Achilles tendon ultrasound findings in triathletes before and after the Ironman Triathlon

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November 2006

Declaration

I, Karen Schwabe, hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

Signed by candidate

(Signature)

22/11/2006

(Date)

Acknowledgements

- Professor Martin Schwellnus, the Course Convener, for his interest, advice and encouragement during the course of the study and in the preparation of the thesis.

- Dr Malcolm Collins, for the statistical analysis of the data, his interest and advice in the preparation of the thesis.

- Ms Yvonne Blomkamp, for technical assistance and her continuous encouragement.

- Dr Richard de Villiers, radiologist, for his assistance in doing all the ultrasound scans.

- Ms Nerle Neethling-du Toit, radiographer, for her assistance with the ultrasound scans.

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List of abbreviations

BMI	Body Mass Index
CD	Colour Doppler
COX	Cyclo-oxygenase
HLA	Human Leucocyte Antigen
IL	Interleukin
MRI	Magnetic Resonance Imaging
NSAIDS	Non-Steroidal Anti-Inflammatory Drugs
PD	Power Doppler
PGE-2	Prostaglandin E-2
US	Ultrasound

Abstract

Background: Overuse injuries, especially of the lower extremity, are common in Ironman triathletes. Achilles tendon injuries in particular have been documented in triathletes who either train or participate for the Ironman event. The effects of competing in ultra-endurance events, such as the Ironman triathlon, on the development of morphological changes and/or changes in blood flow in the Achilles tendon and surrounding tissue have not been investigated.

Objective: The aim of this prospective cohort study therefore was to assess the morphological and blood flow changes in the Achilles tendons of triathletes competing in the 2006 South African Ironman Triathlon.

Methods: 109 triathletes who entered the 2006 Ironman Triathlon in South Africa were recruited as subjects during the registration period. In the pre-race period (1-3 days before the race), all the subjects were asked to complete a questionnaire containing demographics, past injury and training history, medical history including medication use and family history. Subjects then underwent a pre-race clinical examination of the Achilles tendons (n=181), as well as an Ultrasound (US) examination to assess Achilles tendon morphology and blood flow (CD). The clinical assessment and US examinations were repeated immediately after the race (at the finish line) and in a sub-sample of tendons (n=58), 6-8 weeks after the race.

Results: The main findings of the study were that 19/109 (17.4%) of the triathletes had evidence of Achilles tendinosis on US and clinical examination before the race. Factors associated with Achilles tendinosis were increased

age ($p<0.001$), faster running times ($p<0.05$), a past history of any tendon injury ($p=0.022$), a past history of Achilles tendon injury ($p<0.001$), and a history of oral corticosteroid use ($p=0.003$). Immediately post-race, there was a significant increase (% tendons) in blood flow in the Achilles tendon and surrounding tissues (pre=3.4%, immediately post=14.3%, $p=0.007$). Furthermore, after 6-8 weeks, this increased flow returned back to pre-race values (0% of tendons). The immediate post-race increased blood flow was strongly related to the presence of pre-race Achilles tendinosis (US) (odds ratio=4.7, 95% confidence interval 1.8 to 12.2, $p=0.002$), and tenderness to palpation ($p=0.011$) and a positive shift test ($p=0.025$).

Conclusion: About 17% of triathletes who participate in an Ironman have evidence of Achilles tendinosis, and this will result in an increased blood flow in the tendon and surrounding tissues immediately after the race. The increase in blood flow returns to normal at 6-8 weeks after the race, but long term consequences of repetitive increases in blood flow in the Achilles tendon are not known and require further investigation.

Keywords: Achilles tendon, blood flow, triathlon, injuries, tendinosis, ultrasound, Ironman

Chapter 1

Introduction and scope of the thesis

The first Ironman ultra-endurance race was held in Hawaii in 1978. It is said that during an Awards ceremony following a Hawaii running race, the competitors disputed who is better conditioned – swimmers, runners or other athletes. In order to settle this dispute, a unique race was designed, which consisted of a Waikiki Roughwater swim (2.4 miles), the Around-Oahu Bike Race (112 miles) and the Honolulu Marathon (26.2 miles). This race was the beginning of what is now an international ultra-endurance race that attracts more and more interest with a number of Ironman competitions around the world (http://ironman.com/mediacenter/history/ironman-triathlon-world-championship). The medical care required for Ironman triathletes, has also received attention. There is a growing body of scientific literature dealing with the training, nutritional needs, and medical problems encountered by these athletes [1]. In addition, injuries as a result of the intensive training and competition in Ironman triathlon can also occur [2;3].

It has been documented that injuries in triathletes are common, especially overuse injuries [2;3]. In the 1986 Hawaii Ironman Triathlon, 91% of the triathletes reported at least one soft tissue overuse injury during the previous year's training [4]. In another study, at least one injury was reported by 74.8% of the triathletes during their participation in triathlon [5]. It has been shown that 55% of the injuries occurred in the lower extremity, and of those, tendon injuries was the main type of injury [6]. In another study on injuries in triathletes,

the most common injuries reported were of the Achilles tendon, lower back and the knee [7].

Achilles tendon injuries are therefore common in triathletes, and this was chosen as the focus of this thesis because the risk factors and pathogenesis of Achilles tendon injuries have not been well studied in triathletes. In Chapter 2, various aspects related to Achilles tendon injuries in triathletes will be reviewed. The review will however focus on the possible relationship between morphological changes and/or changes in blood flow in the Achilles tendon and surrounding tissues that may occur as a result of competing in an Ironman triathlon. In Chapter 3, the methods and results of a study to assess the effects of competing in an Ironman triathlon on the Achilles tendon will be presented. Finally, in Chapter 4, a summary of the findings of this thesis and practical and the clinically relevant outcomes of this thesis will be presented.

Chapter 2

A review of Achilles tendon injuries in triathletes with specific reference to morphological and blood flow changes in the tendon

2.1. Introduction

The triathlon is a 3-event endurance sport in which athletes compete sequentially in swimming, cycling and running [8]. It is the youngest official Olympic medal event, making its debut at the Sydney Olympics in 2000.

There are four different levels of triathlon competitions: (1) Sprint (2) Olympic (3) Half Ironman and (4) the Full Ironman (Table 1).

Table 2.1.: The four different types of triathlon and the various distances

Event	Sprint	Olympic	Half Ironman	Ironman
Swim	0.8km	1.5km	1.9km	3.8km
Cycle ride	21km	40km	90km	180km
Run	5km	10km	21km	42.2km

The full Ironman is the most physically demanding of the 4 types of triathlons, and consists of a 3.8 km swim, a 180 km cycle ride and a 42.2 km run. Triathletes face a wide range of environmental conditions and physical

demands that are in excess of those found in individual sport events of comparable duration [8]. Therefore, the sports physician looking after these triathletes is faced with many potential medical problems and complications during this event [1]. These medical complaints have recently been reviewed and include musculoskeletal injuries, trauma, muscle cramps, heat illness, postural hypotension, excessive exposure to ultraviolet radiation, gastrointestinal problems, post-race bacterial infections, immunosuppression, sympathetic nervous system problems, psychological exhaustion and haemolysis [1].

However, the focus of this thesis is on musculoskeletal injuries in triathletes, in particular injuries of the Achilles tendon. This review chapter is divided into the following main sections. In Section 2.2., the epidemiology of injuries in triathletes with specific reference to Achilles tendon injuries, classification of Achilles tendon disorders, and risk factors for Achilles tendon injuries will be briefly reviewed. In Section 2.3., the various hypotheses relating to the pathogenesis of Achilles tendon injuries will be explored. This will be followed in Section 2.4. by an overview of the normal anatomy and blood supply of the Achilles tendon, and the imaging modalities that may be used to assess morphological changes and/or changes in blood flow in the Achilles tendon (Section 2.5.). Section 2.6. will focus on the relationship between symptoms/signs of Achilles tendon injury and changes in morphology and blood flow in the tendon. Finally, Section 2.7. will review the possible effects of an acute exercise bout on changes in Achilles tendon morphology and blood flow.

2.2. A brief overview of Achilles tendon injuries (Epidemiology, classification, and risk factors)

2.2.1. Epidemiology of injuries in triathletes with specific reference to Achilles tendon injuries

The data on the epidemiology of injuries in triathletes is sparse and there are wide differences in injury prevalence reported in the literature. Because the sport is relatively young, the long-term physical effects of injuries in triathletes has not been well studied [9], and most of the studies on epidemiology were only undertaken in the last 10 years. The main results of some of these studies will now be briefly reviewed, highlighting the main injuries that occur and possible risk factors for these injuries.

In a recently published questionnaire-based study [5] among 656 triathletes, it was found that 75% of the triathletes experienced at least one injury during the event. More than 50% of the respondents reported one or more contusion or skin abrasion, 33% reported suffering from muscle/tendon injuries, 29% from ligament/capsule injuries, and 12% reported fractures. Chronic complaints in the Achilles tendon, the knee, and the back were reported by 76% of the triathletes. The majority of the injuries (55%) were sustained during the cycling training sessions, whilst 19% of all the injuries occurred during the competition. In this study, factors associated with injuries were age, performance level and weekly training hours. High-performance triathletes

and those with a large number of weekly training hours incurred largely muscle/tendon injuries.

In the Hawaiian Ironman Triathlon (1986), a case series study [4] in 95 triathletes showed that virtually all of the triathletes (91%) sustained at least one soft tissue overuse injury during the previous year. In this study the back was the area most commonly involved (72%), followed by the knee and thigh (63%) and the ankle and foot (61%). Most triathletes reported injuries involving more than one anatomical area (79%).

In another study, injuries and training history was documented in 257 triathletes over a period of one year prior to an event [10]. Forty-nine percent of the triathletes suffered from a training-related overuse injury in one year. Two injuries were reported in 18% and 3 injuries in 7% respectively of the injured triathletes. Running was reported as the cause of 62%, and cycling was identified as the cause in 12,5% of the injuries. Swimming contributed to 11% of the injuries, all being shoulder-related. Knee injuries (25%) were the most common, followed by the shoulder (14%), the Achilles tendon (10%), the ankle (7%) and lower leg (7%). In this study, it was concluded that triathletes training for a triathlon have about a 50% likelihood of sustaining an overuse injury during a 1-year period prior to the event. However, in this study there was no relationship between injuries and training mileage, age, gender or other characteristics of the triathletes [10].

In a retrospective cohort study [3], over a period of 8 weeks, among 155 British triathletes, 37% of triathletes reported at least one injury. Overuse was reported as the cause in 41% of the injuries sustained. Sixty-five percent of the injuries occurred during running, and the most common anatomical sites for injury were: (1) ankle/foot 27% (2) thigh 20% (3) knee 19% (4) lower leg 16% (5) back injuries. Injuries by tissue type were reported as: (1) muscle 40% (2) ligament/joint 28% (3) tendon 15% (4) skin 12% and (5) other body tissue 2%. In this study, the average injury rate was reported as 5.4 injuries per 1000 hours of training versus 17.4 injuries per 1000 hours of competition. In this study, the injury incidence was not related to the mean amount of weekly training or competition, intensity of training or frequency of training [3].

In another report [9], in a group of 92 Japanese triathletes, at least one overuse musculoskeletal injury related to triathlon was reported by 72% of the triathletes in the previous year. Thirty percent of the triathletes reported 2 injuries, and 14% had 3 or more injuries. The lower limb was the most commonly involved area (61%), with the knee again being the most frequently injured joint (54% of all lower limb injuries sustained). Only 8% of injuries were to the upper limb, and similar to that reported in previous studies [10], they were all shoulder-related. The anatomical distribution of the injuries was as follows: (1) knee 33%, (2) low back 28%, (3) shoulder 9%, (4) ankle 8% and (5) calf 5%. In this study, 3% of all the injuries were Achilles tendon injuries. Running was found to be the most common cause of lower limb injury.

In a retrospective, questionnaire-based study [7] of the injury and training characteristics of male elite, development squad and club triathletes over a 5-year period prior to the competition, an overuse injury occurred in 75% of the male elite and development squad athletes, and in 56% of the club athletes. The most common injuries reported were: (1) knee (14-22%) (2) low back (16-18%) and (3) Achilles tendon (10-18%). Once again, running was reported as the cause of most of the injuries in all 3 groups. The number of running injuries sustained was associated with triathlon training distance, cycling distance, swimming distance, number of triathlon workouts and number of running sessions within one week's race training. There was also a correlation between the number of overuse injuries sustained during cycling and the time spent running and cycling. It is important to note that the Achilles tendon was again shown to be one of the most common sites of injury in triathletes.

In the most recently published study on injuries sustained by triathletes, 258 triathletes completed a questionnaire in which they were asked to report injuries they had sustained during the previous 3 triathlon seasons [11]. In this cross-sectional survey there was an association between hours of training and the risk of sustaining an injury. The relationship between training and injury risk was reported as U-shaped, with triathletes training at either high levels or low levels of training were at increased risk of sustaining an injury. The results suggested that, for non-elite triathletes, the risk of sustaining an injury is lowest when training for a total of 8 to 10 hrs per week, and more specifically, cycling for 5 - 6 hrs and running for 3 - 4 hours per week. Training time spent on swimming did not affect the injury risk.

In summary, there is limited research available documenting the precise incidence and risk factors for injuries in triathletes. This is because there are 1) no well-conducted prospective cohort studies of injuries in triathletes, and 2) that injuries are mostly self-reported from questionnaires, which has a potential to introduce recall bias. However, it appears from available case series, retrospective cohort studies and cross sectional studies, that the following is known about injuries in triathletes:

- The reported annual incidence of injury in triathletes varies, but is about 50%
- The incidence of injury during training has been documented as 5.4/1000 hours of training and during competitions as 17.4/1000 hours
- The lower limb (in particular the knee, ankle and foot, and lower leg), followed by the lower back and shoulder are the most common sites for injury in triathletes
- Most injuries are soft tissue injuries involving muscles and tendons
- Of the three modalities, running appears to be associated with a higher risk of injury
- Either increased or decreased training volume and possibly increased age are factors associated with the development of injury in triathletes
- The precise incidence of Achilles tendon injury in triathletes is not well documented
- However, in most studies, where this has been examined, Achilles tendon injury has been reported as one of the more common injuries in triathletes

2.2.2. Classification of Achilles tendon disorders

The classification and terminology that is used to describe disorders of the Achilles tendon is not consistent [12;13]. Previously, the term 'Achilles tendonitis' had been used for any chronic pain in the posterior part of the heel [13]. However, it is now well accepted that this term is a misnomer. Histologically, areas of chronic injury of the tendon are characterized by a degenerative non-inflammatory process [14;15] and it is now well accepted that the term "tendinosis" for this chronic condition is more appropriate [13]. Most researchers in the field do however adopt the classification first presented by Puddu et al. (1976)[16], with some modifications (Table 2.2.). This has led to a review and re-classification of the disorders (injuries) of the Achilles tendon, and each of these injuries will now be briefly reviewed.

Table 2.2.: Classification of Achilles tendon disorders

a. Paratenonitis
b. Tendinosis
c. Insertional tendonitis
d. Retrocalcaneal bursitis
e. Haglund's deformity
f. Ruptures
Partial rupture
Complete rupture

Adapted from Puddu G et al, 1976 [16]

a. Paratenonitis

The term 'paratenonitis' refers to the pathology of the paratenon surrounding the Achilles tendon [17]. Clinically, acute paratenonitis is characterized by

oedema and hyperaemia of the paratenon and is associated with infiltration of inflammatory cells. Hours to days following injury, the tendon 'sheath' is filled with fibrinous exudates, which causes the crepitus that can often be felt on clinical examination. Acute paratenonitis can also progress to chronic paratenonitis, where fibroblasts and perivascular lymphocyte infiltrates appear. The peritendinous tissue becomes thickened and new connective tissue adhesions can occur [18].

b. Tendinosis

Tendinosis is defined as tendon degeneration, without clinical or histological signs of an inflammatory process. It may be associated with paratenonitis. The characteristic histopathological features are collagen degeneration with fiber disorientation, increased mucoid ground substance, and an absence of inflammatory cells [14;15]. Tendinosis may be associated with or without neovascularisation, and focal necrosis or calcification [18]. It is important to note that it is not clear whether this is one condition only, and whether it is purely degenerative in nature. Hence, some authors [19] prefer to use the term 'tendinopathy' which is a non-encompassing term implying that there is underlying pathology in and around the tendon. Furthermore, several pathologic conditions may co-exist in the tendon [13], and this often justifies the use of the term tendinopathy.

c. Insertional tendonitis

Insertional tendonitis can start as peritendonitis and progress to distal tendinosis with cellular necrosis and calcification. This may in turn lead to partial or full avulsion [17]. Symptoms are specific and related to pain at the bone-tendon junction, which frequently become worse after exercise [20].

d. Retrocalcaneal bursitis

Retrocalcaneal bursitis is a specific condition in which the retrocalcaneal bursa becomes inflamed, hypertrophied and may adhere to the tendon [17]. Clinically, it presents with pain anterior to the tendon, just superior to the insertion on the calcaneus.

e. Haglund's deformity

Haglund's deformity is an enlarged postero-superior and lateral calcaneal tuberosity. It occurs when the bursal projection is compressed for example by a poorly fitting heel counter. This, in turn, leads to subcutaneous irritation and bursitis of the adventitial bursa [20]. Clinically, it presents with pain in the posterior aspect of the heel.

f. Achilles tendon rupture

Achilles tendon rupture can be defined as a tear of a tendon, either partially or completely. It has been previously suggested that partial ruptures do not exist [21]. It is suggested that what is referred to as 'partial ruptures', are actually areas of focal degeneration [21;22]. Clinically, a history of sudden onset of severe pain is indicative of an acute tendon rupture (partial or complete), while a more gradual onset of pain and discomfort is the most common clinical presentation of a tendinopathy [13;23;24].

2.2.3. Risk factors associated with Achilles tendon injuries

The aetiological risk factors (both extrinsic and intrinsic) associated with Achilles tendon acute and overuse injuries have been recently extensively reviewed [25-31]. However, these proposed factors have mostly been based on the results of retrospective studies or the clinical experience of sports medicine experts [32;33]. Most reviewers agree that well-conducted prospective studies to identify possible risk factors for Achilles tendon overuse injuries are lacking [34]. A detailed analysis of the scientific evidence for each risk factor has recently been conducted [35], and is beyond the scope of this review and this thesis. However, a summary of the extrinsic and intrinsic risk factors are presented In Table 2.2. and a brief description of each risk factor will follow the summary.

Table 2.3. Risk factors for Achilles tendon injury

Extrinsic risk factors

1. occupation
2. physical activity and type of sport
3. training errors
4. environmental conditions
5. running surface
6. shoes
7. smoking
8. nutrition
9. medication

Intrinsic risk factors

1. age
2. gender
3. previous injury
4. somatotype
5. blood flow
6. biomechanical and alignment factors
7. systemic diseases
8. genetic factors

Adapted from [31]

a. Extrinsic risk factors for Achilles tendon injury

Occupation

It has been suggested that most of the patients with Achilles tendon injuries have white collar jobs and lead sedentary lives [36]. It has also been proposed that a sedentary lifestyle is a primary reason for poor basal circulation of the tendon and that this is, at least in part, responsible for the high number of tendon injuries in people with a sedentary lifestyle that occasionally take part in high intensity sports events [29].

Physical activity and type of sport

The majority of Achilles tendon injuries are associated with participation in sport [36;37]. A relationship between the type of physical activity and injuries to the Achilles tendon was found in these studies. Achilles tendinopathy is common among endurance athletes such as long distance runners [38], while Achilles tendon rupture is largely associated largely with explosive ball and racquet type games [23].

Training errors

Training errors that have been associated with the development of Achilles tendon injury include sudden increases in training load, frequency, or intensity, or excessive hill training [38]. Other possible training errors include excessive interval training and also resumption of training after periods of inactivity. It has been documented that the injury rate in novice athletes is higher than in experienced athletes [39].This could possibly be explained by the higher probability of training errors in the novice group or due to the fact that the muscle-tendon unit adaptation requires time and that novices, being less experienced in training, are more likely to load the unit more quickly [31].

Environmental conditions

Environmental conditions that have been associated with the development of Achilles tendon injuries, include extreme cold or heat, low or high humidity, high altitude, darkness (e.g. in sport orienteering) and strong wind [14]. The increased risk of developing Achilles paratendinitis while exercising in cold

weather outdoor training could perhaps be explained by a decreased temperature of the Achilles paratenon [40]. The paratenon, which is rich in mucopolysaccharides, serves as a lubricant for the gliding of the tendon and epitenon and the viscosity of the lubricant may be increased by low temperatures. This in turn may increase friction and therefore the risk for Achilles tendon paratendinitis. However, there is no scientific evidence to support this hypothesis, but it may explain why it is commonly suggested that 'warm-up' before exercise is important to prevent Achilles tendon injuries.

Running surface

Training surfaces which are cambered, sloping, very hard or slippery, have all been associated with increased risk of developing chronic Achilles tendinopathy [13;41]. However, there is little scientific evidence to support these associations. An association an increased risk of injuries, including Achilles tendon injuries, in soccer players who played more frequently on an artificial turf has also been reported [42].

Shoes

Appropriate footwear can reduce the impact forces of running, and may also provide mediolateral stability, which in turn prevents excessive subtalar joint 'pronation' and 'supination' [14]. An insufficient heel wedge may contribute to compensatory 'over-pronation', which is thought to be associated with Achilles tendon injuries [43]. Other factors associated with footwear that may predispose the athlete to Achilles tendon injuries, are worn midsoles, insufficient heel height, over-rigid soles, lack of shoe cushioning, and inflexibility [38]. However,

these are all associations, and there are no well conducted prospective studied to directly link footwear type and Achilles tendon injuries in a cause-effect relationship.

Smoking

The effect of cigarette smoking as a potential risk factor for distal biceps tendon injuries was studied. In this study, medical records of patients who presented with elbow injuries over a 5-year period were examined [44]. One of the findings was that smokers have a 7.5 times increased risk of distal biceps tendon rupture compared to non-smokers.

In a more recent study, the effect of smoking on rotator cuff injuries was examined in 72 shoulders of 36 cadavers [45]. Of the 36 shoulders with macroscopic rotator cuff tears, 23 were from cadavers with a history of smoking. Advanced microscopic rotator cuff pathology was more than twice as likely in those with a smoking history. It was therefore hypothesized that smoking, being a known risk factor for microvascular disease, may compromise the blood supply to the supraspinatus / infraspinatus tendon, thereby increasing the risk of developing tendon pathology.

However, in both studies, the sample size was small. In contrast to the two above studies, an earlier retrospective study [46] reported that in 23 patients with acute flexor tendon rupture, no relationship between smoking and tendon rupture was found. Also, no relationship between smoking and rotator cuff tears was found in a prospective study on 42 patients who underwent rotator

cuff repair [47]. There is, however, a lack of prospective studies relating smoking and injury risk to the Achilles tendon.

Nutrition

Vitamin C is a cofactor for proline hydroxylase, the enzyme required for the hydroxylation of proline, which is essential in the formation of collagen cross-links [48]. However, to date, no study could be found investigating the role of nutrition and the incidence of Achilles tendon injuries.

Medication

Fluoroquinolone antibiotics, such as ciprofloxacin, have been associated with increased risk of Achilles tendon rupture and tendinopathies [49;50]. In a population case control study it was found that 2-6% of all Achilles tendon ruptures in people older than 60 years could be attributed to the use of quinolones. In this study, the risk for Achilles tendon rupture was found to increase >4 times with the use of quinolones. The risk was found to even be higher in patients concomitantly treated with corticosteroids [49]. However, a recent prospective study was performed to determine if sub-clinical tendinopathy occurs in asymptomatic adults who were treated with fluoroquinolone antibiotics [51]. In this study, serial ultrasounds on 38 adults could not identify any relationship between the use of fluoroquinolone antibiotics and changes in the Achilles tendon.

Pathological mechanisms to explain the possible relationship between quinolone use and Achilles tendon injury have been reported [52;53]. In one

study, it was found that the incubation of Achilles tendon, Achilles paratenon, and shoulder capsule fibroblasts with ciprofloxacin resulted in a significant (66% to 68%) reduction in cell proliferation compared to control cells at Day 3 in culture [52]. It was also found that ciprofloxacin resulted in a statistically significant (14% to 60%) decrease in proteoglycan synthesis in all the fibroblast cell lines and a statistically significant increase in matrix-degrading proteolytic activity after 72 hours in culture. Ciprofloxacin exerted an inhibitory effect on fibroblast metabolism and stimulated matrix-degrading protease activity. All of these factors may contribute to tendinopathy of the Achilles tendon. It has also been documented that ciprofloxacin reduced IL-1β-induced PGE2 output in tendon-derived cells, and that various cellular activities of IL-1β could be modulated by the reduction of PGE2 output, which could therefore be implicated in fluoroquinoline-induced tendinopathy [53].

It has also been suggested that corticosteroids are associated with an increase risk for Achilles tendon rupture [49;54]. However, a recent retrospective study showed that low doses of corticosteroids were harmless to human tendon structure using flouriscopically guided injection [55]. Finally, it has also been suggested that anabolic steroids have been associated with Achilles tendon injuries [56].

b. Intrinsic risk factors

Age

In civilian populations there has been little evidence to suggest that increased age is related to injury risk [57]. In contrast to this, there appears to be an inverted 'U'–shaped risk curve for injury with age in the military population [58].

Gender

Several case studies have shown that there is a greater risk of Achilles tendon rupture in males [59-61]. Case-control studies have also shown that Achilles tendinopathy occurs more frequently in males compared to females [38].

Previous injury

In a large case series of 891 patients who had sustained various tendon injuries, including Achilles tendon rupture, 34% had previous symptoms of tendinopathy suggesting that an Achilles tendon rupture may be preceded by symptomatic injury [41]. It has also been documented that patients with Achilles tendon injuries have more degenerate tendons than asymptomatic subjects [25;62]. This may indicate that, in most cases, a rupture may well be preceded by tendinopathy.

Somatotype

In a recent review, it was concluded that there is only weak evidence to suggest that increased body mass and size are independent risk factors for Achilles tendon injury [31].

Blood flow

Although it has been suggested that there is a relationship between blood flow and Achilles tendon injury, scientific evidence for this relationship is weak [31]. However, this requires further investigation, and this relationship will be explored further in this thesis (Section 2.4.).

Biomechanical and alignment factors

Biomechanical and alignment factors that have been associated with Achilles tendon injuries are flexibility, muscle-tendon unit stiffness, leg length discrepancy, an excessively 'supinated' or 'pronated' foot, excessively high or low arches of the foot, and a large Q-angle [57]. Other factors include muscle imbalance, leg dominance and the presence of a plantaris tendon [13;38]. In a recently published cohort study, it was also identified that increased dorsiflexion range of motion and decreased plantar flexion strength as risk factors for the development of an Achilles tendon overuse injury [34].

Systemic diseases

Several reviews have indicated that a range of systemic diseases may be associated with the pathology of Achilles tendon rupture and Achilles

tendinopathy. Common diseases include inherited systemic diseases, such as Marfan's and Ehlers Danlos syndrome [14;27]. Endocrine and metabolic diseases that have been associated with an increased risk of Achilles tendon injury include diabetes mellitus, renal disease, thyroid disorders and rheumatic diseases such as rheumatoid arthritis and gout [14]. An elevated blood cholesterol has also been associated with Achilles tendon injury [63;64].

Genetic factors

Traditionally, genetic factors associated with Achilles tendon injury have been proposed based on the association in some studies of the ABO blood groups and the human leucocyte antigen (HLA) system with these injuries [14;31;37;41;65-70]. However, the evidence for these three factors as specific risk factors for Achilles tendon injury is weak [31]. In contrast, two recently published studies document an association between Achilles tendon injury and genes encoding for specific structural proteins in the tendon structure [31;71]. In one of these studies, it was shown that persons with specific variants of the tenascin-C gene appear to have a 6-fold increased risk of developing an injury of the Achilles tendon [71]. In the second study variants of the *COL5A1* gene were shown be associated with Achilles tendinopathies [31]. Therefore, recent evidence suggests that genetic factors are important risk factors for Achilles tendon injuries.

In summary, there are a number of intrinsic and extrinsic risk factors for Achilles tendon injuries. In general, the association between specific risk

factors and Achilles tendon injury is not based on strong evidence from either prospective cohort studies, or randomized controlled intervention studies.

2.3. Pathogenesis of Achilles tendon injuries

Three main hypotheses for the development of Achilles tendon injuries have been proposed in the literature. These are known as the degeneration hypothesis, the pathological hypothesis and the failure of the neuro-inhibitory mechanism hypothesis. Each of these hypotheses will be briefly discussed.

2.3.1. Degeneration hypothesis

In the degeneration hypothesis, it is suggested that the exposure of the tendon to repetitive mechanical loads, results in microscopic damage [12]. When the tendon is unable to repair fully, it leads to tendinosis (tendon degeneration) or chronic 'tendonitis" (an inflammatory process). This causes an abnormal, weak and inflexible tendon. This could then further result in pain and impair sports performance [12]. The findings from various studies [62;72-74], but not all studies [73;75] support this hypothesis.

2.3.2. Pathological hypothesis

In this hypothesis, it is believed that systemic diseases weaken the tendon, through mainly unknown pathological mechanisms [27]. The systemic diseases that have been associated with Achilles tendon injuries have been well

documented [14;27], and can be divided into metabolic and endocrine, inherited and rheumatic diseases.

Common examples of metabolic and endocrine diseases associated with Achilles tendon injuries include diabetes mellitus and renal diseases. Commonly inherited systemic diseases include Marfan- and Ehler's Danlos syndrome, while rheumatic diseases associated with Achilles tendon injuries are rheumatoid arthritis and gout [14]. In a study of 292 individuals with Achilles tendon rupture, only 23 (7.8%) had a history of a systemic disease [36].

2.3.3. Failure of the neuro-inhibitory mechanism hypothesis

This hypothesis suggests that when a sudden excessive load is applied to a normal tendon, it ruptures due to an inability to withstand the load [75]. It is suggested that the inhibitory neuroprotective (Golgi tendon organs) mechanisms are overcome by the excessive loading of the tendon. It has been suggested [31] that genetic factors may determine the adaptation to different stimuli, which in turn makes the susceptibility to tendon injuries variable amongst individuals.

