Determination of risk factors causing overuse injuries in runners

New aspects and critical considerations

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Bestimmung von Risikofaktoren, die zur Entstehung von Überlastungsbeschwerden bei Läuferinnen und Läufern führen

Neue Aspekte und kritische Betrachtungen

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List of abbreviations

AF	Abdominal flexion
AT	Achilles tendon pain or Achilles tendinopathy
BE	Back extension
bHAB	Bilateral hip abduction
bHAD	Bilateral hip adduction
CRF	Case Report Form
CRP	Continuous Relative Phase
EVA	Ethylene-vinyl acetate
GPS	Global Positioning System
HAB	Hip abduction
HAD	Hip adduction
HEX	Hip extension
HFL	Hip flexion
IE	Initial examination
ISB	International Society of Biomechanics
ITBS	Iliotibial band syndrome
KEX	Knee extension
KFL	Knee flexion
OI	Overuse injury
PF	Plantar fasciitis
PFPS	Patellofemoral pain syndrome
PT	Patellar tendinopathy
ROP	Roll-over process, stance phase
SD	Standard deviation
SE	Second examination
SP	Scientific paper
SS	Shin splints or medial tibial stress fracture
ÜB	Überlastungsbeschwerde
uHAB	Unilateral hip abduction
uHAD	Unilateral hip adduction
uHEX	Unilateral hip extension
VCRP	Variability of Continuous Relative Phase

Abstract

Introduction

The aetiology of overuse injuries (OI) in runners appears to be injury-specific with influences from clinical, biomechanical and training-related risk factors. However, there is currently no consensus regarding evidence-based multifactorial mechanisms of risk factors leading to injury. Thus, the focus of this doctoral thesis is the prospective determination of extrinsic and intrinsic risk factors and injury-specific mechanisms for the development of OI in recreational runners.

Current state of scientific research

Clinical risk factors. A wide variety of clinical variables has been measured and analyzed with regard to injury, such as leg length discrepancies, high foot arches, restricted or excessive ranges of joint motion, abnormal alignments of the lower extremity, and so forth.

Biomechanical risk factors. The analysis of kinematic variables, in particular frontal rearfoot motion, has been the most commonly-performed biomechanical OI research approach over the past several decades. Recently, new methods such as Vector Coding, Continuous Relative Phase (CRP) and CRP variability (VCRP) have been presented to evaluate joint coordination patterns. An association between abnormal joint loadings resulting from altered kinematics and subsequent contribution to the development of injury appears reasonable. Additional factors such as muscular imbalances or deficits in muscular strength are also presumed to foster the development of OI.

Training-related risk factors. OI are considered to be caused by training errors such as excessive mileage or running volume, duration, frequency or intensity, and therefore could be avoided with different training programs.

Summary. Numerous extrinsic and intrinsic variables have been considered as potential risk factors leading to OI in runners. However, agreement regarding the interrelationships between variables which lead to injury has not been reached. The main reasons for the lack of experimental support are the retrospective designs and inappropriate measurement technologies of previous studies, as well as the constituencies and sizes of previous study populations.

Research questions

The first research question is whether VCRP can provide new insights into the coupling mechanisms and coordination patterns of the lower extremity with regard to the development of injury. The second research question focuses on the prospective determination of injury-specific mechanisms of clinical, biomechanical and training-related risk factors for the development of OI in recreational runners. The third research question concerns whether prospective study approaches are required in order to determine biomechanical risk factors causing OI in runners. Each research question is addressed and answered within a dedicated scientific paper (SP).

Methodology

A prospective study was performed to determine multifactorial risk factors in the development of OI in recreational runners. Well-established, reliable measurement techniques and transparent calculation methods were implemented to acquire relevant clinical and biomechanical data. Training was continuously monitored, which enabled the prospective evaluation of individual training programs, and medical examinations were performed to assure absence of injury at intake and to consistently diagnose presenting complaints in the case of injury.

Results

In SP1, female runners suffering from iliotibial band syndrome do not demonstrate any significant differences in VCRP compared to healthy controls.

In the current prospective study, runners generating Achilles tendon pain already demonstrate decreased knee flexor strength and abnormal lower leg kinematics compared with a matched control group in an injury-free state. Additional changes in individual training programs are also found for injured runners prior to the onset of injury.

Regarding the necessity of prospective study design and research question 3, differences between the healthy and injured state cannot be detected for hip, knee and ankle/rearfoot kinematics for the same group of runners.

SP3 notes that differences between the healthy and injured state have not been identified for hip, knee and ankle/rearfoot kinematics for the same group of runners,

Discussion and Conclusion

Based on the findings of SP1, VCRP does not appear to be an appropriate method to differentiate between lower leg kinematics of healthy runners and injured runners suffering from iliotibial band syndrome who reveal differences in commonly-used kinematic variables,

and consequently is inappropriate to use to determine kinematic risk factors leading to OI in runners.

With regard to the second research question, prospective studies can reveal potential injury-specific mechanisms causing OI in runners as outlined for Achilles tendon pain in SP2. However, if the number of participants can be increased in the future, more detailed and specific information about the interacting mechanisms leading to injury can be determined.

Prospective studies do not appear necessary to determine kinematic risk factors causing OI in runners as postulated in SP3. However, injury-specific analyses of clinical variables and isometric strength measurements need to be performed in order to ultimately determine whether retrospective study designs are sufficient for all future investigations.

Perspective

In order to determine injury-specific mechanisms, the focus on future studies must be on multi-dimensional approaches analyzing extrinsic and intrinsic risk factors, independent of whether retrospective or prospective study designs are implemented. Consequently, the number of subjects needs to be expanded. This will permit cluster, regression or principal component analyses to be performed, which will allow the determination of evidence-based mechanisms between clinical, biomechanical and training-related risk factors causing OI in runners. Additional factors such as physiological, social and neuromuscular factors might also be taken into consideration for future investigations into the development of OI in runners.

Zusammenfassung

Einleitung

Die Ätiologie von Überlastungsbeschwerden (ÜB) bei Läuferinnen und Läufern scheint überlastungsspezifisch von diversen klinischen, biomechanischen und trainingsrelevanten Parametern beeinflusst zu sein. Evidenzbasierte multifaktorielle Mechanismen von Risikofaktoren, die zu ÜB führen, konnten allerdings noch nicht aufgezeigt werden. Daher liegt der Fokus dieser Dissertation auf der prospektiven Bestimmung extrinsischer und intrinsischer Risikofaktoren, sowie überlastungsspezifischer Entstehungsmechanismen von ÜB bei Läuferinnen und Läufern.

Aktueller wissenschaftlicher Stand

Klinische Risikofaktoren. Eine hohe Anzahl unterschiedlicher klinischer Parameter wurde untersucht und mit der Entstehung von ÜB in Verbindung gebracht. Dazu gehören Unterschiede in Beinlänge und Fußgewölbehöhe, eingeschränkte sowie erhöhte Gelenkbeweglichkeit, veränderte statische Ausrichtung der unteren Extremität und viele mehr.

Biomechanische Risikofaktoren. Die Analyse kinematischer Parameter, insbesondere der frontalen Rückfußbewegung, zeigte sich als meist verbreitetster biomechanischer Untersuchungsansatz im Verlauf der letzen Jahrzehnte. Aktuell wurden neue Ansätze wie „Vector Coding“, „Continuous Relative Phase“ (CRP) und „Continuous Relative Phase Variability“ (VCRP) präsentiert, um Koordinationsmuster der unteren Extremität zu untersuchen. Es scheint zudem eine Verbindung zwischen erhöhter Gelenkbelastung durch veränderte Bewegungsabläufe und der Entstehung von ÜB zu geben. Zudem gelten Defizite in der muskulären Kraftfähigkeit sowie muskuläre Dysbalancen als weitere Einflussfaktoren auf die Entstehung von ÜB bei Läuferinnen und Läufern.

Trainingsspezifische Risikofaktoren. ÜB scheinen außerdem durch Fehler in der Trainingsgestaltung wie beispielsweise erhöhte Kilometerleistung oder Laufumfänge, Laufdauer, Trainingsfrequenz und Intensität ausgelöst zu werden und können somit durch verändertes Trainingsverhalten vermieden werden.

Zusammenfassung. Zahlreiche extrinsische und intrinsische Parameter wurden mit ÜB in Verbindung gebracht. Einigkeit über deren Einfluss auf die Entstehung von ÜB besteht allerdings nicht. Das zumeist retrospektive Studiendesign, inadäquaten Messverfahren vergangener Studien sowie deren untersuchte Studienpopulation sind als Hauptgründe für das Fehlen evidenzbasierter Studienergebnisse zu nennen.

Fragestellungen

Die erste Fragestellung untersucht, ob VCRP neue Einblicke in die Kopplungsmechanismen und Koordinationsmuster der unteren Extremität hinsichtlich der Entstehung von ÜB bei Läuferinnen und Läufern liefern kann. Die zweite Fragestellung bezieht sich auf die prospektive Bestimmung überlastungsspezifischer Mechanismen aus klinischen, biomechanischen und trainingsrelevanten Risikofaktoren, die zu ÜB bei Läufern führen. Die dritte Fragestellung hinterfragt die Notwendigkeit prospektiver Studienansätze zur Definition von biomechanischen Risikofaktoren für ÜB bei Läuferinnen und Läufern. Jede der drei Fragestellungen wird anschließend durch jeweils einen Artikel beantwortet.

Methode

Eine prospektive Studie wurde unter Verwendung von etablierten und reliablen Messverfahren sowie transparenten Berechnungsmethoden durchgeführt, um relevante klinische sowie biomechanische Daten zu erheben. Medizinisch orthopädische Untersuchungen dienen der Sicherung der Beschwerdefreiheit zu Beginn einer Teilnahme sowie der Diagnose im Falle einer auftretenden akuten ÜB. Kontinuierliche Trainingsüberwachung ermöglicht zudem die Auswertung individueller Trainingsprogramme.

Ergebnisse

Es zeigen sich keine signifikanten Unterschiede in VCRP zwischen Läuferinnen, die an iliotibialem Bandsyndrom leiden und beschwerdefreien Läuferinnen.

Die aktuelle Längsschnittstudie zeigt hingegen, dass Läuferinnen und Läufer, die Achillessehnenbeschwerden generieren, verglichen mit einer gesunden Kontrollgruppe, bereits in einem beschwerdefreien Zustand schwächere Kniebeugemuskulatur und veränderte Kinematik der unteren Extremität aufweisen. Zudem können Modifikationen in der individuellen Trainingsgestaltung kurz vor der Entstehung einer UB nachgewiesen werden.

In Bezug auf die Notwendigkeit prospektiver Studienansätze und Fragestellung 3 können keine Unterschiede zwischen beschwerdefreiem und verletztem Zustand in der Hüft-, Knie- und Sprunggelenk-/Rückfußkinematik für die gleiche Gruppe von Läuferinnen und Läufern festgestellt werden.

Diskussion und Schlussfolgerung

Basierend auf den Ergebnissen von Artikel 1 erscheint VCRP zur Differenzierung von Koordinationsmustern der unteren Extremität zwischen beschwerdefreien Läufern und Läufern mit iliotibialem Bandsyndrom, die bereits Unterschiede in herkömmlichen kinematischen Parametern aufweisen, und somit zur Bestimmung kinematischer Risikofaktoren nicht geeignet zu sein.

Wie am Beispiel der Entstehung von Achillessehnenbeschwerden in Artikel 2 gezeigt wird, können prospektive Studien potentielle überlastungsspezifische Mechanismen, die zur Entstehung von ÜB führen, aufzeigen. Sollte die Anzahl der Teilnehmer im Laufe der kommenden Jahre erhöht werden, können detaillierte überlastungsspezifische Entstehungsmechanismen definiert werden.

Allerdings scheint die Durchführung prospektiver Studien zur Bestimmung kinematischer Risikofaktoren von ÜB bei Läuferinnen und Läufern auf Grund der Ergebnisse von Artikel 3 nicht erforderlich zu sein. Überlastungsspezifische Analysen klinischer Parameter sowie isometrischer Maximalkraftmessungen müssen in Bezug auf die Notwendigkeit von prospektiven Studien noch durchgeführt werden, um final entscheiden zu können, ob retrospektive Studienansätze für zukünftige Anwendungen ausreichen.

Ausblick

Um überlastungsspezifische Entstehungsmechanismen von ÜB erkennen zu können, muss der Fokus zukünftiger Studien, unabhängig des retrospektiven oder prospektiven Studienansatzes, auf der multifaktoriellen Analyse von extrinsischen und intrinsischen Risikofaktoren liegen. Zudem muss die Anzahl der Studienteilnehmer erhöht werden, um Cluster-, Regressions- oder „Principal Component“-Analysen durchführen zu können, die eine Bestimmung evidenzbasierter Mechanismen aus klinischen, biomechanischen und trainingsspezifischen Risikofaktoren für ÜB bei Läuferinnen und Läufern ermöglicht. Weitere Einflüsse wie zum Beispiel physiologische, soziale oder neuromuskuläre Faktoren sollten in zukünftige Untersuchungen zur Entstehung von ÜB bei Läuferinnen und Läufern mit einfließen.

1. Introduction

1.1. Theoretical background

Running is probably the most popular sport in the world and the number of runners has steadily increased since the 1980s (Buist et al., 2010; Lopes et al., 2012; Verhagen, 2012). Besides positive health effects on the metabolism of the human organism, cardiovascular system, immune system and the musculoskeletal system (Predel and Tokarski, 2005; Williams, 1997), running is often accompanied by the occurrence of some acute but most commonly overuse injuries (OI) to the lower extremities (Buist et al., 2008; Hreljac, 2005; Walther et al., 2005). Despite numerous studies over the last decades which deal with the determination of risk factors, there has been no decline in injury rates (Daoud et al., 2012) and between roughly 20% and 90% of all runners still develop an OI every year (Satterthwaite et al., 1996; van Gent et al., 2007). A recent review by van Gent et al. (2007) shows that OI are mostly located at the knee joint (patella tendinopathy, iliotibial band syndrome), however the Achilles tendon (tendinopathy or peritendinopathy), the tibial crest (medial tibial stress syndrome or shin splints) and the plantar fascia (plantar fasciitis) are also considered dominant sites of running injuries (Lopes et al., 2012; Walther et al., 2005) all of which usually lead to training reductions or rest.

Over the past few years in particular, numerous studies have presented a wide variety of potential risk factors for OI which can be categorized as clinical or anthropometrical, biomechanical and training-related factors (Hreljac, 2005). Among clinical factors are leg length discrepancies, high foot arches, restricted or excessive ranges of joint motion and abnormal alignments of the lower extremity, and many more (Murphy et al., 2003; Neely, 1998a). Excessive pronation, poor muscular strength or muscular imbalance as well as high impact forces or loading rates acting on the human system are proposed as additional risk factors and can be summarized as biomechanical risk factors (Hreljac, 2005). With regard to training-related risk factors or training errors, a high diversity of training parameters have been associated with the occurrence of injury including weekly running mileage, frequency, duration and speed (Nielsen et al., 2012). In particular, running mileage per week and a history of previous OI is acknowledged to increase the risk of generating a new OI in runners (Bovens et al., 1989; Fields et al., 2010; Neely, 1998b; van Gent et al., 2007). Individual running experience also appears to be related to the incidence of injury (Buist et al., 2008;

Taunton et al., 2003). Hreljac (2004) even states that OI, in general, are a consequence of training errors and, therefore, can be assumed to be a final trigger for the generation of OI. However, since equal amounts of running mileage do not necessarily lead to OI in each runner, individual anatomic and biomechanical characteristics need to be identified which either cause or prevent the development of OI (Hreljac, 2004).

In the context of past research, the main deficits lie in the use of one-dimensional biomechanical or clinical approaches to identify factors causing OI in runners. This means, that most studies have focused only on one potential risk factor as e.g. hip abductor strength (Niemuth et al., 2005), ranges of ankle and knee joint motion (van Mechelen et al., 1992) or rearfoot pronation (Ryan et al., 2009) and their potential influence on OI in general while the studies neglected other factors (Wen, 2007). Thus, contradicting results have been found whereby some studies associated abnormal movement patterns with OI while other studies could not find any relationship or demonstrated opposing results (Wen, 2007). There are many more deficits that can be named as well which lead to confounding results such as poor measurement techniques as two-dimensional motion analysis systems (McCrory et al., 1999; Messier and Pittala, 1988), differing approaches to document training information (Nielsen et al., 2012), over-interpretation of results due to missing clinical relevance (Mahieu et al., 2006) or small and inhomogeneous study samples (Hamill et al., 1999; Miller et al., 2007).

To date, the aetiology of OI in runners appears to be injury-specific and multifactorial with influences from clinical, biomechanical and training-related risk factors (Barton et al., 2009; Cheung et al., 2006; Hreljac, 2005; Marti et al., 1988; Wen, 2007). However, interactions between evidence-based risk factors have not been identified. Reasons for the missing identifiers are diverse. The fact that most studies are based on a retrospective study design appears to be the major problem since differences between healthy and injured runners can neither be specified as causes of, nor as a compensatory effect of an injury (Almekinders and Temple, 1998). Prospective study designs are considered essential for future studies in order to clarify cause-effect relationships and to determine interrelationships between different risk factors leading to injury (Almekinders and Temple, 1998; Barton et al., 2009; Dierks et al., 2011; Fredericson et al., 2000; Grau et al., 2011; Ireland et al., 2003; Montgomery et al., 1989; Pohl et al., 2008; Pohl et al., 2009; Thijs et al., 2008; van der Worp et al., 2011; van Gent et al., 2007; van Mechelen, 1992; Wen et al., 1998; Wen, 2007).

Further, since variations in the definition of injuries, the periods of observation, the included populations and their levels of experience affect incidence rates, sample characteristics must be well-defined in order to effectively determine risk factors (Hoeberigs, 1992; Ryan et al., 2006; Wen, 2007).

Thus, the focus of this doctoral thesis is the determination of evidence-based and injury-specific mechanisms of clinical, biomechanical and training-related risk factors for the development of OI in recreational runners.

1.2. Structure of doctoral thesis

Following a discussion of the theoretical background, the thesis provides a more detailed overview of clinical, biomechanical and training-related risk factors, outlines deficits and problems in the implementation of recent studies and illustrates why the prevalence of OI in runners has not yet been reduced.

Based on the presented deficits and aspects of the current state of literature, three research questions are proposed. The first research question deals with the applicability of a recently published mathematical algorithm, called Continuous Relative Phase, which is based on spatial-temporal kinematic information. In contrast to typical discrete variables used to analyse kinematic data, Continuous Relative Phase is applied to quantify joint coordination patterns of the lower extremity which might be associated with OI. The second research question deals with the implementation of prospective studies to determine clinical, biomechanical (three-dimensional kinematics and muscular strength) and training-related risk factors and their potential interrelationships leading to the development of OI in recreational runners. The third research question investigates whether prospective studies are necessary to achieve this goal.

In chapter 4, study design, population and methodology of the current prospective study are detailed from which two manuscripts of the scientific program are derived from. In chapters 5 and 6, the results of the scientific papers are displayed, discussed and placed into context with respect to the current scientific state of research. Finally, based on the conclusions of the current thesis, recommendations are made for future studies with regard to design and focus. The structure of this doctoral thesis is displayed in Figure 1.

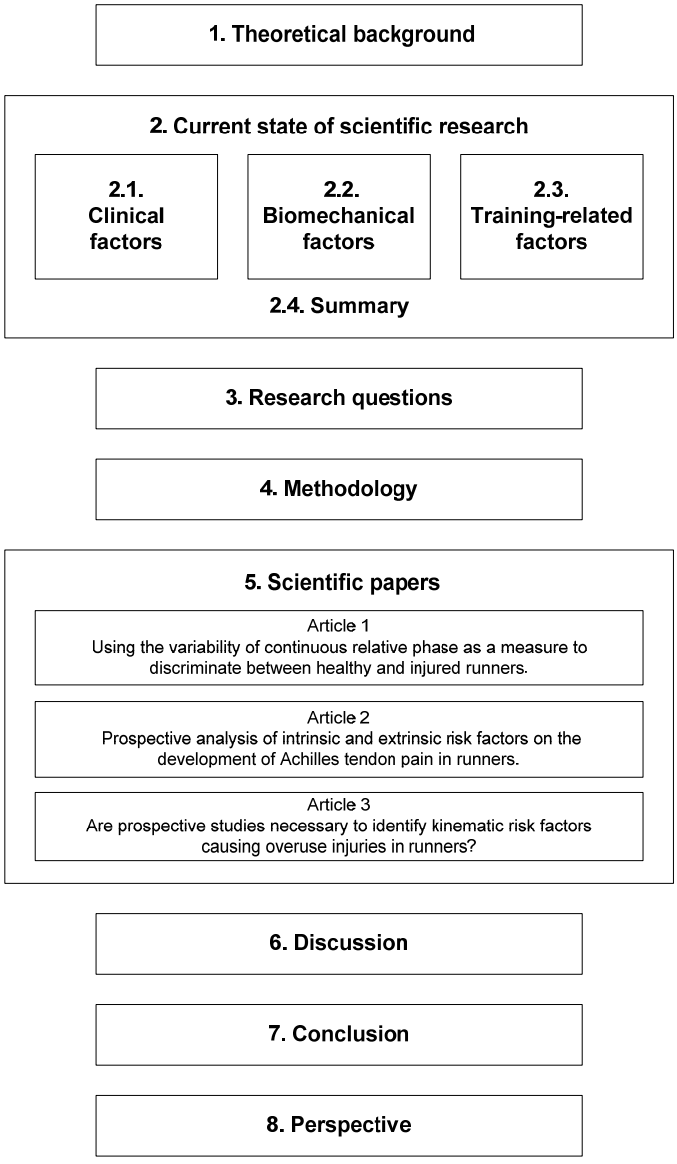


Figure 1: Structure of this doctoral thesis

2. Current state of scientific research

This chapter provides an overview of the current state of research considering the influence of clinical, biomechanical including kinematic, kinetic and muscular strength variables and training-related risk factors on the development of OI in runners.

2.1. Clinical factors

The influence of clinical (or anthropometrical) factors on the development of OI has been a topic of numerous studies and reviews. Consequently, a wide variety of variables has been measured and analyzed with regard to injury such as ranges of hip, knee and ankle joint motion, muscle tightness, leg-length discrepancies, foot arch heights and others (Clement et al., 1984; Fields et al., 2010; Haglund-Akerlind and Eriksson, 1993; Hreljac, 2005; Kannus, 1997; Kaufman et al., 1999; Lankhorst et al., 2012; Lun et al., 2004; Mahieu et al., 2006; McCrory et al., 1999; Messier and Pittala, 1988; Montgomery et al., 1989; Murphy et al., 2003; Neely, 1998a; Rolf, 1995; van der Worp et al., 2011; van Gent et al., 2007; van Mechelen, 1992; Wang et al., 1993; Wen et al., 1998; Wen, 2007; Witvrouw et al., 2000; Witvrouw et al., 2001).

Recent reviews by Hreljac (2005), Murphy et al. (2003), Neely (1998a) and Wen (2007) indicate that consistent and evidence-based clinical risk factors for OI in runners have not yet been determined. Murphy et al. (2003) conclude that a comparison of results between different studies is not possible since diverse measurement techniques between the studies are used to assess variables such as ranges of motions (ROM) or muscle tightness of various joints among athletes of different sports suffering from diverse symptoms. For example, Kaufman et al. (1999) show increased gastrocnemius muscle tightness in combination with Achilles tendon pain (AT) in runners whereas Witvrouw et al. demonstrate in 2000 and 2001 relationships between lower quadriceps and gastrocnemius muscle tightness and anterior knee pain (Witvrouw et al., 2000) or between lower gastrocnemius and hamstring muscle tightness and the development patellar tendinopathy (PT) in students (Witvrouw et al., 2001). There is a disagreement about the relationship between sagittal ankle ROM and OI: while Messier and Pittala (1988) did not find any differences in ankle flexibly between healthy und injured runners, Kibler (1991) and Montgomery et al. (1989) describe lower ankle ROM as potential risk factor for injury.

Even studies which investigate clinical variables of the same symptom in a comparable population of runners, demonstrate contradicting results. Haglund-Akerlind and Eriksson (1993) describe a lower ROM of the ankle joint in runners suffering from AT whereas Mahieu et al. (2006) find that runners with AT have a higher ankle ROM compared to healthy runners. These contradictory results appear to be a consequence of overestimating the obtained results. A difference of 2° in passive ankle joint ROM between the left and the right side as shown by Mahieu et al. (2006) cannot be regarded as a clinically relevant factor for the development of AT in runners. Regarding the development of PT, van der Worp et al. (2011) present in a systematic review several potential variables leading to PT; however evidence-based risk factors have not been identified. Finally, van der Worp et al. (2011) recommend the implementation of prospective studies using multifactorial approaches to determine evidence-based risk factors and to clarify the cause-effect relationship.

A prospective study design thus appears essential in order to define abnormalities causing injury and not to characterize differences resulting from an injury as risk factors (Murphy et al., 2003; Almekinders and Temple, 1998). However the implementation of a prospective study is not the entire solution. Lun et al. (2004) carried out a prospective study to investigate a relationship between lower limb alignment and injury. They were not able to determine anthropometric risk factors for OI due to a high drop-out rate of nearly 50% and a large diversity of overuse symptoms. Even prospective studies including a large number of subjects present contradicting results and thus, have not established a link between clinical risk factors and injury. Walter et al. (1989) measured several clinical variables in about 1000 runners and Montgomery et al. (1989) included more than 500 subjects; neither found any relationships between anthropometric data and injury. Associations between higher longitudinal foot arches and the development of OI and stress fractures have been reported by Cowan et al. (1996) and Giladi et al. (1987). In contrast, Wen et al. (1998) identify higher foot arches as a protective factor against OI. In a recent review by Lankhorst et al. (2012) concerning the development of patellofemoral pain syndrome (PFPS), no clinical risk factors were clearly identified.

In summary, differences in methodology, heavy dependence on retrospective study designs, challenges with included populations and sample sizes or the overestimation of results with regard to their clinical relevance have inhibited the definition of clinical risk factors for OI in runners. Further, the author agrees with Hreljac (2005) who states that anthropometric variables can only predict the development of OI in combination with other biomechanical or training-related risk factors and therefore, multifactorial analyses of risk factors are required.

2.2. Biomechanical factors

Under the term “biomechanical factors”, kinematic and kinetic variables as well as muscular strength can be considered. Thus, this section is divided into three subsections to discuss these factors and to outline the current state of research.

2.2.1. Three-dimensional kinematics

The analysis of kinematic variables, in particular frontal rearfoot motion, has been the most commonly-performed biomechanical OI research approach during the past several decades (Ryan et al., 2006). Pronation is considered an important and necessary part of lower extremity movement patterns to attenuate impacts to the musculoskeletal system between heel strike through mid-stance during walking or running. Since pronation induces internal tibia rotation which is coupled with knee flexion (Tiberio, 1987), alterations in pronation affect the kinematic chain of the entire lower extremity and lead to higher stresses on these structures. Usually, continuous joint excursion angles and discrete variables such as initial, maximal and terminal joint excursions as well as ROMs or motion velocities serve as a basis for further kinematic analyses (Azevedo et al., 2009; Barton et al., 2009; Clement et al., 1984; Dierks et al., 2010; Dierks et al., 2011; Donoghue et al., 2008; Ferber et al., 2010; Grau et al., 2008a; Grau et al., 2008b; Grau et al., 2011; Hreljac et al., 2000; McCrory et al., 1999; Messier and Pittala, 1988; Milner et al., 2007; Munteanu and Barton, 2011; Pohl et al., 2008; Pohl et al., 2009; Ryan et al., 2009; Souza and Powers, 2009; Stergiou et al., 1999; Tiberio, 1987; Willems et al., 2006; Willems et al., 2007).

Subtalar pronation is a combined movement of upper ankle joint dorsiflexion, rearfoot eversion and forefoot abduction, wherein frontal rearfoot motion can be considered to be an accurate indicator of overall subtalar pronation (Ferber et al., 2009). Thus, increased rearfoot eversion in particular, as well as eversion velocity and eversion ROM have been presented as potential risk factors for OI in runners. Studies associate greater rearfoot eversion with OI in general (Messier and Pittala, 1988; Willems et al., 2006) but also with AT (Clement et al., 1984; Donoghue et al., 2008; Ryan et al., 2009), shin splints (SS) or medial stress fractures (Messier and Pittala, 1988; Pohl et al., 2008) and PFPS (Messier et al., 1991). Hreljac et al. (2000), in contrast, did not find any relationship between excessive eversion and OI and in a study by Dierks et al. (2011) runners with PFPS showed reduced frontal rearfoot excursion compared to healthy runners. Ferber et al. (2010) could not find any differences in rearfoot eversion between female runners who suffered from iliotibial band syndrome (ITBS) and

healthy female runners. Instead, the ITBS group exhibited greater knee internal rotation and hip adduction. Pohl et al. (2009) also could not demonstrate differences in frontal rearfoot kinematics for runners who sustained plantar fasciitis (PF) compared to healthy controls. In contrast, in a study by Grau et al.(2008a) runners with ITBS exhibited decreased rearfoot eversion compared to healthy controls. Thus, in 2013, Ferber et al. emphasize that currently, no definitive connection between OI and excessive pronation has been established since contradicting results associating pronation with the development of injury have been found.

