SUB-MAXIMAL BLOOD LACTATE ASSESSMENT OF PROFESSIONAL YOUTH SOCCER PLAYERS THROUGHOUT THE SOCCER SEASON

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ABSTRACT

Aerobic fitness is recognised as a very important fitness component in soccer (Reilly, 1994, Science and Soccer. E&FN Spon, UK). Thus, it is of importance to monitor the aerobic fitness of professional soccer players periodically throughout the soccer season. The aim of this study was to examine the changes in aerobic fitness of professional youth soccer players from the pre-season period to the start of the competitive season and throughout the competitive season.

Thirty-seven male professional youth soccer players aged (mean $\pm$ sd) 18.9 $\pm$ 1.8 years participated in this study. The players were tested at six time-points throughout the playing season – sub-maximal running was performed on a treadmill (Woodway Ergo ES2, Cranlea, UK) and consisted of at least six progressive four-minute stages. Fingertip blood samples were collected at the end of each running speed and analysed for whole blood lactate concentration using an Analox GM7 analyser (Analox Instruments, UK). Although 37 soccer players participated in this study, not all players were tested at each time point (Table A). In order to account for the within-subjects design and the fact that there are missing data at certain testing time-points (which is assumed to be missing at random), a Repeated Measures Analysis of Variance with Bonferroni Multiple comparisons, and Bonferroni adjusted paired t-tests were used to determine any significant changes in aerobic fitness, using running speeds at the lactate threshold [vLT] and 4 mmol.l^{-1} [vLac4].

Aerobic fitness increased from the start of pre-season training to October (5 weeks into the competitive season) as evidenced by an increase in mean vLT and mean vLac4 ($p<0.001$) (Table 1). The mean vLT was highest in December ($p<0.001$) compared with January and June.

Other fluctuations in the mean vLT throughout the competitive playing season were found to be non-significant. No significant differences in mean vLac4 were found during the competitive season.

In conclusion, aerobic fitness increased from the start of pre-season training to the early weeks of the competitive playing season. The mean vLT was found to be highest in December. These findings demonstrate that the pre-season training was effective in improving aerobic fitness. The fact that the January, April and June vLT scores were lower than those in December suggests that coaches should examine the aerobic training regimens in the second half of the season.

Table A. Lactate threshold (vLT) and 4 mmol.l^{-1} (vLac4) running velocity (mean ± sd) and number of observations for each testing time-point

Testing Date	vLT (km.h^{-1})	vLac4 (km.h^{-1})	vLT Players tested (n)	vLac4 Players tested (n)
Pre-Season	11.39 ± 1.05	13.02 ± 0.70	16	14
October	12.62 ± 0.73[a]	14.17 ± 0.82[a]	37	34
December	12.69 ± 0.82[b]	14.14 ± 0.82	30	29
January	12.35 ± 0.94	13.97 ± 1.11	20	17
April	12.37 ± 0.84	14.27 ± 1.02	17	18
June	12.39 ± 0.84	14.37 ±0.80	21	18

a – significantly higher than pre-season ($p<0.001$)

b – significantly higher than January and June vLT ($p<0.001$)

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ANOVA	analysis of variance
Apr	April
AT	anaerobic threshold
ATP	adenosine tri-phosphate
BASES	British Association of Sport and Exercise Sciences
bpm	beats per minute
CHO	carbohydrate
CO_2	carbon dioxide
CP	creatine phosphate
Dec	December
FBLC	fixed blood lactate concentration
FFA's	free fatty acids
FIO_2	fraction of oxygen in the inspired air
FT	fast twitch muscle fibres
h	hour(s)

hand-not.	hand notation
HR	heart-rate
Jan	January
kg	kilogram(s)
km	kilometre(s)
$[Lac]^b$	blood lactate concentration
l	litre(s)
LT	lactate threshold
m	metre(s)
max	maximum
ml	milli-litre(s)
min	minute(s)
MLSS	maximum lactate steady state
$mmol.l^{-1}$	milli-moles per litre
n	number
O_2	oxygen
OBLA	onset of blood lactate accumulation

Oct	October
PFK	phosphofructokinase
RER	respiratory exchange ratio
s	second(s)
sd	standard deviation
ST	slow twitch muscle fibres
tape-rec.	tape recording
VE	ventilation
vel.	velocity
vLT	running velocity ($km.h^{-1}$) at the lactate threshold
vLac4	running velocity ($km.h^{-1}$) which elicits a blood lactate concentration of 4 $mmol.l^{-1}$
$\dot{V}O_2$	oxygen uptake
$\dot{V}O_2 max$	maximum oxygen uptake (maximum aerobic power)

A BRIEF HISTORY OF SOCCER

Soccer (or association football) seems to have its origins in Medieval Britain. A record states that London schoolboys began to play a form of football in 1175 after dinner on Shrove Tuesday, while legend says that the first game was played amongst Anglo-Saxons, using the severed head of a defeated Dane soldier as a "ball". These embryonic forms of soccer were violent affairs, had little or no rules, except for rules actually forbidding the game. Edward III, Richard II and Henry IV banned the game because they were concerned that injuries sustained by soldiers while playing would prevent them from maintaining a strong army for battle against the French (1337-1453), and because the popularity of the game was interfering with archery practice.