2.4. Anatomy and blood supply to the Achilles tendon

2.4.1. Normal anatomy and blood supply of the Achilles tendon

The normal anatomy of the Achilles tendon has been well documented [13;20;76;77] and will not be discussed further in this thesis. However, because potential changes blood flow in the Achilles tendon and the surrounding tissue is a focus of this thesis, the normal blood supply to the Achilles tendon will be briefly reviewed.

The blood supply to the Achilles tendon is from three areas: (1) The musculotendinous junction, (2) osseotendinous junction and (3) the paratenon. The posterior tibial artery provides the major contribution to the blood supply [78].The peroneal artery also makes small contributions, probably through anastomoses with the posterior tibial artery. The anterior tibial artery has not been shown to be involved [79].

In the rabbit Achilles tendon, it was shown that only 35% of the total supply to the midsection of the Achilles tendon was from the paratenon [80]. Thus the remaining 65% is from sources that vascularise the tendon at the insertion and origin. However, in the human Achilles tendon, it is thought that the vascular supply is primarily by the paratenon.

Additional blood supply to the proximal third of the tendon is through vessels from the muscle that continues into the endotenon. However, this is not believed to be significant. Additional blood supply to the distal third is mainly by vessels of less than 300µm in diameter of the rete arteriosum calcaneare [81]. These vessels are fed by the fibular and posterior tibial arteries. This supply stretches from the insertional margin, proximally up the endotenon ± 2cm [78;81-83].

2.5. Imaging of the Achilles tendon

In this thesis, the morphology and blood supply of the Achilles tendon was studied. It is therefore appropriate to briefly review the imaging modalities that are available to assess morphology and blood flow in the Achilles tendon.

The most commonly used imaging modalities in the assessment of the Achilles tendon are ultrasonography (US) and Magnetic Resonance Imaging (MRI) [84]. Plain X-rays usually do not reveal any pathology. In general, excellent morphological information can be obtained from MRI and US, and the use of these two modalities to define morphology and blood flow in the Achilles tendon will now be reviewed.

2.5.1. Ultrasound

Utrasound (US) is a known, cost-effective and accurate method for evaluating the Achilles tendon [85;86]. US can be reliably used to locate Achilles tendon abnormalities, estimate severity, and determine most of the conditions

requiring surgical intervention [86]. Examples of pathologies that can be diagnosed by ultrasound are tendinosis (tendinitis), complete and partial ruptures, various degrees of calcification, peritendinous lesions, retrocalcaneal bursitis, lipomas, xanthomas and foreign bodies [87-89]. US is also used to assess the tendon's surrounding tissue, and is able to differentiate between functional and morphologic conditions [90]. However, US is not completely reliable for diagnosing peritendinitis and tendinosis and it cannot be used accurately to differentiate partial tendon ruptures from focal degenerative lesions [86]. US is a valuable tool to determine the Achilles tendon's thickness and echotexture [91]. Grey-scale sonography is, however, also operator-dependent [92]. In one study it was documented that of quantitative tendon measurements could be achieved when using two observers and a defined scanning protocol [93]. However, it was recommended that the same observer(s) perform the serial assessments.

US has additional features of Colour Doppler (CD) and Power Doppler (PD) ultrasonography. CD is an established technique which is useful in the investigation of tendons. The sensitivity and specificity of CD has been shown to be high. Colour flow is related to blood flow. It does not register normal circulation due to the low blood flow and only registers increased blood flow [94;95]. In contrast to the CD, PD is independant of the angle of the incident beam [96]. The CD and PD are seen as useful adjuncts to grey-scale sonography in the examination of Achilles tendinosis, especially because the presence of blood flow has been associated with pain, discomfort and physical restriction of the tendon [97]. Thus the CD and PD features of US can

assist the sports physician in detecting neovascularisation that can assosiated with chronic tendinosis [95].

However, it has been suggested that PD identifies more tendon microvascularity than the CD in the Achilles tendon [97]. This feature has recently been used in treatment, and it has been found that in patients with chronic Achilles tendinosis, ultrasound guided electro-coagulation into areas of neovascularisation, could be effectively used to treat symptomatic patients [98].

In a review of the recent literature, it was found that besides imaging of abnormalities US can also be used for various other functions, including:

- US can be used for guided electrocoagulation into areas of neovascularisation in patients with chronic Achilles tendinosis [98]
- US can be used as a selection tool for the treatment of acute Achilles tendon ruptures [99]
- Pulsed US treatment in animal studies was found to accelarate the healing in tenotomized Achilles tendons [100]
- US can be used in tendon injuries by stimulating the expression of type I and type 3 collagen via a process most likely mediated by the upregulation of TGF-beta [101;102]
- US can be used in the management of Achilles tendinopathy by ultrasound-guided percutaneous tenotomy [103]
- US is also used as a tool to guide local steroid injections into the peritendinous space in patients with Achilles tendinitis (tendinosis). The

injection of long-acting steroids have been shown to normalize the ultrasonographic pathological lesions [55;104]

2.5.2. Magnetic Resonance Imaging (MRI)

MRI is a non-invasive modality that does not use any ionising radiation. Furthermore, it has the capability of imaging in any plane. Therefore it is well suited to imaging the tendons in the ankle, because their course is not in standard orthogonal planes [105]. MRI also provides excellent soft tissue contrast and is well able to differentiate the tendons from surrounding fat, and to detect the presence of haemorrhage and oedema. Abnormalities of the Achilles tendon detected include complete and partial tears, tendinosis (tendinitis) and oedema [105;106]. However, it should be noted that the MRI has a false positive rate of 6-19% in asymptomatic tendons [85;92], and therefore the MRI findings should be interpreted with caution. Abnormal signals in the MRI of asymptomatic and active athletes have been found in numerous studies [107-109].

2.5.3. US versus MRI in the imaging of the Achilles tendon

There is an ongoing debate as to whether US or MRI is superior to diagnose Achilles tendon disorders in general. US is easily available, more cost-effective, portable and it has the capability of demonstrating physiological movement [87;88]. The US's real time feature helps to correlate the investigation with the symptomatic areas [88].

In contrast, it has been shown that MRI was superior for diagnosing ruptures, especially partial ruptures [110;111]. Futhermore, in a two-year prospective study in patients with chronic Achilles tendinopathy, a graded MRI appearance rather than US assessment was assosiated with clinical outcome [92].

It has been shown that US and MRI to have a comparative accuracy when using clinical criteria as a diagnostic yardstick [92]. Similar findings have been shown between between US and MRI's diagnostic accuracy in patients who were referred for surgery [85]. However, when the extended-field-of-view sonography was compared with MRI in Achilles tendon disease, it was found that US can challenge the MRI as the preferred imaging method in diagnosing symptomatic Achilles tendon disease, especially with respect to saving time and cost [112].

2.5.4. Other methods used to assess blood flow in the Achilles tendon

Although it is widely accepted that Colour Doppler (CD) and Power Doppler (PD) are means to measure blood flow in the Achilles tendon [98;113], [88;114], other methods to assess tendon blood flow have been described. These methods to quantify blood flow are: radioactive isotope Xenon-133 injection ventrally into the Achilles tendon [115], positron emission tomography [116], near-infrared spectroscopy, and indocyanine green [117].

In the research component of this thesis, it was decided to use US as the preferred method of investigation because it is valid, reliable (using the same operator), and also has the added advantage of being portable, Finally, US can accurately assess morphology and blood flow in the tendon, which was a focus of this thesis.

2.6. The relationship between symptoms/signs of Achilles tendon injury and changes in morphology and blood flow in the tendon

2.6.1. Morphological changes in the Achilles tendon and symptoms/signs of Achilles tendon injury

The relationship between morphological changes in the Achilles tendon, and symptoms and signs of Achilles tendon injury has only been investigated in a few studies. In a two-year prospective study the value of US and MRI in the assessment of Achilles tendon disorders was documented [92]. In this study, tendinopathy was diagnosed in 45 patients with symptoms in 57 Achilles tendons. All of the patients had an US examination, with CD and PD, and 25 patients also had an MRI. The results showed that the US identified abnormal morphology in 65% (37 of the 57) symptomatic tendons and normal morphology in 68% (19 of 28) asymptomatic tendons). It was found that baseline ultrasound findings did not predict the 12 month clinical outcome and that CD and PD did not improve the diagnostic ability of the US. The MRI identified abnormal pathology in 56% (19 of 34) symptomatic tendons and

normal morphology in 94% (15 of 16) asymptomatic tendons. Finally, it was documented in this study that lesser grades of MRI signal abnormalities at baseline were assosiated with an improved clinical status at 1-year follow-up. The authors concluded that there was only a moderate correlation between clinical assessment of chronic Achilles tendinopathy and ultrasound or MRI findings. However, graded MRI findings were associated with the clinical outcome whereas US findings were not.

In an observational 8-year follow-up study on 83 patients, it was found that mild-to-moderate changes were observed frequently in both the involved and the initially uninvolved Achilles tendon, and that these changes were not clearly related to the patients symptoms [26].

From these limited data, it appears that abnormalities of the tendon may persist, even if a patient becomes asymptomatic. Therefore it is suggested that the appearance on imaging should not be used as a guide whether a player is fit to return to play after Achilles tendinopathy [92].

2.6.2. Blood flow changes in the Achilles tendon and symptoms/signs of Achilles tendon injury

The relationship between changes in Achilles tendon blood flow, and the development of symptoms or signs of Achilles tendon injury has also been investigated in only a limited number of studies. In one study of 25 patients with chronic Achilles tendinopathy, the relationship between symptoms and

the PD ultrasound as well as tendon thickness was examined [118]. In this study, there was no strong relationship between symptoms and PD ultrasound of the Achilles tendon. However, tendon blood flow on PD ultrasound was positively related to tendon degeneration and functionality (as indicated by tendon size, patient age and functional test), but longitudinal effects were not studied.

In a study where the relationship between PD ultrasound and clinical severity in Achilles tendinopathy was examined, PD ultrasound of the Achilles tendon was not related to symptoms, but rather to functionality and chronicity of tendinopathy [118]. Similarly, it was also shown that a positive colour flow correlated with age, but no correlation was found with the presence of symptoms or sports participation. However, in this study all patients with positive colour flow and PD also had positive findings on the grey scale US [92].

In contrast, studies in animals (horses) showed that neovessels were seen in all the symptomatic tendons, but not in any of the control group. Neovessels in the animals had a similar appearance to those in human tendons [119]. More recently, it was shown in human subjects presenting with chronic painful tendinosis that, by using US and CD, neovascularisation was identified in the tendons, both inside and outside the area with structural tendon changes and pain [120]. This was not found in normal pain-free tendons. Furthermore, under guidance of US and CD, the area with neovascularisation could be sclerosed by injecting the sclerosing agent polidocanol into the vessels. In response to this sclerosis, 8/10 of the subjects were pain-free and had no remaining

neovessels. The 2 patients that were not pain-free still had remaining neovessels [120]. These results appear to indicate that neovascularization in the tendon is associated with pain.

However, there is also some eivdence to suggest that blood flow in the tendon in asymptomatic athletes, may in part be a physiological response to physical activity. In a recent study on elite badmington players, the intratendinous flow in the Achilles tendon, as detected by CD ultrasound, was evaluated [121]. This study included 72 players who were interviewed regarding Achilles tendon pain over the preceding 3 years. Of the 72 players, 64 players had a CD ultrasound examination before a match and this was repeated in 46 players after the match. Before the match, almost all of the athletes had evidence of CD flow -39 (84%) and only 7 (16%) of the players had no CD flow in either tendon. All the players had some CD flow, in one or both tendons after the match. Furthermore, the investigators report that there was a positive association between self-reported pain and an increase in CD flow in the non-dominant Achilles tendon. The fact that all the players exhibited CD flow after the match may indicate that blood flow was a physiological response to activity.

In a follow-up study by the same author, patients with chronic Achilles tendinosis with neovascularisation were treated by electrocoagulation and followed for 6 months [98]. After the 6 months period, 91% of the patients reported being effectively treated, but it had no effect on the intratendinous Doppler activity. The results of this study suggested that localization of

hypereamia could be the key to the pathology and identify the area for treatment, but that obliteration of the blood vessels may not be the key to treating the pain. A possible explanation of this effect and the use of sclerosing agents that was previously reported, could be that the destruction of nerves accompaning the vessels is more important that obliterating the blood vessles.

In a follow-up study, the investigators combined US and CD with immunohistochemical analysis of biopsy and diagnostic injections [120]. The results of this study showed that in the area with tendon changes and neovascularisation, nerve structures were in close relation to blood vessels and that injections of local anaesthesia temporarily relieved the pain in all of the patients. Thus these findings indicated that the area with neovascularisation (vessels and nerves) may be responsible for the symptoms of pain in chronic Achilles tendinosis [120].

Several studies indicate that Achilles tendinosis is accompanied by neovascularisation [18;95], but the precise role of neovascularisation in chronic tendinopathy remains an area of investigation. Tendons are generally a poorly vascularised tissue that relies largely on synovial fluid diffusion [41]. The hypothesis that Achilles tendinopathy is at least in part caused by an inadequate vascular supply to the affected area of the Achilles tendon has been advocated [118]. In apparent complete contrast to this, are the findings of hypervascularity seen in biopsies on patients presenting with Achilles tendinopathy [15]. The findings of neovascularity are similar to that found with

laser doppler flow [122]. Therefore it may be that the neovascularity in tendinopathy is not assosiated with an attempt of tendon repair, but rather with tendon degeneration [118].

2.6.3. Mechanism/s for changes in blood flow in the Achilles tendon

Possible mechanisms to explain the transient increase in blood flow in the Achilles tendon have been investigated. In one study on mechanical loading of calf muscles in vivo [123], it was shown that a COX-2 specific mechanism was responsible for the exercise-induced increase in prostaglandin synthesis, and that this increase played an important role in the peritendinous connective tissue blood flow during physical loading in vivo. However, testing was only done with isometric testing, and there was no follow-up.

In a study by the same author [124], it was shown that an exercise-induced hyperaemia in skeletal muscle is related to an increase in the concentrations of bradykinin and adenosine, in both skeletal muscle and connective tissue. The interstitial concentration of bradykinin and adenosine rose in response to exercise in the peritendinous tissue and skeletal muscle. This finding supported the fact that bradykinin and adenosine are potential regulators of peritendinous tissue blood flow. However, no follow-up was done to assess the time factor related to the decrease in the concentrations of the bradykinin and adenosine.

An association between peritendinous blood flow around the Achilles tendon (during exercise) and age could not be found [125].

Another mechanism that was examined was the association between the rise in calf muscle and peritendinous blood flow, to a fall in oxygen saturation during dynamic exercise in humans [126]. This study found a parallel rise between the above with a 7-fold increase in Achilles' peritendinous blood flow. A limitation of the study was the small sample size (7 individuals).

2.7. The effects of an acute exercise bout on morphology and blood flow in the Achilles tendon

2.7.1. The effects of an acute exercise bout on morphology of the Achilles tendon

The effects of regular training on tendon morphology has been studied [127], but we are not aware of any studies that examined the effects of an acute exercise bout on the morphology of the Achilles tendon. It is therefore not known whether an endurance event such as an Ironman triathlon can result in swelling of the tendon or surrounding tissues, or whether this would result in other changes in and around the tendon that may be visible using US.

2.7.2. The effects of an acute exercise bout on blood flow in the Achilles tendon

The effect of an acute bout of exercise on blood flow in a tendon has been documented in only a few studies. These studies differ with respect to the model used (animal vs. human), the exercise protocol, blood flow measurement techniques, measurement of peritendinous and/or intratendinous blood flow, and whether subjects had evidence of increased blood flow in the tendons before the exercise bout.

In one of the first studies to be undertaken, in an animal model, it was shown that blood flow to the Achilles tendon increased significantly during exercise as measured by the microsphere technique [128].

It has been shown that dynamic calf muscle contractions can increase peritendinous blood flow around the Achilles tendon [129]. This study utilized a technique where xenon-133 was injected into the peritendinous space ventrally to the Achilles tendon, 2 and 5 cm proximal to the calcaneal insertion. Following dynamic muscle contractions, blood flow in the 5 cm proximal to the Achilles tendon insertion increased 4-fold from rest to exercise, compared to the 2.5-fold increase, measured 2 cm proximal to the insertion of the Achilles tendon. Limitations of this study were a small sample size and no repeat follow-up over time.

The effect of a standardized intermittent static exercise protocol on peritendinous blood flow in the human leg has been studied [115]. Peritendinous blood flow was evaluated by the injection of the radioactive isotope xenon-133 ventrally to the Achilles tendon, 5 cm proximal to the tendon's insertion. The results of this study showed that the exercise protocol induced an average increase in the blood flow of 3-4-fold, which was equivalent to results obtained in an earlier study during dynamic heel raises [129]. The main limitations of these studies were the small sample size (6 subjects), and the fact that only intermittent static exercise was performed. The intermittent static exercise was also of a relatively low frequency compared with normal gait. Furthermore, there was no repeat follow-up over time.

The effects of an acute exercise bout on blood flow in a tendon have also been reported in the patellar tendon. The effect an exercise bout on patella tendon vascularity was studied in 17 volleyball players [130]. The main finding of this study was that there was increased vascularity in the Achilles tendon following activity ($p<0.001$). However, in this study all the players were known to have increased vascularity of the patella tendon prior to exercise and once again no follow-up assessment was done.

More recently, in the largest known study to date [121], 72 badminton players at an international badminton tournament were studied. In this study it was shown that in most of the players there was intratendinous blood flow (measured using CD) before the match, and that this blood flow increased immediately after the match. Furthermore, the increase in baseline blood flow

was associated with self-reported symptoms in the preceding 3 years in the non-dominant leg. Pain in the Achilles tendon was common in these athletes, with 40% reported having pain in the past 3 years and 13% reporting ongoing symptoms. The limitations of this study were that morphological changes in the tendon were not examined, and there was no follow-up to determine whether the changes observed in blood flow were transient or more permanent.

The same authors also report on Doppler activity before and after exercise in 10 non-trained healthy subjects and 11 subjects with chronic Achilles tendinosis in a recent study (unpublished) [131]. In this study, 30% of the asymptomatic tendons exhibited intratendinous Doppler activity before the exercise, and this increased to 80% after running exercise. Furthermore, all the symptomatic subjects exhibited increased Doppler activity before the activity, and this remained present after the exercise. The limitations of this study were once again that there was no measure of changes in morphology, there was no follow-up, and that the sample size was small.

In summary, it does appear from the findings of these studies that intratendinous blood flow is present in a percentage of asymptomatic athletes, and that this increases following an acute bout of exercise. Furthermore, there may be an association between the observed increases in tendon blood flow, and symptoms, but this requires further investigation.

2.8. Summary

- Injuries, in particular overuse injuries, are common in triathletes and the reported annual incidence is about 50%
- Lower limb injuries are one of the most common sites of injury, especially involving muscles and tendons
- The Achilles tendon has been widely reported as a common site of overuse injury in triathletes
- A number of intrinsic and extrinsic risk factors have been associated with Achilles tendon injuries
- It has been suggested that there are three main hypotheses for the development of Achilles tendon injury. These are the degeneration hypothesis, the pathological hypothesis, and the failure of the neuro-inhibitory mechanism hypothesis
- MRI and US are both excellent tools for the investigation of the Achilles tendon
- Using US to evaluate the Achilles tendon has the added advantage of being portable, more cost-effective and has an excellent ability to evaluate both the morphology and blood flow (by using CD and PD)
- There is not always is clear relationship between morphological changes in the Achilles tendon and the presence of symptoms
- There is no clear association between blood flow changes in the Achilles tendon and symptoms

- The majority of studies show that there is a relationship between neovascularisation and chronic painful tendinosis (neovessels that were injected with a sclerosing agent resulted in decreased pain in most patients)
- More recent studies show that blood flow in a tendon increases in response to an acute bout of exercise, but this response may not always be pathological – this however requires further investigation

Chapter 3

Achilles tendon morphology and blood flow changes following an Ironman triathlon: A prospective cohort study

3.1. Introduction

The triathlon is an endurance sport consisting of a swim, cycle and run components. Triathletes can participate in a variety of triathlon events, ranging from shorter sprint triathlons to an ultra-endurance triathlon, such as the Ironman triathlon. An Ironman triathlon consists of a 3.8 km swim, a 180 km cycle, followed by a 42.2 km run. The participation in, and more importantly, the preparation and training for this event can predispose triathletes to injuries, in particular, overuse injuries [2;3].

O'Toole et al. [4] have documented that 91% of the triathletes participating in an Ironman triathlon reported at least one soft tissue overuse injury during the previous year's training. In a subsequent study, at least one injury was reported by 75% of the triathletes during their participation in a triathlon [5]. The specific risk factors for injuries in triathletes have however not been well documented. Recently, Egermann et al. [5] reported that injuries in triathlon are

related to age, performance level, weekly training hours and previous history of injury. The most significant factor associated with an injury in triathletes is increasing years of triathlon experience [132].

The anatomical distribution and type of injury in triathletes has, in general, not received much attention. In one study it was documented that 55% of the injuries in triathletes occurred in the lower extremity [5], while in another study 'tendonitis' was reported to be the predominant type of injury [6]. Vleck et al. [7] have reported that injuries of the Achilles tendon, lower back and the knee were the most common injuries in triathletes.

It therefore appears, from the available limited literature, that injuries to the Achilles tendon are common in triathletes. Although the risk factors [25-31] and pathogenesis [27] of Achilles tendon injuries in athletes in general have recently received attention, there are no specific studies documenting factors associated with Achilles tendon injuries in triathletes. In particular, it is not documented what the effects of regular training for a triathlon, as well as the effect of participating in an Ironman triathlon are on the Achilles tendon.

Any study that assesses the Achilles tendon changes requires the use of specialized imaging. In recent years, soft tissue diagnostic US (with CD and PD) and MRI have both been successfully used to assess Achilles tendon morphology [84;85;121;133].

The use of soft tissue diagnostic ultrasound (from here on referred to as "ultrasound" - US) has the following specific advantages over other imaging techniques: 1) US has a high sensitivity and specificity similar to that of MRI [134], 2) US is portable and can be used in field studies, 3) US evaluation of the Achilles tendon can be performed rapidly and is very cost-effective, and 4) US can be used to assess both morphology and blood flow in the tendon and surrounding tissues [135;136].

The importance of measuring blood flow in the Achilles tendon and surrounding tissue has recently received attention. It has been reported that, in general, increased blood flow is not seen in normal resting tendons and seldom in joints [95]. Increased blood flow (Doppler activity) has however been documented in patients with clinical signs of tendinosis [97;137;138]. It is therefore suggested that the presence of intratendinous blood flow may be a sign of pathology in tendons.

However, it has also recently been documented that increased blood flow in tendons and surrounding tissue can occur after an acute bout of exercise. In one study in elite badminton players, increased blood flow in the Achilles tendon was reported in the majority of players following a match, and this was not related to symptoms [121]. This increase in blood flow after repetitive loading was believed to be a physiological hyperemic response rather than a pathological response (Boesen et al. 2006 - In press).

However, the effect of an acute bout of exercise where there is extensive prolonged mechanical loading of the Achilles tendon, such as during the Ironman triathlon, on morphology and blood flow has not been documented. Furthermore, there are no prospective studies that have followed any changes that may occur in Achilles tendon morphology or blood flow immediately after exercise during subsequent weeks. Therefore, it is not known if these changes are transient, and whether they are related to the development of symptoms and signs of Achilles tendon injury.

Therefore, the aims of this study were 1) to determine if the participation in an Ironman triathlon alters Achilles tendon morphology and/or blood flow immediately after the event, 2) whether these changes in morphology and/or blood flow are transient, and 3) if any changes in morphology and/or blood flow are related to the development of symptoms and signs of Achilles tendon injury in triathletes.

3.2. Methods

3.2.1. Type of study

Prospective cohort study.

3.2.2. Subjects

All 1136 triathletes (970 male, 85.4% and 166 female, 14.6%) who entered the 2006 "Spec-savers" Port Elizabeth South African Ironman Triathlon (3.8 km swim, 180 krn cycle and 42.2 km run), which was held during March 2006, were considered as potential subjects. In the 2 months prior to the event, information about the study was posted on to the official race website (Appendix 1). This information included details about the study procedures (Appendix 2), the informed consent form (Appendix 3) and copies of the questionnaires (Appendix 4) to be completed. In addition, a service for triathletes to ask questions about the research by telephone or email was established. Prior to the study, the protocol was approved by the research Ethics Committee of the University of Cape Town (REC ref n° 425/2005) (Appendix 5), as well as the general organizing committee and the medical sub-committee of the 2006 "Spec-savers" Port Elizabeth South African Ironman Triathlon.

Recruitment for the study took place at the registration area in the 3 days prior to the event. A research area was established in close proximity to the registration desk. As triathletes reported at the registration desk, they were informed about the nature of the study, and could then volunteer to take part in the study. Triathletes then reported to the research staff, where further information was given, and any questions were answered. Once triathletes gave written consent to be part of the study, they continued with the

completion of a pre-race questionnaire and a pre-race soft tissue diagnostic US of both Achilles tendons. Of the 1136 triathletes who entered the event, 992 (87.2%) started the race, 304 (26.8%) triathletes complete the pre-race questionnaire, and 109 triathletes (9.6%) consented to undergoing the Achilles tendon diagnostic US investigation.

In addition, triathletes who consented to a pre-race US investigation also agreed to report to the immediate post-race US scanner which was located in the "tunnel" just after the finish line. A subgroup of the triathletes who consented to the pre- and immediate post-race US was also approached to undergo a further US of the Achilles tendon 6-8 weeks after the event. This subgroup was chosen on the basis of their ability to undergo this investigation in Cape Town (South Africa).

3.2.3. Pre-race questionnaires

For the purposes of this study, a previously validated pre-race questionnaire, which consisted of a number of sections, was modified and then used (Appendix 4) [139-141]. The information obtained from this questionnaire was used in a number of studies that were conducted at this event. In this particular study, information from the following sections was used: (a) personal details (including age, height, body weight, gender, ethnic group, country of birth and dominant hand/foot), (b) racing, training and equipment use history, (c) history of medication and supplement use, which included a

section on lifestyle and habits history, (d) family medical history and finally (e) personal general medical history.

The section on racing, training and equipment use history included specific details about previous participation in triathlons, running events and cycling events. Personal best performance times in a number of these events were also obtained. Training details, which included training distances and hours spent training, were obtained for the last 15 weeks and 1 week before the event. All triathletes were also asked to record if they performed stretching exercises and how many times per week and times per day these were performed. The muscle groups that were included in the stretching exercises as well as the duration of holding the stretch for and how many times the muscle is stretched were also included in the questionnaire. In the equipment use history section, the specific brand and type of running shoe was recorded.

In the section on the history of medication and supplement use, subjects were specifically asked to report on the usage of corticosteroids and fluoroquinolone antibiotics. Corticosteroid usage was further divided into oral ingestion or injection, particularly a history of injection in or around the Achilles tendon. Smoking status was also recorded. In the section on family medical history triathletes were asked to report if any of their biological (blood) relatives ever had a chronic Achilles tendon injury, an Achilles tendon rupture or any ligament injuries.

Personal medical information was also obtained on any current or previous tendon (tendinopathy or rupture) or ligament (sprain or complete tear) injuries in any anatomical area. Triathletes were also asked if they ever took any medicines to treat injuries, during their triathlon career, in the week before or during a race – including anti-inflammatory drugs, cortisone (tablets or injection), or analgesics. Finally, triathletes were required to report a history of any connective tissue disorder or known rheumatological disease.

All the triathletes were encouraged to complete the questionnaire whilst remaining in the research area and the majority completed it in this manner. Alternatively, triathletes took the questionnaire with them and returned it the next day or the morning of the race (< 10% of questionnaires were returned this way).

3.2.4. Pre-race Achilles tendon clinical assessment and ultrasound (US)

The triathletes were asked if they were currently experiencing any pain, swelling or stiffness and a clinical examination was performed, assessing for tenderness or swelling (nodular of diffuse) and a shift test (tenderness in the area of the Achilles tendon that moves with plantar- or dorsiflexion) was performed [13].

One experienced musculoskeletal radiologist conducted the soft tissue diagnostic ultrasound investigation of the Achilles tendons in a cohort of 109 triathletes. The method of imaging the Achilles tendon, using ultrasound, has

been described previously [142]. A Toshiba Nemio scanner (Nemio 20, Toshiba Corporation, 1385, Shimoishigami, Otawara-shi,Tochigi-Ken, 324-8550, Japan) and a Toshiba linear probe multifrequency, set on 14-MHz were used.

For the examination, the triathletes were in a prone position with their feet overhanging the examination couch. The ankles were in their naturally relaxed position with the Achilles tendon fairly taut. Both Achilles tendons were scanned from the musculotendinous junction to the calcaneal insertion in both the antero-posterior and transverse diameter. The sonographic probe was placed parallel to the Achilles tendon for the transverse scans to avoid anisotrophy [143].

The tendon shape was classified as either angular or fusiform, and the margin was classified as either sharply or poorly defined. The contour was assessed as smooth, nodular or tapered. The tendon sagittal and transverse diameters in the antero-posterior positions were measured (mm) at the thickest point of the Achilles tendon [85]. For the internal architecture of the tendon, the following abnormalities were noted: (1) hypoechoic foci, (2) disrupted fibres, (3) haematoma's, or (4) calcifications and/or acoustic shadowing. The ultrasonographic diagnosis of Achilles tendinosis in each tendon of the triathletes was made based on the presence of one or more of these abnormalities.

Other abnormal features in the surrounding tissues of the Achilles tendon were also examined. The presence of fluid and any soft tissue swelling in the

paratenon area was documented and will be referred to as 'paratendonitis' [142]. Other characteristics such as Kager's fat pad, presence of fluid in the retrocalcaneal bursa, the myotendinious junction and the insertion on the calcaneus were also recorded to exclude other conditions such as inflammation of the retrocalcaneal bursa (bursitis), formation of bone spurs and triceps surae muscle injuries that may be associated with the musculotendinous junction (Appendix 6) [142]. Collectively, these abnormal features were grouped together and referred to as 'Other abnormal features' or 'Other features' in the results section of this thesis.

