se SIM responda às questões 11.1 e 11.2, se NÃO passe à questão 12

11.1. Imediatamente após a lesão? **Sim** ☐ **Não** ☐

Especifique: ______________________________

11.2. Posteriormente teve necessidade de:

Operação	**Sim**	☐	**Não**	☐
Fisioterapia	**Sim**	☐	**Não**	☐
Outro: especifique: ____________	**Sim**	☐	**Não**	☐

12. Recuperava de outra lesão? **Sim** ☐ **Não** ☐

Se sim, de qual e de que lado (direito/esquerdo)?

____

13. A lesão é reincidente? Ou seja, já se lesionou anteriormente no mesmo local e com os mesmos sintomas? **Sim** ☐ **Não** ☐

Se sim, explique as diferenças?

____

14. Dias sem treinar e/ou jogar:

0 dias	☐	2 a 4 semanas	☐
1 ou 2 dias	☐	4 a 12 semanas	☐
3 dias a 2 semanas	☐	Mais tempo: quanto?_______	☐

15. Teve actividade desportiva condicionada? **Sim** ☐ **Não** ☐

Se sim, durante quanto tempo? ____________

CIRCUNSTÂNCIAS DA LESÃO

16. Actividade e momento da lesão:

No domínio	Do clube	Treino	☐
		Jogo treino	☐
		Competição regional	☐
		Competição nacional	☐
		Competição internacional	☐
	Da selecção	Treino	☐
		Jogo treino	☐
		Competição nacional	☐
		Competição internacional	☐
Jogo	Activação		☐
	Primeira parte	0 – 10 minutos	☐
		11 – 20 minutos	☐
		21 – 30 minutos	☐
	Intervalo		☐
	Segunda parte	31 – 40 minutos	☐
		41 – 50 minutos	☐
		51 – 60 minutos	☐
Treino/Jogo treino	Activação inicial		☐
	Exercício		☐
	Jogo		☐
Outra ☐	Qual?		

Se lesão ocorreu num Jogo ou Jogo Treino passar à questão seguinte.
Se lesão ocorreu num Treino passar à questão nº. 18

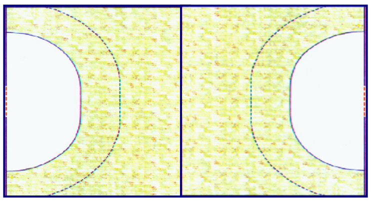
U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

QUESTIONÁRIO NÚMERO (não preencher)

17. Se a lesão ocorreu num JOGO OFICIAL ou num JOGO TREINO:

Grau de **importância** do jogo	Decisivo	☐
	Muito importante	☐
	Importante	☐
	Pouco importante	☐
	Nada importante	☐
Fase do jogo	Ataque posicional	☐
	Contra-ataque/ataque rápido	☐
	Recuperação defensiva	☐
	Defesa posicional	☐

Assinale com uma cruz (**X**) o local do campo **onde ocorreu a lesão**



Defesa Ataque

Assinale no **cronograma:**
- com uma linha os momentos do jogo em que esteve a jogar
- com uma cruz o momento em que se lesionou

Primeira parte:

5' 10' 15' 20' 25'

Segunda parte:

35' 40' 45' 50' 55'

18. A lesão ocorreu (assinale as várias respostas certas):

Em posse da bola	☐	Em impulsão para salto	☐
Em contacto com o adversário	☐	Em recepção de um salto	☐
A sofrer falta sancionada pelo árbitro	☐	Num movimento brusco	☐
A fazer falta sancionada pelo árbitro	☐	Por movimentos repetidos	☐
Houve sanção disciplinar	☐	Acção agressiva do adversário	☐
Numa mudança de direcção	☐	Em contacto com um obstáculo:	☐
Outro: Qual?			☐

19. Descreva os equipamentos de protecção utilizados na altura da lesão (Direita / Esquerda):

	Descrição – lado **Direito**	Descrição – lado **Esquerdo**
Ombro		
Cotovelo		
Pulso		
Dedos da mão		
Coxa		
Joelho		
Pé		
Outro		
Coluna		

Se a lesão não atingiu o JOEHO ou o TORNOZELO passe à questão 22.

20. Piso utilizado na altura da lesão:

Sintético	☐	Madeira – *comcaixa-de-ar*	☐
Cimento	☐	Madeira – *sem caixa-de-ar*	☐
Outro:	☐	Qual? ______	

21. Calçado utilizado na altura da lesão:

Estado:	Bom	☐	**Modelo:**	Andebol	☐
	Razoável	☐		Outros Indoor	☐
	Mau	☐		Outros	☐

Marca:

CARACTERIZAÇÃO da DOR

22. Na realização de que habilidade técnica surgiu a dor (assinale as várias respostas correctas):

Passe	☐	Recepção de salto	☐
Remate	☐	Impulsão para salto	☐
Queda	☐	Intercepção de uma bola - Bloco	☐
Deslocamento defensivo	☐	Defesa - Guarda-redes	☐
Outra(s): qual(ais)?______			☐

23. Na Actualidade ainda sente DOR? **Sim** ☐ **Não** ☐

Se NÃO passe à questão 24; se SIM responda às questões 23.1 e 23.2

23.1. Como se caracteriza a dor:

Dor que aparece após a actividade desportiva	☐
Dor que aparece durante a actividade desportiva	☐
Dor em repouso que desaparece durante a actividade desportiva	☐
Dor em repouso que se mantém durante a actividade desportiva	☐
Outra: qual?______	☐

