

THE EFFECTS OF COMPRESSION GARMENTS ON THE  
RECOVERY OF LONG DISTANCE RUNNERS AFTER  
PROLONGED EXERCISE

BY

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## **DECLARATION**

I, the undersigned, hereby declare that the work contained in this thesis is my own original work and that I have not previously in its entirety or in part submitted it at any university for a degree.

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## SUMMARY

Various types of post-exercise recovery strategies have become part of the modern athlete's daily routine. It is well known that inadequate recovery will prolong the time it takes for the runner's body to adequately adapt between training sessions and competitions. Anecdotal claims have been made about compression garments as a beneficial method to assist recovery after training sessions and competitions. Until now limited scientific research has addressed the influence that compression garments have on the recovery process after sporting activities. The benefits of compression garments, as a possible recovery modality, are that it is cost-effective, practical and easily obtainable.

This study endeavored to investigate the possible influence that compression garments may have on middle-aged long distance runners' recovery rate after a prolonged run. This is the first study that has focused on compression garments as a post-exercise recovery modality for experienced middle-aged long distance runners. The other unique aspect of this study is the prolonged two-hour treadmill protocol that was used to induce muscle soreness.

In addressing the aims, a randomized, crossover study design was used to investigate the possible benefits that the high pressure (CCL II 23-32 mmHg (mercury millimeter)) graduated compression garments may bring about. Seven competitive male long distance runners (height:  $176.0 \pm 8.6$  cm; body mass:  $92.5 \pm 11.8$  kg;  $VO_{2max}$ :  $45.7 \pm 5.0$  mL.kg<sup>-1</sup>.min<sup>-1</sup>) between the ages of 36 to 51 years volunteered for the study. The runners had to complete a two-hour treadmill run at 70 % of their predetermined maximum aerobic capacity, followed by a monitored 72-hour recovery period. The first part of the prolonged run was a 90-minute variant gradient run, followed by a 30-minute downhill run. Each subject acted as his own control and visited the Stellenbosch University's Sport Physiology Laboratory (South Africa) on two occasions, separated by 7 to 28 days. One test was done with a compression garment (23 to 32 mmHg) and the other without.

Testing included the measurement of lower limb circumferences (ankle, calf, mid- and proximal thigh), plasma lactate, lactate dehydrogenase and creatine kinase concentrations and the completion of subjective questionnaires on perceived muscle soreness (visual analog scale (VAS)). The lower extremities' functional ability was determined with a time to exhaustion (TTE) step test, a vertical jump test (VJ) and modified sit-and-reach flexibility test. Pre-exercise measurements were taken and immediately after and during the 72 hour after the

treadmill run and repeated for the second bout.

The main outcomes of this study showed that the two-hour treadmill run induced delayed onset of muscle soreness, with and without the compression garment. Evidence of this was a significant rise in plasma creatine kinase (CK<sub>p</sub>) over the duration of both trials ( $P < 0.05$ ). The compression garment significantly reduced swelling in the calf muscle ( $41.0 \pm 0.2$  vs.  $41.5 \pm 0.5$  mm;  $P < 0.002$ ). Runners showed a lower perceived muscular pain and discomfort while performing functional knee movements at 24 and 48-hours after the two-hour run with the compression garment ( $1.2 \pm 1.6$  vs.  $3.8 \pm 2.4$  cm and  $0.9 \pm 1.8$  vs.  $3.0 \pm 2.6$  cm on VAS, respectively;  $P < 0.05$ ). Significant differences in perceived muscle soreness between the WCG and WOCG trials were observed at 24-hours after the run during rest ( $0.1 \pm 0.2$  vs.  $0.4 \pm 0.8$  cm;  $P = 0.02$ ) and with stretching ( $1.9 \pm 1.2$  vs.  $3.5 \pm 2.5$  cm on VAS  $P = 0.02$ ). The perceived pain associated with pressure was significantly lower with the compression garment at 24 (307 %) and 48-hours (237 %) after the run ( $P < 0.05$ ).

Blood lactate levels were reduced during the acute phase of recovery at 10 ( $1.8 \pm 0.5$  vs.  $2.2 \pm 0.9$  mmol.L<sup>-1</sup>;  $P = 0.05$ ) and 30 minutes ( $1.8 \pm 0.5$  vs.  $2.4 \pm 0.4$  mmol.L<sup>-1</sup>;  $P = 0.01$ ) after the run, as well as plasma creatine kinase concentrations were statistically significantly lower at 24-hours ( $238.3 \pm 81.3$  vs.  $413.3 \pm 250.8$  units.L<sup>-1</sup>;  $P = 0.005$ ) after exercise with the compression garment. The two-hour treadmill run and the compression garment had no significant influence on the runners' lower limb strength, power, endurance or flexibility ( $P > 0.05$ ).

Compression garments demonstrated the potential to enhance recovery after prolonged strenuous exercise in well trained middle-aged runners. In addition, runners did not experience additional fatigue from the moderate to high pressure garments. The effect of higher pressure compression garments on athletic performance and the psychological influence of the garment need further investigation.

## OPSOMMING

Verskillende tipes na oefening herstelstrategieë, vorm deel van die moderne atleet se daaglikse routine. Dit is wel bekend dat onvoldoende herstel sal beteken dat die atleet se liggaam langer sal neen om aan te pas tussen inoefen sessies en kompetisies. Sekere bewerings word al gemaak omtrent die voordeligheid van kompressiesokkies tydens die herstelperiode na oefening sessies en kompetisies. Tot nou toe was daar beperkte wetenskaplike navorsing oor die invloed van kompressie sokkies of die herstel proses van sport aktiwiteite. Die voordeel van kompressie sokkies as 'n moontlike herstelmetode, is dat dit koste-effektief, prakties en maklik verkrybaar is.

Hierdie studie poog om 'n ondersoek in te stel na die moontlike invloed wat kompressie sokkies op middeljarige lang-aftstandatlete se herstelperiode sal hê na 'n verlengde hardloopsessie. Hierdie is die eerste studie wat konsentreer op kompressie sokkies as 'n na oefenings hersteltegniek vir ervare middeljarige lang-aftstandatlete. Die ander unieke aspek van die ondersoek is die langdurige tweeuur trapmeul protokol wat gebruik word om spierpyn te veroorsaak.

Om die doel te bereik, is 'n lukrake oorkruis studie gebruik om ondersoek in te stel na die moontlike voordele van die hoë druk (CCL II 23-32 mmHg) kompressie sokkies. Hierdie sokkies toon 'n progressiewe verhooging van druk vanaf die enkele tot onder die knieskyf. Sewe mededingende langafstand atlete (lengte :  $176.0 \pm 8.6$  cm; liggaams massa:  $92.5 \pm 11.8$  kg;  $VO_{2maks}$ :  $45.7 \pm 5.0$  mL.kg<sup>-1</sup>.min<sup>-1</sup>) tussen die ouderdomme van 36 en 51 jaar, het aan die studie deel geneem. Die wedlopers moes 'n twee-uur lange trapmeul toets voltooi, teen 70% van hul vooraf bepaalde maksimum aerobiese kapasiteit. Dit is gevolg deur 'n gemonitorde 72-uur herstel periode. Die eerste deel van die twee-uur hardloop sessie was 'n 90-minuut afwisselende opdraende en afdraende hardloop stel, wat gevolg is deur a 30-minuut afdraende deel. Elke deelnemer was sy eie kontrole en het op twee geleenthede die Stellenbosch Universiteit se Sport Fisiologiese Laboratorium (Suid Afrika) besoek. Die twee besoeke is tussen 7 en 28 dae geskei. Een toets is met kompressie sokkies gedoen (23 – 32 mmHg) en die ander sonder.

Die toetse het die volgende behels: laer been omtrekke (enkel, kuit, middel- and bo dy), die versameling en ontleding van bloed monsters vir plasma laktaat, laktaat dehydrogenase and kreatine kinase konsentrasies en die voltooiing van subjektiewe vraelyste oor die graad van

spierpyn ervaring (“*visual analog scale*” (VAS)). Die onderlyf funksionele vermoëns is bepaal met ’n tyd tot uitputtings traptoets, ’n vertikale sprong toets en ’n gewysige sit-en-strek soepelheids toets. Data is voor die oefeninge in gevorder asook direk daarna, en gedurende die 72 uur na die trapmeul draf. Die metings vir die tweede sessie is herhaal.

Die hoof uitkomst van die studie het gewys dat die twee-uur trapmeulsessie het spierpyn veroorsaak, met en sonder die kompressie sokkies. Die bewys hiervan was ’n betekensvolle toename in plasma kreatien kinase (CK<sub>p</sub>) oor die tydperk van albei oefening toetse ( $P < 0.05$ ). Die kompressie sokkies het die swelling in die kuitspiere verminder, in vergelyking met die toetse sonder kompressie sokkies ( $41.0 \pm 0.2$  vs.  $41.5 \pm 0.5$  mm;  $P < 0.002$ ).

Wedlopers met die kompressie sokkies het minder spierseerheid en ongerief aangeteken toe hulle knie bewegings gedoen het op 24 en 48-uur na die twee-ure trapmeul toets ( $1.2 \pm 1.6$  vs.  $3.8 \pm 2.4$  cm op VAS en  $0.9 \pm 1.8$  vs.  $3.0 \pm 2.6$  cm op VAS, onderskeidelik;  $P < 0.05$ ). Betekenisvolle verskille is waargeneem tussen die toetse met en sonder kompressie sokkies, op 24-uur na die twee-ure toets gedurende rus ( $0.1 \pm 0.2$  vs.  $0.4 \pm 0.8$  cm op VAS;  $P = 0.02$ ) en met strek oefeninge ( $1.9 \pm 1.2$  vs.  $3.5 \pm 2.5$  cm op VAS  $P = 0.02$ ). Die pyn wat ervaar was met drukking, was betekenisvol minder met die kompressie sokkies op 24 (307 %) en 48-uur (237 %) na die trapmeul sessie ( $P < 0.05$ ). Bloed laktat konsentrasie in die sirkulasie was verlaag gedurende die akute fase van die herstelings periode op 10 ( $1.8 \pm 0.5$  vs.  $2.2 \pm 0.9$  mmol.L<sup>-1</sup>;  $P = 0.05$ ) en 30 minute ( $1.8 \pm 0.5$  vs.  $2.4 \pm 0.4$  mmol.L<sup>-1</sup>;  $P = 0.01$ ) na die hardloop sessie, sowel as die plasma kreatine kinase konsentrasie was statisties betekenisvol laer by 24 uur ( $238.3 \pm 81.3$  vs  $413.3 \pm 250.8$  eenhede L<sup>-1</sup>;  $P = 0.005$ ) na die hardloop sessie met die kompressie sokkies. Die twee-ure trapmeul toets en die kompressie sokkies het geen betekenisvolle invloed gehad op die wedlopers se onderlyf ledemate se plofkrag, uithouvermoë of soepelheid ( $P > 0.05$ ) nie.

Kompressie sokkies het gewys dat dit potensiaal het om met herstel te help na lang en harde oefening in geoefende middeljarige atlete. Nietemin is daar verdere wetenskaplike navorsing nodig om dit te bevestig. Wedlopers het nie addisionele vermoëns van die drukking van kompressie sokkies ervaar nie. Sterker drukkende kompressie sokkies sowel as die sielkundige invloed van die sokkies benodig verdere navorsing.

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“No man is an island, entire of itself...”

-John Donne (1572-1631)-

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## LIST OF ABBREVIATIONS

ACT	:	active recovery
ADP	:	adenosine diphosphate
ANOVA	:	analysis of variance
ANS	:	autonomic nervous system
AS	:	anterior pressure
AT	:	anaerobic threshold
ATP	:	adenosine triphosphate
ave or $\bar{x}$	:	average
BF	:	<i>Biceps Femoris</i>
BIA	:	bioelectrical impedance analysis
BM	:	body mass
BMI	:	body mass index
BMT	:	best marathon time
bpm	:	beats per minute
$c$	:	filtration coefficient
C	:	control
$Ca^{2+}$	:	calcium
Cells.mm <sup>-2</sup>	:	cells per square millimeters
CG	:	compression garment
CK	:	creatine kinase
CK <sub>p</sub>	:	plasma creatine kinase (units.L <sup>-1</sup> )
cm	:	centimeter
CMPF	:	calf muscle pump function
C-NMR	:	C-nuclear magnetic resonance
CNS	:	central nervous system
CO <sub>2</sub>	:	carbon dioxide
CP	:	creatine phosphate
Cr	:	creatine
CV	:	coefficient of variation
CWT	:	contrast water therapy
°C	:	degrees Celsius
DHR	:	downhill running
DOMS	:	delayed onset of muscle soreness



DT	:	maximal throwing distance
EIH	:	exercise induced hypoxemia
EMG	:	electromyography
EPOC	:	excess post-exercise oxygen consumption
ESWT	:	extracorporeal shock wave therapy
ET	:	elastic tights
F	:	filtration force
$f_{bmax}$	:	maximum breathing frequency (breaths.min <sup>-1</sup> )
g	:	gram
g.kg <sup>-1</sup>	:	gram per kilogram
GA	:	<i>Gastrocnemius</i>
GFIT	:	graduated compression technology
GI	:	glycemic index
GLUT 4	:	glucose transporter protein (4)
H <sup>+</sup>	:	hydrogen ion
H <sub>baseline</sub>	:	reaching height
HbO <sub>2</sub>	:	oxyhaemoglobin
hh:mm:ss	:	hour(s):minute(s):second(s)
H <sub>max</sub>	:	maximum velocity
HR	:	heart rate (bpm)
hr(s)	:	hour(s)
HR <sub>max</sub>	:	maximum heart rate (bpm)
HRR	:	heart rate recovery
HRV	:	heart rate variability
H <sub>top</sub>	:	jumping height
Hz	:	hertz
IPC	:	intermittent pneumatic compression
ISAK	:	international standards for advancement of kinanthropometric
IU.L <sup>-1</sup> or u.L <sup>-1</sup>	:	units per liter
kcal	:	kilocalorie
kg	:	kilogram(s)
kg.m <sup>-2</sup>	:	kilogram per square meter
kg.min <sup>-1</sup>	:	kilogram per minute
kJ	:	kilojoule(s)

km	:	kilometer(s)
$K_M$	:	Michaelis-Menten constant
$\text{km.h}^{-1}$	:	kilometres per hour
$\text{L.min}^{-1}$	:	liters per minute
L/Q	:	lactate uptake per perfusion
LCA	:	leukocyte common antigen
LDH	:	lactate dehydrogenase ( $\text{units.L}^{-1}$ )
LT	:	lactate threshold
LV	:	left ventricular
LVDF	:	left ventricular diastolic function
[La]	:	lactate concentration
m	:	meter
MAS	:	massage therapy
MCT	:	monocarboxylate transporters
MES	:	microcurrent electrical stimulation
$\text{Mg}^{2+}$	:	magnesium
MHz	:	megahertz
min	:	minute(s)
$\text{min}^{-1}.\text{kg}$	:	minutes per kilogram
ml	:	milliliter
$\text{mL.kg}^{-1}.\text{min}^{-1}$	:	milliliter per kilogram per minute
$\text{mL.L}^{-1}$	:	milliliter per liter
$\text{ml.min}^{-1}$	:	milliliter per minute
mM or $\text{mmol.L}^{-1}$	:	millimole per liter
mm	:	millimetre(s)
mmHg	:	millimetres mercury
$\text{mmol.kg}^{-1}$	:	millimole per kilogram
MPF	:	mean power frequency
MRC	:	medical research council
$n$	:	number of subjects
n.a.	:	none available
n.d.	:	no data
$\text{N}_2$	:	nitrogen
NADH	:	nicotinamide adenine dinucleotide hydrogen
nm	:	nanometer

NO	:	nitric oxide
NSAID	:	non-steroidal anti-inflammatory drugs
O <sub>2</sub>	:	oxygen
OBLA	:	onset of blood lactate accumulation
<i>P</i>	:	probability value
PAO <sub>2</sub>	:	partial pressure of oxygen in arterial
PAS	:	passive recovery
<i>P<sub>c</sub></i>	:	capillary pressure
PCr	:	phosphocreatine
PDE	:	phosphodiester
pH	:	hydrogen ion concentration
<i>P<sub>i</sub></i>	:	inorganic phosphate
PME	:	phosphomonoester
PNS	:	parasympathetic nervous system
PO <sub>2</sub>	:	partial pressure of oxygen
PPO	:	peak power output (W)
PS	:	posterior pressure
Pt	:	tissue pressure
PTV	:	peak treadmill velocity (km.h <sup>-1</sup> )
Q	:	cardiac output
Q <sub>max</sub>	:	maximal cardiac output
<i>r</i>	:	correlation coefficient
RE	:	running economy
RER	:	respiratory exchange ratio
RF	:	<i>Rectus Femoris</i>
RM	:	repetition maximum
ROM	:	rang of motion
RPE	:	ratings of perceived exertion
rpm	:	revolutions per minute
s	:	second(s)
SaO <sub>2</sub>	:	oxygen saturation in arterial blood
SCI	:	spinal cord injury
SD	:	standard deviation
SE	:	systolic ejection
SNS	:	sympathetic nervous system

SPSS	:	statistical package for social sciences
SR	:	sarcoplasmic reticulum
SV	:	stroke volume
$^{\circ}\text{s}^{-1}$	:	degree per second
TA	:	<i>Tibialis Anterior</i>
TENS	:	transcutaneous electrical nerve stimulation
TPR	:	total peripheral resistance
TTE	:	time to exhaustion
TV	:	treadmill velocity
VAS	:	visual analog scale (mm)
VE	:	minute ventilation ( $\text{L}\cdot\text{min}^{-1}$ )
$\text{VE}_{\text{max}}$	:	maximum minute ventilation ( $\text{L}\cdot\text{min}^{-1}$ )
VJ	:	vertical jump
$\text{VO}_2 \text{ SC}$	:	surplus in oxygen uptake over time
$\text{VO}_2$	:	volume of oxygen consumption
$\text{VO}_{2\text{max}}$	:	maximum oxygen consumption ( $\text{L}\cdot\text{min}^{-1}$ , $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ )
$\%\text{VO}_{2\text{max}}$	:	fractional utilization of oxygen (%)
vs.	:	versus
$V_T$	:	tidal volume
W	:	watt (s)
WCG	:	with compression garment
WOCG	:	without compression garment
ww	:	wet weight
$\pi\text{c}$	:	capillary oncotic pressure
$\pi\text{t}$	:	tissue oncotic pressure
~	:	about
$^{\circ}$	:	incline (%)

### CONVERSIONS

1 mile	:	1.16	kilometers
1 kilocalorie	:	4.184	kilojoules
1 pound	:	0.454	kilograms
1 ounce	:	28.38	grams
0 $^{\circ}\text{C}$	:	32	Fahrenheit

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## CHAPTER ONE

### INTRODUCTION

Long distance running is one of the most popular sports, not only in South Africa, but also throughout the world. In 2006 and 2007 about 28 599 runners participated in long distance events (21.1, 30, 36, 42.2 and 56 kilometers) in the Western Province alone (South Africa) (Jacobs, 2007). Soft tissue injury or damage is one of the most common problems associated with prolonged running. Typically, about 17 % of the reported injuries immediately after a marathon are related to musculoskeletal damage (Sanchez *et al.*, 2006). Muscle damage may cause a 40 to 50 % reduction in a marathon runner's functional ability immediately after a race (Ball and Herrington, 1998). The most popular age groups to participate in long distance events are between the ages of 30 and 50 years. In 2007, 5949 runners completed the Comrades marathon (about 89 km) in this age group, of which 49 % were between 40 and 50 years old. In addition, the average age in the Comrades marathon is 40 years (Jones, 2007).

The runners' reduced muscular strength, power, and endurance as well as stiffness in their joints may limit subsequent athletic performance (Ball and Herrington, 1998; Byrne and Eston, 2002; Kraemer *et al.*, 2004; Miller *et al.*, 2004; Takahashi *et al.*, 2006; Rimaud *et al.*, 2007). In addition, it has been suggested that lactate, acidosis and increased inorganic phosphate accumulation after strenuous exercise may be possible causes for muscle soreness (muscle tears or damage to the muscle wall) and fatigue (Rimaud *et al.*, 2007). This may also inhibit muscular action and exaggerate the risk of injury. Recovery strategies have been investigated with regard to the ability to clear lactate and to lessen the symptoms associated with delayed onset of muscle syndrome (DOMS) (Barnett, 2006).

If the athlete does not recover efficiently after a race or training, future problems may develop (Kraemer *et al.*, 2004; Miller *et al.*, 2004; Barnett, 2006; Gill *et al.*, 2006). Thus, recovery after training and competition is an accepted practice by athletes and their management. However, the literature shows that there is a need for more scientific research on post-exercise recovery strategies, as most research has demonstrated inconclusive findings. A practical and inexpensive post-exercise recovery method will not only aid recovery in trained athletes but also will (i) prevent future injuries and (ii) indirectly improve athletic performance by

allowing the athlete to recover more efficiently and return to training sooner. Some of the most popular post-exercise recovery strategies include: active and passive recovery, water immersion, sport massage, stretching, and prophylactic therapies (ultrasound, iontophoresis, transcutaneous electrical nerve stimulation and microcurrent electrical stimulation).

Traditionally passive recovery has been recommended for athletes after exercise. However, for some athletes this is not viable, since they cannot afford to take a day or even more off to recover. Hence, other possible strategies were investigated. Most of the available recovery studies have indicated that active recovery is the preferred recovery strategy after exercise (Martin *et al.*, 1998; Hemmings, 2001; Chatard *et al.*, 2004; Reilly and Ekblom, 2005; Gill *et al.*, 2006). When active recovery is continued for long enough periods and not restricted by time constraints and resource limitations, it tends to aid lactate clearance, as well as, recovery after exercise. However, some researchers suggest that active recovery may deplete muscle glycogen stores in-between sessions (Wilcock *et al.*, 2006). In addition, McAinch *et al.* (2004) concluded that active recovery does not aid athletic performance between subsequent aerobic sessions, regardless of the associated lower blood lactate concentrations shown.

Water immersion, involving contrast therapy and separate cold, neutral, or warm water recovery sessions is another possible recovery strategy, which works in a similar fashion to active recovery. Possible benefits may be found with contrast water therapy, but further research is warranted. Standardized water temperature or the ratios between hot and cold therapies vary between studies and findings are inconclusive. This strategy is expensive and requires specialized equipment, which is not always readily available and may be impractical between repeated short intervals of exercise (Wilcock *et al.*, 2006).

Sport massage therapy may have a psychological and relaxing influence. However, results are conflicting as to whether massage aid the removal of lactic acid after exercise or reduce DOMS and related symptoms (Ernst, 1998; Hemmings, 2001; Zainuddin *et al.*, 2005). The findings for prophylactic therapies show that these modalities do not aid acute post-exercise recovery, but does have a beneficial effect in rehabilitation of sport injuries (Stracciolini *et al.*, 2007). Traditionally, it was believed that stretching aids recovery after exercise. The latest research indicates that stretching may actually increase the risk of injury, as well as, impairs an athlete's performance (Andersen, 2005; Dawson *et al.*, 2005).

In 2003, Paula Radcliffe broke her own woman's world marathon record (2:15:25) by two minutes in her knee-high graduated compression socks. There has also been an increase lately in the popularity of compression garments across a range of sports, such as cricket, track and field, cycling and middle distance running (Doan *et al.*, 2003; Gill *et al.*, 2006; Ali *et al.*, 2007; Duffield and Portus, 2007). Little scientific evidence is available to support the anecdotal claims that have been made by manufactures, namely that compression garments aid post-exercise recovery and performance.

There is however, a significant amount of research that shows the beneficial role of compression garments in conditions such as venous insufficiencies and other associated vascular problems. Athletes however, have adapted to their training and do not generally demonstrate problems related to the venous system or a weak calf muscle-pump function. On the other hand, several authors suggest that compression is a realistic approach in aiding recovery, limiting strength loss, reducing muscle damage and the perception of muscle soreness (Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>; Kraemer *et al.*, 2004; Bringard *et al.*, 2006<sup>b</sup>; Trenell *et al.*, 2006; Ali *et al.*, 2007).

To date only a small amount of research supports the notion that compression garments may provide some benefits in sports performance and aid recovery from exercise (Barnett, 2006; Gill *et al.*, 2006). The advantage of compression therapy as an aid to post-exercise recovery and performance is that it is a cost-effective and practical method (Kraemer *et al.*, 2004; Barnett, 2006). The question is whether compression garments would be an effective strategy to aid post-exercise recovery in training and competition.

It is hypothesized that compression garments create an external pressure gradient favouring the removal of oedema through skeletal muscle action, local pressure gradients and the influence of gravity (Partsch, 2003; Kraemer *et al.*, 2004). Furthermore, the compression garment exerts pressure on the lower limb thus reducing the diameter of the blood vessel. Both superficial and deep venous blood flow is accelerated, shunting the blood towards the heart, and increasing the cardiac output (Agu *et al.*, 1999; Partch, 2003). Thus, the compression garments function in a similar way to the calf muscle-pump function.

To conclude, results from post-exercise strategies are conflicting and thus far have not revealed a modality as the “gold standard” for recovery. The most successful modalities are those mimicking the calf muscle-pump function and accelerating blood flow to the affected area, such as active recovery and contrast water therapy. This suggests that compression is a realistic post-exercise recovery strategy that warrants further scientific investigation.



## CHAPTER TWO

### THE PHYSIOLOGY OF ENDURANCE TRAINING

#### A. INTRODUCTION

Legaz-Arrese *et al.* (2007) classified long distance runners as those trained in 5000m, 10 000m and marathon events, while middle distance runners are trained in 800m to 3000m distances. In exercise physiology the term endurance, according to Bergero *et al.* (2005), represent the physical and mental ability to withstand exhaustion. In addition, endurance training is characterized as activities lasting for more than 20 minutes, with a rise in heart rate up to 60 – 80 % of the athlete's maximum heart rate (Carter *et al.*, 2003). Ball and Herrington (1998) more specifically define endurance training as low resistance and high repetition exercise.

Training is a process of stress and adaptation. Therefore, one could also consider that prolonged distance running requires well-developed endurance ability of athletes, which entails immediate and numerous adaptations of the whole body to stressors (Sztajzel *et al.*, 2006). Stress might be a physical, chemical, or psychological stimulus or a combination of these factors, which pose a threat to an individual's homeodynamic state. Adaptation, according to Väänänen (2004), is an individual's non-genetic ability to respond to repeated stimuli over a prolonged period.

Bergero *et al.* (2005) highlights the fact that to maximize the physiological performance of an athlete, sport scientists need to possess the necessary knowledge of the metabolic and functional processes, particularly the physiological adaptations involved in the specific athletic discipline. Endurance performance, however, does not rely solely on one physiological system, but rather a combination of physiological, biochemical, biomechanical, histological, and neurological characteristics of the athlete (McArdle *et al.*, 2001; Myburgh, 2003). The closer these physiological systems get to the requirements of the specific event, the better the athlete is adapted for prolonged distance activities.

On account of the difficulty to separate the physiological systems into compartments, as they have interactive characteristics, it is easier to first of all discuss the various physiological

variables that determine running performance and then the specific adaptations that occur with endurance training.

## **B. PHYSIOLOGICAL PERFORMANCE DETERMINANTS**

Numerous authors discuss various characteristics of prolonged running performance that determines successful endurance performance and assist in differentiating between endurance trained (from elite to recreational athletes) and untrained individuals. They specifically mention maximal aerobic capacity ( $VO_{2max}$ ), running economy, fractional use of maximal aerobic capacity (%  $VO_{2max}$  or submaximal  $VO_{2max}$ ), lactate threshold (LT) and peak treadmill velocity (PTV) (Noakes *et al.*, 1990; Ball and Herrington, 1998; Bassett and Howley, 2000; Myburgh, 2003; Armstrong *et al.*, 2006; Legaz-Arrese *et al.*, 2007).

More recent research suggests that skinfold thickness, muscle fiber typing (percentage slow compared to fast twitch fibers), oxidative enzyme activity, time to exhaustion at 100% of peak treadmill speed and end diastolic left ventricular (LV) internal diameter at rest are also good performance predictors (Myburgh, 2003; Armstrong *et al.*, 2006; Legaz-Arrese *et al.*, 2007). However, these variables are presently not often used to assess athletes due to impracticality and the high cost involved in these specialized tests.

Consequently, it is important to understand the underlying physiological, biochemical and biomechanical mechanisms which produce these performance prediction factors. Failure in any of these mechanisms could lead to performance decrements (Ball and Herrington, 1998; Bassett and Howley, 2000; Myburgh, 2003; Armstrong *et al.*, 2006; Legaz-Arrese *et al.*, 2007). Armstrong *et al.* (2006) identified the three primary factors, according to priority, which determine endurance performance, i.e. running economy,  $VO_{2max}$  and %  $VO_{2max}$  at race pace.

### **1. Maximum aerobic capacity ( $VO_{2max}$ )**

Maximum oxygen capacity ( $VO_{2max}$ ) is measured during a progressive incremental exercise test and is the greatest amount of oxygen that an athlete can extract, transport and utilize at maximal effort, during aerobic adenosine triphosphate (ATP) synthesis (Bassett and Howley,

2000; Shave and Franco, 2006; Corazza *et al.*, 2007). In other words,  $VO_{2max}$  is an indicator of how effective a runner's cardiorespiratory system functions and is commonly used as a laboratory assessment tool for endurance capacity (Bassett and Howley, 2000; Corazza *et al.*, 2007).

Several studies have shown that elite endurance athletes have very high  $VO_{2max}$  values (Noakes *et al.*, 1990; Bassett and Howley, 2000; Myburgh, 2003; Abbiss and Laursen, 2005; Shave and Franco, 2006; Legaz-Arrese *et al.*, 2007). Therefore,  $VO_{2max}$  can be used to differentiate between endurance trained and untrained individuals. According to Legaz-Arrese *et al.* (2007) and Shave and Franco (2006), there is a substantial relationship between  $VO_{2max}$  and running performance among diverse groups of athletes competing in various distance events. In spite of this,  $VO_{2max}$  is not a choice predictor of performance in runners at the same level, in events like, 800m, 1500m and 3000m to marathons (Legaz-Arrese *et al.*, 2007). Thus, the more homogenous the group of athletes (i.e. elite and subelite athletes), the weaker the relationship ( $r = 0.55$  to  $-0.86$ ;  $P < 0.01$ ) between  $VO_{2max}$  and running performance (Noakes *et al.*, 1990; Myburgh, 2003; Shave and Franco, 2006).

Legaz-Arrese *et al.* (2007) analyzed their data, as well as, data from the literature to reveal the differences in  $VO_{2max}$  values between various performance levels and gender in runners. The authors found that the  $VO_{2max}$  values of athletes participating in events from 100 to 3000m events increased gradually in runners with the same performance levels. Thus a high  $VO_{2max}$  value becomes increasingly more important the longer the distance and could even be used as a tool for talent identification. However, the  $VO_{2max}$  values of athletes of the same performance level, who participated in 3000m to marathon events, do not differ considerably (Legaz-Arrese *et al.*, 2007). This confirms the importance of a well developed aerobic capacity ( $VO_{2max}$ ) in long distance events, although the  $VO_{2max}$  *per se* may not differentiate between athletes at the same level of performance.

Legaz-Arrese *et al.* (2007) thus came to the conclusion that the total contribution of the aerobic energy system in an event determines the importance of  $VO_{2max}$  in the various distances. In 3000m to marathon events the athlete relies predominantly on the aerobic system for energy, while the anaerobic system plays a lesser role.

## 2. Running economy (RE)

Over time and with intense aerobic training,  $VO_{2max}$  plateaus even though running performances seem to continue to improve. This is especially evident in elite endurance athletes, which indicates that there are other factors that play a role in endurance running performance (Myburgh, 2003; Legaz-Arrese *et al.*, 2007). One of these possible factors influencing running performance is running economy, described as the mechanical efficiency of a runner to utilize oxygen to produce a specific running speed (Bassett and Howley, 2000; Shave and Franco, 2006; Chen *et al.*, 2007).

Running economy, along with the fractional utilization of oxygen (%  $VO_{2max}$ ), determines the athlete's running velocity that can be sustained during a prolonged distance run (Bassett and Howley, 2000). A better running economy means a more economical athlete, given that running economy is associated with a lower fractional utilization ( $VO_{2max}$ ) of oxygen during exercise, with a lower rate of fuel consumption for a given speed, thus sustaining glycogen stores (Shave and Franco, 2006).

Therefore it has been established that improved running economy is associated with better performances in well-trained distance runners (Bassett and Howley, 2000; Myburgh, 2003; Chen *et al.*, 2007), but on the other hand, runners with similar race times, might have different running economies (Shave and Franco, 2006). According to Myburgh (2003), the ratio  $VO_2:VO_{2max}$  (the energy cost of running ( $VO_2$ ) at a set submaximal workload to the metabolic power at maximum workload ( $VO_{2max}$ )), accounts for half the variability in subelite athletes' performances in 1500 to 5000m distance events, with the same running times. In addition, several factors influence RE, i.e. muscle temperature, the respiratory exchange ratio (RER), catecholamine concentration in the circulation, muscle glycogen and muscle damage (Chen *et al.*, 2007).

Consequently, each athlete's running economy differs from the next and it is obvious that running economy is velocity specific, for instance, endurance athletes tend to exhibit better running economy at a slower pace, and sprinters have a better RE at a fast pace (Bassett and Howley, 2000; Shave and Franco, 2006). This might explain the difference between running

performances in athletes with a similar  $VO_{2max}$  (Bassett and Howley, 2000; Shave and Franco, 2006).

### **3. Fractional use of aerobic capacity (% $VO_{2max}$ )**

A greater fractional utilization of  $VO_{2max}$  (%  $VO_{2max}$ ) at race pace, indicates a better running performance (Bassett and Howley, 2000; Myburgh, 2003; Shave and Franco, 2006) and is associated with the muscle's ability to adapt to endurance training (Bassett and Howley, 2000). Furthermore, percentage  $VO_{2max}$  at lactate threshold improves with training (Bassett and Howley, 2000). This brings about improvements in the  $VO_{2max}$  that can be sustained during a prolonged run (Bassett and Howley, 2000). In other words, a well-trained endurance athlete can sustain a higher %  $VO_{2max}$  for a longer duration (Shave and Franco, 2006). The percentage of  $VO_{2max}$  at  $16 \text{ km}\cdot\text{h}^{-1}$  ( $r = 0.76$  to  $0.90$ ;  $P < 0.01$ ) in a treadmill incremental test could be used as a performance predictor in endurance trained runners, specifically marathon and ultramarathon runners (Noakes *et al.*, 1990).

However, Legaz–Arrese *et al.* (2007) and Bassett and Howley (2000) made a valid point, that runners do not run endurance races (3000m to marathon distances) at maximum velocity. Generally endurance athletes run races at the highest velocity that can be maintained before metabolic waste products accumulate (%  $VO_{2max}$  at LT) (Shave and Franco, 2006). Usually this occurs at velocities below the athlete's  $VO_{2max}$ , since they can not sustain running velocity at  $VO_{2max}$  for longer than approximately eight minutes (Shave and Franco, 2006). For instance, Bassett and Howley (2000) reported that trained athletes can run at 83 % of their maximum aerobic capacity for two hours.

Additionally, a higher  $VO_{2max}$  is necessary in these long distance events to achieve a greater race velocity from the same %  $VO_{2max}$  and superior performance is determined by an athlete's ability to run at a higher percentage of  $VO_{2max}$  than his competitors (Bassett and Howley, 2000; Legaz–Arrese *et al.*, 2007). Myburgh (2003) also mentions a theoretical model which hypothesizes that if an athlete has optimal capacities in  $VO_{2max}$ , running economy and lactate threshold, a sub two-hour marathon time is achievable.

#### 4. Lactate threshold (LT)

Lactate threshold is the velocity, heart rate or fraction of  $VO_{2max}$  ( $\%VO_{2max}$ ) at the level of lactate accumulation (Shave and Franco, 2006). Four different lactate parameters exist to indicate the oxygen uptake ( $VO_2$ ) at various lactate concentrations, i.e. lactate threshold (at the initial increase of lactate above resting level during incremental test), LT1 (as blood lactate increases one millimole (mM) above resting value), LT2 (as blood lactate reaches two mM) and OBLA (at the onset of blood lactate accumulation at four mM) (Yoshida *et al.*, 1987).

However, even though all four of these parameters correlate well with one another (at least  $r = 0.87$ ), indicating the inter-related reliance of these parameters, lactate threshold (LT) correlates the best with endurance running performance ( $r = 0.73$ ;  $P < 0.01$ ) and aerobic capacity ( $r = 0.84$ ;  $P < 0.01$ ) in a 12-minute run according to Yoshida *et al.* (1987). A strong relationship of  $r = 0.88$  and  $r = 0.99$  exist between LT and a runner's endurance performance at varying durations (Shave and Franco, 2006). The speed at lactate threshold ( $r = -0.80$  to  $-0.92$ ;  $P < 0.01$ ) integrates running economy,  $\% VO_{2max}$  and  $VO_{2max}$  and is the best physiological predictor of an athlete's normal marathon running pace (Noakes *et al.*, 1990; Bassett and Howley, 2000; Shave and Franco, 2006).

In a study by Coetzer *et al.* (1993), the authors investigated the superior performance and fatigue resistance ability of black South African long distance runners compared to white distance runners. What they found was that the black distance runners maintained a higher percentage of their  $VO_{2max}$  at distances above five kilometers (km). Furthermore, the black distance runners were smaller and lighter than their white counterparts ( $69.9 \pm 5.6$  vs.  $56.0 \pm 5.4$  kg); as a result corrections were made for body mass and the authors illustrated that there was no significant difference in  $VO_{2max}$  ( $71.0 \pm 5.3$  vs.  $71.5 \pm 4.6$  mL.min<sup>-1</sup>. kg<sup>-1</sup>), maximal ventilation ( $VE_{max}$ ;  $1.9 \pm 0.3$  vs.  $2.0 \pm 0.3$  L.min<sup>-1</sup>.kg<sup>-1</sup>) and submaximal running economy between the black and white runners. The only difference between the two groups was the lower lactate accumulation in the circulation ( $12.8 \pm 2.2$  vs.  $8.7 \pm 1.7$  mmol.l<sup>-1</sup>;  $P < 0.001$ ) at any running speed in the black long distance runners, which might contribute to their resistance to fatigue (Coetzer *et al.*, 1993).

According to Bassett and Howley (2000) and Myburgh (2003), lactate threshold is the best physiological predictor of distance running over five kilometers. In addition, running velocity at OBLA correlates well ( $r = 0.96$ ;  $P < 0.001$ ) with marathon running performance (Sjodin and Jacobs, 1981). The ability to maintain a high running velocity goes hand in hand with sustained high oxidative ATP production rate (Bassett and Howley, 2000). Yoshida *et al.* (1987) pointed out that one of the reasons why LT is a better indicator of endurance performance is the closer relationship to muscle oxidative capacity ( $r = 0.94$ ). Legaz-Arrese *et al.* (2007) also points out that a higher  $VO_{2max}$  is necessary to obtain a greater velocity in competition from the same %  $VO_{2max}$ .

## **5. Peak treadmill velocity (PTV)**

Peak treadmill velocity (PTV) or maximum aerobic velocity is also an indicator of running performance (Myburgh, 2003). PTV is determined during a progressive incremental test and is the velocity related to  $VO_{2max}$  (Berthon and Fellmann, 2002). PTV is influenced by both running economy and maximal aerobic capacity (Bassett and Howley, 2000; Myburgh, 2003; Armstrong *et al.*, 2006). The athletes that can achieve a higher running velocity during the maximal test are the most economical (Noakes *et al.*, 1990). Noakes *et al.* (1990) defines PTV as the highest speed ( $km \cdot h^{-1}$ ) maintained for 60 seconds during a maximum incremental test. In the situation that an athlete can not complete one minute at a set speed, the preceding workload is taken as the PTV.

The objective of the study by Noakes *et al.* (1990) was to determine which factors are the best predictors of ten to 90 kilometer performance, in trained long distance runners (age:  $32.1 \pm 7.2$ ;  $VO_{2max}$   $66.2 \pm 8.0$   $mL \cdot kg^{-1} \cdot min^{-1}$ ). Forty three experienced runners, specializing in marathon or longer distances, completed a progressive treadmill test. At a second visit, these athletes had to perform three, six minute submaximal runs. The middle interval was derived from the athlete's average marathon velocities. The first and last six minute running bouts were  $1.5$   $km \cdot h^{-1}$  slower and faster, respectively, than this average marathon speed. Several other performance parameters were assessed besides PTV, such as blood lactate concentrations, running economy and  $VO_2$  at various speeds.

The researchers (Noakes *et al.*, 1990) came to two important conclusions. Firstly, that performance at different distances are the best predictor ( $r = 0.91$  to  $0.97$ ;  $P < 0.01$ ) of running performances at any distance from 10 to 90 kilometers in trained long distance runners (marathon and ultramarathon). In other words, those runners that run shorter distances the fastest are most likely to have shorter running times over longer distances. Furthermore, this also meant that the physiological factors involved in longer races do not differ from shorter distances. The second finding was that PTV is the best laboratory performance predictor ( $r = -0.88$  to  $-0.97$ ) at all distances in ultramarathon runners. In marathon runners, PTV was a predictor of performance in all the distances except in marathons. As mentioned before, in respect to marathon performance and trained marathon runners, the lactate turnpoint was a better predictor of performance (Noakes *et al.*, 1990; Bassett and Howley, 2000).

## **C. ADAPTATIONS TO ENDURANCE TRAINING**

Long distance runners undergo various central, i.e. stroke volume and heart rate, and peripheral adaptations in the skeletal muscle with regular endurance training. In the following sections these adaptations will be highlighted.

### **1. Cardiorespiratory system**

The cardiorespiratory system consists of the heart, lungs and blood (Bassett and Howley, 2000). For muscles to produce movement, energy (ATP) must be provided by these physiological systems. The respiratory and cardiovascular systems develop a synergy with similar mechanisms (pressure gradients), to supply fuel and oxygen, along with the removal of metabolic waste products to and from the mitochondria. Throughout prolonged activity the need for oxygen and nutrients increases and these two systems must therefore adapt to sustain muscle activity (Plowman and Smith, 2003).

#### **1.1 Respiratory system**

It is commonly believed that the respiratory system is not a limiting factor during maximal exercise in healthy individuals and because of the large breathing reserve, the respiratory system does not undergo major adaptations with endurance training (McArdle *et al.*, 2001;



Caine *et al.*, 2001; Sheel, 2002; Plowman and Smith, 2003). In fact, according to Bassett and Howley (2000) arteries are 95 % saturated with oxygen during maximal exercises. Thus the respiratory system provides sufficient oxygen for all of the muscles' metabolic needs. However, recent studies indicate that the respiratory system may possibly limit maximal aerobic capacity (Bassett and Howley, 2000; Spanoudaki *et al.*, 2004; Sanchez *et al.*, 2006).

It seems that some well-trained ( $\text{VO}_{2\text{max}}$  above  $55 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) endurance athletes' metabolic and cardiovascular capacities exceed their respiratory capacities (Stewart and Pickering, 2007). This is referred to as exercise-induced hypoxemia (EIH) (Bassett and Howley, 2000; McArdle *et al.*, 2001; Hopkins, 2002; Spanoudaki *et al.*, 2004) and affects about 50% of well-trained male endurance athletes, especially in events lasting longer than 15 minutes (Stewart and Pickering, 2007). Previous studies suggest that EIH is more prevalent in treadmill running than in cycle ergometry in mixed groups. However, Laursen *et al.* (2005) have shown that triathletes show no difference between the two modalities, whereas Stewart and Pickering (2007) indicate EIH at a lower workload is more prominent and common in women and master endurance athletes, compared to young male subjects.

EIH is the result of an increasing alveolar-arterial difference for oxygen and an inadequate increase in alveolar oxygen pressure ( $\text{PAO}_2$ ). It results in a drop in arterial oxygen partial pressure (10 mmHg reduction of  $\text{PaO}_2$ ) at the onset of exercise and oxygen saturation ( $\text{SaO}_2$ ) below resting values (Stewart and Pickering, 2007). This causes haemoglobin desaturation at exercise intensities nearing maximal oxygen consumption (Hopkins, 2002; Spanoudaki *et al.*, 2004; Laursen *et al.*, 2005; Stewart and Pickering, 2007).

McArdle *et al.* (2001), Hopkins (2002) and Spanoudaki *et al.* (2004) list possible causes for EIH, i.e. hypoventilation (mechanical restriction of breathing), impaired ventilation – perfusion ratio (difference between capillary blood flow in the pulmonary and alveolar ventilation), factors restricting gas diffusion (integrity of the alveolar capillary membrane, stress failure of capillary endothelium, interstitial pulmonary oedema), shunting (bypassing areas for diffusion because the blood is redirected between venous and arterial circulations), changes in cytokine concentration (which affects histamine degranulation), deformed red blood cells (influencing respiratory blood flow distribution and diffusion ability), pressure imbalances at the end-capillaries between alveolar oxygen pressure and oxygen pressure in

pulmonary capillaries and increased blood lactate concentration, histamine and blood viscosity.

As aerobic exercise intensity and duration increase, the arterial desaturation worsens (McArdle *et al.*, 2001; Stewart and Pickering, 2007). This means that during maximal and submaximal intensities less oxygen is transported to the active muscles and this reduces  $\text{VO}_{2\text{max}}$  and peak heart rate. This in turn diminishes performance in endurance athletes (Spanoudaki *et al.*, 2004; Grataloup *et al.*, 2007; Stewart and Pickering, 2007). EIH thus leads to an earlier onset of fatigue. The onset of  $\text{VO}_{2\text{max}}$  deterioration differs between individuals. However, it is estimated to be where oxygen desaturation is three to four percent below resting values (Spanoudaki *et al.*, 2004, Stewart and Pickering, 2007).