Finally, CD was used to detect an increased blood flow in both the tendon substance (intra-tendon), Kager's fat pad and the paratenon. Blood flow was only documented as being either 'present' or 'absent' in the tendon or surrounding tissues. The reasons for documenting blood flow in this manner, rather than quantifying blood flow are as follows: Firstly, there was limited time available to conduct the US examination of each triathlete, and secondly, at the time of finalizing the protocol of this study there were no well established standardized methods of quantifying blood flow in the Achilles tendon using CD ultrasound.

3.2.5. Immediate post-race clinical assessment and Achilles tendon US

An immediate post-race clinical assessment and US of the Achilles tendon was performed on 81 of the 109 triathletes (74% of the original cohort). Of the 18 triathletes who did not undergo immediate post-race US, 6 did not finish

the race and 12 did not report to the immediate post-race research area which was located in the "tunnel" within 100 m from the finish line. All the race finishers walked through this area on their way to the recovery area. At this time, triathletes were also asked to report any pain, swelling or stiffness they may have experienced during or immediately after the race. A brief clinical examination was performed at that time to note any tenderness or swelling (nodular or diffuse) in the Achilles tendon. A shift test was performed in those triathletes who did have areas of tenderness in the Achilles tendon. The same radiologist then performed all the immediate post-race scans using the same US scanner, and the same technique was used.

3.2.6. Six to 8 weeks post-race Achilles tendon US

A 6-8 weeks post-race US of the Achilles tendon was conducted in a subgroup of triathletes 6-8 weeks after the race. At the time of registration, 44 of the 109 triathletes (40%) were eligible for the 6-8 weeks US investigation (based on the fact that they were from the geographical area near Cape Town). The reason for choosing the above demographic area was due to the location of the same radiologist and a similar US scanner in that region. All the triathletes in the original cohort were eligible for the 6-8 weeks post-race ultrasound investigation gave consent, provided they were able to come to the venue 6-8 weeks after the race. Following the race, the 44 triathletes in this subgroup were contacted telephonically and asked to report for the follow-up US.

Of this subgroup, 29 (out of 44 eligible subjects) triathletes (26.6% of the original cohort of 109) agreed and subsequently underwent the 6-8 weeks post-race US 6-8 weeks after the race. During this visit, triathletes were again asked about any pain, swelling or stiffness in the 6-8 weeks after the race. A brief clinical examination for the presence of tenderness, swelling (nodular/diffuse) was repeated. The shift test was performed in those triathletes where there was tenderness. Triathletes with symptoms were also asked about treatment during the 6-8 week post-race period (including ice, stretching, physiotherapy, eccentric training or the use of medication). Finally, aspects relating to training history and races performed in the 6-8 weeks after the Ironman triathlon were recorded.

The Achilles tendon US examination was then repeated by the same radiologist, using the same machine and the same protocol (Appendix 7).

3.2.7. Environmental conditions on race day

Data on the weather conditions on race day were obtained from the South African Weather Service. The average temperature during the race was 20°C (maximum temperature of 21°C, minimum temperature of 19°C). The average relative humidity during the race was 70%, the average wind speed was 37 km/h, and the sea water temperature was 19.2°C.

3.2.8. Statistical analysis of data

Data were analysed using the Statistica 7.0 (Stat-soft Inc, Tulsa, Oklahoma, USA) and GraphPad InStat 2.05a (GraphPad Software, San Diego, California, USA) statistical programs. Data are presented as a mean ± standard deviation or as a frequency (%). The number of subjects or observations (n) is usually in parenthesis. A one way analysis of variance, Peason's chi-square (χ^2) analysis or a Fisher's exact test was used to determine any significant differences between groups. Where the overall F value was significant, a Turkey's honest significant difference post hoc test was used to identify where the differences were. Statistical significance was accepted when $p<0.05$.

3.3. Results

3.3.1. Subject characteristics

One hundred and nine (94 males and 15 females) triathletes were recruited for this study of which 96 (88.1%) (82 males and 14 females) completed the pre-race questionnaires. The general characteristics, as well as the overall and split times of the triathletes are presented in Table 3.1. Two of the triathletes did not start the race, while an additional 2 did not finish the race. As expected the male triathletes were on average significantly taller and heavier, with a corresponding higher BMI, than the female triathletes. The male and female athletes were however, similarly matched for age, dominant

hand and leg, ethnicity, country of birth and smoking status. Eight-two percent and 77% of the triathletes were right handed and legged, respectively. Only one (1.0%) triathlete reported that he was ambidextrous with his hands, while 7 (7.8%) triathletes reported that they were ambidextrous with their feet. Ninety-five percent of the triathletes reported that they were Caucasian and 76 % of the triathletes were born in South Africa. Twenty-seven percent of the triathletes were either current or ex-smokers. Only 3 of these triathletes reported that they were smoking at the time of the race.

Except for the cycle split, where the male triathletes were on average significantly faster than the female triathletes, there were no significant differences between the swim, run and overall times of the male and female triathletes.

The triathletes who participated in this study were significantly slower for the swim leg (90 ± 14 min, range 63 - 142 vs. 86 ± 16 min, range 54 - 146; p=0.007) and the overall (792 ± 80 min, range 624 - 1021 vs. 772 ± 97 min, range 516 - 1021; p=0.039) times of the triathlon when compared to the entire field of 947 competitors who completed the race. There was however no significant differences in the cycle (407 ± 36 min, range 338 - 524) nor run (295 ± 46 min, range 199 – 434) leg times between the study participants and the entire field (cycle: 401 ± 44 min, range 282 - 529; p=0.176; run: 286 ± 50 min, range 168 - 481; p=0.070). There were also no significant differences in the gender (p=0.921) or age-group categories (p=0.582) distributions between the groups (data not shown).

Table 3.1: The general characteristics, demographics, and race performance data of all the triathletes (male and female)

	All (n=109)	Male (n=94)	Female (n=15)	p-value [a]
Age (years)	37.8 ± 8.8 (91)	38.3 ± 9.0 (77)	35.4 ± 7.4 (14)	0.266
Height (cm)	178.7 ± 8.0 (91)	180.4 ± 6.3 (79)	167.1 ± 8.8 (12)	<0.001
Weight (kg) [b]	75.4 ± 10.7 (91)	77.8 ± 9.5 (79)	61.1 ± 4.1 (12)	<0.001
BMI (kg/m^2) [c]	23.5 ± 2.4 (90)	23.8 ± 2.3 (78)	22.1 ± 2.9 (12)	0.022
Dominant Hand (% right) [d]	82.3 (96)	81.7 (82)	85.7 (14)	0.883
Dominant Leg (% right) [e]	76.7 (90)	77.9 (77)	69.2 (13)	0.718
Ethnic Group (% white) [f]	94.8 (96)	95.1 (78)	92.9 (13)	0.769
Country of Birth (% South Africa) [g]	76.1 (92)	74.7 (79)	84.6 (13)	0.669
Current and Ex-Smokers (%) [h]	27.1 (96)	29.2 (82)	14.2 (14)	0.401
Swim Time (min)	90 ± 14 (107)	90 ± 14 (92)	89 ± 14 (15)	0.813
Cycle Time (min)	408 ± 37 (106)	405 ± 35 (91)	427 ± 41 (15)	0.028
Run Time (min)	295 ± 46 (105)	294 ± 47 (90)	304 ± 38 (15)	0.431
Overall Time (min)	792 ± 80 (105)	788 ± 80 (90)	820 ± 80 (15)	0.152
Finished Ironman Triathlon (%) [i]	96.3 (109)	95.7 (94)	100.0 (15)	0.718
Completed Questionnaires (%)	88.1 (109)	87.2 (94)	93.3 (15)	0.499

Values are expressed as either an average ± standard deviation or a frequency. The number of subjects (n) is in parentheses.

[a] Male vs. female triathletes.

[b] Weight is the athletes self-reported normal body weight.

[c] Body mass index (BMI) is calculated as weight (kg) divided by height (m) squared.

[d] 1 male triathlete is ambidextrous.

[e] 6 male and 1 female triathletes are ambidextrous.

[f] 4 of mixed ancestry and 1 Indian.

[g] 6 Zimbabwe, 4 Namibia, 2 Germany, 5 United Kingdom, 1 Ireland, 1 Italy, 1 New Zealand and 2 USA.

[h] 3 current smokers. The current and ex-smokers smoked on average 13 ± 8 cigarettes per day for 10.3 ± 6.2 years. The ex-smokers stopped smoking on average 9.6 ± 6.6 years ago.

[i] 2 male triathletes did not start and 2 male triathletes did not finish the event.

3.3.2. Performance and training history

The personal best times of the triathletes for the various Ironman and road running disciplines, as well as, their best times achieved in (a) triathlons during the last 12 months, (b) road running during the last 15 weeks and (c) a cycling race over 80 km during the last 15 weeks prior to the triathlons are presented in Table 3.2.

The triathletes have completed on average 7.8 ± 7.1 (n=58), ranging from 1 to 36, and 2.8 ± 4.0 (n=44), ranging from 1 to 23, standard and Ironman triathlons, respectively. On average the triathletes (n=55) first competed in an Ironman triathlon during 2003, ranging from 1984 to 2005. During the last 2 years, the triathletes participated in, on average, 4.1 ± 4.0 (n=67), ranging from 0 to 20, standard triathlons. In addition, the triathletes completed on average 23.1 ± 34.8 (n=69), ranging from 1 to 250, half and 14.9 ± 22.2 (n=62), ranging from 1 to 150, standard marathons starting on average during 1995.

The training frequency of the athletes during the 15 weeks prior to the triathlon, as well the swimming, cycling, running and total training distance and duration during the 15 week period and the 1 week before the Ironman Triathlon is presented in Table 3.3.

Table 3.2: Triathlon (sprint, standard, half-Ironman and Ironman) and road running (5 km, 10 km, 21.1 km and 42.2 km) personal best times (PB), as well as, triathlon, running and cycling, best times achieved over the last 12 months for 15 weeks of all the triathletes (male and female)

	All (n=96)	Male (n=82)	Female (n=14)	p-value [a]
Sprint PB (min)	73 ± 15 (54)	73 ± 15 (47)	76 ± 8 (7)	0.624
Standard PB (min)	144 ± 25 (52)	145 ± 26 (47)	136 ± 14 (5)	0.460
Half Ironman PB (min)	330 ± 36 (54)	329 ± 38 (45)	335 ± 26 (9)	0.661
Ironman PB (min)	751 ± 81 (39)	755 ± 84 (35)	719 ± 52 (4)	0.413
Sprint 12 Months PB (min)	76 ± 18 (37)	76 ± 18 (35)	73 ± 10 (2)	0.836
Standard12 Months PB (min)	151 ± 28 (41)	151 ± 29 (35)	152 ± 21 (6)	0.960
Half Ironman 12 Months PB (min)	338 ± 42 (39)	340 ± 43 (33)	332 ± 31 (6)	0.683
Ironman 12 Months PB (min)	763 ± 76 (33)	767 ± 78 (29)	741 ± 66 (4)	0.528
5 km PB (min)	19 ± 2 (56)	19 ± 2 (46)	21 ± 2 (10)	0.015
10 km PB (min)	42 ± 6 (72)	41 ± 6 (61)	47 ± 6 (11)	0.009
21.1 km PB (min)	95 ± 13 (76)	93 ± 12 (64)	105 ± 12 (12)	0.002
42.2 km PB (min)	216 ± 29 (69)	213 ± 29 (58)	230 ± 24 (11)	0.092
5 km 15 weeks PB (min)	21 ± 3 (26)	21 ± 3 (24)	23 ± 3 (2)	0.307
10 km 15 weeks PB (min)	45 ± 6 (37)	45 ± 5 (30)	49 ± 5 (7)	0.042
21.1 km 15 weeks PB (min)	103 ± 13 (48)	102 ± 13 (39)	109 ± 10 (9)	0.118
42.2 km 15 weeks PB (min)	226 ± 30 (29)	227 ± 28 (26)	221 ± 49 (3)	0.750
Cycle 15 weeks PB speed (km/hr) [b]	32.1 ± 4.3 (78)	32.2 ± 4.3 (68)	29.8 ± 3.0 (10)	0.070
Cycle 15 weeks PB distance (km) [b]	109 ± 26 (78)	108 ± 24 (68)	113 ± 40 (10)	0.581

[a] Male vs. female
[b] The best time achieved in a cycle race over 80 km during the 15 weeks before the triathlon.

Table 3.3: The swimming, cycling, running and/or total training frequency, distances and durations for the 1 week and 15 weeks period before the Ironman of all the triathletes (male and female)

	All (n=96)	Male (n=82)	Female (n=14)	p-value [a]
Training Frequency (days/wk)	5.8 ± 0.8 (92)	5.8 ± 0.8 (79)	6.2 ± 0.7 (13)	0.117
Swim Time 15 wk (min/wk)	165 ± 69 (91)	161 ± 67 (79)	188 ± 80 (12)	0.214
Cycle Time 15 wk (min/wk)	545 ± 171 (82)	548 ± 178 (72)	525 ± 121 (10)	0.695
Run Time 15 wk (min/wk)	297 ± 111 (83)	293 ±113 (72)	325 ± 101 (11)	0.386
Total Time 15 wk (min/wk)	1000 ± 277 (83)	993 ± 284 (72)	1045 ± 231 (11)	0.565
Swim Dist 15 wk (km/wk)	6.5 ± 2.9 (92)	6.2 ± 2.6 (78)	8.4 ± 3.8 (14)	0.007
Cycle Dist 15 wk (km/wk)	246 ± 77 (81)	249 ± 80 (70)	225 ± 57 (11)	0.351
Run Dist 15 wk (km/wk)	49 ± 17 (87)	49 ± 17 (75)	49 ± 14 (12)	0.883
Total Dist 15 wk (km/wk)	297 ± 91 (87)	301 ± 95 (75)	277 ± 63 (12)	0.407
Swim Time 1 wk (min)	62 ± 54 (91)	62 ± 56 (79)	61 ± 34 (12)	0.967
Cycle Time 1 wk (min)	149 ± 112 (92)	153 ± 117 (79)	127 ± 72 (13)	0.444
Run Time 1 wk (min)	67 ± 60 (90)	68 ± 62 (77)	60 ± 39 (13)	0.669
Total Time 1 wk (min)	284 ± 188 (89)	287 ± 198 (77)	264 ± 105 (12)	0.694
Swim Dist 1 wk (km)	2.4 ± 2.0 (91)	2.3 ± 2.0 (78)	3.0 ± 2.4 (13)	0.266
Cycle Dist 1 wk (km)	65 ± 47 (91)	68 ± 49 (78)	48 ± 28 (13)	0.151
Run Dist 1 wk (km)	11 ± 10 (87)	11 ± 10 (74)	11 ± 7 (13)	0.921
Total Dist 1 wk (km)	79 ± 54 (87)	82 ± 57 (74)	62 ± 30 (13)	0.212

Values are expressed as average ± standard deviation, with the number of subjects (n) in parentheses.
[a] Male vs. female
wk, week; Dist, distance.

3.3.3. History of tendon and ligament injuries

Forty percent (n=38) of the triathletes reported a lifelong history of tendon and/or ligament injuries, of which 37.5 % (n=36) and 21.9 % (n=21) were a

history of tendon and ligament injuries, respectively (Table 3.4). Twenty-six of the tendon injuries were chronic, 4 were acute and 5 were unknown. There were 8 athletes (8%) who reported a history of multiple tendon injuries and most of the triathletes (*n*=18) reported a history of Achilles tendon injuries. The types and numbers of tendon injuries are summarized in Figure 3.1A. Thirteen of the reported ligament injuries were sprains, 6 were complete tears and 2 were unknown. Six athletes reported a history of multiple ligament injuries (Table 3.4). The types and numbers of ligament injuries are summarized in Figure 3.1B.

There were only 5 athletes who reported a history of joint capsule injuries of which 3 included acute shoulder dislocations and 2 were chronic instability of the shoulder (Table 3.4). There were no significant differences between male and female athletes regarding history of previous tendon and ligament injuries. Of the females, 2 (2%) reported a history of connective or rheumatological disease, whilst none of the men reported such a history. This included 1 with rheumatoid and 1 with reactive arthritis.

Five percent of the 95 athletes reported a family history of chronic Achilles tendon injuries and 1% gave a family history of chronic Achilles tendon rupture. There were 22 athletes (23%) with a family history of ligament injuries (Table 3.4). There were no significant differences in the history of tendon and ligament injuries between the male and the female triathletes.

Table 3.4: Personal and family history of tendon and ligament injuries in all of the triathletes (male and female)

	All (n=96)	Male (n=82)	Female (n=14)	p-value [a]
History of Tendon and/or Ligament Injuries (%)	39.6 (96)	37.8 (82)	50.0 (14)	0.571
All Tendon Injuries (%)	37.5 (96) [b]	35.3 (82)	50.0 (14)	0.455
Multiple Tendon Injuries (%)	8.3 (96)	8.5 (82)	7.1 (14)	0.727
Achilles Tendon Injuries (%)	18.8 (96)	19.5 (82)	14.3 (14)	0.846
All Ligament Injuries (%)	21.9 (96) [c]	20.7 (82)	28.6 (14)	0.760
Multiple Ligament Injuries (%)	6.3 (96)	6.1 (82)	7.1 (14)	0.727
Joint Capsule Injuries (%)	5.2 (96) [d]	3.7 (82)	14.3 (14)	0.316
Connective Tissue or Rheumatological Disease (%)	2.1 (96) [e]	0.0 (82)	14.3 (14)	0.014
Family History of Chronic Achilles Tendon Injuries (%)	5.3 (95)	6.2 (81)	0.0 (14)	0.759
Family History of Chronic Achilles Tendon Ruptures (%)	1.1 (95)	1.2 (81)	0.0 (14)	0.317
Family History of Ligament Injuries (%)	23.4 (95)	23.5 (81)	23.1 (14)	0.747

Values are expressed as a frequency, the number of subjects (n) in parentheses.
[a] Male vs. female
[b] 26 longstanding, 4 acute and 5 unknown tendon injuries
[c] 13 sprains, 6 complete tears and 2 unknown ligament injuries
[d] 3 acute shoulder dislocation and 2 chronic instability
[e] 1 rheumatoid and 1 reactive arthritis

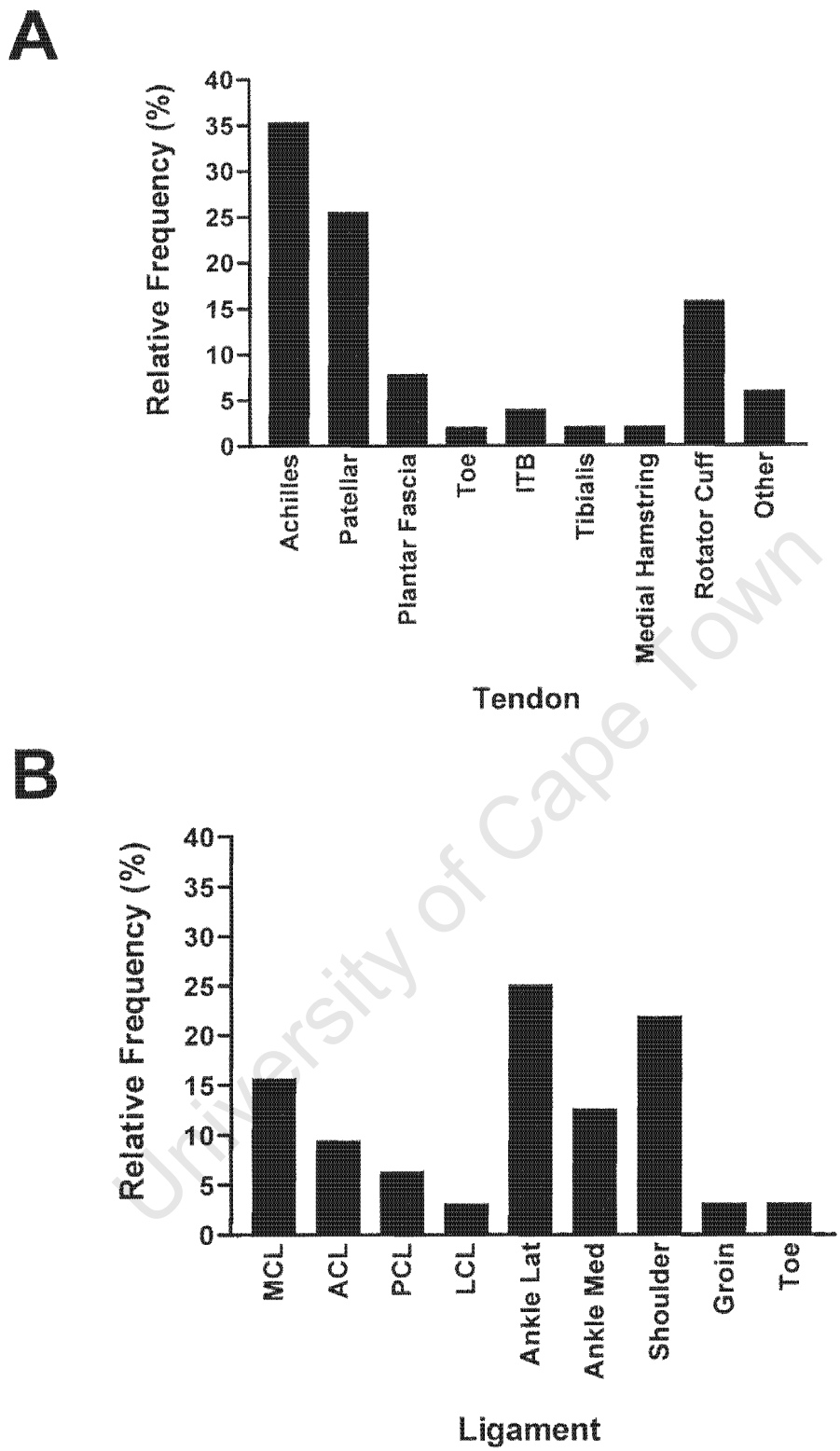

Figure 3.1. The relative number of triathletes who reported injuring the specific (A) tendon and/or (B) ligament

ITB, iliotibial band; MCL, medial collateral ligament; ACL, anterior cruciate ligament; PCL, posterior cruciate ligament; LCL, lateral collateral ligament; Lat, Lateral; Med, Medial.

3.3.4. History of medication and current running shoe use

As shown in Table 3.5, 9 (9.6%) and 24 (25.8%) of the triathletes reported previous oral corticosteroid intake or a previous history of a corticosteroid injection, respectively. Only 2 of the triathletes reported having a single injection in their Achilles tendon. None of the athletes reported ever having more than one injection of corticosteroids in the Achilles tendon.

Table 3.5: History of corticosteroids, fluoroquinoline antibiotics and analgesics or anti-inflammatory medication usage by all the triathletes (male and female)

	All (n=96)	Male (n=82)	Female (n=14)	p-value [a]
Oral Corticosteroids (%)	9.6 (94)	8.6 (81)	15.4 (13)	0.795
Corticosteroid Injection (%)	25.8 (93)	22.2 (81)	50.0 (12)	0.089
Single Corticosteroid Achilles Tendon Injection (%) [b]	2.1 (94)	1.2 (81)	7.7 (13)	0.644
Flouroquinoline Antibiotics (%)	23.1 (91)	23.1 (78)	23.1 (13)	0.722
Analgesic / Anti-inflammatory Medication Pre-1 Week (%) [c]	31.3 (96)	28.1 (82)	50.0 (14)	0.185
Analgesic / Anti-inflammatory Medication during Race (%) [d]	19.8 (96)	18.3 (82)	28.6 (14)	0.597

Values are expressed as a frequency (%) with the number of subjects (n) in parentheses.
[a] Male vs. female triathletes.
[b] None of the triathletes reported ever having 2 or more corticosteroid injections in their Achilles Tendon.
[c] 15 painkillers, 20 NSAIDS, 13 anti-inflammatory gels and 3 cortisone injections.
[d] 15 painkillers, 8 NSAIDS and 4 anti-inflammatory gels.

Twenty-three percent (n=21) of the triathletes reported previous use of fluoroquinoline antibiotics. Analgesics or anti-inflammatory medication use, in the 1 week prior to the race, was reported in 30 (31.3%) of the triathletes. This

included 15 athletes taking painkillers, 20 using NSAIDS, 13 using anti-inflammatory gels and 3 cortisone injections. Nineteen (19.8%) of the triathletes reported taking analgesics or anti-inflammatory medication, which included 15 painkillers, 8 NSAIDS and 4 anti-inflammatory gels, during a race.

There was no significant difference in the history of corticosteroids, fluoroquinoline antibiotics and analgesics or anti-inflammatory medication usage between the male and female triathletes.

The current running shoe types used by the triathletes are summarized in Table 3.6.

Table 3.6: Current running shoe use by all the triathletes (male and female)

	All (n=94)	Male (n=81)	Female (n=13)	p-value [a]
Neutral (%)	50 (53.2)	44 (54.3)	6 (46.2)	0.067
Motion Control or Anti-pronation (%)	31 (33.0)	28 (34.6)	3 (23.1)	
Light Racing (%)	6 (6.4)	3 (3.7)	3 (23.1)	
Unknown (%)	7 (7.4)	6 (7.4)	1 (7.7)	

Values are expressed as the number of observations (n) with the frequency (%) in parentheses.
[a] Male vs. female triathletes.

3.3.5. Flexibility training

The history of flexibility training by the triathletes is summarized in Table 3.7. There was no significant difference in the flexibility training between the male

and female triathletes. The triathletes that stretch reported stretching the following muscle groups: soleus (n=44), gastrocnemius (n=49), groin (n=33), hamstring (n=50), quadriceps (n=49), upper body (29) and other muscle groups (n=20).

Table 3.7: Flexibility training history of all the triathletes (male and female)

	All (n=95)	Male (n=82)	Female (n=13)	p-value [a]
Stretch Training (% yes)	52.6 (95)	48.8 (82)	76.9 (13)	0.112
Frequency (days/wk)	3.9 ± 1.7 (51)	4.1 ± 1.6 (41)	3.1 ± 1.9 (10)	0.076
Frequency (times/day)	1.2 ± 0.5 (50)	1.1 ± 0.5 (40)	1.4 ± 0.6 (10)	0.207
Duration (sec/stretch)	26 ± 12 (50)	27 ± 12 (40)	24 ± 13 (10)	0.448
No. of Times each Muscle Group Stretched	2.2 ± 1.1 (53)	2.3 ± 1.2 (43)	1.9 ± 0.6 (10)	0.278
Total Duration (min/wk) [b]	5.1 ± 5.2 (46)	5.4 ± 5.4 (37)	3.8 ± 4.7 (9)	0.415
Stretch before Exercise (%)	46.2 (52)	45.2 (42)	50.0 (10)	0.935
Stretch during Exercise (%)	27.5 (51)	26.8 (41)	30.0 (10)	0.846
Stretch after Exercise (%)	94.1 (51)	92.7 (41)	100.0 (10)	0.895

Values are expressed as a frequency (%) or as a mean ± standard deviation. The number of subjects is in parenthesis.
[a] Male vs. female triathletes.
[b] The total weekly stretching time (min/week) was calculated as duration of stretch (min) X number of times each muscle group was stretched per day (number)X stretch sessions per day (number) X days of stretching per week (number)

3.3.6. Clinical assessment (history and examination) of the Achilles tendons

The pre- and immediate post-race clinical assessment (history and examination) of the Achilles tendons is presented in Table 3.8. In the pre-race

clinical examination, 217 Achilles tendons were examined; of which 109 was of the left tendon and 108 of the right tendon (one triathlete was an amputee). Tendons having any symptoms and/or signs (tenderness, swelling, positive shift test), included a total of 42 (19.4%), 27 (12.4%) tendons had symptoms while 30 (13.8%) tendons had at least one of the clinical signs. Of these, 23 (10.6%) of the tendons had tenderness, 16 (7.4%) had diffuse or nodular swelling and 5 (2.3%) and a positive shift test was only found in 5 (2.3%) of the tendons.

In the immediate post-race clinical assessment, a total of 181 Achilles tendons were examined, which included 91 of the left and 90 of the right tendon. At least one of the symptoms and/or signs were present in 37 (20.4%) of the tendons, Twenty seven (14.9%) tendons had symptoms and 28 (15.5%) of the 181 tendons presented with at least one of the clinical signs of Achilles tendon injury. Twenty-four tendons (13.3%) had tenderness, while swelling was documented in 16 (8.8%) of the tendons. A positive shift test was found in 7 (3.9%) of the tendons.

There were no significant differences in the distributions of the various individual or combinations of the symptoms and signs when the pre-race assessments were compared to the immediate post-race assessments (Table 3.8).

Table 3.8: Pre- and immediate post-race clinical assessment (history and examination) of the Achilles tendons in the triathletes

	Left	Right [a]	Tendons	Unilateral	Bilateral	p-value [b]	p-value [c]
Pre-race:-	n=109	n=108	n=217	n=109	n=109		
Symptoms / Signs (%)	25 (22.9)	17 (15.7)	42 (19.4)	18 (16.5)	12 (11.0)	0.229	-
Symptoms (%)	17 (15.6)	10 (9.3)	27 (12.4)	11 (10.1)	8 (7.3)	0.227	-
Signs (%)	16 (14.7)	14 (13.0)	30 (13.8)	16 (14.7)	7 (6.4)	0.845	-
Tenderness (%)	11 (10.1)	12 (11.1)	23 (10.6)	13 (11.9)	5 (4.6)	0.981	-
Diffuse or Nodular Swelling (%)	10 (9.2)	6 (5.6)	16 (7.4)	10 (9.2)	3 (2.8)	0.437	-
Shift Test (%)	4 (3.7)	1 (1.0)	5 (2.3)	3 (2.8)	1 (1.0)	0.371	-
Immediately Post-Race:-	n=91	n=90	n=181	n=91	n=91		
Symptoms / Signs (%)	19 (17.4)	18 (20.0)	37 (20.4)	17 (18.7)	10 (11.0)	1.000	0.802
Symptoms (%)	12 (13.2)	15 (16.7)	27 (14.9)	13 (14.3)	7 (7.7)	0.538	0.557
Signs (%)	16 (17.6)	12 (13.3)	28 (15.5)	14 (15.4)	7 (7.7)	0.538	0.671
Tenderness (%)	13 (14.2)	11 (12.2)	24 (13.3)	10 (11.0)	7 (7.7)	0.827	0.438
Diffuse or Nodular Swelling (%)	9 (9.9)	7 (7.8)	16 (8.8)	6 (6.6)	5 (5.5)	0.794	0.712
Shift Test (%)	4 (4.4)	3 (3.3)	7 (3.9)	3 (3.3)	2 (2.2)	1.000	0.393

Values are expressed as the number of observations (n) with the frequency (%) in parentheses.
[a] 1 triathlete was an amputee.
[b] Left vs. right Achilles tendon.
[c] Pre- vs. immediate post-race clinical assessments of all (left and right) the tendons.