23.2. Na realização de que habilidade técnica ainda sente dor (assinale as várias respostas correctas):

Passe	☐	Recepção de salto	☐
Remate	☐	Impulsão para salto	☐
Queda	☐	Intercepção de uma bola - Bloco	☐
Deslocamento defensivo	☐	Defesa - Guarda-redes	☐
Outra(s): qual(ais)?______			☐

ESTADO EMOCIONAL/PSICOLÓGICO

24. Na altura da lesão como se sentia a nível psicológico (Ansiosa, Depressiva, Irritada, Normal, etc.)?

______

25. Na altura da lesão passava por alguma situação conflituosa com familiares, amigos, colegas de trabalho ou equipa ou com o treinador? Se sim, com quem e em que sentido?

______

CICLO MENSTRUAL

Se a lesão não atingiu o JOELHO ou o TORNOZELO passe à questão 28

26. Na fase da lesão tomava algum tipo de contraceptivo oral? **Sim** ☐ **Não** ☐

27. Fase do ciclo menstrual em que se encontrava?

Pré-menstrual	☐	Menstrual	☐
Pós-menstrual	☐	Não sei	☐

NA ACTUALIDADE

28. Na actualidade esta lesão ainda lhe condiciona a prática desportiva/andebol? **Sim** ☐ **Não** ☐

Se sim, de que forma? ______

Muito obrigada pela colaboração!

QUESTIONÁRIO NÚMERO (não preencher) ☐☐☐

Nome: ______________________________ **Data de Nascimento:** ____/____/________
Clube: ______________________________ **Data de preenchimento do questionário:** ____/____/________

5. CICLO MENSTRUAL

Instruções de preenchimento:
Deve ler, com muita atenção, as questões colocadas e esclarecer eventuais dúvidas com a investigadora.
As questões têm espaços próprios para responder, mas se necessário pode utilizar outros espaços, que deve identificar devidamente.
Em função das questões são apresentados espaços para resposta com vista à marcação de uma cruz (X) ou preenchimento numérico ou por extenso.

1. Idade da menarca (Primeira menstruação): ☐☐

2. O seu ciclo menstrual costuma ser regular:

Sempre	☐
Algumas vezes	☐
Nunca	☐

Se respondeu **Nunca** ou **Às vezes** como o caracteriza?
____
____

3. Duração média do Ciclo Menstrual (em dias): ☐☐

Nota: **Um ciclo** começa no primeiro dia do aparecimento da menstruação e termina quando se inicia novo ciclo, ou seja, o 1º dia de menstruação seguinte.

4. Duração média do Período Menstrual (sangramento) (em dias): ☐☐

5. Nos últimos 6 meses, antes de participar no estudo, quantas vezes teve o período? ☐☐

6. Toma algum tipo de contraceptivos (ex.: pílula)? **S** **N**

Se sim, qual? (nome comercial, ex.: Diane-35, Gynera, Minigeste, Miranova)
____

Há quanto tempo (anos/meses) **toma/usa contraceptivos orais:** ________________

E porque motivo(s) começou a tomar/usar contraceptivos: ________________
____

Outras informações que considere relevante acerca deste assunto (por exemplo: se teve necessidade de mudar de contraceptivos orais): ________________
____
____

7. Nos últimos 6 meses antes de participar no estudo tomou algum tipo de contraceptivos? **S** **N**

Se sim, qual? ______________________________

8. Toma algum medicamento regularmente? **S** **N**

Se sim, qual? (nome comercial) ______________________________

Outras informações importantes acerca da sua saúde: ________________
____
____

9. Fuma e / ou bebe álcool?

Fuma	**S**	**N**
Bebe álcool	**S**	**N**

Se sim, explique com que regularidade (diária, semanal, mensal ou outra) **e em que quantidade por dia?**

Fumo: ______________________________

Bebo: ______________________________

10. No contexto do treino/jogo, quando está com o período sente-se:

Limitada	☐	Inibida	☐	Normal	☐
Bem	☐	Mal disposta	☐		

11. Durante o pré-menstrual (PM) ou período menstrual (P) quais os sintomas que se manifestam com maior incidência:

Irritabilidade	**PM**	**P**	Diminuição do desejo sexual	**PM**	**P**
Insónia	**PM**	**P**	Aumento de peso	**PM**	**P**
Acne	**PM**	**P**	Dificuldades de concentração	**PM**	**P**
Nervosismo	**PM**	**P**	Inchaço ou dor abdominal	**PM**	**P**
Depressão	**PM**	**P**	Pele oleosa	**PM**	**P**
Sono	**PM**	**P**	Mastalgia (Inchaço / dores no peito)	**PM**	**P**
Fadiga / Cansaço	**PM**	**P**	Cefaleias (dores de cabeça)	**PM**	**P**
Apetite	**PM**	**P**	Dores nas articulações	**PM**	**P**
Falta de apetite	**PM**	**P**	Inchaço ou dor nas pernas	**PM**	**P**
Tonturas	**PM**	**P**	Dores lombares ("nos rins")	**PM**	**P**
Ansiedade	**PM**	**P**	Outro: Qual? ______________	**PM**	**P**

12. Durante o seu ciclo menstrual percepciona diferenças no seu rendimento desportivo?

	Aumento de Rendimento	Não se altera	Diminuição de Rendimento
Fase pré-menstrual			
Fase menstrual (período)			
Fase pós-menstrual			