It is also evident that in addition to rearfoot kinematics, the evaluation of knee and hip joint kinematics is also essential in determining risk factors for OI in runners. In 2011, Grau et al. identified decreased hip adduction and tibial internal rotation until mid-stance and greater external tibial rotation at push-off between ITBS and healthy runners. Azevedo et al. (2009) found lower sagittal knee joint range of motion for runners with AT, and Souza and Powers (2009) observed greater hip internal rotation in runners suffering from PFPS. In 2010, Dierks et al. reported reduced and slower movement patterns for a group of runners with PFPS compared to healthy controls at the beginning of a long run and detected increases in joint excursion at the end. So, they assumed that diverse kinematic mechanisms as well as alterations due to fatigue may be related to PFPS in runners.

In summary, confounding evidence has been reported regarding lower leg kinematics as a risk factor for development of OI (Barton et al., 2009; van der Worp et al., 2011). Reasons for the contradictory findings may be ascribed to diverse limitations in methodology, study samples and design. Currently, most new studies use three-dimensional motion analysis systems which can be considered as the gold standard for the acquisition of kinematic data. However, there are numerous studies which still refer to findings originating from the 1980s based on either two-dimensional high-speed systems or goniometers (Clement et al., 1984) although it is well-known that these techniques are inaccurate and contain an indeterminable measurement error (Areblad et al., 1990). Additionally, the study performed by Clement et al. in 1984 did not even include healthy controls as comparisons. To date, it has not only been demonstrated that control groups are essential for analyses, it has also been shown by Grau et al. (2008b) that sex-related differences also influence biomechanical variables and that anthropometric variables affect biomechanical outcomes as well. Thus, a matched control group according to gender and anthropometric data is necessary in order to determine differences between two groups of runners. Therefore, results such as presented by Donoghue et al. (2008) need to be questioned since they included only high pronators with AT and ultimately associate excessive pronation with AT. Additionally, differences in individual striking patterns and used methodology affect the kinematic results; running

overground or on a treadmill (Nigg et al., 1995; Riley et al., 2008; Wank et al., 1998), running barefoot or shod (De Wit et al., 2000; Stacoff et al., 2000) and running on a hard or soft surface (Hardin et al., 2004; Gruber et al., 2013). Here, researchers must be cautious when comparing their own results with other findings. Further, the marker setup used to capture lower leg kinematics (Arnold and Bishop, 2013), the running speed (De Wit et al., 2000) as well as the calculation methods (for example the order of rotation sequences for quantifying joint excursion) all lead to different results and are not accounted for when comparing results between studies. In a review of kinematic risk factors for PFPS, Barton et al. (2009) conclude that prospective studies evaluating hip, knee and ankle/rearfoot kinematics are needed to fully determine evidence-based risk factors and to clarify cause-effect relationships.

Due to the missing evidence-based relationship between lower leg kinematics and injury, new methods for evaluating lower leg kinematics and their joint couplings have recently been presented in literature. Vector coding techniques or even more sophisticated algorithms such as continuous relative phase (CRP) and continuous relative phase variability (VCRP) are examples of possible methods to evaluate coordination patterns of the lower extremities (Burgess-Limerick et al., 1991; Burgess-Limerick et al., 1993; Chang et al., 2008; DeLeo et al., 2004; Dierks and Davis, 2007; Hamill et al., 1999; Heiderscheit et al., 2002; Kurz and Stergiou, 2002; Li et al., 1999; Miller et al., 2008; Peters et al., 2003; Sparrow et al., 1987; Tepavac and Field-Fote, 2001; van Emmerik and Wagenaar, 1996). Both approaches are based on the premise that one specific joint motion affects the motion of the adjacent joint and consequently influences lower leg joint coordination.

Vector coding unites continuous excursion angles of two motions of interest and describes their coordination patterns as in-phase or out-of-phase relationships (Chang et al., 2008; Dierks and Davis, 2007; Peters et al., 2003; Tepavac and Field-Fote, 2001). CRP combines qualitative information resulting from joint excursion angles with spatial-temporal information as joint excursion velocities in one variable (Hamill et al., 1999). This approach is considered as a quantitative way to analyze human locomotion which, again, may lead to new insights in injury mechanics (Burgess-Limerick et al., 1993; DeLeo et al., 2004; Hamill et al., 1999; Miller et al., 2008). VCRP is presumed to give information about the flexibility of human locomotion and to allow conclusions about healthy and injured movement patterns since a reduced VCRP is associated with pathology (Hamill et al., 1999; Heiderscheit et al., 2002). However, greater VCRP might also be an indicator for the existence of an injury as proposed by Miller et al. (Heiderscheit et al., 2002; Miller et al., 2008) which means that the relationship between VCRP and the generation of an OI is still not clarified.

2.2.2. Kinetics

There appears to be a reasonable association between abnormal joint loadings resulting from abnormal kinematics and subsequent contribution to the development of injury. Not only vertical joint loadings, resultant forces and joint moments (Cavanagh and Lafortune, 1980; Lieberman et al., 2010; Milner et al., 2006; Nigg and Wakeling, 2001; Pohl et al., 2009; Stefanyshyn et al., 2001; Stefanyshyn et al., 2006; Thijs et al., 2008), but also excessive impact forces (Cavanagh and Lafortune, 1980; Lieberman et al., 2010; Thijs et al., 2008) acting on the musculoskeletal systems at touchdown, active push-off forces during terminal stance phase (Thijs et al., 2008; Winter, 1983), or an altered pressure distribution under foot (Grau et al., 2008a; Thijs et al., 2008; van Ginckel et al., 2009; Willems et al., 2005; Willems et al., 2006; Willems et al., 2007) are of great interest in current scientific research. Again, as in clinical and kinematic data, contradictory results are presented in literature to date, and thus, no evidence-based kinetic risk factor for the development of OI in runners has yet been determined (Hreljac, 2005). Despite the findings Hreljac et al. (2000) who showed a relationship between excessive vertical impact force and loading rates for runners who suffered from stress fractures, the requirement for prospective studies is clear since only prospective approaches can clarify cause-effect relationships and finally lead to preventive measures.

Female runners suffering from PF exhibit high impact forces and loading rates compared to a healthy control group (Davis et al., 2004) however differences between the injured and uninjured side in a group of patients with PF have not been found (Liddle et al., 2000). Milner et al. (2006) and Pohl et al. (2009) report a relationship between high loading rate and the development of tibial stress fractures and PF whereas the results as shown by Stefanyshyn et al. (2006) do not support this wide spread theory. In contrast, Stefanyshyn et al. (2006) associate increased knee joint moments with OI in runners.

Several prospective studies have been carried out using pressure plates to acquire plantar pressure distribution under foot during running by injury-free novice runners (Thijs et al., 2008; van Ginckel et al., 2009) and by injury-free physical education students (Willems et al., 2005; Willems et al., 2006; Willems et al., 2007). Thijs et al. (2008) and van Ginckel et al. (2009) analyzed data from the same cohort and found greater impact forces at heel strike and higher propulsive forces during stance phase for runners generating PFPS (Thijs et al., 2008) whereas runners developing AT revealed a more inverted touchdown and roll-over-process as well as reduced propulsive forces during stance phase (2009). In contrast,

Willems et al. (2006) observed a central heel strike, greater pronation and higher loads under the medial side of the foot and greater inversion and inversion velocity for students generating OI.

However, kinetic approaches to measure ground reaction forces or plantar pressure distribution patterns have not proved to be useful investigating the development of OI in runners (Grau, 2006). For this reason, kinetic measurement techniques have not been implemented in the current study.

2.2.3. Muscular strength

Deficits in muscular strength, or muscular imbalances, are presumed to be additional factors leading to the development of OI. It can be assumed that sufficient and well-balanced muscular strength might reduce the risk of developing an OI since eccentric strength training is regularly implemented in rehabilitation programs.

The quantification of muscular strength is used to diagnose acute and chronic OI in runners. In recent studies, hip abductor strength was of major interest since weak hip abductors are considered to have great influence on lower extremity kinematics and the development of knee and ankle OI (Krauss et al., 2007; Niemuth et al., 2005). Novacheck (1998) describes the hip abductors as one of four tissues which are activated during initial stance phase to attenuate the shock on the musculoskeletal system caused by heel strike while running. Decreased hip abductor strength or abductor-adductor imbalance as presented by Niemuth et al. (2005) consequently disables an effective shock absorption mechanisms, leading to more hip adduction during stance phase and, due to kinematic coupling mechanisms, to greater internal tibial rotation and finally to excessive rearfoot eversion. This relationship suggests that abnormal kinematics of the rearfoot, for instance, might be a consequence of weak muscles surrounding the hip joint and therefore demonstrates the necessity of future studies that carry out multifactorial analyses of biomechanical factors leading to OI in runners.

Beside such functional testing procedures as circumferential measurements, dynamic isokinetic strength measurements appear to be particularly reasonable in identifying a causal relationship between muscular strength or muscular imbalance and the generation of injury. Since running patterns are characterized by alternating concentric and eccentric contractions of the corresponding muscle groups (hip abductors and adductors, quadriceps, hamstrings), maximal strength is measured according to the muscles' main directional movement in their

corresponding form of contraction. Isokinetic measurement devices can be used to measure maximal concentric (positive dynamic), isometric (static) and eccentric (negative dynamic) muscle contractions. As part of clinical gait analyses, the measurement of maximal isometric strength of running relevant muscle groups (hip abductors and adductors, knee flexors and extensors, abdominal flexors and back extensors) has proven to be essential in determining potential deficits causing injury and to define therapeutic interventions for the treatment of injury.

In detail, injured runners exhibit weak hip joint surrounding muscles and side-to-side muscular imbalances in hip flexion, abduction and adduction (Niemuth et al., 2005). It needs to be noted that these authors did not differentiate between different symptoms in their evaluation of hip muscle strength. Fredericson et al. (2000), however, focused on the evaluation of hip abductor strength for long distance runners suffering from ITBS and found significant weaker hip abductor strength compared to healthy runners. For the generation of PT, Devan et al. (2004) revealed an imbalanced hamstring-quadriceps ratio as a potential risk factor whereas Krauss et al. (2007) concluded that neither quadriceps and hamstring strength nor their calculated balance had any influence on the development of injury. With regard to the influence of muscular strength on PFPS, Lankhorst et al. (2012) reviewed recent literature and determined lower knee extensor strength to be a major risk factor for its generation. However, due to the small number of studies they reference, it is important to interpret the results cautiously. Hirschmüller et al. (2005), Mahieu et al. (2006) and McCrory et al. (1999) found a decreased strength performance capacity for the plantarflexor muscle group for runners suffering from AT. Comparable results were presented by Haglund-Akerlind and Eriksson (1993) who reported lower eccentric torques of the gastrocnemius/soleus complex for AT runners.

In summary, the author agrees with Almekinders and Temple (1998) and Ryan et al. (2006) who emphasize that, to date, no distinct and causative connection between muscular weakness or imbalance and the development of OI has been demonstrated. Different measurement devices (handheld dynamometers, isokinetic dynamometers) were used to measure eccentric, isokinetic or isometric maximal muscular strength and endurance of diverse muscle groups so that comparability between results of different studies is not guaranteed. Since most studies were based on a retrospective study design, prospective studies which focus on different symptoms are required to assess muscular strength of hip, knee and ankle joint surrounding muscles and to finally clarify cause-effect relationships.

2.3. Training-related factors

The investigation of the influence of training on the development of OI has been part of numerous studies. Hreljac (2005) stated that all OI are caused by training errors and therefore could have been avoided by different training programs since training programs can be controlled by the athlete or coach and therefore can be adjusted for and consequently modified according to individual sensation. Yeung and Yeung (2001) showed a link between great training intensity and the increased risk of OI and therefore stated that modifications in training programs, especially reductions in running frequency and mileage lead to a reduction of incidence rates.

In a recent review, Nielsen et al. (2012), pointed out that due to differences in the periods of observation, included running populations and documentation techniques utilized, no evidence-based and distinct risk factors have been found leading to OI in runners. Approaches analyzing training data with regard to the development of OI are manifold and differ between one another. Thus, Nielsen et al. (2012) revised four main variables which have been associated with injury in past investigations to determine training-related risk factors: mileage or running volume, duration, frequency and intensity. They concluded that an identification of training-related risk factors has not yet been successful and future attention should be focused on possible interactions between training variables as well as on the impact of sudden increases in training volume, duration and frequency. A sudden increase in training volume in particular has often been associated with the development of injury (Buist et al., 2008; Fields et al., 2010; Lysholm and Wiklander, 1987; Rolf, 1995). Rolf (1995) presumed that slow and gradual increases in training volume could lead to a positive adaptation to the new demands the body is exposed to and consequently reduced risk of injury. This assumption has not been confirmed since Buist et.al (2008) showed no difference in incidence rates between novice runners participating in either a standard training program or a graded training program. Other training variables such as running surface, distance profile or worn footwear have also been associated with OI in runners (Clarke et al., 1983; James, 1995; McKenzie et al., 1985; Stacoff et al., 1988). However, an evidence-based identification of risk factors causing injury has not yet been presented (Hreljac, 2005).

The investigation of training-related risk factors leading to OI in runners has been proven to be difficult. The analysis of supervised training programs for novice runners, military recruits or students leads to the conclusion that increases in training volume or high mileage favors the development of injury (Buist et al., 2008). These approaches enable easy analyses of

training variables, but do not represent a real situation for most recreational hobby runners since individual training programs often imply training breaks and phases of regeneration throughout the year. Consequently, the analysis of prospective training data considering increases and modification in training variables as potential risk factors is essential to determine their influence on the generation of OI in runners. Hoeberigs (1992), Nielsen et al. (2012) and Rolf (1995) emphasized that a large variation in injury definitions does exist, so that the comparability of past findings needs to be questioned. The subjective reporting of training data by mail, questionnaire or surveys appears to be an additional deficit in the execution of prior studies, since data might be reported incorrectly due to recall bias (Nielsen et al., 2012). The usage of GPS (Global Positioning System) technology to track the subjects during training is recommended by Nielsen et al. (2012) to enable an effective and objective evaluation of individual training programs.

Individual running experience and performance may also bias the presented results since a wide variety of runners are included in the various studies. A common theory describes greater running experience as a possible preventing factor in the development of OI, since novice runners have reported higher amounts of overuse symptoms in several studies. Experienced runners have a better “feeling” for their bodies which leads to a higher injury threshold compared to novice runners and they may consider running-related pain as not severe enough to be a reportable acute overuse symptom.

2.4. Summary

It has been demonstrated above that numerous variables have been considered as potential risk factors leading to OI in runners. However, it has also been shown that there is currently no consensus regarding evidence-based and multifactorial mechanisms of clinical, bio-mechanical and training-related risk factors leading to injury. Even the implementation of higher-dimensional calculation methods such as Vector Coding, CRP or VCRP have not led to new insights about how joint coordination patterns may influence the generation of OI.

The main reason for this lack of evidence lies in the designs of previous studies, since most studies have been performed retrospectively and have been based on naive approaches to identify mechanisms of injury. Thus, future studies should be conducted on a prospective basis to clarify cause-effect relationships. While the focus of past studies lay mostly with one-dimensional analyses of risk factors, future studies need to focus instead on the investigation

of multiple risk factors in order to understand how risk factors interact, and how these interactions lead to the development of an injury.

As there appear to be injury-specific risk factors and/or injury-specific mechanisms, different injury symptoms should not be summarized collectively under the broad terms “overuse injury” or “running related pain”. Consequently, effective studies are obliged to use an experienced physician or clinician not only to provide a diagnosis of the current complaint but also to verify the complaint against signs and symptoms of other diagnoses. Therefore, it is mandatory to rationalize definitions of injury on a medically diagnostic basis in order to appropriately include each injury in the study’s evaluation process. However, a well-designed study sample is not sufficient. A control group is critical in order to determine variables causing OI in runners, since, for example, the analysis of training-related risk factors for both novice and elite runners would lead to differing results. Thus, the definition of appropriate inclusion and exclusion criteria is essential in order to obtain a homogenous study sample. Further, to ensure that results are not dominated by anthropometric differences which may exist between two populations such as injured and healthy runners, the samples should be matched according to sex, height and weight.

Lastly, the overvaluation of results, the use of poor and diverse measurement techniques as well as differences in the applied calculation methods for kinematic and kinetic data can be listed as additional reasons for the disagreement regarding risk factors causing OI in runners.

Based on the outlined results and deficits of past studies, a prospective study has been carried out to determine multifactorial risk factors for the development of OI in recreational runners. Well-established, reasonable and reliable measurement techniques and transparent calculation methods were implemented to acquire relevant clinical and biomechanical data. Continuous training monitoring enabled the prospective evaluation of individual training programs, and medical examinations were utilized to assure the absence of injury in intake and to consistently diagnose current complaints in case of an injury.

3. Research questions

Based on the outlined state of research, three research questions are proposed which deal with new aspects considering the determination of risk factors causing overuse injuries in runners. Each research question is addressed and answered within a dedicated scientific paper.

Research question 1:
Is the Variability of Continuous Relative Phase an adequate calculation method to differentiate between lower leg kinematics of healthy and injured runners who reveal differences in commonly-used kinematic variables?

Research question 2:
Can prospective studies reveal multifactorial mechanisms causing the development of overuse injuries in runners?

Research question 3:
Are prospective studies necessary to identify kinematic risk factors causing overuse injuries in runners?

4. Methodology

In the following chapter, the prospective design of the current study is presented, the included running population is described and the implemented methodology, including clinical and biomechanical testing procedures and training documentation, is detailed.

4.1. Study design

The current study is based on a prospective approach which included an initial examination (IE) for each participating healthy runner at the beginning and a second examination (SE) in the case of an acute and diagnosed OI as the injury becomes symptomatic. A schema of the current study design is presented as Figure 2.

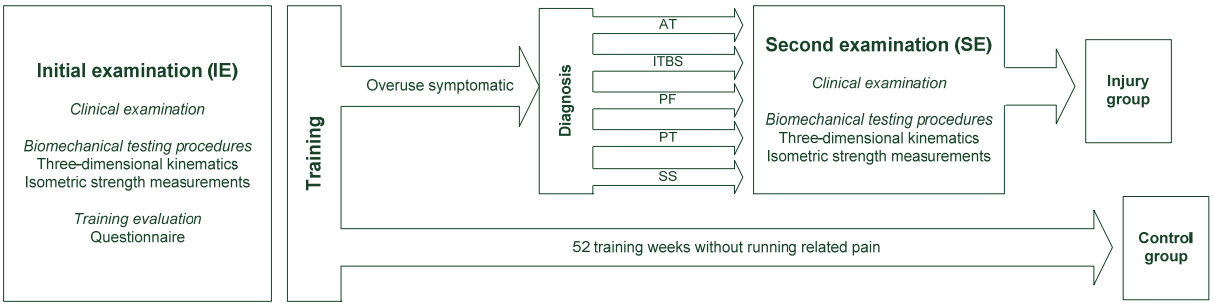


Figure 2: Schema of the current study design.

Note: AT = Achilles tendon pain, ITBS = iliotibial band syndrome, PF = plantar fasciitis, PT = patellar tendinopathy, SS = shin splints

Prior to the IE, the inclusion and exclusion criteria were consistently checked either via telephone or by e-mail to determine whether participation in the study is possible. If the subject met the criteria and the absence of an injury was confirmed by an orthopaedic surgeon, the IE was performed. Here, several clinical variables were assessed, three-dimensional kinematics were recorded, isometric maximal muscular strength was measured and documented, and a questionnaire about individual training habits was completed according to a standardized testing protocol. A detailed description of the conducted clinical and biomechanical testing procedures follows later in this chapter.

After the IE, each subject continued running according to their individual training programs without any further assistance or advice. Over a maximum period of 52 weeks, all runners

were requested to hand in training diaries on a weekly basis, providing information about training habits and pain due to running. A detailed list of training parameters is shown in section 4.6.2. No further examination was carried out if no pain occurred throughout the participation period. In contrast, if a runner suffered from an acute overuse symptom that was diagnosed as AT, ITBS, PF, PT or SS, the SE became necessary.

The existence of an overuse injury was defined as one of the following: medical attention was desired, 66% of all training sessions within two weeks were accompanied by pain, or 50% of all training session within four weeks were accompanied by pain and an OI was diagnosed by a surgeon.

4.2. Study sample

Diverse approaches were utilized to recruit runners for the current study. E-mails were sent to numerous running groups in and around Tübingen containing a flyer and an official information paper. Brochures and posters were also distributed and posted in sports shops or at running events and uploaded on the homepages of the Sports Medicine Tübingen, the "Nikolauslauf" and "Stadtlauf" in Tübingen as well as on diverse other homepages for running events. Lastly, advertisements were placed in local newspapers to arouse people's interest in the current study.

The advertisements focused on the recruitment of recreational and hobby runners which met the following inclusion criteria:

- All runners are between 18 and 55 years of age
- All runners are injury-free for at least the last six months prior to the participation
- All runners run a minimum of 20km per week

The following exclusion criteria were defined:

- Any runners who wear orthopaedic insoles in their running shoes
- Any runners who have had physical therapy during the last six months prior to the participation

Ultimately, 269 uninjured runners were recruited and passed the IE, including clinical examination, biomechanical testing procedures (three-dimensional kinematics and isometric strength measurements) and a questionnaire about individual training habits. 127 runners (47%) were subsequently excluded from the study due to missing feedback, other injuries or

personal and scheduling problems. Consequently, 142 runners (53%) completed their participation, sent in their training diaries on a regular basis and were therefore included in the data analysis. A detailed overview of the initial cohort, all subjects who passed the initial examination, is shown as Table 1.

97 of 142 runners remained uninjured, whereas 45 runners (32%) generated an OI. Ten runners suffered from AT, seven from PF, six from PT, three from SS and four runners from an OI located at the hip joint. Seven additional runners showed overuse symptoms located at the knee joint and five runners at the shank or foot. However, these symptoms were either not diagnosed, since the participants refrained from coming to the laboratory for an examination, or differed from the initially proposed five main running OI.

For the SE, 19 of the 45 injured runners were recruited. Five of these presented AT, six presented PF, five suffered from PT and three from ITBS.

Table 1: Overview of all subjects who were recruited for the study

Subjects	**Initial examination [n]**	**Percentage [%]**	**Second examination [n]**
Passed initial examination	269	100	
Completed participation	142	53	
Drop-outs	127	47	
Completed participation	142	100	
Uninjured runners	97	68	
Injured runners	45	32	
Injured runners	45	100	19
Achilles tendon pain	10	22	5
Plantar fasciitis	7	16	6
Patellar tendinopathy	6	13	5
Iliotibial Band Syndrome	3	7	3
Shin Splints	3	7	
Hip overall	4	9	
Knee unknown / other	7	16	
Foot unknown / other	5	11	

4.3. Clinical examination

The clinical examination, as the first part of the IE, was performed by an orthopaedic surgeon from the Department of Sports Medicine, Medical Clinic, University of Tübingen, Germany. All measurements performed during the IE are part of daily clinical routines and are included in clinical gait analyses, which have proved itself years in practice over the last several. The current Case Report Form (CRF), which was used to document all measurements of the IE, is attached as Appendix 10.1.

The examining surgeon quantified active and passive ranges of motion for hip, knee and ankle joints as well as for the rearfoot, according to the neutral-zero method in a supine position (Debrunner and Hepp, 1973; Ryf and Weymann, 1995). The application of a goniometer was neglected. Instead, joint mobility was assessed on a subjective basis according to Debrunner (1973) as displayed in Figure 3 and compared between both sides. Thus, a characterization of joint mobility as normal, restricted or increased was done by comparing with standard values whereby a difference of at least 10°-15° between both sides was necessary to define joint mobility as either restricted or increased.

Figure 3 displays the measurements of active ranges of motion as carried out in the clinical examination for the hip joint (a,b,c,d,e), knee joint (f), ankle joint (g) and rearfoot (h,i).

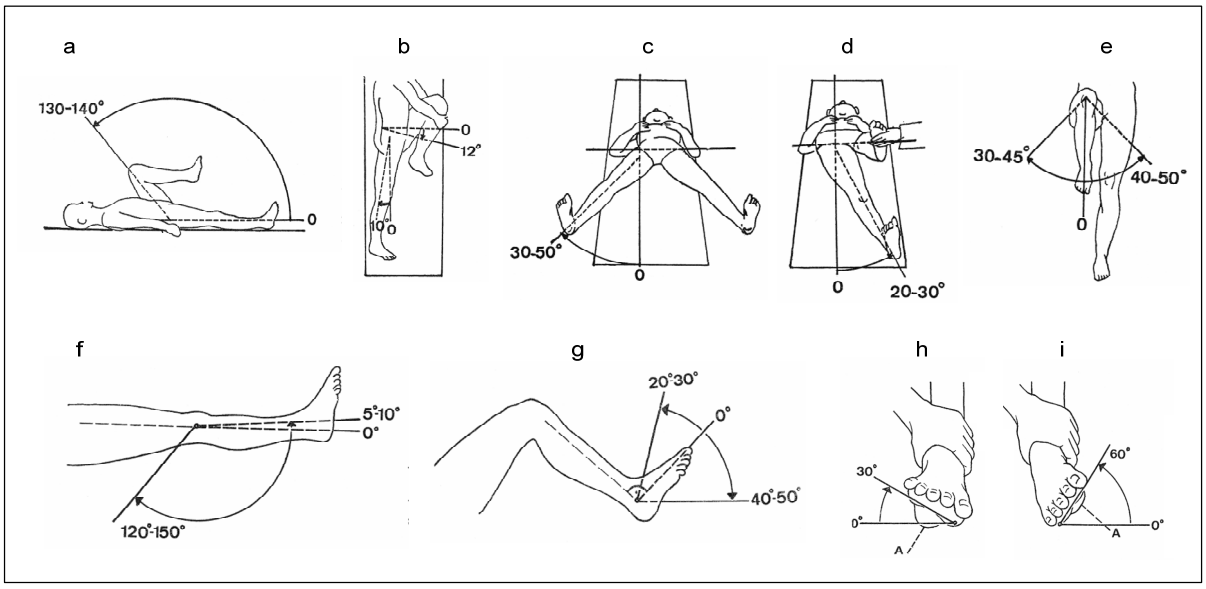


Figure 3: Measurements of active ranges of motion of hip, knee and ankle joint as well as of the rearfoot according to neutral-zero method[1]

[1] Adapted from Orthopädisches Diagnostikum, 2.Auflage, p.103 ff, ISBN: 3 13 324002 1

Standard values for active ranges of motion were defined as follows:

a) Hip flexion (knee flexed): 130°-140°

b) Hip extension (knee extended, lying on the side): 10°-20°

c) Hip abduction (knee extended): 30°-50°

d) Hip adduction (knee extended): 20°-30°

e) Hip internal rotation (knee flexed): 30°-45°

e) Hip external rotation (knee flexed): 40°-50°

f) Knee flexion: 120°-150°

f) Knee extension: 0°-10°

g) Ankle dorsiflexion (knee extended): 20°-30°

g) Ankle plantarflexion (knee extended): 40°-50°

h) Rearfoot Eversion (knee extended): 20°-40°

i) Rearfoot Inversion (knee extended): 40°-60°

Standards for passive ranges of motion are 5°-10° greater than the corresponding active values.

Further, finger-floor and heel-buttock distances were quantified, pelvic obliquity was checked and if necessary quantified. Tightness of the rectus femoris muscle, the iliopsoas muscle, the hamstrings and the iliotibial band (Obers' Test) was categorized as normal, slightly or clearly restricted. Pressure pain was assessed for the iliotibial tract, the gastrocnemius/soleus complex, the piriformis muscle, the gluteus medius muscle, the medial tibial crest, the lateral femoral epicondyle, the patella and the Achilles tendon and categorized as no pain, slight pain or clear pain. All tests were carried out in comparison between the left and the right sides.

Lastly, the incidences and diagnoses of past surgeries to the lower extremities as well as sustained OI were documented.

In the case of an acute overuse symptom arising during the time of participation, a clinical examination was necessary. Here, a diagnosis of the symptoms was deemed essential in order to determine whether it was possible to carry out the complete SE. The presence of one of the five major overuse injuries (AT, ITBS, PF, PT, SS) was used as the criterion for carrying out the second and final SE examination. The diagnosis of the presenting symptoms was based on the surgeons' experience and was accepted by the researcher.

4.4. Three-dimensional kinematics

Three dimensional kinematics were always recorded after the clinical examination and prior to the isometric strength measurements in order to avoid fatigue of the hip and knee joint surrounding muscles and consequently to avoid any influence on running patterns.