In Table 3.9, the pre-, immediate post- and 6-8 weeks post-race clinical assessment (history and examination) of the Achilles tendons is presented. A total of 58 tendons were assessed, both pre- and 6-8 weeks post-race, while only 56 tendons were assessed immediately post-race, because 1 athlete (2

tendons) did not have an immediate post-race clinical examination or ultrasound.

As shown in Table 3.9, 6-8 weeks post-race there were significantly more 1) tendons with any symptoms and/or signs (p value for linear trend=0.031), 2) tendons with any symptoms (p value for linear trend=0.014), and 3) tendons with difffuse or nodular swelling (p value for linear trend=0.047), compared with pre-race or immediately post-race. The prevalence of 1) any clinical signs (p value for linear trend=0.280), 2) tenderness (p value for linear trend=0.613) or 3) positive shift test (data not shown) during the clinical assessment were not different between pre-race, immediately post-race and 6-8 weeks post-race.

Table 3.9: Pre-, immediate post- and 6-8 weeks post-race clinical assessment (history and examination) of the Achilles tendons in 29 triathletes

	Pre-Race (n=58)	Immediately Post-Race (n=56) a	6-8 Weeks Post-Race (n=58)	p-value	p-value linear trend
Symptoms / Signs (%)	16 (27.6)	16 (28.6)	27 (46.6)	0.054	0.031
Symptoms (%)	12 (20.7)	14 (25.0)	24 (41.4)	0.035	0.014
Signs (%)	13 (22.4)	11 (19.6)	18 (31.0)	0.334	0.280
Tenderness (%)	10 (17.2)	10 (17.9)	8 (13.8)	0.844	0.613
Diffuse or Nodular Swelling (%)	8 (13.8)	5 (8.9)	16 (27.6)	0.022	0.047

Values are expressed as the number of observations (n) with the frequency (%) in parentheses.
[a] 1 athlete (2 tendons) did not have a post-race clinical examination or ultrasound.

Five tendons, had pre-, immediately post- and 6-8 week post-race symptoms. Three tendons had only pre- and immediately post-race symptoms. Four tendons had only immediately post- and 6-8 week post-race symptoms. There

were no tendons with only pre-race symptoms and 2 tendons had only immediate post-race symptoms. Symptoms were reported only during the 6-8 weeks post-race clinical assessment in 11 tendons. There were 29 tendons from 19 triathletes with no symptoms during any of the clinical pre- and post-race assessments.

Regarding signs, 5 tendons presented with at least one of the clinical signs during all three examinations. Five tendons had only pre- and immediately post-race signs. One tendon had immediately post- and 6-8 week post-race signs. Three tendons had only pre-race signs. There were no tendons with only immediate post-race signs and 12 tendons with only 6-8 week post-race signs. Twenty-six tendons from 12 athletes had no signs during all the clinical examinations.

3.3.7. Ultrasound examination findings

The pre-race US findings of a total of 217 tendons, including 109 and108 of the left and right tendons respectively, are presented in Figure 3.2. and Table 3.10. The ultrasonographic diagnosis of Achilles tendinosis in each tendon of the triathletes was made based on the presence of one or more of the following abnormalities: (1) hypoechoic foci, (2) disrupted fibres, (3) haematoma's, or (4) calcifications and/or acoustic shadowing. Blood flow in the tendon, paratenon and Kager's fat pad was documented, and other morphological features including soft tissue swelling, paratenon fluid, fluid

filled bursa, hyperechogenic Kager's fat pad, abnormal myotendinous junction and an abnormal calcaneus were grouped as 'other features'.

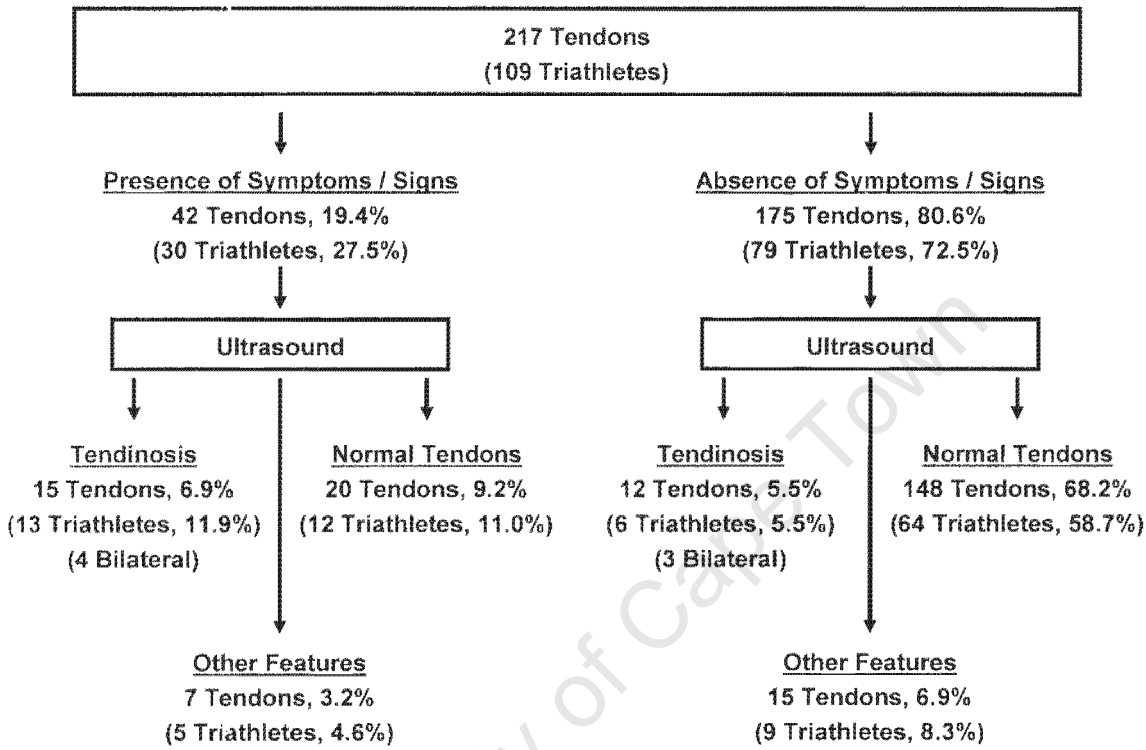

Figure 3.2. Pre-race clinical and ultrasound findings in the triathletes

Table 3.10: Pre-race ultrasound abnormal morphological, blood flow, and other abnormal features in the Achilles tendons of the triathletes

	Left (n=109)	Right [a] (n=108)	Unilateral (n=109)	Bilateral (n=109)	p-value [b]
Morphological Features:-					
Any Morphological Features (%)	15 (13.8)	15 (13.9)	12 (11.0)	9 (8.3)	1.000
Fusiform Shape (%)	13 (11.9)	9 (8.3)	10 (9.2)	6 (5.5)	0.515
Poorly Defined Margin (%)	7 (6.4)	7 (6.5)	4 (3.7)	5 (4.8)	0.796
Nodular or Tapered Contour (%)	12 (11.1)	8 (7.5)	10 (9.2)	5 (4.8)	0.482
Internal Architecture (%)	13 (11.9)	14 (12.9)	11 (10.1)	8 (7.3)	0.840
Blood Flow:-					
Any Blood Flow (%)	4 (3.7)	2 (1.9)	2 (1.8)	2 (1.8)	0.683
Intra-Tendon Blood Flow (%)	2 (1.8)	0 (0.0)	2 (1.8)	0 (0.0)	0.482
Paratenon Blood Flow (%)	3 (2.8)	2 (1.9)	1 (0.9)	2 (1.8)	0.992
Kager's Fat Pad Blood Flow (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Other Features:-					
Any Other Features (%)	15 (13.8)	18 (16.7)	11 (10.1)	11 (10.1)	0.576
Soft Tissue Swelling (%)	1 (0.9)	1 (0.9)	0 (0.0)	1 (0.9)	0.482
Paratenon Fluid (%)	1 (0.9)	0 (0.0)	1 (0.9)	0 (0.0)	0.996
Fluid Filled Bursa (%)	4 (3.7)	6 (5.6)	6 (5.5)	2 (1.8)	0.735
Hyperechogenic Kager's Fat Pad (%)	6 (5.5)	5 (4.6)	5 (4.8)	3 (2.8)	0.988
Abnormal Myotendenous Junction (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Abnormal Calcaneus (%)	6 (5.5)	7 (6.5)	3 (2.8)	5 (4.5)	0.986

Values are expressed as the number of observations (n) with the frequency (%) in parentheses.
[a] 1 triathlete was an amputee.
[b] Left vs. right Achilles tendon.

As summarized in Table 3.11, the triathletes diagnosed with either uni- (n=12) or bilateral (n=7) pre-race tendinosis were 1) older, 2) generally reported better personal best times for the various road running events during their running careers, 3) reported a higher incidence of a history of any tendon, or more specifically Achilles tendon injury, 4) increased previous use of oral

corticosteroids compared with the triathletes with normal bilateral Achilles tendons or those with any other abnormal feature. There were no significant differences in the performance times during the triathlon between these three groups of triathletes. In addition, there were no other significant differences in the general characteristics, training, history of ligament injuries, other medication usage or current running shoe usage between these three groups (data not shown).

Table 3.11: General characteristics, performance, tendon and ligament injury history, as well as, oral corticosteroids usage of the triathletes with uni- or bilateral tendinosis, any other uni- or bilateral abnormal feature and bilateral normal tendons and surrounding tissue during the pre-race ultrasound examination of the Achilles tendon

	Tendinosis (n=19)	Other Abnormal Features (n=14)	Normal Tendons (n=76)	p-value	p-value [d]
Age (years)	45.1 ± 8.5 (15)	38.3 ± 8.7 (12)	36.1 ± 8.1 (64)	[a]<0.001 [b]0.089 [c]0.682	n.d.
5 km PB (min)	17 ± 2 (8)	20 ± 4 (4)	20 ± 2 (44)	[a]0.009 [b]0.061 [c]0.897	n.d.
10 km PB (min)	39 ± 5 (12)	47 ± 9 (6)	42 ± 6 (54)	[a]0.215 [b]0.042 [c]0.246	n.d.
21.1 km PB (min)	87 ± 13 (13)	102 ± 17 (9)	96 ± 11 (54)	[a]0.057 [b]0.015 [c]0.328	n.d.
42.2 km PB (min)	203 ± 22 (14)	218 ± 23 (8)	220 ± 31 (47)	0.154	n.d.
Swim Time (min)	93 ± 13 (19)	89 ± 15 (13)	89 ± 14 (75)	0.539	0.980
Cycle Time (min)	422 ± 43 (19)	398 ± 26 (13)	406 ± 36 (74)	0.142	0.500
Run Time (min)	292 ± 47 (18)	282 ± 51 (13)	298 ± 45 (74)	0.479	0.172
Overall Time (min)	804 ± 88 (18)	770 ± 77 (13)	793 ± 80 (74)	0.499	0.240
Any Tendon Injury (%)	60.0 (15)	8.3 (12)	37.7 (69)	0.022	n.d.
Achilles Tendon Injury (%)	53.3 (15)	8.3 (12)	13.0 (69)	<0.001	n.d.
Any Ligament Injury (%)	26.7 (15)	8.3 (12)	23.2 (69)	0.459	n.d.
Oral Corticosteroids (%)	33.3 (15)	8.3 (12)	4.5 (67)	0.003	n.d.

Values are expressed as either an average ± standard deviation or a frequency. The number of subjects (n) is in parentheses.
[a] Tendinosis vs. normal tendons and surrounding tissue.
[b] Tendinosis vs. other abnormal features.
[c] Other abnormal features vs. normal tendons and surrounding tissue.
[d] Co-varied for age.
n.d., not determined; PB, personal best time.

The immediate post-race ultrasound data of a total of 181 tendons (83.4% of the pre-race tendons), including 91 and 90 of the left and right tendons respectively, are presented in Table 3.12.

Table 3.12: Immediate post-race ultrasound abnormal morphological features, blood flow and other abnormal features in the triathletes' Achilles tendons

	Left (n=91)	Right [a] (n=90)	Unilateral (n=91)	Bilateral (n=91)	p-value [b]
Morphological Features:-					
Any Morphological Features (%)	11 (12.1)	14 (15.6)	13 (14.3)	6 (6.6)	0.526
Fusiform Shape (%)	9 (9.9)	6 (6.7)	9 (9.9)	3 (3.3)	0.591
Poorly Defined Margin (%)	3 (3.3)	5 (5.6)	4 (4.4)	2 (2.2)	0.497
Nodular or Tapered Contour (%)	4 (4.4)	7 (7.8)	7 (7.7)	2 (2.2)	0.371
Internal Architecture (%)	9 (9.9)	12 (13.3)	9 (9.9)	6 (6.6)	0.496
Blood Flow:-					
Any Blood Flow (%)	16 (14.7)	20 (18.5)	18 (16.5)	9 (8.3)	0.471
Intra-Tendon Blood Flow (%)	5 (5.5)	4 (4.4)	9 (9.9)	0 (0.0)	1.000
Paratenon Blood Flow (%)	10 (11.1)	12 (13.3)	10 (11.0)	6 (6.6)	0.656
Kager's Fat Pad Blood Flow (%)	12 (13.2)	14 (15.6)	14 (15.4)	6 (6.6)	0.677
Other Features:-					
Any Other Features (%)	12 (13.2)	15 (16.7)	11 (12.2)	8 (8.8)	0.538
Soft Tissue Swelling (%)	2 (2.2)	3 (3.3)	3 (3.3)	1 (1.1)	0.682
Paratenon Fluid (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Fluid-filled Bursa (%)	4 (4.4)	7 (7.8)	5 (5.5)	3 (3.3)	0.371
Hyperechogenic Kager's Fat Pad (%)	3 (3.3)	3 (3.3)	2 (2.2)	2 (2.2)	1.000
Abnormal Myotendinous Junction (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Abnormal Calcaneus (%)	4 (4.4)	4 (4.4)	2 (2.2)	3 (3.3)	1.000

Values are expressed as the number of observations (n) with the frequency (%) in parentheses.
[a] 1 triathlete was an amputee.
[b] Left vs. right Achilles tendon.

There were no significant differences in the distribution of any of the individual or combined abnormal morphological nor any of the other individual or combined abnormal other features of the triathletes' Achilles tendons between the pre- and immediate post-race ultrasound soft tissue investigation (Table 3.13). There were however significant increases in the intra-tendon ($p=0.027$), paratenon ($p<0.001$) and Kager's fat pad ($p<0.001$) blood flow immediately after the triathlon when compared to the pre-race investigation. There was also a significant increase in blood flow in any of three specific sites in the immediate post-race tendons ($p<0.001$). Similar results were obtained when the numbers of triathletes, instead of tendons, were analyzed (Table 3.13).

Table 3.13: Comparison of the pre- and immediate post-race ultrasound abnormal morphological features, blood flow and other abnormal features in the triathletes' Achilles tendons

	Pre-race Tendons (n=217) [a]	Post-race Tendons (n=181) [a]	p-value [b]	Pre-race Athletes (n=109)	Post-race Athletes (n=91)	p-value [c]
Morphological Features:-						
Any Morphological Features (%)	30 (13.8)	25 (13.8)	1.000	21 (19.3)	19 (20.9)	0.860
Fusiform Shape (%)	22 (10.1)	15 (8.3)	0.605	22 (20.2)	15 (16.5)	0.585
Poorly Defined Margin (%)	14 (6.5)	8 (4.4)	0.510	14 (12.8)	8 (8.8)	0.497
Nodular or Tapered Contour (%)	20 (9.2)	11 (6.1)	0.266	20 (18.3)	11 (12.1)	0.245
Internal Architecture (%)	27 (12.4)	21 (11.6)	0.878	19 (17.4)	15 (16.5)	1.000
Blood Flow:-						
Any Blood Flow (%)	6 (2.8)	36 (19.9)	<0.001	4 (3.7)	27 (29.7)	<0.001
Intra-Tendon Blood Flow (%)	2 (0.9)	9 (5.0)	0.027	2 (1.8)	9 (9.9)	0.025
Paratenon Blood Flow (%)	5 (2.3)	22 (12.2)	<0.001	3 (2.8)	16 (17.6)	<0.001
Kager's Fat Pad Blood Flow (%)	0 (0.0)	26 (14.4)	<0.001	0 (0.0)	20 (22.0)	<0.001
Other Features:-						
Any Other Features (%)	33 (15.2)	27 (14.9)	1.000	22 (20.2)	19 (20.9)	1.000
Soft Tissue Swelling (%)	2 (0.9)	5 (2.8)	0.253	1 (0.9)	4 (4.4)	0.179
Paratenon Fluid (%)	1 (0.5)	0 (0.0)	1.000	1 (0.9)	0 (0.0)	1.000
Fluid Filled Bursa (%)	10 (4.6)	11 (6.1)	0.654	8 (7.3)	8 (8.8)	0.797
Hyperechogenic Kager's Fat Pad (%)	11 (5.1)	6 (3.3)	0.461	8 (7.3)	4 (4.4)	0.552
Abnormal Myotendinous Junction (%)	0 (0.0)	0 (0.0)	-	0 (0.0)	0 (0.0)	-
Abnormal Calcaneus (%)	13 (6.0)	8 (4.4)	0.510	8 (7.3)	5 (5.5)	0.775

Values are expressed as the number of observations (n) with the frequency (%) in parentheses.
[a] 1 triathlete was an amputee.
[b] Pre- vs. immediate post-race tendons
[c] Pre- vs. immediate post-race athletes

There were no significant differences in the general characteristics, performance, training, past history of injuries, medication usage, current running shoe usage and flexibility training between the triathletes with a

presence and absence of blood flow in the tendon and surrounding tissues immediately after the triathlon (data not shown). As illustrated in Figure 3.3B, significantly more Achilles tendons diagnosed with tendinosis before the triathlon, presented with blood flood in the tendon and surrounding tissues immediately after the event when compared to the normal tendons and those with other abnormal features (p=0.002). Triathletes with pre-race Achilles tendinosis were more likely to exhibit increased blood flow within the affected tendon or surrounding tissue immediately after the race (odds ratio=4.7, 95% confidence interval 1.8 to 12.2, p=0.002). In addition, there was a significant association between the presence of immediate post-race blood flow in the Achilles tendon and/or surrounding tissues of the triathletes and the presence of tenderness to palpation (p=0.011) and a positive shift's test (p=0.025), but not the presence of swelling (p=1.000) or symptoms (p=0.593) before the race (Figure 3.3A).

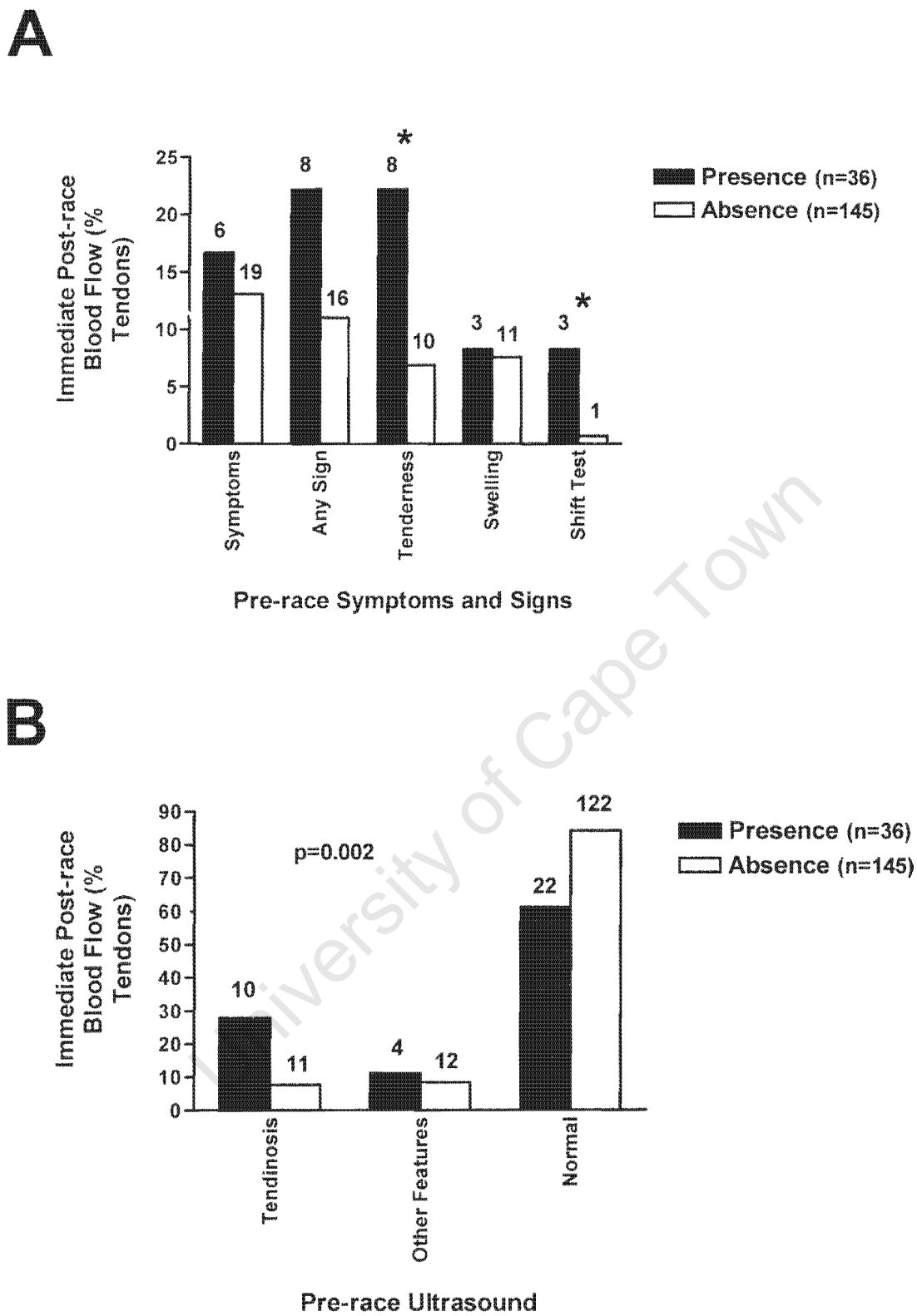

Figure 3.3.: The relationship between pre-race (A) symptoms and signs and (B) ultrasound findings of the Achilles tendon and the presence of immediate post-race increases in blood flow in and around the Achilles tendon in the triathletes

The numbers of observations (n) are indicated above each bar. The asterisk denotes $p<0.05$.

The 6-8 weeks post-race US abnormal morphological features, presence of blood flow and abnormal other features of a total of 58 tendons (26.7% of the 217 pre-race tendons), from 29 triathletes, are presented in Table 3.14.

Table 3.14: Six to 8 week post-race ultrasound abnormal morphological features, blood flow and other abnormal features in the triathletes' Achilles tendons

	Left (n=29)	Right (n=29)	Unilateral (n=29)	Bilateral (n=29)	p-value [a]
Morphological Features:-					
Any Morphological Features (%)	3 (10.3)	7 (24.1)	6 (20.7)	2 (6.9)	0.297
Fusiform Shape (%)	2 (6.9)	3 (10.3)	3 (10.3)	1 (3.4)	1.000
Poorly Defined Margin (%)	1 (3.4)	4 (13.8)	3 (10.3)	1 (3.4)	0.353
Nodular or Tapered Contour (%)	2 (6.9)	5 (17.2)	3 (10.3)	2 (6.9)	0.423
International Architecture (%)	3 (10.3)	7 (24.1)	6 (20.7)	2 (6.9)	0.297
Blood Flow:-					
Any Blood Flow (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Intra-Tendon Blood Flow (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Paratenon Blood Flow (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Kager's Fat Pad Blood Flow (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Other Features:-					
Any Other Features (%)	6 (20.7)	5 (17.2)	3 (10.3)	4 (13.8)	1.000
Soft Tissue Swelling (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Paratenon Fluid (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Fluid Filled Bursa (%)	2 (6.9)	2 (6.9)	0 (0.0)	2 (6.9)	1.389
Hyperechogenic Kager's Fat Pad (%)	1 (3.4)	1 (3.4)	2 (6.9)	0 (0.0)	1.509
Abnormal Myotendinous Junction (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-
Abnormal Calcaneus (%)	4 (13.8)	3 (10.3)	1 (3.4)	3 (10.3)	1.000

Values are expressed as the number of observations (n) with the frequency (%) in parentheses.
[a] Left vs. right Achilles tendon.

Table 3.15 is a comparison of the pre-, immediate post- and 6-8 week post-race ultrasound findings of the Achilles tendons of the 58 pre-, 56 immediate post- and 58 6-8 weeks post-race tendon investigations. Although there was a significant difference in the distribution of blood flow in at least one of the three anatomical areas (intra-tendon, paratenon or Kager's fat pad) (p=0.007) and specifically in the Kager's fat pad (p=0.005), no blood flow was detected in any of the three anatomical areas in the triathletes at 6-8 weeks after the race. There was however a tendency (p=0.052) for the blood flow distribution within the paratenon to be different between the three time points. There were no significant differences in the distributions of intra-tendon blood flow (p=0.123), any of the morphological abnormalities (p=0.537) or any of the other feature abnormalities (p=0.797) between the pre- and two post-race US investigations.

There was a significant increase in post-race antero-posterior (AP) Achilles tendon (n=58) diameter (p=0.030) but not transverse (TRV) diameter (p=0.562) following the race, compared with the pre-race diameters.

On average the 29 triathletes who volunteered for the 6-8 weeks post-race clinical and US investigations did not exercise for 17.1 ± 15.8 days (n=25), ranging from 0 to 47 days, after the triathlon. When they trained they swam on average 1.7 ± 3.3 km/week (n=22), ranging from 0 to 12, cycled on average 61 ± 79 km/week (n=19), ranging from 0 to 200, and ran on average 29 ± 20 km/week (n=21), ranging from 0 to 60. In addition these triathletes

participated in cycle races up to 180 km, on average 22 ± 45 km (n=26) and running races up a maximum distance of 77 km, on average 29 ± 25 km (n=25) during the 6-8 weeks after the triathlon.

During this period only 9 of 29 athletes had specific treatment for their Achilles tendons. The treatment included icing (n=3), stretching (n=5), physiotherapy (US and manipulation) (n=1), eccentric training (n=3) and application of a topical NSAID (n=1)

Table 3.15: Comparison of the pre-, immediate post- and 6-8 week post-race ultrasound findings (morphology and blood flow) of Achilles tendons

	Pre-Race (n=58)	Immediately Post-Race (n=56) [a]	6-8 Weeks Post-Race (n=58)	p-value
Achilles tendon morphology				
Antero-Posterior Diameter (mm)	4.6 ± 0.5	nd [b]	5.0 ± 1.1	0.030
Transverse Diameter (mm)	15,8 ± 1.5	nd [b]	15.9 ± 1.8	0.562
Any Morphological Features (%)	6 (10.3)	7 (12.5)	10 (17.2)	0.537
Any Other Features (%)	10 (17.2)	8 (14.3)	11 (19.0)	0.797
Achilles tendon blood flow				
Any Blood Flow (%)	2 (3.4)	8 (14.3)	0 (0.0)	0.007
Intra-Tendon Blood Flow (%)	0 (0.0)	2 (3.4)	0 (0.0)	0.123
Paratenon Blood Flow (%)	2 (3.4)	5 (8.9)	0 (0.0)	0.052
Kager's Fat Pad Blood Flow (%)	0 (0.0)	5 (8.9)	0 (0.0)	0.005

Values are expressed as the number of observations (n) with the frequency (%) in parentheses, or as Mean ± Standard deviation (SD).
[a] 1 athlete (2 tendons) did not have a post-race clinical examination or ultrasound.
[b] Immediate post-race Achilles tendon diameter measurements were not determined (nd)

3.3.8. The relationship of blood flow with symptoms and signs

As shown in Table 3.16 there were no significant associations between the presence of immediate post-race blood flow in the Achilles tendon and/or surrounding tissues of the triathletes and the presence of any symptoms or tenderness to palpation at 6-8 weeks after the event. There was however, a significant association between the presences of blood flow immediate after the race and the presence of swelling 6-8 weeks after the event (p=0.035). In addition there was no significant association between the presence of immediate post-race blood flow in the Achilles tendon and/or surrounding tissues of the triathletes and the presence of tendinosis 6-8 weeks after the event (p=0.576) (data not shown).

Table 3.16: The relationship between symptoms and signs of Achilles tendon injury (6-8 weeks after the event) and the presence of immediate post-race increases in blood flow in and around the Achilles tendon in triathletes

	Immediate Post-race Blood Flow		p-value
	Present (n=8)	**Absent** (n=48)	
Presence of any signs and/or symptoms at 6-8 weeks post-race	5 (62.5)	20 (41.7)	0.445
Presence of any symptoms at 6-8 weeks post-race	5 (62.5)	17 (35.4)	0.241
Presence of any signs at 6-8 weeks post-race	5 (62.5)	13 (27.1)	0.095
Presence of any swelling at 6-8 weeks post-race	5 (62.5)	11 (22.9)	0.035
Presence of any tenderness at 6-8 weeks post-race	2 (25.0)	6 (12.5)	0.320

Values are expressed as the number of observations with the frequency (%) in parentheses.