Explique as alterações que assinalou (exemplos: variações na força, velocidade, concentração, etc.):
____
____

13. Alguma vez deixou de treinar / jogar por motivos inerentes ao período menstrual?

Treinar	**S**	**N**
Jogar	**S**	**N**

Se sim, **indique as razões** dessa paragem:
____
____

Muito Obrigada pela colaboração

U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

QUESTIONÁRIO NÚMERO (não preencher) ☐☐☐

ANEXO 1: LESÕES NO JOELHO

LESÃO – identificação da lesão e joelho (esquerdo / direito):

____

____

1. Época e idade da ocorrência da lesão:

____

2. Actividade no momento da lesão:

Treino	☐
Jogo de treino	☐
Competição oficial	☐

3. A lesão ocorreu (assinale as várias respostas certas):

Em posse da bola	☐
Em contacto com o adversário	☐
Numa mudança de direcção	☐
Em impulsão para salto	☐
Em recepção de um salto	☐
Outros – quais?	

4. Piso utilizado na altura da lesão:

Sintético		☐
Madeira	Com caixa-de-ar	☐
	Sem caixa-de-ar	☐
Cimento		☐
Outro	Qual?	

5. Gravidade da lesão (tempo sem jogar):

0 dias	☐	2 a 4 semanas	☐
1 ou 2 dias	☐	4 a 12 semanas	☐
3 dias a 2 semanas	☐	Mais tempo: quanto?	

6. Foi operada? Sim ☐ Não ☐

7. Esta lesão ainda lhe condiciona a actividade desportiva? Sim ☐ Não ☐

Se sim, de que forma?

____

____

____

8. Outras informações significativas relativas às condições de ocorrência da lesão, sintomas e recuperação:

____

____

____

ANEXO 1: LESÕES NO JOELHO

LESÃO – identificação da lesão e joelho (esquerdo / direito):

1. Época e idade da ocorrência da lesão:

2. Actividade no momento da lesão:

Treino	☐
Jogo de treino	☐
Competição oficial	☐

3. A lesão ocorreu (assinale as várias respostas certas):

Em posse da bola	☐
Em contacto com o adversário	☐
Numa mudança de direcção	☐
Em impulsão para salto	☐
Em recepção de um salto	☐
Outros – quais?	

4. Piso utilizado na altura da lesão:

Sintético		☐
Madeira	Com caixa-de-ar	☐
	Sem caixa-de-ar	☐
Cimento		☐
Outro	Qual?	

5. Gravidade da lesão (tempo sem jogar):

0 dias	☐	2 a 4 semanas	☐
1 ou 2 dias	☐	4 a 12 semanas	☐
3 dias a 2 semanas	☐	Mais tempo: quanto?	

6. Foi operada? Sim ☐ Não ☐

7. Esta lesão ainda lhe condiciona a actividade desportiva? Sim ☐ Não ☐

Se sim, de que forma?

8. Outras informações significativas relativas às condições de ocorrência da lesão, sintomas e recuperação:

Ex.ma

Atleta

Novembro 04, 2004

Assunto: informação acerca do estudo centrado nas lesões no andebol feminino.

Informamos que os gabinetes de Traumatologia do Desporto e de Andebol da Faculdade de Ciências do Desporto e Educação Física pretendem realizar uma avaliação clínica e morfo-funcional às atletas de andebol feminino no âmbito do estudo acerca das lesões nesta modalidade.

A avaliação referida não contempla qualquer teste de natureza invasiva ou cruente. Trata-se tão-somente de uma avaliação somática (ex.: peso, % de massa gorda, altura), de força e de flexibilidade ao nível dos membros inferiores e clínica (ex.: desvio das rótulas, laxidez ligamentar ao nível do joelho). As medições serão realizados por técnicos especializados e um médico ortopedista/investigador.

A situação de avaliação implica que as atletas se desloquem às Faculdade de Ciências do Desporto e que sejam medidas com vestuário apropriado (fato de banho ou biquini, ou calções e um *top*).

Os melhores cumprimentos

(Prof. Doutor Leandro Massada, responsável pelo Gabinete de Traumatologia e pelo estudo)

✂---

Declaração

A atleta (nome)____

declara que aceita participar no estudo acerca das lesões no andebol feminino.

Data:___/___/2004 ____

(Assinatura da Atleta)

Ex.$^{\underline{mo}}$

Encarregado de Educação da Atleta

____

Novembro 04, 2004

Assunto: informação acerca do estudo centrado nas lesões no andebol feminino.

Informamos que os Gabinetes de Traumatologia do Desporto e de Andebol da Faculdade de Ciências do Desporto e Educação Física pretendem realizar uma avaliação clínica e morfo-funcional às atletas de andebol feminino no âmbito do estudo acerca das lesões nesta modalidade.

A avaliação referida não contempla qualquer teste de natureza invasiva ou cruente. Trata-se tão-somente de uma avaliação somática (ex.: peso, % de massa gorda, altura), de força e de flexibilidade ao nível dos membros inferiores e clínica (ex.: desvio das rótulas, laxidez ligamentar ao nível do joelho). As medições serão realizados por técnicos especializados e um médico ortopedista/investigador.

A situação de avaliação implica que as atletas se desloquem às Faculdade de Ciências do Desporto e que sejam medidas com vestuário apropriado (fato de banho ou biquini, ou calções e um *top*).