A six-camera infrared system (MCam1, Vicon®, Oxford, United Kingdom) was used to record the lower leg kinematics for both barefoot and shod conditions. Marker trajectories were captured with a sampling frequency of 250Hz. All subjects ran with a controlled running speed of 12km/h (SD 5%), first barefoot on a 13m ethylene-vinyl acetate (EVA) foam runway and second shod on a hardwood floor and wearing their own footwear. Running speed was controlled by two light barriers. Figure 4 shows the laboratory setup. A familiarization period of five to ten minutes was allowed for the subjects to become accustomed to the laboratory setup, the unfamiliar barefoot condition, as well as meeting running speed targets and performing on the soft substrate used to enable a natural striking pattern and an individual running style. The software Workstation V4.6 (Vicon®, Oxford, United Kingdom) was used to capture all static and dynamic kinematic trials and to control visually for measurement errors or artifacts, such as gaps in marker trajectories, ghost markers, etc. In case of measurement errors or other artifacts, the recorded trial was discarded and a next trial was captured. The subjects performed one static trial in a neutral standing position as well as 25 dynamic trials for each condition.

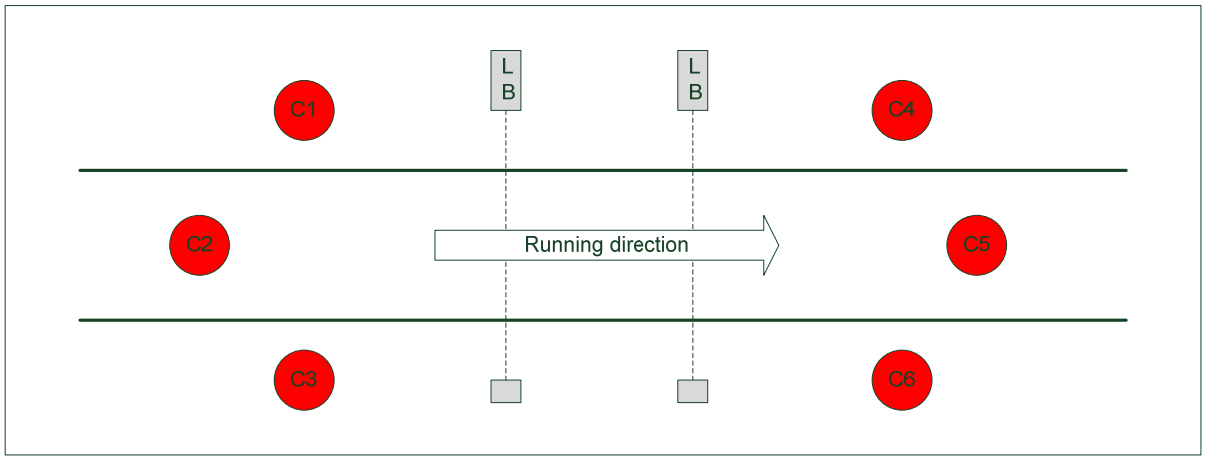


Figure 4: Laboratory setup for recording three-dimensional kinematics including six infrared cameras (C1-C6) and two light barriers (LB)

Since the calculation of three dimensional joint motions is based on the rigid-body model, 34 spherical reflective markers are placed on anatomical landmarks according to ISB recommendations to define seven segments for each subject, namely the pelvis and both lower extremities, each consisting of a thigh, a shank and a foot (Wu et al., 2002; van Sint, 2007). In detail, two markers were positioned on the anterior superior iliac spine and two markers on the posterior superior iliac spine to determine the pelvis segment. Three markers were attached to each thigh (greater trochanter, lateral and medial femoral epicondyle), six to each shank (medial and lateral ridge of tibial plateau, tibial tuberosity, medial crest of tibia, medial and lateral malleolus) and six to each foot (lateral, medial and posterior calcaneus, metatarsals 1 and 5, hallux). For the shod condition, the markers were sited on comparable positions on the upper of the subjects' footwear (lateral, medial and posterior heel counter, metatarsals 1 and 5, tip of the shoe). The marker placement for the barefoot condition, right-sided, is shown in Figure 5.

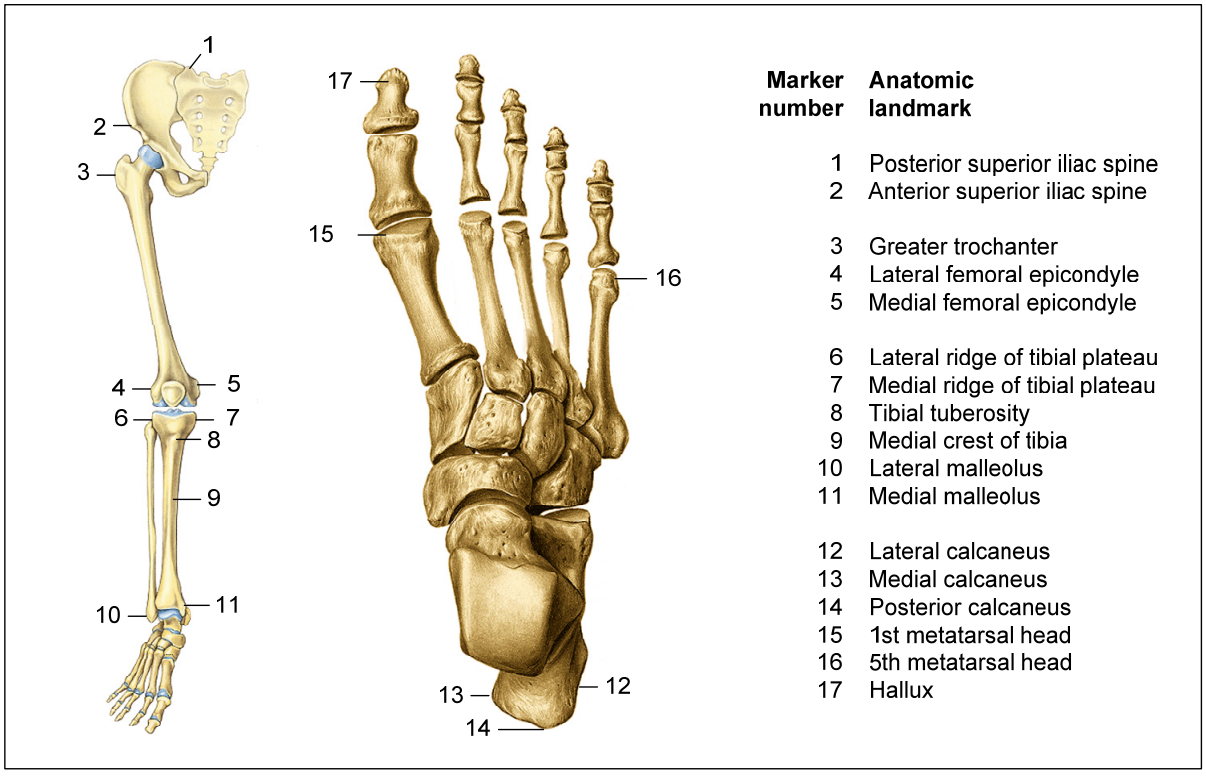


Figure 5: Standardized marker setup for the three-dimensional kinematics (right side)[2]

Joint excursion angles were quantified by calculating Cardan angles according to a calculation rule by Söderkvist and Wedin (1993) with the distal segment rotating with respect to the proximal segment. The first rotation always occurs around the sagittal joint axis describing flexion and extension of a joint, followed by a rotation about the frontal axis

[2] Adapted from Sobotta Atlas of Human Anatomy, 13th Edition, Urban & Fischer with own annotations

(abduction/adduction or inversion/eversion) and lastly by a rotation about the transversal axis (external and internal rotation).

The analysis of joint motion was restricted to the stance phase which was detected according to an algorithm described by Maiwald et al. (2009) and afterwards normalized to 100 data points. Joint excursion curves were calculated relative to the static neutral trial and averaged over 10 trials for each subject and calculated for sagittal and frontal hip motion, sagittal and transversal knee motion as well as sagittal ankle and frontal rearfoot motion. Eight discrete variables were then calculated for each joint motion. The entire computation of continuous and discrete lower leg kinematics was accomplished by a self-written MATLAB evaluation algorithm. Figure 6 displays typical continuous joint excursion curves and corresponding discrete variables for hip flexion/extension (A), hip abduction/adduction (B), knee flexion/extension (C), knee external/internal rotation (D), ankle dorsi/plantarflexion (E) and rearfoot inversion/eversion (F).

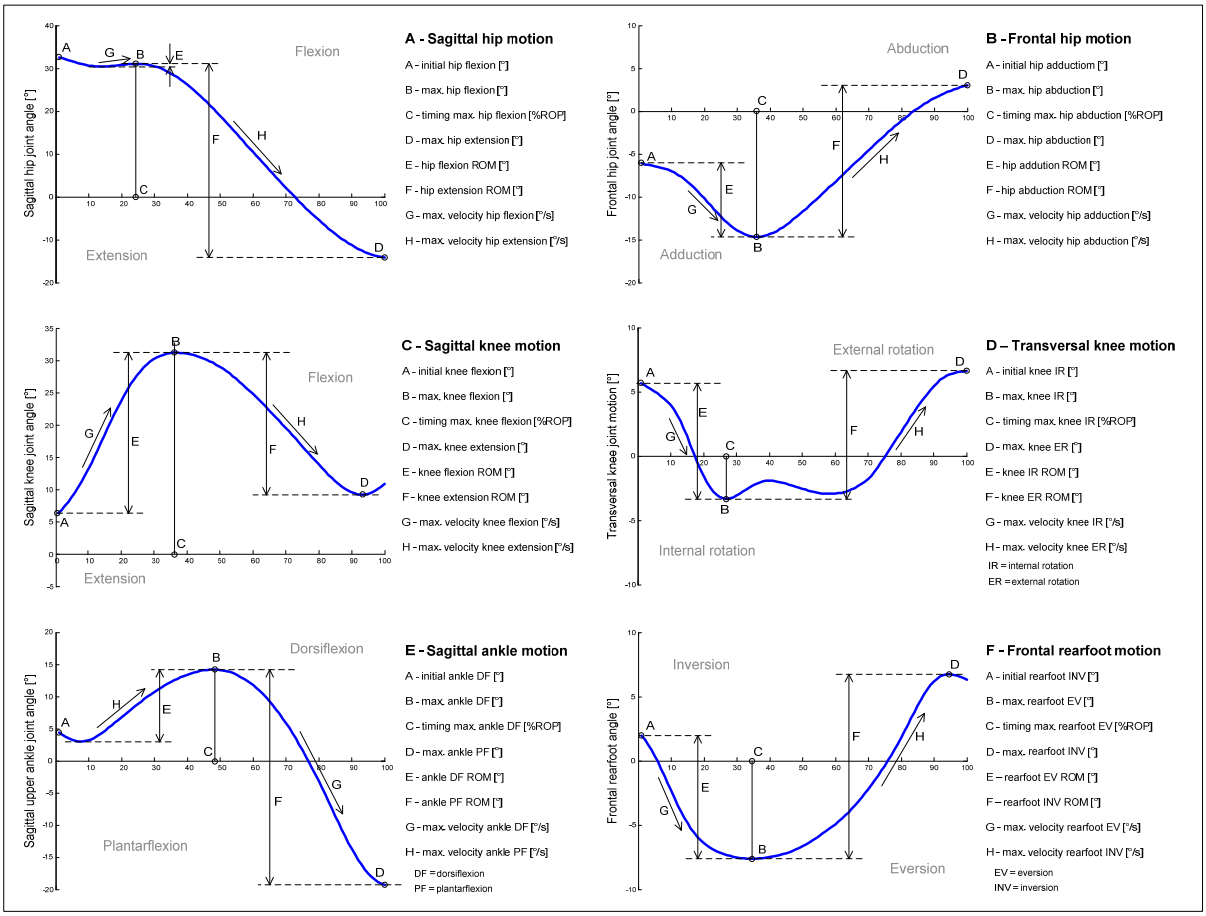


Figure 6: Continuous joint excursion curves and discrete variable for sagittal hip motion (A), frontal hip motion (B), sagittal knee motion (C), transversal knee motion (D), sagittal ankle motion (E) and frontal rearfoot motion (F).

4.5. Isometric strength measurements

Isometric strength measurements were performed after the recordings of three-dimensional kinematics. The standardized testing protocol began with measurements, in a seated position, of the upper body muscles, followed by the bilateral hip joint and unilateral knee joint surrounding muscles using DAVID devices (David GmbH & CO KG, Neu-Ulm, Germany). Subsequently, unilateral hip joint surrounding muscle strength was measured using the GENIUS ECO® HipMachine (FREI AG, Thalwil, ZH, Switzerland) in a standing position.

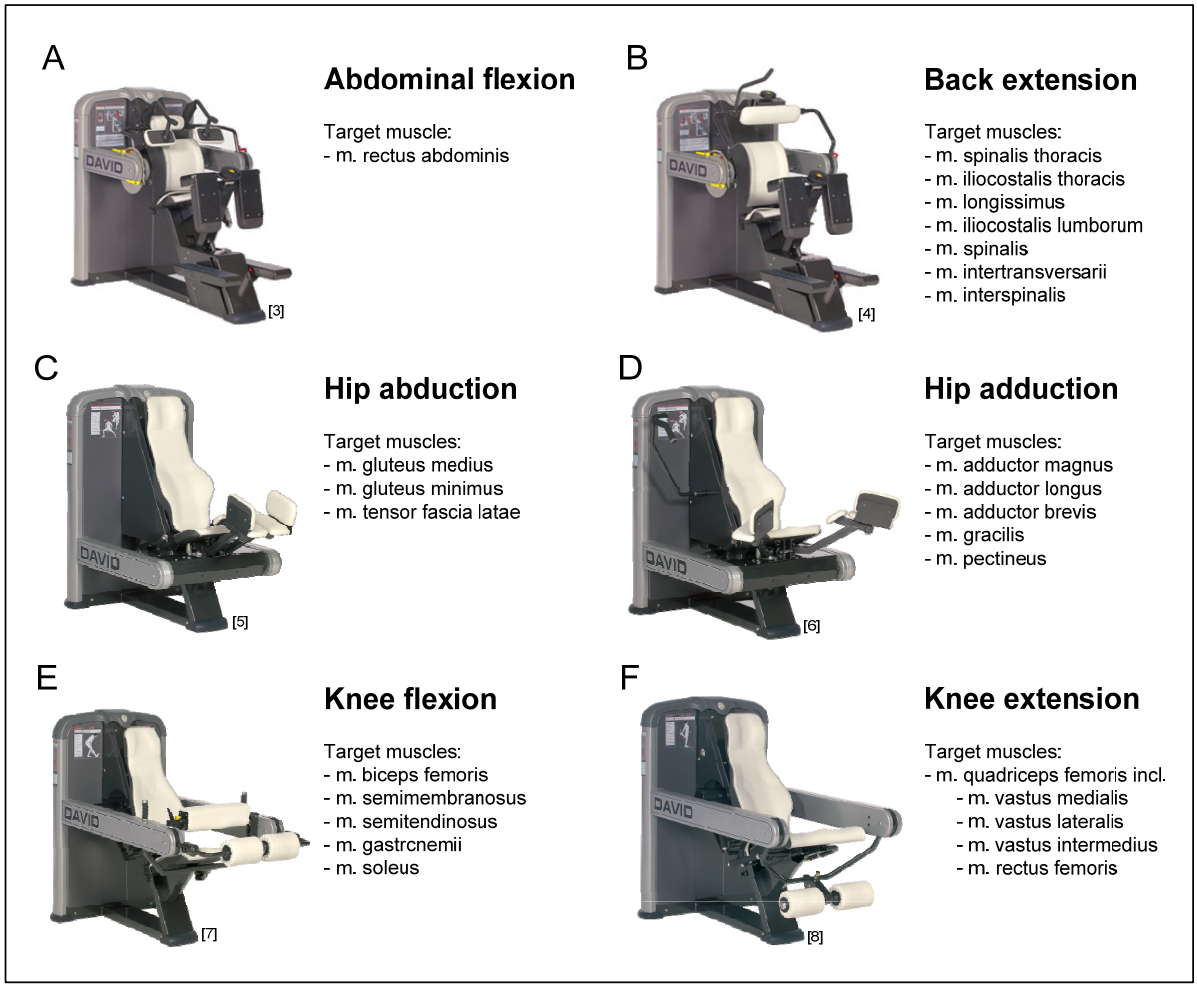


Figure 7: DAVID devices to measure maximal isometric strength of the upper body: abdominal flexors (A)[3] and back extensors (B)[4] as well as of the lower extremity: hip abductors (C)[5] and hip adductors (D)[6], knee flexors (E)[7] and knee extensors (F)[8] including the corresponding target muscles.

[3] From: http://www.david-international.com/cms/images/stories/F-LINE/F130h.jpg
[4] From: http://www.david-international.com/cms/images/stories/F-LINE/F110h_dms.jpg
[5] From: http://www.david.fi/images/made/uploads/products/F310_420_413.png
[6] From: http://www.david.fi/images/made/uploads/products/F320_420_395.png
[7] From: http://www.david.fi/images/made/uploads/products/F300_420_348.png
[8] From: http://www.david.fi/images/made/uploads/products/F200_420_385.png

All measurements were supervised and directed by an experienced physiotherapist. During a familiarization period the participants became accustomed to the direction of movement, as dynamic tasks were performed against an increasing resistance. After two sub-maximal contractions, two maximal isometric contractions were then performed at a standardized joint angle. All contractions were executed slowly up to maximum effort without explosive applications of force. The maximum torque or force was documented. The choice of joint angles was based on recent studies (Johnson et al., 2004; Knapik et al., 1983; Overend et al., 1992) in order to enable the possibility of comparing these results with other findings, or on device-specific presets.

For the upper body, the strength of straight abdominal muscles (abdominal flexion, AF, Figure 7A) and straight back muscles (back extension, BE, Figure 7B) was measured in a seated position with a knee flexion angle of 90° and an additional seat belt to fixate the pelvis to the device. During measurement of AF, the upper body was in an upright position (0°), whereas for the measurements of BE the upper body was positioned at a 30° forward incline. Hip joint surrounding muscles, here hip abductors (bHAB, Figure 7C) and adductors (bHAD, Figure 7D), were tested bilaterally in a seated position with a hip abduction angle of 30° (15° each leg). Unilateral knee flexor (KFL, Figure 7E) and extensor (KEX, Figure 7E) strength was again assessed in a seated position with a knee flexion angle of 30° for KFL and 60° for KEX.

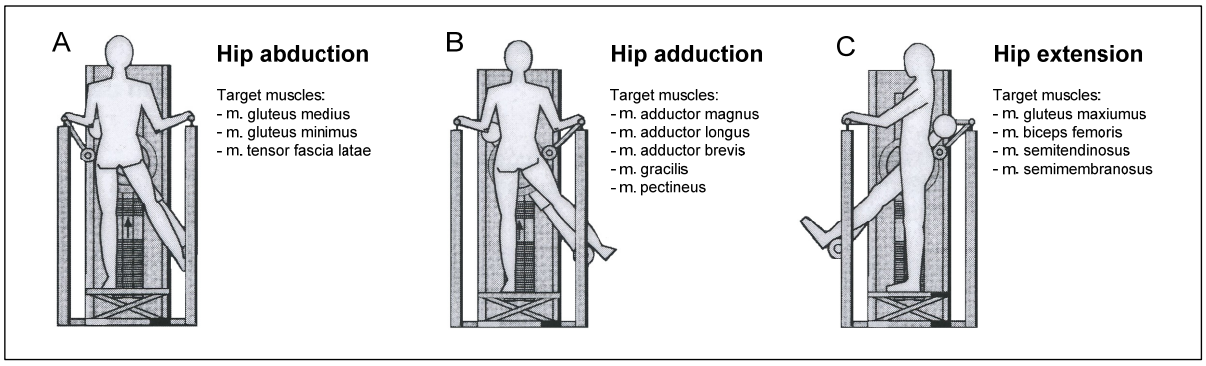


Figure 8: Unilateral maximal isometric strength measurements of hip abductors (A), adductors (B) and extensors (C) using the GENIUS ECO® Hip Machine (Frei AG, Switzerland) including the corresponding target muscles[9]

The unilateral measurements of hip abductor (uHAB, Figure 8A) and adductor (uHAD, Figure 8B) strength were carried out in a standing position with a hip abduction angle of 20° using the GENIUS ECO® HipMachine. Unilateral hip extensor strength (uHEX, Figure 8C) was also measured in a standing position with an extension angle of 0°.

[9] From: Gebrauchsanweisung GENIUS ECO® Hip Machine, Frei AG, Thalwil, Switzerland

During all measurements, the subjects were affixed to the device by flanking pads or seatbelts which could be individually adjusted; subjects were not allowed to self-stabilize by using their hands.

The following variables have been measured:

- Maximal abdominal flexor strength [Nm]
- Maximal back extensor strength [Nm]
- Maximal bilateral hip abductor strength [Nm]
- Maximal bilateral hip adductor strength [Nm]
- Maximal unilateral hip abductor strength [N]
- Maximal unilateral hip adductor strength [N]
- Maximal unilateral hip extensor strength [N]
- Maximal unilateral knee flexor strength [Nm]
- Maximal unilateral knee extensor strength [Nm]

Based on the maximal strength measurements, the following ratios were calculated:

- Upper body strength ratio (AF/BE)
- Bilateral hip strength ratio (bHAB/bHAD)
- Unilateral hip strength ratio (uHAB/uHAD)
- Knee strength ratio (KFL/KEX)

4.6. Training-related variables

4.6.1. Questionnaire

In the course of the IE, each participant completed a questionnaire about individual running experience, and provided records and training habits throughout the previous 12 months leading up to the participation.

The following information was required:

- Running experience [years]
- Average mileage per week [km]
- Average number of training sessions per week [n]
- Average running time per week [min]
- Average running pace [km/h, min/km or heart beats/min]
- Distribution of hard, medium and soft running surfaces during training routines [%]

- Personal records for 10km, half-marathon and marathon races [min]
- Forms of additional exercises performed besides running and duration per week [min]
- Footwear utilized [neutral or supported]

4.6.2. Training documentation

During participation, all subjects were asked to keep a weekly training diary (see Figure 9) with information about their individual training programs, and to submit it regularly via e-mail or by post.

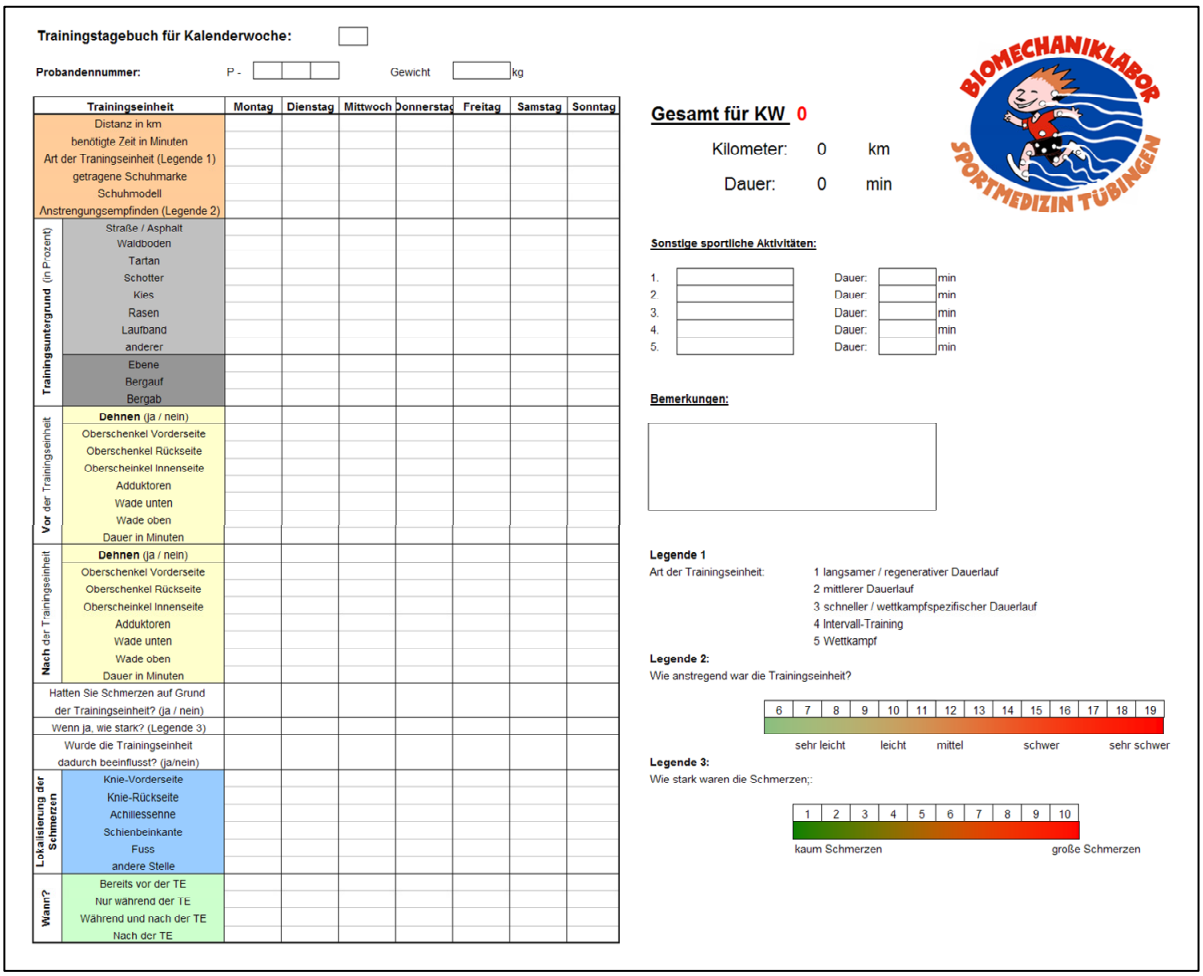
The following information was therefore provided for each training session of the week:

- Mileage [km]
- Running time [min]
- Type of training sessions [slow, medium, fast, intervals or competition]
- Exertion [Borg-Scale]
- Additional exercising [min]
- Distribution of hard, medium and soft running surfaces [%]
- Distribution of level, uphill and downhill running terrain [%]
- Distribution of even and uneven running substrates [%]
- Footwear utilized [neutral or supported]
- Occurrence of pain [yes/no; before, during, after the training session]
- Location of pain [left/right, knee, Achilles tendon, etc.]

Based on the individual training diaries, a variety of variables were calculated and analysed:

- Weekly mileage [km]
- Weekly running time [min]
- Mean exertion [Borg-Scale]
- Weekly additional exercising [min]
- Distribution of types of training sessions [%]
- Distribution of hard, medium and soft running surfaces [%]
- Distribution of level, uphill and downhill running terrain [%]
- Distribution of even and uneven running substrates [%]
- Distribution of neutral and supported footwear [%]

A specific MATLAB code was written to calculate all relevant training variables. For the analysis of data, two different approaches were utilized to determine training-related risk factors. First, training variables were averaged over the whole time of participation for each subject. Second, for a prospective analysis of training data, the last four weeks prior to the onset of injury were averaged and compared with the averages over the rest of the participation period.



Trainingstagebuch für Kalenderwoche:

Probandennummer: P - Gewicht kg

Trainingseinheit		Montag	Dienstag	Mittwoch	Donnerstag	Freitag	Samstag	Sonntag
Distanz in km								
benötigte Zeit in Minuten								
Art der Traningseinheit (Legende 1)								
getragene Schuhmarke								
Schuhmodell								
Anstrengungsempfinden (Legende 2)								
Trainingsuntergrund (in Prozent)	Straße / Asphalt							
	Waldboden							
	Tartan							
	Schotter							
	Kies							
	Rasen							
	Laufband							
	anderer							
	Ebene							
	Bergauf							
	Bergab							
Vor der Traningseinheit	**Dehnen** (ja / nein)							
	Oberschenkel Vorderseite							
	Oberschenkel Rückseite							
	Oberscheinkel Innenseite							
	Adduktoren							
	Wade unten							
	Wade oben							
	Dauer in Minuten							
Nach der Traningseinheit	**Dehnen** (ja / nein)							
	Oberschenkel Vorderseite							
	Oberschenkel Rückseite							
	Oberscheinkel Innenseite							
	Adduktoren							
	Wade unten							
	Wade oben							
	Dauer in Minuten							
Hatten Sie Schmerzen auf Grund der Traningseinheit? (ja / nein)								
Wenn ja, wie stark? (Legende 3)								
Wurde die Traningseinheit dadurch beeinflusst? (ja/nein)								
Lokalisierung der Schmerzen	Knie-Vorderseite							
	Knie-Rückseite							
	Achillessenne							
	Schienbeinkante							
	Fuss							
	andere Stelle							
Wann?	Bereits vor der TE							
	Nur während der TE							
	Während und nach der TE							
	Nach der TE							

Gesamt für KW 0

Kilometer: 0 km

Dauer: 0 min

BIOMECHANIKLABOR SPORTMEDIZIN TÜBINGEN

Sonstige sportliche Aktivitäten:

1. Dauer: min
2. Dauer: min
3. Dauer: min
4. Dauer: min
5. Dauer: min

Bemerkungen:

Legende 1

Art der Traningseinheit:
1 langsamer / regenerativer Dauerlauf
2 mittlerer Dauerlauf
3 schneller / wettkampfspezifischer Dauerlauf
4 Intervall-Training
5 Wettkampf

Legende 2:

Wie anstregend war die Traningseinheit?