Another possible mechanism which may lower running performance is exercise-induced respiratory muscle fatigue, particularly during high-intensity exhaustive and prolonged exercises in healthy subjects (Sheel, 2002; Verges *et al.*, 2007). However, the physiological explanation of diaphragmatic fatigue and whether diaphragm fatigue occurs during or after exercise is controversial and needs further investigation (Sheel, 2002; Kabitz *et al.*, 2007; Verges *et al.*, 2007). Typically, no fatigue occurs at intensities below 80 % of  $\text{VO}_{2\text{max}}$  (Sheel, 2002). Kabitz *et al.* (2007) questioned the subject of exercise-induced diaphragmatic fatigue, since the diaphragm is the most fatigue-resistant skeletal muscle. It is highly oxidative and consists of a high volume of capillaries (Sheel, 2002). Tidal volume, breathing frequency and thus minute ventilation progressively increase as exercise continues (Kabitz *et al.*, 2007). Furthermore, during the final part of an event, athletes tend to increase their exercise performance and if diaphragmatic fatigue occurred, this would not be possible (Kabitz *et al.*, 2007).

Theoretically, when an athlete exercises, an increasing demand is placed on the diaphragm, which may result in fatigue and limit performance (Kabitz *et al.*, 2007). The respiratory muscles can fatigue during high intensity exercise owing to a reduced blood flow. The latter is caused by shunting the cardiac output to the active locomotor muscles instead of the respiratory muscles, or secondly by the circulating metabolic byproducts which might interfere with diaphragm contractility (Sheel, 2002). Verges *et al.* (2007) also explains that fatigue influences both inspiratory and expiratory respiratory muscles, in other words the diaphragm,

the abdominal muscles and intercostals. It has also been shown that respiratory muscle training reduces respiratory muscle fatigue and improves exercise performance, indicating that perhaps the respiratory muscles can limit athletic performance (Verges *et al.*, 2007).

## 1.2 Cardiovascular system

The cardiovascular function of endurance trained athletes (i.e. heart, blood vessels and blood) is the key to performance capacity. It has traditionally been recognized as the central and primary limiting factor and endures the most modification with exercise. Consequently, extensive research has been done by sport scientists on the cardiovascular system, as it is relatively easily measurable and predictable. Generally, structural, neural and local adaptations to the cardiovascular system are associated with exercise training (Carter *et al.*, 2003).

### 1.2.1 *Maximum cardiac output*

Various authors (Bassett and Howley, 2000; Rowland and Roti, 2004; Vella and Robergs, 2005; Midgley *et al.*, 2007) suggest that maximal cardiac output ( $Q_{\max}$ ), or more precisely maximal stroke volume, is the predominant, though not the only limiting  $VO_{2\max}$  factor in well-trained athletes, as well as in normal, healthy individuals. In 2000, Bassett and Howley explained that cardiac output contributes about 70 – 85 % to maximum aerobic capacity ( $VO_{2\max}$ ). During exercise two factors influence cardiac output; namely an increased heart rate (HR) and augmented stroke volume (SV) (Plowman and Smith, 2003; Vella and Robergs, 2005). Endurance training results in a further improvement in cardiac output (Krip *et al.*, 1997; Myburgh, 2003; Rowland and Roti, 2004) owing to enlarged hearts with superior pumping capacity and reduced heart rates at rest, when compared to untrained individuals (Bassett and Howley, 2000; McArdle *et al.*, 2001; Rowland and Roti, 2004; Du *et al.*, 2005).

The classic theory (Bassett and Howley, 2000; Vella and Robergs, 2005) assumes that there is a linear relationship between  $VO_{2\max}$  and the cardiovascular variables (Q, SV and HR) during progressive, incremental load exercises. According to this theory  $VO_{2\max}$  is limited by central factors. However, it has not been established yet whether cardiac output plateaus as exercise intensity approaches maximal values (Vella and Robergs, 2005). In a more recent study (Vella and Robergs, 2005), it was suggested that there is a non-linear relationship (continued increase

in cardiac output), therefore suggesting that  $VO_{2max}$  may be limited by peripheral factors, such as the skeletal muscles.

#### 1.2.1.1 Factors influencing cardiac output

(a) Heart rate (HR): Endurance training alters neural factors, i.e. central command, neural reflex and peripheral factors, which result in cardiovascular adaptation, specifically reducing the athlete's heart rate (Carter *et al.*, 2003).

Nervous control during resting heart rate is mainly sustained by the parasympathetic nervous system's (PNS) vagal tone (Freeman *et al.*, 2006). With endurance training, the resting heart rate is lower when compared to untrained individuals. It is attributable to a more pronounced vagal tone (increased parasympathetic hormone acetylcholine) and reduced sympathetic activity (Braith *et al.*, 1999; McArdle *et al.*, 2001; Carter *et al.*, 2003; Freeman *et al.*, 2006). Regular endurance training reduces sympathetic nervous activity, possibly due to the reduction of the reflex heart rate response to the associated myocardial stretch. This reduction in the sympathetic activity decreases the efferent sympathetic neural outflow to the sinoatrial node in the heart (Carter *et al.*, 2003).

In 2003, Carter *et al.* reviewed the adaptations associated with endurance training and indicated that parasympathetic withdrawal occurs in activities at about 60 % of  $VO_{2max}$ . Certain studies have shown that an increase of more than  $12 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$  in  $VO_{2max}$  after regular endurance training is associated with an increase in parasympathetic control of an athlete's heart rate, but this is not a universal finding. Carter *et al.* (2003) did not find increased parasympathetic activity in rowers. It might be that the training history of an athlete influences this particular training response (Carter *et al.*, 2003).

With low-intensity exercise the parasympathetic outflow increases the heart rate, and during moderate to high-intensity exercise the athlete's heart rate increases up to a 100 beats per minute, primarily due to the sympathetic nervous system (SNS), accompanied by the withdrawal of the parasympathetic vagal tone (Coyle and González-Alonso, 2001; Plowman and Smith, 2003; Carter *et al.*, 2003; Du *et al.*, 2005; Carter *et al.*, 2005; MacMillan *et al.*, 2006; Freeman *et al.*, 2006). A further increase of the heart rate, above a 100 beats per minute

(bpm), is caused by the increase in noradrenalin by means of sympathetic stimulation of the cardiac  $\beta$ -adrenergic receptors (Carter *et al.*, 2003). This increase in heart rate contributes to an increase in cardiac output (Q) (Krip *et al.*, 1997).

Some studies (Braith *et al.*, 1999; Rowland and Roti, 2004) have shown that there is no significant difference in maximal heart rate ( $HR_{max}$ ) between endurance trained and untrained individuals. Carter *et al.* (2003), also highlights the fact that continuing endurance training decreases heart rate at submaximal exercise, by reducing sympathetic activity.

A higher aerobic capacity and heart rate variability in trained compared to non-athletes is associated with a faster heart rate recovery (HRR) post-exercise in male and female marathon runners (Du *et al.*, 2005). This is mainly owing to the athlete's enhanced ability to reactivate parasympathetic activity and a further gradual withdrawal in sympathetic activity reduces the HR even more (Braith *et al.*, 1999; Carter *et al.*, 2003; Du *et al.*, 2005; Freeman *et al.*, 2006). The reduced resting heart rate, due to increased parasympathetic stimulation, could be responsible for the increased heart rate recovery (HRR) in trained runners (Du *et al.*, 2005). The mechanism that results in lowering the heart rate during recovery is not well understood (Carter *et al.*, 2005). A faster HRR indicates higher levels of heart rate variability (HRV), higher aerobic capacity and exaggerated blood pressure response to exercise in trained athletes compared to untrained individuals (Du *et al.*, 2005).

Heart rate recovery after exercise depends on several factors of which exercise intensity has a prominent influence (Du *et al.*, 2005). For instance, heart rate during low-intensity exercise demonstrates an exponential decline when returning to resting levels, while heart rate during moderate to high-intensity exercise is characterized by two distinct phases, i.e. an initial exponential drop followed by a slower decline to resting level. This is observed in untrained and trained groups (Du *et al.*, 2005). Additionally, a trained runner's cardiorespiratory fitness from long-term endurance training, cardiac neural modulation, specifically of the autonomic nervous system and baroreflex sensitivity, contributes to heart rate recovery (Du *et al.*, 2005). Hormonal changes might also be a possible factor, although Du *et al.* (2005) pointed out that catecholamine concentrations increase during exercise and is not removed faster post-exercise.

Besides this autonomic regulation during exercise, a change in baroreflex sensitivity and peripheral adaptations to exercise also accommodates the lower heart rate in endurance athletes (Braith *et al.*, 1999; Carter *et al.*, 2003; Du *et al.*, 2005; Carter *et al.*, 2005). Peripheral receptors in the muscles, blood vessels and joint proprioceptors stimulate the cardiovascular centre in the brain and either modifies the parasympathetic or sympathetic outflow (McArdle *et al.*, 2001; Plowman and Smith, 2003; Freeman *et al.*, 2006). This increases heart rate at the onset of exercise by 30 to 50 beats per minute (McArdle *et al.*, 2001; Plowman and Smith, 2003; Freeman *et al.*, 2006).

Circulatory steady state is regulated by a rapid response of the pressure-sensitive arterial baroreceptor reflex to a change in arterial blood pressure (McArdle *et al.*, 2001; Du *et al.*, 2005). An excessive increase in blood pressure during exercise increases the firing rate of baroreceptors. These send negative feedback to the command centre to slow the heart rate, with an increase in efferent cardiac parasympathetic activity and a decrease in sympathetic activity. Furthermore, the reflex inhibits vasoconstriction and vasodilates the blood vessels, thereby decreasing the blood pressure (McArdle *et al.*, 2001; Carter *et al.*, 2003). Baroreflex sensitivity is usually enhanced in well-trained runners (Du *et al.*, 2005). In addition, in the active muscles the metabolic byproducts through chemoreceptors, the increase in temperature by means of the thermoreceptors and the contracting muscle (stretch and tension) also activate the neural reflex mechanism (Carter *et al.*, 2005). This intramuscular feedback stimulates the heart and increases peripheral perfusion.

Central command also shifts the baroreflex response to a higher blood pressure threshold, which means that during exercise higher blood pressure levels are regulated more efficiently (McArdle *et al.*, 2001; Carter *et al.*, 2003). The carotid baroreflex is altered, during low to moderate intensity exercise, so that no gain or sensitivity of the baroreflex is obtained and the athlete's physiological responses work around the blood pressure response. During dynamic exercises the reflex is reduced. Furthermore, it is possible that long-term endurance modifies the autonomic function and reduces baroreflex control and decreases baroreceptors sensitivity (Carter *et al.*, 2003).

(b) Stroke volume (SV): As mentioned before, endurance trained athlete's heart rate is lower at rest and submaximal exercises intensities. Therefore, the athlete's stroke volume is

augmented at the onset of exercise, to compensate for the reduced heart rate. The increased stroke volume contribute to the increase in the maximum oxygen uptake (Bassett and Howley, 2000; McArdle *et al.*, 2001; Rimaud *et al.*, 2007).

Endurance training improves the athlete's stroke volume at rest and at maximum exercise more than in untrained individuals. Trained cyclists and distance runners, on average, exhibit a 35 – 50 % greater maximal stroke volume than untrained individuals (Rowland and Roti, 2004). This improved stroke volume is mainly due to an increase in ventricular preload and explains the larger aerobic capacity in trained compared to untrained athletes (Krip *et al.*, 1997; Rowland and Roti, 2004).

According to Sundstedt *et al.* (2003 and 2004) stroke volume depends on a fine balance between left ventricular (LV) filling and systolic ejection (SE). Several mechanisms have been proposed to assist in this function, consequently increasing stroke volume. They are:

(a) *Increased left ventricular diastolic function (LVDF):* Enlarged ventricular filling volume (preload) and filling rate may be two factors responsible for the increase in stroke volume (Plowman and Smith, 2003; Vella and Robergs, 2005; Kivisto *et al.*, 2006; Midgley *et al.*, 2007). Endurance training results in improved LVDF by enhancing left ventricular (LV) compliance, size, mass and relaxation rate (Krip *et al.*, 1997; McArdle *et al.*, 2001; Du *et al.*, 2005; Kivisto *et al.*, 2006). This in turn allows for greater ventricular filling.

Increased end diastolic rate is a result of a decreased heart rate or an increased venous return (McArdle *et al.*, 2001). Endurance trained athletes have a reduced diastolic filling time, but more improved diastolic filling rate at rest and during exercise (Krip *et al.*, 1997; Kivisto *et al.*, 2006). Additional cardiovascular adjustments to increase preload by means of increased venous return are a greater calf muscle-pump and higher transmural filling pressure. This is as a result of decreased intrathoracic pressure at higher ventilations (Krip *et al.*, 1997).

(b) *Improved systolic emptying:* Endurance athlete's systolic emptying is enhanced by a combination of factors. Either by longer ventricular ejection time, which allows more blood to be ejected, or a more forceful systolic ejection due to enhanced myocardial

contractility, or a reduced total peripheral resistance (TPR) that lessens the cardiac afterload (Krip *et al.*, 1997; McArdle *et al.*, 2001; Plowman and Smith, 2003; Vella and Robergs, 2005; Midgley *et al.*, 2007). Myocardial contractility is improved by endurance training (Krip *et al.*, 1997; McArdle *et al.*, 2001). This may either be the result of higher catecholamine levels during exercise, or an augmented Frank-Starling mechanism, which will produce a more forceful systolic ejection (McArdle *et al.*, 2001).

The advantage of these systolic and diastolic left ventricular modifications along with an increased diastolic reserve is that it produces a better-developed aerobic capacity that allows runners to increase their performance ability. Another possibility that increases stroke volume is increased blood volume to the heart (venous return), accompanied by reduced peripheral resistance.

(c) *Venous return:* Endurance athletes have greater blood volumes than their untrained counterparts (Krip *et al.*, 1997; McArdle *et al.*, 2001). The blood that returns to the heart (venous return), together with decreased venous pooling is possibly the greatest contributor of increased stroke volume during endurance activities (Saladin, 2001; Plowman and Smith, 2003; Rowland and Roti, 2004). Several authors have indicated that not only will a larger blood volume increase the athlete's stroke volume, but also the cardiac output (Krip *et al.*, 1997; Midgley *et al.*, 2007).

There are five possible mechanisms through which the body improves venous return, which results in an increased preload (Saladin, 2001; Plowman and Smith, 2003; Rowland and Roti, 2004):

- (i) *Pressure gradient:* A pressure gradient throughout the circulation causes blood to flow from a high to a low pressure. However, in the veins this pressure gradient is much lower than in the arteries. The pressure difference between the central venous pressure (average 4.6 mmHg) and average pressure in the veins (between 12 to 18 mmHg) is about seven to 13 mmHg, which supports blood flow toward the heart (Saladin, 2001; McArdle *et al.*, 2001).



- (ii) Muscle-pump function: Muscular activity, specifically of the calf muscle, together with functioning venous valves as well as increased venoconstriction, improves venous return to the heart and is essential for healthy circulation (Plowman and Smith, 2003; Rowland and Roti, 2004; Qiao *et al.*, 2005). Several authors compare the calf muscle-pump function (CMPF) to the action of the left side of the heart. In other words, the large deep veins surrounded by the *Gastrocnemius* and *Soleus* muscles form a compartment that includes a pump chamber and outflow tract comparable to the left ventricle and aorta of the heart (Qiao *et al.*, 2005; O'Donovan *et al.*, 2006 and 2005).

The muscles massage the blood, with a rhythmical action towards the heart, and the valves prevent back flow, thereby increasing the preload (Frank – Starling mechanism) (McArdle *et al.*, 2001; Saladin, 2001; Rowland and Roti, 2004). This mechanism supports venous return and prevents blood pooling. It works in a similar fashion as pressurized suits worn by pilots, and compression garments worn by individuals with varicose veins. When an individual stands upright, the calf muscle reduces hydrostatic shifts of blood in the veins of the lower extremities (McArdle *et al.*, 2001), thus reducing oedema. Adequate functioning of venous valves, forceful muscular actions of the calf muscle, full range of motion in the ankle joint and normal muscle fasciae are essential for effective CMPF (Qiao *et al.*, 2005).

(iii) Respiratory pump: The venous blood flows from the abdominal to the thoracic cavity by means of a drop in internal pressure during inhalation, and an increase in abdominal cavity pressure, due to the lowering of the diaphragm during expiration. Through pressure differences in the inferior vena cava, along with the help of the one-way valves, blood is returned to the heart (Saladin, 2001). It is thus possible that if endurance training causes improved respiratory muscle function (i.e. respiratory muscle strength and endurance) that the function of the respiratory pump may also be enhanced. This will then contribute to the increase in venous return.

(iv) Cardiac suction: When the atrial opening enlarges, it creates a vacuum. This suction extracts blood from the venae cavae and pulmonary veins (Saladin, 2001).

(v) Gravity: The force of gravity that works against the lower extremities, works in favour of the veins in the neck and head. Blood flows downhill towards the heart. In the upright position, however, gravity counteracts the return blood flow to the heart, which decreases preload diminishing stroke volume and cardiac output (McArdle *et al.*, 2001; Saladin, 2001).

### 1.2.2 Blood flow

Regular endurance training increases muscle capillaries in the working muscle (Ball and Herrington, 1998; Nielsen *et al.*, 2003). This allows for an improved distribution of blood flow to the exercising muscles, increases oxygen delivery to the active muscles and is paralleled by an elevated  $VO_{2max}$  (Bassett and Howley, 2000).

Furthermore, the increase in metabolic activity in muscle cells at the onset of exercise serves as a signal for increased blood flow to the active muscles via several stimuli that cause active vasodilation. These stimuli include a decrease in  $PO_2$  and an increase in  $PCO_2$ , an increase in blood temperature and blood flow, a decrease in pH, adenosine, magnesium, potassium ions and an increase in nitric oxide, which are all associated with exercise (McArdle *et al.*, 2001).

## 2. **Musculoskeletal system**

Internal muscle structure performs three main functions: it specifically causes force production in the myofibrils, it produces excitation–contraction coupling and its reversal within sarcoplasmic reticulum (SR), and assists in oxidative ATP production in the mitochondria (Myburgh, 2003). Therefore one can conclude that muscle action provides the foundation for all human movement, by converting chemical energy (ATP) to mechanical energy. Endurance training is one of the three methods to stimulate an adaptive response, via the overloading principle, in the highly adaptable musculoskeletal system (Ball and Herrington, 1998).

## 2.1 Skeletal muscle fiber types

Different muscle fiber types meet functional (muscle contractility) and metabolic demands, and it is these factors that classify muscle fibers into two main types i.e. Type I (slow twitch fibers) and Type II (fast twitch fibers) (Ball and Herrington, 1998; Saladin, 2001).

Type I fibers have a high oxidative and low glycolytic capacity, with a slow twitch, are highly resistant to fatigue, and are therefore ideal for prolonged running (Ball and Herrington, 1998). Type I slow twitch fibers are thought to be more common in endurance athletes due to the increased capillary density (Ball and Herrington, 1998; Myburgh, 2003). Type II fibers are characteristically fast twitch, highly fatigueable and are used predominantly during activities where anaerobic metabolism (immediate or rapid glycolytic system) is used to generate energy; like stop-and-go, sprinting and /or vigorous activities. Muscle actions are fast and powerful during these activities and this is possibly due to the high content of myosin ATPase in Type II muscle fibers (Ball and Herrington, 1998; McArdle *et al.*, 2001).

Even though athletes recruit both fiber types during submaximal activities, it is traditionally accepted that slow-twitch fibers (type I) are the fibers that are predominant in prolonged activities (Ball and Herrington, 1998; McArdle *et al.*, 2001; Myburgh, 2003). The reason for this is that type I fibers contract and relax more slowly, have a high oxidative capacity (oxygen-dependent metabolism), and low glycolytic ability (low ATPase activity), unlike the fast-twitch muscle fibers (Ball and Herrington, 1998; Noakes, 2001). This is further confirmed by the abundant and large mitochondria (with high concentration mitochondrial enzymes and myoglobin), as well as the fiber's red colour due to the magnitude of capillaries in the fiber, allowing enhanced blood flow to the fibers (Noakes, 2001; McArdle *et al.*, 2001).

## 2.2 Musculoskeletal adaptations

Increased capillary growth in the active muscles, oxidative enzyme activity (for example succinate dehydrogenase and cytochrome oxidase) necessary for aerobic energy production, mitochondrial number and volume (the site of energy production at cellular level), improved resting myoglobin and in some cases increased muscle fiber size, especially slow twitch fibers, which are linked with the improved oxidative capacity in skeletal muscle, are some of the

musculoskeletal adaptations associated with endurance training (Ball and Herrington, 1998; Noakes, 2001; Nielsen *et al.*, 2003; Abbiss and Laursen, 2005; Mizuno *et al.*, 2005).

Oxidative enzyme activity as well as capillary density is related to enhancement of the central factors, like  $VO_{2max}$  in sub-elite athletes (Myburgh, 2003; Abbiss and Laursen, 2005). Skeletal muscle mitochondrial capacity depends on training volume, training composition (duration, frequency and intensity) and associated intracellular signaling or gene transcription (Myburgh, 2003). Furthermore, Myburgh (2003) observed that some individuals with the same  $VO_{2max}$  might even have different mitochondrial capacity. The benefits of an enhanced oxidative capacity are sustained high intensity running and lower plasma lactate accumulation. All of these factors improve peripheral adaptation (Myburgh, 2003).

Mizuno *et al.* (2005) described more training adaptations to cardiac and skeletal muscles. For instance, cardiac muscle demonstrates increased contractility as well as resistance to ischemia and skeletal muscle has augmented glucose uptake and reactive hyperemia. Some animal studies indicate that tendons demonstrate an increased collagen deposit as well as improved number and size of fibrils, after endurance training, which could make the tendons more elastic (Ball and Herrington, 1998).

### **3. Metabolic system**

Prolonged activities are extremely demanding on the metabolic system (Bergero *et al.*, 2005). Aerobic and anaerobic endurance is specified according to the fuel utilized and energy metabolism involved (Legaz–Arrese, 2007; Bergero *et al.*, 2005). Prolonged running activities primarily use carbohydrates for fuel, supported by fat metabolism, with no more than 5% from protein in marathon runners (Atwood and Bowen, 2007).

During prolonged activities where the runner's heart rate is below 70 % of his maximum (moderate intensity), ATP production is produced mainly via oxidative phosphorylation (Vander, 1998; Bassett and Howley, 2000). The oxygen utilized is equal to the ATP generated during submaximal endurance activities. In other words, sport scientists can assess the ATP production rate by measuring the oxygen uptake (Bassett and Howley, 2000). For a runner to

maintain a high running velocity, he/she must have the ability to maintain a high oxidative ATP production rate (Bassett and Howley, 2000).

In strenuous activities the oxygen consumption fails to keep up with the increasing oxygen demands of the metabolic processes. Subsequently the energy supply shifts to predominantly oxygen-independent pathways, i.e. anaerobic glycolysis (Bassett and Howley, 2000). The glycolytic system is a multisubstrate and-enzymatic pathway. It operates in the absence of oxygen, and lactic acid is produced as a by-product. This lactic acid dissociates in the circulation and forms hydrogen and lactate ions (Vander, 1998).

### 3.1 Metabolic adaptations

The intracellular (ATP):(ADP)(P<sub>i</sub>) ratio that indicates the rate of ATP production to the rate of ATP breakdown is an important cellular metabolism signal (Noakes, 2001). For the first five to ten minutes of exercise at submaximal intensity, the body utilizes mainly muscle glycogen to produce ATP (adenosine triphosphate), followed by blood glucose and fatty acids. In exercise that lasts longer than 20 minutes, more and more fatty acids and less glucose are utilized (Lowery, 2004). Several metabolic adaptations take place with endurance training, which have a profound effect on the fuel utilization of the body and enhanced endurance performance. Abbiss and Laursen (2005) maintain that proof of all of these endurance-training adaptations is the elevated anaerobic threshold of endurance-trained athletes at a higher fraction of VO<sub>2max</sub>. In other words these adaptations are manifested in a rightward shift of the anaerobic/lactate threshold.

#### 3.1.1 *Structural modifications*

With endurance training, the mitochondria in the active muscle develop an enhanced ability to utilize oxygen, due to the increase in size and number of the mitochondria, as well as increased mitochondrial enzymatic activity. These changes allow the long distance runner to produce more ATP aerobically (McArdle *et al.*, 2001) and might result in a slightly improved VO<sub>2max</sub> in endurance athletes (Bassett and Howley, 2000; McArdle *et al.*, 2001, Noakes, 2001). Furthermore, the high enzyme activity allows an athlete to run for prolonged periods at a higher aerobic capacity, before lactate accumulates in the circulation (McArdle *et al.*, 2001).

The reason for this is that increased mitochondria allows the trained athlete to use more blood borne free fatty acids for fuel at any exercise intensity, whereas untrained individuals start to use carbohydrates at a low-intensity (Noakes, 2001). This means that endurance trained individuals' mitochondria will work at a higher (ATP):(ADP)(Pi) ratio and use more fat for energy than carbohydrates at high-intensities (Noakes, 2001).

### 3.1.2 *Carbohydrate metabolism*

About 400 – 600g of carbohydrates are stored in the muscle and liver, but the energy input of carbohydrates depends on the intensity and duration of the exercise (Lowery, 2004; Atwood and Bowen, 2007). Even though fat contains more than double the energy (9 kcal) than carbohydrates (4 kcal) (Lowery, 2004), at higher intensity levels more energy is obtained from carbohydrate metabolism, owing to the greater production of ATP per O<sub>2</sub> from glucose (ATP: O<sub>2</sub> = 3), than the ATP per O<sub>2</sub> from fat (ATP: O<sub>2</sub> = 2.8) (Atwood and Bowen, 2007). The increase in exercise intensity parallels the progressive decrease in the (ATP):(ADP)(P<sub>i</sub>) ratio, which signals the increase in carbohydrate metabolism via glycolysis (Noakes, 2001). Thus, carbohydrates are the preferred fuel during high-intensity exercise (Noakes, 2001).

Endurance training leads to an enhanced capacity to oxidize carbohydrates during maximal exercise, but during submaximal exercises glycogen is spared and instead fatty acids are utilized (McArdle *et al.*, 2001, Noakes, 2001; Myburgh, 2003; Lowery, 2004; Abbiss and Laursen, 2005). Possible reasons for this reduced carbohydrate metabolism is due to stored muscle and liver glycogen, as well as a glycogen sparing effect and increased rate of fat oxidation (Noakes, 2001; Myburgh, 2003; Abbiss and Laursen, 2005). As little as four days of endurance training enhances glycogen-storing capacity (Noakes, 2001).

Regular endurance training leads to a further increase in insulin sensitivity in the skeletal muscles of the runner compared to untrained individuals (Nielsen *et al.*, 2003). It is suggested that increased sensitivity to insulin shifts the catabolic environment to an anabolic state, with the right nutrients (Ivy, 2004). Theoretically the insulin assists glucose uptake and muscle damage recovery after exercise, by increasing amino acid uptake, protein synthesis and guards against further protein degradation (Ivy, 2004). (Nielsen *et al.*, 2003; Ivy, 2004). Several authors assume that the enhanced glucose transporter protein (GLUT 4) might have a possible

impact on the improved insulin sensitivity. However, the mechanism, by which increased insulin sensitivity enhances glucose uptake, protein synthesis and amino acid uptake, is still not fully understood (Nielsen *et al.*, 2003; Ivy, 2004).

These aerobic training adaptations assist recovery, maintain glucose homeostasis and improve endurance in strenuous activities, since the amount and rate of glucose utilization is the limiting factor in long strenuous exercise where the perception of fatigue and the “hitting the wall” effect affects athletes (McArdle *et al.*, 2001; Ivy, 2004; Lowery, 2004; Atwood and Bowen, 2007).

### 3.1.3 *Fat metabolism*

The longer the duration and the lower the intensity of the exercise, the more energy supply relies on fat oxidation (Lowery, 2004; Atwood and Bowen, 2007). As mentioned in the previous section endurance training enhances fat utilization during submaximal activities (McArdle *et al.*, 2001). This is a result of increased mitochondrial enzyme activity, improved blood flow in trained muscles, increased fatty acid metabolic enzymes, additional resting muscle and plasma fatty acids, more intramuscular utilization of triglycerides (McArdle *et al.*, 2001; Noakes, 2001; Atwood and Bowen, 2007) and attenuated catecholamine release for the same absolute power output (McArdle *et al.*, 2001).

In other words endurance training allows an athlete to run at a higher (ATP):(ADP)(Pi) ratio in mitochondria (Noakes, 2001). This will allow the athlete to utilize fat for longer as the main energy fuel and be less dependent on carbohydrate metabolism (Noakes, 2001). Therefore endurance-trained athletes will produce less and clear more acidic by-products of carbohydrate metabolism, like lactate. Furthermore, endurance training also enhances the ability of other organs like kidneys, liver and the heart as well as active and non-working skeletal muscle to remove and metabolize lactate during exercise (Noakes, 2001).

Dietary glucose cannot sustain the glucose stores. Therefore increased fat metabolism conserves glycogen stores, which allows more glycogen during high intensity prolonged exercise, delays the accumulation of byproducts of glycolysis and aids recovery (McArdle *et al.*, 2001; Shave and Franco, 2006; Atwood and Bowen, 2007). Furthermore, improvement in

fatty acid beta-oxidation and respiratory ATP production contribute to maintaining the cell's integrity and high functioning level, thus enhancing endurance capacity independent of increases in glycogen reserves or aerobic capacity (McArdle *et al.*, 2001).

#### 3.1.4 *Metabolite accumulation*

During strenuous activities, such as the last stretch of a prolonged run or throughout high intensity activities, where the respiratory system cannot keep up with the energy demand, the glycolytic system is implemented. The abnormal accumulation of lactate and CO<sub>2</sub> signifies the initiation of anaerobic energy metabolism (Vander, 1998; Corazza *et al.*, 2007). Since it is the highest VO<sub>2</sub> value where the focus shifts from aerobic energy production (at muscular level) to the anaerobic system (Corazza *et al.*, 2007), this point is called the anaerobic threshold (AT) and above this point endurance capacity is compromised (Corazza *et al.*, 2007).

For this reason lactate is one of the exercise metabolites which have received a lot of attention. Traditionally it was thought that lactate causes muscle soreness and fatigue, and is a primary cause of O<sub>2</sub> debt. Therefore, lactate was seen as a waste product of exercise with no additional purpose.

Contemporary research suggest now that (i) skeletal muscle is not only the producer of lactate but also consumes lactate, (ii) skeletal muscle may be able to reverse the glycolytic pathway and convert lactate into glycogen, (iii) lactate is transported by a series of protein transporters, namely monocarboxylate transporters (MCT), and does not just diffuse through membranes, (iv) that lactate is not just produced due to inadequate oxygen supply, but also via the biochemical manipulation of glycolysis, increased sympathadrenal activity, recruitment of glycolytic motor units and the disrupted lactate removal: production ratio and finally (v) the Lactate Shuttle Theory, which stipulates that since lactate can be exchanged between tissues, it acts as an metabolic intermediate (Brook *et al.*, 1985; Gladden, 2000<sup>a</sup>; Billat *et al.*, 2003).

In the recovery period from high intensity short duration or prolonged exercise, such as middle or long distance races, lactate is transferred from the blood, either to the resting muscles, moderately active muscles or previously active muscles. There are six possible factors that



enhance the consumption of lactate by skeletal muscle, i.e. the metabolic rate, blood flow, lactate and hydrogen concentration, the fiber type and the athlete's level of exercise training.

A high metabolic rate, which does not produce more extracellular lactate, assists lactate removal. Elevated metabolic rate is associated with a faster glycolytic rate and related lactate production. However, during steady state submaximal activities the metabolic rate is increased, but without the associated glycolysis. Lactate utilization is increased due to the faster rate of pyruvate and NADH oxidation. Furthermore, it has been suggested that exercise increases the lactate transport across the sarcolemma membrane, in a similar fashion as glucose transport (~62 to 400%). Nevertheless, metabolic rate only seem to have a minimal effect on lactate transport across the sarcolemma (0 to 28%) (Gladden, 2000<sup>b</sup>; Billat *et al.*, 2003).

In addition, adequate blood flow should ideally be equal to the rate of net lactate uptake. This would mean the athlete would need an optimal lactate uptake/perfusion (L/Q) ratio, to consume lactate more efficiently. This will maintain the gradient difference between lactate and hydrogen concentration across the membrane (Gladden, 2000<sup>b</sup>). However, Gladden (2000<sup>b</sup>) explains that equivocal evidence exists that increased blood flow will assist lactate exchange.

Lactate and hydrogen concentration also have an influence on lactate consumption in the skeletal muscle. For instance, an increased arterial lactate concentration stimulates an increased lactate uptake by the muscle. This elevated lactate concentration gradient continues up to the point of saturation, where it plateaus (~20–30 mM), for both the metabolic systems and/or the monocarboxylate transport system (Gladden, 2000<sup>b</sup>). It has been suggested that if the intramuscular hydrogen concentration ( $[H^+]$ ) is increased, which then inhibits lactate production, while enough extracellular lactate is available, then lactate consumption will be increased. This increased  $[H^+]$  will not inhibit muscle contractility. The  $[H^+]$  gradient will stimulate the uptake of lactate through the sarcolemma (Gladden, 2000<sup>b</sup>).

The fiber type and the athlete's level of exercise training also have an influence on lactate utilization by the muscle. Lactate is mainly produced by Type IIx fibers, and is then transported to type I or IIa to be oxidized. The oxidative muscle fibers will enhance lactate

consumption more than glycolytic fibers (Gladden, 2000<sup>b</sup>). The reason being that these fibers are more adapted to lactate oxidation. The slow twitch fibers have a greater capillary density with a high content of the enzyme lactate dehydrogenase (Martin *et al.*, 1998). In addition, the oxidative fibers begin to oxidize lactate at a lower blood lactate concentration and at a faster rate, since the oxidative fibers have a greater ability to transport lactate across the sarcolemma (37 to 109%). This might be due to the fact that oxidative fibers have more type I monocarboxylate transporters (Gladden, 2000<sup>b</sup>).

Then finally, endurance training increases lactate utilization by the skeletal muscles, as well as the clearance from the muscles and blood (Gladden, 2000<sup>b</sup>; Rimaud *et al.*, 2007). The faster lactate clearance from the muscles and blood is mainly due to increased blood flow, i.e. higher cardiac output and improved redistribution of blood to the active muscles (Draper *et al.*, 2006; Rimaud *et al.*, 2007). Additionally, highly endurance trained athletes have a better ability to buffer H<sup>+</sup>. Even though research is inconsistent with regards to the relationship between lactate clearance and exercise performance, some authors believe that these changes contribute to a higher AT/LT threshold enabling the athlete to maintain a higher running pace for longer (McArdle *et al.*, 2001; Noakes, 2001).

The mechanism through which endurance training improves lactate consumption is not fully understood. The metabolic adaptations in the skeletal muscle due to training include (i) increased mitochondria with increased oxidative enzymes, which enhances the athlete's oxidative capacity and leads to less lactate production (Noakes, 2001; McArdle *et al.*, 2001; Myburgh, 2003; Abbiss and Laursen, 2005). , (ii) when lactate is converted to pyruvate, the athlete has an enhanced ability to utilize the pyruvate due to increased pyruvate dehydrogenase concentration; (iii) improved malate-aspartate shuttle activity which facilitates oxidation of NADH, resulting from lactate to pyruvate conversion and (iv) a shift in lactate dehydrogenase isozyme pattern from muscle isoform to heart isoform (Gladden, 2000<sup>b</sup>).

#### **4. Neuroendocrine system**

The neurohormonal systems act as a mediator between several physiological systems and the shifting environments from rest to exercise. The primary aim is to maintain homeostasis, while enough ATP is produced to meet the demands of the active muscles (Myburgh, 2003).

The autonomic nervous system (ANS) consists of parasympathetic and sympathetic events. MacMillan *et al.* (2006) describes the autonomic nervous activity as the balance between these two actions. Endurance training enhances the autonomic effect, by increasing the parasympathetic nervous activity and reducing the sympathetic nervous activity (Braith *et al.*, 1999; Carter *et al.*, 2003; MacMillan *et al.*, 2006). The evidence for this is the lower resting and recovery heart rates of endurance athletes (Väänänen, 2004; MacMillan *et al.*, 2006).

Catecholamine release improves left ventricle compliance and myocardial contractility (McArdle *et al.*, 2001). This supports the heart's ability to accommodate more blood during diastole and to enhance systolic ejection with a more powerful contraction (McArdle *et al.*, 2001). An increased catecholamine concentration is released during moderate to high-intensity exercise (Du *et al.*, 2005) and is augmented by endurance training (McArdle *et al.*, 2001). The removal post exercise however, does not seem to significantly change due to endurance training (Du *et al.*, 2005).

Another endogenous modulator of several physiological functions in the CNS and peripheral organs is the Adenosine receptor subtype, namely Adenosine<sub>A<sub>2A</sub></sub>. It is more abundant in skeletal and cardiac muscles of endurance athletes than in untrained groups (Mizuno *et al.*, 2005). In the myocardium Adenosine<sub>A<sub>2A</sub></sub> limits ischemia and controls heart contractions, while in the cytosol and plasma membrane of skeletal muscle, specifically in type I fibers, Adenosine<sub>A<sub>2A</sub></sub> helps to regulate glucose uptake, blood flow and contractile force production in the muscle (Mizuno *et al.*, 2005).

## **5. Conclusion**

To conclude, human endurance is potentially reaching its peak during the 21<sup>st</sup> century, in that athletes know how to train, to optimally develop all the physiological systems. This means that athletes will start to rely increasingly on supplementary or enhancement techniques in addition to mental staying power to improve their physical abilities (Noakes, 2006). Compression garments may be a possible method to enhance the circulation's pressure gradient, assist calf muscle-pump function and counteract the forces of gravity, to reduce oedema and support blood flow toward the heart, thus enhancing performance.

## CHAPTER THREE

### POST-EXERCISE RECOVERY

#### A. INTRODUCTION

Demanding running events like marathons and ultramarathons may deplete energy reserves and trigger exercise-induced muscle damage and injury (Myburgh, 2003; Ivy, 2004). The process and consequences of muscle damage and injury are associated with various physiological systems. Muscle damage may prompt inflammation and swelling (oedema) as well as decreased lactate clearance rates. These factors are associated with diminished muscular strength, impaired functional capacity (up to 40 – 50 % immediately post-marathon races), augmented muscle soreness and impaired range of motion (flexibility) (Ball and Herrington, 1998; Byrne and Eston, 2002; Kraemer *et al.*, 2004; Miller *et al.*, 2004; Dawson *et al.*, 2005; Takahashi *et al.*, 2006; Rimaud *et al.*, 2007).

When an athlete runs, the body has to absorb powerful forces up to two or three times the body mass of the individual (Sanchez *et al.*, 2006). Mountainous endurance races place even additional stress on the athlete's body. The reason is that mountainous runs involve steep inclines and descents that necessitate a specific running technique to compensate for the uneven, steep, and rough surface. This could further contribute to the onset of fatigue (Sztajzel *et al.*, 2006). Furthermore, it places extreme stress on the cardiovascular system and is certainly associated with very high levels of catecholamines (Sztajzel *et al.*, 2006). Running on uneven surfaces causes muscle action to generate unaccustomed repetitive and excessive forces, which produces overuse-injuries and muscle damage (Ball and Herrington, 1998).

All of these factors may lead to a long recovery period, which would mean an extended absence from races and training sessions (Kraemer *et al.*, 2004; Barnett, 2006). In addition, the more a runner trains, the less chance there is for injuries on the race day, provided that sufficient recovery is allowed. In other words, an extended absence from training would make the athlete more prone to muscle damage and injury when running long distance races (Miller *et al.*, 2004; Sanchez *et al.*, 2006).

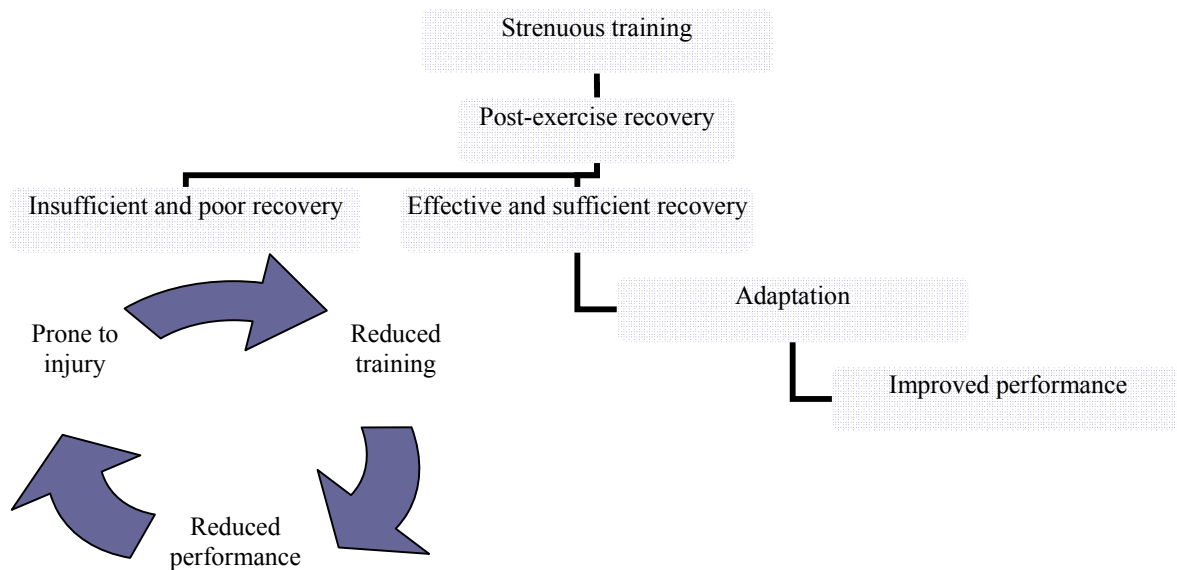


Figure 1. Post-exercise recovery and the impact on athletic performance

In addition, ineffective recovery could be detrimental to running performance and cause injury in future endurance running activities (Figure 1.) (Kraemer *et al.*, 2004; Miller *et al.*, 2004; Barnett, 2006; Gill *et al.*, 2006). Athletes and their management will seek any advantage when training and preparing for events. Optimizing post-exercise recovery from training and races may benefit the subsequent training and performance of competitive athletes over a period of time (Gill *et al.*, 2006).

## B. RESEARCHING MUSCULAR RECOVERY

To determine the factors involved in exercise-induced muscle damage and injury, sport scientists attempt to simulate muscle actions that lead to muscle damage, in a controlled environment. In addition, functional tests are used to establish whether the involved muscles have recovered. This helps sports scientist to determine if and how recovery techniques work and which are the best possible means, related to a specific type of sport.

### 1. Delayed onset of muscle syndrome (DOMS)

One of the common methods by which sport scientists study the recovery process, is to induce delayed onset of muscle soreness (DOMS) (Cleather and Guthrie, 2006). DOMS is the result

of eccentric, unaccustomed, and/or strenuous exercise that damages the skeletal muscle (Craig *et al.*, 1996; Ernst, 1998; Hemmings, 2001; Cleather and Guthrie, 2006). This exercise-induced muscle soreness is evident when the muscle is palpitated, stretched, or contracted (Zainuddin *et al.*, 2005). There have been incidences where muscle soreness peaked as soon as ten hours post-exercise but generally it peaks between 24 – 72 hours and may possibly last as long as seven to ten days (Craig *et al.*, 1996; Ball and Herrington, 1998; Miller *et al.*, 2004; Cochrane, 2004; Zainuddin *et al.*, 2005; Cleather and Guthrie, 2006; Takahashi *et al.*, 2006).

The exact process that leads to DOMS is not fully understood. However, DOMS is found to be more severe after eccentric muscle actions (Miller *et al.*, 2004; Cochrane, 2004; Zainuddin *et al.*, 2005). There are several theories with regard to the possible causes of DOMS. The most widely accepted aetiological mechanism of DOMS suggests that the repeated stretching of muscles during exercise results in severe mechanical stress and breakdown of the ultrastructure of muscle fibers, proteins and connective tissue (Cochrane, 2004; Miller *et al.*, 2004; Takahashi *et al.*, 2006). This results in inflammation, cellular degradation and the perception of pain (Cochrane, 2004; Miller *et al.*, 2004; Takahashi *et al.*, 2006). DOMS may also be aggravated by direct physical impact, such as with contact sports (Reilly and Ekblom, 2005).

Interestingly, as the muscle soreness and discomfort associated with DOMS peak, the muscle tissue damage is almost completely healed (Miller *et al.*, 2004). Another report points out that there is a time delay between the onset of muscle soreness and the peak of muscle soreness (Takahashi *et al.*, 2006). A possible explanation for this is given by Zainuddin *et al.* (2005), which indicate that DOMS induced by eccentric exercise does not necessarily indicate muscle damage and that the level of DOMS does not reflect the extent of muscle damage. Conversely, other researchers do not agree with this assumption that DOMS do not indicate muscle damage, since biochemical markers i.e. creatine kinase, lactate dehydrogenase and glutamic oxaloacetic transaminase are released when DOMS occur (Cleather and Guthrie, 2006).

Furthermore, the magnitude of DOMS depends on the intensity and the duration of exercise, as well as the physiological conditioning of the individual (Cleary *et al.*, 2006). DOMS has a detrimental effect on athletic performance in that muscle endurance, strength and power are reduced (Cleary *et al.*, 2006).

Additionally, the time course for DOMS does not always correspond to other indicators of muscle damage, such as swelling, pain, reduced ROM or muscle function. This means that some interventions might aid the recovery of DOMS, while others alleviate the symptoms of DOMS and restore functional capacity of the involved muscles. Further research needs to be done to study the exact causes of DOMS.

### 1.1 Symptoms associated with DOMS

DOMS is associated with several signs and symptoms, such as a dull and diffuse pain, tenderness and swelling that are generally linked with the inflammatory response. Individuals also show reduced muscular strength, stiffness and diminished length-tension in muscle (Cleary *et al.*, 2006; Cleather and Guthrie, 2006). These signs and symptoms may last as long as 24 to 96 hours after exercise (Cleary *et al.*, 2006). Sport scientists must therefore use practical and realistic tests to assess these factors and these tests are usually functional or sport specific. They allow the sport scientists to establish if exercise-induced muscle soreness has occurred and whether the intervention has an effect on recovery.