Os melhores cumprimentos

(Prof. Doutor Leandro Massada, responsável pelo Gabinete de Traumatologia e pelo estudo)

✂--

Autorizo que a minha filha realize a referida avaliação:

O Encarregado de Educação da atleta ____

Data:___/___/2004 ____

(Assinatura do Encarregado de Educação)

U. PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

DECLARAÇÃO DE CONSENTIMENTO INFORMADO

Titulo do Projecto: Estudo acerca do perfil lesional e ginecológico das atletas de Andebol e da força muscular e estabilidade ao nível da articulação do joelho.

Por favor, leia atentamente a Declaração de Consentimento antes de decidir participar no estudo.

Objectivo do estudo: Pretendemos observar se existe uma relação entre a estabilidade da articulação do joelho, o perfil ginecológico da atleta, a força muscular e a lesão do Ligamento Cruzado Anterior (LCA). Para este propósito, propomo-nos avaliar a capacidade de resposta do LCA à aplicação de uma força com intensidade crescente (de ligeira a moderada), ou seja, a translação (deslocação) anterior da tíbia em relação ao fémur, em atletas de diferentes idades, com e sem lesão ao nível do LCA e com e sem efeito de contraceptivos orais (vulgar pílula).

Importância do estudo: A importância deste estudo decorre de uma possível relação entre níveis elevados de laxidez, deficits de força muscular e a incidência da lesão do LCA.

Tempo requerido e local das avaliações: são necessários cerca de 15 minutos para avaliar a laxidez de ambos os joelhos, com um instrumento desenvolvido para o efeito (KT-1000), e para avaliar os saltos verticais, com o tapete “Ergo Jump” (plataforma de contactos). As respectivas medições serão executadas por técnicos especializados, no local de treino e em horário/momento a aferir com a atleta e o(a) treinador(a).

Procedimentos: Antes de se iniciarem as avaliações propriamente ditas, as atletas preencherão um questionário acerca do seu perfil lesional e ginecológico e será realizada (se justificado) uma avaliação clínica à articulação do joelho (por médico especialista), no sentido de se verificar e garantir a ausência de qualquer patologia associada a esta articulação.

Riscos: A laxidez e a força muscular serão avaliadas com instrumentos específicos desenvolvidos para o efeito, de natureza não invasiva, cujos riscos potenciais de lesão são praticamente inexistentes.

Confidencialidade: Será garantida a confidencialidade de toda a informação recolhida. A cada atleta será atribuído um código (número); quando o estudo terminar e os dados forem analisados, a lista de códigos com a identificação das atletas será destruída. Os dados recolhidos poderão a vir a ser utilizados em estudos futuros, porém não contendo qualquer informação de identificação pessoal dos sujeitos. O nome da atleta não será usado em qualquer relatório relacionado com este estudo.

Participação Voluntária: A participação nesta investigação é absolutamente voluntária.

Desistência do Estudo: A atleta pode cessar a sua participação no estudo a qualquer momento, sem qualquer tipo de penalização. Se a atleta pretender abandonar o estudo deve informar o investigador e as avaliações terminam imediatamente.

Investigadores: Prof. Doutor Leandro Massada, médico especialista em Ortopedia e Medicina Desportiva e docente da FADE-UP; Prof. Doutor João Bernardes, médico especialista em Ginecologia e Obstetrícia; e Mestre Maria Luísa Estriga, docente da FADE-UP.

Contacto em caso de dúvidas acerca do estudo: Luísa Estriga (93******* ou 225074770)

U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

Declaração

A atleta (nome) ____ declara que leu e entendeu esta **Declaração de Consentimento Informado** e que aceita participar no estudo acerca do perfil e ginecológico das atletas de Andebol e da estabilidade ao nível da articulação do joelho, de acordo com o descrito nesta declaração.

Opcional: permito que os dados recolhidos sejam facultados ao _______________ (treinador e/ou médico) mediante solicitação _______ (sim/não)

Atleta com idade inferior a 18 anos:

A validade deste consentimento expira quando a atleta atinge os 18 anos.

Assinatura da Atleta: ____ Data: __/__/______
*Assinatura do Enc. de Educação: ____ Data: __/__/______

* Confirmo que tenho autoridade legal para assinar esta declaração em nome da Atleta menor.

A Atleta e/ou Encarregado de Educação receberá uma cópia desta Declaração

U. PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

DECLARAÇÃO DE CONSENTIMENTO INFORMADO

Titulo do Projecto: Estudo do impacto das variações hormonais do ciclo menstrual na estabilidade da articulação do joelho e na força muscular.

Por favor, leia atentamente a <u>Declaração de Consentimento</u> antes de decidir participar no estudo.

Objectivo do estudo: (1) Pretendemos observar se o Ligamento Cruzado Anterior (LCA) apresenta alterações da laxidez ao longo do ciclo menstrual, em atletas com e sem efeito de contraceptivos orais (vulgar pílula). O LCA funciona como elemento estabilizador do joelho em todos os seus movimentos (translações e rotações). Para que o LCA rompa deverá haver um excesso de translação anterior da tíbia ou a rotação do fémur em relação à tíbia (movimento vulgarmente designado de entorse). Neste estudo pretendemos avaliar a capacidade de resposta do LCA à aplicação de uma força com intensidade crescente (de ligeira a moderada), ou seja, a translação (deslocação) anterior da tíbia em relação ao fémur. **(2)** Objectivamos, também, verificar se as variações hormonais produzidas ao longo do ciclo menstrual afectam a capacidade de salto das atletas. A capacidade força ao nível dos membros inferiores (particularmente dos músculos anteriores e posteriores da coxa) é fundamental para a estabilização da articulação do joelho no desempenho das habilidades técnicas específicas do andebol. Os dados recolhidos serão utilizados para comparar as alterações de laxidez e de força explosiva ao longo do ciclo menstrual e entre as atletas com e sem efeito de contraceptivos orais.