Despite attempted repression, the game was still extremely popular in post-modern British life. Indeed, in 1655 Samuel Pepys described the London Streets as "full of footballs".

By the 18th Century, the game was a large part of public school tradition with the rules varying dramatically from school to school. However during the early years of the 18th Century efforts were made to make rules that were "fair to all the schools". In 1840, a set of rules was drawn up at Cambridge and by 1848 various public schools were playing the game using these "Cambridge rules". No holding, tripping or pushing was permitted, and kicking the ball "through the posts and under the string" scored a goal. However, handling the ball was still allowed, but only to stop the ball or to catch it and then kick.

A code of ten rules under the title of "The Simplest Game" was established in 1862, giving the game a structure that has shaped the game as we know it today. On the basis of these rules the Football Association (FA) was established in 1863. A challenge cup was set up in 1873, with the game now becoming distinctly different from the game of rugby (no handling was permitted, except for the "goal-tender"). It is said that soccer derived its name from an Oxford University student whom when asked if he would like to play a game of "rugger" replied, "No, I'm playing soccer".

Professionalism in soccer was legalised by the FA in 1885, with a professional league consisting of 12 clubs starting in 1888.

By the start of the 1900's, soccer had become organised on an international level. Several European countries merged in 1904 to form FIFA (the Federation of the International Football Association) and in 1930, the first FIFA World Cup was held in Uruguay. Since then, several international competitions for both national and club sides have been established.

Today, soccer is the world's major sport, with approximately 60 million registered players around the world. The "peoples game" has now become a worldwide multi-million pound industry. It is estimated that the 1998 FIFA World Cup attracted some 40 billion television viewers (Shephard, 1999). Elite professional soccer players these days now have earnings of thousands of pounds per week, with the worlds top soccer clubs willing to pay millions of pounds to secure their services. All this is a far cry from the early days of soccer as described by Carew (1602) –

"...Whoever gains possession of the ball generally finds himself pursued by the other side and they will not leave him alone until he is laid flat on God's dear earth...the players play over hills, dales, hedges; yes, and through bushes, briars, bogs, pools and rivers so that you will sometimes see 20 or 30 lie tugging in the water, scrambling and scratching for the ball.... the ball in this game may be compared to an infernal spirit. Whoever catches it behaves immediately like a madman, struggling and fighting with those who try to hold him...I cannot decide whether I should commend this game for its manliness and exercise, or condemn it for its boisterousness and the harm it causes...when the playing is ended you will see them returning home as if from a pitched battle, with bloody heads, bones broken and out of joint, and such bruises as will shorten their lives. Yet it is a good game...."

CHAPTER 1

THE PHYSIOLOGICAL DEMANDS OF SOCCER

(with special reference to aerobic fitness)

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INTRODUCTION

The physiological demands of soccer are more complex than in many individual sports. In soccer, the players perform many different multi-directional movements – the exercise intensity can alternate at any time, ranging from standing still to maximal sprinting. Therefore, soccer is classified as a high-intensity, intermittent exercise (Bangsbo, 1994). The intermittent nature of the game makes it distinct from sports in which continuous exercise is performed, such as marathon runs or an 800m race.

Individual soccer performance is determined by a player's technical, tactical, physiological and psychological/social characteristics (Bangsbo, 1994). These elements are closely linked to each other. For example, a player with poor technical skills may often lose possession of the ball, and must sprint/tackle in order to regain possession.

There is a large variability in the physiological demands imposed on soccer players during a game. The activity of an individual during a match is influenced by several factors such as physical fitness, tactics, positional role of the player, importance of the game, environmental conditions and quality of the opposition.

The aim of this study was to monitor the aerobic fitness levels of male professional youth soccer players belonging to a Scottish Premier League Club throughout a soccer season. The author is a sports scientist for a professional soccer club. The development and maintenance of aerobic fitness of soccer players are considered to be of great importance as several soccer-related studies have emphasised that a high aerobic fitness level is of benefit to the professional soccer player. This chapter highlights the importance of aerobic fitness in soccer players by providing evidence from scientific studies based on the:

A) motion characteristics of soccer,
B) physiological demands of soccer, and
C) physiological profiles of elite soccer players
(with special attention given to aerobic fitness).

(A) MOTION CHARACTERISTICS OF SOCCER

In order to gain a correct impression of the physiological load imposed on soccer players during competitive soccer, observations have to be made during competitive match-play. Motion analysis of soccer players can provide data about the physiological demands of soccer in general. Activities may be classified according to mode, intensity, duration and frequency. In this way an overall picture of the physiological demands of soccer can be gathered as a whole. A different method is to set the activity profile alongside a time-base so that the average work-rest ratio can be determined (Reilly, 1994). Work-rate profiles can also supply important information about the physiological demands of soccer.