6 7 8 9 10 11 12 13 14 15 16 17 18 19

sehr leicht leicht mittel schwer sehr schwer

Legende 3:

Wie stark waren die Schmerzen,:

1 2 3 4 5 6 7 8 9 10

kaum Schmerzen große Schmerzen

Figure 9: Weekly training diary for the participants

5. Scientific papers

This chapter of the doctoral thesis consists of three research papers dealing with the determination of risk factors causing OI in runners. In the first paper, a higher dimensional calculation method is presented, applied and critically examined to investigate joint coordination patterns in healthy and injured runners. The second research paper deals with the prospective evaluation of clinical, biomechanical and training-related risk factors on the development of overuse injuries in runners. The third article examines the need for prospective studies to determine risk factors causing OI in runners and to clarify cause-effect relationships. An overview of these manuscripts is given in Table 2. The first and second manuscripts correspond with the original article accepted for publication by the corresponding journal. The third manuscript has been submitted and is under review. The acceptance letter of scientific paper 2 is attached as Appendix 10.2; the proof of submission of scientific paper 3 as Appendix 10.3.

Table 2: Overview of the scientific papers of the doctoral thesis

Title	**Authors**	**Year**	**Journal**
Using the variability of continuous relative phase as a measure to discriminate between healthy and injured runners.	Hein T, Schmeltzpfenning T, Krauss I, Maiwald C, Horstmann T, Grau S	2012	Human Movement Science, Volume 31, Issue 3, June, 2012, pages 683–694
Prospective analysis of intrinsic and extrinsic risk factors on the development of Achilles tendon pain in runners.	Hein T, Janssen P, Wagner-Fritz U, Haupt G, Grau S	2013	Scandinavian Journal of Medicine and Science in Sports (accepted 4. Sept. 2013) doi: 10.1111/sms.12137
Are prospective studies necessary to identify kinematic risk factors causing overuse injuries in runners?	Hein T, Janssen P, Wagner-Fritz U, Barisch-Fritz, B, Grau S	2013	Scandinavian Journal of Medicine and Science in Sports (submitted 8. Oct. 2013, under review)

5.1. Using the variability of continuous relative phase as a measure to discriminate between healthy and injured runners.

Tobias Hein Dipl-Ing (FH)[1], Timo Schmeltzpfenning Dipl-Ing (FH) [1], Inga Krauss PhD[1], Christian Maiwald PhD[2], Thomas Horstmann MD[3], Stefan Grau PhD[1]

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Abstract

Several studies use variability of continuous relative phase (CRP) to investigate overuse injuries, since low variability is thought to be related to running injuries. This study investigates whether the analysis of CRP variability leads to additional information about possible differences or similarities between healthy and injured runners. Further, a decision about future applications of CRP variability should be based on the ability to implement and interpret data.

18 healthy female runners (CO) and 18 female runners who suffered from iliotibial band syndrome (ITBS) were evaluated by calculating CRP variability for 4 coupling pairs. Besides analysing continuous variability of CRP, we also averaged it for the whole stance phase and for four predefined stance phase intervals. Confidence intervals were displayed and independent t-tests for comparing the two groups were conducted.

During initial and terminal stance phase as well as after heel-off an increase in CRP variability was detected for both groups of runners. In contrast, the foot flat period was characterized by stable joint coordination and a decrease in variability. This paper presents possible interpretations of CRP variability but no statistically significant differences in CRP variability were found between the two groups of runners. Despite the missing statistical significance, a relationship between high CRP variability and injury seems to be conceivable, since the injured runners demonstrated an increased variability for all couplings in the first half of the stance phase.

Further application of CRP variability in biomechanical research is essential to determine whether a relationship exists between injury and coordination variability.

Keywords: variability; joint coupling; iliotibial band syndrome; running kinematics; overuse injuries

1. Introduction

Running is one of the most popular recreational activities worldwide (van Mechelen, 1992). Recreational athletes often increase the amount and intensity of training as soon as their fitness levels improve. Consequently, the human body reacts to this increase in training, often leading to overuse injuries which are predominantly associated with the lower extremities, particularly the knee joint (Hreljac et al., 2000; James, 1995; Marti et al., 1988; Taunton et al., 2002). The causes of overuse injuries, in general, are still unclear and seem to be multi-factorial (Kannus, 1997). This is also true for the occurrence of iliotibial band syndrome (ITBS). Besides diverse intrinsic risk factors such as muscular deficits (Fredericson et al., 1997; Messier et al., 1995) and kinetic abnormalities (Messier et al., 1995), there are several kinematic variables that can be associated with ITBS, e.g. maximal angular displacements and their maximal velocities, as well as the timing of maximal values (Grau et al., 2010; Messier et al., 1995; Miller et al., 2007; Noehren et al., 2007; Orchard et al., 1996). However, since precise reasons for the development of overuse injuries as ITBS could not be clearly identified, several studies investigate joint coordination to get more information about human movement. The main idea behind these approaches is that the movement of one joint is influenced by and will affect the action of an adjacent joint. In the case of irregularities occurring in kinematic variables during a motion task, it is apparent that the transmission of emerging anomalies on the next joint will result in a disturbed joint coordination.

Besides discrete values describing joint coordination, such as excursion ratios (Dierks and Davis, 2007; McClay and Manal, 1998; Williams, III et al., 2001), the importance of continuous methods and their variability has increased in recent biomechanical research (Burgess-Limerick et al., 1993; Dierks and Davis, 2007; Hamill et al., 1999; Hamill et al., 2000; Kurz and Stergiou, 2002; Li et al., 1999; Miller et al., 2008; Scholz, 1990; van Emmerik and Wagenaar, 1996; Wang et al., 2009; Yen et al., 2009). Furthermore, the variability of joint coordination may be a reason for the development of a running injury (DeLeo et al., 2004). For this purpose, researchers use a calculation method that originates from the Dynamical Systems Theory; the continuous relative phase (CRP) (Scholz, 1990). It unites spatial and temporal aspects of human movement and allows the continuous analysis of the entire stance phase or stride cycle. CRP also provides information about the flexibility of human gait and thus allows the differentiation between a healthy and an injured state, since reduced flexibility is often associated with pathology (Hamill et al., 1999; Heiderscheit et al., 2002). Hamill et al. (1999) state that CRP leads to further information about the incidence of running injuries, and that a less variable CRP is symptomatic for the presence of an injury.

Runners suffering from patellofemoral pain (PFP) are thought to use a reduced range of motion to complete their movement tasks which would result in decreased flexibility followed by a permanent stress on the local tissue and consequently in the development of an overuse injury. According to Miller et al. (2008), not only a reduced, but also an increased CRP variability seems to be a possible indicator for a running injury. They consider the increased CRP variability to be a consequence of an adapted running style that enables pain-free running. Therefore, the connection between CRP variability and the development of running injuries is still unclear (DeLeo et al., 2004).

The main objective of this study was to decide whether or not CRP variability is an effective and beneficial method for providing information about possible differences or similarities between injured and non-injured runners. Please note that the intention of this study was not to determine precise reasons for the incidence of ITBS. Therefore, we evaluated the same population of runners as presented by Grau et al. (2010), who compared healthy and injured runners suffering from ITBS and found that the injured runners with ITBS exhibited significantly reduced maximal velocities for hip flexion, hip abduction, knee flexion and ankle flexion, as well as reduced maximal hip adduction, reduced range of motion for the frontal hip movement and earlier maximal hip flexion (Grau et al., 2010). Based on these findings, we assume that differences will also emerge by the analysis of CRP variability, in general. Due to the study by Hamill et al. (1999) we hypothesize that symptomatic runners (ITBS) display a reduced CRP variability in contrast to healthy controls (CO).

To investigate whether knee joint motion is affected by, or possibly affects the motion of the hip or ankle joint, it is insufficient to calculate the CRP variability of hip and ankle joint motions. The same is true when calculating the variability of two motions of the same joint, for example HIPabd/ad – HIPflex/ex. Since ITBS evolves into pain over the lateral aspect of knee joint due to the repetitive rubbing of the iliotibial band over the lateral epicondyle of the femur while running (Fredericson and Wolf, 2005; Khaund and Flynn, 2005; Messier et al., 1995), the inclusion of knee joint motion is the logical consequence for all calculations. Thus, we mainly focused on the following three couplings: HIPflex/ex – KNEEflex/ex, HIPabd/ad – KNEEflex/ex and KNEEflex/ex – ANKLEflex/ex. Abnormalities in frontal plane motion of the subtalar joint or of foot motion are also associated with the incidence of ITBS. Stergiou et al. (2001) stated that a lack of coordination between subtalar joint motion and knee joint motion possibly leads to the development of a running injury. Anomalies in kinematic variables were also detected for runners suffering from ITBS as an increased supination velocity at push-off (Messier et al., 1995), a smaller subtalar inversion angle at touchdown (Messier et al., 1995) or an increased maximum foot inversion (Miller et al., 2007). Although the included

symptomatic runners did not show significant differences in kinematic data for subtalar joint motion, the combination of KNEEflex/ex – ANKLE ev/in was also included in this study.

When examining the results by Grau et al. (2010), who reported three significant differences between CO and ITBS for frontal hip motion (reduced maximal hip abduction velocity, reduced range of motion and reduced maximal adduction for ITBS), we expect the largest discrepancies between CO and ITBS for HIPabd/ad – KNEEflex/ex. We also expect differences between CO and ITBS for the other couplings. Finally, we intend to determine whether or not CRP variability is reasonable for future biomechanical analyses. The decision about any further application will be based on the ability to calculate and to interpret data, as well as on the possible limitations in dealing with CRP variability.

2. Materials and methods.

2.1 Subjects

3-D-kinematics of 18 healthy female runners (CO: mean age 37 years (SD 9), mean height 177 cm (SD 9); mean weight 70 kg (SD 10), BMI 22 kg/m² (SD 2) and 18 female runners suffering from Iliotibial Band Syndrome (ITBS: mean age 36 years (SD 7), mean weight 177 cm (SD 8), mean weight 71 kg (SD 12), mean BMI 23 kg/m² (SD 3)) were evaluated using CRP variability for assessing the flexibility of lower extremity joint coordination. The subjects of both groups were matched according to gender, age, height and weight.

The inclusion and exclusion criteria and an orthopaedic examination of the knee and lower extremity were carried out by an orthopaedic surgeon following a standardized testing protocol (Grau et al., 2010). This study complies with the declaration of Helsinki, and all subjects signed a written consent form approved by the university ethics committee.

2.2 Protocol

All subjects performed the same standardized protocol using the same marker set and laboratory setup. After a static trial, all subjects performed a minimum of 7 valid trials on a 13m EVA foam runway recorded using a 6-camera infrared system (Viconpeak, MCAM M1, 250Hz, Oxford, UK). The runway's density of 100kg/m³ was soft enough to enable natural and comfortable barefoot running.

A valid trial was characterized by a defined speed of 3.3 m/s (+- 5%). This study only included rearfoot or heel strikers who showed a natural heel-toe running style. This criterion was observed and rated by the researcher. The measurements were conducted unilaterally:

the affected leg of subjects from the ITBS group and the corresponding leg of the matched healthy subjects. The applied marker set consisted of 18 spherical reflective markers marking the pelvis (4th lumbar vertebra, 2x ASIS), thigh (greater trochanter, lateral and medial condyle), shank (tibial tuberosity, tibial crest, lateral and medial malleolus) and foot (posterior calcaneus, medial and lateral calcaneus, navicular and cuniform bones, metatarsals 1, 2/3 and 5).

Three dimensional joint motions were filtered by a Woltring filter which is provided by the software (Workstation V4.6, Vicon Peak, Oxford). The filter routine is based on a quintic spline algorithm with a mean square error of 2 mm². Further, joint motions were quantified by calculating Cardan angles (Cappozzo et al., 2005; Grood and Suntay, 1983) using the software BodyBuilder (ViconPeak, Oxford). The joint centres and the joint axes of the four segment model of the lower extremity were calculated according to Isman & Inman (1969) and Bell et al. (1989). The stance phase (roll-over process = ROP) was defined by a calculation method presented by Maiwald et al. (2009) and normalized to 100 data points. Angular displacements for hip flexion and extension, hip abduction and adduction, knee flexion and extension, plantar flexion and dorsiflexion in the upper ankle joint, as well as inversion and eversion in the subtalar joint were calculated relative to the static calibration trial.

2.3 CRP Variability

Several methods to calculate CRP have been presented in scientific literature (Burgess-Limerick et al., 1993; Dierks and Davis, 2007; Hamill et al., 1999; Kurz and Stergiou, 2002; Li et al., 1999; Miller et al., 2008; Scholz, 1990; van Emmerik and Wagenaar, 1996; Wang et al., 2009; Yen et al., 2009). To enable us to compare our results with those from other studies, we decided to use the calculation method described by Hamill et al. (1999), Dierks & Davis (2007) and Miller et al. (2008).

Two normalization techniques to calculate CRP were applied to reduce the influence of frequency discrepancies of the signals and their high amplitudes of angular velocity (Peters et al., 2003). Therefore, angular displacement (see Equation 1) was normalized to a range of -1 to +1 and angular velocity (see Equation 2) was normalized by the absolute maximum (Hamill et al., 1999; Li et al., 1999; Miller et al., 2008; Stergiou et al., 2001; Wang et al., 2009; Yen et al., 2009). Both angular displacement and angular velocity serve as the basis for a phase plot where the x-axis represents the angular displacement and the y-axis corresponds with the angular velocity of the relevant joint. For all of the 100 data points, a

phase angle was calculated that was defined as the angle between the connecting line of the data point and the origin of the coordinate system and the positive x-axis.

Equation 1: $\theta_i = \frac{2 * [\theta_i - \min(\theta)]}{\max(\theta) - \min(\theta)}$ with θ = joint angle, i = data point

Equation 2: $\omega_i = \frac{\omega_i}{\max[\max(\omega), \max(-\omega)]}$ with ω = joint angle velocity; i = data point

Diverse methods to quantify the angle can be found in previous studies (Burgess-Limerick et al., 1993; Dierks and Davis, 2007; Hamill et al., 1999; Kurz and Stergiou, 2002; Li et al., 1999; Scholz, 1990; van Emmerik and Wagenaar, 1996). We decided to quantify the phase angles by a range of [0°, 180°] as shown by Hamill et al. (1999) to avoid possible discontinuities which can appear at the transition from quadrant 2 (180°) to quadrant 3 (-180°). Figure 1 shows the phase angle definition (A) and a phase plot of $KNEE_{flex/ex}$ with two examples of how to calculate the corresponding phase angles (B). The difference between the two relevant phase angles forms the CRP, as the phase angle of the proximal joint is subtracted from the distal phase angle (Kurz and Stergiou, 2002). Finally, the possible range of CRP [-180°, 180°] is halved to a range from [0°, 180°]. Due to the presented calculation methods and the bisection of the CRP range, which has no influence on calculating CRP variability, circular statistic methods can be avoided, since data are no longer directional and linear statistics can be used.

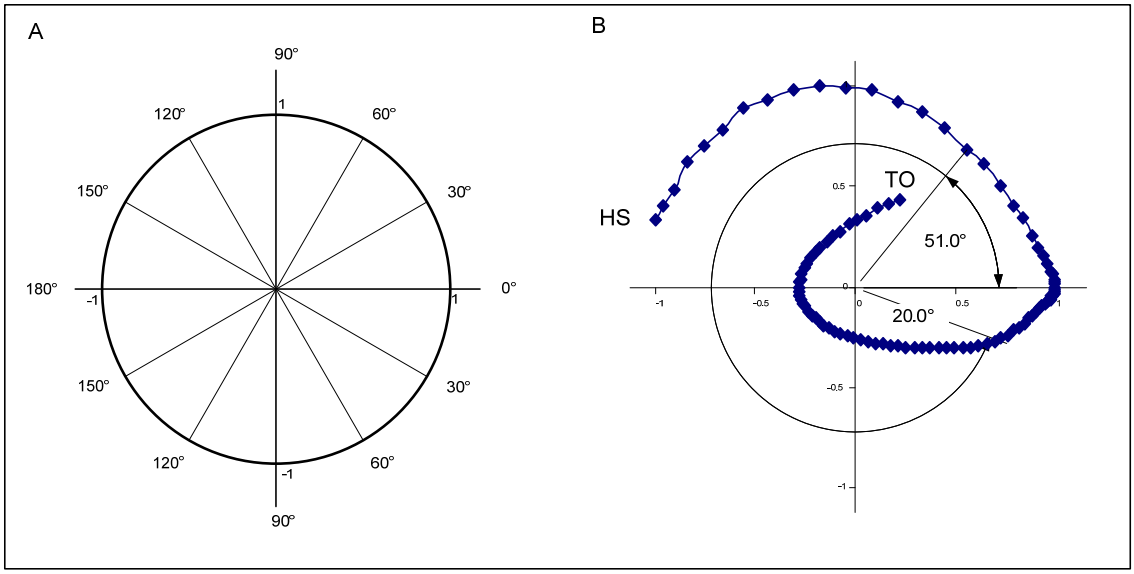

Figure 1: A) The phase plot angle definition with a range from 0° to 180°. B) Two examples of calculating phase plot angles for knee flexion/extension. TO represents toe-off and HS stands for heel strike.

The calculation of CRP variability is based on the CRP of five individual randomly selected stance phases for which standard deviations were calculated on a point-by-point basis for each percent of the ROP. Thus, an individual continuous standard deviation was calculated that represents CRP variability and is displayed in three different ways for this study. These three methods have been applied in previous studies (Dierks and Davis, 2007; Hamill et al., 1999; Miller et al., 2008), and since we are interested in an applicable technique to present and analyse data, all representations were included. First, the calculated individual CRP variability was averaged across the complete stance phase for each subject and then compared between the two groups. Second, the stance phase was divided into four stance phase intervals (SPI) and CRP variability of each subject was averaged for each SPI. According to J. Perry (1992), SPI1 combines initial stance and loading response and is defined as the first twenty percent of stance phase. SPI2 (midstance) starts at 21% ROP and continues up to 50% ROP, followed by SPI3 that lasts up to 80% ROP and corresponds with terminal stance. The last twenty percent of the stance phase (pre swing) is defined as SPI4. Third, CRP variability is displayed continuously for CO and ITBS over the whole stance phase by averaging the individual continuous standard deviations for CO and for ITBS on a point-by-point basis.

To investigate whether CRP variability provides additional information about differences or similarities between CO and ITBS, we calculated the following four couplings: HIPflex/ex – KNEEflex/ex, HIPabd/ad – KNEEflex/ex, KNEEflex/ex – ANKLEflex/ex and KNEEflex/ex – $ANKLE_{ev/in}$. Please note that due to a plausibility check prior to the calculations of Grau et al. (2010) one healthy runner had to be excluded from further calculations, since an unrealistic value for the maximum inversion velocity of the ankle was detected. Thus, both groups consisted of 18 runners for HIPflex/ex – KNEEflex/ex, HIPabd/ad – KNEEflex/ex and KNEEflex/ex – ANKLEflex/ex, and 17 runners for KNEEflex/ex – $ANKLE_{ev/in}$.

2.4 Statistical Analysis

Statistical procedures were conducted using the software JMP 7.0 (SAS Institute Inc, NC, USA; 2007). To analyse whether there are differences in means between the two groups of runners (CO vs. ITBS), the averaged CRP variability over the entire stance phase and over several stance phase intervals allowed the implementation of t-tests for independent samples to compare the averaged CRP variability of the two groups. To conduct these independent t-tests we proposed the null hypothesis that there are no differences in CRP variability between the healthy and the symptomatic group of runners. Significant differences were reported for $p<0.05$ (**), tendencies towards one group were reported for $p<0.1$ (*).

3. Results

Group means of the individually averaged entire stance phases, 95% confidence intervals and resultant p-values of an independent t-test for CO and ITBS are graphically presented in Figure 2 (left side). CO and ITBS show almost equal amounts of CRP variability for all couplings. Slightly higher CRP variability was noticed for HIPabd/add – $KNEE_{flex/ex}$ in ITBS (Figure 2B; p=0.19), but without any statistical significance or tendency. The highest group averages for CRP variability were also found for HIPabd/add – $KNEE_{flex/ex}$.

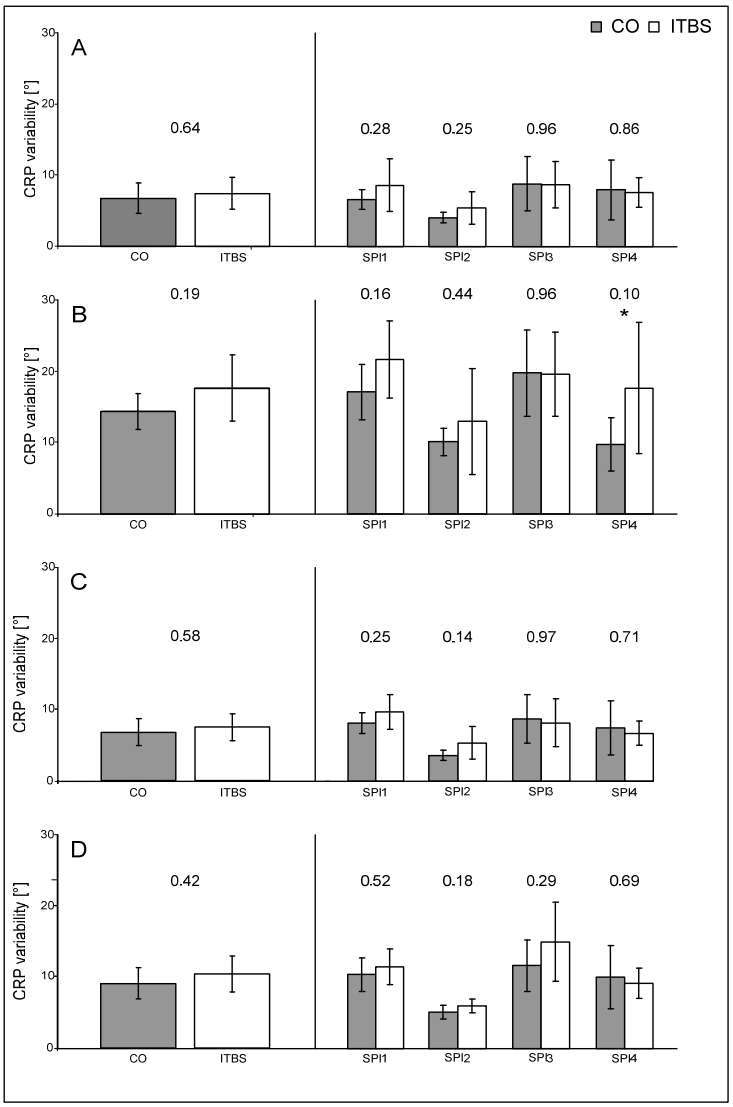

Figure 2: Continuous relative phase (CRP) variability for the healthy group of runners (CO, gray) and the injured group of runners suffering from ITBS (white). 95% confidence intervals and p-values are also presented. CRP is averaged over the complete stance phase on the left side. CRP is averaged according to the four defined stance phase intervals (SPI) according to Perry (1992) (SPI1 = 1-20% ROP, SPI2 = 21-50% ROP, SPI3 = 51-80% ROP, SPI4 = 81-100% ROP) on the right side. A) HIPflex/ex – KNEEflex/ex, B) HIPabd/ad – KNEEflex/ex, C) KNEEflex/ex – ANKLEflex/ex and D) KNEEflex/ex – $ANKLE_{ev/in}$.

Averaged CRP variability for the four defined stance phase intervals, their 95% confidence intervals and p-values of the conducted t-tests for independent samples are shown in Figure 2 (right side). Here, similar overall patterns for all coupling pairs were detected: CRP variability in SPI1 was consistently higher than in SPI2. SPI3 showed an increase in CRP variability, whereas SPI4 was associated with a decrease in CRP variability.

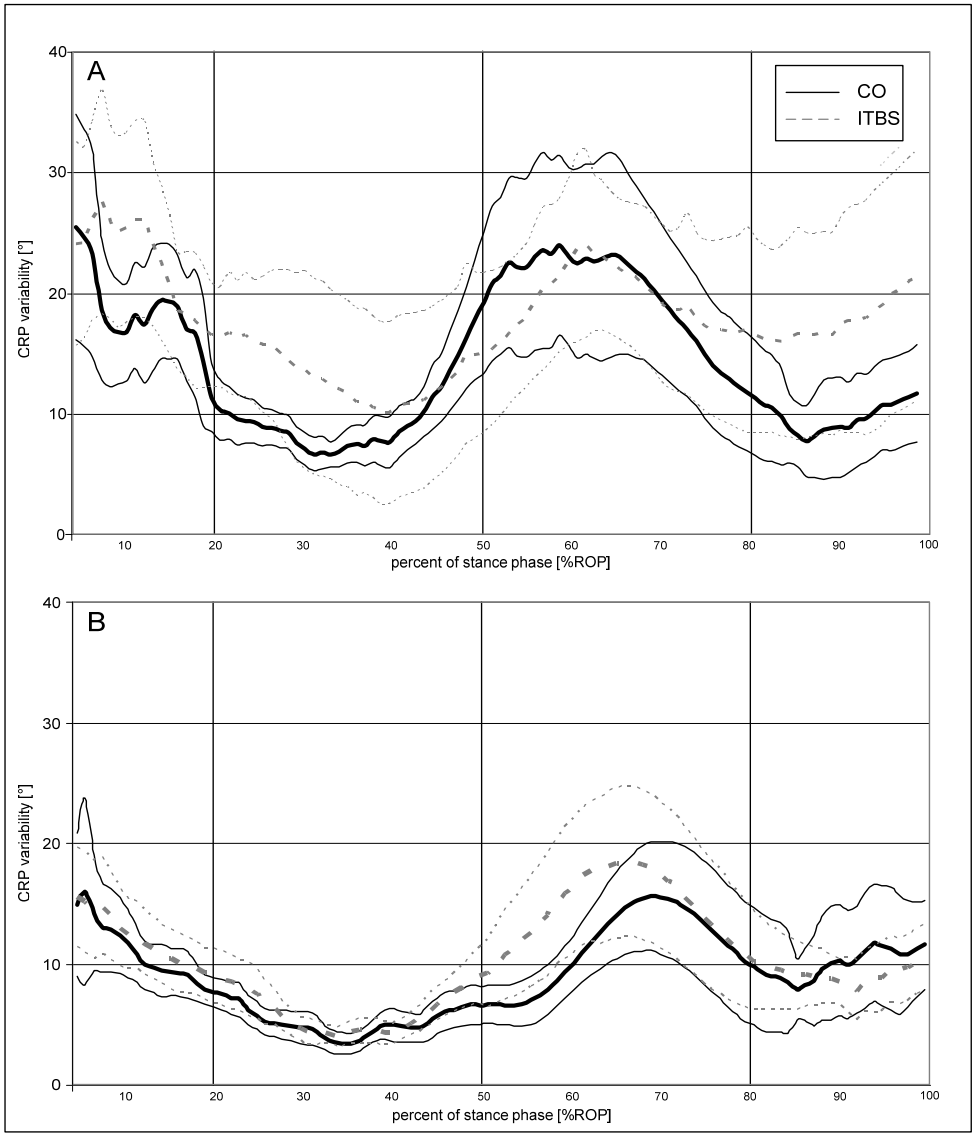

Figure 3: Averaged continuous relative phase variability for the healthy control group (CO: black, solid) and for the injured group of runners suffering from ITBS (gray, dashed) normalized to a 100% of stance phase: A) HIPabd/ad – KNEEflex/ex and B) KNEEflex/ex – $ANKLE_{ev/in.}$ The vertical lines divide stance phase into four stance phase intervals according to J. Perry (1992). 95% confidence intervals are also included for CO (black, solid) and for ITBS (gray, dashed).

Comparing the differences between CO and ITBS during the first half of stance phase (SPI1 and SPI2), CRP variability tended to be increased for ITBS for all coupling pairs. Again, no statistically significant differences between the two groups were found. During stance phase interval 3, the two coupling pairs that include hip motion (2A and 2B) as a coupling partner showed an almost equal amount of CRP variability in addition to a more variable CRP for HIPabd/ad – KNEEflex/ex in SPI4 for the symptomatic runners (Figure 2B, p=0.1).