Muscle soreness is related to the muscle tissue damage, which is characterized by decreased range of motion, pain with stretch and palpitation of exercised muscle, and swelling (Kraemer *et al.*, 2001<sup>a</sup>). Muscle soreness due to eccentric muscle actions has demonstrated a decline in the muscle's functional capacity and athletic performance (Gill *et al.* 2006). Therefore, diminished athletic performance is characterized by reduced muscle strength, limited flexibility and increases muscle stiffness (Stay *et al.*, 1998; Almekinders, 1999; Stupka *et al.*, 2000; Cochrane, 2004; Takahashi *et al.*, 2006). The inability to generate tension in muscle following eccentric protocols may be due to both pain (secondary to local accumulation of inflammation agents) and structural damage in muscle sarcomeres (Kraemer *et al.*, 2001<sup>a</sup>).

Eccentric exercises reduce athletes' jumping ability (power and strength) due to the associated muscle damage (Wilcock *et al.*, 2006). The decreased force-generation in the affected muscle could be assessed by the vertical jump test. The vertical jump test measures the stretch-shortening cycle of the quadriceps extensor muscles and muscle power. The stretch – shortening cycle describes the combination of eccentric and concentric muscle actions and

provides an indication of the ability to use the stored elastic energy in the muscle. Thus, the vertical jump test is used to assess muscular power or the impact of muscular fatigue.

As mentioned in the first paragraph, muscle damage also results in a muscular tenderness and soreness, which is associated with inflammation (Stay *et al.*, 1998; Cochrane, 2004; Cleary *et al.*, 2006; Cleather and Guthrie, 2006). Inflammation causes swelling in the exercised extremities of the individual. Oedema may reduce the athlete's range of motion, cause pain and muscular function may also be hindered (Kraemer *et al.* 2001<sup>b</sup>; Miller *et al.*, 2004). Hence, subjective questionnaires such as the visual analog scale (VAS) are generally used to assess perceived muscle soreness, stiffness, functionality, and discomfort (Cleary *et al.*, 2006; Cleather and Guthrie, 2006). Measurements of limb circumferences are typically used to determine if swelling has taken place in the athlete's extremities (Kraemer *et al.*, 2001<sup>b</sup>; Zainuddin *et al.*, 2005).

## **2. Eccentric muscle action**

The time (duration per velocity), displacement (changing muscle fiber length), and the ability to produce force, determine the type of muscle action i.e. isometric (static), concentric (shortening) and eccentric (lengthening) action. The lengthening actions, such as eccentric and/or plyometric, result in the most muscle damage (Plowman and Smith, 2003; Close *et al.*, 2005).

Eccentric action produces powerful dynamic forces, at a low metabolic cost, due to the tension that develops while the muscle fibers' lengthen (Ball and Herrington, 1998; Noakes, 2001). Isometric action demand the highest energy cost (Close *et al.*, 2005). Repeated eccentric lengthening produces structural damage and mechanical disruption in the runner's muscle filaments (z discs) as a result of the cross-bridges that are cycling (Ball and Herrington, 1998; Noakes, 2001; Kraemer *et al.*, 2004; Takahashi *et al.*, 2006). Fewer motor units are recruited than are required for the mechanical demands during the lengthening action of eccentric muscle action. Consequently the muscle under-performs, leading to an increased tensile load per motor unit (Kraemer *et al.*, 2004). As a result the unequalled strain on the muscle fiber's cross-bridges causes mechanical disruption of the myofibrils. This modifies the contractile



mechanism and leads to muscle damage (Kraemer *et al.*, 2004; Miller *et al.*, 2004). Therefore, it is the muscle's ability to produce tensile force that damages the muscle.

As a result the muscle's Z-band malfunctions and disrupts the sarcomere's integrity (Ball and Herrington, 1998; Kraemer *et al.*, 2004; Miller *et al.*, 2004). The multiphasic muscle injury mechanism is related to the initial damage to the myofiber structure, the sarcoplasmic reticulum and the sarcolemma (myofibril). Within 15 minutes of eccentric actions disruptions to the cytoskeleton occurs (Stupka *et al.*, 2000; Kraemer *et al.*, 2004).

Damage to the sarcolemma causes localized haemorrhage and a flood of calcium ( $\text{Ca}^{2+}$ ) into the cell, thereby compromising  $\text{Ca}^{2+}$  homeostasis (Stupka *et al.*, 2000; Kraemer *et al.*, 2004; Miller *et al.*, 2004). The calcium accumulates in the mitochondria (Stupka *et al.*, 2000; Kraemer *et al.*, 2004; Miller *et al.*, 2004). As the calcium concentration increases it inhibits cellular respiration as well as activates proteases and phospholipases and a further cellular organelle damage occur (Stupka *et al.*, 2000; Miller *et al.*, 2004). Subsequent to muscular damage, the proteolytic enzymes clear the injured cell of internal debris, along with the movement (via diffusion) of intracellular components into the interstitium and plasma (oedema) (Stupka *et al.*, 2000; Miller *et al.*, 2004). This signals monocytes macrophages to clear the area via phagocytosis (Miller *et al.* 2004).

In addition, type II fibers are mostly damaged after muscle lengthening (eccentric) exercise and this is associated with an extensive reduction in muscle glycogen levels and GLUT 4 transporters of the athlete (Byrne and Eston, 2002; Burke, 2006; Chen *et al.*, 2007). Also, a reduction in the high-energy containing phosphate occurs at about 24 hours after muscle lengthening exercise. This influences the muscle's ability to generate force (Byrne and Eston, 2002).

## 2.1 Downhill running

Some of the methods to induce eccentric muscle actions include various resistance exercises, with isolated eccentric muscle actions. However, many of these eccentric exercises are not sport specific. Another method to induce DOMS is through downhill running (DHR)

protocols. Greater blood creatine kinase levels as well as myosin heavy chain activity are observed after downhill running compared to isolated muscle actions (Sanchez *et al.*, 2006).

Running involves lengthening of the muscle under tension (eccentric muscle actions) in the lower extremities of a runner. In addition, the runners' muscles persistently have to work against gravitational forces and this leads to muscle damage (Kraemer *et al.*, 2004; Takahashi *et al.*, 2006; Chen *et al.*, 2007). Throughout downhill running eccentric actions in the *Quadriceps* muscles act like a brake to slow down movement (Ball and Herrington, 1998; Noakes, 2001; Sanchez *et al.*, 2006). Also, glycogen resynthesis is impaired after DHR although a 30 minutes DHR protocol will not deplete glycogen stores (Byrne and Eston, 2002). Consequently, DHR protocols simulate the eccentric muscle actions experienced by runners, although the muscle damage caused by DHR protocols may differ from the painful and abrupt acute injuries, like strains and sprains, seen in races (Kraemer *et al.*, 2004; Miller *et al.*, 2004; Takahashi *et al.*, 2006).

Numerous authors indicate that downhill running ensue heightened muscular soreness and discomfort, owing to the more powerful lengthening forces of eccentric muscle actions (Ball and Herrington, 1998; Noakes, 2001; Byrne and Eston, 2002; Miller *et al.*, 2004; Ivy, 2004; Takahashi *et al.*, 2006; Chen *et al.*, 2007). The extent of muscle damage from strenuous exercise is related to the maximum force produced by the eccentric muscle action (Kraemer *et al.*, 2004). This means that the more forceful the eccentric actions the worse the associated muscle damage.

The reason why the runner might experience more pain in the *Quadriceps* than in the *Hamstrings* muscles after downhill running protocols (DHR) is explained by the muscle activation pattern during the downhill run (Takahashi *et al.*, 2006). A higher amount of stress (force) is placed on the anterior aspect of the thigh (*Quadriceps* muscles) during downhill than on level or uphill running, given that this muscle group must slow down the athlete's descending body mass, which is transferred onto the swing leg, at the end of the gait cycle (Sanchez *et al.*, 2006; Takahashi *et al.*, 2006; Chen *et al.*, 2007).

Therefore, downhill running (DHR) produces more muscle damage, especially in the knee extensors, than during level or uphill running. As explained before, this is owing to the

repeated vigorous eccentric muscle lengthening, which absorbs the more forceful impact forces (Sanchez *et al.*, 2006; Takahashi *et al.*, 2006; Chen *et al.*, 2007). In addition, the changes in the angle of the runner's knee are more apparent during DHR at a decline of 10% (35.1°) than during level running (Chen *et al.*, 2007).

Muscle soreness starts immediately after DHR and steadily increases over the next 48 hours (Miller *et al.*, 2004). Furthermore, additional eccentric exercise (i.e. a light jog) after high intensity exercise which causes damage may aggravate existing muscle damage (Takahashi *et al.*, 2006). The muscle damage that result from this eccentric muscle actions of DHR is associated with impaired muscle structure, metabolism, and function (Byrne and Eston, 2002; Braun and Dutto, 2003). As a result of this muscle damage the athlete's running style and muscle function becomes compromised, which may diminish running economy for up to 72 hours after the run (Byrne and Eston, 2002; Braun and Dutto, 2003; Miller *et al.*, 2004; Chen *et al.*, 2007). Muscle function is generally reduced immediately after eccentric exercise and could last up to a few hours to one week or one month (Byrne and Eston, 2002). It has therefore been shown that not only is DHR an effective method of inducing DOMS, it is also a realistic approach to test runners since it simulates the demanding influence of prolonged running (Chen *et al.*, 2007).

Braun and Dutto (2003) found after inducing DOMS with a downhill run protocol, in nine well trained male distance runners and triathletes, that muscle damage altered the athletes' stride biomechanics. Furthermore, subjects relied more on their anaerobic energy system, which meant an increase in metabolic stress. Thus the athletes' running economies were compromised due to DOMS after running downhill. Eston *et al.* (2000) also found small changes in stride mechanics after a downhill run following an initial downhill run, which was ran five weeks prior. Eston *et al.* (2000) showed that plasma creatine kinase was significantly lower ( $P < 0.05$ ) during the second downhill run, which would suggest a repeated bout effect.

### **3. Creatine kinase kinetics**

Exercise-induced muscle damage causes cellular disruption and the loss of muscle membrane structure. This brings about a temporary increase in cytoskeletal and myofibrillar proteins (for example myosin heavy chain and actin), along with an increase in inflammatory mediators in

the circulation (Kraemer *et al.*, 2004). Increased enzyme activity in the circulation serves as indicators of injury and/or muscle damage and are often included in testing batteries assessing exercise-induced muscle soreness (Stupka *et al.*, 2000; Byrne and Eston, 2002; Väänänen, 2004; Kraemer *et al.*, 2004; Cleather and Guthrie, 2006; Takahashi *et al.*, 2006; Gill *et al.*, 2006). Some of the biochemical markers that might be used to assess DOMS are lactate dehydrogenase (LDH), myoglobin, myosin heavy chain fragments, glutamic oxaloacetic transaminase activity and creatine kinase (CK) (Kraemer *et al.*, 2004; Cleather and Guthrie, 2006; Takahashi *et al.*, 2006).

Plasma creatine kinase (CK<sub>p</sub>) is the most frequently used marker of muscle damage (Kraemer *et al.*, 2004; Cleather and Guthrie, 2006; Sanchez *et al.*, 2006; Takahashi *et al.*, 2006). The enzyme creatine kinase is the catalyst involved in the metabolism of adenosine triphosphate (ATP) and creatine phosphate (CP). The enzyme aids in maintaining ATP homeostasis. For instance, creatine phosphate is a high-energy phosphorylated compound, and is found in greater concentrations (five to six times) than ATP in the resting muscles. When ATP is hydrolyzed to ADP in the muscle, CP rephosphorylates the adenosine diphosphate (ADP) with another inorganic phosphate (P<sub>i</sub>). This reaction releases immediate energy in power activities that last less than 10 to 15 seconds. The creatine kinase enzyme has a very low K<sub>M</sub> (Michaelis-Menten constant) and a very high H<sub>max</sub> (maximum velocity) characteristic. Both of these are kinetic factors of the enzyme, which describes the rate at which the enzyme functions. This means that there is a low interaction between the substrate and the enzyme creatine kinase, with a fast functioning rate. In other words, creatine kinase is not sensitive to the physiological changes in ATP. When there is a high concentration of ATP, creatine kinase activity will be inhibited (Brooks *et al.*, 2005).

When microtrauma is inflicted, the enzyme creatine kinase, along with other substances, seeps through the sarcolemma into the circulation. Creatine kinase starts to accumulate in the blood and is visible within one to twelve hours after muscle damage has occurred. The plasma CK<sub>p</sub> reaches a peak between 24 to 72 hours after and then returns to baseline values (Byrne and Eston, 2002; Väänänen, 2004; Reilly and Ekblom, 2005; Sanchez *et al.*, 2006). Plasma creatine kinase is the most sensitive muscle damage marker, as it shows the best relationship with perceived muscle soreness scores (Kraemer *et al.*, 2004; Reilly and Ekblom, 2005; Cleather and Guthrie, 2006; Sanchez *et al.*, 2006; Takahashi *et al.*, 2006). In other words, the

highest subjective muscle soreness scores on a rating scale are related to a high amount of plasma creatine kinase level for each individual (Reilly and Ekblom, 2005).

The normal reference values for creatine kinase are between 38 – 174 IU.L<sup>-1</sup> (Chen *et al.*, 2007). Creatine kinase (CK<sub>p</sub>) levels have been found to be elevated by 540% four hours after a marathon race. Evidence suggests that enhanced blood flow and distribution, along with reduced stiffness may possibly increase the CK<sub>p</sub> clearance rate after exercise (Gill *et al.*, 2006). Consuming fluids may also contribute to the removal of excess CK<sub>p</sub> (Sanchez *et al.*, 2006). Furthermore, creatine kinase levels can be reduced when athletes taper seven days prior to a half marathon event, by reducing training volume to 15% of the normal training volume (Myburgh, 2003).

#### **4. Conclusion**

To investigate exercise induced muscle soreness and to assess the effectiveness of various recovery strategies, sport scientists induce delayed onset of muscle soreness (DOMS) via eccentric protocols. However, most of these protocols are not sport-specific. Downhill running, however, is an effective means to investigate exercise-induced muscle soreness and its associated symptoms. This means that studies generally include tests for muscular strength, endurance, and power as a measure of functional performance. In addition, swelling, range of motion, perceived muscle soreness, pain, and discomfort together with markers of inflammation, i.e. plasma creatine kinase are also assessed. Plasma creatine kinase activity is the most frequently used and sensitive marker of muscle damage.

#### **C. POST-EXERCISE RECOVERY**

Most athletes nowadays, especially elite athletes, have very strenuous training programs. These programs generally involve one or more prolonged and/or high intensity bouts per day. This typically allows about six to 24 hours for recovery between the workouts (Burke, 2006). In addition to these demanding programs, some sports have competitions that are conducted in series of events or in stages. For instance, sports like swimming as well as track and field events frequently include a number of short races or heats, which may also happen on the same day.

Prolonged endurance events have become increasingly popular, which may involve seven to 21 days of exercise, for instance in cycling, the road races Tour de France and the Giro d'Italia and the Cape Epic mountain bike race or in running, the Iron man or Comrades (~ 89.9 km). Other examples are training camps that may last from seven to 14 days. Then there are weekly matches, events, or races in which athletes participate.

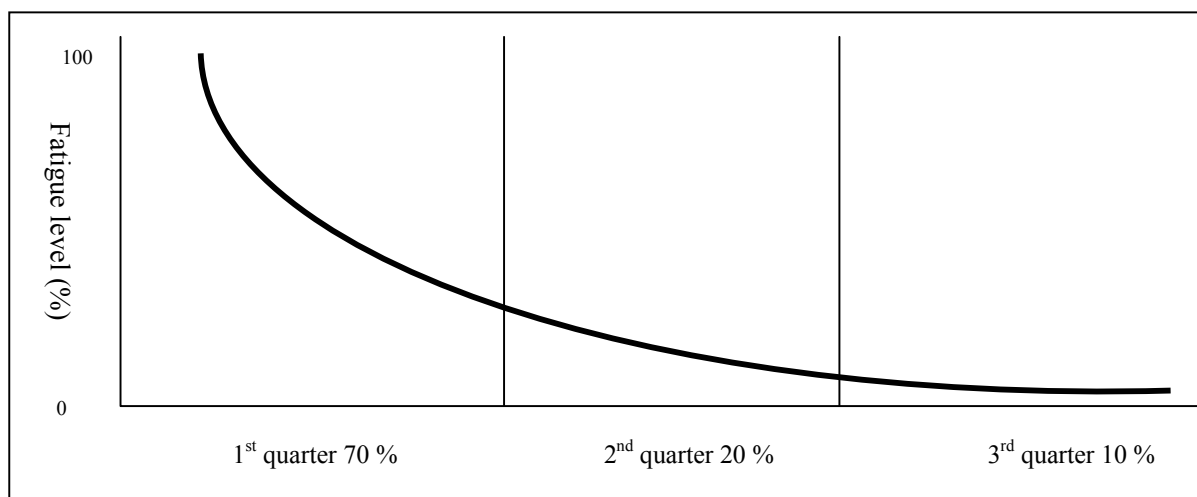
The athlete's musculoskeletal, nerve, immune, and metabolic systems are stressed by these consecutive training bouts or events (Reilly and Ekblom, 2005). Therefore, athletes need optimal recovery to adapt, prevent injuries and to allow the athlete to train in-between races or matches. The main objective of post-exercise recovery is to restore athletic ability to normal levels as soon as possible (Cochrane, 2004; Reilly and Ekblom, 2005; Burke, 2006; Shave and Franco, 2006). Ineffective recovery is harmful to the athlete and will lead to further reduced athletic performance, fatigue, and/or injury or in the worst cases, to overtraining or burn out (Reilly and Ekblom, 2005; Burke, 2006; Lambert and Borresen, 2006).

### **1. Post-exercise recovery rate**

The rate of post-exercise recovery after prolonged exercise differs between the various physiological systems that are exhausted or injured (Miller *et al.*, 2004). Furthermore, inter-individual differences exist in recovery rates amongst runners when they dramatically increase their training volume and intensity (Myburgh, 2003). This means, depending on the affected functions or areas, some athletes might need a longer recovery time than others. Other factors that may play a role in recovery rates are intrinsic and extrinsic factors such as physical conditioning, experience, age, sex, external conditions, nutrition and time between events, certain hormones, type as well as the level of exertion of the exercise (Bompa, 1999; Burke, 2006; Shave and Franco, 2006). More experienced athletes will also recover faster than inexperienced athletes. Furthermore, endurance trained athletes have a better adapted physiological system as explained in chapter two (*pages 12 to 31*), which means that they will remove blood lactate faster and transfer energy more efficiently than untrained individuals (Bompa, 1999).

The multidimensional process of recovery has a series of consecutive responses, which takes place in a curvilinear pattern (see *Figure 2.*). Within the initial third time period (~30 minutes to 6 hours) the recovery response is quick and 70% of the biological factors are restored. In the next 6 to 24 hours, the process slows down and the athlete only recovers by 20% in the second period.

Then finally about 10% of the affected factors take 24 hours or more to recover. The recovery process from the first to the third phase might take several minutes to several months. The recovery rate depends on the energy system(s) that were utilized or exhausted and if the athlete is recovering from short-term fatigue and exhaustion or long term overtraining (Bompa, 1999; Koutedakis *et al.*, 2006).



*Figure 2.* The recovery curve lasting from a few hours to several days or even months if the athlete is overtrained or depending on energy system used (adapted from Bompa, 1999).

Within the initial seconds post-exercise, the electrical properties of nerves and muscle speedily return to resting values (Sesboüé and Guincestre, 2006). The heart rate and blood pressure generally return to resting values within 20 to 60 minutes post-exercise (Bompa, 1999; Koutedakis *et al.*, 2006). Phosphagen stores take three to five minutes to fully recover and lactate, together with pH, will take about 60 minutes or more (Cochrane, 2004).

However, the sluggish aerobic energy system and mechanical properties of the damaged muscle, take longer (Koutedakis *et al.*, 2006; Sesboüé and Guincestre, 2006). O'Reilly *et al.*

(1987) stated that muscle glycogen, as well as the ultrastructure of the muscle may take up to 10 days to recover after eccentric exercises. Generally with activities, such as prolonged and exhausting endurance running, the glycogen stores may return to resting values within ten to 48 hours, at the earliest. The intensity and the duration of the aerobic activity have an inversely proportional relationship with the recovery rate. In other words, the more intense and the longer the training or the race, the slower the recovery process to return the used glycogen to pre-exercising levels. With anaerobic interval activities only five to 24 hours are needed to recover. Protein regeneration takes twelve to 24 hours, while more than 24 hours is needed for fat, vitamin and enzyme restoration (Bompa, 1999).

Activities involving eccentric muscle actions, such as resistance training, generally require 24 to 36 hours to recover from muscle damage and injury. Muscle damage has a severe impact on runners, since performance is influenced until the muscle damage and soreness are fully recovered (Miller *et al.*, 2004). Several authors suggest that muscular recovery is a multiphase response (Miller *et al.*, 2004; Burke, 2006; Tarnopolsky, 2006; Koutedakis *et al.*, 2006; Sesboüé and Guincestre, 2006). This two-fold process is accomplished when (i) pain is reduced and the athlete's feeling of well-being is restored, and (ii) the muscle structure is repaired (Miller *et al.*, 2004). The reduction in muscle function could be evident for as long as seven days post-events (Ball and Herrington, 1998).

It has been said that swelling and microtrauma to tissue are acute responses and with sufficient recovery, it may resolve from one training bout to the next (Ball and Herrington, 1998). After strenuous training or events, however, the athlete will need a longer recovery period, or this will lead to overuse and overtraining (Ball and Herrington, 1998). Then finally the nervous system might take up to 48 hours to recover, depending on the intensity of the exercise (Koutedakis *et al.*, 2006).

When recovery techniques are usually applied within the initial nine hours post-exercise, supercompensation and adaptation of the athlete's functional capacity is improved (Bompa, 1999). Successful post-exercise recovery strategies usually entails either (i) the restoration of muscle and liver glycogen stores, (ii) the replacement of fluid and electrolytes lost in sweat and (iii) the regeneration and repair following the catabolic stress and damage caused by the exercise, or a combination of these factors (Tarnopolsky, 2006).



Post-exercise recovery may be acute (hours), medium (about two weeks) or long term (months to years). The objective of post-exercise recovery is to restore the athlete's homeostasis. This occurs when the runner's body adapts to the stress inflicted by the earlier exercise bout (Myburgh, 2003; Väänänen, 2004). Hereafter, supercompensation follows, thus the athlete not only adapts but also improves (Myburgh, 2003; Väänänen, 2004). The recovery strategy that will be used depends on the type of activity. For instance, endurance training mostly exhausts the nutritional factors (i.e. rehydration and fuel stores) of the athlete, then the physiological (muscle cell) and neurological (peripheral nervous system) factors and finally the psychological factors (central nervous system) (Cochrane, 2004).

Optimal recovery involves replenishing muscle glycogen and fat reserves, with or without protein supplementation along with repairing the muscular damage sustained during prolonged running events (Myburgh, 2003; Ivy 2004; Atwood and Bowen, 2007), which leads to adaptation (Ivy, 2004). Optimal recovery allows an athlete to run longer and for more intense periods, which improves endurance performance (Burke, 2006; Atwood and Bowen, 2007). Optimal training, on the other hand, is coupled to recovery and will lead to the greatest adaptation and thus enhance the athlete's running performance.

## **2. Post-exercise musculoskeletal recovery**

Athletics is associated with eccentric and repetitive prolonged muscle actions that result in muscle injury and damage. Predominantly this is due to the abrupt and vigorous contractions, mechanical overload, and severe structural disruption of the muscle fibers. These repetitive impact forces might be two to three times the runner's body mass (Sanchez *et al.*, 2006).

Muscular soreness due to the lengthening muscle action is associated with ultrastructural damage and an increase in muscle enzyme activity in the blood, such as creatine kinase. Athletes also experiences reduced muscular strength, endurance and power as well as flexibility (Miller *et al.*, 2004; Close *et al.*, 2005; Cleary *et al.*, 2006; Takahashi *et al.*, 2006). All of these actions are associated with the reduction in the muscle's ability to generate tension and therefore interferes with the athletic performance of the athlete (Kraemer *et al.*, 2001; Kraemer *et al.*, 2004; Miller *et al.*, 2004; Takahashi *et al.*, 2006).

Sanchez *et al.* (2006) reviewed some of the medical problems associated with marathon running. Seventeen percent of the injuries observed during a specific marathon event (Twin Cities marathon), over a 12-year period, were as a result of musculoskeletal problems. Typically, the problems included cramps, blisters, and serious ankle and knee injuries. In addition, it was found that knee injuries are more common in road racing and ankle injuries are found in track races. There is currently no evidence linking regular running with chronic musculoskeletal disability. Furthermore, the dehydrated marathon runners rarely demonstrated swelling, tenderness, and weakness (Sanchez *et al.*, 2006).

Immediately after eccentric muscle action, microtrauma occurs in the athlete's muscle fiber. This happens because the lengthening muscle action tears the smaller vulnerable areas, which leads to necrosis of the muscle fiber. Three to four days after the trauma immune cells infiltrate the portion of tissue that has died. These tears are manifested as disrupted sarcomeres in the myofibrils, Z-line streaming, over extended or half sarcomeres, damage to the t-tubule and local disorganization of myofilaments (Close *et al.*, 2005).

## 2.1 Inflammatory response

Musculoskeletal injury is accompanied by the well-investigated inflammatory response (Miller *et al.*, 2004; Kraemer *et al.*, 2004; Stupka *et al.*, 2000). Inflammation increases over the following 24 to 48 hours after eccentric exercise has ceased (Kraemer *et al.*, 2004). Within the next three to five days the inflammation response levels off, before returning to normal at day seven (Kraemer *et al.*, 2004). A study by Real *et al.* (2005) indicated that within four hours after a race, middle-aged male marathon runners demonstrated an increase in the inflammatory response and prothrombotic markers.

Even though inflammation is the first step in the healing process (Kraemer *et al.*, 2004) and an essential part of the recovery process, uncontrolled inflammation according to Miller *et al.* (2004) may delay healing from skeletal muscle injury. Kraemer *et al.* (2004) pointed out that the inflammatory mechanism is triggered by mechanical as well as chemical injury. The mechanical mechanism is the physical disruption in the muscle fiber, whereas the chemical mechanism involves the mediators of inflammation.

Signs and symptoms of inflammation involve localized redness, heat, pain and loss or reduction in functional capacity (Prentice, 2001; Kraemer *et al.*, 2004; Zainuddin *et al.*, 2005). Furthermore, upon palpitation, contracting and/ or stretching of the damaged muscle, pain is apparent (Ball and Herrington, 1998). This is similar to DOMS induced by eccentric exercise, with the only exception that DOMS does not reveal signs of redness and heat (Zainuddin *et al.*, 2005). The degree of inflammation in the injured area depends on the extent of the muscle damage (Kraemer *et al.*, 2004).

Stupka *et al.* (2000) studied the differences in the inflammatory response between healthy university male and female students after performing unilateral eccentric exercises. Previous human and animal studies indicated that there is a difference in the muscle inflammatory response between male and females. Females tend to have an attenuated response to muscle damage compared to males. Possible reasons for the lower CK<sub>p</sub> in females were attributed to the female hormone 17 $\beta$ -estradiol and its anti-oxidant properties and some speculate that women do not work as hard as men, and therefore have a lower CK<sub>p</sub> concentration (Stupka *et al.*, 2000).

Stupka *et al.* (2000) showed that even though there was a difference in the CK<sub>p</sub> response between the untrained men and women, it was not statistically significant. Furthermore, men had a significantly greater total number of bcl-2-positive inflammatory cells.mm<sup>-2</sup> of tissue, than the women. The LCA bcl-2-positive cells.mm<sup>-2</sup> demonstrates that females had an overall reduced cellular infiltrate compared to the men. Stupka *et al.* (2000) came to the conclusion that there is no difference in muscle damage between the two genders, in spite of the weakened inflammation response in females.

## 2.2 Peripheral oedema

The role of the lymphatic system is to collect and remove fluid and proteins from the interstitial tissues, which is then returned to the venous system (Cho and Atwood, 2002; Partsch, 2003). When the athlete's muscle is damaged, chemical mediators, e.g. creatine kinase and myoglobin are released into the circulation (Kraemer *et al.*, 2004) and an inflammation response is produced. The capillary walls become more permeable and proteins

are released into the intercellular fluid (Kraemer *et al.*, 2001<sup>b</sup>; Kraemer *et al.*, 2004). This alters the gradient between the two compartments by increasing the tissue oncotic pressure and elevating the hydrostatic pressure in the veins (Kraemer *et al.*, 2001<sup>b</sup>; Cho and Atwood, 2002; Kraemer *et al.*, 2004). In an attempt to equalize the osmotic gradient across the capillary membrane, fluid is drawn out of the veins into the interstitial compartment (Kraemer *et al.*, 2001<sup>b</sup>; Cho and Atwood, 2002). This in return disturbs homeostasis and oedema will form (Kraemer *et al.*, 2001<sup>b</sup>; Cho and Atwood, 2002; Kraemer *et al.*, 2004; Miller *et al.*, 2004).

Thus, oedema (swelling) is the result of an imbalance between the plasma volume (intravascular) and interstitial compartment (extravascular) (Cho and Atwood, 2002). This imbalance leads to an uncharacteristic accumulation of fluid in the interstitial space (Saladin, 2001; Cho and Atwood, 2002). Oedema in the muscular compartment leads to increased intra-compartmental pressure, which in return inhibits lymphatic and venous drainage of the area, promoting further swelling. As the muscle compartment becomes more turgid, motion is reduced (Kraemer *et al.*, 2001<sup>b</sup>; Cho and Atwood, 2002). About one-hour post-exercise, the mechanical alteration that brings about a transfer of plasma volume and the extracellular compartment can be observed (Maton *et al.*, 2006). Swelling is intramuscular during the first 24 to 48 hours, however in the following 72 hours it becomes subcutaneous (Kraemer *et al.*, 2004).

Kraemer *et al.* (2004) also explained that the inflammatory mediators' bradykinin, serotonin, and histamine augment the responsiveness of the pain afferents in the injured areas and may contribute to the pain stimulus to some extent. Additionally swelling may also have part in producing pain, by increasing intramuscular pressure and stimulating the pain receptors (type IV sensory neurons) (Ball and Herrington, 1998; Miller *et al.*, 2004; Kraemer *et al.*, 2004).

Severe swelling often persists long after muscle soreness has subsided. Kraemer *et al.* (2004) stated that the pain receptors may adapt to the swelling response, or otherwise swelling might be a secondary response to the primary cytokine and histamine mediators (Kraemer *et al.*, 2004). Swelling hampers the function of muscle and cause pain (Miller *et al.*, 2004). Therefore diminished swelling and the decrease in stimulation of the pain receptors are used by sport scientists as recovery indicators (Miller *et al.*, 2004).

An increase in blood flow to these areas will help to reestablish the equilibrium between the two compartments, by lowering the hydrostatic pressure and aiding the lymphatic system. In other words, the fluid and proteins are then collected via the lymphatic system and returned to the venous system (Cho and Atwood, 2002; Partsch, 2003). This will result in a reduced oedema and inflammatory response and allow the athlete to start to heal (Cochrane, 2004; Dawson *et al.*, 2005; Gill *et al.*, 2006).

### **3. Post-exercise metabolic recovery**

All the energy systems are stressed during strenuous events or training bouts. This means that liver and muscle glycogen, as well as muscle triglycerides are either depleted or reduced post-exercise. Below average glucose levels are related to cognitive impairments. This could make the athlete feel drained or mentally exhausted (Reilly and Ekblom, 2005). When glycogen levels are not restored by the following event or training session, athletic performance will be negatively affected (Ivy, 2004; Reilly and Ekblom, 2005).

During the post-exercise recovery phase, the athlete's oxygen consumption (aerobic metabolism) is elevated. The oxygen consumption returns to pre-exercise levels via a curvilinear response. This is known as excess post-exercise oxygen consumption (EPOC) (Cochrane, 2004; Brooks *et al.*, 2005; Laforgia *et al.*, 2006). The purpose of this elevated metabolism is to restore the fuel stores, after they are depleted or lowered by exercise.

Excess post-exercise oxygen consumption is a two part recovery-mechanism, by first restoring immediately, within 30 seconds, 70% of the energy stores (ATP and PCr) and supplying oxygen to haemoglobin and muscle myoglobin and then restoring the energy stores after prolonged exercise by the slow component that could take up to 24 hours to recover. This sluggish recovery rate is associated with an increase in the functions of the cardiorespiratory system, increased body core temperature and aiding in metabolic clearance, such as lactate and phosphate (Cochrane, 2004; Laforgia *et al.*, 2006).

### 3.1 Glycogen restoration

Endurance training alters the relationship between the ventilatory respiratory exchange ratio (R) and the relative exercise intensity in well-nourished individuals, by shifting it to the right. This means that fewer carbohydrates are used as a source of fuel by trained individuals as by untrained, at the same exercise intensity. However, glycogen remains the main source of fuel for muscle exercise, especially during demanding aerobic training bouts and/or prolonged races (Ivy *et al.*, 2002; Ivy, 2004; Brooks *et al.*, 2005; Burke, 2006).

When soccer players, trained hard the day before a game, they experienced the onset of fatigue earlier, than their teammates who rested (Reilly and Ekblom, 2005). In other words, the amount of glycogen present before the onset of exercise will determine the athlete's endurance time (Ivy *et al.*, 2002; Reilly and Ekblom, 2005; Brooks *et al.*, 2005). Therefore, an athlete needs to have recovered glycogen stores prior to the following event or training session for optimal performance (Ivy *et al.*, 2002; Burke, 2006). The athlete will need 20 to 24 hours at an average rate of five to six mmol/kg wet weight (ww) per hour, to restore his glycogen stores (Burke, 2006).

Ivy (2004) substantiates this by stating that aerobic endurance is linked to the initial muscle glycogen stores and that demanding exercise cannot be sustained once these stores are depleted. Therefore, when glycogen stores are returned to pre-exercise levels an athlete can train at the same intensity and/or volume the following day. Furthermore, glycogen restoration improves the anabolic: catabolic ratio of metabolic processes in endurance athletes (Myburgh, 2003). An athlete will demonstrate underperformance when homeostasis is not returned and catabolism overpower the anabolic processes (Reilly and Ekblom, 2005). Thus, athletic performance depends on the athlete's ability to restore glycogen (Burke, 2006).

Muscle glycogen stores are favoured and thus replaced before the liver's stores. The exhaustion of muscle glycogen stores, stimulate the resynthesis of glycogen by activating glycogen synthase. For example, after high-intensity exercises, where the athlete's lactate concentration is elevated and no additional carbohydrate supplementation is taken, glycogen is quickly restored. However, after moderate-intensity exercise athletes need to consume additional carbohydrates post-exercise to aid glycogen restoration (Burke, 2006).

Muscle glycogen synthesis is a sluggish process of at least 1 – 2 mmol/kg wet weight (ww) of muscle per hour without additional carbohydrates. A maximum of 5 – 10 mmol/kg wet weight per hour during the initial twelve hours post-exercise have also been reported (Ivy, 2004, Burke, 2006). The rate of resynthesis is influenced by the factors controlling glucose uptake into the cell and/or those involved with glucose removal (Burke, 2006).

This explains why the optimal time for glycogen replacement is within two hours of cessation of exercise. As the enzymes associated with glycogen synthesis are most active during this period. Moreover, during this initial post-exercise period, glucose sensitivity is increased and greater GLUT 4 transporters are available in the muscle (Reilly and Ekblom, 2005). Nutritional guidelines suggest a carbohydrate intake of 1.5 g. kg<sup>-1</sup> body mass within the first 30-minutes of recovery.

This type of recovery strategy is more applicable to events or exercise sessions that are only separated by four to eight hours. For other activities that are separated by more than twelve hours, this strategy has less of an impact. For longer periods it is more important to take in the required carbohydrates, until glucose threshold is reached (Burke, 2006). The amount of carbohydrates consumed affects the storage of glycogen in a direct and positive way post-exercise, up until the muscle's storage threshold of about seven to ten gram per kilogram of the athlete's body mass is reached (Burke, 2006).

The type of carbohydrates consumed after exercise, warrants further investigation, however to date, it is recommended to consume moderate to high GI (glycemic index) foods (Reilly and Ekblom, 2005; Burke, 2006). The higher GI food increases glycogen storage by about 30% during the 24 hours post-exercise. The reasons are not clear, but it has been purposed that the higher GI foods enhance glucose and insulin responses or that low GI foods are not properly digested. In addition, the form of carbohydrate loading, i.e. liquid or solid or the frequency of meals or regular snacks does not seem to have an influence on glycogen storage (Burke, 2006).

In contrast to concentric exercise or rest, eccentric muscle actions alters the previously active muscle's metabolic properties (Byrne and Eston, 2002; Ball and Herrington, 1998) by giving

rise to more insulin resistant muscles and reduce glucose clearance by 37 % (Ball and Herrington, 1998) and an extended reduction in muscle glycogen levels (Byrne and Eston, 2002). It is thought that the inability of the metabolism to complete its function could result in decreased force production, especially during high intensity activities, which utilizes the anaerobic energy system (Byrne and Eston, 2002). Prolonged runs, on the other hand, place immense stress on the athlete's physiological systems, which may harm muscle glycogen restoration post-exercise. Glycogen storage is impaired by damage to the injured fiber, such as that caused by eccentric exercise or direct contact injury. Increased glycogen consumption within the 24 hours post-exercise, might be partially overcome this reduced glycogen restoration (Burke, 2006). In addition, a decrease in glycogen replenishment has been associated with the greater tendency for injury in runners (Ball and Herrington, 1998).

### 3.2 Dietary fat and athletic recovery

The role of fatty acid and recovery post-exercise is equivocal. Some studies report no effect on DOMS and CK<sub>p</sub>, while other studies have indicated that it aids post-exercise CK levels. A chronic high fat intake may support the endurance athlete's immune system (Lowery, 2004).

### 3.3 Protein supplementation

Additional protein intake is not necessary for recreational athletes or for exercise at low to moderate intensities ( $\leq 50\%$   $VO_{2max}$ ). These athletes consume enough total energy during their training or do not reduce their protein content enough, to justify additional protein intake. However, it has been suggested that for exercise intensities above 65 to 85% of  $VO_{2max}$ , and elite endurance athletes that train at very high intensities for long durations might benefit from protein supplementation (Tarnopolsky, 2006).

Additional dietary protein is usually only necessary at the onset of an endurance program and to aid the recovery of muscle degradation in well-trained athletes (Reilly and Ekblom, 2005, Tarnopolsky, 2006). Well-trained athletes are those athletes that train at intensities of 60 % of their  $VO_{2max}$ , four or five times a week for 45 minutes. These athletes need to consume 20 to 25% more dietary protein, while elite athletes may increase their protein intake by 1.6 grams per kilogram ( $g \cdot kg^{-1}$ ) per day (Tarnopolsky, 2006).



Muscle damage induced by exercise is associated with unaccustomed or strenuous exercise. Therefore, muscle degradation is also coupled with metabolic recovery, since post-exercise muscle break down continues due to protein utilization, such as amino acids (Myburgh, 2003; Ivy, 2004; Tarnopolsky, 2006), inflammation, delayed hormonal regulation and augmented free radicals (Ivy, 2004). This means that the hormonal changes which aids in protein, fat and glycogen break down for fuel, may cause muscle damage (Ivy, 2004).

It has been recommended to consume extra proteins in conjunction with carbohydrate intake within four hours post-exercise. This combination would assist muscle repair, replenish glycogen stores, and result in adaptation in athletes (Myburgh, 2003; Ivy, 2004; Reilly and Ekblom, 2005; Burke, 2006; Tarnopolsky, 2006). Greater protein synthesis is associated with protein supplementation with carbohydrates as soon as one to three hours immediately post-exercise (Reilly and Ekblom, 2005). Previous studies suggested that the additional protein consumption with the carbohydrates causes a greater insulin response. As a result, carbohydrates are absorbed at a greater rate and this leads to a faster restoration of glycogen stores. Others suggest that this combination provided a greater total energy intake compared to just consuming carbohydrates on its own (Ivy *et al.*, 2002).

### 3.4 Post-exercise lactate kinetics

Anaerobic breakdown of glycogen leads to intracellular accumulation of inorganic acids (lactic acid and inorganic phosphate) (Maton *et al.*, 2006). Generally during rest the muscle releases lactate, but to some extent also takes up a small concentration of lactate (Gladden, 2000<sup>b</sup>). Conversely, training and races result in an increased lactate production and hydrogen ions ( $[H^+]$ ) accumulation (Connolly *et al.*, 2003; Draper *et al.*, 2006). The accumulated  $[H^+]$  will make the blood pH more acidic. When the body's pH drops below the normal 7.4, it is known as metabolic acidosis (Martin *et al.*, 1998). The amount of accumulated lactate and  $[H^+]$  will depend on the duration, intensity and the recovery time.

The accumulated lactate, acidosis and increased inorganic phosphate are believed to be responsible for muscular fatigue by interfering with the metabolic function and muscular contractility. This reduces the muscles ability to produce force, as well as retards the transport

and functioning of the metabolic pathways (Martin *et al.*, 1998; Cochrane, 2004; Maton *et al.*, 2006). For optimal performance it would make sense that lactate exchange and removal are critical (Connolly *et al.*, 2003; Draper *et al.*, 2006; Maton *et al.*, 2006). Whether lactate concentration *per se* is a contributor of fatigue is debatable (Connolly *et al.*, 2003; Draper *et al.*, 2006; Wilcock *et al.*, 2006). The exact mechanisms of muscle fatigue are complex and not fully understood. Accumulated lactate, and the associated hydrogen ions, is at least partially responsible for slowing down the recovery from fatigue (Hemmings, 2001).

According to Martin *et al.* (1998) several mechanisms have been proposed to explain how metabolic acidosis diminishes muscular ability. When the pH drops due to lactate and hydrogen ions accumulation it inhibits two of the important regulating enzymes of anaerobic energy production, i.e. lactate dehydrogenase and phosphofructokinase. This reduces the mobilization of free fatty acids and delays glycolysis. Furthermore, the increased hydrogen ions disrupt muscle action by dislodging calcium from troponin. It also stimulates the pain receptors and increases the athletes perceived level of exertion.

Following the cessation of strenuous exercise lactate still continuously filters into the circulation from the earlier working muscles, and then for several minutes increases exponentially to a maximum value where after it progressively returns to the baseline level, via the same exponential decline (Fukuba *et al.*, 1999). Therefore, post-exercise recovery strategies aim to speed up the removal of lactate and H<sup>+</sup> (Martin *et al.*, 1998; Cochrane, 2004; Rimaud *et al.*, 2007). Optimal lactate recovery immediately after strenuous exercise is very important, especially if there are more exercise bouts or matches to follow (Martin *et al.*, 1998).

Although a large amount of lactate is removed during the exercise, the majority of lactate removed during recovery is by direct oxidation or gluconeogenesis and some lactate is shunted to other sites, such as the heart, liver, kidneys and skin. Lactate clearance is further influenced by the internal metabolism. For instance, if there is normal liver glycogen and blood glucose levels, increased lactate will be oxidized. After prolonged activities, such as long distance running, glycogen is depleted or severely exploit (hypoglycemia), then converting lactate into glucose precursors is favored (Brooks *et al.*, 2005).

Enhanced blood flow is suggested to be an important factor in the removal of lactate post-exercise, as it enhances oxidation and diffusion out of the muscles (Hemmings, 2001; Brooks *et al.*, 2005). Active recovery strategies increase blood flow more compared to passive recovery, and is believed by some to be more beneficial for lactate metabolism and subsequent performance (Chatard *et al.*, 2004). Both Connolly *et al.* (2003) and Fukuba *et al.* (1999) maintained that lactate concentrations are reduced promptly by active recovery, more so than with passive recovery.

As mentioned before, several organs, including the skeletal muscles, actively and inactively, aid lactate clearance. The skeletal muscle transforms lactate back to pyruvate, which then re-enters the Krebs cycle. Thus, the suggested reasons why active recovery is more effective than passive recovery is that (i) a greater lactate concentration is oxidized by the moderately active muscles and (ii) a swift transfer of lactate to the site of removal, either to the liver, heart and/or inactive muscle, as a result of improved or maintained blood flow take place (Draper *et al.*, 2006; Connolly *et al.*, 2003; Fukuba *et al.*, 1999).

#### **4. Post-exercise rehydration**

The athlete's core temperature increases proportionally to the intensity of the exercise and the environmental temperature (Reilly and Ekblom, 2005). About 75 % of the energy runners generate, dissipate from the body as heat, primarily as sweat loss (Reilly and Ekblom, 2005; Sanchez *et al.*, 2006; Burke, 2006). About one liter of water from the skin equals 580 kcal of heat from the body (Burke, 2006). Individual sweat rates vary among athletes, but athletes might lose more than 1800 ml sweat per hour during a marathon (Sanchez *et al.*, 2006). Moreover, exercising at an intensity of about 75% of one's  $VO_{2max}$  could result in a rate of two liters per hour of sweat loss (Reilly and Ekblom, 2005). It is this loss in plasma volume that affects thermoregulation and athletic performance as well as places strain on the cardiovascular system (Carter *et al.*, 2005; Burke, 2006).

Endurance performance is impaired when an athlete is dehydrated by about one to two per cent of body mass (BM) (Reilly and Ekblom, 2005; Burke, 2006). Five percent dehydration could reduce the athletic ability by 30%. Some athletes have demonstrated a 45% impairment in their performance capacity during prolonged activities, with a 2.5% loss in body weight

(Burke, 2006). Hypohydration is not only detrimental to the athlete's running performance, it also debilitates thermoregulation, alters muscle metabolism, reduces gastric emptying, affects baroreceptor responsiveness, arterial blood pressure is not sustained, catecholamines levels are increased and also cognitive function is influenced in such a way that perceived exertion is increased (Carter *et al.*, 2005; Burke, 2006). The more dehydrated the athlete becomes, the more progressively the effects worsen (Burke, 2006).