Importância do estudo: A importância deste estudo decorre de uma possível influência hormonal na superior e preocupante taxa de incidência de lesão deste ligamento nas atletas do sexo feminino comparativamente aos atletas do sexo masculino.

Tempo requerido e local das avaliações: em cada uma das sessões de avaliação são necessários cerca de 10 minutos para avaliar a laxidez dos joelhos, com um instrumento desenvolvido para o efeito (KT-1000), e para avaliar os saltos verticais, com o tapete “Ergo Jump” (plataforma de contactos). As respectivas medições serão executadas por técnicos especializados, no seu local de treino, numa frequência equivalente às sessões de treino e jogo e em horário/momento a aferir com a atleta e o(a) treinador(a).

Procedimentos: Antes de se iniciarem as avaliações propriamente ditas, as atletas preencherão um questionário acerca do seu perfil lesional e ginecológico e, quando justificado, será realizada uma avaliação clínica à articulação do joelho (pelo Dr. Leandro Massada), no sentido de se verificar e garantir a ausência de qualquer patologia associada a esta articulação. A ocorrência de uma qualquer lesão ao nível da articulação do joelho, consequência de actividade desportiva ou outra, implicará a cessação imediata das avaliações. O ciclo menstrual das atletas que não tomam contraceptivos orais será monitorizado com base nos seguintes procedimentos: (a) recolha diária de amostras de saliva, cuja cristalização se relaciona com a fase do ciclo menstrual; (b) observação da presença da LH na urina, com base no teste da ovulação (Clear Plan, facilmente adquirível em qualquer farmácia), a realizar de acordo com as instruções de utilização; (c) observação da temperatura basal corporal, com um termómetro fornecido para o efeito e instruções próprias e (c) colheita de sangue em três momentos, correspondentes às três fases do ciclo menstrual, a qual será realizada mediante deslocação da atleta às instalações do Laboratório Endoclab ou por um técnico do referido laboratório que se deslocará ao local das avaliações. Para monitorização do ciclo menstrual das atletas que tomam contraceptivos orais serão realizadas três colheitas de sangue, cujo procedimento será idêntico ao descrito para as atletas que não tomam contraceptivos orais.

Início e duração do estudo: As atletas serão avaliadas durante um ciclo menstrual. No primeiro dia do período menstrual a atleta avisa o investigador de campo (Luísa Estriga) e imediatamente se iniciam as avaliações até ao início do ciclo menstrual subsequente.

Riscos: A laxidez e a capacidade de salto serão avaliadas com instrumentos específicos desenvolvidos para o efeito, de natureza não invasiva, cujos riscos potenciais de lesão são praticamente inexistentes.

Confidencialidade: Será garantida a confidencialidade de toda a informação recolhida. A cada atleta será atribuído um código (número); quando o estudo terminar e os dados forem analisados, a lista de códigos com a identificação das atletas será destruída. Os dados recolhidos poderão vir a ser utilizados em estudos futuros, porém não contendo qualquer informação de identificação pessoal dos sujeitos. O nome da atleta não será usado em qualquer relatório relacionado com este estudo.

Participação Voluntária: A participação nesta investigação é absolutamente voluntária.

Desistência do Estudo: A atleta pode cessar a sua participação no estudo a qualquer momento, sem qualquer tipo de penalização. Se a atleta pretender abandonar o estudo deve informar o investigador e as avaliações terminam imediatamente.

Investigadores: Prof. Doutor Leandro Massada, médico especialista em Ortopedia e Medicina Desportiva e docente da FCDEF-UP; Prof. Doutor João Bernardes, médico especialista em Ginecologia e Obstetrícia; e Mestre Maria Luísa Estriga, docente da FCDEF-UP.

Contacto em caso de dúvidas acerca do estudo: Luísa Estriga (93******* ou 225074770)

Declaração

A atleta (nome) ____ declara que leu e entendeu esta **Declaração de Consentimento Informado** e que aceita participar no estudo acerca do impacto das variações hormonais do ciclo menstrual na estabilidade da articulação do joelho, de acordo com o descrito nesta declaração.

Opcional: permito que os dados recolhidos sejam facultados ao ________________ (treinador e/ou médico) mediante solicitação _______ (sim/não)

Atleta com idade inferior a 18 anos:

A validade deste consentimento expira quando a atleta atinge os 18 anos.

Assinatura da Atleta: ____ Data: __/__/______

*Assinatura do Enc. de Educação: ____ Data: __/__/______

* Confirmo que tenho autoridade legal para assinar esta declaração em nome da Atleta menor.