Continuous variability of CRP and the corresponding confidence intervals of two coupling pairs are displayed in Figure 3. Note that KNEEflex/ex – $ANKLE_{ev/in}$ (B) is representative of HIPflex/ex – KNEEflex/ex and for KNEEflex/ex – ANKLEflex/ex since all three graphs show similar trends and the levels of confidence overlap throughout the entire stance phase. CRP variability decreased after heel-strike which lasted until 30% ROP. Afterwards it increased until its maximum was reached in the third stance phase interval. Another decrease of CRP variability, with its minimum occurring at about 85% ROP, was followed by a final increase during terminal stance phase until its peak was reached at toe-off. Further, similar trends were found for HIPflex/ex – KNEEflex/ex, KNEEflex/ex – ANKLEflex/ex and KNEEflex/ex – $ANKLE_{ev/in}$ (Figure 3B) throughout the entire stance phase with higher CRP variability and an earlier increase in variability for ITBS in the second half of the stance phase.

In contrast to the other three couplings, the control group showed an earlier increase to a higher CRP variability for HIPabd/ad – KNEEflex/ex (Figure 3A). However, the symptomatic group of runners was characterized by a slightly more variable CRP in the fourth stance phase interval. Significant differences were also not detected, as the width of the confidence band was larger for ITBS. In summary, differences in the graphs of continuous CRP variability were noticeable but not statistically significant.

4. Discussion

The purpose of this study was to decide whether or not variability of continuous relative phase provides additional information about differences or similarities between healthy and injured runners. We hypothesized that there are differences between healthy and injured states and that the healthy controls would demonstrate a higher CRP variability. Since significant differences between symptomatic and asymptomatic runners were found in several kinematic variables of the used data (Grau et al., 2010), CRP variability was implemented to examine whether this method confirms these findings in a way that differentiates between CO and ITBS. Another aim was to decide whether CRP variability is

applicable for further biomechanical research by solving possible difficulties in calculating and interpreting CRP variability adequately.

When examining CRP variability averaged over the entire stance phase (Figure 2, left side), equal amounts of CRP variability were detected for three of the four joint coordination patterns. Minimally higher variability for the symptomatic runners was noticed for HIPabd/ad – KNEEflex/ex (Figure 2B). Despite the missing statistical significance, a connection between ITBS and an increased CRP variability was observed. Hamill et al. (1999) showed a general connection between a less variable CRP and pathological states. However, it is important to notice that this connection found by Hamill et al. (1999) is based on the variability during the swing phase. In fact, they detected greater variability for symptomatic runners during stance phase, which agrees with our findings, in part.

The calculated averages over the stance phase shown in Figure 2, which are accompanied by high between-subject variability, and the findings by Miller et al. (2008) demonstrate that a single value representing variability of the entire stride, swing or stance phase does not lead to consistent results. Consequently, we propose that dividing stance phase into several intervals would lead to additional temporal information, which in turn would lead to more consistent results. This is illustrated by our results for ITBS which showed consistently more variable coordination patterns during the first half of the stance phase in all couplings, but without any significant differences between the two groups.

It is difficult to draw a direct comparison between the presented results and the results of other studies (Hamill et al., 1999; Dierks & Davis, 2007) since they divided their stance phase according to rearfoot motion. However, the consistently higher CRP variability for ITBS during SPI1 and SPI2 corresponds with the results from Hamill et al. (1999), as pathological runners suffering from PFP also demonstrated higher variability in the first two intervals of the stance phase. The tendencies towards a greater CRP variability of ITBS for HIPabd/ad – KNEEflex/ex (2B) in SPI4 ($p=0.1$) demonstrate that at least some temporal aspects of stance phase should be maintained to allow a better handling of CRP variability, since these differences between CO and ITBS were not found when the entire stance phase is averaged for each group. Further, the presented results support the findings by Grau et al. (2010), since the reported differences in frontal hip motion were also detected by CRP variability. This supports our hypothesis that there is a connection between the quantity of differences in discrete kinematic variables and an increase in CRP variability.

Comparing averaged CRP variability over temporal periods does not lead to satisfying results, since distinct differences between the two groups cannot be identified. Subsequently, continuous graphs of CRP variability (Figure 3) are helpful to analyse data more accurately. The continuous graphs show that symptomatic runners exhibited a more variable CRP pattern for HIPabd/ad – KNEEflex/ex (Figure 3A) during three of the four stance phase intervals. Furthermore, during the second stance phase interval CRP variability was higher for ITBS, but the corresponding p-value p=0.44 (Figure 2B) does not reflect this difference. Another example is during the third stance phase for KNEEflex/ex – $ANKLE_{ev/I}$ (Figure 3B), where an earlier increase in CRP variability was detected for the symptomatic runners, but with no statistical significance or tendency (p=0.29), as shown in Figure 2D. The authors are aware that the confidence bands of the presented graphs still overlap and statistical evidence is missing, but a tendency towards an increased CRP variability for the injured runners can be observed.

As seen in Figure 3, the entire stance phase is described by 100 data points, whereas heel strike still corresponds with the first data point. The observed increases in variability at the beginning and at the end of the continuous graphs coincide with the initial and terminal stance phases. Kelso (1984) and Hamill et al. (1999) have both described this phenomenon of high variable transition periods. The great variability during the initial stance phase may be the consequence of the emerging impact forces which the human system has to compensate. This adaptability of the system requires an essential amount of flexibility and variability of the relevant joints and their movement coordination to cope with external influences. The stability deficits during terminal stance phase may be associated with the loss of ground contact and the synchronous application of the required forces for the final push off phase. After heel strike, variability of CRP decreases and reaches a minimum at about 20 to 30 percents of stance phase. The higher stability of the system is simultaneously accompanied by the foot flat phase as defined by De Cock (2005). During the foot flat phase, from about 20 to 55 percent of stance phase when the foot has complete contact with the ground, all couplings demonstrate a plateau of minimal variability. The following increase in CRP variability can be associated with the synchronous lifting of the heel, which disturbs the entire system. In addition, the reversals of the involved joint movements may also influence the stability of the system.

Additionally, the main problem in using CRP and CRP variability is the impossibility to establish a relationship between CRP and time series data, since it is remains unclear how the normalization procedures of time series data affect the resultant CRP and its variability. Normalization of the time series data reduced the effects of the signals' frequencies and

amplitudes so that the interpretation of CRP and its variability is restricted to an analysis of the phase plane portraits and their relationship (Peters et al., 2003). It is therefore undetermined why CRP variability of the injured group increased earlier and reached its maximum earlier than the controls for HIPflex/ex – KNEEflex/ex , KNEEflex/ex – ANKLEflex/ex, and KNEEflex/ex – $ANKLE_{ev/in}$ (3B). In contrast the greater variability during terminal stance phase in HIPabd/ad – KNEEflex/ex for ITBS (Figure 3A) may be a result of the significantly reduced range of frontal hip motion (Grau et al, 2010), since angular displacement was normalized to a range of -1 to +1 in the calculation procedures. This highly variable final stance phase interval for HIPabd/ad – KNEEflex/ex suggests that the injured runners each respond differently to cope with the injury, which would explain the higher within-group standard deviation.

In summary, the depiction of the continuous variability of CRP (Figure 3) shows additional information, such as the earlier increase around midstance which could not be detected in the averaged graphs of Figure 2. Since these periods do not coincide with the four predefined stance phase intervals, statistical significance for existing differences between healthy and injured runners was not found. We implemented the different types of CRP variability presentation to decide which was the most reasonable for our purpose and also allowed a statement about possible differences or similarities between healthy and injured runners. To interpret data adequately and to obtain a better understanding of the flexibility of joint coordination, we recommend that CRP variability should at least be visualized using averages over several stance phase intervals, or, more precisely, using continuous graphs.

Although the calculation of averages always implicates the disadvantage of lost temporal information, a division of stance phase into several periods is conceivable to maintain some temporal aspects. An advantage of calculating averages, however, is the output of discrete numbers compared to continuous graphs and thus the possibility of carrying out simple statistical tests, such as a t-test for independent samples. Our results show that the most effective method for analysing CRP variability data is the presentation of continuous graphs throughout the entire stance phase. However, a compromise, such as calculating averages for different stance phase intervals, would simplify the analysis and the handling of CRP variability data. Therefore, we suggest that a division of the stance phase by temporal characteristics as shown by De Cock et al. (2005) may lead to a more practicable and comprehensible way to implement CRP variability.

5. Conclusion

Based on the presented results, we do not consider CRP variability to be a solid method for discriminating between healthy and injured runners, since differences which exist in discrete kinematic variables were not detected by CRP variability. High within-group variations were evident, but most likely were consequences of the small populations the calculations were based on. These problems should be solved by increasing the number of runners for both groups. It is well known that CRP variability is difficult to access, since its calculation is complex and its interpretation is not intuitive. This paper has presented a possible connection between CRP variability and time-series data which enables the interpretation of CRP variability. We think it is necessary to apply this method in future biomechanical research to determine whether CRP variability is valid and can be used as an analytical method or not.

Runners suffering from ITBS in this study did not demonstrate any significant differences in CRP variability compared to the healthy controls, so that we cannot be sure if there is a relationship between injury and increased CRP variability. Further, our hypothesis of less variable joint coordination for injured runners could not be proven, but it was noticeable that higher CRP variability was associated with the injured group of runners during some periods of the stance phase. This relationship has never been mentioned before in previous studies. It is also worth mentioning that the quantity of differences in discrete variables seems to influence the magnitude of CRP variability.

A prospective study may also be helpful to decide whether CRP variability increases when runners develop an injury, or whether CRP variability is initially greater in healthy individuals who later develop injuries. Further, a distinct conclusion about a positive or negative relationship between high CRP variability and injury should be established, and it should be determined, if possible, how much variability is normal in healthy individuals.

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5.2. Prospective analysis of intrinsic and extrinsic risk factors on the development of Achilles tendon pain in runners.

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Abstract

There are currently no generally accepted, consistent results which clearly characterize factors causing Achilles tendon pain (AT) in runners. Therefore, we carried out a prospective study to evaluate the multifactorial influence of clinical, biomechanical (isometric strength measurements and 3D-kinematics) and training-related risk factors on the development of AT. 269 uninjured runners were recruited and underwent an initial examination. 142 subjects completed their participation by submitting training information on a weekly basis over a maximal period of one year. 45 subjects developed an overuse injury, with ten runners suffering from AT.

In an uninjured state, AT runners already demonstrated decreased knee flexor strength and abnormal lower leg kinematics (sagittal knee and ankle joint) compared with a matched control group. A relationship between years of running experience or previous overuse injuries and the development of new symptoms could not be established.

The interrelationship of biomechanical and training-specific variables on the generation of AT is evident. A combination of alterations in lower leg kinematics and higher impacts caused by fast training sessions might lead to excessive stress on the Achilles tendon during weight bearing and thus to AT in recreational runners.

Keywords: Overuse injury, Achilles tendon pain, running, prospective, clinical data, 3D-kinematics, isometric strength measurement, training, multifactorial

Introduction

Running has become increasingly popular over the last decades and by association the amount of runners suffering from overuse injuries has also risen. Epidemiologic studies show that 19% - 80% of all runners develop an overuse injury every year (Hreljac, 2005; van Gent et al., 2007) whereby 5% - 34% generate Achilles tendon pain or Achilles tendinopathy (Clement et al., 1984; Haglund-Akerlind and Eriksson, 1993; Mahieu et al., 2006). Hence, the Achilles tendon is one of the most prevalent sites for overuse injuries for both recreational and elite runners, leading to training reductions or rest. Determining potential risk factors for developing overuse injuries, not only Achilles tendon pain, in runners is of major interest for biomechanical research and is the subject of daily clinical routines.

Numerous studies and reviews have been published investigating intrinsic and extrinsic risk factors for developing AT to understand the multifactorial mechanisms causing these symptoms. In particular, runners suffering from AT have limitations in ankle joint motions and a poor flexibility in the gastrocnemius/soleus complex resulting from regular training (Clement et al., 1984; Haglund-Akerlind and Eriksson, 1993; Kaufman et al., 1999; Kvist M., 1991). According to Smart et al. (1980) and Clement et al. (1984), the unsuccessful compensation of decreased ankle flexibility by additional knee flexion leads to greater pronation which, again, may cause a whipping action of the Achilles tendon generating microtears in the tendon and finally cause AT. Lower muscular strength or muscular imbalances are also cited in reviews as potential risk factors for AT (Alfredson and Lorentzon, 2000; Paavola et al., 2002). This assumption is mostly based on data showing that strengthening calf muscles leads to a quicker rehabilitation and earlier return to training. Abnormalities in movement patterns have been a topic of discussions about risk factors for AT since the 1980s. The findings by Smart et al. (1980) and Clement et al. (1984), who defined increased pronation as a major risk factor for AT, are supported by more recent studies (Donoghue et al., 2008; McCrory et al., 1999; Ryan et al., 2009). Over the last decades, training or training errors have also been considered to be potential risk factors for developing AT, e.g. excessive training distance, changes in training routines, increases in training intensity, faster training pace, running surface and terrain, and footwear to name a few (Clement et al., 1984; Kannus, 1997; Murphy et al., 2003; van Gent et al., 2007).

The development of AT seems to be multifactorial, with influences of clinical, biomechanical and training-specific variables. One main reason for the lack of success in defining evidence-based risk factors is the retrospective design the majority of the described studies are based on. Clarifying cause and effect is not possible using a retrospective study design and

interpreting obtained results is hardly possible (Almekinders and Temple, 1998). Low measurement accuracy based on goniometers or two-dimensional high speed video systems in order to analyse rearfoot and ankle kinematics might be another reason. Further, the lack of a control group (Clement et al., 1984; Smart et al., 1980) or the comparison of injured runners with a non-matched control group (Ryan et al., 2009) do not allow distinct causes for the development of AT to be identified. No direct link between a distinct training parameter and the occurrence of AT can be found. Reasons for the diversity of results are studies that include and compare different populations of runners (novice, recreational or elite runners, military recruits, etc.) over various time periods using different approaches (supervised training program, non-influenced training) and inconsistent definitions of injury (Hoeberigs, 1992; Rolf, 1995).

Consequently, prospective studies are essential to define possible intrinsic and extrinsic risk factors or a combination of different factors for developing AT, and to clarify the principle of cause and effect (Bovens et al., 1989; Kader et al., 2002; Murphy et al., 2003; Paavola et al., 2002; Ryan et al., 2009). Van Ginckel et al. (2009) carried out one of three prospective studies on the generation of AT and show a more laterally shifted force distribution underneath the forefoot and a decreased forward progression of the centre of force for runners generating AT. Mahieu et al. (2006) name decreased strength of the plantar flexors and a greater dorsiflexion range of motion as predictors of AT. In contrast, Kaufman et al. (1999) describe restricted ankle dorsiflexion and increased hindfoot inversion as potential risk factors. Despite the prospective study design, the relevance of these findings needs to be questioned, since these studies carry out a supervised training program including either novice runners (van Ginckel et al., 2009) or military recruits (Kaufman et al., 1999; Mahieu et al., 2006) and therefore do not reflect the situation for experienced recreational runners.

There are currently no generally accepted and consistent results which clearly characterize factors causing AT, and a reduction of the incidence rate has not been achieved. Van Gent et al. (2007) state that future well-designed prospective studies, focusing on one distinct symptom, including clearly defined running populations and using a universal definition of running injury are required to achieve comparable results. Therefore, we carried out a prospective study including experienced recreational runners to evaluate the multifactorial influence of clinical, biomechanical and training-related risk factors on the development of AT.

Based on previous findings, three research hypotheses are proposed: (1) Runners who generate AT already show restricted sagittal ankle joint mobility in an uninjured state. (2)

Increased pronation and, as a consequence to coupling mechanisms, additional alterations in lower leg kinematics are found for AT runners in an uninjured state compared with healthy controls. (3) Excessive mileage and modifications in training programs favour the development of AT.

Material and Methods

Subjects

Healthy recreational runners were included in the prospective study starting with an initial examination (IE) which comprised standardized clinical examinations, biomechanical testing procedures (isometric strength measurements, 3D-kinematics) and a questionnaire about training behaviour and years of running experience. After the IE, every subject was urged to keep a weekly training diary over a period of 52 weeks with information about their individual training habits. In the event that a subject incurred a running-related overuse injury, a second examination including the same clinical and biomechanical testing procedures as in the IE with additional diagnostics was necessary. The following inclusion and exclusion criteria were defined: all runners needed to be between the age of 18 and 55 and had to have a minimum weekly running volume of 20km. If a runner suffered from any running-related injury or had visited a physical therapist during the last six months before their participation or wore orthopaedic insoles in their running shoes, participation in the study was not possible. This study complies with the declaration of Helsinki, and all subjects signed a written consent form approved by the university ethics committee prior to IE.

Two hundred sixty-nine uninjured runners were recruited and passed the initial examination. One hundred twenty-seven subjects (47%) had to be excluded from the study due to missing feedback, other injuries and personal or timing reasons which did not allow any further training. One hundred forty-two subjects (53%) completed their participation and handed in their training data on a regular weekly basis. Ninety-seven of the included runners remained uninjured and serve as controls (CO), 45 subjects (32%) developed an overuse injury with 10 runners suffering from Achilles tendon pain (AT). A detailed list is shown in Table 1.

Since literature shows a sex-related influence and an effect of anthropometric differences on the biomechanical results (Grau et al., 2008a; Krauss, 2006), the subjects of both groups were matched according to gender, BMI, height, weight and age. Consequently, two groups of ten runners including eight males and two females each (CO: mean BMI 23kg/m² (SD 2), mean height 177cm (5), mean weight 72kg (8), mean age 40years (7); AT: mean BMI

23kg/m² (3), mean height 177cm (4), mean weight 72kg (8), mean age 45years (5)) were included in the data analysis.

Table 1: Overview of all subjects who passed the initial examination

Subjects	**Number**	**Percentage**
Passed initial examination	269	100
Completed participation	142	53
Drop-outs	127	47
Completed participation	142	100
Uninjured runners	97	68
Injured runners	45	32
Injured runners	45	100
Achilles tendon pain	10	22
Plantarfasciitis	7	16
Patella tendinopathy	6	13
Iliotibial Band Syndrome	3	7
Shin Splints	3	7
Hip overall	4	9
Knee unknown / other	7	16
Foot unknown / other	5	11

Definition of overuse injury

A runner was classified as injured if medical attention was needed, more than 66% of all training sessions in two consecutive weeks or more than 50% of all training sessions in four consecutive weeks were accompanied by running-related pain and an overuse injury was diagnosed by the orthopaedic surgeon.

Experimental procedures

Clinical examination

All clinical examinations were carried out by an experienced orthopaedic surgeon and sports physician including the measurement of active and passive ranges of motion for hip, knee and ankle joints according to the neutral-zero method (Ryf and Weymann, 1995). All measurements were performed in a supine position and compared with standard values to determine whether joint mobility was normal, limited or excessive. The following standards for active ROM measurements were defined according to the neutral-zero method: hip

flexion (with flexed knee): 130°-140°; hip extension (Thomas Test): 10°-20°; hip abduction (knee extended): 50°-80°; hip adduction (knee extended): 20°-30°; hip internal rotation (knee flexed): 30°-40°; hip external rotation (knee flexed): 40°-50°; knee flexion: 120°-150°; knee extension: 0°-10°; ankle dorsiflexion (knee flexed): 10°-20°; ankle plantarflexion (knee flexed): 40°-50° (Ryf and Weymann, 1995). Passive standards were 5°-10° larger than the corresponding active values. The quantification of angular values was neglected since the reliability and comparability of ROM measurements is considered to be more critical (Roaas and Andersson, 1982). Therefore, the joint amplitudes were compared between both legs to discriminate between normal and abnormal range of motion (Boone and Azen, 1979; Roaas and Andersson, 1982; Ryf and Weymann, 1995). A difference of at least 10°-15° between sides was necessary for a definite classification into limited or increased mobility. The incidence of past operations and overuse injuries to the lower extremities was also documented.

Biomechanical measurements

a) Isometric strength measurements

The isometric strength measurements were carried out for the upper body and lower extremities according to a standardized testing protocol. This protocol is implemented in the daily clinical routine and has proved itself in practice over the last 15 years. During a short familiarization period, all subjects were allowed to get used to the direction of movement by performing the dynamic task against an increasing resistance. Following this, each subject had to perform two maximum isometric contractions at a standardized angle according to recent studies and its functional relevance (Johnson et al., 2004; Murray et al., 1980). All measurements were supervised by an experienced physiotherapist who determined whether the task was accomplished successfully by increasing the applied force slowly to a maximum without explosive maximal contractions. The maximal torque was documented.

The maximal isometric strength was assessed for the upper body by measuring the maximal isometric torque of the straight abdominal muscles, called “abdominal flexion” (AF) and the straight back muscles, called “back extension” (BE). For both measurements, subjects were fixated in a seated position with a knee flexion angle of 90° and not allowed to use their hands for stabilization. Assessing AF, the upper body was in an upright position (0°). For BE, the upper body was positioned at a 30° forward incline. Hip abduction and adduction was tested both bilaterally and unilaterally. Bilateral measurements were performed in a seated position (bHAB, bHAD) with a hip abduction angle of 30° (15° each leg). Unilateral measurements were conducted in a standing position (uHAB, uHAD) with a hip abduction angle of 20° for uHAB and uHAD. The realization of a functionally relevant hip abduction

angle of 15° as described by Johnson et al. (2004) was not possible due to device-specific limitations. Therefore, a hip abduction angle of 20° was chosen, which also enabled a comparison with the seated measurements. Knee flexion and extension (KFL, KEX) was tested unilaterally in a seated position with a knee flexion angle of 30° for KFL and 60° for KEX (Knapik et al., 1983; Overend et al., 1992). Performing the seated measurements, all subjects were fixated with an additional seatbelt and not allowed to self-stabilize during the measurement by using their hands. In the standing position, the subject's pelvis was fixated by individually adjustable flanking pads to enable a stable upright position for the testing procedures. The unilateral hip strength measurements were accomplished using the Hip Machine (FREI SWISS AG, Thalwil, ZH, Switzerland), whereas all other isometric strength measurements were performed using DAVID devices (David GmbH & CO KG, Neu-Ulm, Germany).

b) Three dimensional kinematics

All subjects ran barefoot with a controlled speed of 12km/h (SD 5%) on a 13m EVA foam runway in the laboratory. Sufficient time was allowed for the subjects to get used to the laboratory, running surface and speed, enabling an individual and natural running style. A minimum of 25 running trials was recorded for each subject using a six-camera infrared system (ViconPeak, MCam, M1, Oxford, UK) with a sampling frequency of 250Hz. The applied marker set consisted of 34 spherical markers according to ISB recommendations (Wu et al., 2002) marking pelvis (2xASIS, 2xPSIS) and both lower extremities, each consisting of three segments: thigh (greater trochanter, lateral and medial femoral epicondyle), shank (lateral and medial tibia plateau, tibial tuberosity, tibial crest, lateral and medial malleolus) and foot (lateral, medial and posterior calcaneus, metatarsals 1 and 5 and hallux).

Three dimensional joint motions were quantified by calculating Cardan angles according to Söderkvist and Wedin (1993) with the distal segment rotating with respect to the proximal segment. Here, the first rotation occurred around the sagittal axis (extension/flexion), followed by a rotation around the frontal axis (abduction/adduction or eversion/inversion) and lastly by a rotation around the transversal axis (internal/external rotation). Data analysis was restricted to the stance phase, which was detected according to Maiwald et al. (2009). Joint angle curves were time-normalized to 100 data points. Mean angular displacements and discrete variables were based on 10 valid trials and calculated for hip flexion/extension (HFL, HEX), hip abduction/adduction (HAB, HAD), knee flexion/extension (KFL, KEX), knee external/internal rotation (KER, KIR), ankle dorsi/plantarflexion (ADF, APF) and rearfoot inversion/eversion (RFINV, RFEV).

The discrete kinematic variables were:

- Initial joint excursion [°] at touchdown for hip flexion (HFL_{init}), hip abduction (HAB_{init}), knee flexion (KFL_{init}), knee external rotation (KER_{init}), ankle dorsiflexion (ADF_{init}) and rearfoot inversion ($RFINV_{init}$)
- Maximal joint excursion [°] and its timing [% ROP = roll-over process/stance phase] for hip flexion (HFL_{max}, t HFL_{max}), hip adduction (HAD_{max}, t HAD_{max}), knee flexion (KFL_{max}, t KFL_{max}), knee internal rotation (KIR_{max}, t KIR_{max}), ankle dorsiflexion (ADF_{max}, t ADF_{max}) and rearfoot eversion ($RFEV_{max}$, t $RFEV_{max}$)
- Maximal joint excursion [°] for hip extension (HEX_{max}), hip abduction (HAB_{max}), knee extension (KEX_{max}), knee external rotation (KER_{max}), ankle plantarflexion (APF_{max}) and rearfoot inversion ($RFINV_{max}$)
- Range of motions [°] for hip flexion and extension (HFL_{ROM}, HEX_{ROM}), hip adduction and abduction (HAD_{ROM}, HAB_{ROM}), knee flexion and extension (KFL_{ROM}, KEX_{ROM}), knee internal and external rotation (KIR_{ROM}, KER_{ROM}), ankle dorsiflexion and plantarflexion (ADF_{ROM}, APF_{ROM}) and rearfoot eversion and inversion ($RFEV_{ROM}$, $RFINV_{ROM}$)
- Maximal motion velocity [°/s] for hip flexion and extension (HFL_{velmax}, HEX_{velmax}), hip adduction and abduction (HAD_{velmax}, HAB_{velmax}), knee flexion and extension (KFL_{velmax}, KEX_{velmax}), internal and external knee rotation (KIR_{velmax}, KER_{velmax}), ankle dorsiflexion and plantarflexion (ADF_{velmax}, APF_{velmax}), and rearfoot eversion and inversion ($RFEV_{velmax}$, $RFINV_{velmax}$)

Training-specific variables

Individual training diaries were submitted on a weekly basis for a maximal period of 52 weeks and contained information about running frequency, distance, duration, type of training session (slow, medium, fast, interval or competition), running terrain (hard, medium or soft underground; even, medium or uneven surface), occurrence of running-related pain and its location as well as any additional exercising.

Statistical analysis

The analysis of clinical and biomechanical variables, except the maximal strength measurements of bilateral hip abduction and adduction, was conducted either for the injured leg of an injured runner or for a randomly selected leg of a non-injured runner. The randomization of legs was performed prior to the statistical analysis.

Because of the low number of subjects and high amount of variables, the current study design is an explorative evaluation of risk factors influencing the development of AT without any statistical tests. Instead, descriptive statistical methods, such as, means, standard

deviations, medians and 95% confidence intervals were included in data analysis. Data is graphically presented by box plots with 25th and 75th percentiles and whiskers extending to 1.5 interquartile range (IQR). Prior to the descriptive analysis, Pearson's correlation coefficients were computed to detect redundancies and to reduce the quantity of variables for presentation. Variables were pooled for r>0.6.

Results

Clinical examination

The clinical examination revealed no limited or excessive mobility of the hip, knee or ankle joints for any CO or AT runner, so that clinical data were not presented. Six of ten CO runners suffered from an overuse injury to the lower extremity in the past; one runner underwent an operation of the lower extremity. Two of ten AT runners suffered from an overuse injury in the past; three underwent an operation.

Biomechanical measurements

a) Isometric strength measurements

As high correlations exist between unilateral and bilateral measurements of hip joint surrounding muscles, unilateral measurements were excluded from the upcoming analysis. Hence, data evaluation includes AF, BE, bHAB, bHAD, KFL and KEX. Results are displayed in Table 2 and Figure 1.

Table 2: Isometric strength measurements of the control group (CO, n=10) and runners generating Achilles tendon pain (AT, n=10).

	AF [Nm]		**BE [Nm]**		**bHAB [Nm]**		**bHAD [Nm]**	
	CO	**AT**	**CO**	**AT**	**CO**	**AT**	**CO**	**AT**
Mean (SD)	133 (33)	116 (29)	215 (84)	242 (75)	216 (41)	224 (32)	314 (66)	318 (79)
Median	124	113	207	233	215	220	304	289
Up 95%CI	153	134	267	288	241	244	355	367
Low 95%CI	113	98	163	196	191	204	273	269

	KFL [Nm]		**KEX [Nm]**	
	CO	**AT**	**CO**	**AT**
Mean (SD)	149 (22)	124 (26)	201 (54)	193 (50)
Median	149	119	207	186
Up 95%CI	163	140	234	224
Low 95%CI	135	108	168	162

Upper body including abdominal flexion (AF) and back extension (BE). Lower extremity including bilateral hip abduction (bHAB) and adduction (bHAD), unilateral knee flexion (KFL) and extension (KEX). Displayed are means (and standard deviations, SD), medians and upper and lower limits of the 95% confidence interval (CI).