Athletes do not generally ingest enough fluids during competitions and cannot keep up with the rate of fluid loss (Reilly and Ekblom, 2005). They usually only replace 30 to 70 % of their sweat loss during exercise and are limited between exercise bouts (six to eight hours) to replace loss fluids. Moderate to severe hypohydration (two to five percent BM), may take four to 24 hours to recover the body's fluid levels (Burke, 2006).

Post-exercise rehydration is therefore an important strategy to restore fluid losses between exercise bouts and events. The aim would be to restore the athlete's autonomic control, as well as to start the next exercise session or event euhydrated (Carter *et al.*, 2005; Burke, 2006). Some of the recommendations specify that for moderate fluid loss, athletes should consume 150% of fluid over the following hours post-exercise. In other words, the athlete needs to consume more volume of fluid than what he has lost during and post-exercise. Generally, to restore the fluid balance the athlete needs to rehydrate by 50 to 70% over two to four hours post-exercise (Burke, 2006).

About seven grams of sodium may be lost through sweating after exercising in hot environments (Burke, 2006). Sodium losses via sweat are typically between 20 to 80 mmol.L<sup>-1</sup>, but this varies between individuals (Burke, 2006). Sweat rates are dependent on factors such as body size, physiological differences, exercise intensity and duration, ambient temperature, humidity and acclimatization. This makes sodium the main electrolyte of concern for long-distance runners (Sanchez *et al.*, 2006; Burke, 2006).

Various recommendations are given about the correct amount of sodium given post-exercise. However, Burke (2006) suggests that athletes should preferably ingest 50 to 90 mmol.L<sup>-1</sup> of sodium. The sodium facilitates the absorption of water through the intestinal wall and therefore restores plasma volume more rapidly than just plain water. The reason being that

pure water dilutes the plasma osmolality and the sodium content in return, urination increases and thirst is reduced. This inhibits the success of the rehydration process. In other words, sodium aids optimal rehydration by maximizing the retention of consumed fluids. Sodium may be ingested in the form of fluids, such as sports drinks, food or salty snacks (Reilly and Ekblom, 2005; Burke, 2006).

## **5. Neural recovery**

During exercise, the parasympathetic activity is reduced and sympathetic activity is increased, as explained in chapter two (*pages 16 to 18*). During exercise the athlete's heart rate and myocardial contractility increases, the periphery vasodilate and the airways constrict and dilate *via* nervous stimulation. After exercise, with efficient recovery the heart recovers to resting levels.

However, when athletes train repeatedly and at high intensities or prolonged durations, the sympathetic nerve stimulation increases due to insufficient recovery or rest (Cochrane, 2004). In addition, after matches or events, the central nervous system might still be stimulated and this could lead to reduced sleep or disturbed sleeping patterns. Active recovery strategies may lessen the nervous systems response and thereby promote sleep (Reilly and Ekblom, 2005).

Athletes are endangered by the possibility of conditions such as overreaching and/or staleness (overtraining), if they do not give the nervous system time to recuperate (Bompa; 1999; Cochrane, 2004). The sympathetic nervous system is predominantly affected when an athlete experience overtraining. This in return affects the endocrine function and recovery may take months to restore the neural input (Ball and Herrington, 1998).

More flexible muscles post-exercise, improved reflexes and positive feelings and/or reduced perceived muscle soreness are associated with an improved neurological recovery, specifically in the CNS. Appropriate recovery techniques, such as contrast therapy and hydrotherapy, might assist the restoration of the peripheral and central nervous system (Cochrane, 2004).

## **6. Conclusion**

Therefore, strategies to aid recovery after training or events have become increasingly important for modern athletes. The intention is to restore muscle and liver glycogen; replenish fluid and electrolyte loss in sweat as well as aid regeneration, repair and adaptation to catabolic stress (Burke, 2006). Furthermore, recovery depends on the exercise intensity and duration. The more strenuous the activity and effort demanded from the athlete, the longer the recovery period.

### **D. RECOVERY STRATEGIES**

Recovery is defined as the return of the muscle to its pre-exercise condition after exercise (Cochrane, 2004). In other words, recovery is a method by which the athlete's body restores homeostasis. Recovery also allows the athlete to adapt its physiological functions and systems at a higher level (Shave and Franco, 2006). Optimal recovery permits an athlete to train for longer and harder (Barnett, 2006; Atwood and Bowen, 2007). However, the rate of post-exercise recovery after prolonged exercise depends on which physiological systems are debilitated (Miller *et al.*, 2004). This means that some athletes might need longer recovery time than others.

Myburgh (2003) refers to the non-biological factors that influence endurance performance, for instance optimal training, years of training, performance level, pace and optimal recovery. Very little research has actually been done on post-exercise or in-between training recovery techniques; if one considers that it is an accepted practice after competitions and training for most athletes (Myburgh, 2003; Dawson *et al.*, 2005).

Overreaching is generally used as a training tool when the athletes are building up to a big event. Overreaching results in a temporary decrease in athletic performance, this may take a few days to several weeks to recover. Approximately 15–50% of competitive endurance athletes experiences overreaching during their competitive training phase (Coutts *et al.*, 2007). Athletes, who train on a regular basis at these high-intensities or for prolonged periods (overreaching), together with limited recovery might be at risk of overtraining syndrome (Väänänen, 2004).

Overtraining occurs when an excessive repeated training stimulus is applied with limited recovery, in such a manner that the athlete does not make any positive adaptations to the stimulus, but rather responds negatively through injury or overreaching (Ball and Herrington, 1998; Väänänen, 2004). It is therefore evident that sufficient recovery is an important part of an endurance-trained athlete's training program. The perfect balance between training volume and duration of recovery is necessary to optimize athletic performance and minimize injury.

Endurance training mostly exhausts the nutritional factors, physiological, neurological, and psychological properties of the athlete (Cochrane, 2004). Cochrane (2004) gave possible examples of recovery techniques, which are used for these above-mentioned aspects. To aid the recovery of the metabolic factors, for example, techniques like rehydration and carbohydrate loading, might be employed.

With muscle fatigue, injury or damage athletes could make use of hydrotherapy, cooling down and massage techniques to increase the blood flow to the areas affected, and this will help with neurological recovery, together with passive rest. For psychological recovery visualization techniques, progressive muscular relaxation, meditation, flotation and massage are recommended (Cochrane, 2004).

## **1. Active recovery**

Active recovery is a form of tapering involving low intensity aerobic activities, such as light jogging, swimming, and/or walking, which is one of the two most common methods for facilitating recovery in athletes after strenuous exercise (Martin *et al.*, 1998; Myburgh, 2003; Gill *et al.*, 2006). With active recovery, the athlete continues submaximal exercise after the exercise bout for 7.5 to 20 minutes. Some studies have used intensities of less than 65% of the athlete's  $VO_{2max}$  for active recovery (Wilcock *et al.*, 2006). However, the typical criteria for active recovery are suggested to be at intensities of about 30 to 40% of the athlete's  $VO_{2max}$ . Several authors state that active recovery is the preferred recovery modality over passive recovery (Martin *et al.*, 1998; Myburgh, 2003; Gill *et al.*, 2006).

The suggested mechanism of active recovery is that it increases blood flow through the active muscles, increases the turnover of metabolic substrates, distributes blood better and removes lactate and creatine kinase faster. Active recovery also improves flexibility and reduces muscle cramping (Berry and McMurray, 1987; Martin *et al.*, 1998; McArdle *et al.*, 2001; Chatard *et al.*, 2004; Reilly and Ekblom, 2005; Gill *et al.*, 2006). The removal of both lactate as well as creatine kinase after exercise from the damaged muscle is important contributors to the recovery of athletic performance post-exercise. Since these aspects retard the post-exercise recovery rate of soft tissue injury and possibly contribute to fatigue (Berry and McMurray, 1987; Gill *et al.*, 2006).

Additionally, numerous authors suggest that active recovery increases lactate removal faster than passive recovery and reduces body temperature and blood flow more gradually (Martin *et al.*, 1998; McArdle *et al.*, 2001; Chatard *et al.*, 2004; Reilly and Ekblom, 2005). When the athlete continues to exercise at a low intensity after events or training the increased heart rate and the active muscles improves blood flow. It has been proposed that it is this improved blood flow that clears lactate faster, by shuttling the lactate to the elimination sites. At these sites, such as the heart and skeletal muscle, the lactate is used as a source of fuel (Brooks *et al.*, 2005).

Additionally, the increased metabolic rate aids lactate metabolism via oxidation and gluconeogenesis (Martin *et al.*, 1998; Chatard *et al.*, 2004; Draper *et al.*, 2006). As much as 70% of the athlete's lactate is cleared by oxidation (Martin *et al.*, 1998). At intensities below 50% of the individual's  $VO_{2max}$  the lactate clearance is directly related to the exercise intensity. In other words, active recovery will remove lactate from the circulation faster than no activity, such as passive recovery (Reilly and Ekblom, 2005). This may indicate that active recovery post-exercise is a better strategy than the traditional rest. However, it is important to realize that passive recovery also allows lactate clearance, but according to some authors not as efficient as active recovery.

Gill *et al.* (2006) investigated the effects of four post-match recovery techniques on interstitial creatine activity in 23 elite rugby players (age:  $23 \pm 3$  years). Subjects had to be injury free and played for at least 20-minutes to be included in the study. Creatine kinase activity is not only greater due to the running aspect of rugby, but also due to the collisions in the game. The



four recovery strategies i.e. compression garments, active, passive, and contrast water therapy were monitored over a four-week period in the competition season.

Subjects were randomly assigned to one of the four recovery techniques. Interstitial creatine kinase samples were collected three and a half hours prior to matches and then immediately after, 36 and 84-hours post-exercise. The recovery strategies were applied after each of the matches. All of these strategies, except in the cases where the subjects had to wear compression garments, were executed before showering, rehydration, snacking, and getting ready for the post-match function. With the passive recovery (PAS) protocol subjects had to sit for nine minutes after a match. Subjects in the active recovery group (ACT) cycled for seven minutes at a low-intensity (150 Watts; 80 to 100 rpm) on a stationary bicycle. The contrast water therapy (CWT) and compression garments (WCG) protocols will be discussed under the appropriate sections.

Post-match interstitial creatine kinase activity was significantly higher (Pre:  $1023.0 \pm 308.3$  u.L<sup>-1</sup> vs. post:  $2194 \pm 833.7$  u.L<sup>-1</sup>;  $P < 0.01$ ). No significant differences were noted, throughout the experiment, between the ACT, CWT, and WCG recovery strategies in the male rugby players. There was a significant difference between passive recovery (PAS) and in the individuals participating in the other three recovery modalities, at 36 and 84-hours post-exercise ( $P < 0.05$ ). At 84-hours passive recovery indicated a 39.0% improvement in interstitial creatine kinase activity, while active recovery was 49.2% more pronounced than passive recovery with a 88.2 % recovery ( $P < 0.05$ ).

According to Gill *et al.* (2006), active recovery is one of the best recovery interventions to increase blood flow and distribution to the active muscles. Gill *et al.* (2006) hypothesized that active recovery would yield the greatest reduction in post-match creatine kinase concentration. Even though active recovery showed the highest relative percentage of recovery (88.2 %) at 84-hours, no significant difference was found when compared to contrast water and compression garment strategies (85.0% and 84.4 %, respectively;  $P < 0.05$ ). Due to the time constraint and resource limitations seven minutes were selected for active recovery, but the authors suggest that a longer active recovery period would be even more beneficial.

Martin *et al.* (1998) performed a counterbalance experimental study with repeated measures, in which they investigated the effectiveness of three experimental conditions, namely i.e. active recovery (ACT), sport massage (MAS) and rest (C). The subjects were competitive male cyclist with ages ranging from 21 to 34 years ( $n = 10$ ; age:  $24.5 \pm 3.98$  years;  $VO_{2max}$ :  $55.87 \pm 3.87$  mL.kg<sup>-1</sup>.min<sup>-1</sup>). Prior to the main testing subjects had to perform a  $VO_{2max}$ , to obtain baseline values.

They were randomly requested to complete three supramaximal Wingate cycling tests. Each Wingate test entailed a 30-second all out sprint at a high braking force that was related to body weight, followed by a two minute rest. The third Wingate test was followed by a five minute rest period and then the 20-minute experimental procedure, which involved active recovery, sport massage or rest. Subjects participated in all three conditions, separated by 48 hours and acted as their own control.

Blood lactate was assessed 10 minutes before exercise and immediately after, then five minutes after the last Wingate and every five minute interval in the 20-minute experimental condition. The active recovery protocol required that the cyclist pedaled at a rate of 80 rpm at an intensity of 40% of his  $VO_{2max}$ . For the sport massage intervention, athletes were massaged for 20 minutes using typical post-exercise recovery techniques. For the resting condition, the subjects lay supine for 20 minutes.

The Wingate tests significantly increased the athlete's lactate concentration for all conditions ( $P < 0.05$ ). Active recovery significantly reduced lactate concentration by 59.38% (6.79 mL.L<sup>-1</sup> decrease) compared to the sport massage reduction of 36.21% (4.39 mL.L<sup>-1</sup> decrease) and resting conditions by 38.67% (4.33 mL.L<sup>-1</sup> decrease).

One of the possible problems with a long active recovery between bouts is that it depletes muscle glycogen stores. This may be harmful to athletic performance. McAinch *et al.* (2004) investigated the difference between passive and active recovery in repeated 20-minute aerobic sessions. Each aerobic session was separated by a 15-minute active (ACT) or passive (PAS) recovery bout. For the passive recovery the subjects laid supine. In the active recovery bout the subjects continued to cycle at 40% of their  $VO_{2max}$  after the aerobic exercise. Each session was repeated on a separate occasion.

The subjects were seven trained men (age:  $22 \pm 4$  years;  $VO_{2max}$ :  $58.3 \pm 9.4$  mL.kg<sup>-1</sup>.min<sup>-1</sup>). The investigators collected blood samples and muscle biopsies to assess glycogen levels in addition to blood and muscle lactate samples. Muscle samples were collected before and immediately after the first experimental bout. Blood samples were collected at rest and every five minute interval throughout each main trial.

Before the main exercise bout on a separate visit, the subjects'  $VO_{2max}$  values were obtained via an incremental exercise test. Seven days after the initial test, subjects visited the laboratory for a familiarization time trial. In this session the subjects had to perform as much work as possible for 20 minutes, which was similar to the main testing sessions. The 48 hours before both the main sessions, subjects cycled at 70 %  $VO_{2max}$  for 60 minutes. For each of the main experimental trials the subjects completed two 20-minute full out cycle sessions, separated by 15 minutes. In this 15-minute interval the subjects randomly performed active or passive recovery. After five days the same protocol was repeated for the second experimental trial. Between exercise and recovery periods, there was a one-minute transition interval. In this interval blood samples and biopsies were collected. Both trials showed a significant decrease in performance in the second 20-minute bout (ACT:  $\sim 320$  W vs.  $\sim 300$  W; PAS:  $\sim 319$  W vs.  $\sim 299$  W;  $P < 0.01$ ). Muscle glycogen did not significantly differ between the two conditions at any point ( $P < 0.05$ ).

Blood lactate concentrations were higher in the passive recovery group ( $7.7 \pm 1.4$  mmol.L<sup>-1</sup>), while the active recovery group demonstrated a lower blood lactate level before starting the second bout of each trial ( $4.4 \pm 0.7$  mmol.L<sup>-1</sup>). The difference was statistically significant between active and passive recovery ( $P < 0.05$ ). No significant difference was found between the active and passive group's muscle ATP ( $26.0 \pm 0.72$  mmol.kg<sup>-1</sup>dry weight vs.  $25.8 \pm 0.85$  mmol.kg<sup>-1</sup>dry weight, respectively) before and after each bout. Muscle PCr was reduced in both ACT and PAS by  $\sim 228$  % ( $P < 0.05$ ). After the first bout the Cr was elevated by  $\sim 164$ % and  $\sim 144$  % in the active and passive recovery group, respectively ( $P < 0.05$ ) compared to before the exercise. Nevertheless, there was no difference between the two main trials. McAinch *et al.* (2004) concluded that active recovery does not aid improvement in performance between subsequent aerobic sessions, regardless of decreased lactate circulation levels.

Some of the associated methods of conventional active recovery include: stretching, low intensity weight training and aqua walking/running. These methods predominantly function in a similar fashion as active recovery (Dawson *et al.*, 2005; Gill *et al.*, 2006; Stracciolini *et al.*, 2007).

Young (2007) defines static stretching as an activity by which the body position is taken to the end of the joint's range of motion and held for about ten to 30 second intervals. Traditionally it has been suggested that stretching the involved muscles post-exercise, would assist recovery, prevent injury, reduce perceived muscle soreness, and improve performance in the athlete. Apparently stretching would apply tensile force to the affected tissue and if applied progressively this will aid in tensile strength of the damaged area (Ball and Herrington, 1998).

Recent research however, indicated little or no effect of stretching on muscle strength, delayed onset of muscle soreness (DOMS) and muscle damage (assessed by creatine kinase concentration) (Dawson *et al.*, 2005). In addition, pre-exercise static stretching has been associated with impaired athletic performance, such as reduced muscle strength, strength endurance and power. The reasons for this negative influence on athletic ability are associated with reduced neural activation, lesser musculotendinous stiffness or even a combined neural and muscular effect (Young, 2007).

A review by Andersen (2005) on pre- and post-exercise stretching also concluded that stretching does not produce any additional benefits. Specifically post-exercise stretching only showed a two per cent reduction in muscle soreness over the initial 72 hours post-exercise. This is not significant or practical in athletic populations (Andersen, 2005). The protocols assessed in this review typically involved a post-exercise stretching routine of four to ten repetitions held for about 30 to 120 seconds. Anderson (2005) suggested that the risk of injury would be less than 5% in athletes who followed a stretch routine. In fact, by stretching the muscle up to 120% of its resting length, may lead to muscle damage in some cases. However, protocols that sustained stretching for longer intervals might produce different results.

Aqua walking or -running at low intensities also require further investigation (Dawson *et al.*, 2005). Theoretically, water places less strain on the body and the joints and aids venous return

via hydrostatic pressure differences (Dawson *et al.*, 2005). It has also been suggested that the subtle massage-effect of the water resistance reduces muscle stiffness and the increase in temperature and blood flow in the muscle could reduce muscle soreness. Water furthermore limits eccentric muscle action and therefore it will not augment existing muscle damage. Thus, water-exercise, such as walking or running might aid recovery from DOMS (Reilly and Ekblom, 2005; Takahashi *et al.*, 2006).

Takahashi *et al.* (2006) provided evidence for aqua exercise as a therapeutic modality that improves DOMS after a downhill run. Ten male long-distance runners (age:  $20 \pm 1$  years) were included into the study. The study consisted of two groups, i.e. the intervention group, which was the aqua-exercise group, and the control group, which performed no recovery strategies.

Subjects were requested to perform three sets of five-minute downhill runs (DHR) at  $-10\%$ . Each subject ran at speeds that corresponded to his best 5000m-race time ( $335.7 \pm 6.1$  meters per minute). In addition, each set was separated by a five-minute rest-interval. Immediately after the DHR and during the following 48-hours after the DHR protocol, the aqua-exercise group performed 30-minutes of walking, running and jumping in a pool ( $29 \pm 0.8^\circ\text{C}$ ) everyday. The control group performed no recovery exercises.

The DHR protocol caused significant muscle damage, since the 24-hour post-exercise creatine kinase levels were increased in both groups (Pre: C:  $347 \pm 195 \text{ u.L}^{-1}$  and Aqua:  $278 \pm 137 \text{ u.L}^{-1}$  vs. 24-hours: C:  $555 \pm 155 \text{ u.L}^{-1}$  and Aqua:  $615 \pm 337 \text{ u.L}^{-1}$ ;  $P < 0.05$ ). Muscle power in the control group was reduced 24-hours after the DHR ( $1652 \pm 418 \text{ W}$  vs.  $1379 \pm 477 \text{ W}$ ;  $P < 0.05$ ), but not in the intervention group ( $1568 \pm 148 \text{ W}$  vs.  $1504 \pm 130 \text{ W}$ ;  $P > 0.05$ ). At 72-hours post-exercise the perceived muscle soreness was greater in the calf muscles of the control group ( $P < 0.05$ ). The sit and reach, stride length and ankle range of motion to assess flexibility were not significantly different between the groups ( $P > 0.05$ ). Both groups demonstrated reduced flexibility after the DHR, but it was returned to baseline values by 72-hours. Reaction time followed a similar pattern, but was not returned to baseline values within 72-hours ( $P > 0.05$ ).

This study demonstrated that aqua-exercise aids the recovery rate of muscle power, stiffness, and perceived soreness, compared to just rest. According to the authors, these results support the assumption that water exercise mildly massages the legs; increase muscle temperature and blood flow and the non-eccentric contractions associated with water- exercises helps to reduce muscle damage (Takahashi *et al.*, 2006).

## **2. Passive recovery**

Traditionally passive recovery or inactive recovery as it is sometimes called is when the athlete lies down or ceases activity after exercise. Athletes believe that the complete rest reduces the resting energy demand and this permits more oxygen to the muscles for the recovery processes (Cochrane, 2004). In other words, the inactivity restores the physiological homeostasis (Wilcock *et al.*, 2006).

Traditional forms of passive recovery include sleeping, doing nothing, or staying seated, standing and lying supine post-exercise. With long-term passive recovery an athlete continues to perform daily activities (McArdle *et al.*, 2001; Wilcock *et al.*, 2006). More contemporary forms of passive recovery might be used in conjunction with other post-exercise recovery strategies, such as massage, contrast therapy, and flotation (Cochrane, 2004).

## **3. General therapeutic interventions**

Therapeutic interventions are traditionally used to aid the recovery of acute sporting injuries and for rehabilitation purposes. However, athletes want to recover as quickly and as effectively as possible, so that they can participate in the following event or training bout. As a result, athletes use various additional recovery methods, which includes prophylactic or therapeutic techniques (Cochrane, 2004; Dawson *et al.*, 2005). In other words, athletes might use preventative or therapeutic modalities associated with injury-recovery, to aid post-exercise recovery for muscle damage and/or to prevent injury. Some of these therapies include: sauna or spa treatments, iontophoresis with anti – inflammatory medication, acupuncture, flotation, hyperbaric oxygenation, various ultrasound and electrical stimuli, alternating hot/cold (contrast baths) therapy or separate cryotherapy (cold) and thermotherapy (hot) (Cochrane, 2004; Dawson *et al.*, 2005; Gill *et al.*, 2006; Stracciolini *et al.*, 2007).

### 3.1 Electrotherapeutic and associated techniques

Examples of electrical stimulation are transcutaneous electrical nerve stimulation (TENS) and microcurrent electrical stimulation (MES). It is believed that both of these strategies reduce pain, oedema and increase strength, ROM as well as aid the healing of fractures. Various electrical waves are utilized. These waves have different physiological responses, which are associated with the characteristics of the waveforms (Stracciolini *et al.*, 2007).

However, Craig *et al.* (1996) demonstrated no significant improvement in recovery from DOMS with TENS. The subjects ( $n = 48$ ; 24 male and 24 female) of this test first had to complete a concentric exercise, to obtain a one repetition maximum. To induce DOMS they had to perform three sets of eccentric exercises to exhaustion in their non-dominant arm. The subjects were randomly divided into four groups, i.e. the control, placebo, low TENS (200  $\mu$ sec; 4Hz) and high TENS (200  $\mu$ sec; 100Hz) groups. During the 72 hours after the eccentric exercises, the subjects received treatment and investigators assessed range of motion and pain in the upper arm.

All of the measurements indicated no significant differences between the four groups. All the groups, however, reported increased pain and reduced range of motion after the eccentric exercise ( $P < 0.05$  for all measurements over time). Thus, the authors concluded that TENS do not aid recovery from DOMS. Almekinders' review (1999) supported this observation that TENS and MES showed no conclusive indication of recovery after exercise-induced muscle damage.

Another form of an electrophysical technique is the high ultra frequency sound waves, namely ultrasound. With ultrasound the electrical energy is converted into inaudible high-frequency mechanical vibrations (Speed, 2001). Ultrasound is frequently used and widely available in sports medicine, but mainly in a rehabilitation context to aid injury recovery (Warden, 2003).

Ultrasound varies in frequency, intensity, amplitude, focus, and beam uniformity. This means that the amount of energy that reaches the specific site depends on these characteristics and the tissue through which the waves must propagate. Therapeutic ultrasound tends to have a frequency range between 0.75 to three MHz. For deeper injuries (three to five cm),

frequencies at one MHz are utilized. Tissues that are rich in protein, such as skeletal muscle, have a high absorption ability. Ultrasound could be continuous or pulsed, where the pulsating ultrasound alternate between on and off intervals (Speed, 2001).

The physical effects exerted by ultrasound might be thermal or non-thermal. The thermal ultrasound increases the tissue temperature up to 40 – 45°C. Increased blood flow, reduced muscle spasms, less stiff collagen fibers, and a mild inflammatory response are induced by thermal ultrasound. Non-thermal ultrasound involves cavitation and acoustic microstreaming. It is this non-thermal ultrasound that is used on soft tissues. Cavitation results in accelerated flow, due to the gas-filled bubbles that expand, and compress in tissue fluid because of the pressure changes associated with ultrasound waves. Stable cavitation is beneficial for tissue injury, while unstable cavitation could result in soft tissue damage. Microstreaming could change a cell membrane's structure, function and permeability, due to the mechanical pressure that cause a one-way movement of fluids along cell membranes (Speed, 2001).

It is assumed that the mechanical effects, stable cavitation, and the microstreaming of the ultrasound aids tissue regeneration and healing. The acoustic microcurrent and cavitation accelerate the diffusion of ions and metabolites across the membrane of the damaged tissue. Ultrasound increases the cell membrane's permeability to calcium and sodium ions, which aids the healing process and lessens pain and spasms, respectively (Stay *et al.*, 1998). In addition, ultrasound helps to increase mobility and limits scar tissue formation, by limiting fibroblastic activity and reducing acute inflammation. All of these factors supposedly augment the tissue renewal process (Ball and Herrington, 1998; Stay *et al.*, 1998; Speed, 2001; Stracciolini *et al.*, 2007).

Even though ultrasound is a common practice in soft tissue injuries, little evidence exists that it actually assists soft tissue damage recovery (Stay *et al.*, 1998; Speed, 2001). According to Speed (2001), some studies found that ultrasound increases tissue tensile strength; while others found no evidence that ultrasound modify the structure or mechanical characteristics of the connective tissue. This would mean that it does not strengthen tensile strength when applied to the damaged or injured tissue (Ball and Herrington, 1998). Warden (2003) insists that the effect of ultrasound in a clinical sports medicine setting is minor, but does indicate



improvement in fractures and associated musculoskeletal injury and inflammation. Almekinders (1999) also reported no improvements in DOMS related symptoms via ultrasound.

Modified forms of ultrasound are phonophoresis and extracorporeal shock wave therapy (ESWT). Phonophoresis is similar to Iontophoresis. The difference is ultrasound waves are used instead of electrical currents. ESWT is focused high-energy ultrasound energy (Speed, 2001). Iontophoresis is a painless method of delivering medication via electrical currents, through the skin, to the injured or damaged area. Generally, it is used in conjunction with anti-inflammatory drugs, like dexamethasone, to treat the inflammatory response and muscle soreness. These inflammatory responses might be manifested as bursitis, tendonitis, and enthesopathy (Straccolini *et al.*, 2007).

Athletes often use anti-inflammatory medication (nonsteroidal anti-inflammatory drugs (NSAID) and/or corticosteroids) to alleviate pain and muscle discomfort post-exercise. However, the treatment of eccentric exercise-induced muscle soreness with anti-inflammatory drugs has been shown to be mainly ineffective (Reilly and Ekblom, 2005). Almekinders (1999) reviewed some of the studies that have investigated the effects of anti-inflammatory medication, and found contradictory results. Overall, it seems that NSAID interventions are associated with only a small inhibition of the initial inflammatory response and related symptoms.

The NSAIDs hinder the enzyme cyclo-oxygenase's function, which then inhibits the inflammatory mediator prostaglandin. Furthermore, some animal studies show a reduction in oedema by NSAIDs. Even though improved muscle contractility has been associated with NSAID, it seems to be only temporary and is related to retarded muscle regeneration (Almekinders, 1999). In other words, it appears that the NSAID medication helps to control the inflammation, but lengthens the natural recovery time-line, since a prolonged low grade inflammatory response is evident (Ball and Herrington, 1998). Conversely, corticosteroids have demonstrated an adverse effect in the recovery process of muscle injuries and contusions (Almekinders, 1999).

### 3.2 Hot and cold therapy

Hot and cold therapy could be used either individually or in combination, such as in contrast therapy. Cryotherapy is the application of a cold stimulus like ice packs, cold baths or whirlpools and ice massage (Cochrane, 2004; Stracciolini *et al.*, 2007). Generally cold therapy is applied during the acute recovery phase (24 – 48 hours) (Cochrane, 2004; Stracciolini *et al.*, 2007).

Cryotherapy (cold) reduces the skin, subcutaneous and muscle temperature and this reduced temperature stimulates the sympathetic cutaneous receptors. The sympathetic stimulation constricts the blood vessels, which reduces the swelling and inflammation by slowing the metabolic rate (Cochrane, 2004). Furthermore, it is believed that the cold therapy diminishes the sensation of pain and reduces muscle spasms, by reducing the rate by which the nerve impulse is conducted (Cochrane, 2004).

Thermotherapy assists in the recovery of chronic conditions, spasms, reduced range of motion and scar tissue formation; it is the opposite of cold therapy (Stracciolini *et al.*, 2007). Heat is applied to the injured area, such as heat packs, infrared lamps, warm baths, showers and paraffin wax (Cochrane, 2004; Stracciolini *et al.*, 2007). Thermotherapy increases tissue temperature. The heat reduces sympathetic nerve stimulation and this dilates the blood vessels. Blood flow is accelerated to the area to which heat is applied (Cochrane, 2004). The increase in local circulation carries more oxygen and antibodies to the affected area and also assists in the removal of metabolites. Thermotherapy also increases muscle flexibility and reduces muscle spasms (Cochrane, 2004).

Water immersion is another therapeutic modality that improves recovery of athletic performance. There are four possible types of water immersion strategies, i.e. contrast therapy, separate hot or cold techniques and neutral water temperature. Water immersion varies in temperature and usually ranges between six to 20 minutes (Wilcock *et al.*, 2006). It is assumed that water immersion works in a similar fashion as active recovery (Wilcock *et al.*, 2006). Thus, water immersion increases blood flow and blood lactate removal. Furthermore, the additional benefit of water immersion is that it does not expend additional energy as with active recovery (Wilcock *et al.*, 2006). It is important with water immersion that a large

portion of the body is submerged, since this will increase the extra-intravascular movement of fluid and cardiac output and blood flow. Smaller body segments may not have the same impact (Wilcock *et al.*, 2006).

In the review by Wilcock *et al.* (2006) the authors concluded that ten to 20 minutes of water immersion will not have a negative impact on athletic recovery. Furthermore, some of the reviewed studies indicate a sustained strength and jumping ability due to water immersion, but no influence was observed in cycling or running performance. Furthermore, athletes who experienced prolonged fatigue associated with muscle damage after strenuous exercise indicated that water immersion helped. Oedema was also reduced in some of the studies, and this might also contribute to aid performance recovery.

Of the four types of water immersion strategies, contrast therapy is one of the most popular post-exercise recovery techniques (Wilcock *et al.*, 2006). With contrast water therapy (CWT) hot and cold techniques are alternated, like ice baths/showers alternating with warm baths/showers, or warm spas with cool pools. Hot-cold therapy, as it is also known, has become increasingly popular in sport as a post-exercise recovery modality.

Some speculate that the temperature changes created by contrast therapy lets the blood vessels dilate and constrict, thereby creating a mechanical shunting or pumping action. The result is that blood flow is increased to the affected area and hydrostatic pressure lowered and this reduces the swelling (Cochrane, 2004; Dawson *et al.*, 2005; Gill *et al.*, 2006). It has also been proposed that this pumping action may aid in the clearance of exercise-induced metabolites (i.e. lactate), repair muscle damage and reduce muscle spasms. In addition, it is thought that contrast therapy slows down the athletes' metabolic rates and revitalizes their psychological state (Cochrane, 2004). This will mean that a lesser amount of metabolites are produced.

In the previously mentioned study by Gill *et al.* (2006) (*pages 60 to 61*) elite rugby players had to randomly participate in four different recovery strategies. The subjects in the CWT group were immersed for nine minutes, up to their anterior superior iliac spine, in alternating warm (two-minutes at 40 to 42 °C) and cold-water baths (one-minute at 8 to 10 °C). They found that contrast therapy enhanced interstitial creatine kinase recovery after the rugby match compared to passive recovery. Eighty-four hours after a rugby match subjects showed a 46%

greater recovery with CWT (85 %) than with passive recovery (39 %;  $P < 0.05$ ) (Wilcock *et al.*, 2006).

The ratio of hot vs. cold application, for injury and post-exercise treatment is usually 3:1 or 4:1, and the therapy always ended with the cold treatment. This final cold application allows the blood vessels to constrict in the injured area. The temperature and application time between hot and cold spas/baths differs from showers. Baths are heated up to 37 – 43 °C and the athlete stays in the bath for three to four minutes, and then alternates with 12 – 15 °C cold baths for 30 to 60 seconds. Treatments generally last for 20 – 30 minutes twice a day. Whereas with showers it is usually one to two minutes hot, and alternated with ten to 30 seconds of cold therapy. The reason for these time differences between the two methods has not been specified (Cochrane, 2004). Some of the studies reviewed by Cochrane (2004) did indicate that 60 minutes of cold therapy after warm therapy is not enough time to lower the tissue temperature.

Further research, to indicate whether contrast therapy is an effective post-exercise recovery technique and the most effective application time, mode and ratio between hot and cold, is warranted (Cochrane, 2004; Gill *et al.*, 2006).

### 3.3 Sport massage therapy

Massage therapy involves the manipulation, precise pressure application, friction, rubbing and kneading of the body (Hemmings, 2001). Sport massage also incorporates Swedish massage strokes together with other techniques, such as compression, trigger points and cross fiber friction (Martin *et al.*, 1998; Hemmings, 2001). Though evidence supporting sport massage is limited and conflicting, it is still a very common post-exercise technique (Martin *et al.*, 1998; Almekinders, 1999; Reilly and Ekblom; 2005; Zainuddin *et al.*, 2005).

Massage incorporates mechanical and reflectory effects. The mechanical effect of massage shunts blood, stretch soft tissue and breaks scar tissue and fibrous adhesions, which would improve microcirculation, tissue flexibility and also increase tissue permeability. The reflectory aspects of massage lead to relaxation and improved microcirculation (Ernst, 1998; Hemmings, 2001).

Certain studies show that massage improved the athlete's flexibility, lymph drainage, oedema and blood flow in the previously active muscles, while other studies demonstrated no improvements in recovery (Ernst, 1998; Martin *et al.*, 1998; Almekinders, 1999; Zainuddin *et al.*, 2005). Additionally, it is believed that massage helps to improve performance, reduce fatigue and aid relaxation as well as recovery from injury (Hemmings, 2001; Zainuddin *et al.* 2005).

Theoretically, sport massage facilitates lactate removal by improving and accelerating skeletal muscle blood flow. Blood flow is accelerated to the involve tissue by the direct mechanical pressure on the blood vessels and the indirect influence on the circulation of the muscle, like the mechanoreceptor stimulation that releases vasodilators and decreases sympathetic tone. Even though most studies produced conflicting results, it has been reported that the compression technique increased blood flow significantly (Martin *et al.*, 1998).

As described in the active recovery section (*page 61 to 63*), Martin *et al.* (1998) compared sport massage, active recovery and rest and the influence these treatments had on blood lactate clearance. The results demonstrated no significant difference between sport massage (36.21%) and conventional rest (38.67%) interventions in absolute or relative decrease in blood lactate. As mentioned before, there was a significant reduction in blood lactate with active recovery, compared to massage recovery (decrease: 4.39 vs. 6.79 ml.L<sup>-1</sup>, sport massage and active recovery, respectively;  $P < 0.05$ ).

The fact that sport massage did not have an influence on lactate clearance in the circulation could be attributed to several possible factors. For example the increased blood flow to the involved skeletal muscle might not be substantial enough to impose a reduction in blood lactate. Additionally, it has been suggested that increased blood flow does not aid lactate clearance. This could mean that massage does not have an influence on the processes that accelerate lactate clearance or metabolism (Martin *et al.*, 1998).

Zainuddin *et al.* (2005) showed that a ten-minute massage three hours after maximal elbow flexion exercises had a significant effect (30 % reduction) on muscle soreness and reduced swelling. No significant differences were noted in maximal isometric and isokinetic strength. After massage range of motion decreased by 30 % immediately post-exercise and did not

recover to pre-exercise values by day four in the massage group. Massage thus showed no significant effect on muscle function and reduced stiffness in the muscle joints.

A review by Hemmings (2001) of the scientific literature, on massage as a therapeutic intervention in sport, addressed the possible influences of massage on the athlete's physiology, psychology and performance. The review indicated that massage has demonstrated conflicting results on the physiological aspects, such as improved blood flow, lactate clearance and alleviating delayed onset of muscle syndrome (DOMS). Hemmings (2001) and Martin *et al.* (1998) is in agreement that active recovery seems to be more effective than massage in accelerating lactate removal. Massage seems to have a positive psychological effect on the athlete's perceptions of recovery. However, evidence is lacking for the support of massage as a performance enhancing aid. The author concluded that the literature reports conflicting evidence on the possible effects of massage and that further research is needed (Hemmings, 2000). Furthermore, both Ernst (1998) and Hemmings (2000) pointed out that methodological flaws in past studies may have contributed to this uncertainty about the benefits of massage therapy.

## **E. CONCLUSION**

Various physical, psychological and nutritional recovery techniques are recommended by coaches, athletes, and trainers to speed up the recovery process. These strategies are intended to aid recovery of the athlete's athletic performance and functional capacity immediately to seven days post –event or training bouts. This will theoretically allow the athletes to return to training or events as soon as possible, without experiencing fatigue, overreaching, burnout, or poor performances (Cochrane, 2004).

However, research on the various post-exercise recovery practices are contradicting. The conflicting results in the literature could be attributed to the methodological variation in mode of inflicting muscle damage, interindividual differences or the duration of therapy employed. This makes it difficult for sport scientist to compare results and to draw conclusions. Thus, recovery treatments and outcome measure depend on a combination of factors, including interindividual differences, lifestyle, and the type of activity. It is recommended that post-exercise recovery should be seen as a holistic approach and not as an isolated recovery

technique (Cochrane, 2004; Reilly and Ekblom, 2005). Prevention is better than cure, which means that athletes would benefit a lot from more research in post-exercise recovery.

## CHAPTER FOUR

### COMPRESSION GARMENTS

#### A. INTRODUCTION

Compression therapy (intermittent or continuous) is typically used in clinical settings, for instance physical therapy and rehabilitation, to prevent oedema and to enhance blood circulation in individuals with chronic venous insufficiencies such as deep vein thrombosis, venous stasis, and varicose veins. Furthermore, it is also used in patients with spinal cord injuries, patients with certain cancers (i.e. breast cancer), orthostatic hypotension, and chronic inflammatory problems, like lymphoedema. It is used extensively in the treatment of burns and scars (Brennan and Miller, 1998; Choucair and Phillips, 1998; Gniadecka *et al.*, 1998; Bergan and Sparks, 2000; Benkö *et al.*, 2001; Harris *et al.*, 2001; Hirai *et al.*, 2002; Doan *et al.*, 2003; Partsch, 2003; Chatard *et al.*, 2004; Kraemer *et al.*, 2004; Van den Kerckhove *et al.*, 2005; Maton *et al.*, 2006; Trenell *et al.*, 2006; Ali *et al.*, 2007; Rimaud *et al.*, 2007). Compression garments may also protect the limbs from burns, insect bites, or lacerations (Harris, *et al.*, 2001).

In a clinical setting, compression has been shown to reduce or manage oedema, diminish the effect of venous hypertension and associated symptoms, assist or restore calf muscle-pump function, increase the velocity of venous return and support and protect the injured limb (Choucair and Phillips, 1998; Bergan and Sparks, 2000; Blecken *et al.*, 2005). In addition, compression, in conjunction with other components (i.e. rest, ice, compression and elevation), is one of the most common treatments of acute injuries, like strains. However, limited research has been done on these simple modalities (Prentice, 2001; Kraemer *et al.*, 2004; Trenell *et al.*, 2006).

Recently the focus of the application and utilization of compression therapy has shifted from the clinical setting to sports-related rehabilitation and injury management. However, according to several authors, more research is necessary to determine whether compression garments are a practical and effective recovery method in sports medicine, especially for subelite and elite athletes (Kraemer *et al.*, 2004; Barnett, 2006; Trendell *et al.*, 2006; Rimaud *et al.*, 2007). In



other words, it needs to be established whether there is any truth in anecdotal evidence that compression garments enhance post-exercise recovery and athletic performance.

## **B. ANECDOTAL CLAIMS**

For some reason it has simply been accepted by trainers and athletes that compression garments enhance recovery after exercise. These anecdotal claims are subjective or are based on isolated case studies. Athletes motivate their reasons for wearing compression garments by claiming that the garments are fashionable, lessen chaffing, prevent injury and enhance performance (Doan *et al.*, 2003; Bringard *et al.*, 2006<sup>a</sup>; Duffield and Portus, 2007).

Modern internet marketing claims that compression garments have the following benefits: it improves circulation in the lower body, thus attenuating muscle fatigue; it increases aerobic capacity, proprioception and power production; it lowers perceived exertion; diminish swelling, reduce muscle soreness and lessen the enzyme marker serum creatine kinase (“What Marathon Runners Know about Healthier Legs.” *GFIT (graduated compression) TECHNOLOGY*.” [Hyperlink <http://www.healthylegs.com/whmaruknabhe.html>]. Accessed on 2 August 2007). Other claims that have been made are that compression garments, together with individualized exercise programs, may enhance lymphatic drainage, restore muscle strength, flexibility and lower limb endurance, as well as reduce the energy cost of running (Bringard *et al.* 2006<sup>a</sup>).

## **C. TYPES OF COMPRESSION GARMENTS**

Compression garments can be custom made or prefabricated and are either elastic or non-elastic (Brennan and Miller, 1998; Bergan and Sparks, 2000). These garments can be worn as upper, lower or full body pieces (Duffield and Portus, 2007). The three types of compression garments currently available are: (i) graduated compression stockings with a pressure gradient (Brennan and Miller, 1998; Agu *et al.*, 1999; Iwama *et al.*, 2000; Barnett, 2006), (ii) compression sleeves worn over limbs and joints for support, as well as to limit swelling (Barnett, 2006); these garments can be inflatable as well (Brennan and Miller, 1998), (iii) elastic garments generally used for exercise (Barnett, 2006), which apply a uniform

compression and have the ability to recover its size and shape after it has been deformed (Agu *et al.*, 1999; Bergan and Sparks, 2000).

Elastic garments are the traditional type of graduated compression garments. Graduated compression garments are generally worn by individuals suffering from venous insufficiencies, i.e. deep vein thrombosis and other lower limb circulatory problems (Bergan and Sparks, 2000; Barnett, 2006). However, more recently graduated compression garments have been used during exercise. As mentioned above, graduated compression creates a pressure gradient over the length of the limb. In other words, the distal compression (i.e. ankle) is greater than the proximal compression (i.e. calf or thigh), thereby applying various degrees of gradual pressure to different segments of the limb (Agu *et al.*, 1999; Brennan and Miller, 1998; Iwama *et al.*, 2000). Graduated compression garments have three main advantages, i.e. (a) it reduces ambulatory venous pressure, (b) increase venous filling time and (c) assist or restore the calf muscle-pump function (CMPF). Graduated compression garments are available in different sizes and with various pressure gradients.

Compression garments may also exert mechanical pressure, either intermittently or continuously (Brennan and Miller, 1998; Blecken *et al.*, 2005). Intermittent pneumatic pressure pumps usually exert pressures between 30 - 80 mmHg (Partsch, 2003). Other forms of compression therapy include orthotic devices and bandages (Choucair and Phillips, 1998; Bergan and Sparks, 2000; Blecken *et al.*, 2005).

#### **D. EXTERNAL COMPRESSION MECHANISM**

Various therapeutic modalities, as mentioned in chapter three (*page 58 to 73*), are questionable treatments for post-exercise recovery. Compression therapy, on the other hand, may possibly be a cost-effective and practical method to aid post-exercise recovery and performance (Kraemer *et al.*, 2004; Barnett, 2006). One could hypothesize that compression accelerates effective recovery, thereby allowing more training bouts and, as Sanchez *et al.* (2006) suggested, the more the athlete runs during training, without overtraining, the lower the risk of injury on the race day. Kraemer *et al.* (2004) suggested that compression is a realistic approach in aiding recovery, limiting strength loss, reducing muscle damage and the

perception of muscle soreness. However, compression as a possible post-exercise recovery strategy has received little attention in scientific research.

It has been shown in previous studies that compression garments assist in reducing perceived pain (Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>), minimize peripheral swelling and inflammation associated with muscle damage (Gniadecka *et al.*, 1998; Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>; Kraemer *et al.*, 2004; Trenell *et al.*, 2006); enhance peripheral circulation and increase venous return (Partsch, 2003; Ali *et al.*, 2007; Duffield and Portus, 2007), support scar tissue healing (Kraemer *et al.*, 2001<sup>b</sup>; Kraemer *et al.*, 2004), enhance blood lactate clearance (Berry and McMurray, 1987; Chatard *et al.*, 2004; Rimaud *et al.*, 2007), reduce muscle oscillation (Bringard *et al.*, 2006<sup>b</sup>) and improve proprioception in injured joints as a consequence of eccentric exercises (Kraemer *et al.*, 2001<sup>b</sup>; Kraemer *et al.*, 2004). Furthermore, compression garments have been shown to reduce markers of muscle damage (Kraemer *et al.*, 2001<sup>b</sup>; Doan *et al.*, 2003; Gill *et al.*, 2006; Trenell *et al.*, 2006) and maintain functionality of the muscle (Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>).