There were no significant differences in (a) the number of rest days (p=0.247); (b) swimming (p=0.094), cycling (p=0.458), running (p=0.860) and total (p=0.970) training distance covered per week; (c) cycling (p=0.947) and running (p=0.471) race distances participated in; as well as; (d) specific treatment received to the Achilles tendon (p=0.243) during the 6-8 weeks after the Ironman triathlon between those triathletes who had increased blood flow within the Achilles tendon or surrounding tissue immediately after the race.

3.4. Discussion

The main finding of this study showed a significant increase in the blood flow in the Achilles tendon and surrounding structures (paratenon and Kager's fat pad) of triathletes immediately following the Ironman triathlon. These changes in blood flow were transient and returned back to normal 6-8 weeks later. The observed increase in blood flow (tendon and surrounding structures) was strongly associated with 1) the presence of pre-race ultrasonographic evidence of Achilles tendinosis, 2) pre-race tenderness to palpation of the Achilles tendon, and 3) a positive 'shift' test for Achilles tendinosis.

A second finding was that triathletes had a significant increase in the symptoms and signs of Achilles tendon injury 6-8 weeks after participation in the Ironman triathlon. With the exception of the presence of swelling (at 6-8 weeks post-race), other symptoms/signs of Achilles tendon injury were unrelated to the observed increase in blood flow observed immediately after the race. To our knowledge, this represents the largest prospective cohort

study of athletes where the morphology and blood flow in the Achilles tendon was documented before immediately after, 6-8 weeks after an ultra endurance event.

There are only a few studies to which we can compare our findings. In the only other similar study published to date, intratendinous blood flow (but not morphology or changes in blood flow in surrounding tissue) was measured before and after a match in 72 badminton elite players during an international tournament [121]. In contrast to the findings from our study, where only 0.9% of Achilles tendons in the triathletes had evidence of an increase in intratendinous blood flow pre-race, most of the badminton players exhibited intratendinous blood flow before the match. In our cohort of triathletes, we showed that the presence of an immediate post-race increase in blood flow was related to pre-race evidence of Achilles tendinosis (ultrasonographic and tenderness). Although we did not find that blood flow changes immediately after the triathlon were related to self-reported symptoms of Achilles tendon injury, self-reported symptoms in the preceding 3 years in the non-dominant leg were associated with the presence of intratendinous blood flow in the study on badminton players. This is most likely due to the reporting of current symptoms in the triathletes compared to a 3-year history of self reported symptoms in badminton players. Also, in our triathletes study, we did show that the incidence of intratendinous blood flow in the Achilles tendon increased from 0.9% pre-race to 5.0% tendons immediately post-race. This incidence was still much lower than the 100% reported post-match intratendinous blood flow in the study on badminton players.

The precise explanation for these differences in the two study findings is not apparent. However, there are important methodological differences between the two studies that may explain the differences in this finding. Firstly, in our study the triathletes significantly decreased their training before the event (as indicated by the data presented in the results section), whereas it appears that the badminton players were playing in a tournament where daily play is not unusual. This would mean that in the triathlete study, subjects would not have performed significant training (loading the Achilles tendon) in the few days before the US examination. It is likely, but not confirmed, that the badminton players loaded the Achilles tendons during matches in the day or two before the event. Changes in blood flow induced by an acute exercise bout may well be transient. In our study, we showed that blood flow changes returned back to normal 6-8 weeks post-race. We did not measure blood flow changes serially in the 6-8 weeks post race, therefore the duration of how long the increased blood flow remained, is not known. Secondly, it is important to note that badminton and triathlon (swimming, running and cycling) will result in different types of loading of the Achilles tendon and this may account for the differences that were observed incidences in blood flow. Finally, differences in the results between the two studies may also be related to the techniques that were used to document intratendinous blood flow.

Our observations that blood flow increases immediately after the triathlon also occurred in the surrounding tissues (paratenon, Kager's fat pad) have, to our knowledge, not been reported before following an endurance event. However,

increased peritendinous blood flow has been shown to increase following 40 min of dynamic contraction of the triceps surae [129], and following static isometric contractions [115]. It has been suggested that the mechanism for the increased peritendinous blood flow following exercise is a reduced interstitial tissue pressure [144].

In our study, changes in peritendinous blood flow and surrounding tissues following the triathlon were more dramatic that those observed within the tendon (paratenon – increased from 2.3% pre-race to 12.2% immediately post-race; Kager's fat pad increased from 0% pre-race to immediately 14.4% post-race) (Table 3.13.). The possible significance of these findings is that in triathletes with pre-race Achilles tendinosis, increases in blood flow are not limited to the tendon, but increase to a greater extent in the surrounding tissues. The increase in blood flow in the tendon as well as the surrounding tissue immediately post-race may well explain the observation that there was increased swelling documented at 6-8 weeks later in this group (Table 3.16). It has been shown that in cases of Achilles tendon pathology, blood vessels are evident and originate from the peritendinous soft tissue and this may well explain the development of swelling [97;145].

Although our study was not aimed at defining any mechanism/s for changes in the blood flow in the Achilles tendon following an acute exercise bout, there are studies that have examined this question. Increases in blood flow in peritendinous connective tissue have been observed in studies using a model where mechanical loading was applied to calf muscles in vivo [123]. In this study

it was reported that a COX-2 specific mechanism was responsible for an exercise-induced increase in prostaglandin synthesis, and that this increase played an important role in the peritendinous connective tissue blood flow during physical loading in vivo. The same investigators [124] also reported that, in response to exercise, there is hyperemia in the skeletal muscle and that this is related to an increase in the concentrations of bradykinin and adenosine, in both skeletal muscle and connective tissue. It was suggested that bradykinin and adenosine are potential regulators of peritendinous tissue blood flow. A limitation of both these studies is that there was no longer term follow-up to document changes in blood flow over time. The nature and duration of the loading during exercise is also not similar to that of competing in a triathlon, which makes direct comparison to the results of our study difficult. Finally, in one other study, a possible mechanism to explore the association between dynamic exercise in humans and the increase in calf muscle and peritendinous blood flow was investigated [126]. In this study, it was reported that there is a relationship between a decrease in oxygen saturation during exercise and a rise in Achilles' peritendinous blood flow [126].

In summary, it therefore appears that in response to loading of the Achilles tendon during exercise there is an increase in intratendinous and peritendinous blood flow immediately after exercise. Our study has shown that this increase is strongly related to the pre-exercise presence of Achilles tendinosis. The mechanism/s for this increase in blood flow is not clear but may be related to decreased oxygen saturation, and/or the production of

mediators via the COX-2 mechanism. Mediators that have been related to this increase are bradykinin and adenosine.

A further novel finding of our study was that 6-8 weeks following the race, intratendinous and peritendinous blood flow changes had returned to pre-race values. This finding has to our knowledge not been reported previously. Therefore, in our group of triathletes, immediate post race changes in blood flow of the Achilles tendon and surrounding structures were of a transient nature. During the 6-8 week post-race period triathletes continued with training, but 9/29 did have some treatment for the Achilles tendon. Although this requires further study in other sports, and perhaps more serial measurements are needed, we can conclude that these findings of increased blood flow are not permanent. However, it is not known whether repeated exposure to loads resulting in these transient increases in blood flow could lead to long-term permanent detrimental effects. Further studies are needed to determine this.

In our study, the morphological features of the Achilles tendons did not change from the pre- to immediate post-race and 6-8 weeks post-race. A possible limitation of our study is that the diameter of the Achilles tendon was not measured at the immediate post-race ultrasound. This was due to the time limitation during the post-race period as many athletes finished at the same time, and only one ultrasound machine and radiologist was available to perform the US examinations.

To our knowledge there are no studies with which to compare our findings of changes in morphological features from pre- to immediate post race and 6-8 weeks later. Our finding that, at 6-8 weeks post race, there was a significant increase in the antero-posterior diameter of the Achilles tendon (p=0.030), but not the transverse diameter is of interest. There were no other features to suggest that this was related to fluid, partial tears with swelling or degenerative changes in the tendon and hence the precise reason for this increase in diameter is therefore not clear and would require further investigation.

Other findings in our study were that 38% of the triathletes reported a previous history of any tendon injury, 22% reported a history of any ligament injury, while 19% reported a previous history of an Achilles tendon injury. Although methodological differences exist between studies, these frequencies are generally comparable for previously reported ligament injuries [3;5], any tendon injuries [3;5], and Achilles tendon injuries [7] in triathletes.

The factors associated with pre-race Achilles tendinosis in the triathletes in our study were increasing age, performance at a higher level, past history of tendon injury and Achilles tendon injury, and a history of oral corticosteroid use. Increased training volume prior to the race, gender, previous fluoroquinolone antibiotic use, smoking, and somatotype were not associated with Achilles tendinosis in our cohort of triathletes. These findings are similar to that previously reported [5] for increasing age, performance level and previous history of Achilles tendon injury. However, increased training and the

number of previous triathlon races were not associated with Achilles tendon injury in our study, but were previously documented [5;132].

Finally, in any cohort study it is important to note whether the cohort is representative of the population. In our study, the cohort of triathletes that we followed did not differ from the triathletes representing the entire field in gender and age distribution. However, triathletes in our cohort were significantly slower in the overall race, as well as the swim component of the race, but were not different in the cycle and running components of the race. All the findings we report therefore have to take into account that the triathletes we studied may represent the more 'average' rather than the 'elite' triathlete. It is also possible that triathletes with existing Achilles tendon injury were more likely to volunteer to participate in this study (selection bias). This is a limitation of this study, and it would mean that the prevalence of Achilles tendon injury during the pre-race assessment could be falsely elevated. However, this possible selection bias is unlikely to affect the main results of the study, which were related to changes in blood flow from the pre-race to 6-8 weeks post-race.

3.5. Summary and conclusions

In summary, the main results of this prospective cohort study in 109 triathletes entering an Ironman race show that immediately post-race, there was increased blood flow in the Achilles tendon and surrounding tissues, which returned back to normal after 6-8 weeks, with the exception that swelling in

the tendon remained. This increase in blood flow was related to the pre-race presence of Achilles tendinosis, which was diagnosed clinically (tenderness) and on US. The long-term effects of this transient increase in blood flow in response to a single prolonged exercise bout, such as an Ironman race, require further investigation. Furthermore, at the start of the race, 19.4% of the triathletes in our cohort had symptoms and/or signs of Achilles tendon injury. The factors associated with Achilles tendinosis in these triathletes were increased age, better personal best times, a higher prevalence of past tendon injury, and previous use of oral corticosteroids.

Chapter 4

Summary and conclusion

The Ironman Triathlon is an endurance event comprising a 3.8 km swim, a 180 km cycle, and a 42.2.km run. Triathletes participating in this event can develop injuries, in particular overuse injuries. One of the more common specific overuse injuries in these triathletes is Achilles tendinosis. However, there are very few studies that have documented the factors associated with Achilles tendinosis in triathletes. In this study, we recruited triathletes during the pre-race period for an Ironman competition, and then followed a cohort of these triathletes from pre-race, to immediately after the race and a subsequent follow-up at 6-8 weeks after the race in order to study Achilles tendon injuries.

We observed that 19% of the triathletes in our group had pre-race evidence of Achilles tendinosis. Furthermore, the factors associated with Achilles tendinosis were increased age, better previous race performances, a past history of any tendon injury, a past history of Achilles tendon injury, and a history of oral corticosteroid use. All these risk factors have previously been identified, and the results of our study confirm the findings of others.

However, the novel aspect of our study was that we were able to show that blood flow in the Achilles tendon and surrounding tissue was increased immediately after the race, and more importantly that these changes were related to the presence of a pre-race diagnosis of Achilles tendinosis (using

US and clinical criteria). Our results also showed that immediately post race increased blood flow was associated with increased swelling in the Achilles tendon region at 6-8 weeks after the race, despite the fact that blood flow changes had returned to normal by 6-8 weeks. There was also some evidence of morphological changes in the Achilles tendon with an increased in tendon diameter that occurred at 6-8 weeks following the race.

The precise mechanism for these changes in blood flow, as well as the possible long-term consequences of repeated increases in tendon blood flow will require further investigation.

Clinical implications of the findings in this study are as follows:

- About 20% of triathletes participating in the Ironman triathlon have some evidence of Achilles tendinosis
- Possible risk factors for Achilles tendinosis in triathletes are increased age, performing at a higher level, past history of Achilles tendon injury, and oral corticosteroid use
- Triathletes with Achilles tendinosis will have an increase in blood flow in the Achilles tendon and surrounding tissues immediately after the race, but this will return to normal 6-8 weeks later
- Ultrasonography to assess blood flow in tendons has to be standardized and an exercise bout performed immediately before assessment can influence the findings

References

1. Dallam GM, Jonas S, Miller TK. Medical considerations in triathlon competition: recommendations for triathlon organisers, competitors and coaches. *Sports Med.* 2005;**35**:143-61.

2. Fitzpatrick MJ. Triathlon injuries. The swim-bike-run how-to for medical practitioners. *Aust.Fam.Physician* 1991;**20**:953-8.

3. Korkia PK, Tunstall-Pedoe DS, Maffulli N. An epidemiological investigation of training and injury patterns in British triathletes. *Br.J.Sports Med.* 1994;**28**:191-6.

4. O'Toole ML, Hiller WD, Smith RA, Sisk TD. Overuse injuries in ultraendurance triathletes. *Am.J.Sports Med.* 1989;**17**:514-8.

5. Egermann M, Brocai D, Lill CA, Schmitt H. Analysis of injuries in long-distance triathletes. *Int.J.Sports Med.* 2003;**24**:271-6.

6. Pen LJ, Barrett RS, Neal RJ, Steele JR. An injury profile of elite ironman competitors. *Aust.J.Sci.Med.Sport* 1996;**28**:7-11.

7. Vleck VE,.Garbutt G. Injury and training characteristics of male Elite, Development Squad, and Club triathletes. *Int.J.Sports Med.* 1998;**19**:38-42.

8. O'Toole ML,.Douglas PS. Applied physiology of triathlon. *Sports Med.* 1995;**19**:251-67.

9. Manninen JS,.Kallinen M. Low back pain and other overuse injuries in a group of Japanese triathletes. *Br.J.Sports Med.* 1996;**30**:134-9.

10. Collins K, Wagner M, Peterson K, Storey M. Overuse injuries in triathletes. A study of the 1986 Seafair Triathlon. *Am.J.Sports Med.* 1989;**17**:675-80.

11. Shaw T, Howat P, Trainor M, Maycock B. Training patterns and sports injuries in triathletes. *J.Sci.Med.Sport* 2004;**7**:446-50.

12. Paavola M, Kannus P, Orava S, Pasanen M, Jarvinen M. Surgical treatment for chronic Achilles tendinopathy: a prospective seven month follow up study. *Br.J Sports Med* 2002;**36**:178-82.

13. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. *Am.J Sports Med* 2002;**30**:287-305.

14. Jozsa L, Kannus P. Human tendons. Champaign, IL: Human Kinetics, 1997.

15. Astrom M,.Rausing A. Chronic Achilles tendinopathy. A survey of surgical and histopathologic findings. *Clin.Orthop.* 1995;151-64.

16. Puddu G, Ippolito E, Postacchini F. A classification of Achilles tendon disease. *Am.J.Sports Med.* 1976;**4**:145-50.

17. Chazan I. Achilles tendinitis part 1:Anatomy,histology,classification,etiology and pathomechanics. *The Journal of Manual and Manipulative Therapy* 1998;**6**:63-9.

18. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. Update and implications for clinical management. *Sports Med* 1999;**27**:393-408.

19. Almekinders LC. Tendinitis and other chronic tendinopathies. *J Am Acad Orthop Surg* 1998;**6**:157-64.

20. Myerson MS,.McGarvey W. Disorders of the Achilles tendon insertion and Achilles tendinitis. *Instr.Course Lect.* 1999;**48**:211-8.

21. Smart GW, Taunton JE, Clement DB. Achilles tendon disorders in runners--a review. *Med Sci Sports Exerc.* 1980;**12**:231-43.

22. Gibbon WW, Cooper JR, Radcliffe GS. Sonographic incidence of tendon microtears in athletes with chronic Achilles tendinosis. *Br.J Sports Med* 1999;**33**:129-30.

23. Leppilahti J,.Orava S. Total Achilles tendon rupture. A review. *Sports Med* 1998;**25**:79-100.

24. Maffulli N,.Wong J. Rupture of the Achilles and patellar tendons. *Clin Sports Med* 2003;**22**:761-76.

25. Maffulli N,.Kader D. Tendinopathy of tendo achillis. *J Bone Joint Surg.Br.* 2002;**84**:1-8.

26. Paavola M, Kannus P, Paakkala T, Pasanen M, Jarvinen M. Long-term prognosis of patients with achilles tendinopathy. An observational 8-year follow-up study. *Am.J Sports Med* 2000;**28**:634-42.

27. Riley G. The pathogenesis of tendinopathy. A molecular perspective. *Rheumatology.(Oxford)* 2004;**43**:131-42.

28. Jarvinen M. Epidemiology of tendon injuries in sports. *Clin.Sports Med.* 1992;**11**:493-504.

29. Jarvinen TA, Kannus P, Paavola M, Jarvinen TL, Jozsa L, Jarvinen M. Achilles tendon injuries. *Curr.Opin.Rheumatol.* 2001;**13**:150-5.

30. Jarvinen TA, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: etiology and epidemiology. *Foot Ankle Clin.* 2005;**10**:255-66.

31. Mokone, G. G., Schwellnus, M. P., Noakes, T. D., and Collins, M. The COL5A1 gene and Achilles tendon pathology. *Scand.J Med Sci.Sports* . 2006;**16**(1):19-26.

32. Knapik JJ, Sharp MA, Canham-Chervak M, Hauret K, Patton JF, Jones BH. Risk factors for training-related injuries among men and women in basic combat training. *Med.Sci.Sports Exerc.* 2001;**33**:946-54.

33. Maffulli N, Wong J, Almekinders LC. Types and epidemiology of tendinopathy. *Clin Sports Med* 2003;**22**:675-92.

34. Mahieu NN, Witvrouw E, Stevens V, Van Tiggelen D, Roget P. Intrinsic risk factors for the development of achilles tendon overuse injury: a prospective study. *Am.J.Sports Med.* 2006;**34**:226-35.

35. Mokone, G. G. Intrinsic factors for Achilles tendon injuries: A special emphasis on genetic predisposition. 2006. Unpublished thesis. University of Cape Town.

36. Jozsa L, Kvist M, Balint BJ, Reffy A, Jarvinen M, Lehto M *et al.* The role of recreational sport activity in Achilles tendon rupture. A clinical, pathoanatomical, and sociological study of 292 cases. *Am.J.Sports Med.* 1989;**17**:338-43.

37. Suchak AA, Bostick G, Reid D, Blitz S, Jomha N. The incidence of Achilles tendon ruptures in Edmonton, Canada. *Foot Ankle Int.* 2005;**26**:932-6.

38. Kvist M. Achilles tendon injuries in athletes. *Sports Med.* 1994;**18**:173-201.

39. Stevenson M, Finch C, Hamer P, Elliott B. The Western Australian sports injury study. *Br.J.Sports Med.* 2003;**37**:380-1.

40. Milgrom C, Finestone A, Zin D, Mandel D, Novack V. Cold weather training: a risk factor for Achilles paratendinitis among recruits. *Foot Ankle Int* 2003;**24**:398-401.

41. Kannus P,.Natri A. Etiology and pathophysiology of tendon ruptures in sports. *Scand.J Med Sci.Sports* 1997;**7**:107-12.

42. Ekstrand J,.Nigg BM. Surface-related injuries in soccer. *Sports Med.* 1989;**8**:56-62.

43. McCrory JL, Martin DF, Lowery RB, Cannon DW, Curl WW, Read HM, Jr. *et al.* Etiologic factors associated with Achilles tendinitis in runners. *Med Sci Sports Exerc.* 1999;**31**:1374-81.

44. Safran MR,.Graham SM. Distal biceps tendon ruptures: incidence, demographics, and the effect of smoking. *Clin.Orthop.Relat Res.* 2002;275-83.

45. Kane SM, Dave A, Haque A, Langston K. The incidence of rotator cuff disease in smoking and non-smoking patients: a cadaveric study. *Orthopedics* 2006;**29**:363-6.

46. Harris SB, Harris D, Foster AJ, Elliot D. The aetiology of acute rupture of flexor tendon repairs in zones 1 and 2 of the fingers during early mobilization. *J.Hand Surg.[Br.]* 1999;**24**:275-80.

47. Prasad N, Odumala A, Elias F, Jenkins T. Outcome of open rotator cuff repair. An analysis of risk factors. *Acta Orthop.Belg.* 2005;**71**:662-6.

48. Junqueira LC, Carneiro J, Kelley R. Basic histology. Stamford, Connecticut: Appleton and Lange, 1998.

49. van der Linden PD, Sturkenboom MC, Herings RM, Leufkens HM, Rowlands S, Stricker BH. Increased risk of achilles tendon rupture with quinolone antibacterial use, especially in elderly patients taking oral corticosteroids. *Arch.Intern.Med.* 2003;**163**:1801-7.

50. Vanek D, Saxena A, Boggs JM. Fluoroquinolone therapy and Achilles tendon rupture. *J Am Podiatr.Med Assoc* 2003;**93**:333-5.

51. Roberts JR, Hudson JA, Lindell KK, Finger DR. Serial sonographic evaluation of Achilles tendons in patients taking fluoroquinolone antibiotics. *Hawaii Med J* 2006;**65**:112-4.

52. Williams RJ, III, Attia E, Wickiewicz TL, Hannafin JA. The effect of ciprofloxacin on tendon, paratenon, and capsular fibroblast metabolism. *Am.J.Sports Med.* 2000;**28**:364-9.

53. Corps AN, Curry VA, Harrall RL, Dutt D, Hazleman BL, Riley GP. Ciprofloxacin reduces the stimulation of prostaglandin E(2) output by interleukin-1beta in human tendon-derived cells. *Rheumatology.(Oxford)* 2003;**42**:1306-10.

54. Speed CA. Fortnightly review: Corticosteroid injections in tendon lesions. *BMJ* 2001;**323**:382-6.

55. Gill SS, Gelbke MK, Mattson SL, Anderson MW, Hurwitz SR. Fluoroscopically guided low-volume peritendinous corticosteroid injection for Achilles tendinopathy. A safety study. *J Bone Joint Surg Am* 2004;**86-A**:802-6.

56. Battista V, Combs J, Warme WJ. Asynchronous bilateral achilles tendon ruptures and androstenediol use. *Am.J.Sports Med.* 2003;**31**:1007-9.

57. Neely FG. Intrinsic risk factors for exercise-related lower limb injuries. *Sports Med.* 1998;**26**:253-63.

58. Knapik J, Ang P, Reynolds K, Jones B. Physical fitness, age, and injury incidence in infantry soldiers. *J.Occup.Med.* 1993;**35**:598-603.

59. Maffulli N. Rupture of the Achilles tendon. *J Bone Joint Surg.Am.* 1999;**81**:1019-36.

60. Cretnik A,.Frank A. Incidence and outcome of rupture of the Achilles tendon. *Wien.Klin.Wochenschr.* 2004;**116 Suppl 2**:33-8.

61. Fahlstrom M, Lorentzon R, Alfredson H. Painful conditions in the Achilles tendon region: a common problem in middle-aged competitive badminton players. *Knee.Surg.Sports Traumatol.Arthrosc.* 2002;**10**:57-60.

62. Tallon C, Maffulli N, Ewen SW. Ruptured Achilles tendons are significantly more degenerated than tendinopathic tendons. *Med Sci Sports Exerc.* 2001;**33**:1983-90.

63. Mathiak G, Wening JV, Mathiak M, Neville LF, Jungbluth K. Serum cholesterol is elevated in patients with Achilles tendon ruptures. *Arch.Orthop.Trauma Surg.* 1999;**119**:280-4.

64. Ozgurtas T, Yildiz C, Serdar M, Atesalp S, Kutluay T. Is high concentration of serum lipids a risk factor for Achilles tendon rupture? *Clin.Chim.Acta* 2003;**331**:25-8.

65. Davis JJ, Mason KT, Clark DA. Achilles tendon ruptures stratified by age, race, and cause of injury among active duty U.S. Military members. *Mil.Med.* 1999;**164**:872-3.

66. Olivieri I, Gemignani G, Gherardi S, Grassi L, Ciompi ML. Isolated HLA-B27 associated Achilles tendinitis. *Ann.Rheum.Dis.* 1987;**46**:626-7.

67. Olivieri I, Padula A, Lisanti ME, Braccini G. Longstanding HLA-B27 associated Achilles tendinitis. *Ann.Rheum.Dis.* 1992;**51**:1265.

68. Amor B, Cherot A, Delbarre F, Nunez RA, Hors J. Hydroxyapatite rheumatism and HLA markers. *J.Rheumatol.Suppl* 1977;**3**:101-4.

69. Maffulli N, Reaper JA, Waterston SW, Ahya T. ABO blood groups and achilles tendon rupture in the Grampian Region of Scotland. *Clin.J.Sport Med.* 2000;**10**:269-71.

70. Aroen A, Helgo D, Granlund OG, Bahr R. Contralateral tendon rupture risk is increased in individuals with a previous Achilles tendon rupture. *Scand.J.Med.Sci.Sports* 2004;**14**:30-3.

71. Mokone GG, Gajjar M, September AV, Schwellnus MP, Greenberg J, Noakes TD *et al.* The guanine-thymine dinucleotide repeat polymorphism within the tenascin-C gene is associated with achilles tendon injuries. *Am.J.Sports Med.* 2005;**33**:1016-21.

72. Gibson W. Are "spontaneous" Achilles tendon ruptures truly spontaneous? *Br.J.Sports Med.* 1998;**32**:266.

73. Kannus P,.Jozsa L. Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. *J.Bone Joint Surg.Am.* 1991;**73**:1507-25.

74. Kainberger F, Mittermaier F, Seidl G, Parth E, Weinstabl R. Imaging of tendons--adaptation, degeneration, rupture. *Eur.J.Radiol.* 1997;**25**:209-22.

75. Williams JG. Achilles tendon lesions in sport. *Sports Med* 1986;**3**:114-35.

76. O'Brien M. The anatomy of the Achilles tendon. *Foot Ankle Clin.* 2005;**10**:225-38.

77. Ly JQ,.Bui-Mansfield LT. Anatomy of and abnormalities associated with Kager's fat Pad. *AJR Am.J.Roentgenol.* 2004;**182**:147-54.

78. Ahmed IM, Lagopoulos M, McConnell P, Soames RW, Sefton GK. Blood supply of the Achilles tendon. *J Orthop.Res.* 1998;**16**:591-6.

79. Theobald P, Benjamin M, Nokes L, Pugh N. Review of the vascularisation of the human Achilles tendon. *Injury* 2005;**36**:1267-72.

80. Naito M,.Ogata K. The blood supply of the tendon with a paratenon. An experimental study using hydrogen washout technique. *Hand* 1983;**15**:9-14.

81. Lagergren C,.Lindholm A. Vascular distribution in the Achilles tendon; an angiographic and microangiographic study. *Acta Chir Scand.* 1959;**116**:491-5.

82. Karcz MJ, Skawina A, Gorczyca J, Danilewicz M. The arterial vascularisation of the human calcaneus (Achilles) tendo during the prenatal development. *Folia Morphol.(Warsz.)* 1996;**55**:306-8.

83. Zantop T, Tillmann B, Petersen W. Quantitative assessment of blood vessels of the human Achilles tendon: an immunohistochemical cadaver study. *Arch.Orthop.Trauma Surg.* 2003;**123**:501-4.

84. Bleakney RR,.White LM. Imaging of the Achilles tendon. *Foot Ankle Clin* 2005;**10**:239-54.

85. Astrom M, Gentz CF, Nilsson P, Rausing A, Sjoberg S, Westlin N. Imaging in chronic achilles tendinopathy: a comparison of ultrasonography, magnetic resonance imaging and surgical findings in 27 histologically verified cases. *Skeletal Radiol.* 1996;**25**:615-20.

86. Paavola M, Paakkala T, Kannus P, Jarvinen M. Ultrasonography in the differential diagnosis of Achilles tendon injuries and related disorders. A comparison between pre-operative ultrasonography and surgical findings. *Acta Radiol.* 1998;**39**:612-9.

87. Blankstein A, Cohen I, Diamant L, Heim M, Dudkiewicz I, Israeli A *et al.* Achilles tendon pain and related pathologies: diagnosis by ultrasonography. *Isr.Med Assoc.J* 2001;**3**:575-8.

88. Rawool NM,.Nazarian LN. Ultrasound of the ankle and foot. *Semin.Ultrasound CT MR* 2000;**21**:275-84.

89. Bude RO, Adler RS, Bassett DR, Ikeda DM, Rubin JM. Heterozygous familial hypercholesterolemia: detection of xanthomas in the Achilles tendon with US. *Radiology* 1993;**188**:567-71.

90. Kainberger FM, Engel A, Barton P, Huebsch P, Neuhold A, Salomonowitz E. Injury of the Achilles tendon: diagnosis with sonography. *AJR Am.J Roentgenol.* 1990;**155**:1031-6.

91. Nehrer S, Breitenseher M, Brodner W, Kainberger F, Fellinger EJ, Engel A *et al.* Clinical and sonographic evaluation of the risk of rupture in the Achilles tendon. *Arch.Orthop.Trauma Surg.* 1997;**116**:14-8.

92. Khan KM, Forster BB, Robinson J, Cheong Y, Louis L, Maclean L *et al.* Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. *Br J Sports Med* 2003;**37**:149-53.

93. O'Connor PJ, Grainger AJ, Morgan SR, Smith KL, Waterton JC, Nash AF. Ultrasound assessment of tendons in asymptomatic volunteers: a study of reproducibility. *Eur.Radiol.* 2004;**14**:1968-73.

94. Weinberg EP, Adams MJ, Hollenberg GM. Color Doppler sonography of patellar tendinosis. *AJR Am.J.Roentgenol.* 1998;**171**:743-4.

95. Ohberg L, Lorentzon R, Alfredson H. Neovascularisation in Achilles tendons with painful tendinosis but not in normal tendons: an ultrasonographic investigation. *Knee.Surg.Sports Traumatol.Arthrosc.* 2001;**9**:233-8.