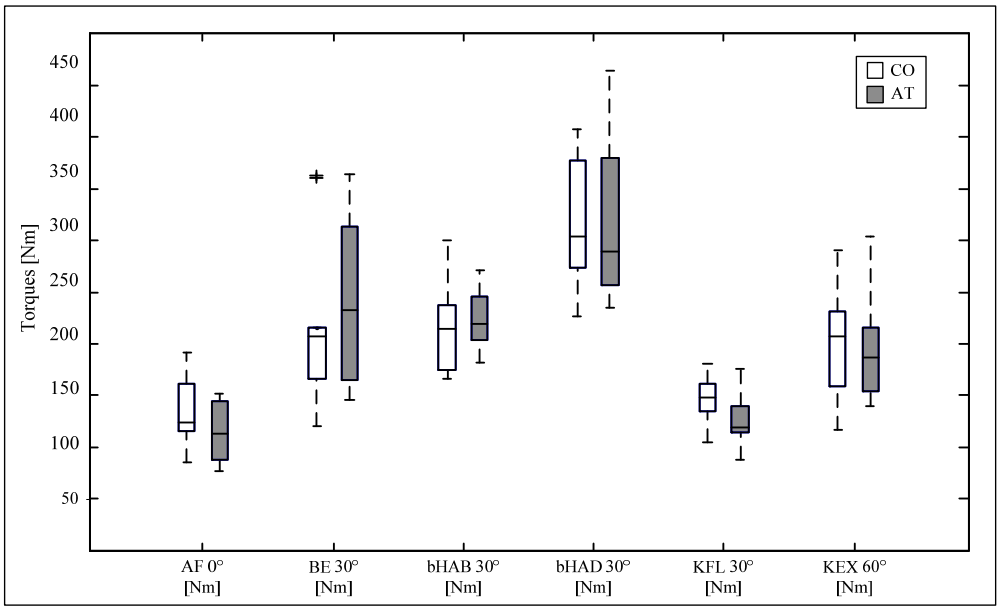

Figure 1: Isometric strength measurements of the control group (CO, n=10, white) and runners generating Achilles tendon pain (AT, n=10, gray).

Upper body including abdominal flexion (AF) and back extension (BE). Lower extremity including bilateral hip abduction (bHAB) and adduction (bHAD), unilateral knee flexion (KFL) and extension (KEX). Displayed are box plots with 25th and 75th percentiles, whiskers extending to 1.5 IQR as well as medians and outliers (marked by +).

Runners who developed AT already showed decreased knee flexor strength compared with CO in an uninjured state even though 95% confidence intervals slightly overlap. No differences in maximal isometric strength were found for the upper body, hip joint surrounding muscles or knee extensors between AT and CO.

b) Three dimensional kinematics

Please note that due to forefoot running one subject had to be excluded from the kinematic analysis. Measurement errors forced the omission of another subject for the evaluation of ankle and rearfoot motion. Therefore, the analysis of hip and knee kinematics contains nine subjects per group. The analysis of ankle and rearfoot motion is based on eight runners per group.

The presentation of hip joint and transversal knee joint kinematics was abandoned since there were no differences between CO and AT. Correlation coefficients greater than 0.6 were found for several discrete knee, ankle and rearfoot variables, so that 12 of 24 were included in data analysis: Sagittal knee motion: KFL_{max}, t KFL_{max}, KFL_{ROM}, KEX_{ROM}. Sagittal ankle motion: ADF_{max}, t ADF_{max}, ADF_{ROM}, APF_{ROM}. Frontal rearfoot motion: $RFEV_{max}$, $RFINV_{max}$, $RFEV_{ROM}$, $RFINV_{ROM}$. Kinematic results are shown in Table 3 and Figure 2.

Although variability in kinematic data is high, the authors intend to highlight some kinematic aspects. AT runners revealed a lower ADF_{max} and a greater $RFEV_{max}$ compared to CO. For sagittal knee joint motion, runners generating AT already showed a reduced KFL_{max} in an uninjured state. As maximal joint excursions correlate with initial joint angles, it can be concluded that AT also show a more extended knee joint, a lower dorsiflexed ankle joint and a more everted rearfoot at touchdown compared with CO. No differences in ranges of motion, timing values and maximal velocities for ankle, rearfoot and knee motions were found between the two groups of runners.

Table 3: Three dimensional kinematics of the control group (CO) and runners generating Achilles tendon pain (AT)

	KFL_{max} [°]		**t KFL_{max} [%ROP]**		**KFL_{ROM} [°]**		**KEX_{ROM} [°]**	
	CO	**AT**	**CO**	**AT**	**CO**	**AT**	**CO**	**AT**
Mean (SD)	41 (4)	37 (7)	35 (3)	34 (2)	26 (3)	26 (4)	25 (6)	25 (6)
Median	41	36	35	34	26	26	24	27
Up 95%CI	44	41	37	36	28	29	29	29
Low 95%CI	38	33	33	32	24	23	21	21
	ADF_{max} [°]		**t ADF_{max} [%ROP]**		**ADF_{ROM} [°]**		**APF_{ROM} [°]**	
	CO	**AT**	**CO**	**AT**	**CO**	**AT**	**CO**	**AT**
Mean (SD)	14 (5)	9 (3)	45 (1)	43 (4)	13 (2)	11 (2)	37 (5)	35 (3)
Median	15	8	45	43	13	11	36	35
Up 95%CI	18	11	46	45	14	13	40	37
Low 95%CI	10	7	44	41	12	9	34	33
	$RFEV_{max}$ [°]		**$RFINV_{max}$ [°]**		**$RFEV_{ROM}$ [°]**		**$RFINV_{ROM}$ [°]**	
	CO	**AT**	**CO**	**AT**	**CO**	**AT**	**CO**	**AT**
Mean (SD)	-3 (4)	-5 (3)	5 (5)	4 (3)	8 (2)	8 (3)	8 (6)	9 (1)
Median	-3	-4	3	5	8	8	8	9
Up 95%CI	-1	-3	9	6	9	10	12	10
Low 95%CI	-5	-7	1	2	7	6	4	8

Note: Selected variables for sagittal knee motion: maximal knee flexion (KFL_{max}), its timing (t KFL_{max}), knee flexion range of motion (KFL_{ROM}) and knee extension range of motion (KEX_{ROM}). Selected variables for sagittal ankle motion: maximal ankle dorsiflexion (ADF_{max}), its timing (t ADF_{max}), ankle dorsiflexion range of motion (ADF_{ROM}) and ankle plantarflexion range of motion (APF_{ROM}). Selected variables for frontal rearfoot motion: maximal rearfoot eversion ($RFEV_{max}$), maximal rearfoot inversion ($RFINV_{max}$), rearfoot eversion range of motion ($RFEV_{ROM}$) and rearfoot inversion range of motion ($RFINV_{ROM}$). Sagittal knee motion (CO: n=9, AT: n=9), sagittal ankle motion (CO: n=8, AT: n=8) and frontal rearfoot motion (CO: n=8, AT: n=8). Displayed are means (and standard deviations, SD), medians and upper and lower limits of the 95% confidence interval (CI).

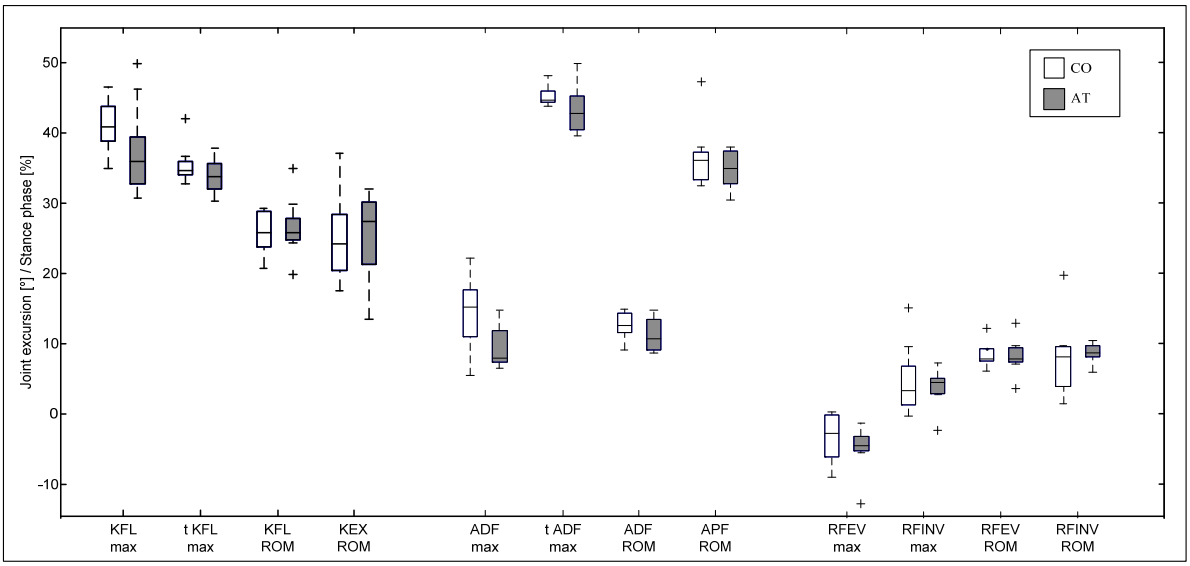

Figure 2: Three dimensional kinematics of the control group (CO, white) and runners generating Achilles tendon pain (AT, gray).

Sagittal knee motion (CO: n=9, AT: n=9), sagittal ankle motion (CO: n=8, AT: n=8) and frontal rearfoot motion (CO: n=8, AT: n=8). Displayed are box plots with 25th and 75th percentiles, whiskers extending to 1.5 IQR as well as medians and outliers (marked by +). Note: Selected variables for sagittal knee motion: maximal knee flexion (KFL_{max}), its timing (t KFL_{max}), knee flexion range of motion (KFL_{ROM}) and knee extension range of motion (KEX_{ROM}). Selected variables for sagittal ankle motion: maximal ankle dorsiflexion (ADF_{max}), its timing (t ADF_{max}), ankle dorsiflexion range of motion (ADF_{ROM}) and ankle plantarflexion range of motion (APF_{ROM}). Selected variables for frontal rearfoot motion: maximal rearfoot eversion ($RFEV_{max}$), maximal rearfoot inversion ($RFINV_{max}$), rearfoot eversion range of motion ($RFEV_{ROM}$) and rearfoot inversion range of motion ($RFINV_{ROM}$)

Training-specific variables

After a correlation analysis, the evaluation of training data was reduced to the following variables: weekly running distance, additional weekly exercising, percentage distributions of slow, medium and fast training sessions (incl. fast endurance runs, interval training sessions and competitions) and the percentage distributions of hard, soft, even and uneven running terrain.

a) Group comparison

Comparing the averaged training variables (see Table 4 and Figure 3), AT and CO documented comparable training concepts with nearly equal amounts of weekly running distance, distributions of training sessions and chosen running terrain. High variability in averaged training data was found for both groups. Two AT runners recorded high amounts of additional exercising during their participation in the study. Both groups of runners had similar running experience of ten years (CO: 10 years (SD 10); AT 10 years (8)).

Table 4: Averaged training data for controls (CO, n=10) and runners generating Achilles tendon pain (AT, n=10) over their time of participation

	distance [km/week]		TS slow [%]		TS medium [%]		TS fast [%]	
	CO	**AT**	**CO**	**AT**	**CO**	**AT**	**CO**	**AT**
Mean (SD)	32 (20)	33 (15)	30 (17)	42 (21)	47 (15)	43 (23)	22 (13)	16 (11)
Median	29	28	29	35	48	44	26	13
Up 95%CI	44	42	41	55	56	57	30	23
Low 95%CI	20	24	19	29	38	29	14	9

	UG hard [%]		UG soft [%]		UG uneven [%]		UG even [%]		add. exercise [min]	
	CO	**AT**	**CO**	**AT**	**CO**	**AT**	**CO**	**AT**	**CO**	**AT**
Mean (SD)	75 (17)	71 (24)	13 (12)	15 (24)	29 (20)	27 (26)	59 (19)	59 (24)	110 (77)	228 (314)
Median	77	76	9	3	31	20	59	48	113	94
Up 95%CI	85	86	20	30	42	43	71	74	158	423
Low 95%CI	65	56	6	0	16	11	47	44	62	33

Displayed are means (and standard deviations, SD), medians and upper and lower limits of the 95% confidence interval (CI). Please note: TS = training session, UG = underground.

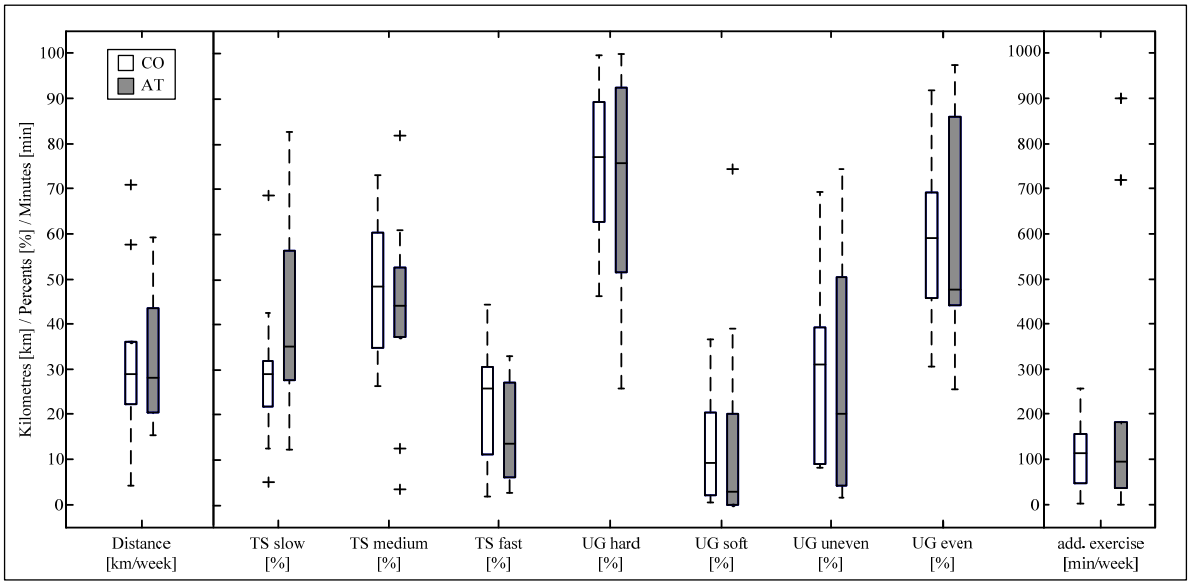

Figure 3: Averaged training data of controls (CO, n=10, white) and runners generating Achilles tendon pain (AT, n=10, gray) over their time of participation.

Displayed are box plots with 25th and 75th percentiles, whiskers extending to 1.5 IQR as well as medians and outliers (marked by +). Please note: TS = training session, UG = underground.

b) Prospective training data

The prospective analysis of ATs' training data is displayed in Table 5 and Figure 4. Here, two subjects were excluded from data analysis since injury occurred in week 4 and week 5 of their participation, respectively. Consequently, the prospective analysis of training data was based on eight runners.

Subjects generating AT reported a slight shift from slow to fast training sessions and an increase in additional exercising throughout the last four weeks prior to injury. Any modifications in weekly mileage and comparable percentage distributions of training sessions and choice of running surface were documented. Again, there was a high variability in all recorded training variables.

Table 5: Prospective training data of runners generating Achilles tendon pain (AT, n=8). Comparison of training data between a period of four weeks before the onset of Achilles tendon pain (last 4 weeks, L4w) and the rest of their participation (Rest).

	distance [km/week]		TS slow [%]		TS medium [%]		TS fast [%]	
	Rest	**L4w**	**Rest**	**L4w**	**Rest**	**L4w**	**Rest**	**L4w**
Mean (SD)	35 (15)	34 (26)	42 (20)	36 (32)	47 (22)	45 (31)	11 (11)	19 (22)
Median	37	39	39	25	51	36	9	10
Up 95%CI	45	52	56	60	62	68	19	36
Low 95%CI	25	16	28	12	32	22	3	2

	UG hard [%]		UG soft [%]		UG uneven [%]		UG even [%]		add. exercise [min]	
	Rest	**L4w**	**Rest**	**L4w**	**Rest**	**L4w**	**Rest**	**L4w**	**Rest**	**L4w**
Mean (SD)	64 (28)	71 (24)	21 (30)	15 (27)	55 (28)	59 (25)	30 (33)	27 (27)	60 (64)	153 (161)
Median	66	67	9	0	48	58	16	17	46	102
Up 95%CI	83	89	42	35	74	78	53	47	104	264
Low 95%CI	45	53	0	-5	36	40	7	7	16	42

Displayed are means (and standard deviations, SD), medians and upper and lower limits of the 95% confidence interval (CI). Please note: TS = training session, UG = underground.

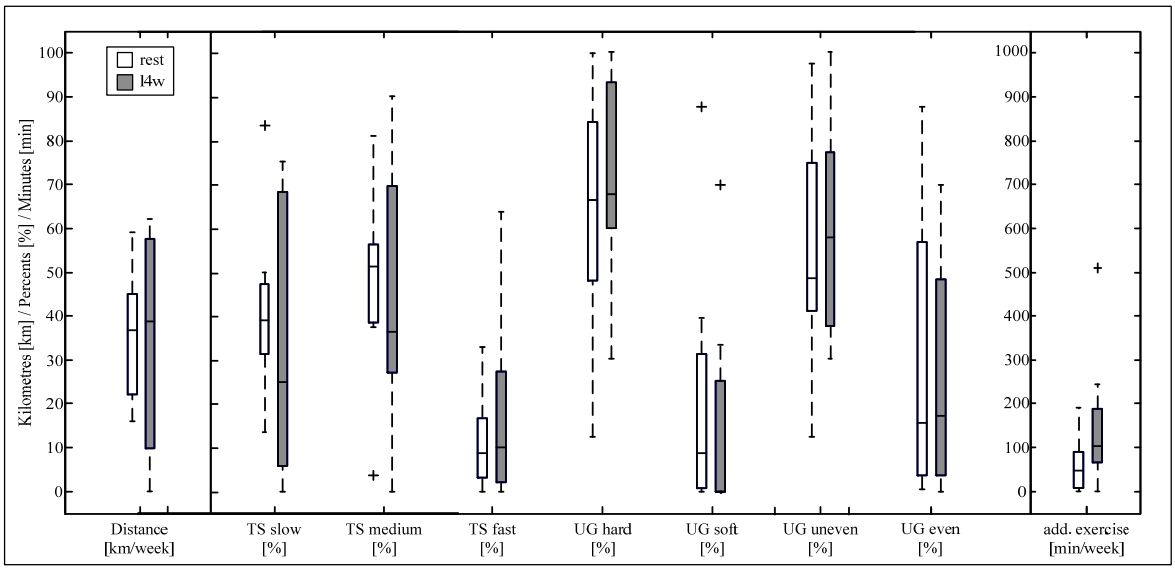

Figure 4: Prospective training data of runners generating Achilles tendon pain (AT, n=8). Comparison of training data between a period of four weeks before the onset of Achilles tendon pain (Last 4 weeks, l4w, gray) and the rest of their participation (rest, white).

Displayed are box plots with 25th and 75th percentiles, whiskers extending to 1.5 IQR as well as medians and outliers (marked by +). Please note: TS = training session, UG = underground.

Discussion

Clinical examination

Clinical examination of the subjects did not find any abnormalities in the ranges of motion for hip, knee or ankle joints in all three planes of motion between the two groups of runners. These findings comply in part with recent studies since hypo- or hypermobility of the hip and knee joints have never been associated with Achilles tendon pain in runners. In contrast, decreased ankle mobility is named as potential risk factor for developing AT by several studies (Haglund-Akerlind and Eriksson, 1993; Kaufman et al., 1999; Kvist M., 1991). This relationship cannot be supported by the results of the current study.

There are three possible reasons for these contradicting results. First, goniometer-based quantitative measurements to assess joint mobility were not carried out in our study. However, subjective estimations were minimized by carrying out measurements with an experienced orthopaedic surgeon and by dividing mobility into three categories (restricted, normal or increased) according to the neutral-zero method (Boone and Azen, 1979; Roaas and Andersson, 1982; Ryf and Weymann, 1995). In addition, a bilateral comparison was conducted to enable a clinically useful and relevant approach to judge restricted or increased joint mobility. The findings of recent studies (Haglund-Akerlind and Eriksson, 1993; Kaufman et al., 1999; Mahieu et al. 2006) have to be considered as over-interpreted since differences between injured and uninjured runners are too small according to the neutral-zero method. Second, most studies are based on a retrospective approach which does not enable the clarification of cause-effect relationships. For example, reduced ankle dorsiflexion might either be the result of tight calf muscles (Haglund-Akerlind and Eriksson, 1993) or be the reason for higher loads on the Achilles tendon (Cook et al., 2002). Therefore, prospective approaches as carried out by Mahieu et al. (2006) and Kaufman et al. (1999) appear to be the appropriate method to determine potential risk factors, not only for clinical parameters. The studies by Mahieu et al. (2006) and Kaufman et al. (1999), however, demonstrate a third limitation, since they include military or naval recruits with no previous running experience. As only experienced recreational runners are included in the current study, a comparison of results seems to be inappropriate.

The findings of the current study do not demonstrate a connection between previous overuse injuries and the development of a new symptomatic as presented in a review by van Gent et al. (2007). The authors speculate that a past overuse injury implies a learning effect leading to a sensible and sophisticated training design to minimize the risk of generating an overuse

injury. A possible influence of gender, age or weight cannot be evaluated in the current study, since AT runners were matched with healthy controls.

Biomechanical measurements

a) Isometric strength measurements

The authors consider the decreased knee flexor strength shown by runners developing AT to be of great importance. The high variability of data (see Figure 1) is a consequence of the diversity of included subjects, since both groups consisted of runners of both sexes with different weights and ages. A normalization of torque according to body weight is omitted since both groups were matched according to BMI and body weight prior to data evaluation. A potential influence of weak knee flexor muscles on developing Achilles tendon pain will be discussed in combination with lower leg kinematics in the next section.

There are currently no studies which implement isometric strength measurements of the upper body, hip or knee joint surrounding muscle groups to investigate the development of AT. Therefore, we have nothing to compare our results to. In contrast, a connection between weak calf muscles and the generation of Achilles tendon pain has been demonstrated in several studies. Haglund-Akerlind and Eriksson (1993), as well as Mahieu et al. (2006), both measured the muscular strength of the gastrocnemius/soleus complex or calf muscles using a dynamometer for isokinetic concentric and/or eccentric measurements. Although their study is based on a retrospective design, Haglund-Akerlind and Eriksson (1993) consider reduced eccentric torques of the gastrocnemius/soleus complex as a possible reason for the development of Achilles tendon pain. Mahieu et al. (2006) found that decreased strength of the plantar flexors leads to the genesis of Achilles tendon pain in a cohort consisting of military recruits. Consequently, the authors admit that measuring the maximal strength of the gastrocnemius/soleus complex using isometric, isokinetic concentric or eccentric measurements would have been a great benefit to their study.

b) Three-dimensional kinematics

Based on the presented kinematic data, a conclusive statement about whether pronation influences the development of Achilles tendon pain in runners as shown in previous studies (Donoghue et al., 2008; McCrory et al., 1999; Ryan et al., 2009) is not possible. Sample sizes are too small and variability of data is too high. The assumption that increased pronation results in a “whipping action” of the Achilles tendon and finally leads to Achilles tendon pain (Clement et al., 1984; Smart et al., 1980) still seems logical and needs to be part of future studies.

In the current study, runners developing AT already revealed small alterations in the sagittal upper ankle joint and in sagittal knee motion in a non-injured state compared to CO. These abnormalities occur especially during the first half of the stance phase until the foot flat phase (De Cock et al., 2005) is terminated. Despite the problems of sample size and data variability, the authors intend to outline two possible mechanisms leading to more tension on the Achilles tendon and finally to Achilles tendon pain.

First, decreased knee flexor strength might be a reason for alterations in lower leg kinematics, since a more extended knee joint is necessary to compensate insufficient stabilization of the knee joint. The stabilization is usually ensured by the knee flexor muscles, especially when the joint flexes during the weight bearing stage. An increased extension of the knee, reaching its maximum at about 35% stance phase, induces more tension on the gastrocnemius/soleus complex. The higher stress is transferred directly to the Achilles tendon as it merges into the Achilles tendon and inserts at the calcaneus. Due to the origin at the medial border of the tibia, a more extended soleus muscle might lead to higher torsion on the Achilles tendon. To relieve this excessive stress on the Achilles tendon, the runners tend to ease the distal tension by decreasing ankle dorsiflexion during the weight bearing stage of running.

A second approach may lead to the conclusion that weak knee flexor muscles might be a result of abnormal lower leg kinematics. In detail, stress on the Achilles tendon is initiated by a higher eversion of the rearfoot, which has been demonstrated in previous studies (Donoghue et al., 2008; McCrory et al., 1999; Ryan et al., 2009) and can be seen in part in our population of AT runners. Higher pronation causes a greater internal rotation of the tibia due to coupling mechanisms as shown by Tiberio (1987) and Bellchamber & van den Bogert (2000) and finally induces a tensed gastrocnemius/soleus complex. Increased gastrocnemius/soleus tension results in a more extended knee joint during running due to its insertion at the femoral epicondyles. Thus, the Achilles tendon is exposed to excessive stress that the runners compensate with reduced dorsiflexion of the upper ankle joint. As a consequence, the knee flexor group might not be innervated as usual for stabilizing the flexed knee joint and subsequently degrades. Based on the presented results, the importance of multifactorial study concepts for the future is apparent. Alterations in movement patterns appear to influence the generation of overuse injuries so that analyses of lower extremity kinematics need to be part of future study designs.

The authors are aware that overuse injuries usually occur running shod and outdoors, and that data from barefoot running in a laboratory setup is difficult to compare to data from shod running outside the laboratory. On the one hand, shod running leads to adaptation processes like a flatter touchdown of the foot. On the other hand, marker placement on individual running shoes does not enable an appropriate measurement of rearfoot and ankle kinematics. Therefore, a compromise needs to be found and the authors decided to ensure equal conditions for every subject in the study and to eliminate the influence of different shoe constructions on lower leg kinematics.

Training-specific variables

a) Group comparison

A relationship between the individual running experience and the generation of Achilles tendon pain cannot be established in our population since both groups consisted of runners with an average training age of 10years. In contrast, recent studies and reviews (Macera et al., 1989; Marti et al., 1988; van Gent et al., 2007; Walter et al., 1989) describe less experienced runners as more exposed to the danger of developing an overuse injury than experienced runners.

According to the results of the current study, training errors do not appear to influence the development of AT, since averaged training data did not differ between CO and AT. Several studies use questionnaires to assess individual training concepts leading to different results about training parameters causing AT (Clement et al., 1984; Haglund-Akerlind and Eriksson, 1993; Kvist M., 1991; McCrory et al., 1999). McCrory et al. (1999) show that runners suffering from AT ran a significantly higher training pace than controls and did not regularly implement stretching habits in their training routines. According to Clement et al. (1984), overtraining, in general, is described as a major risk factor leading to the development of AT, including increases in weekly running distance, single running sessions, increases in intensity, running hilly profiles, returning from training breaks or a combination of these factors. In 1993, Haglund-Akerlind & Eriksson described a longer weekly training distance as a major risk factor for Achilles tendon pain.

The evaluation of averaged training data, either documented on a weekly basis or assessed by questionnaires, does not seem to be appropriate. One-year training periods are often characterized by training breaks, reductions and increases of training intensity so that a prospective analysis of training is essential. As high variability of averaged training data within both groups was detected, the evaluation of individual training concepts is indispensable. In the authors' opinions, identifying training-related risk factors for a small group of runners can only be accomplished by examining individual data. For example, two

subjects who develop AT document excessive amounts of additional exercising during their time of participation, displayed as outliers in Figure 3. These subjects generated Achilles tendon pain in week 4 and 5 during their participation. Hence, excessive additional exercising has to be considered a potential risk factor for generating Achilles tendon pain. The necessity of increasing the study sample in the future is obligatory to define injury-specific training errors using cluster analyses or other statistical methods.

b) Prospective data

The current study is the first prospective study investigating the development of Achilles tendon pain in experienced recreational runners without a supervised training program. Despite the high variability in training data, the authors consider modifications in training concepts as one major risk factor contributing to the development of AT. Conclusive connections cannot be established, but changes in individual training concepts and higher variability in the percentage distributions during the last four weeks prior to the onset of AT were observed. For example, a shift from slow endurance runs to fast training sessions and competitions might lead to higher impacts on the structures of the musculoskeletal system and thus generate Achilles tendon pain. Further, additional exercising, such as soccer or hiking, might also increase eccentric stress on the tendon and consequently affect the development of Achilles tendon pain.

Therefore, the prospective analysis of non-manipulated training programs is a promising method to determine training-related risk factors and should be part of future studies. Supervised training programs which increase exercise volume prior to injury do not lead to individual training-related risk factors (Kaufman et al., 1999; Mahieu et al., 2006; van Ginckel et al., 2009).