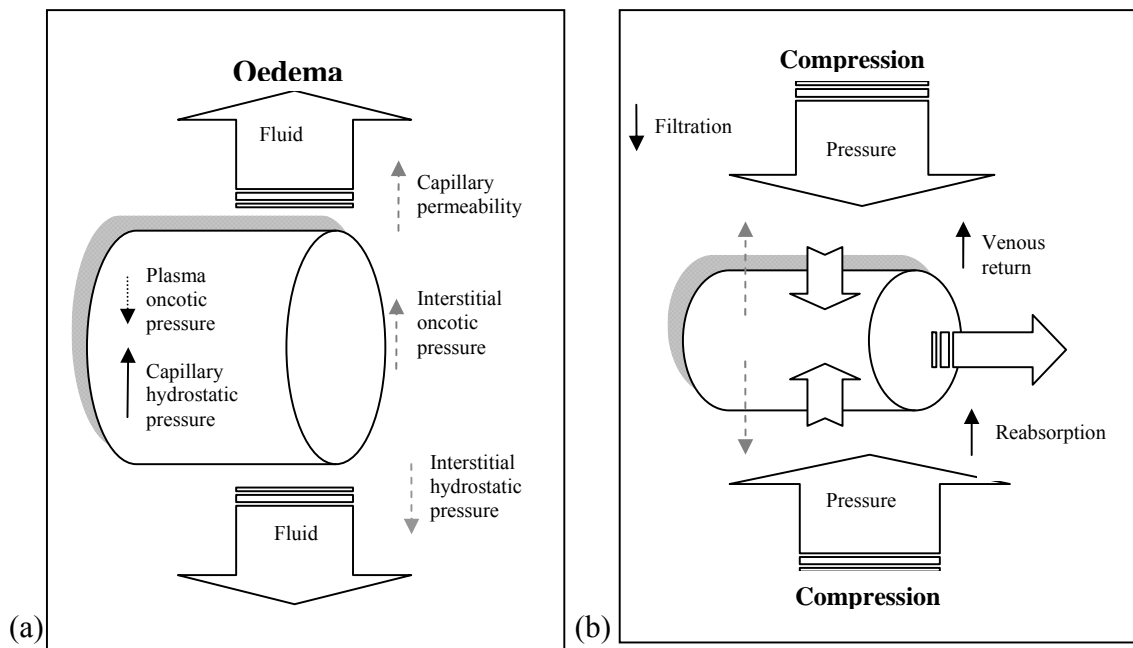


Figure 3. (a) Factors involved in the formation of oedema. (b) Compression mechanism working against filtration and enhances reabsorption of fluid.

It is believed that compression influences the lymphatic system by creating an external pressure gradient favouring oedema attenuation through skeletal muscle action, local pressure gradients and the influence of gravity (Kraemer *et al.*, 2004). Kraemer *et al.* (2001<sup>b</sup>) suggested that the pressure that the external compression creates and stimulates mechanical as well as chemical processes, consequently lessening muscle damage. Both of these mechanisms are supported by various studies.

The compression mechanism is very simple and may be explained by the Starling equation which states that the filtration force ( $F$ ) is equal to the filtration coefficient ( $c$ ) times the pressure difference between the capillary ( $P_c$ ) and tissue ( $P_t$ ) as well as the difference in oncotic pressure of the capillary ( $\pi_c$ ) and tissue ( $\pi_t$ ) (Partsch, 2003). Due to an oncotic pressure gradient that exists across the semi permeable capillary wall, water is drawn across the membrane until the concentrations on both sides of the wall is equal.

The amount of lymph fluid that is drained depends on (i) the permeability of the capillary wall, (ii) the hydrostatic pressure gradient and (iii) the oncotic pressure gradient between the blood and tissue. The hydrostatic pressure difference causes filtration and the oncotic pressure causes reabsorption. According to Starling's equation, external compression acts against the forces of filtration by increasing the local pressure and therefore assists reabsorption of fluid into the veins and the lymph system (*Figure 3b*)(Partsch, 2003). Thus, compression of various pressures influences the volume of the various vessels in the circulation system, i.e. the veins, arteries, and the lymphatic system (Partsch, 2003).

### **1. Reduced swelling and muscle injury**

Prentice (2001) consider compression the single most important technique for controlling swelling. Research indicates that external compression therapies, such as compression garments (20 to 60 mmHg) are one of the most common methods to treat individuals with lymphoedema. These patients experience, among other problems, pain and impaired function in the affected limb. This is due to an abnormal accumulation of tissue proteins, oedema, and chronic inflammation in the extremity. Compression lessens the formation of oedema and removes excess lymph fluid accumulated within the limb (Brennan and Miller, 1998; Harris, *et al.* 2001).

The roles of the lymphatic system are to collect and remove fluid from the interstitial tissues and then return it to the vascular compartment of the veins (Cho and Atwood, 2002; Partsch, 2003). Swelling reduction requires the removal of debris and fluid via the lymphatic system. Thus, the increase in local blood flow alone will not reduce oedema. The lymphatic system requires muscle action and gravity to move fluid contents and is independent on changes in the circulation (Cochrane, 2004). Partsch (2003) believes that the compression garments reduce oedema by the attenuation of the tissue's lymphatic fluid and not because it accelerates the lymphatic drainage (Partsch, 2003).

In the upright position, as in running, a healthy individual's calf muscle reduces the hydrostatic shifts (filtration) of the venous blood in the lower extremities, thus reducing oedema (McArdle *et al.*, 2001). Previous studies have speculated that the oedema, caused by the difference in osmotic gradient, is counteracted due to an increase in the tissue hydrostatic pressure from the mechanical pressure applied by the compression garment. The increase in tissue hydrostatic pressure, as well as by minimizing the available space for fluid accumulation (*Figure 3a and b.*), may stop or reduce the swelling. Furthermore, it may decrease haemorrhage and haematoma formation and enhanced lymph drainage (Brennan and Miller, 1998; Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>; Prentice, 2001; Kraemer *et al.*, 2004).

As pointed out in the section on recovery (*page 47*), reduced oedema is accompanied by decreased inflammation, less inflammatory markers and a lower perception of pain. Plasma creatine kinase concentration (CK<sub>p</sub>) is attenuated, either because of an increased creatine clearance, or because less creatine kinase is released by the muscle fiber (Kraemer *et al.*, 2004; Trenell *et al.*, 2006). In other words, by preventing or reducing oedema with external compression, circulating CK<sub>p</sub> is reduced and therefore muscle function is maintained (Kraemer *et al.*, 2004; Trenell *et al.*, 2006).

Standing for hours (~ eight hours) or long periods of sitting, generally results in oedema, muscle fatigue, discomfort in the lower extremities and it negatively affects venous function. These problems are mainly due to orthostatic stress, such as an increase in capillary hydrostatic pressure and gravity (Hirai *et al.*, 2002; Bringard *et al.*, 2006<sup>a</sup>). Kraemer *et al.* (2000) assessed the physiological responses in healthy women ( $n = 12$ ;  $23.0 \pm 2.1$  years) while

wearing various light to moderate graduated compression garments. The same subjects participated in four identical eight-hour standing protocols, separated by seven days. In one of the four sessions, the subject acted as her own control.

While standing on a hard surface, subjects had to perform various tasks such as carrying a 4.54 kilogram (kg) plate held overhead for fifteen minutes, playing cards and board games, watching videos for eight hours. A 30-minute seated lunch break was allowed with a standardized meal and fluid consumption. Each session took place at 22°C room temperature on the same hard surface and all the subjects wore the same shoes (3.81 centimeter (cm) heel).

Body mass and water content significantly increased throughout the day in all the conditions, possibly due to fluid and food intake. Average heart rate did not differ between those wearing compression garments (WCG) and those who did not wear compression garments (WOCG). There were significant reductions in both ankle (WCG<sub>all</sub>:  $\sim 2.0 \pm 0.2$  mm vs. WOCG:  $\sim 4.0 \pm 0.9$  mm;  $P \leq 0.05$ ) and calf circumferences (WCG<sub>all</sub>:  $\sim 3.7 \pm 0.9$  mm vs. WOCG:  $\sim 6.8 \pm 1.0$  mm;  $P \leq 0.05$ ). However, no statistically significant difference was noted in the thigh ( $P > 0.05$ ). The data also indicated a significant reduction in all of the garments for the popliteal diameter (WCG<sub>All</sub>  $\sim 0.7$  mm<sup>2</sup> vs. WOCG:  $\sim 2.25$  mm<sup>2</sup>;  $P \leq 0.05$ ) and posterior tibial vein (WCG<sub>All</sub>:  $\sim 0.126$  mm<sup>2</sup> vs. WOCG:  $\sim 0.276$  mm<sup>2</sup>;  $P \leq 0.05$ ). A reduction in the size of the veins indicates a reduction in venous pooling.

There was a significantly lower perceived muscle soreness rating in the lower limb in subjects wearing the compression garment (WCG<sub>A</sub>:  $\sim 232$  vs. WOCG:  $\sim 455$  rating;  $P \leq 0.05$ ). Blood creatine kinase concentration was significantly reduced with all three compression garments (WCG<sub>All</sub>:  $\sim 24.3$  % vs. WOCG:  $\sim 14$  %;  $P \leq 0.05$ ), as well as plasma volume (WCG<sub>All</sub>:  $\sim 8$  % vs. WOCG:  $\sim 5$  %;  $P \leq 0.05$ ) over the eight hours. Vertical jump performance was not improved by wearing compression garments over the eight-hour trial. This study shows that compression garments may reduce oedema in the ankles and legs, as well as venous pooling and may cause less perceived discomfort in the muscles. These findings also suggest that the type of garment, as well as the specific pressure should be carefully considered.

Kraemer *et al.* (2001<sup>a</sup>) demonstrated that compression garments (*Raschell* fabric and 25 % Lycra; 10 mmHg) aid recovery of soft tissue damage. Twenty non-strength trained healthy

women ( $21.3 \pm 2.9$  years WCG and  $21.1 \pm 3.3$  years WOCG) were matched according to age, anthropometric data, and one repetition maximum (1 RM) concentric arm strength. They were randomly assigned to a control ( $n = 10$ ; no intervention (WOCG)) and an experimental group ( $n = 10$ ; with compression sleeves (WCG)). Subjects were requested to perform two sets of 50 passive elbow flexor exercises (Biodex System 2 isokinetic dynamometer). To inflict DOMS, every fourth repetition was a strenuous eccentric repetition muscle action. After each set the subject rested for three minutes. Immediately after the eccentric protocol the compression garment was applied to the exercised arm and worn for 120-hours post-exercise. The subjects were allowed to take the garment off once a day when bathing, and to collect measurements.

Plasma creatine kinase activity was significantly lower from day two to five in the recovery period in those subjects wearing compression garments (~20 % increase), compared to the subjects not wearing any compression garments (~117 %;  $P \leq 0.05$ ). No significant changes were observed in cortisol and LDH levels ( $P > 0.05$ ). A significant improvement in force production (power and strength) was observed in the WCG group from day three to five, compared to the WOCG group ( $P \leq 0.05$ ). Subjects in the experimental group also maintained their elbow flexibility, showed significantly reduced circumference measurements, and perceived muscle soreness ( $P \leq 0.05$ ). The relaxed elbow angle in those subjects WCG was unchanged, while the WOCG group demonstrated a significant flexion ( $P \leq 0.05$ ). This was consistent with the reduced circumference measurements thus indicating a reduced swelling response in the WCG group.

In a different study by Kraemer *et al.* (2001<sup>b</sup>) non-strength trained men performed two sets of 50 arm curls (1 RM elbow flexion at  $60^\circ/s$ ) and were then matched and randomly divided into two groups ( $n = 8$  WCG and  $n = 7$  WOCG). The continuous compression sleeves (*Raschell* fabric and 25 % Lycra) exerted a pressure of 10 mmHg. They followed the same protocol as in the previous study of Kraemer *et al.* (2001<sup>a</sup>), but post-exercise follow up was limited to a 72-hour recovery phase.

Plasma creatine kinase activity was significantly increased in both groups at 72-hours post-exercise (~490 vs. ~1390 u.L<sup>-1</sup>;  $P \leq 0.05$ ), however the increase was significantly less in the compression sleeve group. Furthermore, both groups showed reduced elbow extension flexibility in the three days post-exercise ( $P \leq 0.05$ ). However, the compression sleeve group

(WCG) had significantly less elbow extension stiffness at 48-hours (WCG:  $\sim 158^\circ$  vs. WOCG:  $\sim 153^\circ$ ) and 72-hours (WCG:  $\sim 158.5^\circ$  vs. WOCG:  $\sim 154.5^\circ$ ) post-exercise, compared to the WOCG group. Biceps circumferences were significantly increased in the WOCG group at 24 hours (WCG:  $\sim 0.52$  cm vs. WOCG:  $\sim 0.65$  cm;  $P \leq 0.05$ ) and 48-hours (WCG:  $\sim 0.4$  cm vs. WOCG:  $\sim 0.9$  cm;  $P \leq 0.05$ ). The swelling in the subjects' upper arm were less in the WCG group at 24 and 48 hours (hrs), but it was only significantly reduced at 48-hours post-exercise ( $\sim 0.4$  cm vs.  $\sim 0.98$  cm increase;  $P \leq 0.05$ ).

Both groups reported a greater perception of muscle soreness throughout the study. During active range of motion, there was a significant increase in pain in both groups from pre-exercise levels, but significantly less perceived soreness in those subjects WCG, between 60 and 72-hours post-exercise (60hrs:  $\sim 22$  vs.  $\sim 30$  ratings; 72 hrs:  $\sim 21$  vs.  $\sim 30$  ratings;  $P \leq 0.05$ ). At 72-hours all ratings of perceived muscle soreness were less in the WCG group compared to the WOCG group ( $P \leq 0.05$ ). Furthermore, peak-force and power production was not significantly altered in the compression garment group after the protocol. There were indications that the WCG-group had a faster recovery of force production during the one RM elbow flexion test.

In chapter three (*pages 60 to 61*) a study by Gill *et al.* (2006) was briefly reviewed, which investigated the effects of four post-match recovery techniques on plasma creatine activity in 23 elite male rugby players. Compression garments were amongst the four strategies monitored over a four-week period in the competition season. Subjects were randomly assigned to one of the four recovery techniques (i.e. compression garments, active, passive, and contrast water therapy).

Interstitial creatine kinase samples were collected three and a half hours prior to matches and then immediately after and at 36 and 84-hours post-exercise. Unlike most of the other studies on compression garments, the compression garment was not worn immediately after or during the match. Players went about their normal post-match routines before applying the compression garment. The garment was then worn for approximately twelve hours.

Interstitial creatine kinase activity was significantly greater after matches ( $1023.0 \pm 308.3$  u.L<sup>-1</sup> vs. post:  $2194 \pm 833.7$  u.L<sup>-1</sup>;  $P < 0.01$ ). This indicates that muscle damage occurred. When



compression garments (WCG) were compared to active (ACT), passive (PAS), and contrast water (CWT) recovery-techniques after a game, no significant differences were noted between these recovery modalities overall. However, there was a significant difference ( $P < 0.05$ ) between passive recovery and in the individuals wearing compression garments at 36 and 84-hours post-exercise. The subjects with the compression garments showed an 84.4% improvement in muscle damage while passive recovery only indicated a 39.0% improvement at 84-hours post-match. Even though not significant (NS; ~ 67% CG; ~ 59 % ACT; ~ 55% CWT and ~ 28% PAS), compression garments had a greater recovery response at 36-hours post-match than all of the other strategies. However, when compression garments were compared to the contrast (85.0% recovery) and active recovery (88.2% recovery) modalities at 84-hours, compression garments yielded a lower recovery response (Gill *et al.*, 2006).

The creatine kinase activity levels were far greater in the study by Gill *et al.* (2006) than usually observed in studies. Possible reasons for this may be that interstitial creatine kinase concentration ([CK]) was assessed (which may be more sensitive than plasma [CK]), methodological differences and incomplete recovery of the players after matches or training.

### 1.1 Conclusion

All of the above studies suggest that compression garments reduce muscle soreness. In three of the four studies the compression garments showed a significant reduction in the swelling of the extremities over the recovery period, compared to those individuals not wearing the compression garments. This could be contributed to either the decreased release of muscle damage markers, for instance CK from the ultrastructure of myocytes, or the aided clearance of myofibrillar proteins from the injured area. Blood creatine kinase concentration was significantly attenuated after exercise in all of the studies. In the three studies that assessed plasma creatine kinase, the reduced CK<sub>p</sub> levels were associated with significantly lower ratings of perceived muscle soreness.

Furthermore, in two of the studies by Kraemer *et al.* (2000 and 2001<sup>b</sup>) force production (power and strength) by the individuals wearing compression garments did not show significant changes. However in Kraemer *et al.* (2001<sup>a</sup>) significant improvements in force production were reported when subjects wore compression garments. Similarly Kraemer *et al.*

(2001<sup>a</sup>) also found an improvement in flexibility, but this was not supported by the other studies. When contrast water and active recovery were compared to compression therapy, no significant differences were found between the modalities. However, compression garments increased recovery after a match faster than passive recovery.

## **2. Improved venous function and microcirculation**

When a healthy individual stands upright, the venous pressure is about 80 – 100 mmHg and the blood flows slowly through the veins. However, when an individual starts to walk the venous pressure drops to between 10 – 20 mmHg in the foot (Partch, 2003). This is due to the calf and foot muscle-pumps that accelerate the blood flow in the veins, thus preventing blood pooling. In addition, when compression exerts pressure on the lower limb, the pressure reduces the diameter of the vein vessel. For instance, a pressure of 15 mmHg exerted on the limb results in a 20% reduction of the venous luminal diameter (Agu *et al.*, 1999). This also causes a significant acceleration in both superficial and deep venous blood flow, which shunts the blood away from the periphery towards the heart (Agu *et al.*, 1999; Partch, 2003). The heart's preload is enhanced and may increase the cardiac output by about 5% (Partch, 2003). Compression garments function in a similar way than the calf muscle-pump function. The only requirements for the compression garments to increase venous blood flow are that arterial flow must be maintained and sufficient pressure needs to be applied (Agu *et al.*, 1999).

Previous research on conditions such as venous insufficiency, showed that the compression garments' ability to improve the venous return function is probably related to the reduction of blood pooling in the lower limbs of these patients, as well as the faster clearance of muscle damaging and metabolic by-products (Berry and McMurray, 1987; Agu *et al.*, 1999; Kraemer *et al.*, 2001<sup>b</sup>; Doan *et al.*, 2003). Compression garments may improve the femoral blood flow velocity by 138 % in healthy recumbent subjects and decrease venous pressure from 65 mmHg to 25 mmHg while standing upright (Berry and McMurray, 1987).

More recent research is consistent with the findings of previous studies and agrees that elastic compression garments enhance venous function (Chatard *et al.*, 2004; Trenell *et al.*, 2006; Rimaud *et al.*, 2007). By applying mechanical compression to the extremities, the pressure theoretically increases the intramuscular pressure, thereby reducing venous transmural

pressure and also obstructing the blood flow in the cutaneous veins or limiting venous wall expansion of the lower extremities (*Figure 3a and b*). This prevents cutaneous venous pooling and the shunting of blood to the deeper femoral veins, thereby increasing blood flow back to the heart (Iwama *et al.*, 2000; Chatard *et al.*, 2004; Trenell *et al.*, 2006; Maton *et al.*, 2006; Rimaud *et al.*, 2007). In addition, Rimaud *et al.* (2007) suggested that refilling rates and ejection fractions of the heart may also be enhanced and venous pressure reduced by compression garments.

It has also been speculated that compression garments could enhance the calf muscle-pump function (CMPF) during rest and exercise. Furthermore, the garments may improve venous valve function in the case of valve insufficiencies (Maton *et al.*, 2006; Rimaud *et al.*, 2007). Maton *et al.* (2006) explains that the compression garment acts like an unyielding fascia in the muscle during dynamic activities. This improves the efficiency of the calf muscle-pump as explained in chapter two (*page 21*). This mechanism supports venous return and prevents blood pooling. It works in a similar fashion to pressurized antigravity suits worn by pilots (Rimaud *et al.*, 2007).

Iwama *et al.* (2000) investigated the effect of intermittent pneumatic compression therapy on the lower extremity's peripheral circulation. The study assessed the bilateral deep plantar and tympanic temperature, average blood pressure, and heart rate at 15-minute intervals. Deep plantar temperature reflected the lower limb's peripheral circulation. Subjects included in the study were adult patients ( $n = 70$ ; 20 – 85 year old) who had received major gastrointestinal surgery one day before the experiment. Subjects with unsteady circulatory parameters or signs and symptoms of deep vein thrombosis or arteriosclerosis obliterans were excluded. At 09:00 on the day following surgery, a single-pulse calf-length compression garment was fitted to both legs. The subject was requested to lie in the supine position and the thermometers were placed on both plantae. After 30-minutes when deep plantar temperature reached steady state, the experiment started. The intermittent pneumatic compression (IPC) device connected to the garments applied 40 mmHg for 150 minutes at a cycle of 10 seconds of compression and 50 seconds of decompression. Only one of the calves, which were randomly selected, received IPC treatment and the other acted as a control.

A total of 31 left and 39 right calves received IPC treatment. The tympanic temperature, heart rate and mean arterial blood pressure did not change during the course of the experiment. However, there was a significant  $\sim 1.6^{\circ}\text{C}$  increase in deep plantar temperature between the calf receiving compression therapy and the control calf ( $P < 0.05$ ). Temperatures increased by almost  $\sim 5\%$  in the calf receiving IPC treatment. The authors concluded that compression garments enhanced the peripheral circulation. The evidence for this was that no circulatory changes were observed and higher temperatures were found in the compressed calf.

Iwama *et al.* (2000) proposed another possible mechanism by which intermittent pneumatic compression (IPC) prevents deep vein thrombosis. They suggested that the femoral vein blood flow is augmented by the function of nitric oxide (NO) synthesis. As mentioned in chapter two (page 22), nitric oxide production is stimulated by venous compression and causes the vascular endothelium to vasodilate. Furthermore, NO also increases fibrinolytic activity (Iwama *et al.*, 2000; Dai *et al.*, 2002). This prevents blood pooling in the veins of the lower extremities, increases venous return to the essential veins, and impedes thrombosis (Iwama *et al.*, 2000). In animal studies mentioned by Dai *et al.* (2002), external pneumatic compression caused vasodilation in arteries and veins possibly due to NO stimulation. This vasodilation effect increases the peripheral and central circulation (Dai *et al.*, 2002). This could also be a possible mechanism of graduated compression garments, and needs further investigation.

Ali *et al.* (2007) assessed the physiological and perceptual effects of knee-length graduated compression garments (18 to 22 mmHg) in two randomized counterbalanced experiments, i.e. intermittent and continuous trials. Both of these experiments were repeated after at least three days. The objective of this study was to determine whether compression garments worn throughout a running bout, similar to a training bout, would affect the subsequent recovery period. Subjects randomly wore a compression garment (WCG) with a normal sport sock (no compression ankle-height) or just a normal sport sock in the control group (WOCG).

The first experiment was the intermittent running trial. Subjects were recreational athletes ( $n = 14$ ; age:  $22 \pm 0.4$  years;  $\text{VO}_{2\text{max}}$ :  $56.1 \pm 0.4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) and had to participate in at least two running sessions of 30 to 60 minutes per week and/or in running activities like soccer, tennis, and rugby. This experiment consisted of two multi-stage shuttle runs with a one-hour recovery period between each run. Subjects had to complete as many 20-meter shuttle runs as they

could. The initial shuttle was at a slow pace ( $2.22 \text{ m}\cdot\text{s}^{-1}$ ) and the pace progressively increased by  $0.14 \text{ m}\cdot\text{s}^{-1}$  every 60 seconds. If subjects failed to reach the end of the 20-meter line before the next beep (audible signal), they had to stop.

Subjects wore the compression garment before the first shuttle run and removed it after all the measurements were collected at the end of the second shuttle run. The authors only wanted to assess the effects on recovery when graduated compression garments were worn during exercise. Thus, the garments were removed after the second bout and not worn during the 24-hour post-run assessment.

In the second experiment, another group of subjects, similar to those in the intermittent trial, completed a continuous protocol ( $n = 14$ ; age:  $23 \pm 0.5$  years;  $\text{VO}_{2\text{max}}$ :  $55.0 \pm 0.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ). These subjects had to perform a fast paced continuous ten-kilometer (km) road run. Prior to the main 10-km run subjects had to complete a 10-km tarmac route once. These times were then used to determine the running pace during the main run. The athlete's pace was regulated by frequent time checks and with an investigator cycling next to the athlete.

Unfortunately, the intermittent protocol did not succeed in causing muscle damage and the authors speculated that the protocol was not long enough. There were no difference in total distance completed between subjects wearing compression garments and those not wearing compression garments ( $2213 \pm 77$  meter (m) vs.  $2272 \pm 75$  m (first bout) and  $2247 \pm 84$  m vs.  $2225 \pm 67$  m (second bout)). Mean heart rate and maximum heart rate did not differ during the experiment or between those WCG and WOCG. Perceived soreness, RPE and the comfort-and-feel rating of the compression garments did not change throughout the experiment and were not different between WCG and WOCG.

In the second continuous exercise bout, no difference was found in RPE ratings between those subjects WCG ( $17 \pm 0.5$  rating) and WOCG ( $17 \pm 0.5$  rating), and the comfort-and-feel ratings of the compression garments were the same as in experiment one. Perceived muscle soreness was significantly ( $3 \pm 0.6$  rating vs.  $5 \pm 0.4$  rating;  $P < 0.05$ ) lower at 24-hours after the run in the WCG group. A difference was also noticed in the frequency and location of soreness in those with compression garments and those without. A higher running pace (44.7 minutes (min) vs. 45.0 min;  $P = 0.15$ ) and lower heart rate ( $\sim 176$  bpm vs.  $\sim 180$  bpm;  $P = 0.15$ ) were

noticed in runners wearing compression garments, although the differences were not statistically significant. The authors speculated that the lower heart rate might be an indication of improved venous return.

Bringard *et al.* (2006<sup>a</sup>) investigated the effects of compression garments (CG) compared to Lycra<sup>®</sup> elastic tights (ET) and no compression shorts (C) on calf muscle oxygenation and venous pooling in resting conditions in twelve endurance trained men (age  $26.5 \pm 2.6$  years). Subjects' tissue oxygenation index, deoxyhemoglobin and the right *Gastrocnemius Medialis*' blood pooling were monitored continuously by two-hertz (Hz) near-infrared spectroscopy in supine and standing positions. The protocol stated that subjects had to lay supine and stand for five minutes in each position while alternating the different garments in one single session, under a constant room temperature. The compression garment's pressure was also assessed with a homemade transducer and the mean pressure over the calf was 5.6 (ET) and 23.2 mmHg (CG) in the supine position and 5 mmHg (ET) and 24.1 mmHg (CG) during standing. This indicated that there was a significant difference in the exerted pressures of the different clothing (CG>ET>shorts).

The tissue oxygenation index was significantly higher, and deoxyhemoglobin and *Gastrocnemius Medialis*' blood pooling were significantly lower ( $P < 0.001$ ) in CG compared to ET and were similar for the supine vs. standing positions. The compression garments thus had a positive effect on the athlete's calf muscle oxygenation and venous stasis during rest in supine and standing positions, compared to the ET. The improved oxygenation in the muscle might be due to several possible mechanism, such as (i) increased blood flow in the capillary bed, this supplies more oxygen, (ii) better perfusion of the muscle which would mean that the muscle utilized oxygen more efficiently and then (iii) changes in skin blood flow which could lead to an enhanced oxygenation in the muscle. The authors recommended that compression garments be used post-exercise to oxygenate the muscles. This might reduce recovery time from fatigued muscles as well as enhance lactate clearance.

Compression accelerates blood flow in the microcirculation, favours white cell detachment from the endothelium, and prevents further adhesion. As mentioned before in *Section D* of this chapter (*page 78*) the capillary filtration is reduced and reabsorption is increases due to the

applied mechanical pressure. Compression therapy accelerates blood flow by increasing this pressure gradient (Dai *et al.*, 2002; Partsch, 2003).

When comparing the compression shorts with the elastic tights and the standard shorts (Bringard *et al.*, 2006<sup>a</sup>), the results indicate statistically significant differences for all measures ( $P < 0.001$ ), except HbO<sub>2</sub>. Those subjects wearing compression shorts demonstrated lower deoxyhaemoglobin. This indicates that the compression garment did reduce blood pooling during both body positions. The authors speculated that the drop in total hemoglobin might be due to a reduction in venous compliance when the subjects wore compression tights. The significance of this is that venous return will improve, thus leading to an enhanced cardiac output.

## 2.1. Conclusion

All of the studies showed that compression garments tend to improve venous return as well as peripheral circulation. In one of the studies (Bringard *et al.*, 2006<sup>a</sup>) a reduced blood pooling and increase oxygenation to the calf muscles were demonstrated when the compression garments were worn. However, this was a resting protocol, which eliminated the effect of the calf muscle-pump function. Iwama *et al.*, (2000) established that deep plantar temperature significantly increased when wearing compression garments, and suggested that this is evidence for improved peripheral circulation. Conversely, Ali *et al.* (2007) found that compression garments reduced their subjects' heart rate and improved their running pace, but this was not statistically significant. The methodology differs between all three of these studies, with regards to subjects and protocols that were used. Thus, more research is needed to confirm that compression garments will aid venous return and increase blood flow and distribution to the periphery after exercise, especially during prolonged exercise.

## 3. **Enhanced metabolic recovery**

Very little is known about the possible effects of compression garments on intramuscular metabolic functions (Trenell *et al.*, 2006). For instance, factors associated with muscular fatigue and pain, such as intracellular lactate and inorganic phosphate accumulation, as well as

acidosis as a result of anaerobic breakdown of glycogen, have not been studied (Maton *et al.*, 2006).

It is known that lactate exchange and clearance is associated with performance and that reduced lactate concentration depends on several factors, such as a (i) reduced lactate production; (ii) increased plasma volume and/or (iii) altered lactate kinetics. Two possible ways to change lactate kinetics is by either quickly clearing lactate via a fast circulation and thereby increasing lactate oxidation or preserving the lactate within the muscle and not allowing it to diffuse from the muscular bed (Berry and McMurray, 1987; Chatard *et al.*, 2004; Maton *et al.*, 2006).

Consequently, an increased blood flow will assist clearance of metabolic by-products, thus enhancing muscle recovery and reducing fatigue. As mentioned before, active recovery increases blood flow when compared to passive recovery (Chatard *et al.*, 2004). In a similar fashion, the calf muscle-pump function increases blood flow. If compression therapy successfully enhances the function of the calf muscle-pump, this would further increase the restoration of muscle metabolism after exercise, thus aiding recovery and reducing muscle fatigue (Maton *et al.*, 2006; Rimaud *et al.*, 2007). However, other authors like Berry and McMurray (1987) support the theory that compression garments inhibit the release of lactate from the muscle.

Twenty years ago Berry and McMurray (1987) were one of the first researchers to investigate the effects of wearing compression garments on lactate recovery. They hypothesized that the use of compression garments would either (i) lower the production of lactate, (ii) reduce the lactate retained within the muscle, or (iii) improve the blood flow and thus clear and oxidize lactate more efficiently. In their study, well-trained healthy male subjects ( $n = 12$ ) performed two different experiments. The two experiments were completed as a separate but related study.

In the first experiment investigators wanted to establish if compression garments have an effect on maximum oxygen consumption ( $VO_{2max}$ ), time to exhaustion (TTE) and lactate recovery, by using a treadmill  $VO_{2max}$  test. The subjects ( $n = 6$ ; age:  $22.5 \pm 5.4$  years;  $VO_{2max}$ :  $52.8 \pm 8.0$  mL.kg<sup>-1</sup>.min<sup>-1</sup>) performed the same protocol on two separate visits to the laboratory,



once with graduated compression stockings (WCG) and once with no garment (WOCG). The compression garments provided graduated compression, which was the highest at the ankle (18 mmHg) and lowest just below the knee (8 mmHg). During the recovery period the subject walked for five minutes at a speed of ~5.63 kilometers per hour; thereafter the subject rested for 55 minutes in a seated position.

Blood samples to assess the lactate concentration and haematocrit levels were collected at rest and at five, 15, 30, 45 and 60-minutes post-exercise. Oxygen uptake was measured at rest and during the test at the last minute of the 15-minute steady state exercise, in each of the two-minute intervals during the second minute and during the last minute of the test. Furthermore, oxygen uptake was also assessed during the first five minutes of the recovery period and then at 15, 30, 45 and 60 minutes post-exercise. The reason behind this was that if lactate levels were reduced significantly together with increased oxygen consumption post-exercise in the subjects WCG, this would indicate that the CG improved blood flow to the active muscles. The lactate concentration is therefore cleared via the accelerated venous return velocity.

There were no significant differences in  $VO_{2max}$  or TTE between the WCG and WOCG trials. The compression garments caused no difference in oxygen uptake recovery for 60-minutes post-exercise. Although lactate concentrations were lower, throughout the entire 60-minute recovery period, in those subjects WCG, only the values at 15-minutes post-exercise was significantly lower (~ 30.6 %) compared to those WOCG. Plasma volume only demonstrated small, but not significant changes.

In the second experiment, investigators studied the effect of compression garments on lactate retention. The subjects (age:  $21.4 \pm 4.3$  years;  $VO_{2max}$ :  $59.9 \pm 6.8$  mL.kg<sup>-1</sup>.min<sup>-1</sup>;  $n = 6$ ) visited the laboratory on four separate occasions. The first visit was a baseline visit to obtain the subjects'  $VO_{2max}$  and to familiarize them with the protocol. During visits two to four the participants cycled on an ergometer for three minutes at 110% of their  $VO_{2max}$ . Afterwards subjects rested for 30-minutes in a supine position. The only difference between visit two to four was that subjects randomly wore graduated compression stockings. Subjects either wore CG (i) during the exercise (WCG) or (ii) during the exercise and recovery period (WCG<sub>exercise</sub>) or (iii) not at all (WOCG). Oxygen consumption was measured 5-minutes prior to exercise and

during recovery. Blood samples were collected for lactate and haematocrit levels prior to and five, 15 and 30 minutes post-exercise.

There were no significant differences in post-exercise  $\text{VO}_2$  in any of the three trials. However, subjects WOCG tended to have the highest post-exercise oxygen consumption. There were also no significant differences in plasma volume between the groups. The mean lactate concentration was significantly lower in the subjects WCG during the exercise and recovery period (WCG:  $47.0 \pm 13.79$  vs.  $\text{WCG}_{\text{exercise}}$ :  $57.6 \pm 20.17$  and WOCG:  $53.5 \pm 18.54$  mmol;  $P < 0.05$ ). When the compression garment was removed during recovery ( $\text{WCG}_{\text{exercise}}$ ), there was a  $\sim 53\%$  increase in lactate levels in the post-exercise period.

As mentioned before, the two proposed lactate-clearance mechanisms of the compression garment is by (i) shunting the blood away from the muscle so that the diffusion medium is removed, thus lactate stays in the muscle or (ii) the venous blood flow is increased, thus clearing and oxidizing lactate faster. This study provided evidence to suggest that blood lactate concentrations were reduced when wearing compression garments due to the metabolite being preserved intramuscularly and that lactate was not cleared. The compression garment created an inverse pressure gradient or increased the intramuscular hydrostatic pressure, which retained the lactate in the muscle. The evidence for this is that when the garment was removed during the recovery period, the lactate concentration increased. In addition, the oxygen uptake during experiment one would have increased post-exercise if the blood flow increased through the previously active muscle. However, experiment 1 did not show any significant increase in post-exercise oxygen uptake. Furthermore, both experiments indicated that the plasma volume was maintained in those athletes wearing the compression garments.

Chatard *et al.* (2004) found similar results as Berry and McMurray (1987). They tested twelve trained elderly cyclists (age:  $63 \pm 3$  years) for the effect of compression garments (Ganzoni-Sigvaris; 60% polyamide and 40% Lycra) on the recovery of muscle soreness in the lower limbs and the effect on performance. Subjects performed two five-minute maximal exercise bouts, twice a week, for two consecutive weeks on a cycle ergometer at 50 - 60 % of their PPO. Between the two maximal exercise bouts was an 80-minute recovery period, in which the subjects had to sit with their legs elevated. Therefore, all subjects performed four maximal

exercise bouts per week, with two rest days between the two tests. This was repeated for the second week.

Subjects randomly wore the elastic compression stockings (WCG) or not (WOCG), during the 80-minute recovery period. This meant that the subjects each completed two of the four sessions with compression garments, during the two weeks. The compression garment's pressure decreased by 40% from ankle to mid-thigh (33 mmHg (ankle); 18 mmHg (calf); 12.75 mmHg (mid-thigh)). The subjects' perceptions of leg sensations were assessed during the 80-minute recovery period. RPE did not differ significantly between the test weeks meaning that the exertion during all of the maximal bouts was the same.

After both conditions, i.e. with compression garments and without, the maximum power maintained ( $249 \pm 19$  W vs.  $154 \pm 17$  W;  $P < 0.01$ ), heart rate (second:  $152 \pm 11$  bpm vs. first:  $155 \pm 11$  bpm) and blood lactate concentration (second:  $10.6 \pm 2.2$  first:  $11.5 \pm 2.2$  mmol.L<sup>-1</sup>) were statistically significantly lower during the second bout, compared to the first bout. However, those subjects that wore compression garments had a  $2.1 \pm 1.4$  % ( $P < 0.01$ ) lesser reduction in maximum power in the second bout, compared to those WOCG.

The study demonstrated that subjects who wore the compression garment had significantly reduced blood lactate and haematocrit levels from 20 to 80 minutes after the two five-minute maximal exercises. In addition, those wearing compression garments had a 20 % lower lactate concentration than those without compression garments before the second bout. Furthermore, the blood lactate difference, before and after the second maximal bout, in subjects WCG, was higher than those WOCG, ( $8.8 \pm 2.0$  vs.,  $8.0 \pm 2.0$  mmol.L<sup>-1</sup>, respectively;  $P < 0.03$ ).

A relationship was shown between blood lactate clearance and an individual's  $VO_{2max}$  ( $r = 0.37$ ;  $P < 0.01$ ). This was found in all of the subjects and during the entire recovery period. In other words, the higher the subject's  $VO_{2max}$ , the more efficient or faster blood lactate is cleared. This supports the hypothesis that compression garments acts in a fashion similar to active recovery strategies, which would suggest that the compression mechanically increases the blood flow in the veins (*Figure 3b*). This would help to clear lactate from the muscles, and increases lactate oxidation. Furthermore, the increased blood lactate clearance might have

contributed to the higher maximum power during the second bout, indicating an improvement in anaerobic performance.

Subjective opinions of the cyclists indicated that the compression garments reduced perceived leg pain. Ten of the twelve subjects thought that the compression garments contributed to their performance. The investigators concluded that the subjects' performances, together with haematocrit concentrations and lactate removal were enhanced compared to the control group, after the recovery period.

Barnett (2006) reviewed popular recovery strategies in elite athletes and was unconvinced about the practicality and effectiveness of compression garments. He pointed out that passive recovery will also return lactate levels to pre-exercise levels before the following exercise bout, and that sitting with your legs elevated for 80-minutes is not regarded as a practical technique for elite athletes, as an in-between training recovery technique.

In a more recent study, Trenell *et al.* (2006) investigated the benefits of compression garments on the metabolic recovery of eleven recreational male athletes. The subjects (age:  $21.2 \pm 3.1$  years) performed a 30-minute downhill walk protocol ( $6 \text{ km.h}^{-1}$  at 25 % decline). Compression garments (76% Nylon and *Meryl Microfibre*, 24% *Roica Spandex*), covering the calf and thigh muscle of only one leg were worn immediately after exercise until 48-hours. Thus, each subject served as his own control. Muscle metabolites (phosphomonoester (PME), phosphodiester (PDE), phosphocreatine (PCr), inorganic phosphate (Pi) and ATP) and perceived muscle soreness were assessed pre-exercise, one-hour and 48-hours post-exercise.

Although there was a significant increase in the perception of muscle soreness in both legs after the exercise, there were no significant difference in perceived muscle soreness between the WCG and WOCG leg. A significantly lower pH was observed at one hour after the downhill walking exercise in both groups. However, no significant differences were found in the pH values between the two legs throughout the experiment ( $P > 0.05$ ). The leg with the compression garment compared to WOCG indicated an increase of [PDE] ( $\sim 0.653$  vs.  $0.550$ , respectively;  $P < 0.05$ ) in the thigh one-hour post exercise. The PCr/Pi relations reflected non-specific muscle damage, but in this study, no significant alteration in PCr/Pi,  $\text{Mg}^{2+}$ , or PME was found throughout the study in either of the legs ( $P > 0.05$ ).

This study showed that the compression garment had no treatment effect. Thus, the garments did not benefit metabolic recovery from eccentric exercise, although, the elevated [PDE] after the downhill protocol in the leg with the compression garment, could indicate an increased skeletal muscle membrane turnover. This may show a changed phospholipid metabolism, which is involved in the tissue regeneration process. Consequently, Trenell *et al.* (2006) suggested that the increased [PDE] indicates an accelerated inflammatory and repair process. It may also be argued that changes in [PDE] are due to under perfusion. Since vasoconstriction results in an increased [PDE], less oxygen is supplied to the muscle and protons are not cleared. In the end, this stress activates the break down of muscle fibers. Trenell *et al.* (2006) points out that elevated pH is associated with this proton retention and vasoconstriction. However, compression garments did not cause vasoconstriction, since both legs demonstrated an elevated pH after the exercise. They concluded that the 30-minute downhill walk did not significantly influence the creatine kinase equilibrium and it appears that compression garments improve the inflammatory responses to muscle damage and aid intramuscular repair. However, more research is needed to confirm this.

Interestingly Rimaud *et al.* (2007) performed a randomized study on fourteen well-trained subjects with spinal cord injuries (SCI), who were divided into two groups (low SCI  $n = 9$ ; age:  $36.9 \pm 11.79$  years and high SCI  $n = 5$ ; age:  $36.6 \pm 11.3$  years). Subjects had to complete two progressive incremental exercise tests to exhaustion on a wheelchair ergometer. These were separated by seven days. Both tests were similar, except that the subjects randomly performed the exercise either with a graduated compression garment or without. After the maximal exercise, a passive recovery period followed where the subjects rested in the wheelchairs.

The crossover design demonstrated that low pressure (21 mmHg) elastic graduated compression garments enhanced recovery after maximal wheelchair exercises in only low-level SCI. There was a twelve percent difference in the subjects' blood lactate concentrations with compression garments compared to the subjects without compression garments at three minutes after the exercise ( $10.9 \pm 3.9$  mmol.L<sup>-1</sup> vs.  $12.5 \pm 4.6$  mmol.L<sup>-1</sup>, respectively;  $P < 0.05$ ). Apparently, wheelchair athletes often strap their lower extremities to prevent venous pooling and enhance cardiac output (Rimaud *et al.*, 2007). However, in this study no

improvement was found in performance and cardiovascular responses with compression garments. They concluded that lactate recovery is accelerated in well-trained SCI individuals (low-level) wearing graduated compression garments after maximal exercises. Furthermore, the low pressure exerted by the garments might be responsible for the lack of improvement in the athlete's performance.

### 3.1 Conclusion

Studies have reported significant reductions in lactate, with associated lower haematocrit and heart rate. This suggests that improved blood flow aids the removal of lactate. Berry and McMurray (1987) had a different theory. They also showed lower blood lactate concentrations in athletes who wore compression garments. However, the researchers suggested that lactate was trapped inside the muscle due to the reverse pressure gradient created by the compression garment. The evidence for this was that when the garment was removed during the recovery period, the lactate concentration increased; also post-exercise oxygen consumption did not increase significantly in the recovery period.

Blood lactate concentrations were found to be lower in individuals with lower lesion spinal cord injuries, but performance was not enhanced. The researcher speculated that the pressure exerted by the compression garment was not enough to improve venous return (Rimaud *et al.*, 2007). Trenell *et al.* (2006) on the other hand, suggested that compression garments aid muscle repair, but found no significant evidence that it aids metabolic recovery.

## **4. Enhanced functional performance**

Muscle soreness and structural damage after strenuous eccentric exercise contribute to loss of muscle function (Kraemer *et al.*, 2001<sup>b</sup>; Bernhardt and Anderson, 2005). Kraemer *et al.* (2001<sup>b</sup>) observed that subjects with compression garments regained muscle strength quicker after exhausting elbow-flexion exercises. Furthermore, this was consistent with the improved recovery of damaged muscles in the subjects. It has also been shown that compression garments enhance repetitive jumping performances, which might be due to reduced muscle oscillation, improved proprioception, and possible resistance to fatigue (Doan *et al.*, 2003).

Prentice (2001) suggested that intermittent compression provides an active mechanical support to injured extremities. Kraemer *et al.* (2001<sup>b</sup> and 2004) refer to this as a dynamic immobilisation. This dynamic form of mechanical support by the external compression garment facilitates the removal of by-products (Prentice, 2001). Additionally, it also reduces the limb's movement. This in return, lessens the inflammatory response and the structural damage in the particular muscle (Kraemer *et al.*, 2001<sup>b</sup>).

Kraemer *et al.* (2004) explained that the advantage of active mechanical support is that it allows some degree of movement in the limb while immobilising the injured extremity, unlike an unyielding cast. This suggests that more motor units can be activated, therefore enhancing the neural input to the involved limb (Kraemer *et al.*, 2004; Kraemer *et al.*, 2001<sup>b</sup>). However, increased muscle firing while wearing compression garments has not been observed in all studies (Bernhardt and Anderson, 2005). The compression garment also reduces the end range of motion, which is believed to prevent injuries, but still allows functional movement. This in return will enhance performance or aid recovery after injuries.

Proprioception aids the athlete's performance by contributing to body awareness in relation to space, direction, and speed. A number of complex factors stimulate an individual's proprioception, such as mechanoreceptors in the skin, muscles, tendons, ligaments, and joints. The literature indicates that tactile mechanoreceptors increase the sensitivity of an individual's proprioception. Some authors speculate that compression garments enforce this tactile stimulation, which increases proprioceptive sensitivity. Evidence for this is, however, lacking (Bernhardt and Anderson, 2005). Furthermore, it has been said that the effectiveness of the garment will depend on the pressure exerted and the type of compression garment, as well as the athlete's inherent proprioception ability (Bernhardt and Anderson, 2005).