A Atleta e/ou Encarregado de Educação receberá uma cópia desta Declaração

U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

Gráfico de Registo da Temperatura Basal

Nome:______________________________ **Clube:**____________________

Ciclo mais curto conhecido	
Duração do ciclo (total de dias)	
Temperatura mais baixa	
Hora de observação da temperatura	

Dia 1: corresponde ao primeiro dia do período (sangramento)

Dia do ciclo	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40
Dia do mês																																								
Dia da semana																																								
Aparência do muco																																								
Sensação do muco																																								
Teste LH (+/-)																																								
99.00																																								
.9																																								
.8																																								
.7																																								
.6																																								
98.5																																								
.4																																								
.3																																								
.2																																								
.1																																								
98																																								
9																																								
8																																								
.7																																								
.6																																								
97.5																																								
.4																																								
.3																																								
.2																																								
1																																								
97.00																																								
9																																								
8																																								
Outros Sintomas (**notas**)																																								

X – sangue
P – pegajoso
G – grumoso
E - elástico

S - Seco
H – humido
M – molhado
L - lubrificante

(+) ou (-)

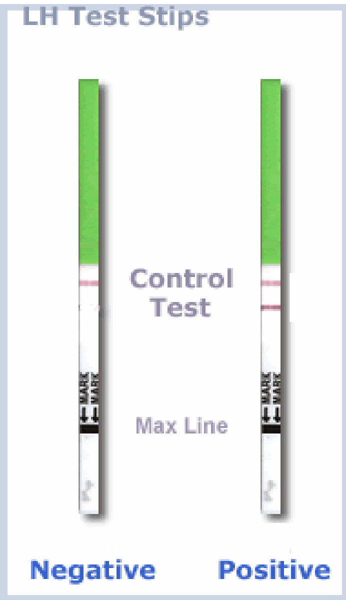


Gráfico de Registo da Temperatura Basal – <u>informações</u>

O registo da temperatura deve ser realizado de forma sistemática e ordenada:

1. A observação diária da temperatura corporal permite-nos verificar em que dia do ciclo menstrual ocorreu a ovulação; o registo da temperatura deve começar no primeiro dia do ciclo, isto é, no primeiro dia do período (menstruação/sangramento) até ao início do próximo ciclo menstrual (novo sangramento).

2. A Temperatura Corporal Basal é a temperatura em repouso, que deve ser medida à mesma hora, imediatamente ao acordar e antes de se levantar da cama; antes de qualquer tipo de actividade física (pois esta irá provocar um aumento da temperatura) ou comer/beber. No caso da observação/medição ser realizada numa hora diferente do habitual a mesma deve ser indicada no mapa de registos.

3. Para a maioria das mulheres, os valores de temperatura considerados normais, antes de ocorrer a ovulação, situam-se entre os 96.0 a 97.5 graus, após a qual se espera um aumento continuado da temperatura.

4. Deverá usar sempre o mesmo termómetro (fornecido para o efeito).

5. Qualquer alteração: hora de medição, termómetro, insónia, constipação ou dores físicas, transtornos emocionais, medicação, viagens, *stress/*ansiedade, ingestão significativa de álcool ou alimentos, jejuns, etc., deverá ser apontada no gráfico para, posteriormente, facilitar a sua interpretação (notas 1, 2, 3...).

<u>Instruções para uso do termómetro – *Digital Basal Thermometer*</u>

Apenas para uso oral

1. Pressione o botão **on/off**, o **termómetro** registará **88.88ºF**, seguido de **97.70ºF** e em seguida “**Lo**”, com um **ºF** intermitente (indicando que o termómetro está pronto para ser usado).

2. Para observar a última temperatura registada pelo termómetro: ao ligar o termómetro pressione continuamente o botão **on/off** e a última temperatura registada aparecerá no mostrador enquanto pressionar o botão; assim que deixar de pressionar o botão o mostrador indicará **97.70ºF,** seguido de **“Lo”** com um **ºF** intermitente (indicando que o termómetro está pronto para ser usado).

3. Coloque a ponta do termómetro debaixo da língua, centrado na boca. Mantenha a boca fechada durante o intervalo de tempo necessário para a medição da temperatura (aproximadamente 2 minutos). Não permita que o termómetro se mova dentro da boca.

4. A medição da temperatura requer cerca de 2 minutos. No máximo da temperatura o ºF deixará de aparecer de forma intermitente e o *beeper* irá apitar cerca de 4 segundos (4 bips), indicando que o termómetro está pronto para ser retirado da boca e a temperatura observada. **Leia e registe a temperatura no Gráfico.**

5. Quando terminado o processo, desligue o termómetro no botão **On-Off**, caso não o tenha feito o termómetro desligar-se-á em 10 minutos.

6. Caso verifique que o termómetro não tem bateria ou qualquer outra anomalia contacte a pessoa responsável pelo projecto.

<u>CUIDADOS A TER:</u>

1. Tenha o máximo cuidado com o termómetro, não deixe cair, não dobre ou desmonte.

2. Guarde o termómetro na caixa de plástico fornecida para o efeito. Não o exponha directamente à luz solar, temperaturas extremas ou humidade.

3. Não deixe que água (ou outro produto) contacte com o mostrador digital da temperatura ou entre no termómetro.

4. Instruções de limpeza: limpe a ponta do termómetro com álcool etílico (diluído com água); limpe sempre entre cada utilização; limpe a área do termómetro (ponta) que toca a boca e evite que o liquido toque no mostrador digital da temperatura; não use água quente.

Observação do muco cervical

<u>Informações:</u>

Cada mulher pode aprender a identificar as características do seu muco cervical para saber ler a sua fisiologia e assim identificar em que momento/fase do ciclo se encontra. As características a identificar são duas: **sensação e aparência**.