Summary

The current study clearly demonstrates the necessity for further complex prospective studies of intrinsic and extrinsic risk factors on the development of overuse injuries in runners. It also reveals several difficulties in carrying out such a prospective study. The subjective measurements of joint mobility and the absence of isokinetic-eccentric maximal strength measurements show that compromises in experimental procedures are necessary. The feasibility of the study in terms of carrying out diverse measurements using adequate techniques within a reasonable period of time is essential to recruit and to include subjects in the study. Additional difficulties such as high drop-out rates of almost 50% complicate the

realization of prospective studies. Further, the occurrence of other overuse injuries results in small sample sizes and slow down the process of defining injury-specific risk factors.

The authors are aware of the small sample size, compromises in experimental procedures and the missing statistical tests, but believe that first insights in possible multifactorial mechanisms favouring the development of Achilles tendon pain in recreational runners have been gained. With reference to the initially proposed research hypotheses, Hypothesis 1 cannot be confirmed since no restrictions in mobility of hip, knee or ankle joint were measured. Hypothesis 2 can be partly confirmed. Runners generating Achilles tendon pain did not show excessive pronation in an uninjured state but demonstrated altered lower leg kinematics, especially in the sagittal planes of motion of the knee and ankle joints. Hypothesis 3 can either be confirmed or rejected. An increase in faster training sessions and resulting higher impacts on the musculoskeletal system seem to be a potential risk factor for Achilles tendon pain but an individual evaluation of training concepts might lead to a better understanding of the relationship between training and injury. Further, additional exercising should also be taken into account as possible reasons for the development of overuse injuries in recreational runners without supervised training programs.
Finally, we feel there is a clear interrelationship between clinical, biomechanical and training-specific variables and the development of Achilles tendon pain. We speculate that a combination of alterations in lower leg kinematics and higher impacts caused by fast training sessions lead to excessive stress on the Achilles tendon during weight bearing and finally to microtears in the tendon. The role of weak knee flexor muscles remains unclear, since they might be a cause or effect of abnormal lower leg kinematics.

Perspective

The increase of the study's population will be the most important step over the next years to enlarge the sample size of controls and injured runners and to determine evidence-based and injury-specific risk factors. A comparison of risk factors between the uninjured and injured state will also be essential to clarify the principle of cause and effect, not only for Achilles tendon pain. And finally, a decision will be made about whether retrospective approaches might be sufficient for future studies to investigate possible interactions of risk factors leading to injury.

The authors encourage other researchers not only to focus on one specific risk factor when carrying out future studies, but to become aware of the interrelationship of intrinsic and extrinsic risk factors on the development of overuse injuries. Thus, the realization of complex

study designs is difficult but might be the only way to reveal the interactions of risk factors contributing to injury and to develop preventive measures against overuse symptomatic.

From a clinical point of view, it seems inevitable not only to include frontal, but also a sagittal perspective to examine hip, knee and ankle joint motions in two-dimensional clinical gait analyses to determine potential risk factors and to prevent the generation of overuse injuries, in general. Furthermore, balanced knee joint-surrounding muscles (flexor/extensor ratio) stabilizing the ankle joint to minimize internal rotation of the tibia during running is essential for preventing and treating Achilles tendon pain in runners.

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5.3. Are prospective studies necessary to determine kinematic risk factors for the development of overuse injuries in runners?

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Abstract

Prospective studies are considered the golden standard to establish cause and effect relationships and to define risk factors leading to overuse injuries (OI) in runners. To determine whether injury influences the mechanics of the lower extremity, the purpose of the current prospective investigation was to compare kinematic variables of runners between an uninjured and an injured state.

142 of 269 included runners (53%) completed their participation whereas 127 runners (47%) had to be disqualified from the study. 97 runners remained injury-free and 45 runners (32%) generated an OI during their time of participation. Kinematic data of 19 runners was collected in a healthy and in an injured state.

In the current group runners, differences between the healthy (M1) and injured (M2) state could not be detected for hip, knee, ankle or rearfoot kinematics. Thus, it can be speculated that lower leg kinematics might cause the development of injury and remain the same after the onset of injury.

If the results of the current study can be confirmed by future prospective investigations based on a larger number of subjects, controlled retrospective studies may enable the determination of injury-specific and evidence-based kinematic risk factors for the development of OI in runners.

Keywords: 3D-kinematics, prospective, running, risk factors, overuse injury

1. Introduction

Studies about risk factors causing overuse injuries (OI) in runners have been the topic of biomechanical research over the last decades. Despite the high number of retrospective studies and reviews, the identification of evidence-based risk factors leading to OI, such as training errors (Nielsen et al., 2012) or kinematic variables, has been unsuccessful (Barton et al., 2009; Grau et al., 2007; Grau et al., 2008b). So far, it has only been proven that the development of OI in runners is multi-factorial and influenced by clinical, biomechanical and training-specific variables (Ferber et al., 2009; Hreljac, 2005; Wen, 2007). Here, differences in study populations (novice vs. recreational vs. elite runners), study designs, definitions of injury and analyzed variables can be named as possible reasons for a missing decrease of the incidence rate in runners, which still varies between 11 and 85% (Nielsen et al., 2012; Rolf, 1995). Several reviews demand the application of prospective studies to clarify the principle of cause and effect and to determine distinct factors for the generation of OIs in runners (Barton et al., 2009; Murphy et al., 2003; van der Worp et al., 2011; van Gent et al., 2007; Wen, 2007). However, realizing prospective studies has been described as challenging with respect to the recruitment of runners and follow-up measurements (Noehren et al., 2007). Further, Hein et al. (2013) and Stefanyshyn et al. (2001) describe high drop-out ratios with nearly 50 percents, so that building a study sample including at least 20 runners per injury and a sufficient amount of controls to enable multivariate statistics appears to be almost impossible.

The question arises whether there is another option to determine ascertained causes for the development of OIs. Barton et al. (2009) demand prospective analyses for kinematic data of hip, knee and ankle/rearfoot to clarify a cause and effect relationship. In 2007, Noehren et al. reported consistent kinematic data from a retrospective and a prospective study showing an increased hip abduction and increased knee internal rotation for runners suffering from ITBS (Noehren et al., 2007; Noehren et al., 2006). They conclude that if the mechanics of the lower extremity do not alter as a consequence of acute injury, retrospective analyses might be sufficient for future investigations. This conclusion is based on kinematic measurements conducted with 18 female runners in a non-injured state for the prospective study and 35 female runners who suffered from ITBS years ago for the retrospective study. Both groups of runners were compared with matched control groups according to age and mileage. However, for the prospective study a second measurement during the acute phase of injury was not carried out so that the absence of injury-induced adaption processes is not proven. Further, no literature has been found carrying out a comparison of biomechanical variables of runners in an injured as well as in an uninjured state. This information is essential to judge

the sense and purpose of prospective studies and to decide whether findings based on retrospective studies are sufficient to determine distinct risk factors and mechanisms for the development of overuse injuries.

In summary, prospective studies are considered the golden standard to establish cause and effect relationships and to define risk factors leading to overuse injuries in runners. Since their realization is extremely difficult and retrospective studies might lead to the same results as prospective studies, the continuation of prospective studies appears to be questionable. To determine whether injury influences the mechanics of the lower extremity, the purpose of the current prospective investigation was to compare kinematic variables of runners between an uninjured and an injured state. Hence, the null hypothesis of the current study is that no differences in biomechanical variables exist between an uninjured and an injured state. Based on the findings, a recommendation will be made as to whether prospective studies should be continued in the future.

2. Methods

2.1 Subjects and study protocol

Healthy recreational runners were recruited for the prospective study if they met the following inclusion criteria: age between 18 and 55 years, a minimum weekly mileage of 20km, no OI or physical therapy for six months prior to the study and no orthopaedic insoles in their running shoes. Each subject underwent an initial examination (IE) including a clinical examination, biomechanical measurements consisting of three-dimensional kinematics and maximal isometric strength, and completed a questionnaire about their individual training habits. Following the IE, all subjects resumed their individual running programs and were encouraged to hand in weekly training logs over a maximal period of 52 weeks containing information about mileage, distance, frequency, intensity, additional exercising, footwear, route profile, running surface and occurring pain. If a runner sustained an OI during the time of participation, a second examination (SE) became necessary equivalent to the IE with additional diagnosis of the current symptoms. This study complies with the declaration of Helsinki, and all subjects signed a written consent form approved by the university ethics committee prior to IE.

142 of 269 included runners (53%) completed their participation and sent in their training logs as requested, whereas 127 runners (47%) had to be disqualified from the study due to missing feedback, other injuries, personal or timing reasons. 97 runners remained injury-free (controls, CO), 45 runners (32%) generated an OI during their time of participation, with 19

runners receiving an SE. A second examination could not be carried out for 26 runners due to timing reasons or pain during measurements. A complete list is displayed in Table 1.

19 runners underwent both examinations of the prospective study protocol and therefore serve as a basis for the comparison of kinematic variables between an uninjured and an injured state. This group of runners consists of 13 male and six female runners with a mean age of 42 years (SD 9), a mean BMI of 24 kg/m² (2), a mean height of 177 cm (8), and a mean weight of 74 kg (11).

Table 1: Overview of subjects who passed the initial (injury-free) and second (acute overuse injury) examination.

Subjects	**Initial examination [n]**	**Percentage [%]**	**2nd examination [n]**
Passed initial examination	269	100	
Completed participation	142	53	
Drop-outs	127	47	
Completed participation	142	100	
Uninjured runners (CO)	97	68	
Injured runners	45	32	
Injured runners	45	100	19
Achilles tendon pain	10	22	5
Plantar fasciitis	7	16	6
Patella tendinopathy	6	13	5
Iliotibial Band Syndrome	3	7	3
Shin Splints	3	7	
Hip overall	4	9	
Knee unknown / other	7	16	
Foot unknown / other	5	11	

2.2 Definition of injury

A runner was classified as injured if medical attention was needed, more than 66% of all training sessions in two consecutive weeks or more than 50% of all training sessions in four consecutive weeks were accompanied with running-related pain, and an overuse injury was diagnosed by the orthopaedic surgeon.

2.3. Experimental procedures

The experimental procedures of IE and SE were identical. A six-camera infrared system (ViconPeak, MCam, M1, Oxford, UK) with a sampling frequency of 250Hz was used to capture all subjects running barefoot with a controlled speed of 12 km/h (SD 5%). A 13m EVA foam runway and sufficient time to familiarize themselves with the laboratory prior to the measurements enabled a natural individual running style. Finally, a static trial and a minimum of 25 dynamic trials were recorded for each subject. Please note that during SE none of the runners felt any pain during the running tasks.

According to ISB recommendations (Wu et al., 2002), 34 spherical markers were applied to the subjects' pelvis (2xASIS, 2xPSIS), thighs (greater trochanter, lateral and medial femoral epicondyle), shanks (lateral and medial tibia plateau, tibial tuberosity, tibial crest, lateral and medial malleolus) and feet (lateral, medial posterior calcaneus, matatarsals 1 and 5 and hallux). The measurements of lower leg kinematics, including marker placement and surveillance of trials were performed by the same researcher.

Three dimensional joint motions were quantified by calculating Cardan angles and rotating the distal segment with respect to the proximal segment (Söderkvist and Wedin, 1993). The first rotation occurred around the sagittal axis (extension/flexion), followed by a rotation around the frontal axis (abduction/adduction or eversion/inversion), and lastly by a rotation around the transversal axis (internal/external rotation). For data analysis, the stance phase was computed according to Maiwald et al. (2009) and normalized to 100 data points. Discrete variables were calculated from each individual continuous joint curve and finally averaged over 10 valid trials for hip flexion/extension (HFL, HEX), hip abduction/adduction (HAB, HAD), knee flexion/extension (KFL, KEX), knee external/internal rotation (KER, KIR), ankle dorsi/plantarflexion (ADF, APF) and rearfoot inversion/eversion (RFINV, RFEV).

The following discrete kinematic variables were calculated:

- Initial joint excursion [°] at touchdown for hip flexion (HFL_{init}), hip adduction (HAD_{init}), knee flexion (KFL_{init}), knee external rotation (KER_{init}), ankle dorsiflexion (ADF_{init}) and rearfoot eversion ($RFINV_{init}$),
- Maximal joint excursion [°] and its timing [%ROP] for hip flexion (HFL_{max}, $tHFL_{max}$), hip adduction (HAD_{max}, $tHAD_{max}$), knee flexion (KFL_{max}, $tKFL_{max}$), knee internal rotation (KIR_{max}, $tKIR_{max}$), ankle dorsiflexion (ADF_{max}, $tADF_{max}$) and rearfoot eversion ($RFEV_{max}$, $tRFEV_{max}$)
- Maximal joint excursion [°] for hip extension (HEX_{max}), hip abduction (HAB_{max}), knee extension (KEX_{max}), knee external rotation (KER_{max}), ankle plantarflexion (APF_{max}) and rearfoot inversion ($RFINV_{max}$)

- Range of motions [°] for hip extension (HEX_{ROM}), hip adduction and abduction (HAD_{ROM}, HAB_{ROM}), knee flexion and extension (KFL_{ROM}, KEX_{ROM}), knee internal and external rotation (KIR_{ROM}, KER_{ROM}), ankle dorsiflexion and plantarflexion (ADF_{ROM}, APF_{ROM}) and rearfoot eversion and inversion ($RFEV_{ROM}$, $RFINV_{ROM}$)
- Maximal motion velocity [°/s] for hip extension (HEX_{velmax}), hip adduction and abduction (HAD_{velmax}, HAB_{velmax}), knee flexion and extension (KFL_{velmax}, KEX_{velmax}), internal and external knee rotation (KIR_{velmax}, KER_{velmax}), ankle dorsiflexion and plantarflexion (ADF_{velmax}, APF_{velmax}) and rearfoot eversion and inversion ($RFEV_{velmax}$, $RFINV_{velmax}$).

The calculation of range of motion and maximal velocity of hip flexion (HFL_{ROM} , HEX_{velmax}) was neglected (n.c. = not calculated) since some runners did not flex the hip joint at the beginning of the stance phase.

2.4 Statistical analysis

The analysis of lower leg kinematics was based on the affected leg of an injured runner.
Dependent t-tests were performed to analyze differences in lower leg kinematics between the non-injured (M1) and injured (M2) state. Since 40 discrete variables were included in the analysis, the Bonferroni correction was used to adjust the alpha level to $p<0.000025$ (=0.001/40).

Mean differences of discrete kinematic variables between the injured (M2) and uninjured (M1) state were calculated and tested against a hypothesized mean of 0. Again, alpha level was adjusted according to the Bonferroni method and set to $p<0.000025$. Additionally means, standard deviations, median and 95% confidence intervals are displayed.

3. Results

Please note that due to forefoot running one subject had to be excluded from the upcoming analysis. Further, three subjects were excluded due to a change of running pattern from rearfoot striking (IE) to forefoot striking (SE) during their time of participation. Consequently, the presented results are based on 15 subjects, all characterized as heel strikers.

In the current group of runners, differences between the healthy (M1) and injured (M2) state could not be detected for hip, knee, ankle or rearfoot kinematics. Group means are displayed in Table 2. Mean differences between M2 and M1 are listed in Table 3.

For joint excursions or ranges of motion, a maximal difference of 1.5 degree was found for KER_{max} and APF_{max}. A maximal shift in timing of 2.4 percent stance phase (%ROP = roll-over process) for $tKIR_{max}$, a decreased maximal motion velocity of 21°/s for KFL_{velmax}, and an increase of 23°/s for ADF_{velmax} were found as the greatest differences between M2 and M1. However, significant or relevant differences between the injured and uninjured state cannot be reported for any of the included discrete kinematic variables.

Table 2: Three dimensional lower leg kinematics of 15 runners before (M1) and after (M2) the onset of an overuse injury. Displayed are means and standard deviations. Alpha level is adjusted according to Bonferroni method and set to p<0.000025 (= 0.001 / 40).

	HFL_{init} [°]	**HFL_{max} [°]**	**t HFL_{max} [%ROP]**	**HEX_{max} [°]**	**HFL_{ROM} [°]**	**HEX_{ROM} [°]**	**HFL_{velmax} [°/s]**	**HEX_{velmax} [°/s]**
M1	32 (6)	32 (6)	15 (11)	-10 (5)	n.c.	41 (4)	n.c.	327 (59)
M2	31 (6)	31 (6)	16 (10)	-10 (4)	n.c.	40 (4)	n.c.	314 (46)
	HAD_{init} [°]	**HAD_{max} [°]**	**t HAD_{max} [%ROP]**	**HAB_{max} [°]**	**HAD_{ROM} [°]**	**HAB_{ROM} [°]**	**HAD_{velmax} [°/s]**	**HAB_{velmax} [°/s]**
M1	-8 (3)	-14 (3)	32 (4)	-2 (3)	6 (3)	12 (4)	138 (66)	120 (49)
M2	-8 (4)	-14 (3)	31 (5)	-3 (3)	7 (4)	11 (4)	151 (82)	115 (42)
	KFL_{init} [°]	**KFL_{max} [°]**	**t KFL_{max} [%ROP]**	**KEX_{max} [°]**	**KFL_{ROM} [°]**	**KEX_{ROM} [°]**	**KFL_{velmax} [°/s]**	**KEX_{velmax} [°/s]**
M1	11 (6)	36 (4)	35 (4)	10 (5)	26 (4)	27 (4)	464 (89)	291 (43)
M2	11 (5)	37 (5)	35 (3)	11 (5)	26 (3)	26 (4)	463 (84)	270 (39)
	KER_{init} [°]	**KIR_{max} [°]**	**t KIR_{max} [%ROP]**	**KER_{max} [°]**	**KIR_{ROM} [°]**	**KER_{ROM} [°]**	**KIR_{velmax} [°/s]**	**KER_{velmax} [°/s]**
M1	-1 (2)	-9 (3)	44 (12)	2 (3)	8 (2)	11 (4)	217 (49)	193 (49)
M2	-1 (2)	-8 (2)	41 (8)	4 (3)	8 (2)	12 (3)	226 (65)	202 (58)
	ADF_{init} [°]	**ADF_{max} [°]**	**t ADF_{max} [%ROP]**	**APF_{max} [°]**	**ADF_{ROM} [°]**	**APF_{ROM} [°]**	**ADF_{velmax} [°/s]**	**APF_{velmax} [°/s]**
M1	-3 (4)	9 (2)	44 (3)	-25 (4)	12 (3)	34 (4)	217 (58)	446 (68)
M2	-2 (5)	10 (3)	44 (4)	-24 (5)	13 (4)	34 (5)	240 (78)	451 (58)
	$RFINV_{init}$ [°]	**$RFEV_{max}$ [°]**	**t $RFEV_{max}$ [%ROP]**	**$RFINV_{max}$ [°]**	**$RFEV_{ROM}$ [°]**	**$RFINV_{ROM}$ [°]**	**$RFEV_{velmax}$ [°/s]**	**$RFINV_{velmax}$ [°/s]**
M1	4 (3)	-3 (2)	28 (8)	5 (3)	8 (3)	8 (3)	253 (87)	135 (45)
M2	4 (2)	-4 (2)	27 (11)	4 (2)	8 (2)	8 (3)	270 (92)	142 (45)

Note: Initial joint excursion [°] at touchdown for hip flexion (HFL_{init}), hip adduction (HAD_{init}), knee flexion (KFL_{init}), knee external rotation (KER_{init}), ankle dorsiflexion (ADF_{init}), and rearfoot inversion ($RFINV_{init}$). Maximal joint excursion [°] and its timing [%ROP] for hip flexion (HFL_{max}, t HFL_{max}), hip adduction (HAD_{max}, t HAD_{max}), knee flexion (KFL_{max}, t KFL_{max}), knee internal rotation (KIR_{max}, t KIR_{max}), ankle dorsiflexion (ADF_{max}, t ADF_{max}) and rearfoot eversion ($RFEV_{max}$, t $RFEV_{max}$). Maximal joint excursion [°] for hip extension (HEX_{max}), hip abduction (HAB_{max}), knee extension (KEX_{max}), knee external rotation (KER_{max}), ankle plantarflexion (APF_{max}) and rearfoot inversion ($RFINV_{max}$). Range of motions [°] for hip extension (HEX_{ROM}), hip adduction and abduction (HAD_{ROM}, HAB_{ROM}), knee flexion and extension (KFL_{ROM}, KEX_{ROM}), knee internal and external rotation (KIR_{ROM}, KER_{ROM}), ankle dorsiflexion and plantarflexion (ADF_{ROM}, APF_{ROM}), rearfoot eversion and inversion ($RFEV_{ROM}$, $RFIN_{ROM}$). Maximal motion velocity [°/s] for hip extension (HEX_{velmax}), hip adduction and abduction (HAD_{velmax}, HAB_{velmax}), knee flexion and extension (KFL_{velmax}, KEX_{velmax}), internal and external knee rotation ($KIR_{vel\ max}$, $KER_{vel\ max}$), ankle dorsiflexion and plantarflexion (ADF_{velmax}, APF_{velmax}) and rearfoot eversion and inversion ($RFEV_{velmax}$, $RFINV_{velmax}$). The calculation of range of motion and maximal velocity of hip flexion (HFL_{ROM} , $HEX_{vel\ max}$) was neglected (n.c. = not calculated) since some runners did not flex the hip joint at the beginning of the stance phase.

Table 3: Differences in lower leg kinematics between the uninjured (M1) and injured (M2) state of 15 runners. Displayed are mean differences (and standard deviations, SD), medians and upper and lower limits of the 95% confidence intervals (CI)

	HFL_{init} [°]	**HFL_{max} [°]**	**t HFL_{max} [%ROP]**	**HEX_{max} [°]**	**HFL_{ROM} [°]**	**HEX_{ROM} [°]**	**HFL_{velmax} [°/s]**	**HEX_{velmax} [°/s]**
Δ M2-M1 (SD)	-0.9 (2.2)	-1.1 (2.2)	1.3 (7.0)	0.0 (2.8)	n.c.	-1.1 (2.8)	n.c.	-14 (19)
Median	-0.4	-1.3	2.5	-0.8		-1.5		-9
Up 95% CI	0.2	0.0	4.8	1.4		0.3		-4
Low 95% CI	-2.0	-2.3	-2.2	-1.4		-2..5		-2..3
	HAD_{init} [°]	**HAD_{max} [°]**	**t HAD_{max} [%ROP]**	**HAB_{max} [°]**	**HAD_{ROM} [°]**	**HAB_{ROM} [°]**	**HAD_{velmax} [°/s]**	**HAB_{velmax} [°/s]**
Δ M2-M1 (SD)	0.2 (1.9)	0.0 (1.3)	-1.4 (2.4)	-0.3 (2.0)	0.3 (1.7)	-0.4 (2.4)	-13 (41)	-6 (26)
Median	-0.3	0.2	-1.7	-0.4	0.1	-0.8	-12	-7
Up 95% CI	1.2	0.6	-0.1	0.7	1.1	0.8	8	34
Low 95% CI	-0.8	-0.7	-2.6	-1.3	-0.6	-1.6	-34	-19
	KFL_{init} [°]	**KFL_{max} [°]**	**t KFL_{max} [%ROP]**	**KEX_{max} [°]**	**KFL_{ROM} [°]**	**KEX_{ROM} [°]**	**KFL_{velmax} [°/s]**	**KEX_{velmax} [°/s]**
Δ M2-M1 (SD)	0.4 (4.5)	0.3 (3.9)	-0.1(1.6)	1.5 (3.8)	-0.1 (2.2)	-1.2 (3.5)	-1 (40)	-21 (29)
Median	-0.4	0.1	-0.2	1.0	-0.1	-1.1	-5	-18
Up 95% CI	2.7	2.2	0.7	3.4	1.0	0.6	20	-7
Low 95% CI	-1.9	-1.7	-1.0	-0.5	-1.3	-2.9	-21	-36
	KER_{init} [°]	**KIR_{max} [°]**	**t KIR_{max} [%ROP]**	**KER_{max} [°]**	**KIR_{ROM} [°]**	**KER_{ROM} [°]**	**KIR_{velmax} [°/s]**	**KER_{velmax} [°/s]**
Δ M2-M1 (SD)	0.4 (4.5)	0.3 (3.0)	-2.4 (13.5)	1.5 (2.5)	0.0 (2.4)	1.2 (3.3)	9 (55)	9 (54)
Median	1.1	-0.6	-3.2	1.2	0.2	0.5	18	0
Up 95% CI	1.7	1.8	4.4	2.7	1.2	2.9	37	37
Low 95% CI	-1.6	-1.2	-9.3	0.2	-1.2	-0.5	-19	-18
	ADF_{init} [°]	**ADF_{max} [°]**	**t ADF_{max} [%ROP]**	**APF_{max} [°]**	**ADF_{ROM} [°]**	**APF_{ROM} [°]**	**ADF_{velmax} [°/s]**	**APF_{velmax} [°/s]**
Δ M2-M1 (SD)	0.5 (2.5)	1.2 (3.0)	0.6 (2.2)	1.5 (4.2)	0.6 (2.1)	-0.4 (6.1)	23 (47)	5 (87)
Median	1.3	0.9	0.3	1.0	0.2	-1.1	12	-17
Up 95% CI	1.8	2.7	1.7	3.7	1.6	2.7	46	49
Low 95% CI	-0.8	0.4	-0.5	-0.6	-0.5	-3.4	-1	-40
	$RFINV_{init}$ [°]	**$RFEV_{max}$ [°]**	**t $RFEV_{max}$ [%ROP]**	**$RFINV_{term}$ [°]**	**$RFEV_{ROM}$ [°]**	**$RFINV_{ROM}$ [°]**	**$RFEV_{velmax}$ [°/s]**	**$RFINV_{velmax}$ [°/s]**
Δ M2-M1 (SD)	-0.3 (2.4)	-0.3 (1.8)	1.2 (8.3)	-0.7 (2.5)	-0.1 (2.3)	-0.5 (2.1)	17 (86)	-3 (38)
Median	0.5	0.4	-0.6	-1.3	0.5	-0.3	9	-11
Up 95% CI	0.9	0.7	3.0	0.6	1.1	0.5	60	16
Low 95% CI	-1.4	-1.2	-5.3	-2.0	-1.2	-1.6	-27	-22

Note: Initial joint excursion [°] at touchdown for hip flexion (HFL_{init}), hip adduction (HAD_{init}), knee flexion (KFL_{init}), knee external rotation (KER_{init}), ankle dorsiflexion (ADF_{init}), and rearfoot inversion ($RFINV_{init}$). Maximal joint excursion [°] and its timing [%ROP] for hip flexion (HFL_{max}, t HFL_{max}), hip adduction (HAD_{max}, t HAD_{max}), knee flexion (KFL_{max}, t KFL_{max}), knee internal rotation (KIR_{max}, t KIR_{max}), ankle dorsiflexion (ADF_{max}, t ADF_{max}) and rearfoot eversion ($RFEV_{max}$, t $RFEV_{max}$). Maximal joint excursion [°] for hip extension (HEX_{max}), hip abduction (HAB_{max}), knee extension (KEX_{max}), knee external rotation (KER_{max}), ankle plantarflexion (APF_{max}) and rearfoot inversion ($RFINV_{max}$). Range of motions [°] for hip extension (HEX_{ROM}), hip adduction and abduction (HAD_{ROM}, HAB_{ROM}), knee flexion and extension (KFL_{ROM}, KEX_{ROM}), knee internal and external rotation (KIR_{ROM}, KER_{ROM}), ankle dorsiflexion and plantarflexion (ADF_{ROM}, APF_{ROM}), rearfoot eversion and inversion ($RFEV_{ROM}$, $RFIN_{ROM}$). Maximal motion velocity [°/s] for hip extension (HEX_{velmax}), hip adduction and abduction (HAD_{velmax}, HAB_{velmax}), knee flexion and extension (KFL_{velmax}, KEX_{velmax}), internal and external knee rotation ($KIR_{vel\ max}$, $KER_{vel\ max}$), ankle dorsiflexion and plantarflexion (ADF_{velmax}, APF_{velmax}) and rearfoot eversion and inversion ($RFEV_{velmax}$, $RFINV_{velmax}$). The calculation of range of motion and maximal velocity of hip flexion (HFL_{ROM} , $HEX_{vel\ max}$) was neglected (n.c. = not calculated) since some runners did not flex the hip joint at the beginning of the stance phase.

4. Discussion

Prospective study designs are assumed to identify distinct risk factors which can be associated with OI and eventually to clarify their cause-effect relationship (Barton et al., 2009; van Gent et al., 2007). As shown in a recent study by Hein et al. (2013), a major problem implementing a prospective study is the high drop-out rate of nearly 50%. Consequently, the investigation of subjects over a period of one year including additional examinations in case of injury is hardly possible. Therefore, the particular aim of the current analysis is to decide whether prospective approaches are necessary to determine kinematic risk factors leading to the development of OI. To accomplish this goal, kinematic data of 18 runners acquired prior to, and after the onset of an overuse injury were compared to determine whether an acute symptomatic leads to compensation or adaption processes and consequently to alterations in lower leg kinematics. All runners included in the study were injury-free for at least six months prior to the initial examination.