96. Newman JS, Adler RS. Power Doppler Sonography: Applications in Musculoskeletal Imaging. *Semin.Musculoskelet.Radiol.* 1998;**2**:331-40.

97. Reiter M, Ulreich N, Dirisamer A, Tscholakoff D, Bucek RA. Colour and power Doppler sonography in symptomatic Achilles tendon disease. *Int J Sports Med* 2004;**25**:301-5.

98. Boesen MI, Torp-Pedersen S, Koenig MJ, Christensen R, Langberg H, Holmich P *et al.* Ultrasound guided electro-coagulation in patients with chronic non-insertional Achilles tendinopathy: a pilot study. *Br.J Sports Med* 2006.

99. Kotnis R, David S, Handley R, Willett K, Ostlere S. Dynamic Ultrasound as a Selection Tool for Reducing Achilles Tendon Reruptures. *Am J Sports Med* 2006.

100. da Cunha A, Parizotto NA, Vidal BC. The effect of therapeutic ultrasound on repair of the achilles tendon (tendo calcaneus) of the rat. *Ultrasound Med Biol.* 2001;**27**:1691-6.

101. Tsai WC, Hsu CC, Tang FT, Chou SW, Chen YJ, Pang JH. Ultrasound stimulation of tendon cell proliferation and upregulation of proliferating cell nuclear antigen. *J Orthop Res.* 2005;**23**:970-6.

102. Tsai WC, Pang JH, Hsu CC, Chu NK, Lin MS, Hu CF. Ultrasound stimulation of types I and III collagen expression of tendon cell and upregulation of transforming growth factor beta. *J Orthop Res.* 2006;**24**:1310-6.

103. Testa V, Capasso G, Benazzo F, Maffulli N. Management of Achilles tendinopathy by ultrasound-guided percutaneous tenotomy. *Med Sci Sports Exerc.* 2002;**34**:573-80.

104. Fredberg U, Bolvig L, Pfeiffer-Jensen M, Clemmensen D, Jakobsen BW, Stengaard-Pedersen K. Ultrasonography as a tool for diagnosis, guidance of local steroid injection and, together with pressure algometry, monitoring of the treatment of athletes with chronic jumper's knee and Achilles tendinitis: a randomized, double-blind, placebo-controlled study. *Scand J Rheumatol.* 2004;**33**:94-101.

105. Chandnani VP,.Bradley YC. Achilles tendon and miscellaneous tendon lesions. *Magn Reson.Imaging Clin.N.Am.* 1994;**2**:89-96.

106. Mammone JF. MR imaging of tendon injuries about the foot and ankle. *Clin.Podiatr.Med Surg.* 1997;**14**:313-35.

107. Shalaby M,.Almekinders LC. Patellar tendinitis: the significance of magnetic resonance imaging findings. *Am.J.Sports Med.* 1999;**27**:345-9.

108. Miniaci A, Dowdy PA, Willits KR, Vellet AD. Magnetic resonance imaging evaluation of the rotator cuff tendons in the asymptomatic shoulder. *Am.J.Sports Med.* 1995;**23**:142-5.

109. Lohman M, Kivisaari A, Vehmas T, Kallio P, Malmivaara A, Kivisaari L. MRI abnormalities of foot and ankle in asymptomatic, physically active individuals. *Skeletal Radiol.* 2001;**30**:61-6.

110. Pfirrmann CW, Zanetti M, Hodler J. Joint magnetic resonance imaging: normal variants and pitfalls related to sports injury. *Radiol.Clin.North Am.* 2002;**40**:167-80.

111. Kayser R, Mahlfeld K, Heyde CE. Partial rupture of the proximal Achilles tendon: a differential diagnostic problem in ultrasound imaging. *Br.J Sports Med* 2005;**39**:838-42.

112. Reiter M, Ulreich N, Dirisamer A, Tscholakoff D, Bucek RA. [Extended field-of-view sonography in Achilles tendon disease: a comparison with MR imaging]. *Rofo* 2004;**176**:704-8.

113. Cook JL, Malliaras P, De Luca J, Ptasznik R, Morris ME, Goldie P. Neovascularization and pain in abnormal patellar tendons of active jumping athletes. *Clin.J.Sport Med.* 2004;**14**:296-9.

114. Richards PJ, Win T, Jones PW. The distribution of microvascular response in Achilles tendonopathy assessed by colour and power Doppler. *Skeletal Radiol.* 2005;**34**:336-42.

115. Langberg H, Bulow J, Kjaer M. Standardized intermittent static exercise increases peritendinous blood flow in human leg. *Clin Physiol* 1999;**19**:89-93.

116. Nuutila P,.Kalliokoski K. Use of positron emission tomography in the assessment of skeletal muscle and tendon metabolism and perfusion. *Scand J Med Sci Sports* 2000;**10**:346-50.

117. Boushel R, Langberg H, Olesen J, Nowak M, Simonsen L, Bulow J *et al.* Regional blood flow during exercise in humans measured by near-infrared spectroscopy and indocyanine green. *J Appl.Physiol* 2000;**89**:1868-78.

118. Peers KH, Brys PP, Lysens RJ. Correlation between power Doppler ultrasonography and clinical severity in Achilles tendinopathy. *Int Orthop* 2003;**27**:180-3.

119. Kristoffersen M, Ohberg L, Johnston C, Alfredson H. Neovascularisation in chronic tendon injuries detected with colour Doppler ultrasound in horse and man: implications for research and treatment. *Knee.Surg Sports Traumatol.Arthrosc.* 2005;**13**:505-8.

120. Alfredson H. Chronic tendon pain--implications for treatment: an update. *Curr.Drug Targets.* 2004;**5**:407-10.

121. Boesen MI, Boesen A, Koenig MJ, Bliddal H, Torp-Pedersen S. Ultrasonographic Investigation of the Achilles Tendon in Elite Badminton Players Using Color Doppler. *Am J Sports Med* 2006.

122. Astrom M,.Westlin N. Blood flow in the human Achilles tendon assessed by laser Doppler flowmetry. *J.Orthop.Res* 1994;**12**:246-52.

123. Langberg H, Boushel R, Skovgaard D, Risum N, Kjaer M. Cyclo-oxygenase-2 mediated prostaglandin release regulates blood flow in connective tissue during mechanical loading in humans. *J Physiol* 2003;**551**:683-9.

124. Langberg H, Bjorn C, Boushel R, Hellsten Y, Kjaer M. Exercise-induced increase in interstitial bradykinin and adenosine concentrations in skeletal muscle and peritendinous tissue in humans. *J Physiol* 2002;**542**:977-83.

125. Wilson AM,.Goodship AE. Exercise-induced hyperthermia as a possible mechanism for tendon degeneration. *J Biomech.* 1994;**27**:899-905.

126. Boushel R, Langberg H, Green S, Skovgaard D, Bulow J, Kjaer M. Blood flow and oxygenation in peritendinous tissue and calf muscle during dynamic exercise in humans. *J Physiol* 2000;**524 Pt 1**:305-13.

127. Rosager S, Aagaard P, Dyhre-Poulsen P, Neergaard K, Kjaer M, Magnusson SP. Load-displacement properties of the human triceps surae aponeurosis and tendon in runners and non-runners. *Scand.J.Med.Sci.Sports* 2002;**12**:90-8.

128. Bulow J,.Tondevold E. Blood flow in different adipose tissue depots during prolonged exercise in dogs. *Pflugers Arch.* 1982;**392**:235-8.

129. Langberg H, Bulow J, Kjaer M. Blood flow in the peritendinous space of the human Achilles tendon during exercise. *Acta Physiol Scand* 1998;**163**:149-53.

130. Cook JL, Malliaras P, De Luca J, Ptasznik R, Morris M. Vascularity and pain in the patellar tendon of adult jumping athletes: a 5 month longitudinal study. *Br.J.Sports Med.* 2005;**39**:458-61.

131. Boesen M I, Koenig M J, Torp-Pedersen S. Tendinopathy and Doppler activity: the vascular response of the Achilles tendon to exercise. *Scand J Med Sci Sports* 2006;**in press**.

132. Burns J, Keenan AM, Redmond AC. Factors associated with triathlon-related overuse injuries. *J.Orthop.Sports Phys.Ther.* 2003;**33**:177-84.

133. Goodwin DW. Imaging of the Achilles' tendon. *Foot Ankle Clin.* 2000;**5**:135-48, vii.

134. Fessell DP,.van Holsbeeck MT. Foot and ankle sonography. *Radiol.Clin.North Am.* 1999;**37**:831-58, x.

135. Ohberg L,.Alfredson H. Effects on neovascularisation behind the good results with eccentric training in chronic mid-portion Achilles tendinosis? *Knee.Surg Sports Traumatol.Arthrosc.* 2004;**12**:465-70.

136. Maffulli N, Regine R, Angelillo M, Capasso G, Filice S. Ultrasound diagnosis of Achilles tendon pathology in runners. *Br.J Sports Med* 1987;**21**:158-62.

137. Richards PJ, Dheer AK, McCall IM. Achilles tendon (TA) size and power Doppler ultrasound (PD) changes compared to MRI: a preliminary observational study. *Clin.Radiol.* 2001;**56**:843-50.

138. Koenig MJ, Torp-Pedersen S, Qvistgaard E, Terslev L, Bliddal H. Preliminary results of colour Doppler-guided intratendinous glucocorticoid injection for Achilles tendonitis in five patients. *Scand J Med Sci Sports* 2004;**14**:100-6.

139. Sharwood KA, Collins M, Goedecke JH, Wilson G, Noakes TD. Weight changes, medical complications, and performance during an Ironman triathlon. *Br.J Sports Med* 2004;**38**:718-24.

140. Sharwood K, Collins M, Goedecke J, Wilson G, Noakes T. Weight changes, sodium levels, and performance in the South African Ironman Triathlon. *Clin J Sport Med* 2002;**12**:391-9.

141. Sulzer NU, Schwellnus MP, Noakes TD. Serum electrolytes in Ironman triathletes with exercise-associated muscle cramping. *Med Sci Sports Exerc* 2005;**37**:1081-5.

142. Gibbon WW, Cooper JR, Radcliffe GS. Distribution of sonographically detected tendon abnormalities in patients with a clinical diagnosis of chronic achilles tendinosis. *J Clin.Ultrasound* 2000;**28**:61-6.

143. Fornage BD. The hypoechoic normal tendon. A pitfall. *J.Ultrasound Med.* 1987;**6**:19-22.

144. Langberg H, Skovgaard D, Bulow J, Kjaer M. Negative interstitial pressure in the peritendinous region during exercise. *J Appl.Physiol* 1999;**87**:999-1002.

145. Silvestri E, Biggi E, Molfetta L, Avanzino C, La Paglia E, Garlaschi G. Power Doppler analysis of tendon vascularization. *Int.J.Tissue React.* 2003;**25**:149-58.

146. Clements K, Yates B, Curran M. The prevalence of chronic knee injury in triathletes. *Br.J.Sports Med.* 1999;**33**:214-6.

Appendix 1

Welcome to the Spec-Savers Ironman South Africa Research programmme - 2006

Dear Triathlete

We have the privilege to inform you that scientific and medical research at the Port Elizabeth Spec-Savers Ironman South Africa triathlon has been planned in collaboration with the UCT/MRC Research Unit for Exercise Science and Sports Medicine based at the Sports Science Institute of South Africa, and Tswane University in Pretoria. This will provide a unique opportunity for a research programme to address important medical and physiological problems that are associated with participation in the Spec-Savers Ironman South Africa triathlon.

The research study will concentrate on the following 6 main components that will ultimately assist you to **improve your performance** and **improve the standard of your medical treatment** at future triathlons and other endurance events:

- Management of the collapsed triathlete
- Causes and treatment of Exercise Associated Muscle Cramping
- Preventing post-exercise decreases in immune function and upper respiratory tract (URT) symptoms
- Genetic basis for performance and physiological responses during an Ironman Triathlon
- Identifying causes of chronic Achilles tendon injuries in triathletes
- Identifying the relationship between training history, perception of effort (RPE) during the race and the subsequent recovery after the race.

How can I volunteer to participate in the research study?

As a participant in the Port Elizabeth Spec-Savers Ironman South Africa triathlon, you will be given the unique opportunity to participate in this research effort. Please understand that your participation is entirely voluntary. Please read through the details of the following six components of the study. You will be given the opportunity to participate in any number, or all the components of the study. The details of each component are summarized below and a detailed explanation of each component can be downloaded as a PDF file. If you wish to participate in the study, please **download** the information related to each component of the study (PDF file), and read through it carefully. Please bring the INFORMED CONSENT FORM of the study with you to Port Elizabeth, and then visit our RESEARCH area at the registration venue. Here we will discuss any questions you may have, and then sign the INFORMED CONSENT FORM with you. In addition please download and complete the QUESTIONNAIRES. We will let you know once the questionnaires are available. Printed copies of all the documentation will also be available at the REGISTRATION research area.

Will my participation in the research affect my preparation, race participation, or recovery after the race?

All the components of this study have been carefully designed NOT to 1) interfere with your preparation or participation in the Ironman, 2) affect your performance on race day, and 3) your recovery after the event. All the tests are not painful and non-invasive (apart from a small blood sample taken at registration and after the race).

Will I have access to the results of the study?

Once the study results are known, you will be able to access a summary of the findings of the study on the website and you can also request, this be sent to you by email. You will also be given the opportunity to attend a feedback meeting where the results of the study will be discussed. The results will only be that of the whole group, and no individual results will be made public.

Who can I contact for more information?

In the next few weeks, please feel free to contact members of the research team should you have any questions related to the study (or any component of the study). Contact details of the research team are as follows: ironman@sports.uct.ac.za or (021) 650 4572.

The following documents can be downloaded:-

1. Subject information sheets (PDF File)
2. Consent form (PDF File)
3. Questionnaires (MS Word document)
4. Summary of the study (This web page) (PDF File)
5. Adobe Acrobat Reader

Summary of each component of the research study:-

1. Management of the collapsed triathlete

The precise causes and best treatment of collapsed endurance athletes is still widely debated. We would like to see if collapsed athletes have a greater incidence of serum sodium and plasma volume abnormalities than athletes who do not collapse at the end of the race. Accordingly, if these abnormalities do exist in collapsed athletes, are intravenous fluids superior to oral fluids in the treatment and restoration of sodium and plasma volume levels? Close monitoring of sodium levels, heart rate and blood pressure and time to discharge will help our team answer these questions.

2. Exercise associated muscle cramping

The precise causes of Exercise Associated Muscle Cramping (EAMC) are still widely debated. Contrary to popular belief, heat, dehydration and electrolyte (salt) abnormalities may NOT be the cause of EAMC. In this component of the study we would like to measure these changes in triathletes who cramp, and then follow what happens once we treat these athletes. We also want to measure the muscle

"twitchiness" during the recovery period, once again trying to see oif these related to changes in serum electrolyte concentrations (salt). Triathletes who are prone to EAMC may well be interested in this component of the study.

3. **Post-exercise upper respiratory tract (URT) symptoms**

It is well documented that intense training, as well as participation in a prolonged strenuous endurance evenst (such as the Ironman) can cause changes in the immune system, and may increase the risk of infections (mainly of the upper respiratory tract). In this component of the study we want to examine the immune changes, as well as find out what causes the upper respiratory tract symptoms in endurance athletes after participation in the Ironman. Triathletes that are prone to developing symptoms such as sore throaot, runny or blocked nose or cough after a race may well be specifically interested in participating in this component of the study.

4. **Genetic basis for performance and physiological responses during an Ironman Triathlon**

Athletic ability is partly determined by an intervals genetic make-up. Various gene (DNA material) have been shown to be associated with endurance performance, including the South African Ironman Triathlons. In addition it has also been suggested that the inter-individual physiological responses, such as blood salt and water imbalance, as well as the development of tendon overuse injuries, during endurance activities is partially determined by ones genes. The aim of this component of the study is to identify genes associated with performance and susceptibility to salt and water imbalances and indicators of underlying tendon pathology during the Ironman Triathlon. Volunteers for this component of the study will be asked to complete a questionnaire. At registration they will be asked to donate a small blood sample from which your genetic material (DNA) will be extracted and your blood salt levels measured. You will also be weighed before the swim and again immediately after the race. A second blood sample will also be taken after the measure to measure your blood salt levels. Some volunteers will also have there Achilles tendons scanned at registration.

5. **Chronic Achilles tendon injuries in triathletes**

Chronic Achilles tendon injuries are common in athletes participating in weight-bearing sports. It is well established that repetitive forces that are applied to the Achilles tendon (such as during running) may cause microscopic damage to the tendon. In the initial phases this may not cause any symptoms (pain or swelling). However, these changes can be observed using a technique known as soft tissue diagnostic ultrasound (non painful scan of the tendon). In this component of the study we wish to assess the changes in the Achilles tendon before and then after (immediately and 6 weeks later) the Ironman. In particular we wish to find out what damage (if any) takes place in the tendon as a result of the race, and how does this recover after 6 weeks. The findings of this study will also be linked to the genetic basis component (described in 4 above). Here we will be able to determine if your genetic make up determines how your tendons respond to a race such as the Ironman.

6. **The relationship between training history, perception of effort during the race and the subsequent recovery after the race.**

   The relationship between training history, perception of effort during the race and the subsequent recovery after the race is poorly understood. Knowing more about this relationship is important as it will have practical implications for the preparation for the race and minimise any health risks associated with too much physical stress which may occur after the race. Volunteers for this study will be asked to complete a short questionnaire on their training habits in preparation for the Ironman. During the race subjects will be asked to shout out a "perception of effort" score at they go past one of the 8 stations along the route. A researcher at the station will record the race number and the score. Volunteers will be sent emails on a daily basis for a week after the race with a short questionnaire on their recovery. Thereafter, they will be sent an email on a weekly basis for 12 weeks. Volunteers living near a big centre will be asked to donate a small blood sample at 1, 3, 5, 7 and 9 days after the race for the measurement of creatine kinase, a marker of muscle damage (however, blood donations are not essential for entry into the study)

If I decide to participate in the research study, what will be required of me?

The following table summarises the details of your participation in the study:-

	Details of Your Participation in the Study
Before Race	1. Download information sheets, questionnaires and consent forms from web page 2. Complete questionnaires using Microsoft Word 3. E-mail completed questionnaires to researchers at ironman@sports.uct.ac.za
At Registration	1. Hand in and sign the informed consent forms 2. Donate a sample of blood Ultrasound scan of both Achilles tendons in some athletes (Achilles Tendon component - No 5) Donate a saliva sample and have a throat swab (URT component - No 3)
Before Swim (Race Day)	1. Have yourself weighed near the start of the swim before donning your wetsuit in your costume
During Race	1. Shout out a "perception of effort" score at they go past one of the 8 stations along the route (RPE component - No 6)

Immediately After Race (Medical Tent)	1. Have yourself weighed in your running gear, without shoes, at the medical tent 2. Donate a sample of blood Ultrasound scan of both Achilles tendons in those athletes who had a scan during registration (Achilles Tendon component - No 5) Donate saliva samples and have throat swabs (URT component - No 3) Treatment of athletes with cramps and testing of unaffected volunteers (cramps component- No 2) Treatment of the collapsed athletes (collapsed athlete component - No 1)
Continuing Follow-up	**At 6 Weeks:** Ultrasound scan of both Achilles tendons (Achilles Tendon component - No 5) **Daily for 2 Weeks:** Complete symptoms questionnaire, available for telephonic surveillance calls every second day, **and only if required,** visit a designated centre for a clinical examination, donation of saliva and blood samples and have a throat swab taken (URT component - No 3) **For 12 weeks after the race:** Complete a short electronic questionnaire on your recovery daily for a week after the race, thereafter, on a weekly basis for 12 weeks. Volunteers living near a big centre will be asked to donate a blood sample at 1, 3, 5, 7 and 9 days after the race (however, blood donations are not essential for entry into this component of the study) (RPE component - No 6)

We look forward to meeting you at the Spec-Savers Ironman South Africa Registration area, and wish you well in your race preparation and participation.

Prof Martin Schwellnus, Dr Malcolm Collins, Prof Tim Noakes, and the rest of the Ironman Research Team

Appendix 2

SUBJECT INFORMATION SHEET

Dear Triathlete

We have the privilege to inform you that scientific research at the Port Elizabeth IRONMAN triathlon has been planned in collaboration with the UCT/MRC Research Unit for Exercise Science and Sports Medicine based at the Sports Science Institute of South Africa. This will provide a unique opportunity for a research programme to address important medical and physiological problems associated with the IRONMAN triathlon. Each participant will be able to access a summary of the findings of the study by email, and the website, once it has been completed. You will also be given the opportunity to attend a feedback meeting where the results of the study will be discussed. The results will only be that of the whole group, and no individual results will be made public.

The research study will concentrate on the following 6 main components that will ultimately improve your performance and improve the standard of your medical treatment at future triathlons and other endurance events:

- Management of the collapsed triathlete
- Exercise-associated muscle cramping
- Post-exercise upper respiratory tract symptoms
- Genetic basis for performance and physiological responses during an Ironman Triathlon
- Chronic Achilles tendon injuries in triathletes
- The relationship between training history, perception of effort during the race and the subsequent recovery after the race.

As a participant in the Port Elizabeth IRONMAN Triathlon, you will be given the choice to participate in this research effort. Your participation is entirely voluntary. Please read through the details of the following six components of the study. You will be given the opportunity to participate in one or more components of the study. The details of each component are explained in this document, and if you wish to participate in one or more components of the study, please read through and sign the INFORMED CONSENT FORMS that relate to each component of the study. Please feel free to contact members of the research team should you have any questions related to the study (or any component of the study). Contact details of the research team are as follows: ironman@sports.uct.ac.za or (021) 650 4572.

SUBJECT INFORMATION SHEET:

COMPONENTS OF THE RESEARCH STUDY TO BE CONDUCTED AT THE 2006 IRONMAN TRIATHLON IN PART ELIZABETH

The research study at the 2006 Ironman Triathlon, comprise of six components. The detailed information on each of these components of the study is as follows:

Component 1: A study on the management of the collapsed triathlete

General information:

The aim of this study is to evaluate the optimum treatment strategies for which to treat collapsed triathletes, after an Ironman race. Although intravenous (IV) fluid replacement is a common practice in the treatment of collapsed triathletes, medical personnel need to be advised of a treatment method that will prevent possible fluid overload (hyponatraemia) which can be a very severe condition. Your participation in this trial will aid in the understanding and management of how best to correct any fluid imbalance following this race.

If you collapse during or after the Ironman Triathlon and are brought into the medical tent, you will be evaluated and treated according to the current best standard of care principles. Your legs will be elevated and your heart rate, blood pressure, mental status and serum sodium concentration will be measured. If you are confused and your sodium level is normal, other laboratory tests will be performed such as an evaluation of your body temperature and blood sugar levels. If your body temperature is normal and do not have evidence for another treatable medical condition, an IV line will be placed in your arm and the appropriate fluid will be administered - IV or oral fluid (ad libitum – you choose how much you wish to drink) - until you recover and can leave the medical tent without assistance. Your discharge will be at the discretion of the supervising medical officer. If your condition deteriorates at any time, you will be immediately removed from the trial, treated appropriately and transported to the nearest hospital.

The risk of adverse affects of placement of an intravenous line include: infection, delayed healing, bruising, physical pain, mental discomfort and possible injury to a nerve or vessel. The risk of these adverse effects are rare and every attempt to minimize these risks will be undertaken by the use of sterile technique and use of disposable, single use, material. Your blood will be used for evaluation of serum sodium or blood glucose concentration only. No other tests will be performed on your blood and your blood samples will be appropriately discarded after these tests are performed.

We will obey the strict practices of confidentiality and anonymity. Each subject's identity will be known only to the researchers and numbers will be assigned to each sample in lieu of names. No results will be publicly available and the scientific publication of results will never disclose subject identity.

Potential risks of this component of the study

- The completion of a questionnaire is not associated with any risk. Questionnaire and other clinical data (paper and electronic) will be kept confidential, will be kept secure, and will not be made available to any party other than the research team without the consent of the individual subjects.
- The potential risks to subjects of blood collection are minimal and are related to 1) blood sample collection technique, and 2) the volume of blood collected prior to racing and the potential risk of a decreased performance in the race. The potential risks associated with blood collection technique from the ante-cubital veins are: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or a vessel. These risks are small and will be minimized by the use of trained phlebotomists, use of sterile techniques and the use of disposable, single use materials. The risk of decreased performance as a result of blood collection will be reduced by not subjecting any participant to the collection of a blood volume exceeding 15ml prior to the race.
- Body weight will be measured using a standard electronic scale, and there is no risk associated with this procedure.
- The risks associated with participation in this component of the study do not exceed the risks associated with competing in the Ironman competition. The administration of IV fluids will involve an invasive placement of an intravenous line. The risks associated with the placement of an intravenous line include: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or vessel. These risks will be minimized by the use of trained phlebotomists, sterile technique and disposable, single use materials. If at any time the condition of a collapsed triathlete deteriorates, the most appropriate treatment will be initiated, the trial terminated and the patient will be transported to the local hospital if necessary. The support form the local hospital is part of the normal standard medical care associated with this event.

Potential benefits of this component of the study

- The data collected in this component of the study will aid in the development of optimal treatment strategies for collapsed triathletes. Although intravenous fluid replacement is a common practice in the treatment of collapsed triathletes, medical personnel need to be advised of a more judicious approach to treatment as to avoid the deleterious effects of fluid overload (hyponatraemia). This information will aid in the understanding and management of serum sodium disorders in collapsed triathletes by scientifically 1)

evaluating the efficacy of intravenous versus oral rehydration and 2) assessing if the normalization of serum sodium levels are important in the recovery of collapsed triathletes.

Component 2: A study to determine the cause of Exercise-Associated Muscle Cramping (EAMC)

General information

The purpose of this component of the study is to determine the possible cause of exercise-associated muscle cramping (EAMC) in endurance athletes. At registration, triathletes will be given the opportunity to volunteer to participate in this component of the study.

Details of the study are as follows:

- Prior to or at registration, a questionnaire detailing personal particulars, medical information, training information, and history of muscle cramping will be completed.
- At registration, a blood sample (5ml – 1 teaspoon) will be collected from the vein in the arm using standard procedures.
- Body weight will be determined at the time of registration, and on the morning before the race starts by stepping onto an electronic scale
- Should you develop muscle cramping during or immediately after the race, and if you agree to participate, you will be admitted to a designated area of the medical facility at the finish of the race.
- At the finish your core body temperature will be measured using a rectal thermometer. This procedure will take place in privacy, and entails placing a thermometer in the rectum (backside) for about 3 minutes. This procedure may be associated with mild discomfort but no pain. Normal precautions will be taken to ensure that the thermometer is clean and properly lubricated. Trained medical staff will perform this procedure.
- Disposable surface patches (electrodes) will be attached to your cramping muscle/s and also to your arm (back of the arm on the triceps muscle) to record the electrical activity of the muscles. This procedure is not associated with any pain or discomfort.
- During the time of your admission to the medial facility you will be treated for cramping using standard accepted medical procedures.
- You will be asked to stand and walk periodically (every 15min), unless you are still actively cramping. Once you are able to stand and walk with no cramping, you will be discharged from the medical facility.
- Should you develop any medical complications or if your condition deteriorates, you will be treated according to normal accepted medical practices, and this can include admission to hospital if required.

Potential risks of this component of the study

- The completion of a questionnaire is not associated with any risk. Questionnaire and other clinical data (paper and electronic) will be kept confidential, will be kept secure, and will not be made available to any party other than the research team without the consent of the individual subjects.
- The potential risks to subjects of blood collection are minimal and are related to 1) blood sample collection technique, and 2) the volume of blood collected prior to racing and the potential risk of a decreased performance in the race. The potential risks associated with blood collection technique from the ante-cubital veins are: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or a vessel. These risks are small and will be minimized by the use of trained phlebotomists, use of sterile techniques and the use of disposable, single use materials. The risk of decreased performance as a result of blood collection will be reduced by not subjecting any participant to the collection of a blood volume exceeding 15ml prior to the race.
- Body weight will be measured using a standard electronic scale, and there is no risk associated with this procedure.
- All medical conditions, including EAMC, will be treated appropriately, based on the current standard of care or evidenced based paradigms. If at any time the condition of a triathlete with EAMC deteriorates, the most appropriate treatment will be initiated, the trial terminated and the patient will be transported to the local hospital if necessary. The support form the local hospital is part of the normal standard medical care associated with this event. Surface electrode placement and measurement of EMG activity is not associated with any known risk to the subject.

Potential benefits of this component of the study

- The anticipated benefits of this component of the study are that the results will further our understanding of the possible cause/s of EAMC in endurance athletes. In particular, once the aetiology of EAMC is better understood, this will improve our ability to prevent this condition, and to treat it effectively if it does occur.

Component 3. A study to determine the cause of post-exercise upper respiratory tract symptoms

General information

Upper respiratory tract (URT) symptoms such as a sore throat, runny or blocked nose, and throat irritation are particularly common in ultra distance athletes including triathletes. These symptoms occur mostly in the 2 weeks after a race. It has been shown to occur in 30-50% of all athletes after endurance events. It is important to understanding the relationship between exercise and URT symptoms as it is known that infections have potential negative effects for the athlete. Having an infection or not may mean the difference between being able to compete safely, performing at a sub-optimum level at risk, or missing the event altogether because of illness. In recent years we have become aware that the symptoms of URT infections that endurance athletes suffer from after a race may NOT be caused by an infection. Instead this may reflect an irritation of the inner cell lining of the nose and throat due to allergy or perhaps pollution. However, we still need more evidence to prove this.

The aim of this component of our research is to determine if the symptoms experienced by athletes after an Ironman race are due to an infective cause (microbial agent such as a virus or a bacteria) or due to a non-infective inflammatory process in the upper respiratory tract.

The study will involve recruiting in excess of 120 triathletes who participate in the Port Elizabeth IRONMAN endurance race. You will be requested to report to a specific area at the registration desks in the 3 days prior to the event. At this time you will be asked to complete a questionnaire, and have a blood sample taken from your vein in the forearm. In addition nasal and throat swabs will be taken and you will be required give a specimen of your saliva (spit).