To this end, Doan *et al.* (2003) investigated the effect of custom-fit compression tights (shorts) on the athletic performance of experienced track athletes, specializing in jump and sprint events. A secondary objective was to investigate the mechanical characteristics of these garments. Twenty university track athletes were included in the study (age men:  $20.0 \pm 0.9$  years and women:  $19.2 \pm 1.3$  years). A randomized block design was used, where subjects wore standardized gym shorts (loose fitting) during the control tests.

The compression tights were unlike those seen in other studies. The garment ran from just above the knee to the waist, and similar to other studies it was custom fitted for each subject. In addition, the garment was 15 to 20 % smaller than the participant's lower body and at 100% expansion it was the same as the subject's original measurements. They were 4.76 mm thick and made from 75% closed cell neoprene and 25% butyl rubber, with a sticky inner to support and enhance compression. The subject's thigh muscle oscillation, skin temperature, jump power, and 60-meter sprinting performance, including ROM, were assessed. Thereafter, the mechanical characteristics of the compression garment were investigated, including elasticity and the ability to reduce impact forces.

Wearing the compression tights (WCG) did not significantly change 60-meter sprint speed, compared to the control group. However, skin temperature was significantly higher during the warm-ups (WCG: 1.09°C vs. C: 0.07°C;  $P = 0.003$ ). An increase in skin temperature usually indicates an improved blood flow. In addition, hip flexion during sprints were significantly reduced WCG (WCG:  $\sim 72^\circ$  vs. WOCG:  $\sim 78^\circ$ ;  $P = 0.04$ ). Furthermore, vertical jump height was statistically significantly improved by 5.2 % ( $P = 0.015$ ) in the compression group (WCG), compared to those WOCG, while the total squat depth was statistically significantly less (1.8 cm;  $P = 0.02$ ) WCG.

The results demonstrated that those subjects wearing the compression garment had reduced impact forces by 26.6% ( $P < 0.001$ ), at a drop height of 38.1 cm and by 11.6% ( $P = 0.066$ ) at a drop height of 76.2 centimeter (cm). The anterior-posterior as well as longitudinal oscillation in the thigh was statistically significantly lower with the compression tights ( $P = 0.01$ ). This would mean that muscle tissue damage may be reduced as well as repetitive jump performance enhanced, by the reduced oscillation on landing from the vertical jump test.

The elasticity of the garment increased the hip joint torque (N.m) by 191 - 285% during extension at  $200^\circ$  and  $195^\circ$  and 53 - 91 % at  $127^\circ$  and  $158^\circ$  flexion ( $P \leq 0.05$ ). In other words, the elasticity of the garment allows more flexion and extension torque in the hip joint at the end of the flexion and extension movement. Additionally, those athletes with the compression shorts might be less injury prone, especially in the hamstrings, due to the considerable torque that is generated during hip flexion and extension (Doan *et al.*, 2003).



The results suggest that these compression tights might enhance athletic performances and reduce the risk of injuries, by several possible means. For example, the reduced hip flexion indicates a possible increase in average stride frequency and the elasticity might even increase the acceleration of the leg after the swing phase. It is clear that the garment does not interfere with the athlete's sprinting biomechanics, except for a small obstruction near full hip extension ( $> 180^\circ$  extension). It was also noted that the materials of which the compression shorts are made can ease impact forces during contact sports, such as rugby.

The improvement in muscle oscillation might reduce injuries as well as increase repetitive jump performance. The garments provided mechanical support during jump power tests, and an enhanced proprioception might also contribute to this. The increased skin temperature could reflect increases in dynamic strength, blood flow and muscle temperature. If the garment raises the muscle temperature during warm ups, the muscle will function better and be less prone to injury. One may speculate that these results are mainly due to more compressive, elastic, and impact-absorbing characteristics of this specific compression garment compared to traditional compression garments. The authors recommend that more research should be aimed at longer sprinting events, such as 100 to 400 meter (m).

Bernhardt and Anderson (2005) assessed 13 healthy active university students (age: 25.7 years) in a crossover randomized study design. The objective was to establish whether moderate compression tights (elasticized) would have an influence on several performance determinants and proprioception at the hip joint. The hypothesis was that increased support around the hip would aid injury prevention.

Subjects performed two sessions, with a series of performance tests, either with the compression tights (WCG) or without (WOCG). Similar to Doan *et al.* (2003), compression tights caused significant reduction in active range of motion during hip flexion (WCG:  $88.50 \pm 8.8^\circ$  vs. WOCG:  $98.25 \pm 8.9^\circ$ ). All the other performance tests (balance, vertical jump agility, speed and endurance) were not statistically significant when WCG was compared to WOCG. Hyperextension of the hip WCG was 2.7 % and hip abduction WCG was 6.8% degrees more, compared to WOCG, but the results were not statistically significant. No differences were observed between WCG and WOCG with regards to flexion, hyperextension and abduction.

The results indicate that compression garments did not have an influence on a healthy individual's proprioception. The investigators speculated that this might be because these subjects did not have any injuries and that different results may be seen with injured athletes. They reasoned that individuals with the worst inherent proprioception usually gain the most benefit from additional support. However, further research is needed to confirm this.

Maton *et al.* (2006) investigated the effects of elastic compression garments (European class I;  $23.6 \pm 3.2$  mmHg to  $6.8 \pm 1.5$  mmHg) on muscular fatigue and recovery after fatigue. Fifteen healthy subjects ( $32 \pm 6$  years) participated in the study. During the experiment the force produced by static ankle dorsiflexion and the pressures exerted by the compression garment on the lower limb (anterior and posterior) were measured.

The investigators recorded surface electromyograms (EMGs) to examine muscle fatigue in the *Tibialis Anterior* (TA), *Gastrocnemius* (GA), *Rectus femoris* (RF), and *Biceps femoris* (BF). For the fatiguing protocol, subjects were requested to sit with their right knee flexed at a 70-degree ( $^{\circ}$ ) angle and the ankle in dorsiflexion at a  $70^{\circ}$  angle (with respect to the tibial line). Subjects had to complete two trials, one without a compression garment (WOCG) and the other with compression garments (WCG).

Each subject had to sustain 50% of his/her maximum voluntary force (MVF), while performing ankle dorsiflexion for as long as possible (WOCG). Then the subject rested for 30-minutes. Within the first 10 minutes of the resting period, the subjects had to perform static maximum ankle dorsiflexion every 30 seconds. Then the protocol was repeated WCG. Thereafter, the subject again had to rest for 30 minutes and perform 10 minutes of static maximum ankle dorsiflexion every 30 seconds. The pressure exerted by the compression garment was measured (Salzmann apparatus) after this final 30-minute rest period.

The endurance time with the compression garments, tended to be sustained for a 5.6% shorter time than WOCG; however, this difference was not statistically significant. No statistically significant difference was noted in the MPF or TP values between WCG and WOCG. The velocity at which the muscle fiber's action potential is conducted affects the mean power frequency (MPF) in the muscle. Peripheral (i.e. metabolic changes) or central fatigue (i.e. motor unit recruitment) may delay this conduction. What this means is that fatigue occurred in

both muscles and in both conditions (WCG and WOCG), because the results indicate a reduction in MFP, together with an increase in total muscle power. However, no difference was noticed between wearing compression garments and not wearing compression garments.

The average time to recovery 95 % of the muscle force was four minutes WCG and five minutes WOCG. The WCG subjects recovered 28.9 % faster than WOCG, but there were no statistically significant differences between recovery times of WCG and WOCG. The authors explained that both peripheral fatigue, which involves the whole leg, and central fatigue, might also have contributed to the results. The authors speculated that compression garments may aid the recovery of short dynamic fast activities, which activates the calf muscle-pump function. Maton *et al.* (2006) also pointed out that these recovery values correspond roughly to the time required for removing lactic acid.

There was a statistically significant difference between the posterior (PS) and anterior (AS) pressures of the lower limb ( $P < 0.001$ ). The anterior side experienced more pressure from the compression garments (AS:  $14.5 \pm 6.2$  mmHg vs. PS:  $12.8 \pm 4.3$  mmHg). The reason for this is that the curvature of the leg is greater at the tibial process than at calf level (Laplace's law). As the investigator pointed out, this might indicate that the influence of the compression garments might be greater on the anterior side, with respect to muscle venous dynamics, recovery of force and muscle fatigue. The variation of pressure exerted between subjects indicated that limb morphology influenced the pressure exerted, even though the garment was specifically fitted for the individual. Maton *et al.* (2006) explained that the average pressures of the garments are not enough to cause fatigue, since the pressures exerted by the muscles are still more pronounced. They concluded that the elastic compression garments did not enhance recovery of force production post-testing, nor imposed fatigue on the lower limbs of healthy subjects.

Aerobic energy cost is an important aspect of long-distance athletic performance. The change in energy cost over time, fatigue, and the clothing that a runner wears might influence running economy. Therefore, Bringard *et al.* (2006<sup>b</sup>) assessed, in a two-part experiment, the effect of compression tights, compared to normal elastic tights or normal shorts (control). They assessed indicators of muscle efficiency, such as aerobic energy cost and the slow component or energy cost over time expressed as the surplus in oxygen uptake over time ( $\text{VO}_2 \text{ SC}$ ) in two

groups of healthy trained middle distance runners (age:  $31.2 \pm 5.4$  years;  $n = 6$  and  $26.7 \pm 2.9$  years;  $n = 6$ ).

Bringard *et al.* (2006<sup>b</sup>) hypothesized that compression garments would reduce the energy cost and  $\text{VO}_2$  SC in trained middle-distance runners at submaximal prolonged running and furthermore enhance comfort, thermal and fatigue sensations. A repeated-measures experimental design was used and subjects were their own control during both experiments. Subjects wore the compression garment during indoor runs till exhaustion and over a 15 minute run at 80 % of the runner's  $\text{VO}_{2\text{max}}$ .

Energy cost was assessed at set speeds (10, 12, 14 and 16  $\text{km} \cdot \text{h}^{-1}$ ) in part one. In part two the runner's energy cost was assessed at a constant speed for 15 minutes (80% of  $\text{VO}_{2\text{max}}$ ). No significant difference was observed WCG, shorts and/or elastic tights for thermal stress, loss of body mass, subjective ratings of perceived clothing comfort, exertion and of sweating in either of the two parts. The subjects in part one that wore compression garments and tights showed a significantly lower aerobic energy cost at 12  $\text{km} \cdot \text{h}^{-1}$  and a similar trend at 10 and 14  $\text{km} \cdot \text{h}^{-1}$ , compared to just wearing shorts. No differences were noticed in heart rate, VE or in the  $\text{VO}_{2\text{max}}$  values between WCG, shorts and elastic tights. In addition, part two (II) demonstrated a significant difference between the three garments' energy cost over a time ( $\text{VO}_2$  SC) ( $P = 0.01$ ). The increase of  $\text{VO}_2$  SC was significantly reduced ( $P = 0.01$ ) in compression tights by 26% compared to normal shorts by 36%. Furthermore,  $\text{VO}_2$  SC was also lower in compression garments than in elastic tights ( $P = 0.04$ ).

The conclusion made from part one was that compression garments reduced the athlete's running economy at some submaximal speeds, but it was only significantly different from conventional shorts. Elastic tights and compression garments, on the other hand, demonstrated similar energy costs during running. Part two illustrated that compression garments used during running could improve the runner's overall circulation, and reduce muscle oscillation. This would result in less muscle fatigue and improved endurance capacity due to a lower energy cost at a prolonged submaximal speed ( $\sim 12 \text{ km} \cdot \text{h}^{-1}$  at 80 %  $\text{VO}_{2\text{max}}$ ). The authors speculated that compression garments and elastic tights may improve the runner's proprioception, muscle coordination, and propulsive force. Moreover, these factors would result in a lower energy cost during running. The authors also pointed out that these

experimental protocols might have been too short to demonstrate the advantages of compression garments above elastic tights (Bringard *et al.*, 2006<sup>b</sup>).

In a more recent study Duffield and Portus (2007), investigated the effects of three different types of compression suits (full body) on cricket performance. Well-trained club level male cricket players (age:  $22.1 \pm 1.1$  years;  $n = 10$ ) were included in the study. Subjects had to participate in a maximal- and accuracy-throwing test as well as in a 30-minute repeat-sprint test. The three different garments had to be worn during all the tests, as well as 24-hours post-exercise.

The subjects first had to complete five maximal cricket ball throws for maximal distance (DT). Between each throw, the cricketer rested for 20 seconds. Next was the accuracy-throwing test (AT), in which the aim was to throw two 10, 20, and 30 meter target throws. The target was a diagram of the actual size wickets, with scoring zones allocated. The objective was not just accuracy but also speed, thus the subject achieved a score according to time to complete (speed) and accuracy of the throws. Wearing the three compression garments did not result in any significant improvement in the repeated-sprint and throwing performances (WCG<sub>1</sub>, WCG<sub>2</sub> and WCG<sub>3</sub>), compared to WOCG. There were also no differences in the heart rate, body mass, capillary blood lactate, pH, CK, haemoglobin, and oxygen partial pressures post-exercise.

The only statistically significant changes noted were (i) an increase in skin temperature in all three WCG – trials, (ii) the 24-hour post-exercise CK levels in WCG<sub>1</sub> and WCG<sub>3</sub> compared to WOCG were significantly less and (iii) perceived muscle soreness in the arms and legs WCG was less 24-hours after exercise, compared to the those WOCG. Perceived muscle soreness was assessed with subjective rating scales, thus it may indicate that the compression garments had a psychological impact. Furthermore, the authors suggest that compression garments might be able to reduce post-exercise trauma and perceived muscle soreness 24 to 48-hours post-exercise.

#### 4.1. Conclusion

It has been suggested that compression sleeves supply mechanical support to the injured extremity and enhanced the repair of the contractility of the involved muscle group. This might restore the athlete's ability to produce muscle action. However, this is not supported by the studies investigated here. More evidence suggests that compression could be used as a recovery tool to reduce muscle soreness after exercise. Thus, more research is needed on the ability of compression to enhance functional capacity of the previously active muscles.

#### **E. SIDE-EFFECTS**

It is clear from the previously studies that the correct amount of pressure needs to be applied to the limb to obtain an effective response (Agu *et al.*, 1999; Rimaud *et al.*, 2007; Bringard *et al.*, 2006<sup>a</sup>). In the clinical setting the optimal pressure varies according to the individual's condition, although no consensus has been reached on the preferred pressure. Some authors are of the opinion that compression garments between 30 to 40 mmHg will prevent fluid from escaping through the capillary wall and enhance venous blood flow in the lower extremities of individuals with severe venous insufficiency conditions (Gniadecka *et al.*, 1998; Bringard *et al.*, 2006<sup>a</sup>).

Nevertheless, pressures as low as eight mmHg (distal) to 18 mmHg (proximal) have also been shown to be beneficial in preventing oedema in individuals with varicose veins and other lower limb circulatory problems (Agu *et al.*, 1999; Benkö *et al.*, 2001; Hirai *et al.*, 2002). Hirai *et al.* (2002) maintains that compression garments with pressures of 22 mmHg and/or 30 to 40 mmHg in the ankle are more superior in preventing oedema, than the lower compression garments. However, pressures that are beneficial for individuals with peripheral conditions, may actually be harmful for healthy or active individuals.

Pressures exerted through compression garments differ according to body position, such as standing or lying supine. For example, in the supine position a pressure of more than 10 mmHg will limit blood pooling in the veins, by reducing the volume and increasing the blood flow velocity in the veins (Partsch, 2003). However, pressure exceeding 30 mmHg in the supine position will not have any effect, since the venous blood volume cannot be reduced

more or shunted to the heart. On the other hand, in the upright position, pressures in the lower leg varies between 20 to 100 mmHg, and thus a higher compression garment (40 - 50 mmHg) is needed to exert an effect on venous blood flow. This might indicate that a different garment will be needed for different types of body positions in different sports.

Berry *et al.* (1990) investigated whether elastic tights would have the same effect on the blood lactate concentrations and the post-exercise response as compression garments. Eight male subjects (age:  $27.0 \pm 1.8$  years;  $VO_{2max}$ :  $56.5 \pm 1.5$  mL.kg<sup>-1</sup>.min<sup>-1</sup>) ran on a treadmill at 110% of their  $VO_{2max}$  for three minutes. This was repeated three times, with the only difference being three random conditions, i.e. with the elastic tights during the exercise and recovery, only during the exercise bout, or without any elastic tights.

No statistically significant differences were found in post-exercise heart rates,  $VO_2$ , lactate or haematocrit levels in any of the three conditions. Thus, elastic tights did not alter blood lactate concentrations post-exercise, which could have been because of the lower pressures of the elastic tights (Berry *et al.*, 1990). Gniadecka *et al.* (1998) used light compression garments (10 - 20 mmHg) and found no increase in venous return. Similarly, Rimaud *et al.* (2007) found no significant enhancement of performance, or increase in venous return in their study with low compression (21 mmHg) socks.

Kraemer *et al.* (2004) explained that it is important that the garment does not exceed diastolic blood pressure (40 to 60 mmHg for the upper extremities and 60 to 100 mmHg for lower extremities). Since pressures that exceed these limits would occlude venous flow and would delay the recovery process of tissue injury and damage. In healthy individuals higher pressures of 35 to 55 mmHg may increase the venous flow velocity by 175%, and aid waste product removal better. A pressure of 30 mmHg would increase the venous velocity by more than 18 mmHg in chronic venous deficiencies, but the lower pressure is considered as being safer and less likely to compromise subcutaneous oxygenation (Agu *et al.*, 1999). According to Bringard *et al.* (2006<sup>a</sup>), healthy individuals do not require high compression, such as 30 – 40 mmHg during recovery. Pressures of 10, 30 and 64 mmHg reduce cutaneous blood flow by 10, 25 and 84 %, respectively. This illustrates the importance of the correct fitting and size of the garment.

One of the suggested contra-indications of compression therapy is that the increased intramuscular pressure and associated ischemia could lead to circulatory problems, fatigue (peripheral or central) and/or ischemic damage, such as necrosis and compartmental syndromes (Ogata and Whiteside, 1982; Prentice, 2001; Maton *et al.*, 2006). This ischemia is due to the limited perfusion and blood flow in venous cutaneous and/or muscle capillaries. Ischemic complications have been reported when individuals with arterial insufficiencies wore elastic compression garments (Bergan and Sparks, 2000). Researchers speculate that the increased pressure exerted on the limb, by the compression garment, restricts perfusion (Ogata and Whiteside, 1982; Maton *et al.*, 2006).

Furthermore, it has been suggested that blood flow ceases even when the pressure applied to the tissue is less than that of diastolic pressure. It also seems that blood circulation in the cutaneous capillaries might be more vulnerable than in the muscle (Ogata and Whiteside, 1982). Ogata and Whiteside (1982) performed an animal study and established that blood flow to the skin ceased when external compression exceeded 30 mmHg, while in the muscle blood flow was sustained up to 50 mmHg. Thus, even when high external pressures do not reduce muscle perfusion, it could be debilitating for skin circulation.

Other possible problems, which are associated with ischemic conditions, and have been noticed in the long term use of compression garments, is that pressures above 40 mmHg might cause a spontaneous burning, prickle or numbness sensation (paresthesia). In addition, the skin's integrity needs to be checked regularly, since the garment might soften the skin (maceration) or irritate the skin, causing a rash (Brennan and Miller, 1998; Van den Kerckhove *et al.*, 2005).

Some tight compression garments have been found to be difficult to manage or to put on and are uncomfortable, especially in elderly, obese or arthritic patients (Choucair and Phillips, 1998; Gniadecka *et al.*, 1998; Harris, *et al.* 2001). Other authors indicated that both knee and thigh-length graduated compression garments have been reported as difficult to use, even though both significantly reduce venous stasis of the lower limb. However, it has been indicated that knee-length garments are more affordable, comfortable to wear and wrinkle less (Agu *et al.*, 1999; Benkő *et al.*, 2001).



Graduated compression garments need to be replaced at least every three to six months or as soon as the garments lose its elasticity. Generally, it is recommended to replace garments at least three times a year in individuals who wear garments daily. This will ensure efficient compression, since the garment might lose its elasticity over time and due to washing (Brennan and Miller, 1998; Choucair and Phillips, 1998; Bergan and Sparks, 2000; Harris, *et al.*, 2001; Van den Kerckhove *et al.*, 2005).

## **F. CONCLUSION**

Most research mainly investigated the effects of compression garments on individuals with vascular disorders. They concluded that compression garments initiate a series of complex physiological and biochemical effects concerning the venous, arterial and lymphatic system. Theoretically, the compression garment mechanism is multifactorial. By increasing the tissue hydrostatic pressure and by the actual mechanical compression, the garment creates a pressure gradient favouring fluid reabsorption and reduces possible space for oedema and the venous blood vessel diameter. These two mechanisms shunt the blood towards the deeper venous system and enhance venous return as well as the blood flow velocity. One could make the assumption that compression garments mimic the function of the calf muscle-pump function during exercise, similar to the active recovery technique.

The latest research focuses more on compression as a post-exercise recovery and performance enhancement strategy. Even though some of the evidence for compression garments is still equivocal, several studies indicate that compression garments may possibly enhance blood flow, reduce oedema and pain, attenuate the inflammatory response, lessen DOMS symptoms, assist waste product clearance and increase proprioceptive sensitivity. However, an appropriate garment according to pressure, materials, body type, sport modality, and level of athletic performance must be considered. This will avoid detrimental effects on the peripheral and subcutaneous circulation's oxygenation and ensure effectiveness of the garment. Further research is warranted, to determine whether compression therapy is an effective post-exercise recovery method.

## **CHAPTER FIVE**

### **PROBLEM STATEMENT**

#### **A. COMPRESSION GARMENTS AND ITS CONTEXT**

Compression garments are not a novel idea in the clinical setting, where they are used to treat inflammation and associated pain or swelling, as well as to improve venous return. Two decades ago Berry and McMurray (1987) conducted the first research investigating compression garments as a post-exercise recovery modality. However, it is only in the last seven years that the focus has shifted more to compression garments as a recovery modality after strenuous exercise.

A significant amount of research exists showing the beneficial role of compression garments on vascular distribution in diseased patients with venous insufficiencies. However, these individuals usually have venous reflux and weak calf muscle strength, as well as calf muscle-pump dysfunction. Trained runners, especially long distance runners, do not demonstrate any of these problems, and their physiological systems are usually well adapted to strenuous events, such as prolonged running.

More evidence is needed to show whether compression garments aid post-exercise recovery and athletic performance. To date only a small amount of research supports the notion that compression garments may provide some benefits in sports performance and aid recovery after exercise.

#### **B. EXISTING LITERATURE ON COMPRESSION GARMENTS**

A few problems and limitations arise when the available literature is reviewed on compression garments as a post-exercise recovery strategy. Manufacturers of commercial compression garments have made several anecdotal claims. However, little scientific evidence is available to support their claims.

The majority of the literature that was reviewed addressed the possibility of compression as a post-exercise recovery strategy or performance enhancement aid in a younger population ( $22.3 \pm 1.87$  years) (Berry and McMurray, 1987; Kraemer *et al.*, 2000; Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>; Doan *et al.*, 2003; Bernhardt and Andersen, 2005; Gill *et al.*, 2006; Bringard *et al.*, 2006<sup>a</sup>; Ali *et al.*, 2007; Trenell *et al.*, 2006; Duffield and Portus, 2007). Only one other study involved elderly cyclists ( $63 \pm 3$  years) (Chatard *et al.*, 2004). Three studies were done on subjects older than 30 years (Bringard *et al.*, 2006<sup>b</sup>; Maton *et al.*, 2006; Rimaud *et al.*, 2007). Rimaud *et al.* (2007) investigated the influence of compression garments in the recovery of well trained athletes with spinal cord injuries. Maton *et al.* (2006) assessed fatigue in healthy untrained individuals, while Bringard *et al.* (2006<sup>b</sup>) studied middle distance runners. Therefore, to date no study has been done on middle-aged long distance runners.

There has been an increase lately in the popularity of compression garments across a range of sports. The literature, however, shows more research on compression therapy in team sports and activities of short duration, such as cricket, sprinting and jumping events and rugby (Doan *et al.*, 2003; Gill *et al.*, 2006; Ali *et al.*, 2007; Duffield and Portus, 2007). Most protocols involved either short bouts of eccentric or maximal exercise (Berry and McMurray, 1987; Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>; Chatard *et al.*, 2004; Bernhardt and Andersen, 2005; Trenell *et al.*, 2006; Maton *et al.*, 2006; Rimaud *et al.*, 2007). Subjects were also assessed after games or with functional tests of a short duration (< 15 minute or 10 km events) (Doan *et al.*, 2003; Bringard *et al.*, 2006<sup>b</sup>; Gill *et al.*, 2006; Duffield and Portus, 2007; Ali *et al.*, 2007). In a few cases, experiments included standing or resting protocols (Kraemer *et al.*, 2000; Bringard *et al.*, 2006<sup>a</sup>). None of the protocols thus far involved prolonged continuous activity of more than 60 minute.

Most individuals that have been included in studies were healthy untrained or recreational athletes (Kraemer *et al.*, 2000; Kraemer *et al.*, 2001<sup>a</sup>; Kraemer *et al.*, 2001<sup>b</sup>; Bernhardt and Andersen, 2005; Maton *et al.*, 2006; Trenell *et al.*, 2006; Ali *et al.*, 2007). A few studies assessed trained athletes (Berry and McMurray, 1987; Doan *et al.*, 2003; Chatard *et al.*, 2004; Gill *et al.*, 2006, Duffield and Portus, 2007; Rimaud *et al.*, 2007), of which only three were performed on runners (Berry and McMurray, 1987; Bringard *et al.*, 2006<sup>a</sup>; Bringard *et al.*, 2006<sup>b</sup>). However, none of these studies tested long distance runners competing in regular

marathon races and only Berry and McMurray (1987) assessed the compression garments for post-exercise recovery.

### **C. THE OBJECTIVE OF THE CURRENT STUDY**

The present study endeavors:

1. to determine whether compression garments aid the recovery process of experienced middle-aged long distance runners, after a prolonged two-hour treadmill run.

#### Specific questions:

- (i) Are compression garments an effective metabolic recovery modality after prolonged runs. In particular, would compression garments reduce the time it takes to recovery so that the runner may return to training as soon as possible?
- (ii) Will compression garments prevent skeletal muscle damage? In other words, will compression garments show attenuated response in muscle damage markers, such as plasma creatine kinase and lactate dehydrogenase activity?
- (iii) Do compression garments reduce the associated symptoms of exercise induced-muscle soreness, i.e. swelling, perceived muscle soreness, diminished muscular strength, power and endurance and limited range of motion?
- (iv) Do blood lactate concentration decrease as a result of compression garments?

## **CHAPTER SIX**

### **METHODOLOGY**

#### **A. STUDY DESIGN**

In this crossover controlled experimental study the effect of compression garments on selected parameters of muscle recovery was studied in long distance runners after a prolonged run, in a controlled laboratory environment.

The following parameters of recovery were assessed, namely

1. blood lactate concentration
2. muscle soreness
3. lower limb oedema
4. muscular strength and endurance
5. lower limb range of motion

The following tests were administered to assess the aforementioned parameters:

1. blood lactate concentration assessment with a portable Lactate Pro analyzer
2. VAS questionnaires
3. enzyme markers: LDH and CK<sub>p</sub> analysis
4. lower limb circumferences
5. vertical jump test and time to exhaustion (TTE) step test
6. modified sit and reach flexibility test

#### **B. SUBJECTS**

Seven injury free male long distance runners, aged 36 to 51 years, volunteered to participate in the study. Each subject acted as his own control and therefore completed two trials in random order, namely with and without compression socks. Since it was impossible to blind participants to the intervention (compression garment), subjects were never informed of the true purpose of the study, so as to avoid prejudiced views which could interfere with the subjective questionnaires.

*Recruitment:* All registered running clubs (Boland and Western Province), within a 70 km radius from the testing facility, were approached for subject recruitment. This was done via word of mouth and advertisements. Informative pamphlets, stating the study design and tests involved, were handed out or faxed to each club. Electronic correspondence was also sent to the members of the respective clubs. If an athlete was interested in participating in the study, they could place their names, contact details and dates available on a list at their club representatives. After two to four weeks the lists and electronic replies were collected and followed up by phone or electronic correspondence. If the athlete met the inclusion criteria, an appointment was scheduled for the baseline visit to the Sport Physiology Laboratory, Stellenbosch (South Africa).

*Inclusion and exclusion criteria:* Subjects were healthy male volunteers, between the ages of 35 to 55 and without a history of musculo-skeletal, metabolic, cardiovascular or endocrine disorders. Subjects were excluded if they participated in any lower body weight training exercises in the three months prior to the study, or were dependent on chronic medication. Subjects needed to train on a regular basis (four to five times a week) in order to be sure that they will be able to complete the laboratory exercise test. Competitive athletes were included but they were not elite, professional and/or ultra marathon athletes.

## **C. EXPERIMENTAL DESIGN**

Subjects performed testing at the Sport Physiology Laboratory, Stellenbosch University (Stellenbosch, South Africa) on five different occasions. All the visits, except the initial laboratory visit (visit one), were repeated twice for each subject (with and without compression socks).

### **1. Laboratory visits**

*Visit one:* One to three weeks prior to the treadmill test subjects were familiarized with the testing procedures, i.e. the treadmill (h/p/cosmos/Saturn Nussdorf-Traunstein, Germany), heart rate monitor (POLAR®, Polar Electro Oy, Finland), vertical jump and time to exhaustion (TTE) step test. Furthermore, athletes were given an explanation of the study protocol, dietary

guidelines, and exercise guidelines for the 24 hours prior to the 120-minute treadmill run. Subjects were requested to complete a general questionnaire about training methods, best marathon times in last 6 months and injuries in the last three months. After informed written consent was acquired, the subject's anthropometric profile, maximum aerobic capacity ( $VO_{2max}$ ) and flexibility (modified sit-and-reach test) were tested to obtain baseline values. The baseline VAS questionnaire, assessing muscle soreness, was also completed.

Visit two: Blood samples were taken prior to the 120-minute treadmill run, along with a resting VAS questionnaire. Thereafter the athletes ran a 90 minute simulated mountainous trail, in the controlled environment of the Sport Physiology Laboratory (18 – 20 °C), on a treadmill (h/p/cosmos/Saturn Nussdorf-Traunstein, Germany), followed by a 30 minute downhill run (DHR) at a ten percent gradient. The resistance (speed) of the treadmill was adjusted to keep the runner's effort level at 70 percent of his  $VO_{2max}$ . Blood samples were taken after the 30-minute DHR at three, ten and 30-minute post-exercise for lactate analysis with a Lactate Pro lactate analyzer (ARKRAY, Inc. Kyoto, Japan). A second blood sample was collected ten minutes after the downhill run (DHR), for enzyme analysis. At 15 minutes the subject completed the VAS questionnaires and flexibility test. Subsequently, the 30-minute lactate sample was taken and then subjects performed two lower body strength tests: the vertical jump and a TTE step test.

Visit three to five: All subjects were followed up for three days, during which blood samples were taken at 24, 48 and 72 hours post-run for lactate,  $CK_p$  and LDH analysis. On each occasion the subject also completed the VAS questionnaires, TTE step test and the modified sit-and-reach flexibility tests to assess muscle fatigue and tightness. Four limb girths were also measured to quantify possible oedema in the lower extremities.

Visits one and two were separated by a one to three week preparation phase, in which the participants were allowed to train and participate in their normal running program. However, the seven days prior to the two-hour treadmill run (visit two), subjects were asked not to perform any hill training, running up or down stairs, jumping or plyometrics, lower body resistance training, strenuous training (heart rate above 70% of maximum heart rate) and running races. In the 72 hours after the treadmill run, the athletes had to refrain from any form of exercise, until the last blood sample was taken at 72 hours post-exercise. In addition,

subjects were requested to abstain from taking any oral pain medications (including non-steroidal anti-inflammatory drugs (NSAID)), as well as any additional interventions to alleviate pain, i.e. cold–or-heat therapy, massage, stretching, and ultrasound. Subjects were asked not to consume any food or drink fluids, except water, two to three hours before, as well as no physical activity 24 hours prior to each assessment. They were instructed to record their daily activities, and fluids and food intake in a journal. Both the with (WCG) and without compression garment (WOCG) trials for each subject were separated by seven to 28 days. All subjects performed all their tests at approximately the same time of the day.

## **2. Place of study**

Data were collected at Stellenbosch University, in the fully equipped Sport Physiology laboratory of the Department of Sport Science. All testing were done at temperatures between 18 and 20 °C.

## **3. Ethics**

During visit one the informed consent form (see *Appendix A*) was verbally explained to each subject and opportunities for questions were allowed. The subjects read through the consent form in their own time as well. The researcher educated the subjects with regards to the relevance as well as the associated risks of the study. Subjects were kept blind to the purpose and benefits of the compression garments. Research was conducted to the Declaration of Helsinki medical research council (MRC) and GCP.

## **4. Compression garments**

During the initial baseline meeting each subject's lower leg length, ankle and mid-calf circumference were documented. These records were used to select the correct compression garment; one which covered the widest circumference of the calf muscle in a standing position, up to just below the patella. A moderate pressure sock was selected for this study. The highest pressure (32 mmHg) was at the foot and ankle and the lowest pressure (23 mmHg) was just below the knee.



The selected compression garments (*C-Sock*) are designed by *Pardes* (Maintal, Germany) according to the *European RALL* guidelines, and are composed of Elasthane (spandex) and Polyamide (nylon). Subjects wore the compression garments throughout the two hours treadmill run, as well as over the three-day recovery period. Socks were removed when the participants' dominant limb and circumferences were measured. Subjects were permitted to remove the garment during bathing or showering and whilst sleeping.

## **D. MEASUREMENTS AND TESTS**

### **1. Anthropometric measurements**

Anthropometrical measurements were taken during the initial baseline consultation, as well as during the 24, 48 and 72 hours follow-up assessments by a HPCSA registered (health professional council of South Africa) and qualified Biokineticist. Anthropometrical measurements included i.e. stature, body mass, lower leg length and four girths (calf, ankle, proximal and mid-thigh). The latter two measurements were necessary for the compression sock fitting. Bioelectrical impedance analysis was used to assess percentage body fat.

For all assessments, subjects were bare feet and were dressed in tight fitting clothes. All testing were done two hours post-prandial and prior to any physical activity. All anthropometrical measurements were taken on the right-hand side of the body, according to the ISAK protocol (Marfell–Jones *et al.*, 2006).

*Body mass:* Body mass was determined by using a calibrated electronic scale (UWE BW – 150 freeweight, 1997 model, Brisbane Australia), and recorded to the nearest 0.1 kilogram (kg). The weighing scale was zeroed on a level surface before the measurement was recorded. The subject was then asked to stand in the centre of the scale, distributing his weight evenly without support.

*Stature:* This determines the distance between the transverse planes of the highest point on the skull and the inferior aspect of the feet, at right angles. Stature was measured with a sliding steel anthropometer (Siber-Hegner GPM, Switzerland), while the subject was positioned against a wall. The anthropometer was held vertically to a wall and perpendicular to the floor.

The subject stood bare feet with his heels together and upper back, buttocks and heels against the wall. The mid-line of the body was positioned in-line with the measuring tape, in front of the subject. The sliding anthropometer was placed firmly on the highest point of the subject's skull (Vertex), while the head was in the Frankfort plane, with the lower edge of the eye socket (Orbitale) in line with the indentation superior to the tragus of the ear (Tragion), in the horizontal plane. The subject was requested to inhale while the measurement was taken. Measurements were taken to the nearest 0.1-centimeter (cm).

*Bioelectrical impedance analysis (BIA):* A portable body composition monitor was used to assess the subjects' lean and fat mass (Bodystat CC BCM 1000, South Africa, 1991). Lean mass comprises the bony skeleton, muscle mass, innards and entire water content of the body, while fat mass is only the adipose tissue. Subjects were requested to lie in the supine position, and the electrodes were connected to the right hand and foot.

The skin was wiped with an alcohol swab before placing the electrodes on the standard anatomical sites. The first electrode was placed on the dorsal side of the hand, one centimeter proximal to the knuckle of the middle finger, with the second electrode on the wrist next to the wrist joint (between the head of the ulnar and radius). The remaining two electrodes were placed on the dorsal surface of the foot, on the base of the foot and one centimeter proximal to the joint of the second toe (next to the big toe), and on the ankle at the level of the protruding bones (medial and lateral malleoli) on the side of the ankle.

The subject's limbs were not to touch his or her center body or one another; the subjects were also requested to empty their bladders beforehand and to avoid exercise and drinking diuretics like alcohol, coffee and certain teas 24-hours prior to testing (nutritional guidelines). The leads were paired into red and black attachments, with the red lead always connected to the current – introducing electrode and the black lead connected to the detector electrode. After the electrodes were positioned, the paired leads were connected to the corresponding electrodes. All measurements were taken while the subject was relaxed for approximately 20 minutes.

*Girths:* All circumference measurements were taken by the same investigator, in the anatomical position, according to the standard ISAK (International Standards for

Advancement of Anthropometry) protocols. All measurements were obtained without the compression garment. Four girths were taken to determine the circumferences of the athletes' lower limbs: calf, ankle, and proximal-and-mid-thigh. Two sets of measurements were obtained. If a set differed by more than 2 mm from the other, subsequent measures were taken. This would indicate whether swelling was present in the lower body of the athlete, after the two-hour treadmill run.

Girths were measured with a spring-loaded, non-extensible anthropometric tape measure (*Rosscraft*, Canada). The flexible steel tape is seven millimeters (mm) wide and two meters (m) long, with four centimeters (cm) blank before the zero line. All measurements, except the ankle, were recorded at the point where the circumference was the greatest. The measuring tape was held horizontally, at right angles to the limb and so as not to compress the subcutaneous tissue with a constant tension. A cross-hand technique was used, with the zero mark on the more lateral side of the subject at the designated anatomical landmark. Measurements were recorded to the nearest millimeter (mm). All measurements were taken while the athlete stood upright, with legs slightly apart and weight evenly distributed.

(i) Proximal thigh: The subject stood with arms crossed at the chest, on a 25cm step (© 2000 *Reebok International LTD*, Taiwan), so that the measurement could be taken at eye level. The anthropometric tape was placed firmly around the lower thigh, and moved up to one centimeter (cm) distal to the gluteal fold, horizontal to the vertical long axis of the limb.

(ii) Mid-thigh: The subject stood on a 25cm step with arms crossed at the chest. The measurement was taken midway between the trochanterion and lateral border of the tibia, at the mid-trochanterion-tibiale laterale site.

(iii) Calf: The subject stood on a 25cm step, with arms hanging by the sides. The steel tape was applied perpendicular to the long axis of the leg, around the maximum girth of the calf muscle (the medial calf site). The anthropometric tape was moved up and down the calf, until the maximum girth was obtained.

(iv) Ankle: The subject stood on a 25cm step, with arms hanging by the sides. The steel tape was placed tightly around the minimum circumference, proximal to the malleolus of the tibia and fibula of the leg and superior to the sphyrion tibiale. The anthropometric tape was moved up and down the lower leg, until the minimum girth was obtained.

Circumferences were taken during the initial meeting (baseline), after the two hour prolonged run and at 24, 48 and 72 hours and during both trials (with and without compression socks).

Lengths: Lengths are the vertical distances from the standing surface, while the subjects stood with their feet together, with their arms hanging at their sides in the upright position. The direct method was used; thus the measurement was taken from landmark to landmark. Only one length was assessed; the tibiale laterale height, by using a segmometer (*Rosscraft*, Canada) and a 25 cm step (*Reebok* aerobic step).

(i) Lower leg length: The vertical distance was taken from a standing position (the fixed part of the segmometer), on a 25 cm step, from the step surface to the tibiale laterale site (sliding part of the segmometer).

## **2. Assessment of perceived muscle soreness**

Visual analog scale: The subjective Visual Analog Scale (VAS) was used, which rated the perceived muscle pain or discomfort before the exercise commenced, directly after the treadmill run and at 24, 48 and 72 hours following the run protocol.

The VAS consists of a ten centimeter (0 to 10 cm) line labeled with “no pain” on the extreme left end (0 mark) and “unbearable pain” on the extreme right end (10 mark). To quantify the perceived muscle pain, the subject marked the VA scale, with a pencil. The score was rounded off to the nearest one millimeter from the left (*Figure 4.*).

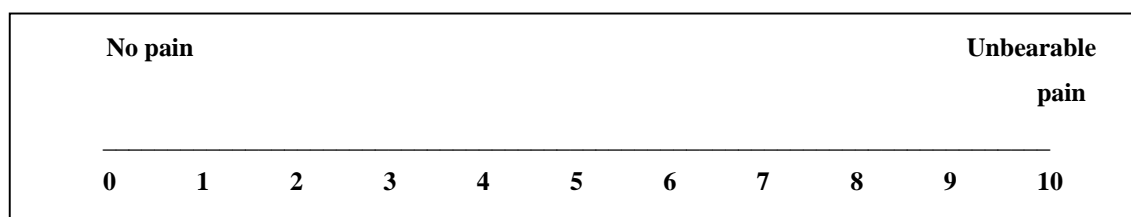


Figure 4. A schematic representation of the visual analog scale, which was used to assess perceived muscular pain and discomfort.

The VA scales assessed muscular pain or discomfort in the front knee extensor muscles, i.e. the *Quadriceps*. Perceived muscle soreness scores were taken while (i) the athlete was in a resting state, lying supine on a plinth. This gave the subject the opportunity to be familiarized with the questionnaire. Furthermore, (ii) while stretching the *Quadriceps* in both legs, (iii) whilst pressure was applied to the muscle belly and (iv) after the TTE step test to assess pain after functional flexion-and-extension movements in the knee joint.

- (i) At rest: Subjects were seated and asked to relax. The subject was then questioned about muscular pain or discomfort in his *Quadriceps* whilst he was inactive. Subjects were not allowed to move or stretch the relevant muscle group.
- (ii) Pressure response: Subjects stood upright with their body weight evenly distributed. Pressure was applied to the muscle belly site of the *Quadriceps* muscles. The muscle belly is a more suitable site for measuring pain related to pressure than the musculo-tendinous sites (Baker *et al.*, 1997). Subsequently muscle discomfort due to the pressure was assessed. Pressure was standardized by pressing inwards into the muscle for three seconds (Medalist stopwatch, South Africa) and then immediately released.
- (iii) During stretching: Subjects were asked to assess the muscular distress, irritation, and/or soreness during stretching in the *Quadriceps* muscles. The *Quadriceps* were stretched in both legs and held for 15 seconds.

- (iv) Functional movement in knee joint: Subjects were asked to rate their muscle soreness after the time to exhaustion (TTE) step test that specifically involved flexion and extension of the knees.

### 3. Blood sample collection

Enzyme analysis: Blood samples for enzymatic analysis were obtained before the two-hour treadmill run, 10 minute post-run as well as at 24, 48 and 72 hours post-exercise, under resting and three hours post-meal conditions (*Figure 5.*).

Blood samples, of about five milliliters per sample, were drawn from an antecubital vein in the forearm using a needle, syringe, and *Vacutainer* set-up, by a trained phlebotomist. EDTA was used as an anticoagulant. Samples were transported to the pathology laboratories within one hour after collection. Serum creatine kinase (CK<sub>p</sub>) and lactate dehydrogenase (LDH) concentrations in the blood samples were analyzed by qualified pathologists (*PathCare* laboratories).



*Figure 5.* Collection of blood samples (photograph by J. Barnard)

The laboratories processed and centrifuged the blood samples (1500 x g) and analyzed it for the specific enzyme markers, with either the *Beckman Synchron LX/CX* (United States of

America) or *Roche Hitachi 911* analyzers. Both of these procedures employed the *Lactate to Pyruvate* method to analyze LDH and the *original Rosalki* method for CK<sub>p</sub> analysis.

*Blood lactate analysis:* Whole blood lactate concentrations were measured with an automated blood lactate analyzer (*Lactate Pro*, Arkray Inc., Kyoto, Japan). The Lactate Pro was chosen for its practicality, accuracy, reliability and demonstrates a strong correlation with other analysers (  $r = 0.98 - 0.97$ ) with a range of mean difference of -0.06 mM to 0.52 mM , over a physiological range of 1.0 – 18.0 mM (Pyne *et al.*, 2000).

A resting blood sample was collected prior to the two-hour treadmill run, by means of a finger prick with a lancet device (Softclix ®, Boehringer Mannheim). Lactate samples were also collected following the two-hour treadmill run, at three, ten and 30-minute intervals, as well as at 24, 48 and 72 hours.

Before each sample was collected the subject was requested to warm up his hands, by pulsating his fingers to enhance blood flow to his fingertips. For each sample, the subject's fingertip was cleaned with an alcohol swab prior to the finger prick. To prevent damaging blood cells and to prevent leakage of interstitial fluid into the sample obtained, the subject's fingers were on no account compressed for blood. This would prevent a false readings of the blood lactate concentrations.

A cotton swab was used to wipe off the first blood droplet and then the next droplet of blood was sampled on the lactate strip. Alternate fingers were used with each finger prick and cotton swabs were used to stop any additional bleeding of the subject's finger.

#### **4. Maximum aerobic capacity test**

A progressive incremental exercise test to exhaustion was performed on the h/p/cosmos Saturn treadmill (h/p/cosmos/Saturn Nussdorf-Traunstein, Germany), to assess maximum aerobic capacity during the baseline session.



*Figure 6.* Subject on h/p/cosmos/Saturn (Nussdorf-Traunstein, Germany) treadmill completing a  $VO_{2max}$  test (photograph by K. Welman)

The treadmill (h/p/cosmos/Saturn Nussdorf-Traunstein, Germany) is interfaced with specialized computer software (*Cosmed Quark b<sup>2</sup> 2000*, Italy). By using breath-by-breath analysis together with a telemetric heart rate monitor (POLAR®, Polar Electro Oy, Finland), the Quark b<sup>2</sup> software calculates and records exercise intensity and selected cardiorespiratory parameters continuously throughout each test (*Figure 6.*).