The current study demonstrates no differences in kinematic data between the healthy (M1) and injured state (M2) of one sample of runners. Since all runners did not mention experiencing any pain during the recording of lower leg kinematics, we presume that individual movement patterns were not intentionally influenced by the acute injury. Thus, it can be speculated that lower leg kinematics might cause the development of injury and remain the same after the onset of injury. These results confirm a statement by Noehren et al. (2007) who conclude that retrospective approaches might be adequate for defining kinematic risk factors for ITBS. Although their results are not based on one but on two different study samples, similar lower leg mechanics were found for retrospective and prospective kinematic data. However, since three subjects changed their running pattern during the current study, it cannot be ruled out that lower leg kinematics do not contribute to the development of OI in runners. Nevertheless, as seen by Hein et al. (2013), additional clinical and training-related risk factors, but also muscular strength deficits have to be taken into account to determine variables causing injury.

The authors emphasize that more research is needed to finally clarify whether retrospective analyses of kinematic data are sufficient to determine kinematic risk factors. If so, the focus of future retrospective studies should lie on the inclusion and examination of runners suffering from distinct overuse injuries which are medically verified by an experienced clinician or surgeon. We believe that evaluations based on injured runners suffering from diverse symptoms will not lead to a successful definition of evidence-based risk factors, since risk factors are thought to be injury-specific and differ between injuries. Further, large sample

sizes of acutely injured runners are also essential to clarify cause-effect relationships. However, the multifactorial composition of risk factors still has to be considered.

The authors are aware of several limitations of the current study referring to the applied methodology and statistics. Barefoot lower leg kinematics recorded on a soft underground in a laboratory setup do not reflect a typical outdoor situation the runners are used to and therefore have to be interpreted with care. It is well known that injuries usually occur running shod and outdoors on uneven surfaces and hilly terrain. Thus, three-dimensional analyses of shod running patterns might mirror a more realistic training situation. However, the placement of markers on the upper material of footwear contains, in turn, other disadvantages (Arnold and Bishop, 2013). An evaluation of knee and hip kinematics may nevertheless be possible. The low number of subjects and the simple statistic tests can be considered as an additional limitation of this study and will be addressed over the next years by increasing the number of participants.

5. Conclusion

The current study is the first evaluation of kinematic data acquired in an injury-free and an injured state from one population of runners. Based on the results of the current study it appears that running patterns do not alter due to an acute OI and therefore the initial null hypothesis can be confirmed. Thus, retrospective analyses may be appropriate to determine kinematic risk factors causing injury in runners as proposed by Noehren et al. (2007).

6. Perspective

If the results of the current study can be confirmed by future prospective injury-specific investigations based on a larger number of subjects, controlled retrospective studies may enable the determination of injury-specific and evidence-based kinematic risk factors for the development of OI in runners. Once kinematic risk factors have been clearly identified, preventive studies can be carried out to decrease the risk and consequently the incidence rate of overuse injuries in runners. Further, clinical gait analyses may also benefit from these results, since suitable therapeutic and rehabilitative measures can be introduced to the runner to shorten the time of injury and to return to training faster.

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6. Discussion

This doctoral thesis focuses on new aspects and methods to determine clinical, biomechanical and training-related risk factors leading to OI in runners. The presented approaches are also examined critically and placed into scientific context with regard to their feasibility and obtained findings. This chapter briefly summarizes and discusses the results of the three scientific papers comprising this thesis.

Scientific paper 1

“Using the variability of continuous relative phase as a measure to discriminate between healthy and injured runners.”

One might speculate that due to the lack of success in defining evidence-based kinematic risk factors for OI in runners over the last several decades of research, CRP and VCRP have increasingly shifted into the focus of biomechanical research to analyse lower leg motion patterns with regard to the development of injury. As described in SP1, CRP unites spatial and temporal information for two adjacent segments in order to enable the evaluation of joint coordination and movement patterns. However, since the calculation and interpretation of VCRP data is so complex, and neither increased VCRP nor decreased VCRP have been associated with injury, the primary objective of SP1 was to evaluate whether differences in common kinematic variables between injured and healthy female runners can also be detected in VCRP data.

Due to diverse calculation methods of CRP and VCRP in literature, it first became necessary to define an appropriate calculation algorithm. Based on the proposed algorithm, a promising connection between time-series data and VCRP has been described. Of particular note is the fact that in particular the initial and terminal stance phases, as well as the heel-off phase, are accompanied with an increase in VCRP, whereas the foot flat period can be associated with a low VCRP. Significant differences in averaged discrete VCRP variables and in continuous VCRP time-series data have not been detected between a healthy and an injured group of runners.

As discussed in SP1, high within-group variation as a consequence of a low number of subjects might be the reason for the absence of an apparent link between VCRP and injury.

Based on the findings of SP1, the author believes that the application of VCRP in future research must be considered critically, since differences in commonly-used kinematic variables have not been found in VCRP data. Additionally, the non-intuitive interpretation of VCRP data does not lead to reliable conclusions about how abnormal VCRP might be changed to normal VCRP, leading to normal lower leg coordination and movement patterns and a reduction in the risk of injury. The author is aware of the fact that the retrospective study design does not allow any inferences about the development of injury to be drawn from the obtained data, but due to the current lack of correlation between VCRP data and injury, a future application of CRP and VCRP has to be questioned.

The question must be asked as to why no studies have been conducted to date including healthy runners to define “normal” VCRP. If VCRP is to find its application in future studies, this has to be a necessary step prior to the evaluation of injured coordination patterns. Further, although SP1 portrays one potential algorithm to calculate VCRP, a generally-applicable calculation method should be determined in order to enable comparisons between different study findings and ways of interpreting data. The lack of consensus in data assessment and processing applies not only to CRP and VCRP; this problem is also evident in the evaluation of lower leg kinematics, since a wide variety of data collection and calculation methods have been used in past studies, leading to inconsistent results.

Based on the presented results of SP1, the answer to research question 1 has been found to be that VCRP does not appear to be an appropriate method to differentiate between lower leg kinematics of healthy and injured runners suffering from ITBS who reveal differences in commonly-used kinematic variables, and consequently is inappropriate to use to determine kinematic risk factors leading to OI in runners.

Scientific paper 2

“**Prospective analysis of intrinsic and extrinsic risk factors on the development of Achilles tendon pain in runners.”**

The current study is the first prospective study evaluating intrinsic and extrinsic risk factors causing OI in runners. Potential multifactorial interrelationships of clinical, biomechanical and training-related risk factors as assumed in literature have been evaluated in SP2. For a group of runners who generate AT, a combination of kinematic abnormalities and lower knee flexor strength appears to cause higher stress on the Achilles tendon. The higher stress on the tendon might be potentiated by training sessions with higher speeds and higher impacts, ultimately leading to AT. Whether weak knee flexors cause alterations in lower leg

kinematics, or are themselves caused by altered lower leg kinematics, cannot be determined. Further, the influence of additional exercise beyond that measured in the running programs has to be evaluated in more detail.

The author notes that these findings are based on results from only ten injured runners, and so considers the results with caution. However, the mechanisms outlined which lead to AT appear to be reasonable and can be partially confirmed through daily clinical routines. Nevertheless, the realization of this prospective study has been constrained by the nearly 50% drop-out rate of subject participation over a one-year period and by the lack of compliance with a second examination to diagnose presenting symptoms in case of injury.

More technically advanced techniques, such as training surveillance via GPS and online applications for smart phones to document training, might be used to minimize the drop-out rate and to improve the applicability of training documentation. Particularly, the use of GPS technology might reveal more significant details about individual training programs, since training sessions often combine different aspects such as slow and fast training intervals, so that calculation of an averaged running speed does not reflect important characteristics of the whole training session.

Further, as described in SP2, the evaluation of shod lower leg kinematics may also reveal more information about mechanisms leading to injury, since barefoot running on a soft substrate in a laboratory setting does not reflect a typical real-world training session. Despite the application of markers on the footwear upper and the resultant difficulties in assessing ankle and rearfoot kinematics, knee and hip joint motion patterns can in fact be analysed. Carrying out such a complex study with different measurement techniques is always accompanied with compromises in terms of feasibility and rational measurements. Therefore, research groups will have to determine which testing procedures can be carried out in a reasonable period of time and still account for the multifactorial aspects of how injury develops prior to the beginning of the study. Additionally, with regard to the transfer into clinical practice, the clinical examination and isometric strength measurements conducted in the current study appear to be beneficial, so that suitable preventive or therapeutic measures can be adjusted to meet the individual needs of each runner. The missing calculation of inverse dynamics, providing resultant joint forces and moments, is a major deficit of this study and therefore must be accounted for in future investigations to gain more insight into joint overloads and inappropriate strain distributions.

Thus, the continuation of the current prospective study appears to be essential in order to increase the number of healthy and injured subjects and to provide better information for the analysis of potential mechanisms not only for AT, but also for other symptoms. Finally, regression analyses, cluster analyses or principal component analyses can be carried out to identify evidence-based and injury-specific risk factors. The design and findings of SP2 might have a significant impact on future studies since the multifactorial interrelationships of different risk factors have been demonstrated. Thus, one-dimensional studies focusing on either clinical, kinematic, strength or training-related risk factors leading to OI will not be able to identify mechanisms for injury and consequently will not be able to propose possible prevention measures in the future. The implemented study protocol and applied measurement techniques in the current study should also serve as an example for future study designs since standardized, practical, reliable and repeatable clinical and biomechanical testing procedures are essential for the comparison of data and for possible multi-centre studies.

With regard to the second research question of this doctoral thesis, the author affirms that prospective studies can reveal potential injury-specific mechanisms causing OI in runners as outlined for AT in SP2. However, if the number of participants can be increased over the coming years, more detailed and specific information about these interacting mechanisms leading to injury will be acquired.

Scientific paper 3:

“Are prospective studies necessary to identify kinematic risk factors causing overuse injuries in runners?”

Due to the high rate of drop-outs in the current study, the question arises whether prospective studies are really necessary for the determination of risk factors causing OI in runners. From a logical point of view, prospective studies are essential to clarify cause-effect relationships since all measurements are carried out with injury-free runners who generate an OI during their study participation independent of its duration.

To date, no study has been carried out that has investigated clinical and biomechanical variables of the same group of runners in both healthy and injured states. This is a unique feature of the current study and, thus, enables the prospective evaluation of kinematic data with regard to alterations in movement patterns as a consequence of injury. However, this evaluation is rather limited as only 19 out of 45 injured runners fulfilled the criteria for a

second examination. Note that all runners included in the analysis were injury-free for at least six months prior to the IE and each eventually suffered from an acute OI. During the measurements of the lower extremity kinematics, no subject showed any signs of pain that would have influenced the movement patterns consciously.

Here, no differences in hip, knee, ankle and rearfoot kinematics have been found between the uninjured and injured states. Three subjects, or one sixth of the sample size of SP3, changed their running style from rearfoot strikers to midfoot or forefoot strikers, so that a possible influence of altered movement patterns on the development of OI cannot be ruled out completely. For runners not changing their running style, it can be concluded that lower extremity kinematics seem to cause injury in combination with other clinical, biomechanical or training-related factors, but do not alter as a consequence of injury. The author is aware of the fact that a quantity of 15 runners is too small for any evidence-based inferences of cause-effect relationships. However, if these first results can be confirmed by a larger number of runners, retrospective studies might lead to the same results as prospective studies. Consequently, new insights in injury mechanism might be gained with the systematic realisation of retrospective studies including acutely injured runners. Clinical variables and isometric strength measurements also need to be analysed with regard to the requirement for prospective studies in order to ultimately determine whether retrospective study designs are necessary for future investigations. If these factors also remain unchanged after the onset of an injury, the need for prospective studies needs to be questioned.

In answer to the third research question considering the findings of SP3, prospective studies do not appear necessary to determine kinematic risk factors causing OI in runners. Nevertheless, these findings must be confirmed in future by a larger number of subjects, since one sixth of the subjects analysed in SP3 altered their running style and therefore have been excluded from the analysis.

7. Conclusion

The calculation of CRP and VCRP represents a multi-dimensional approach in the evaluation of joint coordination patterns, which was thought to be advantageous for future studies. Due to the complex calculation algorithms of VCRP, the difficult and non-intuitive interpretation of data and the lack of correlation with injury, the implementation of VCRP does not seem to serve as a solid method for future studies.

In contrast, multifactorial approaches such as presented in SP2, analyzing the interactions of clinical, biomechanical and training-related risk factors, appear to be essential to determine injury-specific mechanisms leading to OI in runners. However, the high number of subject drop-outs experienced in this study is a major problem in the justification of a prospective study as portrayed in this thesis.

The preliminary results of SP3 showed no differences between kinematic data of hip, knee and ankle joints between injury-free and acute-injury states. Should this be confirmed for additional injury-specific clinical and biomechanical variables, and based on a larger population of runners, multifactorial retrospective studies can be implemented to determine injury-specific mechanisms.

8. Perspective

In order to determine injury mechanisms, the focus on future studies must be on multi-dimensional approaches analyzing extrinsic and intrinsic risk factors, independent of whether retrospective or prospective study designs will be implemented. As a first step to ultimately determining risk factors causing OI in runners, the current study needs to be continued to expand the number of participants, especially with regard to the results of SP3. With a larger sample size, should these results be confirmed for other injury-specific clinical and biomechanical risk factors, the implementation of retrospective studies may be the appropriate course of action for future applications. A well-designed retrospective and multifactorial study protocol might be realized in cooperation with other research groups, and implemented as a multi-centre study in order to provide a sufficiently large study sample size. Ultimately, the implementation of cluster, regression or principal component analyses will allow the determination of evidence-based mechanisms between clinical, biomechanical and training-related risk factors causing OI in runners.

However, for a successful determination of injury-specific interactions of risk factors for different running populations, further measures should be taken into consideration in future applications. First, it is essential to provide standardized and well-designed study protocols for clinical investigations and biomechanical measurement techniques (for example the assessment and calculation of lower leg kinematics or the measurement of maximal strength). Second, consistent differentiation between symptoms is important in upcoming analyses, since injury-specific interactions between clinical, biomechanical and training-related variables are presumed to exist. Therefore, clinician-led diagnostic procedures are required, not only to diagnose current complaints, but to also to verify the complaint against the signs and symptoms of other diagnoses (differential diagnosis). Third, well-defined inclusion and exclusion criteria are essential in order to evaluate risk factors for different running populations, since recreational runners and elite runners might reveal different training-related risk factors causing OI than novice runners. Nevertheless, the implementation of kinetic measurement techniques appears to be essential to allow, in combination with three-dimensional kinematics, the calculation of inverse dynamics, and consequently to provide more information about abnormal joint loading patterns and a potential link with the development of injury.

Potential effects of fatigue of the lower leg muscles on kinematics also have to be considered and investigated with regard to abnormal movement patterns and thus, to the development of OI. There are two approaches to data acquisition in this regard. First, data may be collected in a well-supervised laboratory setting on a treadmill to enable continuous measurements over a specific period of time. Second, the assessment of kinematic data during a prolonged run in a natural outdoor setting must be considered as reasonable alternative to the laboratory, either via inertial sensor systems or, again, via three-dimensional motion capturing systems.

Besides clinical, biomechanical and training-related risk factors as described in this doctoral thesis, additional factors such as physiological, social and neuro-muscular factors might also be taken into consideration for future investigations of the development of OI in runners.

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10. Appendix

10.1. Case Report Form

On the following pages, the case report form (CRF) of the current study for the initial examination is presented.

Remember:

_____

_____

_____

_____

_____

_____

UexPain - prospektiv

Probanden-Nr.: P - _|_|_|

Initialen: _| _| ♂ ❑ ♀ ❑

Geburtsdatum: _|_| . _|_| . _|_|_|_|

Größe: _|, _|_| m

Gewicht: _|_|, _| kg

Eingangsuntersuchung

Datum _|_| . _|_| . _|_|_|_|

Uhrzeit: _|_| : _|_| Uhr

Zweite Untersuchung mit Beschwerde

Datum _|_| . _|_| . _|_|_|_|

Uhrzeit: _|_| : _|_| Uhr

Messtag 1

-beschwerdefrei-

Rekrutierung des Probanden durch:

Lauftreff ❑

Arzt ❑ Name: _____

Presse ❑

Sonstiges ❑ _____

Probandenaufklärung

Der Proband wurde von mir über Wesen, Bedeutung und Tragweite dieser klinischen Prüfung aufgeklärt (gem. AMG § 40, 41), die Probanden-information wurde ausgehändigt, und der Proband hat die Teilnahme freiwillig erklärt**:**

JA ❑ NEIN ❑

Die schriftliche Einverständniserklärung erfolgt e am

_|_| . _|_| . _|_|_|_|

Bestehen irgendwelche *Begleiterkrankungen*?

JA ❑ NEIN ❑

Wenn JA, welche?

_____

Nehmen Sie zurzeit *Medikamente* ein? JA ❑NEIN ❑

Handelsname / Substanzgruppe	Erkrankung	Seit (Monat / Jahr)	bei Bedarf	ständig
			❑	❑
			❑	❑

Bemerkungen:

_____

Hatte der/die Teilnehmer/in bereits eine oder mehrere OPs an der UEX?

□ **NEIN** □ **JA** _____

_____

Hatte der/die Teilnehmer/in bereits eine oder mehrere Überlastungsbeschwerden an der UEX?

□ **NEIN** □ **JA** _____

_____

1. Klinische Eingangsuntersuchung – P1

Untersucher: ……

1.1. Gelenk - Bewegungsausmaß

		LINKS					Vergleich	RECHTS				
	Norm	hypo	norm	hyper	S	Grad	li <=> re	hypo	norm	hyper	S	Grad
Hüfte												
Flexion	130/140	□	□	□	□	__|__|__°	☐	□	□	□	□	__|__|__°
Extension	20/30	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
Abduktion	30/50	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
Adduktion	20/30	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
IR	30/45	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
AR	40/50	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
Knie												
Flexion	120/150	□	□	□	□	__|__|__°	☐	□	□	□	□	__|__|__°
Extension	5/10	□	□	□	□	__|°	☐	□	□	□	□	__|°
Sprunggelenk												
Dorsalext	20/30	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
Plantarflex	40/50	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
Pronation	15	□	□	□	□	__|__°	☐	□	□	□	□	__|__°
Supination	35	□	□	□	□	__|__°	☐	□	□	□	□	__|__°

Bemerkungen: _____

_____

1.2 Muskulatur – Dehnfähigkeit

	LINKS			Vergleich	RECHTS		
	norm	leicht	deutlich	li<=> re	norm	leicht	deutlich
m. rectus femoris	□	□	□	□	□	□	□
m. iliopsoas	□	□	□	□	□	□	□
Ischios	□	□	□	□	□	□	□
Obers Test	□	□	□	□	□	□	□

1.3 Triggerpunkte Muskulatur / Druckschmerz

	LINKS			Vergleich	RECHTS		
	norm	leicht	deutlich	li<=> re	norm	leicht	deutlich
Tractus	□	□	□	□	□	□	□
Wade	□	□	□	□	□	□	□
m. piriformis	□	□	□	□	□	□	□
m. gluteus med.	□	□	□	□	□	□	□
……	□	□	□	□	□	□	□

	ja	nein		ja	nein
Tibia medial	□	□	□	□	□
Epicond. lateral	□	□	□	□	□
Patellaspitze	□	□	□	□	□
Achillessehne	□	□	□	□	□

	frei	blockiert		frei	blockiert
prox. Tibiafib.Gelenk	□	□		□	□

Finger-Boden-Abstand ⌊_⌋⌊_⌋ cm

Abstand Ferse-Gesäß ⌊_⌋⌊_⌋ cm ⌊_⌋⌊_⌋ cm

Beckenschiefstand normal □ □ auffällig re □ li ⌊_⌋,⌊_⌋ cm.

Weitere Bemerkungen:

2. Kinematische Untersuchung - P1

Zuständiger Untersuchungsleiter: Hein ❑

Vertretung:_____ ❑

Probandennummer: **P - ……**

Messablauf

I.Messungen barfuss:

Dateiname: P1bf_01 … P1bf_30
Statische Messung durchgeführt ❑
Seitenwechsel ❑
Laufgeschwindigkeit: 11,4km/h – 12,6 km/h
MIND. 30 MESSUNGEN DURCHGEFÜHRT UND ABGESPEICHERT ❑

❑ Rückfußläufer ❑ Mittelfußläufer ❑ Vorfußläufer

Bemerkungen

II. Messungen Schuh:

getragener Schuh:

Marke: …… Schuhmodell:…….

Alter: …….. getragene KM: …….

Dateiname: P1shoe_01 … P1shoe_30
Statische Messung durchgeführt ❑
Seitenwechsel ❑
Laufgeschwindigkeit: 11,4km/h – 12,6 km/h
MIND. 30 MESSUNGEN DURCHGEFÜHRT UND ABGESPEICHERT ❑

❑ Rückfußläufer ❑ Mittelfußläufer ❑ Vorfußläufer

Bemerkungen

3. Kraft – Test

Untersucher: …….

Rechtshänder ☐ Linkshänder ☐

1. Rumpf - DAVID

Rücken Extension	(30°)	⌐⌐⌐ Nm		Sitz	⌐
Bauch Flexion	(0°)	⌐⌐⌐ Nm		Fuß	⌐
Rumpf Rotation	(30°)	links	⌐⌐⌐ Nm	Rücken	⌐
		rechts	⌐⌐⌐ Nm	Brust	⌐

2. Hüfte

DAVID - Abduktion	(30°)	⌐⌐⌐ Nm	
DAVID - Adduktion	(30°)	⌐⌐⌐ Nm	
		LINKS	RECHTS
FREI - Abduktion	(10°)	⌐⌐⌐⌐ N	⌐⌐⌐⌐ N
FREI - Adduktion	(30°)	⌐⌐⌐⌐ N	⌐⌐⌐⌐ N
FREI - Extension	(0°)	⌐⌐⌐⌐ N	⌐⌐⌐⌐ N

3. Knie

DAVID - Extension	(60°)	⌐⌐⌐ Nm	⌐⌐⌐ Nm	Sitz	⌐
DAVID - Flexion	(30°)	⌐⌐⌐ Nm	⌐⌐⌐ Nm	Sitz	⌐

Proband hatte Schmerzen

❑ nein

❑ ja

Bei ……..

Weitere Bemerkungen:

4. Laufspezifische Anamnese

Trainingsalter ⎽⎸ Jahre ⎽⎸ Monate

Laufpensum pro Woche (letzte 12 Monate) ⎽⎸⎽⎸km

Laufeinheiten pro Woche (letzte 12 Monate) ⎽⎸

Laufzeit pro Woche (letzte 12 Monate) ⎽⎸h ⎽⎸⎽⎸min

Mittleres Lauftempo/ Belastung

⎽⎸⎽⎸ km/h ⎽⎸:⎽⎸⎽⎸min ⎽⎸⎽⎸⎽⎸min^{-1}

Prozentuelle Verteilung Laufuntergrund

Asphalt	⎽⎸⎽⎸ %	Schotter	⎽⎸⎽⎸%
Kies	⎽⎸⎽⎸%	Tartan	⎽⎸⎽⎸%
Rasen	⎽⎸⎽⎸%	Laufband	⎽⎸⎽⎸%
Finnenbahn	⎽⎸⎽⎸%	anderer	⎽⎸⎽⎸%

Weich ⎽⎸⎽⎸⎽⎸% medium ⎽⎸⎽⎸⎽⎸% hart ⎽⎸⎽⎸⎽⎸%

Bisherige Laufbestzeit & ca. Anzahl absolvierte Wettkämpfe

10km: ⎽⎸h ⎽⎸⎽⎸min Jahr: ⎽⎸⎽⎸⎽⎸⎽⎸ Anz: ~ ⎽⎸⎽⎸

HM ⎽⎸h ⎽⎸⎽⎸min Jahr: ⎽⎸⎽⎸⎽⎸⎽⎸ Anz: ~ ⎽⎸⎽⎸

M ⎽⎸h ⎽⎸⎽⎸min Jahr: ⎽⎸⎽⎸⎽⎸⎽⎸ Anz. ~ ⎽⎸⎽⎸

Was haben Sie in den letzten beiden Tagen trainiert?

vorgestern: ……..Schmerzen ja❑ nein❑

gestern: ……....Schmerzen ja❑ nein❑

Andere regelmäßige Sportarten

Sportart	Trainingsalter	TE / Woche	Umfang / W

Laufschuhe

Marke	Modell	Alter [J]	Kilometer

Bemerkungen

10.2. Acceptance letter - Scientific paper 2

From: SJMSSedoffice@wiley.com

To: Tobias.Hein@med.uni-tuebingen.de

CC: Tobias.Hein@med.uni-tuebingen.de, Pia.Janssen@med.uni-tuebingen.de, Ursula.Wagner-Fritz@med.uni-tuebingen.de, Georg.Haupt@med.uni-tuebingen.de, Stefan.Grau@med.uni-tuebingen.de

Subject: Scand J Med Sci Sports: Decision to accept revised manuscript SJMSS-O-260-13.R2

Body: 04-Sep-2013

Dear Mr Hein,

It is my pleasure to accept your manuscript entitled "Prospective analysis of intrinsic and extrinsic risk factors on the development of Achilles tendon pain in runners." in its current form for publication in the Scandinavian Journal of Medicine and Science in Sports.

As part of the Journal's continued commitment to its authors, the Editorial Office and Publisher wish to keep you informed about what will happen next and, as the attached footer contains important information regarding journal publication and services for authors, you may wish to save it for future reference.

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Stephen Harridge
Editor-in-Chief
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SJMSSedoffice@wiley.com

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To: Tobias.Hein@med.uni-tuebingen.de

CC: Tobias.Hein@med.uni-tuebingen.de, Pia.Janssen@med.uni-tuebingen.de, Ursula.Wagner-Fritz@med.uni-tuebingen.de, Bettina.Barisch-Fritz@med.uni-tuebingen.de, Stefan.Grau@med.uni-tuebingen.de

Subject: Scand J Med Sci Sports: SJMSS-O-547-13 has been submitted

Body: 08-Oct-2013

Dear Mr Hein,
Your manuscript entitled "Are prospective studies necessary to identify kinematic risk factors causing overuse injuries in runners?" has been successfully submitted online and is presently being given full consideration for publication in the Scandinavian Journal of Medicine and Science in Sports. Your manuscript ID is SJMSS-O-547-13.
Please mention the above manuscript ID in all future correspondence.

The review process is usually completed within 10 weeks, but can take longer, depending on reviewer availability (e.g. during holiday periods or if an alternative reviewer needs to be approached). This time frame includes selecting and inviting reviewers, awaiting their response to the request, consideration of the reviews by the assigned Editor and, finally, the Editor-in-Chief's decision and communication with the author. Please be patient during this process and it would be much appreciated if you would not email the Editorial Office to enquire about the status of your manuscript until a period of at least 10 weeks has lapsed. You can track the progress of your paper using the tracking facility in your Author Centre.

If there are any changes in your street address or e-mail address, please log in to http://mc.manuscriptcentral.com/sjmss and edit your user information as appropriate.

Please be aware that any accepted article which exceeds 6 printed pages will be charged. Excess pages must be paid for at a rate of GBP 95 per page. Review papers are as a rule not charged for excess pages, but should not exceed 10 printed pages. Papers will be invoiced upon publication. One printed page contains about 5,400 letters, space between words included (but not tables and figures).

Thank you for submitting your manuscript to the Scandinavian Journal of Medicine and Science in Sports.

Yours sincerely,

Tara Noonan
Scandinavian Journal of Medicine and Science in Sports
SJMSSedoffice@wiley.com

Date Sent: 08-Oct-2013

11. Affidavit

I, Tobias Hein, born 28. November 1982 in Rheinfelden, hereby declare that I wrote this dissertation at hand on my own, that I did only use the sources and materials referred to and that each citation is made explicit. Moreover, I declare that I did neither use the dissertation in this or any other form as a thesis nor submit the work as a dissertation to another faculty.

Tübingen, 7. November 2013

Tobias Hein

12. Curriculum Vitae

Tobias Hein, born 28. November 1982 in Rheinfelden, Germany

School

1991 – 1995	Elementary school (Schillerschule), Rheinfelden, Germany
1995 – 06/2002	Secondary school (Georg-Büchner Gymnasium), Rheinfelden, Germany

Civilian service

07/2002 – 04/2003	Frauenklinik, Rheinfelden, Germany

Education

03/2004 – 02/2009	Medical Engineering and Sports Medical Engineering, Fachhochschule Koblenz, Place of location Remagen, Germany
03/2009 – present	Ph.D. student, Eberhard Karls University Tübingen, Faculty of Economics and Social Sciences, Tübingen, Germany

Visit abroad

08/2012 – 10/2012	Human Performance Laboratory, University of Calgary, Calgary, Canada. Supervisor Ph.D. Darren Stefanyshyn

Career

03/2009 – 10/2013	Technical employee, University Clinic Tübingen, Department of Sports Medicine, Research area “Clinical Biomechanics”, Tübingen, Germany
10/2013 – present	Senior researcher and Lecturer, University of Gothenburg, Department of Food and Nutrition, and Sport Science, Gothenburg, Sweden