Immediately after you finished the race, you will be asked to report to a specific section of the medial tent at the finish, where a further blood sample and saliva sample will be taken.

You will then be asked to be available for a follow up in the 14 days after the race. Follow-up will take place in four cities (Cape Town, Port Elizabeth, Durban and Gauteng).You will be required to complete a short symptom chart every day, and you will be contact regularly (every 2 days) by a member of the research team to obtain this information. Should you develop any symptoms of upper respiratory tract irritation (such as blocked nose, runny nose, sore throat, cough) you will be asked to report to a research centre in the city (as mentioned above). There will be no financial compensation to attend this centre, but the medical consultation will be free of charge. During that visit you will be seen by a doctor, who will take a medical history, and conduct a medical examination of your upper respiratory tract (ears, nose throat and chest). In addition a blood and saliva samples will be taken. You will receive treatment and advice for the management of these symptoms.

Potential risks of this component of the study

- The completion of a questionnaire is not associated with any risk. Questionnaire and other clinical data (paper and electronic) will be kept confidential, will be kept secure, and will not be made available to any party other than the research team without the consent of the individual subjects.
- The potential risks to you during blood collection are minimal and are related to 1) blood sample collection technique, and 2) the volume of blood collected prior to racing and the potential risk of a decreased performance in the race. The potential risks associated with blood collection technique from the ante-cubital veins are: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or a vessel. These risks are small and will be minimized by the use of trained phlebotomists, use of sterile techniques and the use of disposable, single use materials. The risk of decreased performance as a result of blood collection will be reduced by not subjecting any participant to the collection of a blood volume exceeding 15ml prior to the race.
- The potential risks associated with the collection of saliva and throat swabs are minimal. Local minimal and transient discomfort in the upper respiratory tract is the only anticipated risk. The collection procedure will be conducted by trained staff.

Potential benefits of this component of the study

- The anticipated benefits to subjects participating in this component of the study are firstly that the knowledge of the cause of the symptoms of the URT after an endurance event will be known, secondly that the treatment of these symptoms will be based on sound scientific and clinical evidence and finally, that triathletes can be given accurate and safe advice on training during the recovery period.

Component 4: A study to determine the genetic basis for performance and physiological responses during an Ironman Triathlon

General information

A study to determine the genetic basis for performance and physiological responses during an Ironman Triathlon will be conducted by the UCT/MRC Research Unit for Exercise Science and Sports Medicine at the University of Cape Town in Cape Town, South Africa, in conjunction with the Molecular Genetics Department B and Laboratory of Forensic Genetics of the Cyprus Institute of Neurology and Genetics in Nicosia, Cyprus.

The study involves donate ten millilitres (2 teaspoons) of venous blood and this will be done at race registration and after the race (five millilitres - 1 teaspoon). Five millilitres of the sample will be used for the extraction and analysis of genetic material (DNA), while the remainder of the sample will be used to measure serum electrolyte (salt) levels. In addition,

body weight will be measured prior to the start of the race and again in the medical tent on completion of the race.

The DNA will only be used for scientific research purposes relating to the genetic basis of (1) athletic ability, (2) tendon and ligament overuse injuries and (3) dysnatraemia during ultra-endurance events. Personal particulars and sporting and medical questionnaires will have to be completed and this information will be treated with the strictest confidentiality and will only be used for scientific research purposes. All data will be analysed anonymously and DNA samples will be destroyed on completion of the study.

Part of the DNA extracted form the donated blood sample will be sent to the Cyprus Institute of Neurology and Genetics in Cyprus for analysis. DNA samples will be shipped to and analysed in Cyprus anonymously. DNA will be genotyped (analysed) for variations (polymorphisms) within genes relating to the genetic basis of athletic ability, tendon and ligament overuse injuries as well as water and salt imbalance during ultra-endurance events only.

Potential risks of this component of the study

- The completion of a questionnaire is not associated with any risk. Questionnaire and other clinical data (paper and electronic) will be kept confidential, will be kept secure, and will not be made available to any party other than the research team without the consent of the individual subjects.
- The potential risks to you during blood collection are minimal and are related to 1) blood sample collection technique, and 2) the volume of blood collected prior to racing and the potential risk of a decreased performance in the race. The potential risks associated with blood collection technique from the ante-cubital veins are: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or a vessel. These risks are small and will be minimized by the use of trained phlebotomists, use of sterile techniques and the use of disposable, single use materials. The risk of decreased performance as a result of blood collection will be reduced by not subjecting any participant to the collection of a blood volume exceeding 15ml prior to the race.

Potential benefits of this component of the study

The anticipated benefits of this component of the research study are to identify genetic factors that may predispose to 1) improved performance or 2) increased risk of medical consequences (such as abnormal electrolyte imbalances). This information will eventually assist triathletes in predicting and improving their performance, and decrease their risk of medical complications during participation in triathlon.

Component 5. A study to determine the genetic risk/s associated with chronic Achilles tendon injuries in triathletes

General information

The purpose of this component of the research study is to determine if there are specific genetic factors (refer to the details for component 4) that are associated with the development of chronic tendon injuries. In addition, we want to determine what the effect of an endurance event (such as the Ironman) is on the structure of the Achilles tendon.

At registration you will be required to complete a questionnaire with personal details, training details, past injury details, and details about family history. In addition, a 5ml (1 teaspoon) blood sample will be taken from a vein in your arm. Finally, a qualified radiologist will examine both you Achilles tendons using a soft tissue diagnostic ultrasound machine. This procedure entails putting a clear jelly on your skin, and then using a probe to examine the tendon by passing it over the skin. This is not associated with any discomfort.

After you complete the race, you will be asked to undergo the same procedure (blood collection and ultrasound examination) in the medical facility at the finish. If possible, you will be asked to report to a medical centre close to your home for a final ultrasound examination approximately 6 weeks after the race. The cost of this will be free, but you will not receive any financial compensation to attend this centre.

Potential risks of this component of the study

- The completion of a questionnaire is not associated with any risk. Questionnaire and other clinical data (paper and electronic) will be kept confidential, will be kept secure, and will not be made available to any party other than the research team without the consent of the individual subjects.
- The potential risks to you during blood collection are minimal and are related to 1) blood sample collection technique, and 2) the volume of blood collected prior to racing and the potential risk of a decreased performance in the race. The potential risks associated with blood collection technique from the ante-cubital veins are: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or a vessel. These risks are small and will be minimized by the use of trained phlebotomists, use of sterile techniques and the use of disposable, single use materials. The risk of decreased performance as a result of blood collection will be reduced by not subjecting any participant to the collection of a blood volume exceeding 15ml prior to the race.
- Soft tissue diagnostic ultrasound is a well described and common clinical diagnostic procedure that is associated with no known risk. This procedure will be undertaken by a trained radiologist.

Potential benefits of this component of the study

- The anticipated benefits of this component of the study are that the results will clarify why certain triathletes may be more or less prone to chronic tendon injuries, based on their genetic make-up. In future, this work may lead to the screening and early identification of an increased risk for tendon injuries, so that preventative measures can be undertaken.

Component 6: A study to determine the relationship between training history, perception of effort during the race and the subsequent recovery after the race

General information

The purpose of this component of the study is to investigate whether the strain experienced in the recovery period after an Ironman is directly proportional to the perception of effort and racing intensity in a group of similarly trained triathletes. The answer to this question has a practical application for training and also contributes to a better understanding of the physiological responses of ultra endurance events.

The research project will involve the following:

- About 1 week before the race you will be asked to complete a questionnaire on your training habits for swimming, cycling and running in preparation for the Ironman and your personal best times for the 3 disciplines. This will take about 30 minutes.
- You will be familiarised with the subjective scores for "perception of effort rating" and "pain assessment" before the race.
- During the race researchers will be allocated to about 12 stages throughout the race. As you swim, run or cycle past these researchers they will hold up two boards with the scores for "perception of effort rating" and "pain assessment". You will be asked to shout out your respective scores as you go past them and they will record these scores against your race number.
- You will be sent an email on a daily basis for a week after the race with a short questionnaire on your subjective perception of recovery. This questionnaire will take about 2 minutes to complete. Thereafter, you will be sent an email on a weekly basis for 12 weeks with the same short questionnaire.
- Blood samples after the race will be obtained 1, 3, 5, 7 and 9 days later for the measurement of creatine kinase.

Potential risks of this component of the study

- The completion of a questionnaire is not associated with any risk. Questionnaire and other clinical data (paper and electronic) will be kept confidential, will be kept secure, and will not be made available to any party other than the research team without the consent of the individual subjects.

- The potential risks to you during blood collection are minimal and are related to 1) blood sample collection technique, and 2) the volume of blood collected prior to racing and the potential risk of a decreased performance in the race. The potential risks associated with blood collection technique from the ante-cubital veins are: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or a vessel. These risks are small and will be minimized by the use of trained phlebotomists, use of sterile techniques and the use of disposable, single use materials. The risk of decreased performance as a result of blood collection will be reduced by not subjecting any participant to the collection of a blood volume exceeding 15ml prior to the race.
- Data for this component of the study will involve contact with subjects during the race. There is a potential risk that in the process of data collection, the performance of subjects in the race will be interfered with. This risk will be minimal, as the nature of the data collection is such that subjects will only be asked to shout out two numbers as they pass members of the research team at designated points in the race. However, should triathletes feel that this affects their performance during the race, they will be free to withdraw from this component of the study. There will be no interference with other race participants during this data collection process.

Potential benefits of this component of the study

- The anticipated benefits of this component of the study are firstly that subjects will receive a full summary of their individual results, as well as the overall findings from this component of the study. Secondly, and more specifically, the individual results will include information about their training and development of fatigue during the race which will be of interest. Finally, these results may assist triathletes in modifying their training to improve their performance.

Appendix 3

INFORMED CONSENT FORM

THE PORT ELIZABETH IRONMAN TRIATHLON 2006: MEDICAL CONSEQUENCES FOLLOWING ENDURANCE SPORTS RESEARCH PROJECT

I, _____, agree voluntarily to participate in the UCT/MRC Research Unit for Exercise Science and Sports Medicine's research project with the following components titled:-

- "A study on the management of the collapsed triathlete",
- "A study to determine the cause of Exercise Associated Muscle Cramping (EAMC)",
- "A study to determine the cause of post-exercise upper respiratory tract symptoms",
- "A study to determine the genetic basis for performance and physiological responses during an Ironman Triathlon",
- "A study to determine the genetic risk/s associated with chronic Achilles tendon injuries in triathletes",
- "A study to determine the relationship between training history, perception of effort (RPE) during the race and the subsequent recovery after the race",

performed by the University of Cape Town and the Sports Science Institute of South Africa. I have read the subject information sheets and the following procedures and concepts have been explained to me in full:

1. Completion of a questionnaire: The completion of a questionnaire is not associated with any risk. Questionnaire and other clinical data (paper and electronic) will be kept confidential, will be kept secure, and will not be made available to any party other than the research team without the consent of the individual subjects.

2. Blood sample collection at registration, immediately after the race, and if required in the 14 days after the race: The potential risks to subjects of blood collection are minimal and are related to 1) blood sample collection technique, and 2) the volume of blood collected prior to racing and the potential risk of a decreased performance in the race. The potential risks associated with blood collection technique from the ante-cubital veins are: infection, delayed healing, haematoma, physical pain, mental discomfort and injury to a nerve or a vessel. These risks are small and will be minimized by the use of trained phlebotomists, use of sterile techniques and the use of disposable, single use materials. The risk of decreased performance as a result of blood collection will be reduced by not subjecting any participant to the collection of a blood volume exceeding 25ml prior to the race.

3. Measurement of body weight before and after the race: Body weight will be measured using a standard electronic scale, and there is no risk associated with this procedure.

4. Treatment if I collapse after the race: **(only for the collapsed athlete component)** If I collapse during or after the race I might receive either IV (drip into arm vein) or oral fluids ad libitum (as much fluid as I want). I will be attended to in a separate section of the medical tent under the supervision of a qualified doctor. I will be assessed regularly (every 15 minutes) and I understand that optimum care will be provided to me according to the current standard of care. Treatment will cease when I am alert, oriented, able to walk and when my laboratory tests are normal. I will be transported to the local hospital if my condition requires more urgent medical attention.

5. Treatment if I develop muscle cramps during or after the race: **(only for the cramps component).** If I develop muscle cramps during after the race I will receive treatment in

a designated area of the medical facility. Optimum care will be provided to me according to the current standard of care. I will be required to have a rectal temperature measurement taken, blood samples will be collected, body weight will be measured, and I will have surface electrodes attached to my muscle to measure electrical activity. Treatment will cease when my cramps have stopped and I am able to stand up and walk. I will be transported to the local hospital if my condition requires more urgent medical attention.

6. Saliva sample collection at registration, immediately after the race, and if required in the 14 days after the race: **(only for the URT component)** The potential risks associated with saliva sample collection are very small. I may experience transient discomfort as the inner lining of my throat is swabbed with a soft swab. I understand that all the normal precautions will be taken during this procedure, and that it will be undertaken by trained staff.

7. Assessment and treatment of symptoms of the upper respiratory tract in the two weeks after the race: **(only for the URT component)** I understand that should I develop any symptoms of the upper respiratory tract in the 14 days after the race, I will be required to report to a research centre in my home town, to be examined by a doctor, give a blood sample and have a throat swab as well a saliva sample taken. I understand that I will then be treated for my symptoms according to standard medical practice. I understand that I will not receive any financial compensation to attend the centre.

8. Soft tissue diagnostic ultrasound examination: **(only for the Achilles tendon component)** I understand that I will be subjected to a soft tissue diagnostic ultrasound examination of my Achilles tendons during the registration period, on completion of the race, and if possible 6 weeks after the race at a medical facility close to my home. I understand that I will not receive any direct financial compensation to attend this centre for the ultrasound, but that the investigation will be free of charge. I understand that these investigations are not associated with any risk, and will be performed by a trained radiologist.

9. The genetic basis for performance and physiological responses during an Ironman Triathlon as well as to determine the genetic risk/s associated with chronic Achilles tendon injuries in triathletes: **(only for the genetics components).** These components of the study are been performed in conjunction with the Molecular Genetics Department B and Laboratory of Forensic Genetics of the Cyprus Institute of Neurology and Genetics in Nicosia, Cyprus. At race registration, I have agreed to donate ten ml (2 teaspoon) of venous blood. Half the sample will be used for the extraction and analysis of genetic material (DNA), while the remainder of the sample will be used to measure serum electrolyte (salt) levels. I also agree to donate an additional five ml (1 teaspoon) of venous blood after the race in the medical tent which will be used to measure post-race serum electrolyte (salt) levels.

   The DNA will only be used for scientific research purposes relating to the genetic basis of (1) athletic ability, (2) tendon and ligament overuse injuries and (3) dysnatraemia during ultra-endurance events. I also understand that all data will be analysed anonymously and my DNA sample will be destroyed on completion of the study. I understand that some of the DNA extracted form the donated blood sample will be sent to the Cyprus Institute of Neurology and Genetics in Cyprus for analysis. I understand that the DNA samples will be shipped to and analysed in Cyprus anonymously. I understand that the DNA will be genotyped (analysed) for variations (polymorphisms) within genes relating to the genetic basis of athletic ability, tendon and ligament overuse injuries and dysnatraemia during ultra-endurance events only.

   I understand that whilst there is no direct benefit to myself, if a genetic predisposition for (1) athletic ability, (2) tendon and ligament overuse injuries and (3) dysnatraemia during ultra-endurance events can be established, then future generations will be able to establish their risk for this condition. This may allow better prevention and treatment options in the future. I understand that I will receive the overall results of the study.

I have read (or, where appropriate, have had read to me) and understood the information about this study, and any questions I have asked have been answered to my satisfaction. I agree to participate in the study, realising that I have the right to request that my DNA sample be destroyed at anytime. I agree that research data provided by me or with my permission during the project may be included in a thesis, presented at conferences and published in journals on the condition that neither my name nor any other identifying information is used.

10. Providing information on my rating of effort and fatigue status during the race: **(only for the RPE component)**I understand that I will be required to study and familiarize myself with two scales of perceived effort and fatigue before the race starts. I understand that during the race, at designated stages, I will be required to report (by shouting) my perception of effort and fatigue to members of the research team.

I have read the preceding subject information sheet and understand the testing procedures outlined therein. I understand any accompanying risks and discomforts. Knowing these risks and discomforts and having had the opportunity to pose questions answered to my satisfaction, I hereby consent to participate in this study. I understand that I may withdraw from this study at any time without further question. I have been informed that the individual data derived from my participation in these protocols will remain confidential. I understand that the medical staff and the research team have professional medical insurance.

Name of the triathlete: _____
Signature of triathlete _____

Date: _____

Name of investigator: ___Prof Martin Schwellnus_____

Signature of Investigator: _____

Date: _____ _____

Appendix 4

Department of Human Biology

UCT/MRC RESEARCH UNIT FOR EXERCISE SCIENCE & SPORTS MEDICINE
Faculty of Health Sciences, University of Cape Town
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2006 IRONMAN – MEDICAL AND TRAINING QUESTIONNAIRES

These questionnaires have been constructed by the Medical Research team, in conjunction with the Medical Director of the Ironman 2006. The information obtained from these questionnaires is essential for the planning of medical care during events such as the Ironman 2006. We acknowledge that the questionnaires are long, but we are asking about 20 minutes of your valuable time to complete them. The completion of the questionnaires is voluntary, all the information will be kept confidential and will only be used for research and medical care planning purposes. We suggest that you consider completing this before the event, or at the time of registration.

Prof Martin Schwellnus (Chairman, Research Team)
Dr Peter Schwartz (Medical Director, Ironman 2006)

Instructions

You can either complete the questionnaires electronically using Microsoft word or print the questionnaires and complete them manually. Please answer each question by filling in the details in the allocated space or checking one or more of the option boxes.

If you complete the questionnaire electronically using Microsoft word, please e-mail the completed forms to ironman@sports.uct.ac.za and bring the signed consent form to the research table at race registration.

If you complete the questionnaire manually, please bring the completed forms together with the signed consent form to the research table at race registration.

Please complete sections A, B, C, D and E

Please complete only the relevant questions in the following section

Section A: Personal details			
2006 Ironman Race Number			
Surname			
First Name			
Postal Address		Postal/ Zip Code	
E-mail address		Phone (day time)	code number
Date of birth	y y y y - m m - d d	Cell	
Height	cm	Gender	Male ☐ Female ☐
Weight	kg	Age	
Ethnic group (Only Required and Used for Research Purposes)	Black/African ☐ White ☐ Indian ☐ Mixed Ancestry (Coloured) ☐ Asian ☐ Other ☐		
Ancestry: Tribal or national background (eg Xhosa, Dutch, Zulu, German, Italian)	Father: Unknown ☐		
	Mother: Unknown ☐		
Country of Birth			
Dominant Hand	Left ☐ Right ☐ Both ☐	Dominant Leg	Left ☐ Right ☐ Both ☐
Occupation			
What **percentage** of your **working** day is spent in the following activities?	Sitting: ____ % Standing: ____ % Walking (Lower body activity) ____ % Manual Labour (upper and body activity) ____ %		

Section B. Racing and training history

Type of triathlon	Sprint	Standard (1.6, 40, 10)	½ Ironman	Ironman
Which triathlons have you **ever** participated in?	Yes ☐ No☐	Yes ☐ No☐	Yes ☐ No☐	Yes ☐ No☐
Year of first event				
How many events have you **ever** participated in?				
How many Olympic (or above) triathlon races have you completed over the **past 2 years**?				
Personal best time **ever**	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min
What was your time for your last triathlon race during the **past 12 months**?	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min

Type of running event	5 km	10 km	21.1 km	42.2 km
Which races have you **ever** participated in?	Yes ☐ No☐	Yes ☐ No☐	Yes ☐ No☐	Yes ☐ No☐
Year of first event				
How many events have you **ever** participated in?				
Personal best time **ever**	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min
What is your best time, in a running race, in the **last 15 weeks**?	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min

Type of event	Two Oceans Marathon	Comrades Marathon
Which races have you **ever** participated in?	Yes ☐ No☐	Yes ☐ No☐
Year of first event		
How many events have you **ever** participated in?		
Personal best time	_____ hrs:min	_____ hrs:min

What is your best average cycling speed (km/h) in a race over 80 km in the **last 15 weeks**?	Average speed: _____ Distance: _____	km/h; km
What is your best swimming performance in the **last 15 weeks**?	Time: _____ Distance: _____	min m
What is your predicted time for the entire 2006 Ironman event and each of the three splits?	Entire event: _____ Swim: _____ Cycle: _____ Run: _____	min min min min

Please answer the following questions, with your answers reflecting your average in the **most recent 15 weeks i.e. beginning December 2005 to 18th March, 2006.**	
How many days a week did you train during the **last 15 weeks**?	days/week
What distances did you train in an average week during the **last 15 weeks**?	Swim: _____ km/week Cycle: _____ km/week Run: _____ km/week
How many hours a week did you train in an average week during the **last 15 weeks**?	Swim: _____ hrs/week Cycle: _____ hrs/week Run: _____ hrs/week
What **distances** did you train in the week before the race?	Swim: _____ km Cycle: _____ km Run: _____ km
How many **hours** did you train in the **week before** the race?	Swim: _____ hours Cycle: _____ hours Run: _____ hours

Flexibility training history	
Do you perform flexibility training (stretching exercises)?	Yes ☐ No ☐
If YES, please complete the rest of the flexibility training history section below:- If NO, continue completing the questionnaire from the top of page 5 (Equipment use history).	
On average, how many days a week do you perform a stretching session?	days/week
On average, how times a day do you perform a stretching session?	times/day
Please tick which muscle groups do you include in your stretching session?	☐ Hamstrings ☐ Quadriceps ☐ Calf (gastrocnemius) ☐ Calf (soleus) ☐ Groin (inner thigh) ☐ Upper body limbs ☐ Other: _____
Please tick when you stretch? (before, during and/or after exercising. You can tick more than one box)	☐ Before Exercise ☐ During Exercise ☐ After Exercise
When you stretch an individual muscle group, on average, **how long do you hold the stretch** for?	seconds
When you stretch an individual muscle group, on average, **how many times do you stretch the muscle for**?	☐ Once ☐ Twice ☐ 3 times ☐ 4 times ☐ 5 times ☐ 6 or more times

Equipment use history	
Please indicate which type of **bicycle** you use?	☐ Kuota ☐ Kestrel ☐ Trek ☐ Aegis ☐ Litespeed ☐ Softride ☐ Felt ☐ Quintana Roo ☐ Javelin ☐ Cervelo ☐ Argon 18 ☐ Scott ☐ Elite ☐ Specialized ☐ Guru ☐ Giant ☐ Other: _____
Please indicate which type of **handle bars** you use?	☐ Bontrager ☐ HED ☐ Zipp ☐ Profile Design ☐ Vision Tech ☐ Oval Concepts ☐ Deda ☐ Easton ☐ Syntace ☐ Pedalsoft ☐ Kestrel ☐ Other: _____
Please indicate which type of **saddle** (Brand - model) you use?	☐ Selle San Marco- Azoto TriathGel ☐ Profile Design- Tri Stryke (with a groove) ☐ Selle San Marco- Rever Profil ☐ Fizik- Arione Tri ☐ Terry ☐ Koobi ☐ Other: _____
Please indicate which brand of **helmet** you use?	☐ Trek ☐ Bell ☐ Giro ☐ MET ☐ Other: _____
Please indicate which type of **cycling shorts** you use?	☐ Thin lycra (no padding) ☐ Padded cycling shorts ☐ Triathlon shorts with some padding ☐ Swimming costume ☐ Other: _____
Do you normally wear **underwear** together with cycling shorts?	☐ Yes ☐ No
Please indicate which type of **cycling shoes** you use?	☐ Olympic ☐ Nike ☐ Diadora ☐ Shimano ☐ Carnac ☐ Sidi ☐ Other: _____
Please indicate which type of **kit** you use?	☐ Anatomic ☐ Nike ☐ Velo ☐ Howzit ☐ Adidas ☐ Orca ☐ De Soto ☐ Louis Garneau ☐ Quintana Roo ☐ Zoot ☐ Other: _____
Please indicate which **brand of running shoe** you use?	☐ Adidas ☐ Asics ☐ Brooks ☐ New Balance ☐ Nike ☐ Mizuno ☐ Puma ☐ Reebok ☐ Saucony ☐ Other: _____
Please indicate which **type of running shoe** you use?	☐ Soft neutral shoe ☐ Mild anti-pronation shoe ☐ Motion control shoe ☐ Light racing shoe ☐ Unknown or not sure ☐ Other: _____

Section C. History of medication and supplement use

	Name of medication	Years taken
What medication, if any, are you currently using? (please list)		

Do you use protective skin sunscreen during training session or when competing?	Yes ☐ No ☐	☐ Every session	☐ Most sessions
		☐ Some sessions	☐ Very occasionally

Are you currently taking dietary supplements/vitamins?	Yes ☐ No ☐

	Name of supplement	Years taken
If **yes** to the above question, please list names of dietary, sports or vitamin supplements.	☐ Multi-vitamins	____
	☐ Anti-oxidants	____
	☐ Immune boosters	____
	☐ Protein powders/supplements, Protein bars. BCAAs	____
	☐ Creatine	____
	☐ Caffeine	____
	☐ Fat cutters	____
	☐ Carbohydrate drinks/powders/gels	____
	☐ Other: _____	____

Have you ever used oral corticosteroids (cortisone tablets)? (If **yes**, how long ago?)	Yes ☐ No ☐	☐ 3 months	☐ 6 months
		☐ 12 months	☐ 24 or more months
Have you ever been given an injection with corticosteroids? (If **yes**, how long ago?)	Yes ☐ No ☐	☐ 3 months	☐ 6 months
		☐ 12 months	☐ 24 or more months
Have you ever been given an injection of corticosteroids in or around the **Achilles** tendon? (If **yes**, how many times?)	Yes ☐ No ☐	☐ Once	☐ Twice
		☐ 3 times	☐ >3 times
Have you ever used fluoroquinoline antibiotics? (refer to the following list)	Yes ☐ No ☐	☐ 3 months	☐ 6 months
		☐ 12 months	☐ 24 or more months

List of some fluoroquinoline antibiotics:

ADCO-CIPRIN	CIPROBAY	SANDOZ CIPROFLOXACIN
AVELON	CIPROGEN	TAFLOC
BACTIDRON	CPL ALLIANCE CIPROFLOXACIN	TARIVID
CIFLOC	DYNAFLOC	TAVANIC
CIFRAN	FACTIVE	TEQUIN
CIPLA-CIPROFLOXACIN	FLOXIN	UNIQUIN
CIPLOXX	MAXAQUIN	UTIN-400
CIPRO-HEXAL	NOROXIN	ZANOCIN
	ORPIC	

Lifestyle and habits history			
Please indicate your smoking status	Current smoker ☐	Ex smoker ☐	Never smoked ☐
If you answered yes, (past or current smoker) please complete the section on the right	Number of years of smoking:	If stopped, how many years ago:	
	What is (was) the average number of cigarettes per day:		
On average, how much alcohol do you drink per week (tots, glasses) of spirits, wine or beer?	_____ glasses beer/cider per week _____ glasses wine per week _____ tots of spirits per week		

Fluid Intake		
How do you best describe your fluid intake during an Ironman triathlon race?	(a) I drink to thirst ☐ (b) I drink as much as tolerable ☐ (c) I drink according to a predetermined fluid intake schedule ☐ (d) I drink to prevent any weight loss during exercise ☐ (e) I combine (a) with (c) ☐ (f) I combine (b) with (c) ☐ (g) Other: _____ ☐	
What percentage of your fluid intake will consist of these beverages?	Water: ☐ 0-25% ☐ 26-50% ☐ 51-75% ☐ 76-100% Sports drink: ☐ 0-25% ☐ 26-50% ☐ 51-75% ☐ 76-100% Coke: ☐ 0-25% ☐ 26-51% ☐ 51-75% ☐ 76-100% Other: ☐ 0-25% ☐ 26-50% ☐ 51-75% ☐ 76-100% Specify other: _____	
What will be your estimated **total** fluid intake be **(if at all)** during the **swim**?		ml
What will be your estimated **total** fluid intake be during the **cycle**?		ml
What will be your estimated **total** fluid intake be during the **run**?		ml
Rank the following sources of information on their importance in formulating your drinking strategy. (1 being most influential and the lowest number being least influential)	_____ Fellow triathletes _____ Coach / trainer _____ Magazines / books _____ Website (please specify: _____) _____ Drinking guidelines from sports associations _____ Adverts _____ Self-experimentation _____ Other: _____	

Section D. Family medical history

Have any of your blood (biological) relatives <u>ever</u> had the following?

Please tick yes or no. If yes, please tick the relationship of that person to you (You may tick more than one of the relationship blocks).

Description		If Yes, please indicate the relationship
Exercise associated muscle cramps	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Night muscle cramps	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Chronic Achilles tendon injury	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Achilles tendon rupture	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Any ligament injury	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Asthma	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Allergies (in general)	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Heart Disease	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother
Diabetes	Yes ☐ No ☐	☐ Father ☐ Mother ☐ Brother ☐ Sister ☐ Child ☐ Grandfather ☐ Grandmother

Section E. Personal general medical history

In this section, you are asked to read through 14 questions about your personal general medical history. If you answer "yes" to any of questions 1 to 12, please complete the additional questions at the end of the section (section F on page 11).