SENSAÇÃO: refere-se àquela que o moço produz em contacto com a pele e as mucosas dos genitais externos da mulher;

<u>Existem três possibilidades</u>:

(a) sensação de secura (o papel não desliza e inclusivamente rasga);

(b) sensação de lubrificação (o papel desliza com facilidade);

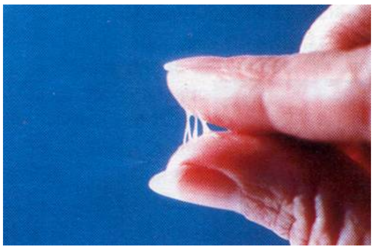
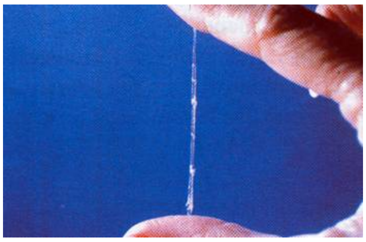
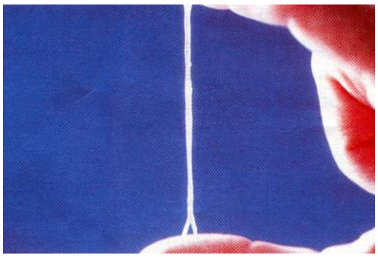
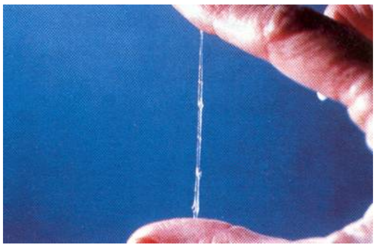
(c) e sensação de estar molhada (sente-se humidade).

<u>Nomenclatura</u>: **S** – seco; **H** – húmido; **M** – molhado; e **L** – lubrificado

APARÊNCIA: refere-se ao aspecto do muco que se pode observar; diferentes características identificáveis pela mulher: (a) transparência; (b) elasticidade; (c) tingido de sangue; (d) quantidade e (e) alteração de cor.

<u>Nomenclatura</u>: **S** – seco; **X** – com sangue; **P** – pegajoso; **G** – grumoso; **E** – elástico.

Tipos de Muco, a registar no **Mapa:**

		
(G) Grumoso, denso, fica num dos lados do papel	**(P) Pegajoso**, ao tentar despegá-lo pode esticar 1 ou 2 cm, mas parte-se logo, ficando na ponta dos dedos	**(E)Elástico**, faz fios, normalmente transparente, tem a aparência da clara de ovo crua.
		
(M) Misto, possui uma parte de aspecto elástico e outra pegajoso ou grumoso		

Anotações no Gráfico:

As anotações acerca do muco devem realizar-se à noite, ao deitar; depois de se ter observado o muco ao longo de todo o dia.

Se ao longo do dia tiver sintomas diferentes, deve anotar-se no gráfico aquele que for indicador de maior fertilidade. As anotações far-se-ão dia após dia.

U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

Teste de Ovulação

Com este teste é possível detectar a subida brusca da **Hormona Luteinizante (LH)** na urina. A LH está sempre presente na urina, porém a rápida e elevada subida do seu nível, a meio do ciclo menstrual, provoca a libertação de um óvulo proveniente dos ovários (24 a 48 horas depois). Este processo é designado de **Ovulação**. Por conseguinte, após ao resultado positivo do teste, os dias seguintes são considerados os dias de máxima fertilidade feminina (isto é, elevada probabilidade de engravidar).

Instruções de utilização:

1. Ser-lhe-ão fornecidos os necessários dispositivos (tiras) para detectar a subida da LH e indicados os dias em que deve fazer o teste. Deve fazer o teste todos os dias até o resultado ser positivo.

2. Deve fazer o teste sempre à mesma hora, de preferência entre as 10:00 e as 20:00 (a altura ideal é durante a tarde), nunca com a primeira urina da manhã, ou em alternativa após no mínimo 2 a 3 horas sem urinar (evite beber muitos líquido antes de fazer o teste para não diluir a hormona).

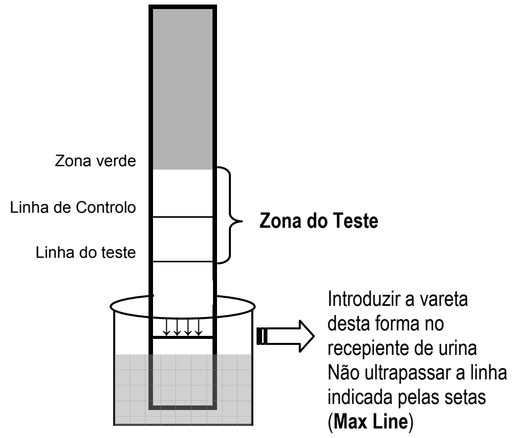
3. Recolha a amostra de urina num recipiente seco e limpo.