*Maximum aerobic capacity protocol ( $VO_{2max}$ ):* Subjects were fitted with an adjustable safety harness. The test commenced with a five minute warm up from speeds starting at seven kilometers per hour ( $km.h^{-1}$ ) for two minutes, one minute at eight  $km.h^{-1}$  and ending at nine kilometers per hour for two minutes. Thereafter the subject was allowed to drink water, before the mask was placed over his face.

A maximal test to exhaustion was performed, with increments of one kilometer per hour, every three minutes, up to thirteen kilometers per hour. Thereafter both the speed and incline was increased by one kilometer per hour ( $km.h^{-1}$ ) and one percent every minute until the subject reached exhaustion. The test was terminated when the subject reached exhaustion, which was verified if (i) the  $VO_2$  did not increase by more than 150 ml per successive



workload, (ii) a respiratory quotient (RER) value equal or above 1.15 was reached, (iii) heart rate was more than 90 % of the theoretical maximal heart rate (bpm) and (iv) the rating of perceived exertion (RPE) was above 19.

Throughout the incremental test, breath-by-breath gases were continuously recorded. Expired gases, flow and volumes were sampled through the turbine flow meter and gas sampling line and analyzed by a cardio-pulmonary metabolic system (Cosmed *Quark b<sup>2</sup>*, Rome, Italy). The gas analyzers were calibrated prior to each test with atmospheric gas and known gas concentrations (16 % O<sub>2</sub>, 4% CO<sub>2</sub>, balance N<sub>2</sub>) and the turbine flow meter was calibrated with a 3 L calibration syringe. Heart rate was measured through telemetry (POLAR®, Polar Electro Oy, Finland) interfaced with the metabolic system.

Anaerobic threshold (AT): The anaerobic threshold was detected at the same time as the VO<sub>2max</sub> through the specialized computer software analysis (Cosmed *Quark b<sup>2</sup>*, Rome, Italy), during the incremental maximum aerobic test. Anaerobic threshold, or ventilatory threshold as it is also known, was defined as the point where a non-linear increase in the consumption of carbon dioxide (VCO<sub>2</sub>) occurred when plotted against the subject's oxygen consumption (VO<sub>2</sub>) (Wasserman *et al.*, 1973). The AT value was expressed as a percentage of VO<sub>2max</sub> (%VO<sub>2max</sub>). The heart rate (HR) and velocity at which AT occurred were also determined through computer software analysis.

## **5. The two-hour prolonged treadmill run**

Participants completed a simulated two-hour undulating run on a h/p/cosmos/Saturn (Nussdorf-Traunstein, Germany) treadmill. The protocol consisted of a 90-minute gradual mountainous trail followed by 30-minute downhill run (-10 % equivalent to 35. 1 °). In the initial part of the run, the objective was to simulate an extended run at a moderate tempo, on an *up-and-down* route (see *Appendix B*).

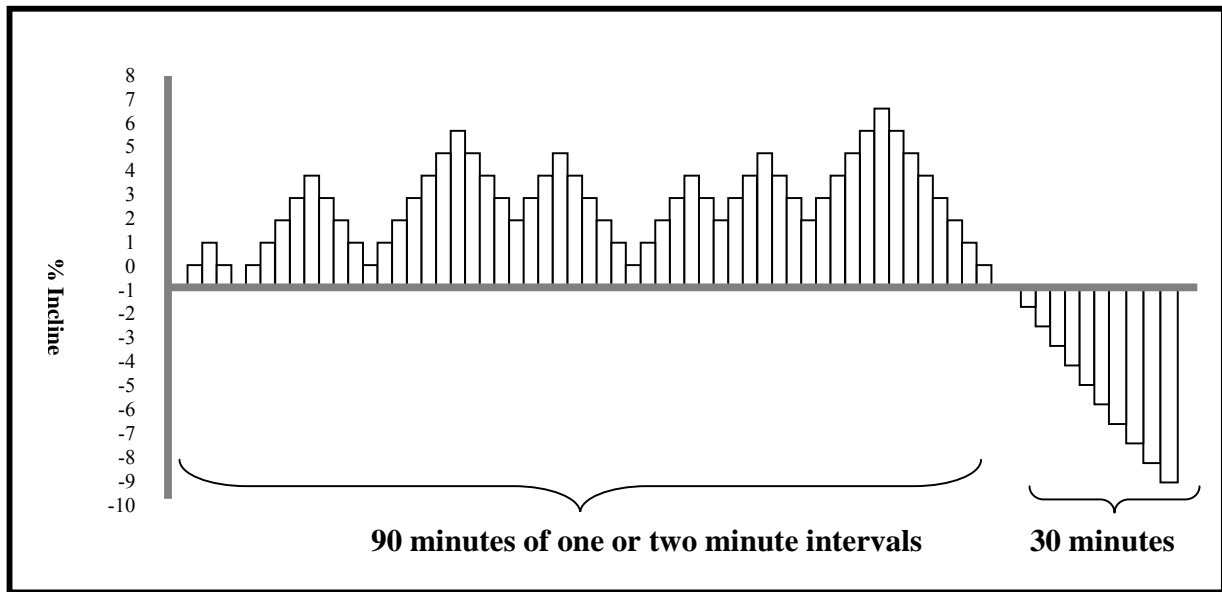


Figure 7. A schematic representation of the prolonged simulated two-hour treadmill run

90-Minute simulated mountainous run on the h/p/cosmos/Saturn treadmill: The elevation of the treadmill was either increased or decreased in one to two-minute intervals (Figure 7.). The treadmill velocity was adjusted so that subjects maintained a pace equal to 70% of their  $VO_{2max}$ . After the 90 minute run, the treadmill was stopped and the subject stepped off the treadmill for a maximum of two minutes. During this period, the treadmill belt was reversed and the safety barrier moved to enable the athlete to run downhill. The downhill run started on a level surface and decreased progressively to minus ten percent (Figure 7.).

Prior to the run the athletes were informed once more of the safety and emergency equipment and allowed to empty their bladders. Next a heart rate monitor (POLAR®, Polar Electro Oy, Finland) and an adjustable safety harness were strapped on, and positioned according to the athlete's comfort. The subject was allowed to consume isotonic liquids throughout the two-hour run. Each subject had to complete two prolonged treadmill run sessions, at separate occasions and in random order, with and without a compression garment.

Pre-run warm up: To make sure the athletes were properly warmed up, prepared, and accustomed to the treadmill, each participant had to warm-up for five minutes. The treadmill speed was progressively increased from eight  $km.h^{-1}$  to nine  $km.h^{-1}$ .

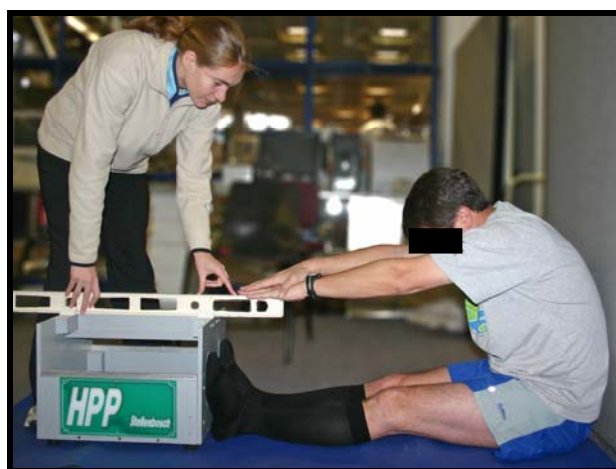
Post test recovery: To prevent blood pooling subjects were cooled down for five minutes from eight to zero km.h<sup>-1</sup>, until their heart rate dropped to 130 bpm or lower.

## 6. Flexibility and range of motion

One practical and portable flexibility test was used to assess rigidity associated with DOMS. The flexibility test was implemented at baseline (visit one) and after the two-hour treadmill run at 15 minutes, 24, 48 and 72 hours.

Modified sit-and-reach test: A portable wooden sit-and-reach box (dimensions: height 30.5 cm; width: 35.5 cm length: 43.5 cm), with a 1.21 meter detachable plastic ruler and nylon carpet, was used to assess the flexibility of the hamstrings and lower back muscles (*Figure 8*). Subjects were seated on the carpet with their legs extended and buttocks, shoulders and head against a wall. The participant's feet (without shoes) were then placed against a 30 cm high sit-and-reach box.

To start the subject was asked to reach with their arms in front of them, hands on top of one another, while the head, shoulders and buttocks pressed against the wall. They then had to inhale and slowly exhale, while the initial measurement was taken, by placing the plastic ruler at the tip of the middle finger (Dactylon). This was the starting point and was taken before each measurement.



*Figure 8.* A modified sit-and-reach hamstring flexibility test (photograph by J. Barnard)

Subjects then had to reach forward as far as possible, by sliding their fingers on top of the ruler, while maintaining their hands on top of one another, palms facing down and legs in extension throughout the test. The athlete's shoulders and head were allowed to move away from the wall. Three practice trials were given to each athlete, to correct any inaccurate technique. Three measurements were recorded at the furthest distance, where the subject could hold the position for at least three seconds. Units of measurement were in centimeters and the best of the three trials was taken as the final score.

## **7. Muscle strength and endurance**

By conducting a vertical jump test and time-to-exhaustion functional step test (TTE) explosive leg power and functional strength in the lower extremities were measured.

Vertical jump test: For this test the subject stood with the dominant hip against the wall-mounted vertical jump; this is the starting position. The subject's heels and feet were on the ground, hip-width apart. Each participant was instructed to reach up with his dominant hand as high as possible (shoulder at 180 degree extension) and to hold this position for three seconds. This is the standing-reach-height or baseline height measurement. Subsequently they were instructed to dip only their index and middle finger of the dominant hand into a chalk container. The next three jumps were recorded.

The objective was to jump as high as possible, using a double foot take-off and knee flexion, swinging the arms and touching the vertical jump panel. At the top of the jump, the subject marked the wall with the chalk on his two fingertips. The score for the jump is the difference between the baseline height ( $H_{\text{baseline}}$ ) and the height achieved at the top of the jump ( $H_{\text{top}}$ ). All three trials were recorded to the nearest centimeter and the best of the three trials was taken as the final score. The vertical jump test was conducted during the initial baseline meeting, after the two-hour treadmill run and immediately following the 30-minute lactate measurement.

Time to exhaustion step test (TTE): After the vertical jump test, the subjects progressed to the TTE test. Athletes were asked to step up and down a 25 cm Reebok aerobic step to a moderate-fast pace (152 beats per minute), set by an electronic metronome (Seiko *DM10*, Digital Metronome, Japan).

During the test participants needed to keep up with the pace or rhythm of the metronome, place their whole foot on the step (not just the balls of their feet) and they were not allowed to run. If they were unsuccessful, the stopwatch (Medalist, South Africa) was terminated and they were brought to a halt by the researcher. The objective was to continue stepping to the pace of the metronome, until fatigue set in. The test was discontinued when the subjects felt they could not adhere to the specific criteria of the test or were too exhausted to continue. The test was conducted during the initial baseline meeting (30 minutes after the maximum capacity test) as well as after the two-hour treadmill run, following the vertical jump tests and 30-minute lactate assessment, and at 24, 48 and 72 hours, with and without compression hosiery.

#### **E. STATISTICAL ANALYSIS**

Statistical analysis was performed with SPSS 14.0 Windows (SPSS inc., Chicago Illinois) and Microsoft Office Excel (Windows 2003). Descriptive data are reported as mean ( $\bar{x}$ ) and standard deviation (SD) unless otherwise specified. The possible influence of a repeated bout effect was assessed by inspecting for a learning effect. A two-way ANOVA for repeated measures were performed on all outcome variables, to assess the effect of compression garments on recovery. The main group effect and the time effect were the two independent factors, with the interaction effect (group\*time) as the dependent factor. Significant differences among mean values were identified using the Fischer LSD (*Least Significant Difference*) test. The level of significance was set at  $P < 0.05$  for all analysis.

## CHAPTER SEVEN

### RESULTS

#### A. INTRODUCTION

The present study aimed to investigate the possible influence(s) of graduated compression garments on experienced long distance runners' recovery responses after a prolonged run. A crossover controlled experimental study design was used, in which each runner acted as his own control. Selected parameters of muscle recovery were documented after a two-hour treadmill run, in a controlled laboratory environment. The parameters included lower limb oedema, perceived muscle soreness, indices of muscle damage i.e. plasma creatine kinase and lactate dehydrogenase, as well as, blood lactate concentration, and functional lower limb ability, which included strength, power, endurance and flexibility.

#### B. DESCRIPTIVE CHARACTERISTICS

##### 1. Subjects

The runners' physical characteristics and running experience are presented in *Table 1*. The subjects were middle-aged (between 36 and 51 years), club-level runners with about 13 years competitive running experience.

*Table 1.* The physical characteristics of the subjects ( $n = 7$ ).

<b>Variables</b>	<b>Mean (<math>\bar{x}</math>) <math>\pm</math> SD</b>		
<b>Age (years)</b>	43.7	$\pm$	5.5
<b>Height (cm)</b>	176.0	$\pm$	8.6
<b>Body mass (kg)</b>	92.5	$\pm$	11.8
<b>Body fat (%)</b>	24.6	$\pm$	4.0
<b>BMI (kg.m<sup>-2</sup>)</b>	29.9	$\pm$	3.0
<b>BMT (hh:mm:ss)</b>	04:04:43	$\pm$	00:16:48
<b>Training (days/week)</b>	4.6	$\pm$	0.5
<b>Running experience (years)</b>	12.6	$\pm$	7.3

BMI, body mass index; BMT, best marathon time

On average, subjects trained four to five times per week and participated in at least three marathons in the six months prior to the study. In this time the athletes' best marathon times were just above four hours, demonstrating a pace of about six minutes per kilometer.

The runners' body fat percentages on average were 4.9 % above the upper limit of the reference values for their age ( $14 \pm 1.0$  % to  $19.7 \pm 0.5$  %) and the average body mass was also 5.9 kilograms (kg) above the age predicted range ( $80.8 \pm 9.2$  kg to  $86.6 \pm 9.9$  kg).

## 2. Maximum aerobic capacity

Table 2. summarizes the maximal exercise responses to the  $VO_{2max}$  testing performed on a treadmill in the laboratory during the initial visit. All the subjects performed a maximum test in accordance with the criteria for a maximum aerobic capacity test, as indicated in Chapter six (pages 124-125). The average maximum aerobic capacity ( $VO_{2max}$  in  $mL.kg^{-1}.min^{-1}$ ) of the subjects falls within the “excellent” category with respect to gender and age (Heyward, 1998; Brooks *et al.*, 2005).

Table 2. The maximum exercise capacity of the runners ( $n = 7$ ).

<b>Variables</b>	<b>Mean (<math>\bar{x}</math>) <math>\pm</math> SD</b>
<b><math>VO_{2max}</math> (<math>mL.kg^{-1}.min^{-1}</math>)</b>	$45.7 \pm 5.0$
<b><math>VO_{2max}</math> (<math>L.min^{-1}</math>)</b>	$4.2 \pm 5.0$
<b><math>HR_{max}</math> (bpm)</b>	$177.6 \pm 6.2$
<b><math>VE_{max}</math> (<math>L.min^{-1}</math>)</b>	$146.9 \pm 14.7$
<b>RER</b>	$1.17 \pm 0.1$
<b>AT (% <math>VO_{2max}</math>)</b>	$65.9 \pm 4.3$
<b>PTV (<math>km.h^{-1}</math>)</b>	$14.6 \pm 0.5$

$VO_{2max}$ , Maximum aerobic capacity; HR, heart rate; VE, minute ventilation; AT, anaerobic threshold; PTV, peak treadmill velocity; RER, respiratory exchange ratio

## 3. The two-hour treadmill run

During the two-hour treadmill run the subjects ran at 70 % of their predetermined maximum aerobic capacity. Three of the subjects randomly wore the garments during their first two-hour treadmill run and four without. The average total distance achieved was  $18.9 \pm 1.1$  km with compression garments and  $19.0 \pm 1.2$  km without compression garments. Athletes were able to maintain an average speed of  $9.9 \pm 0.4$   $km.h^{-1}$  with compression garments (WCG) and  $10.2 \pm 0.8$   $km.h^{-1}$  without compression garments (WOCG).

There were no statistically significant differences in treadmill performance between the with and without compression garment trials. The downhill section of the run placed less stress on the athletes, since the average heart rate was lower, but the athletes ran at a faster

average pace during the 30-minute downhill section compared to the 90-minute variable gradient run (*Table 3*).

*Table 3.* Two-hour treadmill run variables, with and without the stockings ( $n = 7$ ).

<b>Variables</b>	<b>WCG</b>	<b>WOCG</b>	<b>P - values</b>
	$\bar{x} \pm SD$	$\bar{x} \pm SD$	
<b>Distance<sub>90 minutes</sub> (km)</b>	13.3 ± 0.5	13.4 ± 0.9	$P > 0.05$
<b>HR<sub>ave</sub> at 90 minutes (bpm)</b>	146.4 ± 7.4	146.5 ± 6.5	$P > 0.05$
<b>TV<sub>90 minutes</sub> (km.h<sup>-1</sup>)</b>	8.8 ± 0.3	9.1 ± 1.2	$P > 0.05$
<b>Distance<sub>30 minutes</sub> (km)</b>	5.6 ± 0.9	5.7 ± 0.6	$P > 0.05$
<b>HR<sub>ave</sub> 30 minutes (bpm)</b>	137.3 ± 7.5	139.9 ± 5.0	$P > 0.05$
<b>TV<sub>30 minutes</sub> (km.h<sup>-1</sup>)</b>	11.00 ± 0.6	11.4 ± 0.6	$P > 0.05$
<b>Distance<sub>Total</sub> (km)</b>	18.9 ± 1.1	19.0 ± 1.2	$P > 0.05$
<b>HR<sub>ave. total</sub> (bpm)</b>	141.9 ± 6.9	143.2 ± 5.7	$P > 0.05$
<b>TV<sub>Total</sub> (km.h<sup>-1</sup>)</b>	9.9 ± 0.4	10.2 ± 0.8	$P > 0.05$

TV, average treadmill velocity; HR<sub>ave</sub>, average heart rate; WCG, with compression garment (s); WOCG, without compression garment(s)

## C. DETERMINANTS OF POST-EXERCISE RECOVERY

### 1. Lower limb oedema

Swelling in the lower limbs of the athletes were assessed before the run, 40–minutes, 24, 48 and 72-hours after the two-hour treadmill run by measuring ankle, calf, mid- and proximal thigh circumferences. Reduced swelling is associated with a smaller circumference in the limb. *Figure 9.1* to *9.4* show the relative changes (%) in the lower limb circumferences from one assessment to another, with (WCG) and without compression garments (WOCG), while the absolute changes are reported in *Table 4*.

After the two-hour treadmill run swelling in the ankles, calf, mid- and proximal thigh changed statistically significantly over time in both trials from baseline to 72-hours after the exercise ( $P < 0.01$ ). In addition, no repeated bout effect was found in the ankle or calf muscle circumferences.



Table 4. The average circumferences of the lower extremities with and without compression garments from pre-exercise to 72-hours after exercise (n = 7).

Circumferences (mm)		Ankle	Calf	Mid-thigh	Proximal thigh
WCG	Pre- exercise	23.7 ± 1.7	40.7 ± 2.0	55.6 ± 3.5	60.9 ± 4.0
WOCG		23.7 ± 1.7	40.7 ± 2.0	55.6 ± 3.5	60.9 ± 4.0
WCG	40-minutes post-run	† 23.9 ± 1.7	* 41.0 ± 2.0	*55.9 ± 3.6	61.2 ± 3.7
WOCG		24.3 ± 1.6	41.4 ± 2.2	56.6 ± 4.1	61.7 ± 4.5
WCG	24-hours post-run	† 23.9 ± 1.7	† 41.3 ± 2.0	† 56.8 ± 3.5	61.8 ± 4.0
WOCG		24.5 ± 1.8	41.9 ± 2.1	57.6 ± 4.1	62.4 ± 4.1
WCG	48-hours post-run	† 23.8 ± 1.7	† 41.1 ± 2.2	~56.7 ± 3.6	61.5 ± 3.9
WOCG		24.4 ± 1.7	41.9 ± 2.2	57.3 ± 3.5	62.1 ± 3.8
WCG	72-hours post-run	† 23.8 ± 1.7	† 40.9 ± 2.0	*56.1 ± 3.4	61.5 ± 3.8
WOCG		24.8 ± 1.7	41.7 ± 2.1	56.9 ± 3.3	61.7 ± 3.6

\* P < 0.05 and †P < 0.01 Comparison of WCG and WOCG; ~P = 0.06 - 0.09 trend between WCG and WOCG

WCG, with compression garments; WOCG, without compression garments

The ankle circumferences of the subjects peaked at 24-hours in both the WCG and WOCG trials (23.9 ± 1.7 mm and 24.5 ± 1.8 mm; P = 0.001). The ankle circumferences of the runners WOCG trial were 2.4 % more elevated than the WCG, at 24-hours. The following 48-hours the ankle circumferences reached a plateau in the WOCG trials, whereas the ankle circumferences of the subjects WCG started to return to almost pre-exercising values. By 72-hours the ankle circumferences of those WCG returned to baseline, while the subjects not wearing the compression garment still showed an increased ankle circumference.

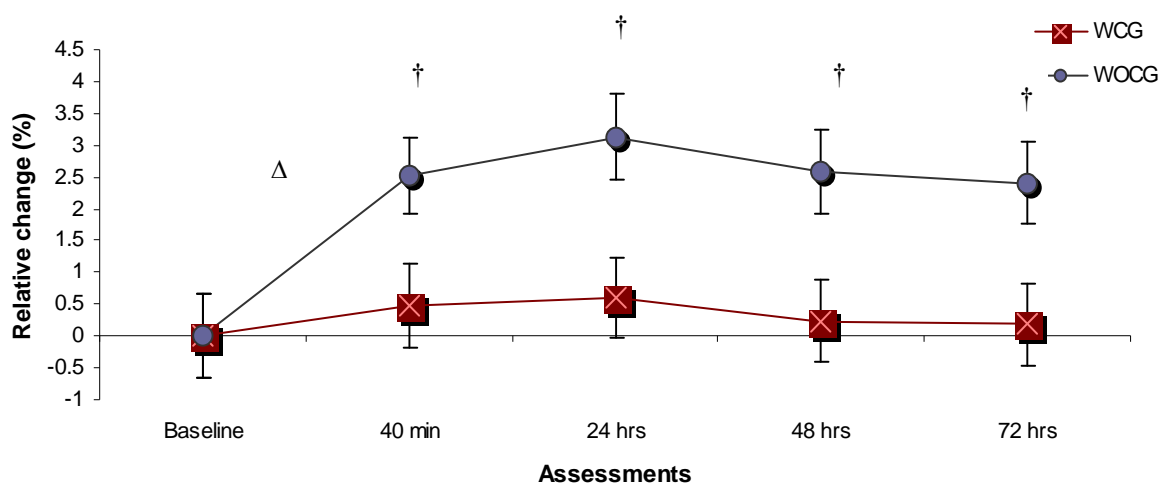
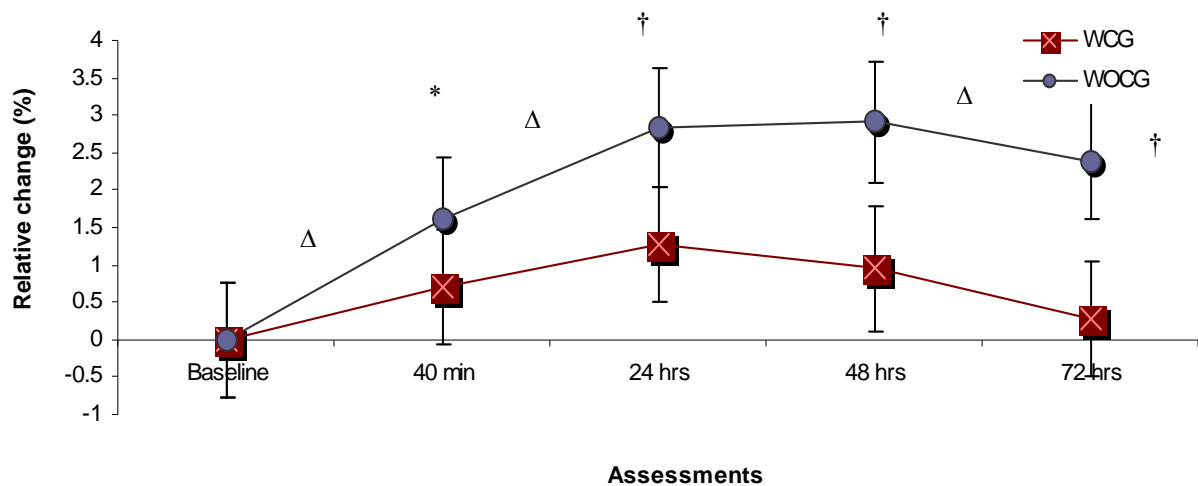


Figure 9.1 The relative change (%) in the ankle circumferences with (WCG) and without compression garments (WOCG). (†P < 0.01 WCG and WOCG; Δ P < 0.05 Change over time for all graphs).

According to the two-way ANOVA analysis, the individuals WCG showed on average a significantly lower ankle circumference compared to those not wearing the garment (*main group effect*;  $P = 0.002$ ). There was an average difference of  $1.8 \pm 1.0$  % in ankle circumferences from pre-run to 72-hours post-exercise between WCG and WOCG (*Figure 9.1*). The interaction between the compression garment's influence and recovery time shows that those with compression garments tend to have more reduced ankle circumferences (*interaction effect*;  $P = 0.09$ , NS).

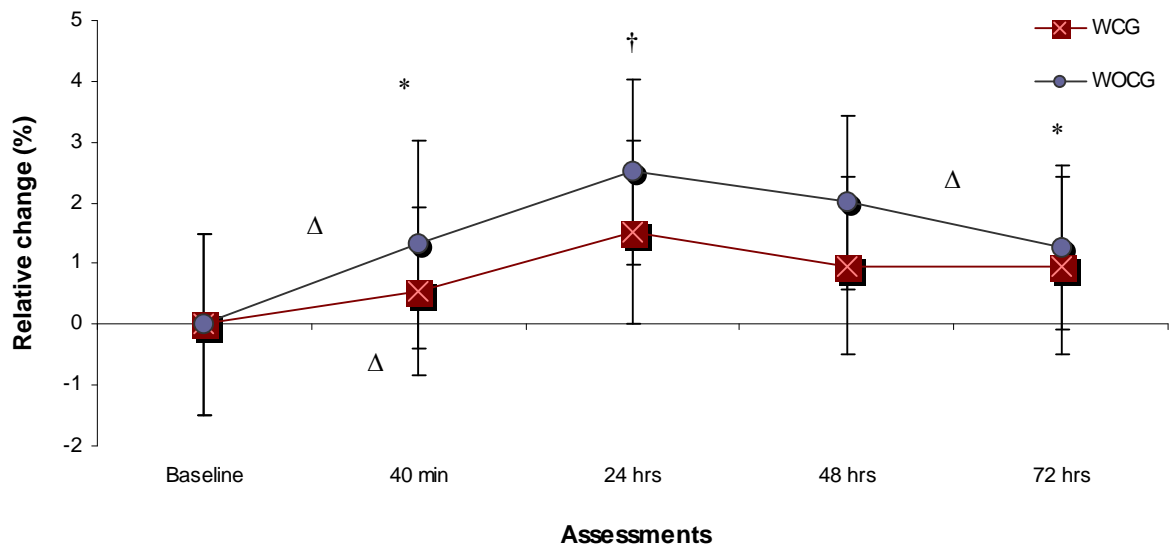
*Figure 9.2* illustrates the relative changes in calf circumferences (%). In the trials WCG and WOCG the calf circumferences peaked at 24-hours ( $41.3 \pm 2.0$  mm and  $41.9 \pm 2.1$  mm, respectively;  $P = 0.0003$ ). The subjects not wearing garments then plateau until 48 hours after the run, before starting to return to baseline. The WCG returned to baseline values ( $40.9 \pm 2.0$  mm) by 72-hours, unlike those subjects WOCG who did not return to baseline values by 72 hours ( $41.7 \pm 2.1$  mm;  $P = 0.0001$ ). A statistically significant difference exists between the average circumferences of the WCG and WOCG groups (*main group effect*;  $P = 0.002$ ). The interaction between the intervention and recovery time of the subjects WCG showed a significantly lower calf circumference, compared to those WOCG (*interaction effect*;  $P = 0.0002$ ).



*Figure 9.2* The relative change (%) in calf circumferences with (WCG) and without compression garments (WOCG). (\* $P < 0.05$ ; † $P < 0.01$  WCG and WOCG; Δ  $P < 0.05$  Change in time).

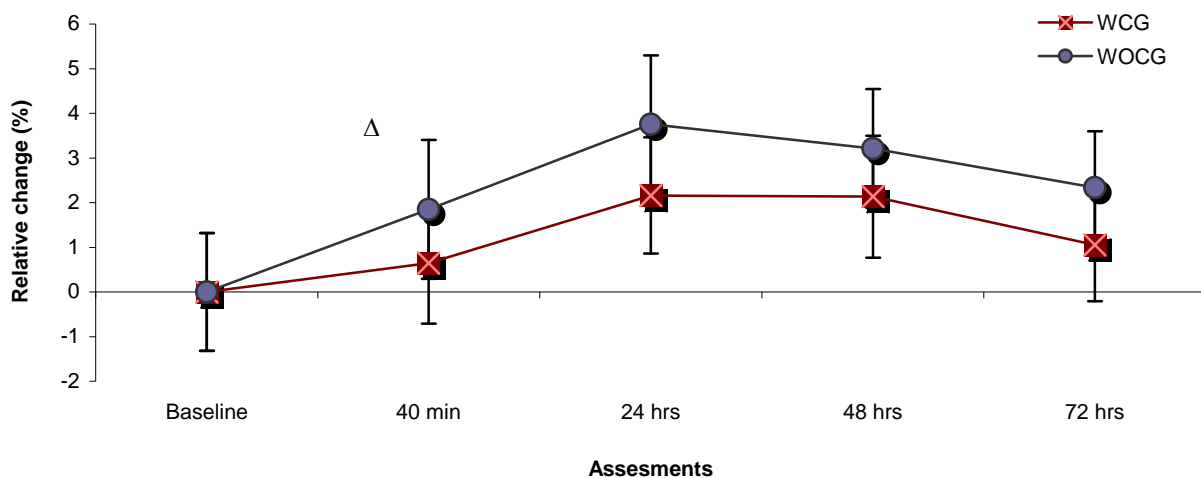
There was no significant influence of the compression garments on the recovery rate of the mid-thigh circumferences (*interaction effect*;  $P > 0.05$ ). There were, however, significantly higher average circumferences in the WOCG trials, compared to all the WCG trials (*main*

group effect;  $P = 0.02$ ). This is illustrated in *Figure 9.3*, where the WCG trials were always showing reduced circumferences, compared to the WOCG trials from 40 minutes to 72 hours after the run.



*Figure 9.3* Mid-thigh circumferences with (WCG) and without compression garments (WOCG). (\* $P < 0.05$ ; † $P < 0.01$  WCG and WOCG;  $\Delta P < 0.05$  Change in time).

Both the mid- and proximal thigh circumferences peaked at 24 hours after the run in WCG ( $56.8 \pm 3.5$  mm and  $61.8 \pm 4.0$  mm, respectively) and WOCG trials ( $57.6 \pm 4.1$  mm and  $62.4 \pm 4.05$  mm, respectively). Only the mid-thigh circumferences showed a significant difference WCG compared to WOCG at 24-hours after the run (*Table 4.*;  $P = 0.008$ ). Neither of the WCG and the WOCG mid- and proximal thigh circumferences returned to baseline by 72-hours after the run.

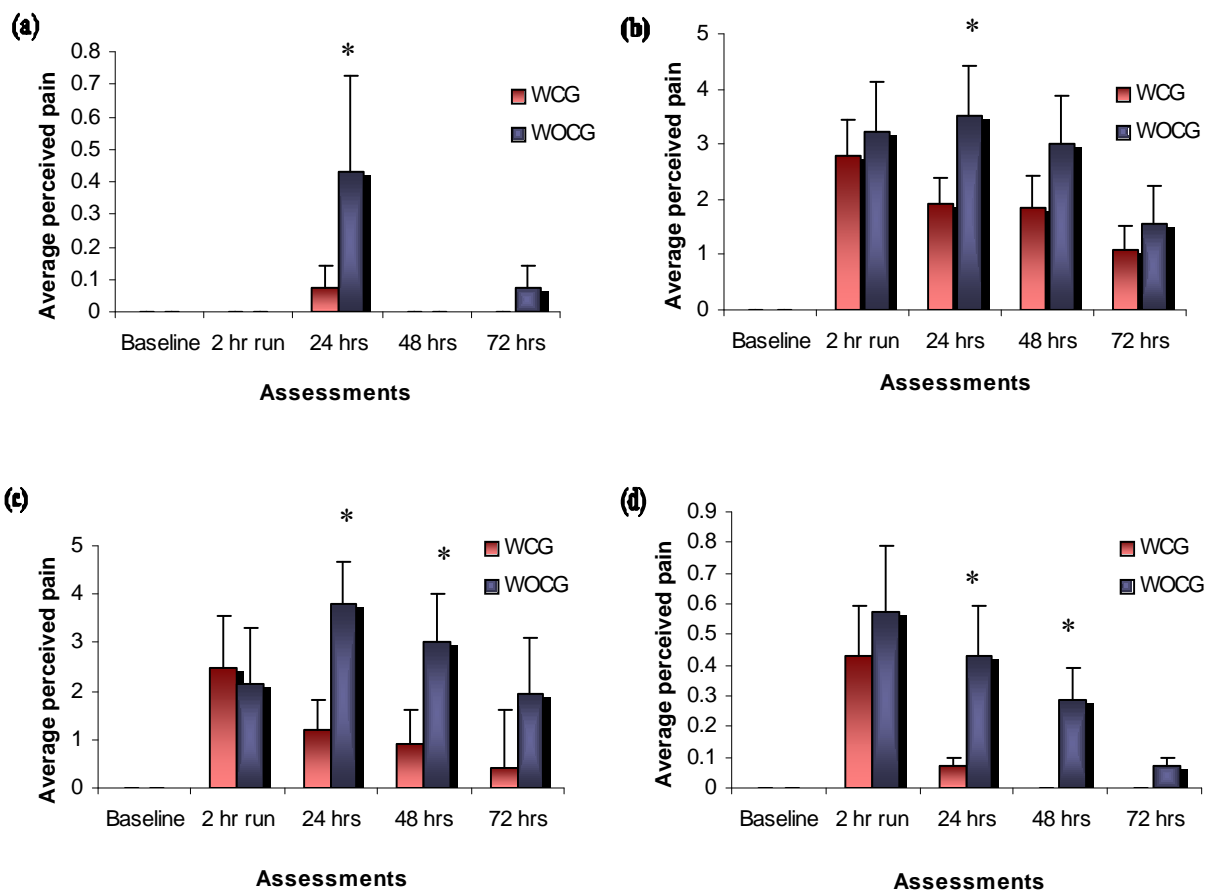


*Figure 9.4* The relative change (%) in proximal thigh circumferences with (WCG) and without compression garments (WOCG). ( $\Delta P < 0.05$  Change in time).

There was a tendency towards statistically significant difference between the average values of the two trials (*main group effect*;  $P = 0.07$ ). No effect was noticed in the WCG compared to WOCCG trial over time ( $P > 0.05$ ). Thus, from the relative changes of all the circumferences (*Figure 9.1 to 9.4*) it shows that the average circumferences were significantly more pronounced, in the ankle, calf and mid-thigh from 24 to 72 hours into recovery in the WOCCG, compared to WCG trials.

## 2. Perceived muscle soreness and discomfort

Perceptual responses of perceived muscle soreness and discomfort in the *Quadriceps* muscles were documented before the two-hour treadmill run and up to 72-hours into recovery. Perceptual responses indicated an increase from no pain experienced before the run, with the most pronounced effect between 24 and 48-hours post-exercise.



*Figure 10* The average perception of pain in *Quadriceps* throughout the trial (baseline to 72 hours post exercise), while (a) seated, (b) stretching, (c) performing

functional knee movements and (d) applying pressure. (\*  $P < 0.05$  Comparison of WCG and WOCG).

*Figures 10.1 (a) to (d)* illustrates the development of perceived pain in the *Quadriceps* muscles, while seated, with stretching, while performing lower body activities and while applying pressure to the belly of the muscle, respectively. The two-way ANOVA showed a significant change across time in the VAS data when subjects were asked to stretch (*time effect*;  $P = 0.01$ ) and to perform functional knee movements (*time effect*;  $P = 0.03$ ).

The WCG trial showed significantly lower perceived muscle pain and discomfort after performing functional knee movements, compared to the WOCG trial at 24 and 48-hours after the run ( $1.2 \pm 1.6$  vs.  $3.8 \pm 2.4$  cm and  $0.9 \pm 1.8$  vs.  $3 \pm 2.6$  cm, respectively;  $P < 0.05$ ). The two-way ANOVA disclosed a significant interaction for time and group during the functional knee movement questionnaire (*interaction effect*;  $P = 0.04$ ).

Significant differences between the WCG and WOCG trials were observed at 24-hours after the run during rest ( $0.1 \pm 0.2$  vs.  $0.4 \pm 0.8$  cm;  $P = 0.02$ ) and with stretching ( $1.9 \pm 1.2$  vs.  $3.5 \pm 2.5$  cm;  $P = 0.02$ ). The perceived pain associated with pressure was significantly lower with the compression garment at 24 (307 %) and 48-hours (237 %) after the run ( $P < 0.05$ ).

### **3. Blood analysis**

Blood samples were analyzed for enzyme markers (LDH and CK<sub>p</sub>) and blood lactate concentrations, to establish if muscle damage has occurred. LDH and CK<sub>p</sub> samples were collected before the treadmill run, 10 minutes after, and at 24, 48 and 72 hours after the run. Blood lactate samples were collected at the same intervals, as well as 3 minutes after the run. No statistically significant ( $P > 0.05$ ) repeated bout effect was found in the blood lactate and creatine kinase concentrations between the first and the second two-hour treadmill run.

#### **3.1 Blood lactate concentration**

Blood lactate concentrations significantly changed over time in both groups (*time effect*;  $P = 0.01$ ). *Figure 11.1* demonstrates the relative percentage change over the experimental

period, WCG and WOCG (from one collection point to another). The relative percentage change was calculated from one time point to another. Blood lactate concentrations reached a maximum value immediately after the two-hour run in both trials. At three minutes there was a trend for the compression garment trials to have lower blood lactate concentrations ( $P = 0.07$ ). The WCG and WOCG trials both demonstrated a plateau hereafter up until 30-minutes. At 10 minutes ( $1.8 \pm 0.5$  vs.  $2.2 \pm 0.9$  mmol.L<sup>-1</sup>;  $P = 0.05$ ) and 30 minutes ( $1.8 \pm 0.5$  vs.  $2.4 \pm 0.4$  mmol. L<sup>-1</sup>;  $P = 0.01$ ) there were significantly lower lactate levels in the circulation.

Subjects wearing compression garments had 45 % lower blood lactate levels 30-minute after exercise, compared to trials where subjects did not wear compression garments. After 30-minutes, the WCG trial illustrated a gradual return to below baseline by 72 hours, while the WOCG started to return to baseline, but at 72-hours the lactate concentrations were still elevated above baseline values. *Figure 11.1* also shows that at 24-hours after the exercise the blood lactate concentrations dropped swiftly in the WOCG trials; this is the result of one subject which indicated an extremely low blood lactate value (when the measurement was retaken, the same value was obtained).

Even though the blood lactate values after the treadmill run were lower throughout the experiment when the compression garments were worn (*main group effect*;  $28.8 \pm 17.0$  %;  $P = 0.02$ ), the compression garments did not result in a significant interaction effect (group\*time;  $P > 0.05$ ). In other words, when the two trials are compared, the mean blood lactate concentrations of the WCG trials were significantly lower compared to the WOCG trials, even though both trials started with similar pre-exercise lactate concentrations. However, the compression garment did not aid the recovery effect over the experimental period.

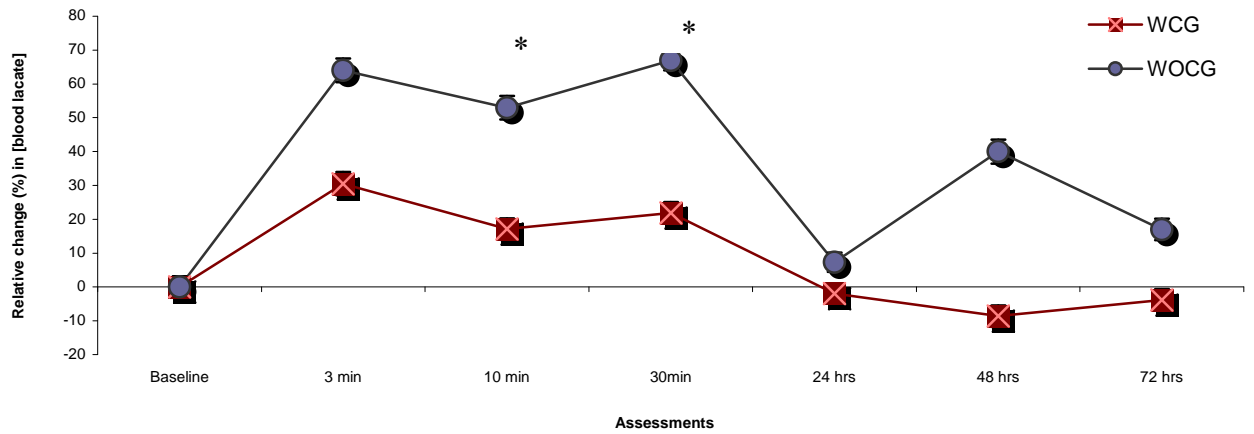


Figure 11.1 The relative percentage change in blood lactate concentrations (mmol. L<sup>-1</sup>) with (WCG) and without compression garments (WOCG). (\*  $P < 0.05$  Comparison of WCG and WOCG).

### 3.2 Muscle damage markers: LDH and CK<sub>p</sub> analysis

Both plasma creatine kinase (CK<sub>p</sub>) and lactate dehydrogenase (LDH) concentrations increased statistically significantly from pre-exercise to 72 hours after the run, in both groups (*time effect*;  $P < 0.01$ ).

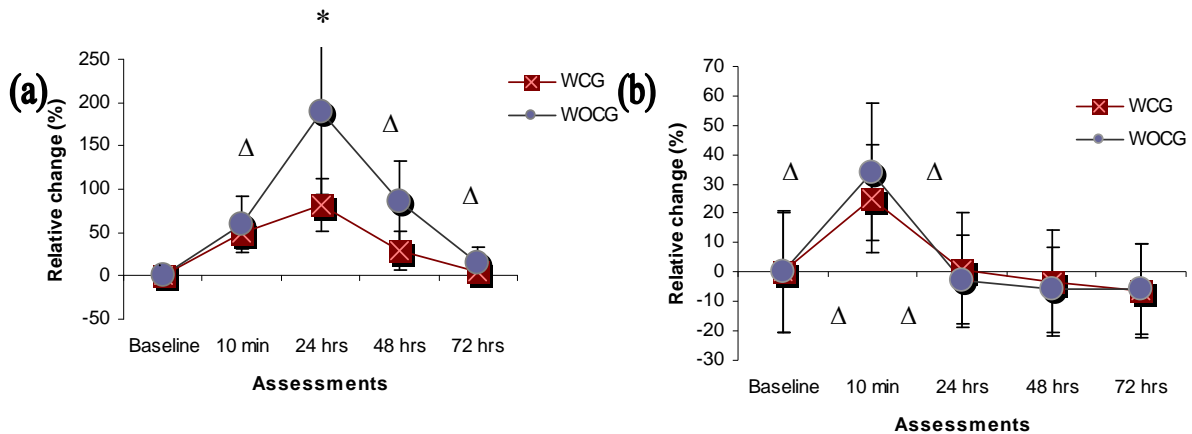


Figure 11.2 The relative change in plasma (a) creatine kinase concentrations and (b) lactate dehydrogenase levels; with (WCG) and without compression garments (WOCG). (\* $P < 0.01$  WCG and WOCG;  $\Delta P < 0.05$  Change in time).

There were no significant difference in lactate dehydrogenase concentration between the two trials (Figure 11.2 (b);  $P > 0.05$ ). The compression garments also did not have an influence on recovery over time (*interaction effect*;  $P > 0.05$ ). LDH peaked at 10 minutes after the run in both trials.

The data that are illustrated in *Figure 11.2 (a)* indicates the relative changes (%) in plasma creatine kinase concentrations. There is a 5.4 % difference from pre-exercise to 72-hours in the trials WCG, whilst the WOCG showed a 14.3 % difference. The CK<sub>p</sub> levels were on average 8.8 % lower in the WCG trials compared to the WOCG trials ( $P > 0.05$ ). WCG trials showed a tendency to return quicker to pre-exercising levels. However, there were no statistical significant differences in the CK<sub>p</sub> levels between the individuals wearing compression garments and without, throughout the test from resting to 72 hours after the run ( $P > 0.05$ ).

The only significant difference observed when the WCG trials are compared to WOCG trials was at 24 hours after the run ( $P = 0.005$ ). This was also the maximum concentration of CK<sub>p</sub> in both trials. Plasma creatine kinase peaked at 24-hours post-exercise WCG and WOCG (81.7 %,  $238.3 \pm 81.3 \text{ u.L}^{-1}$  vs. 189.3 %,  $413.3 \pm 250.8 \text{ u.L}^{-1}$ ). At 48 hours, the creatine kinase concentration in the WCG trial also showed a tendency to be lower ( $P = 0.09$ ; *Table 5*).

*Table 5.* Circulating creatine kinase and lactate dehydrogenase levels from pre-exercise to 72-hours post-exercise in subjects WCG and WOCG ( $n = 7$ ).