1. In the **6 weeks before this race** (from 1st February) did you suffer from any **symptoms of flu** (fever, sore throat, blocked or runny nose, cough, wheeze, muscle aches and pains)?	Yes ☐ No ☐
2. Have you **ever** in triathlon career suffered from **muscle cramping** during or immediately (within 6 hours) after exercise (in training or competition)?	Yes ☐ No ☐
3. Have you **ever** in your triathlon career suffered from **a tendon or ligament injury** (pain, swelling, stiffness) in any tendon (including Achilles tendon, knee tendons, and shoulder tendons) or ligaments (partial or complete tear)?	Yes ☐ No ☐
4. Have you **ever** in your triathlon career **used medicines to treat injuries** in the week **before or during a race** – including anti-inflammatory drugs, cortisone (pills, or injection), or pain killers?	Yes ☐ No ☐
5. Have you **ever** in your triathlon career suffered **gastrointestinal** symptoms **during exercise** including heartburn, nausea, vomiting, abdominal pain, urge to defecate (pass a stool), diarrhoea, or blood in the stools?	Yes ☐ No ☐
6. Have you **ever** in your triathlon career suffered from symptoms of the **nervous system** including exercise induced headaches, nerve tingling or loss of sensation?	Yes ☐ No ☐
7. Have you **ever** in your triathlon or cycling career (in particular with **cycling**) suffered from **injury to the genital area** including genital numbness after cycling, genital pain after cycling, genital swelling or altered sexual function after cycling?	Yes ☐ No ☐
8. Have you **ever** in your triathlon career suffered from **symptoms of allergies** including nose allergies (hay fever), allergic sinusitis, allergic asthma, skin allergies, a past history of allergies to medication, plant material or animal material?	Yes ☐ No ☐
9. Do you **currently suffer from asthma** including exercise induced asthma, or symptoms of asthma such as shortness of breath, wheezing, or chronic coughing?	Yes ☐ No ☐
10. Have you ever **collapsed** (fell down **not because of an accident,** needing medical attention) during, at the finish or after a race or training session?	Yes ☐ No ☐
11. Do you **currently** suffer from any **symptoms of injury** in the muscles, tendons, bones, ligaments or joints?	Yes ☐ No ☐
12. Do you **currently**, or did you **in the last year**, suffer from any symptoms of **exercise related skin disease**?	Sunburn: Yes ☐ No ☐ Skin cancer: Yes ☐ No ☐ Other skin damage resulting sun exposure: Yes ☐ No ☐
13. Please tick in which anatomical area you ever had **surgery** performed.	☐ Head ☐ Neck ☐ Face ☐ Front chest ☐ Back chest ☐ Shoulder ☐ Upper arm ☐ Elbow ☐ Forearm ☐ Wrist ☐ Finger ☐ Lower back ☐ Hip ☐ Thigh ☐ Knee ☐ Lower leg ☐ Achilles ☐ Ankle ☐ Foot ☐ Abdomen ☐ Other (Specify: _____)

14. **Female athletes only:** Please complete the following questions (14a. to 14g.) related to your menstrual cycle and other gynaecological history	
14a. At what age did you start your periods (menstruating)?	(years)
14b. In the last 12 months, how many menstrual cycles did you have?	
14c. Have you ever had irregular menstrual periods in the past? (excluding pregnancy)?	Yes ☐ No ☐
14d. Have you had a hysterectomy/ovarectomy?	Yes ☐ No ☐
14e. How many times have you been pregnant?	(times)
14f. What form of contraception are you currently using?	☐ None ☐ Oral contraceptive pill ☐ Injection ☐ Intra-uterine device ☐ Sterilization (tubes tied) ☐ Other: _____
14g. If yes to question 14f. above, for oral contraceptive pill, for what reason was the pill prescribed?	☐ Not applicable ☐ Dermatological ☐ Contraception ☐ Regulate period ☐ Other: _____

THANK YOU FOR COMPLETING THIS QUESTIONNAIRE

If you have answered **YES** to any of the first 11 questions of the Personal General Medical History questionnaollow in section F.

If you have completed the questionnaire manually, please bring the completed forms together with the signed consent form to the research table at race registration.

If you have completed the questionnaire electronically using Microsoft word, please e-mail the completed forms to ironman@sports.uct.ac.za and bring the signed consent form to the research table at race registration.

Section F. Additional detailed medical history

(Please complete all the sections to which you answered "Yes" in the Personal general medical history)

1. Flu symptoms in the last 6 weeks

If you answered **YES** to **question 1** in section E, please complete the following two questions related to flu symptoms in the last 6 weeks.

(1a) Please tick which of these flu symptoms you suffered from **in the last 6 weeks**.	☐ Fever ☐ Cough ☐ Joint pains ☐ Blocked nose ☐ Wheezing ☐ Runny nose ☐ Muscle aches ☐ Any other flu symptoms (Specify: _____)
(1b) Please tick which of these flu symptoms you suffered from **in the last 7 days**.	☐ Fever ☐ Cough ☐ Joint pains ☐ Blocked nose ☐ Wheezing ☐ Runny nose ☐ Muscle aches ☐ Any other flu symptoms (Specify: _____)

2. Muscle cramping

If you answered **YES** to **question 2** in section E, please complete the following questions (2a. to 2m.) related to your cramping.

(2a) For how many years have you suffered from cramping?	(years)
(2b) Did you suffer from cramping during or after exercise in the **last 12 months**?	Yes ☐ No ☐
(2c) With what **type of exercise** is your cramping associated (You can tick more than one form of exercise)?	☐ Swimming ☐ Cycling ☐ Running
(2d) In the **last 10 races or training sessions**, how many times have you experienced cramping?	Races: _____/10 Training sessions: _____/10
(2e) What treatment/s have you had that **successfully relieved** an acute cramp? (can tick more than one)	☐ Stretching ☐ Resting ☐ Drinking fluid ☐ Ice application ☐ Massage ☐ Magnesium ☐ Salt (tablets or solution) ☐ Other (Specify: _____)
(2f) At **what point in the race or training run** do you usually first experience cramping?	☐ First quarter ☐ Second quarter ☐ Third quarter ☐ Fourth quarter ☐ After the race ☐ No pattern
(2g) In which **muscles** do you usually cramp (please list the muscle by the one which cramps most frequently (as 1) and the others after that (2-4)?	☐ Calves ☐ Hamstrings ☐ Quadriceps (thigh) ☐ Foot muscles ☐ Other (Specify: _____)
(2h) Have you **ever** suffered from cramping in your **whole body** (arms and legs)?	Yes ☐ No ☐
(2i) Have you **ever** been **admitted to hospital** following cramping?	Yes ☐ No ☐
(2j) Have you **ever** been **confused or in a coma** during or after a cramping episode?	Yes ☐ No ☐
(2k) Have you ever had **"dark urine"** in the 3 days following a cramping episode?	Yes ☐ No ☐

(2l) If you cramp, **how long** does the cramp usually last for (min)?	(minutes)
(2m) If you cramp, how **severe** is the cramp usually? (please tick).	☐ Mild: < 5 minutes and you are able to continue exercising ☐ Moderate: 5-15 minutes and you are able to continue exercising ☐ Severe: >15 minutes or if you have to STOP exercising

3. Past Tendon and Ligament Injury History

If you answered **YES** to **question 3** in section E, please complete the following questions (3a. to 3d.) related to your past history of tendon/ligament injury/ies.

(3a) Please tick which **tendon/s** you have injured? (next column on the tright) Also indicate (tick) if your injured tendon was longsatnding pain (tendinopathy) or an acute tear/rupture	**Tendon**		**Longstanding Pain (Tendionapthy)**	**Acute Tear/ Rupture**
	Foot and ankle:	☐ Achilles tendon	☐	☐
		☐ Tibialis posterior	☐	☐
		☐ Plantar fascia	☐	☐
	Knee:	☐ Patellar tendon	☐	☐
	Elbow and wrist:	☐ Wrist extensor tendon	☐	☐
	Shoulder:	☐ Rotator cuff	☐	☐
	Other: _____		☐	☐

(3b) Please tick which **ligament/s** you have injured? (next column on the tright) Also indicate if your sprained or completely tore the ligamant.	**Ligament**	**Sprain**	**Complete Tear**
	☐ Shoulder ligaments	☐	☐
	☐ Elbow ligaments	☐	☐
	☐ Wrist ligaments	☐	☐
	☐ Finger ligaments	☐	☐
	☐ Knee (ACL)	☐	☐
	☐ Knee (MCL)	☐	☐
	☐ Knee (PCL)	☐	☐
	☐ Knee (LCL)	☐	☐
	☐ Ankle lateral ligaments	☐	☐
	☐ Ankle medial ligaments	☐	☐
	☐ Spinal ligaments	☐	☐
	☐ Other: _____	☐	☐

(3c) Please tick if you have you ever suffered from any of the following **joint capsule** injuries?	☐ Acute shoulder dislocation ☐ Chronic shoulder instability ☐ Other: _____
(3d) Do you suffer from any other **connective tissue or rheumatological diseases** or disorders? (If yes, please specify which one)	Yes ☐ No ☐ (refer to the list on the next page) (If yes, specify: _____)

List of some Connective Tissue and/or Rheumatic Diseases and Disorders		
Ankylosing Spondylitis	Lipid Storage Diseases	Pseudogout
Aspartylglycosaminuria (AGU)	Marfan Syndrome	Reactive Arthritis
Behcet's Syndrome	Menkes Kinky Hair Syndrome	Reiter's Syndrome
Crohn's Disease	Mucopolysaccharidoses	Relapsing Polychrondritis
Discoid Lupus Erythematosus	Myopathies and Dystrophies	Scleroderma
Ehlers-Danlos syndrome (EDS)	Ochronosis (Homocystinuria)	Sjogren's Syndrome
Eosinophilic Fascitis	Osteogenesis imperfecta (OI)	Systemic Lupus Erythematosus (SLE)
Giant Cell (Temporal) Arthritis	Polyarteritis Nodosa	Systemic Sclerosis
Gout	Polymyalgia Rheumatica	Wegener's Granulomatosis
Hypersentive Vasulatis	Polymyositis & Dermatomyositis	

4. Use of medicines to treat an injury before or during participation

If you answered **YES** to **question 4** in section E, please complete the following two questions related to medicine use for injuries before or during races.

(4a) Which of the following medicines have you used in the past to treat an injury **in the week just before** a race?	☐ Paracetamol (e.g. Panado, Tylenol) ☐ Non-steroidal anti-inflammatories (e.g. Voltaren, Cataflam) ☐ Cortisone (pills) ☐ Cortisone injection ☐ Codeine ☐ Anti-inflammatory gels/creams/patches ☐ Any other pain killers (Specify: _____)
(4b) Which of the following medicines have you used in the past to treat an injury **during a race**?	☐ Paracetamol (e.g. Panado, Tylenol) ☐ Non-steroidal anti-inflammatories (e.g. Voltaren, Cataflam) ☐ Cortisone (pills) ☐ Cortisone injection ☐ Codeine ☐ Anti-inflammatory gels/creams/patches ☐ Any other pain killers (Specify: _____)

5. Gastrointestinal symptoms during exercise

If you answered **YES** to **question 5** in section E, please indicate which gastrointestinal symptoms you have ever suffered from **during exercise** and, how frequently (in the last 12 months and in the last 10 races), and in which type of exercise.

Symptom	Number of times in the last 12 months **(during exercise)**	Number of times in last 10 races **(during races)**	Tick type of exercise
Nausea			☐ Swimming, ☐ Cycling, ☐ Running
Vomiting			☐ Swimming, ☐ Cycling, ☐ Running
Heartburn			☐ Swimming, ☐ Cycling, ☐ Running
Abdominal pain			☐ Swimming, ☐ Cycling, ☐ Running
Urge to pass a stool (defecate)			☐ Swimming, ☐ Cycling, ☐ Running
Diarrhoea			☐ Swimming, ☐ Cycling, ☐ Running
Passing blood in the stool			☐ Swimming, ☐ Cycling, ☐ Running

6. Diseases of the nervous system

If you answered **YES** to **question 6** in section E, please indicate which nervous disease symptoms you have ever suffered from **during exercise** and, how frequently (in the last 12 months and in the last 10 races), and in which type of exercise.

Symptom	Number of times in the last 12 months **(during exercise)**	Number of times in last 10 races **(during races)**	Tick type of exercise
Headaches			☐ Swimming, ☐ Cycling, ☐ Running
Nerve tingling in the hands			☐ Swimming, ☐ Cycling, ☐ Running
Loss of sensation in the hands			☐ Swimming, ☐ Cycling, ☐ Running

7. Genital tract injury during cycling

If you answered **YES** to **question 7** in section E, please indicate which symptoms of genital tract injury have you suffered from **during or after cycling**, how frequently (in the last 10 sessions), how long symptoms last, and what factors prevent or relieve symptoms?

Symptom	Number of times in the last 10 cycling sessions	Please indicate when the symptoms occur	Please indicate if any of the following reduce or prevent the symptoms (can tick more than one)
Genital numbness		☐ Only during cycling ☐ During and up to 1 hour after cycling ☐ During and 1-24 hours after cycling ☐ During and > 24 hours after cycling	☐ Changing the saddle type ☐ Changing the saddle position ☐ Using padded cycling shorts ☐ Wearing no underwear ☐ Wearing additional underwear ☐ Other (Specify: _____)
Genital pain		☐ Only during cycling ☐ During and up to 1 hour after cycling ☐ During and 1-24 hours after cycling ☐ During and > 24 hours after cycling	☐ Changing the saddle type ☐ Changing the saddle position ☐ Using padded cycling shorts ☐ Wearing no underwear ☐ Wearing additional underwear ☐ Other (Specify: _____)
Genital bruising		☐ Only during cycling ☐ During and up to 1 hour after cycling ☐ During and 1-24 hours after cycling ☐ During and > 24 hours after cycling	☐ Changing the saddle type ☐ Changing the saddle position ☐ Using padded cycling shorts ☐ Wearing no underwear ☐ Wearing additional underwear ☐ Other (Specify: _____)
Altered sexual function following a cycling session		☐ Up to 1 hour after cycling ☐ 1-24 hours after cycling ☐ > 24 hours after cycling	☐ Changing the saddle type ☐ Changing the saddle position ☐ Using padded cycling shorts ☐ Wearing no underwear ☐ Wearing additional underwear ☐ Other (Specify: _____)

8. Allergy history

If you answered **YES** to **question 8** in section E, please complete the following questions (8a. to 8e.) related to your current and past history of allergies.

(8a) Please indicate how long (years) have you been suffering from allergies?					years
(8b) Please tick which <u>type of allergy</u> do you currently suffer from					
Nose (hay fever)	Yes ☐ No ☐	Sinusitis	Yes ☐ No ☐	Asthma (allergic)	Yes ☐ No ☐
Skin allergies	Yes ☐ No ☐	Eye allergies	Yes ☐ No ☐	Allergy to plant material	Yes ☐ No ☐
Allergy to foods	Yes ☐ No ☐	Allergy to animals	Yes ☐ No ☐	Other	
(8c) Please tick which <u>type of allergy</u> do you <u>currently take medication</u> for					
Nose (hay fever)	Yes ☐ No ☐	Sinusitis	Yes ☐ No ☐	Asthma (allergic)	Yes ☐ No ☐
Skin allergies	Yes ☐ No ☐	Eye allergies	Yes ☐ No ☐	Allergy to plant material	Yes ☐ No ☐
Allergy to foods	Yes ☐ No ☐	Allergy to animals	Yes ☐ No ☐	Other	
(8d) Please tick which <u>type of medication</u> do you <u>currently take</u>					
Cortisone nose spray	Yes ☐ No ☐	Cortisone nose inhaler	Yes ☐ No ☐	Anti-histamine tablets	Yes ☐ No ☐
Cortisone cream	Yes ☐ No ☐	Anti-histamine cream	Yes ☐ No ☐	Other inhaler / tablets or cream	Yes ☐ No ☐
(8e) Please tick which <u>symptoms of allergy</u> do you <u>currently suffer</u> from					
Sneezing	Yes ☐ No ☐	Itchy runny nose	Yes ☐ No ☐	Headache	Yes ☐ No ☐
Itchy palate	Yes ☐ No ☐	Streaming eyes	Yes ☐ No ☐	Fatigue	Yes ☐ No ☐
Itchy eyes	Yes ☐ No ☐	Blocked nose	Yes ☐ No ☐	Poor sleep	Yes ☐ No ☐
Post nasal drip	Yes ☐ No ☐	Coughing	Yes ☐ No ☐	Wheezing	Yes ☐ No ☐
In which months of the year do you <u>currently</u> have symptoms of allergies? (You tick more than one)	☐ Jan ☐ Feb ☐ March ☐ April ☐ May ☐ June ☐ July ☐ Aug ☐ Sept ☐ Oct ☐ Nov ☐ Dec				
(8f) Please tick which <u>type of allergy</u> did you suffer from in the past (NOT currently)					
Nose (hay fever)	Yes ☐ No ☐	Sinusitis	Yes ☐ No ☐	Asthma (allergic)	Yes ☐ No ☐
Skin allergies	Yes ☐ No ☐	Eye allergies	Yes ☐ No ☐	Allergy to plant material	Yes ☐ No ☐
Allergy to foods	Yes ☐ No ☐	Allergy to animals	Yes ☐ No ☐	Other	

9. Asthma history

If you answered **YES** to **question 9** in section E, please complete the following questions (9a. to 9k.) related to your current history of asthma

(9a) Do you currently suffer from asthma?	Yes ☐ No ☐
(9b) How many years have you suffered from asthma?	(years)
(9c) How was your asthma diagnosed?	☐ A doctor taking a history and performing an examination ☐ Lung function test (blow test) but no exercise ☐ Lung function test (blow test) before and after exercise ☐ Metacholine challenge test ☐ Eucapnic hyperventilation test (rebreathing test) ☐ Other test (Specify: _____)
(9d) Which **type of asthma** do you currently suffer from?	☐ Asthma that occurs at any time but not during exercise ☐ Asthma that occurs at any time including during exercise ☐ Asthma that only occurs during exercise
(9e) Please indicate **how frequently do you currently experience the symptoms** of asthma (shortness of breath, wheezing, coughing or coughing after exercise)?	**Daytime symptoms (per week)** ☐ < 2 / week ☐ 2-4 / week ☐ >4 / week ☐ All the time **Night time symptoms (per month)** ☐ < 1 / month ☐ 2-3 / month ☐ ≥4 / month ☐ All the time **Exercise related symptoms (per 10 exercise sessions)** ☐ <1 per 10 sessions ☐ 2-3 per 10 sessions ☐ ≥4 per 10 sessions
(9f) Please indicate if you had symptoms of asthma that were severe enough to necessitate **hospital admission in the last 12 months**	☐ No hospital admission for asthma in the last 12 months ☐ 1-2 hospital admissions for asthma in the last 12 months ☐ 3-4 hospital admissions for asthma in the last 12 months ☐ >4 hospital admissions for asthma in the last 12 months
(9g) Which **symptoms of asthma** do you currently suffer from?	☐ Wheezing ☐ Dry cough ☐ Shortness of breath ☐ Tight chest ☐ Chest pain ☐ Other (Specify: _____)

(9h) What **medication do you currently use** for your asthma? (you may tick more than one option)	☐ Cortisone inhaler (e.g. Beclate, Becloforte, Becodisks, Becotide, Budeflam, Flixotide, Inflammide, Pulmicort, Qvar, etc) ☐ Salbutamol (bronchodilator) inhaler (e.g. Ventolin, Venteze, Vomax, Airomir, Asthavent etc.) ☐ Salmeterol (bronchodilator) inhaler (Serevent) ☐ Fenoterol (bronchodilator) inhaler (Berotec) ☐ Terbutaline (bronchodilator) inhaler (Bricanyl) ☐ Formoterol (bronchodilator) inhaler (e.g. Foradil, Foratec, Oxis) ☐ Ipratropium (bronchodilator) inhaler (Atrovent) ☐ Tiotropium (bronchodilator) inhaler (Spiriva) ☐ Combined cortisone and bronchodilator inhaler (e.g. Atrovent, Berodual, Combivent, Duolin, Duovent, Seretide, Symbicord) ☐ Cortisone tablets ☐ Bronchodilator tablets ☐ Leukotriene receptor antagonist tablets (e.g. Acccolate, Singulair) ☐ Other inhaler ☐ Other medication (Specify: _____)
(9i) **When do you use your medication** for your asthma?	☐ Daily (irrespective of exercise) ☐ Only before exercise ☐ Other (Specify: _____)
(9j) **How long before an exercise session** do you use your medication for asthma?	min
(9k) Have you obtained **TUE (therapeutic use exemption forms)** for your asthma medication?	Yes ☐ No ☐

10. History of previous collapse

If you answered **YES** to **question 10** in section E, please complete the following questions (10a. to 10d.) related to your current history of asthma

(10a) Have you collapsed during training or racing?	☐ Training ☐ Racing ☐ Training and racing
(10b) How many times have you collapsed in training session or races during the last **five years**?	_____ training session _____ races
(10c) When you collapse, does it mostly occur before of after the finish line / completion of the training session?	☐ Before the finish ☐ After the finish
(10d) What is the cause of you collapse?	☐ Dehydration ☐ Heat illness ☐ Hyponatremia ☐ Low blood pressure ☐ Low blood sugar ☐ Other condition (Specify: _____)

11. History of any current injury that you suffer from

If you answered **YES** to **question 11** in section E, please complete the following questions (11a. to 11g.) related to each of your current injury/ies (Space is provided for two injuries)

Injury 1	
(11a) What was the approximate date when you first became aware of the injury?	Month Year
(11b) Please indicate which side of your body is injured (if applicable)	☐ Right ☐ Left
(11c) Please indicate which anatomical area is currently injured	☐ Head ☐ Neck ☐ Face ☐ Front chest ☐ Back chest ☐ Shoulder ☐ Upper arm ☐ Elbow ☐ Forearm ☐ Wrist ☐ Finger ☐ Lower back ☐ Hip ☐ Thigh ☐ Hamstring ☐ Quadriceps ☐ Knee ☐ Shin ☐ Achilles ☐ Ankle ☐ Foot Other (Specify: _____)
(11d) Please indicate the type of structure that was injured	☐ Muscle ☐ Tendon ☐ Bone ☐ Ligament ☐ Joint Other (Specify: _____)
(11e) Please indicate in which sport (discipline) the injury occurred	☐ Running ☐ Cycling ☐ Swimming Other (Specify: _____)
(11f) Please indicate the severity of the injury (tick one box please)	☐ I only experience symptoms after exercise - Grade 1 ☐ I experience symptoms during exercise, but it does not interfere with exercise - Grade 2 ☐ I experience symptoms during exercise that may interfere with my training/ competition - Grade 3 ☐ I am so painful that I may not be able to train or compete - Grade 4
(11g) Please indicate how your injury was treated to date (you can tick more than one)?	☐ Rest ☐ Stretches ☐ Physiotherapy ☐ Surgery ☐ Strengthening exercises ☐ Equipment change ☐ Tablets ☐ Cortisone injection ☐ Other injection ☐ Orthotics Other (Specify: _____)

Injury 2	
(11a) What was the approximate date when you first became aware of the injury?	Month Year
(11b) Please indicate which side of your body is injured (if applicable)	☐ Right ☐ Left
(11c) Please indicate which anatomical area is currently injured	☐ Head ☐ Neck ☐ Face ☐ Front chest ☐ Back chest ☐ Shoulder ☐ Upper arm ☐ Elbow ☐ Forearm ☐ Wrist ☐ Finger ☐ Lower back ☐ Hip ☐ Thigh ☐ Hamstring ☐ Quadriceps ☐ Knee ☐ Shin ☐ Achilles ☐ Ankle ☐ Foot Other (Specify: _____)
(11d) Please indicate the type of structure that was injured	☐ Muscle ☐ Tendon ☐ Bone ☐ Ligament ☐ Joint Other (Specify: _____)
(11e) Please indicate in which sport (discipline) the injury occurred	☐ Running ☐ Cycling ☐ Swimming Other (Specify: _____)
(11f) Please indicate the severity of the injury (tick one box please)	☐ I only experience symptoms after exercise - Grade 1 ☐ I experience symptoms during exercise, but it does not interfere with exercise - Grade 2 ☐ I experience symptoms during exercise that may interfere with my training/ competition - Grade 3 ☐ I am so painful that I may not be able to train or compete - Grade 4
(11g) Please indicate how your injury was treated to date (you can tick more than one)?	☐ Rest ☐ Stretches ☐ Physiotherapy ☐ Surgery ☐ Strengthening exercises ☐ Equipment change ☐ Tablets ☐ Cortisone injection ☐ Other injection ☐ Orthotics Other (Specify: _____)

Appendix 5

UNIVERSITY OF CAPE TOWN

Health Sciences Faculty
Research Ethics Committee
Room E53-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6338 • Facsimile [021] 406 6411
e-mail: preaward@curie uct ac za

13 January 2006

REC REF: 425/2005

Assoc Prof MP Schwellnus
Department of Human Biology
UCT/MRC Research Unit for Exercise Science and Sports Medicine
Medical School

Dear Prof Schwellnus

THE PORT ELIZABETH IRONMAN TRIATHLON 2006: MEDICAL CONSEQUENCES FOLLOWING ENDURANCE SPORTS.

Thank you for your letter to the Research Ethics Committee dated 14 December 2005, addressing the issues raised by the committee. It is a pleasure to inform you that the Ethics Committee has formally approved the above mentioned study.

Please quote the REC. REF in all your correspondence.

Yours sincerely

PROF. T ZABOW
CHAIRPERSON

Appendix 6

Athletes Name: _____
Race Number: _____

PRE- AND POST-RACE ULTRASOUND REPORT

Right Achilles tendon:

Current Symptoms:

_____

_____

Clinical Examination:

Site of Tenderness: _____

Swelling
- Nodular ☐
- Diffuse ☐

Shift test
- Positive ☐
- Negative ☐

Tendon:
- Shape:
  - Angular: ☐
  - Fusiform: ☐
- Margin:
  - Sharply defined ☐
  - Poorly defined ☐
- Contour:
  - Smooth ☐
  - Nodular ☐
  - Tapered ☐
- Max. diameter:
  - AP: ☐
  - TRV: ☐
- Internal architecture
  - Organised ☐
  - Central hypoechoic foci (med/lat/ant/post) ☐
  - Disrupted fibres (mild/mod/severe ☐
  - Haematoma ☐
  - Calcification ☐
  - Acoustic shadowing ☐
- Power Doppler vascularity
  - Absent ☐
  - Present ☐
  - Prominent ☐

Paratenon
- Fluid:
  - Absent ☐
  - Present
    - Site ☐
    - Amount ☐
- Soft tissue swelling
  - Absent ☐
  - Present ☐
- Power Doppler Vascularity
  - Absent ☐
  - Present ☐
  - Increased ☐

Left Achilles Tendon:

Current Symptoms:

_____

_____

Clinical Examination:

Site of Tenderness: _____

Swelling
- Nodular ☐
- Diffuse ☐

Shift test
- Positive ☐
- Negative ☐

Tendon:
- Shape:
  - Angular: ☐
  - Fusiform: ☐
- Margin:
  - Sharply defined ☐
  - Poorly defined ☐
- Contour:
  - Smooth ☐
  - Nodular ☐
  - Tapered ☐
- Max. diameter:
  - AP: ☐
  - TRV: ☐
- Internal architecture
  - Organised ☐
  - Central hypoechoic foci (med/lat/ant/post) ☐
  - Disrupted fibres (mild/mod/severe) ☐
  - Haematoma ☐
  - Calcification ☐
  - Acoustic shadowing ☐
- Power Doppler vascularity
  - Absent ☐
  - Present ☐
  - Prominent ☐

Paratenon
- Fluid:
  - Absent ☐
  - Present
    - Site ☐
    - Amount ☐
- Soft tissue swelling
  - Absent ☐
  - Present ☐
- Power Doppler Vascularity
  - Absent ☐
  - Present ☐
  - Increased ☐

- Retrocalcaneal Bursa
  - Normal
  - Fluid filled
- Kager's fat
  - Normal
  - Hyperechogenic

Myotendinous junction

- Normal
- Tear

Calcaneus

- Normal
- Abnormal

- Retrocalcaneal Bursa
  - Normal
  - Fluid filled
- Kager's fat
  - Normal
  - Hyperechogenic

Myotendinous junction

- Normal
- Tear

Calcaneus

- Normal
- Abnormal

Appendix 7

Athletes Name: _____ **Race Number**: _____

6 – 8 WEEK FOLLOW UP ULTRASOUND REPORT

Right Achilles tendon:

SX:
Pain
Swelling
Stiffness

SIGNS:
Tenderness - Site
Swelling – nodular/diffuse
Shift test – positive/ negative

TREATMENT
ICE
STRETCHING
PHYSIO: ULTRASOUND
MANIPULATION
ECCENTRIC TRAINING:

TRAINING HISTORY

_____
_____
_____

RACES DONE

_____
_____
_____

ULTRASOUND REPORT

Tendon:

- Shape:
  - Angular: ☐
  - Fusiform: ☐
- Margin:
  - Sharply defined ☐
  - Poorly defined ☐
- Contour:
  - Smooth ☐
  - Nodular ☐
  - Tapered ☐
- Max. diameter:
  - AP: ☐
  - TRV: ☐
- Internal architecture
  - Organised ☐
  - Central hypoechoic foci (m ☐)
  - Disrupted fibres (mild/mod ☐
  - Haematoma ☐
  - Calcification ☐
  - Acoustic shadowing ☐

Left Achilles Tendon:

SX:
Pain
Swelling
Stiffness

SIGNS:
Tenderness - Site
Swelling – nodular/diffuse
Shift test – positive/ negative

TREATMENT
ICE
STRETCHING
PHYSIO: ULTRASOUND
MANIPULATION
ECCENTRIC TRAINDING:

TRAINING HISTORY

_____
_____
_____

RACES DONE

_____
_____
_____

ULTRASOUND REPORT

Tendon:

- Shape:
  - Angular: ☐
  - Fusiform: ☐
- Margin:
  - Sharply defined ☐
  - Poorly defined ☐
- Contour:
  - Smooth ☐
  - Nodular ☐
  - Tapered ☐
- Max. diameter:
  - AP: ☐
  - TRV: ☐
- Internal architecture
  - Organised ☐
  - Central hypoechoic foci (med/lat/ant/post)
  - Disrupted fibres (mild/mod/severe)
  - Haematoma ☐
  - Calcification ☐
  - Acoustic shadowing ☐

Paratenon

- Fluid:
  - Absent ☐
  - Present
    - Site
    - Amount ☐
- Soft tissue swelling
- Power Doppler Vascularity
  - Absent ☐
  - Present ☐
  - Increased ☐
- Retrocalcaneal Bursa
  - Normal ☐
  - Fluid filled ☐
- Kager's fat
  - Normal ☐
  - Hyperechogenic ☐

Myotendinous junction

- Normal ☐
- Tear ☐

Calcaneus

- Normal ☐
- Abnormal ☐