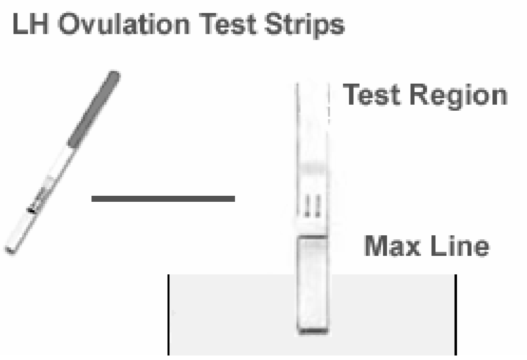
4. Quando estiver pronta para fazer o teste (certifique-se que tem um relógio perto) abra o invólucro da vareta e introduza a parte absorvente do dispositivo (ponta da vareta) na urina durante aproximadamente 4 a 5 segundos; sem ultrapassar a linha das setas. Segure na vareta pela zona verde, mantendo-a direita dentro da urina, com a ponta virada para baixo.

5. Quando a amostra (urina) entra em contacto coma tira, percorre toda a tira absorvente e, assim que chega à Zona do Teste, faz reacção produzindo ou não uma linha vermelha.

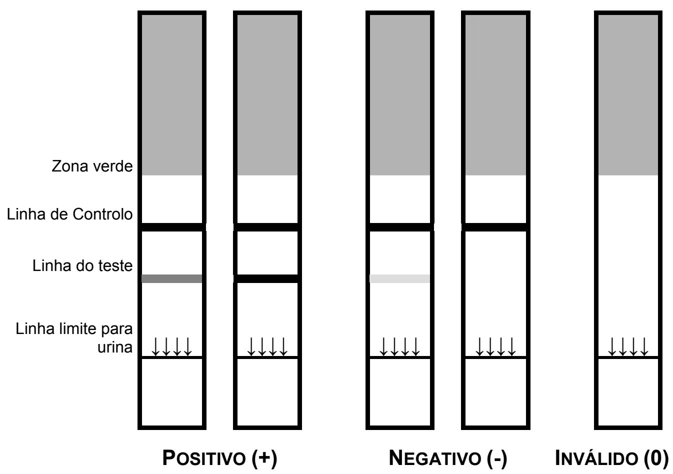
6. Após retirar a tira da urina coloque-a em cima de uma superfície plana e observe o resultado.

7. O resultado deve ser lido conforme abaixo indicado e o resultado registado no gráfico para o efeito. O resultado está pronto para ser lido após 3 minutos, contudo para confirmar um resultado negativo deve esperar até 5 minutos. Rejeite o teste após 10 minutos.





Ler o resultado do teste da seguinte forma:

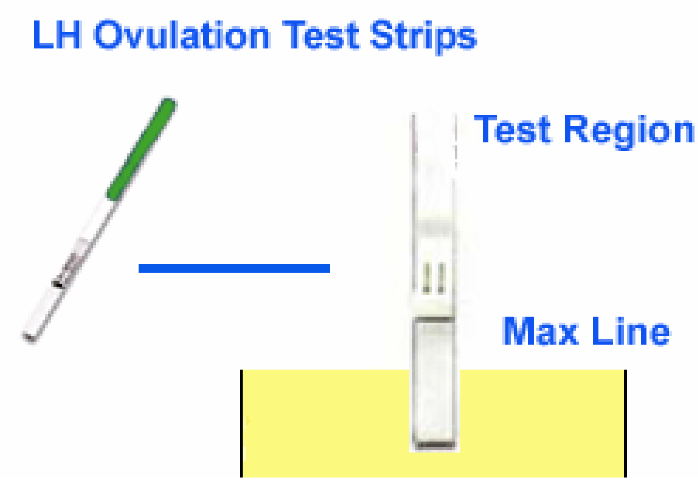


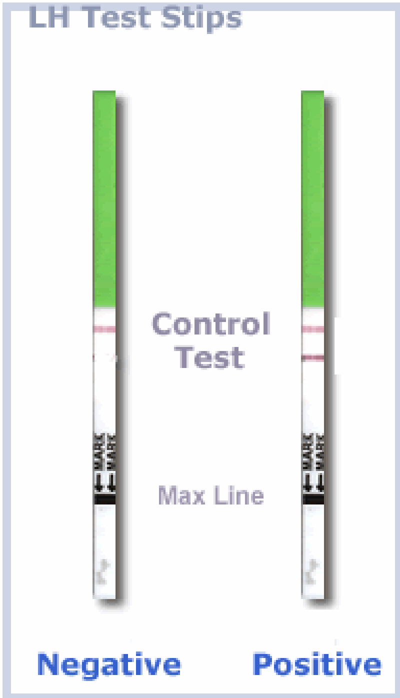
Para observar o resultado do teste deve comparar a intensidade das linhas, a de controlo e a do teste. A linha de controlo é usada para comparação com a linha do teste, ao mesmo tempo que serve para confirmar que o teste foi bem feito.

POSITIVO (Pico LH; +): Se as duas linhas forem visíveis, estando a linha do teste igual ou mais escura que a linha de controlo. Neste caso, a ovulação deverá ocorrer nas próximas 24 a 48 horas.

NEGATIVO (-): Se apenas se observa a linha de controlo ou se observam as duas linhas, mas a linha do teste é de fraca intensidade (ténue). Neste caso, deve continuar a realizar o teste até o resultado ser positivo.

TESTE INVÁLIDO (0): Se a linha de controlo não é visível até 5 minutos após a realização do teste; o resultado considera-se inválido e o teste é rejeitado; neste caso, deve voltar a fazer o teste com uma nova vareta.


LH Ovulation Test Strips
Test Region
Max Line


LH Test Stips
Control
Test
Max Line
Negative
Positive