<b>Blood analysis (<math>\text{u.L}^{-1}</math>)</b>		<b>WCG</b>			<b>WOCG</b>			<b>P- values</b>
		$\bar{x}$	$\pm$	SD	$\bar{x}$	$\pm$	SD	
<i>Pre-exercise</i>	<b>[CK<sub>p</sub>]</b>	131.1	$\pm$ 48.3		142.9	$\pm$ 86.2	$P > 0.05$	
	<b>[LDH]</b>	155.1	$\pm$ 53.8		156.3	$\pm$ 54.8	$P > 0.05$	
<i>10-mins post-exercise</i>	<b>[CK<sub>p</sub>]</b>	194.9	$\pm$ 45.5		227.1	$\pm$ 109.4	$P > 0.05$	
	<b>[LDH]</b>	~193.4	$\pm$ 48.4		~209.6	$\pm$ 61.7	NS; $P = 0.09$	
<i>24-hours post-exercise</i>	<b>[CK<sub>p</sub>]</b>	*238.3	$\pm$ 81.3		*413.3	$\pm$ 250.8	$P = 0.005$	
	<b>[LDH]</b>	156.4	$\pm$ 51.4		152.4	$\pm$ 40.3	$P > 0.05$	
<i>48-hours post-exercise</i>	<b>[CK<sub>p</sub>]</b>	~168.9	$\pm$ 57.9		~263.7	$\pm$ 123.2	NS; $P = 0.09$	
	<b>[LDH]</b>	149.6	$\pm$ 47.5		146.9	$\pm$ 38.2	$P > 0.05$	
<i>72-hours post-exercise</i>	<b>[CK<sub>p</sub>]</b>	163.3	$\pm$ 48.9		138.3	$\pm$ 44.2	$P > 0.05$	
	<b>[LDH]</b>	145.7	$\pm$ 42.3		147.3	$\pm$ 40.6	$P > 0.05$	

\*  $P < 0.05$  Comparison of WCG and WOCG; ~ $P = 0.06 - 0.09$  trend between WCG and WOCG  
WCG, with compression garment(s); WOCG, without compression garment (s); [CK<sub>p</sub>], Plasma creatine kinase concentration; [LDH], plasma lactate dehydrogenase concentration



#### 4. Functional ability

The functional tests were intended to be non-invasive, practical and a measure of some of the fitness components relevant to running performance, i.e. muscle power, strength, endurance, and flexibility.

The absolute changes are reported in *Table 6*. and *Figure 12.1* to *12.3* show the average differences in the lower extremity functional tests, with (WCG) and without compression garments (WOCG). No statistically significant differences or improvements were found in the subjects, while wearing compression garments ( $P > 0.05$ ), compared to the trials WOCG in any of the three functional tests.

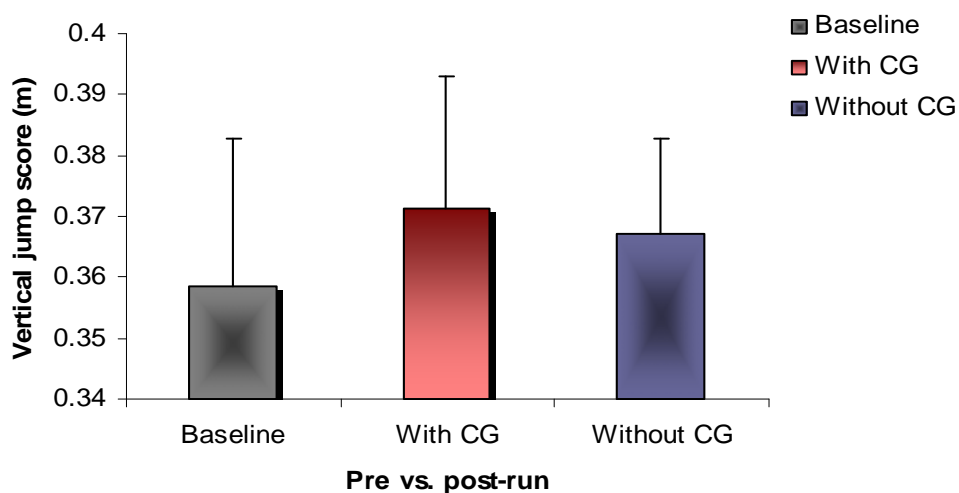
*Table 6.* Summary of functional capacity tests WCG and WOCG ( $n = 7$ ).

<i>Functional tests</i>		<i>WCG</i>		<i>WOCG</i>		<i>P-values</i>
		$\bar{x}$	$\pm$ <i>SD</i>	$\bar{x}$	$\pm$ <i>SD</i>	
<b>Vertical jump test (m)</b>	<i>Pre - exercise</i>	0.2	$\pm$ 0.2	0.2	$\pm$ 0.2	$P > 0.05$
	<i>Post- run</i>	0.4	$\pm$ 0.1	0.4	$\pm$ 0.1	$P > 0.05$
<b>TTE step test (s)</b>	<i>Pre - exercise</i>	244.8	$\pm$ 209.6	244.8	$\pm$ 209.6	$P > 0.05$
	<i>Post- run</i>	225.2	$\pm$ 250.8	207.9	$\pm$ 122.0	$P > 0.05$
	<i>24-hrs post-run</i>	228.2	$\pm$ 245.9	238.6	$\pm$ 218.5	$P > 0.05$
	<i>48-hrs post-run</i>	‡278.2	$\pm$ 234.4	‡301.9	$\pm$ 243.0	* $P > 0.05$ ; ‡ $P = 0.05$
	<i>72-hrs post-run</i>	388.5	$\pm$ 285.0	357.9	$\pm$ 288.7	$P > 0.05$
<b>Modified sit-and-reach (cm)</b>	<i>Pre - exercise</i>	32.4	$\pm$ 9.1	32.4	$\pm$ 9.1	$P > 0.05$
	<i>Post- run</i>	32.9	$\pm$ 7.4	32.1	$\pm$ 7.4	$P > 0.05$
	<i>24-hrs post-run</i>	33.0	$\pm$ 4.9	31.1	$\pm$ 3.6	$P > 0.05$
	<i>48-hrs post-run</i>	33.3	$\pm$ 5.8	32.1	$\pm$ 7.5	$P > 0.05$
	<i>72-hrs post-run</i>	34.4	$\pm$ 5.1	32.7	$\pm$ 5.1	$P > 0.05$

\* Comparison of WCG and WOCG; ‡ Change over time from baseline WCG, with compression garment(s); WOCG, without compression garment (s); TTE, time to exhaustion

##### 4.1 Lower body explosive power and strength

The two-hour treadmill run did not significantly ( $P > 0.05$ ) reduce power and strength in the lower extremities from pre-exercising values to post-exercising values (*Table 6*). Furthermore, no interaction effect was observed between the garment and recovery time ( $P > 0.05$ ). Vertical jump (VJ) scores improved 4% and 2.8% from pre-exercising scores WCG and WOCG, respectively (*Figure 12.1*). VJ scores, however, did not significantly ( $P > 0.05$ ) improve with compression garments compared to without compression garments.

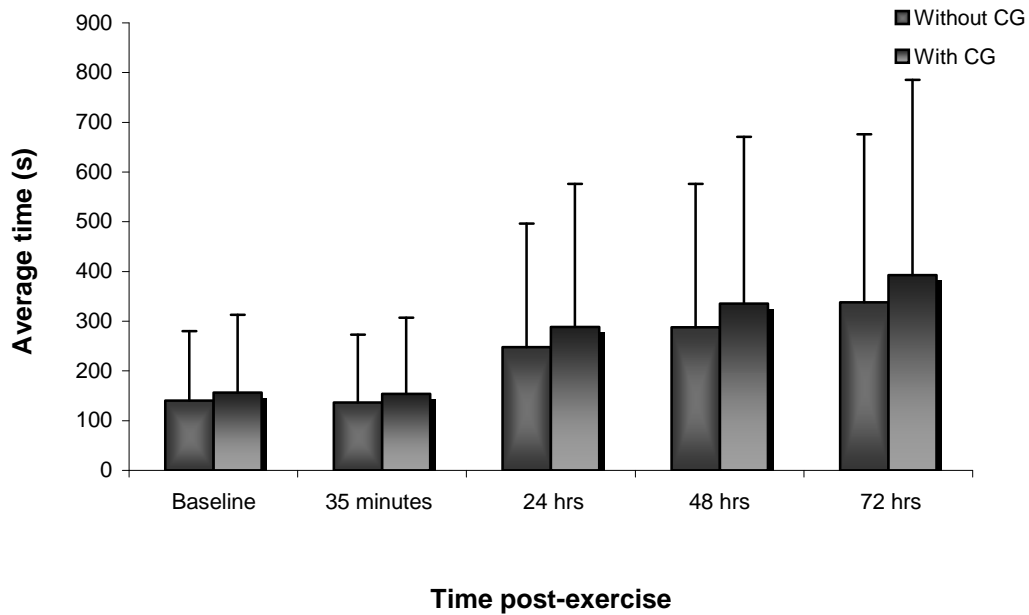


*Figure 12.1* Vertical jump tests prior to run compared to post-run with (WCG) and without compression garments (WOCG).

#### 4.2 Lower body muscle endurance

Both trials (WCG and WOCG) showed a progressive increase in the step-test time from baseline to 72 hours (*Figure 12.2* and *Table 6*). Both trials peaked at 72-hours ( $388.5 \pm 285.0$ s and  $357.9 \pm 288.7$ s, respectively;  $P > 0.05$ ). No statistical significant difference was found in the average times (seconds) between wearing compression garments and no compression garment trials (*main group effect*;  $P > 0.05$ ).

With respect to recovery, the step test did not show a statistically significant change over time from pre-exercise (baseline) to 72 hours (*time effect*;  $P > 0.05$ ). No statistically significant ( $P > 0.05$ ) interaction effect (group\*time) between the groups and time were observed.

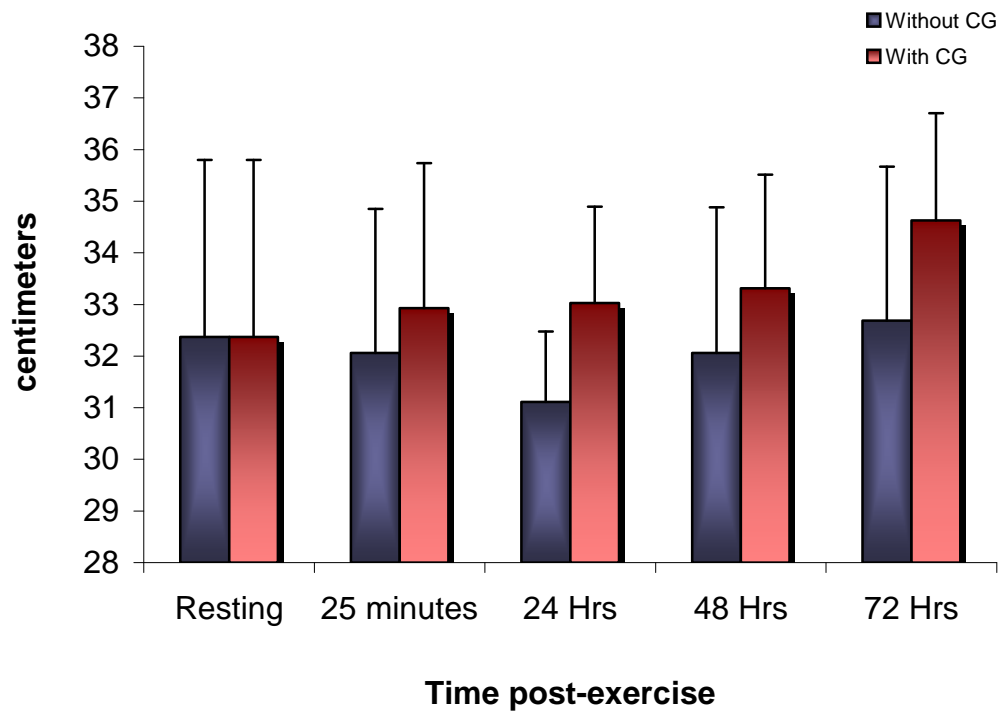


*Figure 12.2* The average measurements of the time to exhaustion (TTE) step test with compression garments (WCG) compared to without (WOCG).

#### 4.3 Range of motion

The modified sit-and-reach flexibility test was used to determine the subject's range of motion of the lower extremities. *Figure 12.3* shows that the subjects not wearing compression garments (WOCG) seems to have experienced a slightly more restricted range of motion compared to the WCG group. However, no statistically significant difference is shown between the two groups ( $P > 0.05$ ).

Whilst the subjects with compression garments (WCG) indicated a six per cent improvement in range of motion over time, peaking at 72-hours ( $34.4 \pm 5.1$  cm;  $P < 0.05$ ) after the two-hour run. The largest differences between the groups were noted at 24-hours after the run, with a 5.8 percentage difference between the trials with compression garments and without. However, no statistically significant differences were observed over time (*time effect*;  $P > 0.05$ ), as well as when the compression garments trials were compared to without the garment trials (see *Table 6. group effect*;  $P > 0.05$ ).



*Figure 12.3* The average scores in the modified sit-and-reach flexibility test, with (WCG) and without compression garments (WOCG).

## CHAPTER EIGHT

### DISCUSSION

#### A. INTRODUCTION

The current study endeavored to investigate the possible influence of compression garments on the recovery process of middle-aged long-distance runners. Compression garments are generally worn to prevent venous blood pooling, post-operative deep venous thrombosis and orthostatic hypotension and associated oedema.

The main findings of the current study are that wearing compression garments were effective in attenuating the swelling in the calf muscles of long distance runners. The compression garments reduced the swelling only to the applied limb, which suggests a reduction in the inflammatory response of trained long distance runners. Runners also experienced less perceived discomfort and pain in the *Quadriceps* muscles of the lower limb. In addition, blood lactate concentrations in the circulation were lower in the participants within the initial 30 minutes after the run with the compression garments. In addition, the DOMS protocol was successful in causing muscle damage. The results suggest that compression garments may have an influence on the acute metabolic responses by either increasing the removal of blood lactate concentration or suppressing muscle lactate. However, further research is needed to distinguish which mechanism the compression garment is responsible for.

Long distance runners are continuously exposed to strenuous training sessions and demanding races. These activities not only places stress on the cardiovascular and metabolic systems, but also cause repeated eccentric lengthening of muscles in the lower extremities. The sarcomere becomes over stretched and eventually disrupts. The structural damage to the membranes of the sarcoplasmic reticulum, transverse tubule and sarcolemma, releases calcium into the cytoplasm. Proteolysis is signaled and thus begins the break down of the muscle fibers. This is associated with the infiltration of macrophages and monocytes accompanied with oedema. The degree of inflammation in the injured area depends on the extent of the muscle damage (Kraemer *et al.*, 2004).

Delayed onset of muscle soreness is associated with several signs and symptoms, such as a dull and diffuse pain, tenderness and swelling that are generally linked with the inflammatory response (Kraemer *et al.*, 2004; Zainuddin *et al.*, 2005). Individuals also show reduced functional capacity, i.e. muscular strength, power, endurance and muscular stiffness (Cleary *et al.*, 2006; Cleather and Guthrie, 2006). These signs and symptoms may last as long as 24 to 96 hours after exercise (Cleary *et al.*, 2006). Compression garments function in a similar fashion as active recovery and mainly supports the calf muscle pump function. The compression creates pressure gradients which accelerate blood flow to the working muscles and increases the metabolic turnover of lactate.

## **B. DATA IN PERSPECTIVE**

### **1. Descriptive characteristics**

Previous studies that investigated compression garments and the role in sport recovery or performance were mainly conducted on untrained individuals or recreational athletes (Rimaud *et al.*, 2007). Furthermore, of all the studies that included highly trained individuals, only three investigated runners. None of the other studies have investigated the middle-aged long distance runner with a prolonged running protocol.

It has been noted that exercise-induced muscle soreness are magnified in older individuals (McArdle *et al.* 2001; Prentice and Voight, 2003). In this study, the runners had about 13 years of running experience and demonstrated very high  $VO_{2max}$  values for their sex and age group (Brooks *et al.*, 2005). Experienced older runners can run marathons at higher percentages (80 to 85 %) of their maximum heart rate than inexperienced younger runners (65 to 75 %). This improved ability to utilize their aerobic capacity is owing to their years of training (Mahler and Loke, 1984). The fact that more than half of the runners were in their transition phase at the time of the initial assessment, might explain why their body fat percentage is about five percent above their normal range for their age, sex and activity levels.

Furthermore, the physiological functions of experienced long distance runners have adapted optimally to years of training. In contrast, individuals with venous insufficiencies and vascular disorders have weak calf muscle-pump capacity and damaged blood vessel valves in the venous system. These individuals use compression garments to aid these

functions, which improves the venous return to the heart. However, it is questionable whether compression garments would improve the circulation of well-trained endurance athletes. These runners may have already reached their physiological limits. It is also difficult, to significantly improve the performances of trained, compared to untrained athletes (Rimaud *et al.*, 2007).

The magnitude of DOMS depends on the intensity and the duration of the exercise, as well as the physiological conditioning of the individual (Cleary *et al.*, 2006). Individuals that are accustomed to eccentric exercise such as trained athletes have a protective effect to exercise-induced muscle soreness (Kraemer *et al.*, 2001<sup>a</sup>). The two-hour treadmill run at 70 % of  $VO_{2max}$  together with the 30-minute eccentric downhill run was chosen to provide a potent stimulus to induce DOMS. This was a unique protocol, which have never before been attempted. The objective was to simulate the forces that act on the runner, during a race. No statistically significant differences were shown between the WCG and WOCG trials for distance, average heart rate or treadmill velocity, which shows that the exact same protocol was followed during both sessions.

During the downhill run, the runners' heart rates were lower than during the 90-minute variable gradient run. This is not uncommon during exercise involving predominantly eccentric muscle actions, which demonstrates less stress on the cardiovascular as well as on the local metabolic system. The eccentric muscle contraction is metabolically less demanding and recruits fewer motor units than concentric contractions. The evidence for this is that concentric exercise produces more lactate and a higher heart rate compared to eccentric exercise (Carrasco *et al.*, 1999; Eston *et al.*, 2000).

## **2. Post-exercise recovery**

Much is known about the exercise responses and training adaptations of endurance athletes, yet very little information is available on the recovery after races and training. The ability to recover quickly after bouts of strenuous exercise is beneficial for the athlete.

## 2.1 Lower limb swelling

Reduced swelling in lower extremities is the result of the mechanical compression exerted by the garment (Kraemer, *et al.*, 2001<sup>a</sup>). Swelling is caused by a difference in the osmotic gradient and is typically associated with inflammation. The compression garment reduces the swelling in the lower extremities by exerting mechanical pressure onto the limb. The mechanical compression neutralizes the osmotic pressure imbalance by increasing the tissue hydrostatic pressure. Additionally, the compression garment also minimizes the available space for fluid accumulation (Brennan and Miller, 1998; Harris, *et al.*, 2001; Kraemer, *et al.* 2001<sup>a</sup>). Limb circumferences have been used in several studies to assess the efficiency of compression garments in the removal or prevention of oedema (Gniadecka *et al.*, 1998; Kraemer *et al.*, 2001<sup>b</sup>). Numerous studies have reported that compression garments lessen oedema in the extremities of healthy and venous insufficiency populations.

In the current study, swelling in the lower extremity followed the classical pattern of exercise-induced muscle damage. Swelling in the ankle, calf, mid- and proximal thigh of both trials peaked 24 hours after the two-hour run. Only the calf muscle showed a significant reduction in swelling with the compression garments trials. This is noteworthy, since the calf muscle plays an important role during running activities and is prone to muscle damage (Bringard *et al.*, 2006<sup>a</sup>). Hence, the reduction calf circumferences would indicate the possible onset of the inflammatory response (Miller *et al.*, 2004; Kraemer *et al.*, 2004). There was also a strong trend ( $P = 0.09$ ) for the ankle circumferences of the runners with the compression garment to be lower. In addition, when the mean ankle circumferences between the treatment groups were compared it definitely showed a more reduced ankle circumference with the compression garment ( $P = 0.002$ ).

No significant attenuation of limb swelling was noted between the mid- and upper thigh. A possible explanation for no effect in the upper thigh is that the compression garment was only applied to the lower leg (ankle and calf). Similar to this study, Kraemer *et al.* (2000) found no significant lower upper thigh circumferences when the authors investigated standing fatigue in younger healthy women wearing three different types of low pressure compression socks. However, the compression garments did significantly reduce the ankle and calf ( $P < 0.05$ ). Kraemer *et al.* (2000) speculated that the low pressure exerted by the garment was ineffective for the larger muscle mass limbs, or that long standing activities



do not result in oedema in the thighs. However, it is well known that downhill running induces muscle damage largely in the knee extensors. Thus, it seems that the compression garments did not have had an influence on the upper thigh circumference.

Gniadecka *et al.* (1998) reported no significant difference in ankle circumferences between two types of compression garments compared to wearing no compression garment i.e. CLASS I ( $23.7 \pm 2.1$  vs.  $23.3 \pm 1.8$ cm) and CLASS II ( $23.7 \pm 1.8$  vs.  $23.3 \pm 1.6$  cm). A higher compression at the ankle may have produced more significant results in the current study.

## 2.2 Perceived muscle soreness and discomfort

Both the WCG and WOCG trials demonstrated some of the common symptoms associated with DOMS in the *Quadriceps* muscles, such as pain and tenderness in the damaged muscle when the belly of the muscle was palpitated, contracted and stretched (Ball and Herrington, 1998; Zainuddin *et al.*, 2005). Downhill running causes muscular damage primarily in the *Quadriceps* muscles. Furthermore long distance runners typically demonstrate muscle pain and discomfort in the *Quadriceps* and *Gastrocnemius* muscles (Lambert and Borresen, 2006). Therefore, visual analog scales were used to indicate the awareness of muscle soreness and discomfort in the *Quadriceps* at rest, while stretching, with functional knee-movements and when applying pressure.

Even though the VAS questionnaires are subjective and not a quantitative approach, when compared in conjunction with the circumferences, the muscle soreness and discomfort experienced during rest, stretching and functional knee movements in the WOCG trials closely resembled the fluid dynamics of the swelling in the lower limb. Whereas with the WCG trials, the runners experienced the most muscular pain directly after the two hour run while stretching, applying pressure and performing functional knee movements and then demonstrated a gradual decline in muscle soreness during the following days. In addition, these signs and symptoms are associated with exercise-induced muscle soreness (Gleeson *et al.*, 1998). Thus, a reduction in swelling together with a lower perception of pain would indicate a diminished inflammatory response (Miller *et al.*, 2004; Kraemer *et al.*, 2004).

The results of the two-way ANOVA indicated that only during stretching and with functional knee movements did the perceived pain response significantly changed over

time. The pain and discomfort associated with the functional knee movement was significantly less when runners wore the compression garment. There was furthermore, a significant intervention effect between the compression garment and perceived muscle soreness during functional knee movements. Similarly, when the runner's damaged *Quadriceps* muscles were palpitated, the trials with the compression garment demonstrated significantly less pain compared without the garment at 24 and 48 hours after the run. This is an important feature since, as mentioned before, the downhill run cause the most muscle damage in the knee extensors. The lower perceived pain during knee flexion and extension shows that compression garments are beneficial in reducing exercise-induced muscle damage.

These findings are in agreement with those of Ali *et al.* (2007) who observed that recreational runners wearing compression garments perceived less muscle soreness at 24-hour after a 10 kilometer run. Other studies have also found less perceived muscle soreness with the compression garment trials, although the differences were not always significantly (Chatard *et al.*, 2004; Trenell *et al.*, 2006; Miller *et al.*, 2004; Kraemer *et al.*, 2001<sup>b</sup>). Reduced pain may be an indication of reduced swelling in the lower extremities. This reduced swelling means that the compression garment attenuates the inflammatory response. If athletes indicate a weakened perception of muscle soreness, it may signify that the compression garment facilitated the inflammatory response.

The athletes were not informed of the real objective of the study. Nevertheless, the placebo effect cannot be dismissed. It may therefore be argued that the reduced perception of muscle soreness were due to the participants expectations and not as a result of the garment.

Thus, wearing compression garments during running and for three days after the prolonged two-hour treadmill run appears to reduce symptoms of delayed onset of muscle soreness in trained runners. This may be beneficial for all runners, since reduced muscle soreness after exercise will allow the athlete to return to training and races sooner.

### 2.3 Blood lactate concentration

The energy cost over time is an important determinant of exercise tolerance in both patients and athletes. When an athlete reduces his running cost, he is able to run for longer

and tolerate higher running demands and may also experience less fatigue. Some factors have been proposed as possible predictors of running cost, such as blood lactate concentration, cardiorespiratory work, the oxygen available to the muscle and motor-unit recruitment patterns (Bringard *et al.*, 2006<sup>b</sup>). Therefore, a reduced blood lactate concentration might be related to a lower energy cost over time. According to Dawson *et al.* (2005), an accelerated lactate removal is considered as an indication that metabolic recovery has taken place.

However, the rate of lactate clearance from the circulation is the function of both metabolic and circulatory dynamics (Rimaud *et al.*, 2007). Chatard *et al.* (2004) and Rimaud *et al.* (2007) suggest that a lower blood lactate concentration may be beneficial for subsequent exercise, especially after strenuous activities. Participants with DOMS typically demonstrate elevated maximal and submaximal blood lactate concentrations. This is primarily owing to the more permeable membrane and the elevated metabolic stress placed on the undamaged muscle fibers. In addition, it may also be possible that more type II muscle fibers are recruited. This increases the rate of glycogenolysis, which in return also contributes to an elevated lactate concentration in the circulation (Gleeson *et al.*, 1998; Semark *et al.*, 1999). The present study found that when runners were wearing compression garments blood lactate concentrations were significantly lower at 10 and 30 minutes during the recovery period after the run. The trials with the compression garment had a tendency to cause lower blood lactate levels after the initial three minutes ( $P = 0.07$ ), compared to the WOCG trials. No significant differences were observed at 24 to 72 hours after the run. There was an outlier at 24 hours after the run, however the training diary of the specific did not reveal any deviation to the protocol, nor when the measurement was retaken was there any difference found.

Other studies have also found that the compression garments had an influence on lactate clearance during the acute recovery period. Berry and McMurray (1987) assessed young well-trained runners (part I) and cyclists (part II) for 60 minutes after a maximal test. The participants wearing the compression garments showed lower blood lactate levels in both parts of their study. Similar to this study, they found a significantly lower lactate concentration at 15-minutes post-exercise with compression garments in the runners. However, the second part of their investigation found significantly lower blood lactate concentrations when wearing graduated compression garments in the cyclist, across time (60 minutes).

Chatard *et al.* (2004) found that compression garments reduced blood lactate concentrations, as well as cleared lactate faster (by 20 %) in elderly cyclists wearing compression garments, during an 80-minute recovery period. Rimaud *et al.* (2007) found significantly lower blood lactate concentrations in well-trained low-level spinal cord injured participants, which wore compression garments at three minutes after the maximal exercise bout.

As explained before, the compression garment exerts a force on the superficial veins and limits dilation of the peripheral venous system. By reducing the blood vessel's diameter the blood is shunted from the periphery towards the deeper veins, thereby increasing the venous return. This leads to two possible theories of how compression reduces blood lactate concentrations, i.e. (i) either the compression accelerates venous blood flow, which aids lactate clearance from the muscle and increases oxidation of lactate, or (ii) the force reduces the diffusion medium available for lactate, inorganic phosphate and oxygen transport. Consequently, the compression garment retains the lactate in the muscle (Berry and McMurray, 1987; Chatard *et al.*, 2004; Rimaud *et al.*, 2007). Unfortunately, blood flow, muscle lactate concentrations, and oxygen consumption were not assessed during the current study. Therefore, one may only speculate which one of the processes were responsible for the reduced blood lactate concentrations.

The lack of a significant interaction effect for the changes in blood lactate concentration may be related to the trained athlete's well-developed physiological systems. For instance, in patients with blood vessel disorders the calf muscle-pump function (CMPF) and venous valves may be compromised. By wearing the compression garments the pressure exerted functions in a similar fashion as the CMPF, thus improving venous return (Bringard *et al.*, 2006<sup>a</sup> and 2006<sup>b</sup>). With experienced long distance runners their CMPF is well developed, which could indicate that the compression garment did not add any additional benefits to their all ready well-developed circulation.

#### 2.4 Muscle damage markers

Plasma creatine kinase is the most sensitive marker of muscle damage and is typically increased after exercise and peaks between 24 and 48 hours after exercise. The significantly elevated plasma creatine concentrations above baseline over the 72-hour

recovery period in this study ( $P < 0.01$ ), demonstrates that muscle damage occurred in both groups due to the two-hour treadmill protocol. The initial two-hour treadmill run did not have a protective effect on the muscle during the second two-hour run (repeated bout). McHugh (2003) suggests that the muscle action's intensity must be close to maximum during the initial eccentric bout to result in a protective mechanism for the following eccentric bouts. The runners had to run at a moderate pace and not at maximum (70 % of their own maximum aerobic capacity).

Achieving statistical significance when measuring CK concentration is difficult, because of the high inter-subject variability in CK response. This might be due to (i) the variability in exercise-induced muscle damage and/or (ii) membrane permeability (Stupka *et al.*, 2000). Stupka *et al.* (2000) and Laursen *et al.* (2007) found large variations between subjects' creatine kinase concentrations. Laursen proposed that individuals could be divided into CK<sub>p</sub> responders and non-responders, and that this is a possible reason for inter-subject variability in creatine kinase concentrations.

Even though the CK<sub>p</sub> responses were less pronounced in the trials with the compression garment compared to the without compression garments, it was only significantly lower at 24 hours after the two-hour run. This is consistent with the results of the visual analog scales, which also demonstrated maximum muscle soreness in the *Quadriceps* and a significantly lower perception of muscle soreness in the trial with compression garments at 24 hours.

There are three suggested mechanisms by which compression garments attenuate muscle damage. By either (i) releasing fewer muscle damage markers (creatine kinase or LDH) from the damaged muscle's ultrastructure, (ii) accelerating the clearance of the muscle damage markers like creatine kinase from the injured area or (iii) the mechanical support of the compression garment reduces muscle oscillation. This results in a reduced muscle fiber tearing and damage (Doan *et al.*, 2003; Bringard *et al.*, 2006<sup>b</sup>).

Previous studies have reported that creatine kinase concentrations may be 20-fold higher, compared to resting levels, after a marathon race (Mahler and Loke, 1984). However, the creatine kinase concentrations in the present study were not as high as in other studies. This is possibly because the runners were trained long distance runners and might have

been partially accustomed to, or have adapted to, downhill running. This is consistent with the findings of Takahashi *et al.* (2006).

Kraemer *et al.* (2001<sup>b</sup>) also showed an elevated creatine kinase concentration in the circulation in untrained men WCG and WOCG, but it was more pronounced at 72 hours after the eccentric exercise WOCG. By 72 hours, in the current study, CK<sub>p</sub> was already reduced to resting levels in both trials. This indicates that trained runners demonstrate a reduced CK<sub>p</sub> response compared to untrained individuals. Duffield and Portus (2007) only found a significant reduction in plasma creatine kinase with the compression garment after 24 hours in trained cricket players. This reduction in creatine kinase concentration was consistent with the lower perceived muscle soreness at 24 hours, experienced by the cricket players. Semark *et al.* (1999) also maintained that creatine kinase and pain responses in trained athletes were not as pronounced as in untrained individuals during DOMS.

Additionally, subjects were allowed to consume water voluntary during the treadmill run and it has been suggested that additional plasma CK may be attenuated by consuming fluids (Sanchez *et al.*, 2006). Another possible reason given is that athletes were requested to reduce their training volume and intensity (below 70 % of maximum heart rate) seven days prior to the two-hour treadmill run. As mentioned before, plasma creatine kinase levels are reduced when endurance athletes taper seven days prior to a half marathon event (15% less than their normal training volume) (Myburgh, 2003). However, even though the creatine kinase were not as elevated as seen in previous studies, all of the CK<sub>p</sub> measurements were elevated above the normal range and did indicate muscle soreness.

Lactate dehydrogenase (LDH) was also significantly elevated above baseline values. However, no differences were found between those WCG and those WOCG. Lactate dehydrogenase is not as sensitive muscle damage marker as plasma creatine kinase. This is consistent with findings of Kraemer *et al.* (2001<sup>a</sup>) who also observed an elevated plasma creatine kinase activity with no significant change in LDH after an eccentric exercise protocol in the upper arm. The different responses in CK<sub>p</sub> and LDH might be partly related to the structurally different areas in the muscle sarcomere from where it is released. Both enzyme markers depend on the site of primary mechanical muscle damage (Kraemer *et al.*, 2001<sup>a</sup>).

## 2.5 Functional ability

Contrary to what was expected, neither of the two trials resulted in a change over time or differences between WCG and WOCG in any of the functional tests. Compression garments and the protocol had no influence on muscle endurance and flexibility throughout the experiment. Unlike Kraemer *et al.* (2001<sup>b</sup>) that found a significant reduction in elbow ROM in both groups after exercise (men), the current study found no significant reduction in ROM. In Kraemer *et al.* (2001<sup>b</sup>), the compression garment group gradually regained their ROM within 24 and significantly by 72 hours. The TTE and modified sit-and-reach test were chosen for their practicality and mobility. However, the inter-subject variability in these tests, including the vertical jump test, is a clear indication that the tests were not sensitive measures for changes in lower body endurance or flexibility.

In addition, muscular power and strength assessed by the vertical jump test also did not show significant differences with or without the compression garment. Kraemer *et al.* (2001<sup>b</sup>) found no reduction in force production after an eccentric protocol in a WCG group of untrained men. Performance, however, decreased over the 48 en 72 hours during recovery in the control group. The current study did not assess muscle power and strength during the three-day recovery period.

Possible reasons for the findings of this study are (i) that the tests that were chosen were not effective measures of functional ability, (ii) the endurance athletes have adapted to prolonged exercise and did not experience muscular fatigue or reduced range of motion, or (iii) the runners pushed through the pain-barrier.

Although the downhill protocol resulted in muscular damage, this does not mean that the runner's functional ability was diminished. Depleted glycogen stores, among other factors, could have caused fatigue. The rate of glycogen depletion is proportionally related to the intensity of the exercise, the initial glycogen content, and the type of predominant muscle fibers. For instance, type I fibers are depleted first when running at 70 % of one's  $VO_{2max}$  and only at a faster pace the type II fibers are depleted. Additionally, athletes running at 70 to 80 % of their  $VO_{2max}$  will only deplete their glycogen stores at 32 to 40 kilometers (km) (Mahler and Loke, 1984). The runners in this study ran at 70 % of their  $VO_{2max}$ , however they only ran about 19 km in both trials. The runner's well-developed calf muscle-pump may also have aided the recovery of force production.

To complete long distance races endurance athletes have to be highly motivated and determined individuals. It has been documented that experienced runners show a better capacity to deal with pain (Mahler and Loke, 1984). During the TTE step test a psychological aspect might also have contributed to the runner's ability to push through their perceived pain and to continue with the exercise.

Even though the current study did not set out to investigate whether compression garments could contribute to muscular fatigue, the results suggest that the higher pressures exerted by the garment used in our study did not result in muscular fatigue. Previous studies have suggested that too high a pressure (30 to 50 mmHg) exerted by the compression garment will result in muscular fatigue, due to ischemia in the muscle. Maton *et al.* (2006) investigated muscular fatigability during sustained muscle contraction and whether compression garments improve recovery after fatigue. They used compression garments that exerted lower pressures (23.6 to 6.8 mmHg) than the current study (32 to 23 mmHg). Maton *et al.* (2006) found no statistically significant difference in the individuals' ability to generate force, between the WCG and WOCG. Their conclusion was that compression garments did not cause muscular fatigue, possibly because the added pressure of the compression garment is negligible in comparison to the pressures exerted by the contracting muscle.

The compression garments in this study may not have resulted in fatigue for the similar reasons. Maton *et al.* (2006) studied healthy subjects, while the trained endurance athletes in this study have well-developed muscle-pump functions. Thus, the higher pressure of the garment may not have been enough to restrict blood flow to the muscles, compared to the forceful contractions of the calf muscle.

### **C. STUDY LIMITATIONS AND FUTURE STUDIES**

The current study was limited by the small sample size, which may be the reason why some results did not reach statistical significance. Future studies of small sample sizes should include a retrospective power analysis. Furthermore, data was collected from personal reports (VAS and training history) and are therefore potentially less reliable than the laboratory-measured data. However, there was no reason to doubt the training data or subjective opinions reported by any of the runners.



This study would be strengthened by the inclusion of additional recovery determinants such as haematocrit, C-reactive protein, and plasma volume, muscle lactate concentrations, heart rate, and post-exercise oxygen consumption. The plasma volume should be incorporated to correct creatine kinase concentrations, for the shifts in plasma volume. C-reactive protein is an inflammatory marker, which will allow scientist to more accurately determine if compression garments aided the inflammatory response. Assessing muscle lactate concentrations will allow the sport scientist to identify the mechanism by which compression garments lower blood lactate concentrations. In addition, the psychological influence of the compression garments needs further investigation.

Previous studies have indicated that the amount of pressure exerted by the compression garment will have a different influence on the various physiological functions. Maton *et al.* (2006) observed that there is a large mean pressure difference between subjects, which demonstrated that the effectiveness of the garment strongly depend on the subject's morphology. Other studies mainly used compression garments with lower pressures of about 23 to 16 mmHg. This study used a higher compression garment and indicated very little negative effects on endurance-trained athletes. One possible question for future research is to investigate what pressure would exert effective or optimal compression for trained athletes in various types of sporting activities. Optimal pressure would result in improved athletic performance and aid recovery post-exercise, without side effects.

Compression garments have become increasingly popular among athletes and have been extensively marketed by the media. Therefore, the placebo-effect can not be ignored. As there was no placebo condition in the current study, it could be argued that some of the findings were as a result of the participants' intuition of expected findings. However, this was a randomized trial and participants were never informed of the exact objectives of this study, merely that more research is needed on the recovery process of long distance runners. Future studies might have to include a garment that exerts no compression as a placebo.

Eccentric activities to induce DOMS may differ from dramatic or sudden acute injuries, such as strains and sprains. In addition, treadmill running reduces the maximum impact force experienced by runners, compared to over ground running on hard surfaces such as tarmac (Ali *et al.*, 2007). Most studies on runners and compression garments, except Bringard *et al.* (2006<sup>b</sup>), were performed in laboratories. Future studies that investigate the

benefits of compression garments while the athlete wears the garment during the actual event need more attention. No study thus far has been performed on long distance runners while wearing compression garment during events. Furthermore, Bennett (2006) commented that post-match muscle damage in rugby is more pronounced than post-training, and that no study has been performed on post-training recovery.

Reasons for the possible inconsistency in findings among the various studies are the different eccentric models that were used, which resulted in different magnitudes of damage to different muscles. Individuals also showed a wide variation in response to the same exercise protocol. Additionally, different modalities may differ from each other, i.e. cycling versus running. This large variability in responses among individuals to the effects of eccentric exercise and various modalities has made comparison with control conditions and other studies difficult. More research on compression garments as a possible recovery strategy is needed.

#### **D. CONCLUSION**

Very little research has been performed on compression garments as a recovery aid and even less on experienced older trained athletes. To date the effects of compression garments on athletic performance have been mainly investigated during power sports and after supramaximal exercises. Very little research has been done on compression garments and its effect on submaximal running exercise. This study was unique, in that it addressed a population that has been overlooked by previous studies. Middle-aged runners are currently the most popular age group in long distance events. These runners would benefit immensely from a practical recovery modality, such as compression garments.

The results of this study showed that long distance runners experienced muscle damage after a two-hour treadmill run and that the compression garments reduced muscle soreness at 24 hours after the run, compared to the runners not wearing compression garments. The evidence for this is the reduced swelling, creatine kinase concentration and perceived muscle soreness. Blood lactate concentration was also lower with the compression garment during the acute recovery phase after the run. In addition, the higher pressure of the garments did not lead to muscular fatigue. However, further research which could more specifically identify whether compression garments aid lactate clearance or suppressed muscle lactate is warranted.

It seems, therefore, that compression garments have the potential to enhance recovery after strenuous exercise and may thus provide athletes with an easy, inexpensive recovery option after training sessions or competitions.

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## APPENDIX A

### THE CONSENT FORM

#### INFORMED CONSENT

**Title of the research project:** *“Investigating long distance runners’ recovery rate.”*

**Reference number:** \_\_\_\_\_00000\_\_

#### STATEMENT BY THE RUNNER:

I, the undersigned, \_\_\_\_\_ [ID \_\_\_\_\_],  
of (address) \_\_\_\_\_

\_\_\_\_\_  
\_\_\_\_\_

#### Confirm that:

1. I was invited to participate in the above mentioned research project that is undertaken by the **Sport Science Department** at **Stellenbosch University**.
2. It was explained to me that:
  - 2.1 the aim of the study is to collect data on long distance runner’s recovery rate.
  - 2.2 from the project I will receive interesting information on my general fitness level that can be beneficial to improving my sport performance.
  - 2.3 I will receive a voucher for a maximal aerobic capacity test with lactate threshold measurement and a 30–minute sports massage, only if I complete the study. The voucher is valid until 30 November 2007.

- 2.4 If I receive additional equipment or gear from the Sport Physiology laboratory, during the study, I will return it to the University of Stellenbosch at the termination of the study.
- 2.5 I will participate in a baseline test, followed by a 7 to 10 days recovery period, in-between the second and baseline assessment. The sixth follow up test, must be completed within 7 to 28 days, after the fifth testing session.
- 2.6 The second assessment, consist out of a two-hour simulated mountainous run on a treadmill, inside a laboratory, followed by three consecutive days of collecting blood and lactate samples, with follow up tests (same as baseline tests).
- 2.7 The sixth assessment session is the follow up tests of the second assessment. This is a repeat of the test performed in the second evaluation session, along with three consecutive days of collecting blood and lactate samples, with follow-up tests (same as baseline tests).
- 2.8 During the seven days prior to the second and third assessment sessions, I will record a logbook of my daily activities and hand it in to the researcher at the next testing date.
- 2.9 I will not take any medication two days prior to or during the tests.
- 2.10 I will visit the Sport Physiology Laboratory possible on three to nine different occasions. During these visits, the following tests will be done:
  - 2.10.1 during the first visit I will complete a questionnaire regarding my health status and activity level, as well as, muscle soreness and fatigue questionnaire. I will undertake the first baseline test: body impedance with the Body stat (fat percentage and hydration levels), vertical jump - (muscle strength), TTE step test (time to exhaustion endurance test), flexibility tests, maximum aerobic capacity tests (oxygen consumption). My height, weight, calf-, mid-thigh, proximal thigh and ankle circumferences, and diameter as well as leg length will be measured.
  - 2.10.2 during the second and sixth visit, I will be retested. Blood samples will be taken for lactate and enzyme markers (this involves finger pricks and drawing blood samples by a

qualified phlebotomist). Some of the baseline tests will be repeated along with a 90-minute mountainous run and a 30-minute downhill run, in a laboratory on a treadmill.

- 2.10.3 24, 48 and 72 hours after the second and sixth assessment, more blood samples will be collected, along with certain of the baseline tests.
- 2.10.4 I will try my best to come to the Sport physiology Laboratory at 24, 48 and 72 hours after the second and sixth assessment sessions to give blood samples. However, if I cannot make it to the laboratory for some unforeseen reason, I will be available for a sport physiologist to come to me.
- 2.11 I will only perform **low to moderate intensity exercises**, by maintaining my heart rate below 70 % of my maximum or **long slow distance training (LSD's)**, in-between my baseline and second assessment, as well as the seven days prior to the second and sixth assessments. I will run no hilly or mountainous trails and/or perform lower body weight training, during this period. I will not participate in any high intensity training the seven days prior to the second and sixth assessment dates.
- 2.12 I will not participate in any events or races the 72 hours after the second and sixth testing days, as well as the seven days prior to the evaluation sessions. I will tell the assessor if I have any muscle fatigue or soreness prior to the tests.
3. The first and second visit will be separated by at least seven days, but not more than 10 days and the fifth and sixth assessment will be separated by at least 7 days, but no more than 28 days.
4. I do not have any problem wearing a heart rate monitor or compression socks during the study.
5. I understand that the researcher/test observers and/or Stellenbosch University may not be held responsible for any injuries that might occur during any of the tests included in the project.

6. I was warned that there is a possibility that I may experience one or more symptoms during the aerobic capacity tests. These symptoms include light-headedness, dizziness, fainting, chest, jaw, neck or back pain or pressure, severe shortness of breath, wheezing, coughing or difficulty breathing, nausea, cramps or severe pain or muscle ache and severe prolonged fatigue or exhaustion, since this is a maximal test to exhaustion. I understand that I may stop the tests at any time when I experience any of these symptoms.
7. I was informed that the information, which will be obtained through this project, would be handled confidentially, but that the results will be published in research journals.
8. The above information was explained to me in English by Karen Bindemann. I was given the opportunity to ask questions and all the questions were satisfactorily answered.
9. It was explained to me that my participation in this project is voluntary and that I may withdraw from the study at any time. I also understand that the researcher or medical doctor may withdraw me from the study if deemed necessary for medical purposes.
10. I was informed that there are no costs involved for my participation in this project.

**I voluntarily agree** to participate in the abovementioned project.

**I take the responsibility** to endeavor to complete all (three visits to the laboratory for fitness tests and the 2 times 3 consecutive days for sample collection) tests.

**I take the responsibility** to be and stay **highly motivated** during the **testing programme** and to complete my **daily logbook** (the seven days prior to the second and sixth assessments) and hand it in to the researcher on at the next assessment.

Signed at \_\_\_\_\_ on \_\_\_\_\_.

\_\_\_\_\_

Subject

\_\_\_\_\_

Witness



**STATEMENT BY RESEARCHER**

I, ***Karen Estelle Bindemann***, declare that I:

1. Explained the information in this document to \_\_\_\_\_;
2. Requested him to ask questions if anything was unclear;
3. That this conversation took place in English/Afrikaans.

Signed at ***Stellenbosch*** on \_\_\_\_\_

\_\_\_\_\_

Researcher

\_\_\_\_\_

Witness

**Research will be conducted according to the Declaration of Helsinki, MRC guidelines and GCP.**

## APPENDIX B

### ILLUSTRATIONS OF THE TWO-HOUR TREADMILL RUN



Illustrations of the two-hour treadmill run to induce DOMS, with compression garments.