DISTANCE COVERED DURING A SOCCER GAME

According to Reilly and Thomas (1976) the total distance covered provides information about the physiological load associated with soccer match-play. Several studies have determined the individual distance covered during a game, which can be used as an indicator of the total work performed (the energy cost of running is related to mechanical work output and is largely independent of the running speed).

Walter Winterbottom, manager of the English national team and the FA Director of coaching (1946-62) was one of the first to analyse the game of soccer. He estimated that players covered a distance of 3361m (cited in Bangsbo, 1994) by tracking their movements on a scale plan of the pitch. Wade (1962) noted a total distance of 1600-5486m for professional players. Since these early observations, several methods have been used to determine distance covered during a soccer game, including the use of hand-notation systems (Knowles and Brookes, 1974), coded commentary (Reilly and Thomas, 1976), video-filming and potentiometer readings (Van Gool et al, 1988), trigonometry (Ohashi et al, 1988), and computer-aided analysis (Moyls, 1988; Ali and Farrally, 1991a).

Some discrepancies exist in the published data for the distance covered by soccer players during match-play. At both extremes, Vinnai (1973) cited 17 km for Russian soccer

players (no reference to methodology), while Knowles and Brookes (1974) reported an average of 4886m for Manchester City players. Distance covered in soccer games from various sources is detailed in Table 1.

The studies on elite Australian (Withers et al, 1982), Belgian (Van Gool, 1988), Canadian (Mayhew and Wenger, 1985), Danish (Bangsbo et al, 1991, Bangsbo and Lindquist, 1992), English League (Reilly and Thomas, 1976), Japanese (Ohashi et al, 1988) and Swedish (Saltin, 1973) all used reliable, objective and valid methodologies, and on the basis of these studies, is it generally accepted **that on average, soccer players cover a distance of 9-12 km during match-play.**

TIME BASED ANALYSES

An alternative strategy to computing distance covered is to operate on a time base to which activity patterns can be related to (Reilly, 1994). From their study on Canadian soccer players, Mayhew and Wenger (1985) found that **88% of match-play time was spent in primarily aerobic activities**, with 12% of match-play time spent utilising anaerobic energy systems.

WORK RATE PROFILE OF SOCCER PLAY

Although there is a large component of unpredictability inherent in individual and team behaviour during match-play, a break-down of the work-rate activities into specific categories can provide information on the more specific demands of the game which are hidden when only the total distance covered is analysed.

Reilly and Thomas (1976) reported that First Division English soccer players exhibited around 1000 – 1200 changes in playing activities during match-play with each activity

TABLE 1

Distance covered (km) during a soccer game according to different researchers (adapted from Reilly, 1994a and Bangsbo, 1994)

Source	Players	Distance covered (km)	Method
Winterbottom (1952)	Prof. Players (England)	3.3	Hand Not.
Wade (1962)	Prof.Players (England)	1.6-5.5	Undisclosed
Saltin (1973)	Non-Elite	11.5	Cine-Film
Knowles and Brookes (1974)	Prof.Players (England) (n = 40)	4.8	Hand Not.
Reilly and Thomas (1976)	Prof.Players (England) (n = 40)	8.7	Tape-Rec.
Withers et al (1982)	First Team Players (Australia) (n = 20)	11.5	Videotape
Ekblom (1986)	1^{st}-4^{th} Division (Sweden) (n = 44)	10	Hand-Not.
Van Gool et al (1988)	University players (Belgium) (n = 7)	10.3	Cine-Film
Ohashi et al (1988)	Elite players	9.8	Trigonometry

lasting for a mean duration of 5 - 6s. These discrete bouts of action incorporate frequent changes of pace and direction as well as involvement with the ball. These English players had short rest periods of 3s on average every 2 mins, with sprints averaging about 15m in distance, and occurring approximately every 90s. Players were found to "cruise" or sprint once every 30s. Although these data were derived 25 years ago, Reilly (1994) states that observations made on World Cup players performing in the English League in 1990 indicate that these profiles from the 1970's are still representative of top-level club soccer.

Reilly and Thomas's (1976) work-rate study on elite English soccer players is considered to be the most appropriate way of monitoring one player per game (Reilly, 1990; Reilly 1996), and indicates that, on average, the overall distance covered by outfield players during match-play consists of –

25% walking
37% jogging
20% cruising sub-maximally
11% sprinting
7% moving backwards,

with sideways and diagonal running contained in these categories. Less than 2% of the total distance covered is with the ball. The ratio of low-intensity to high-intensity exercise was found to be about 2.2 to 1 in terms of distance covered. **On a time-base, this ratio is about 7 to 1 which denotes a predominant sub-maximal stress on the aerobic energy system.**

Work-rate is determined to a large extent by the positional role of the player. Midfield players tend to cover the greatest distances during match-play since they have to act as links between defence and attack (Reilly and Thomas, 1976; Ekblom, 1986; Bansgbo et al, 1991). **The greater overall distance covered by the Danish midfield players in a study by Ekblom (1986) was due to more running at low speeds, denoting a sub-maximal aerobic type of activity profile for the midfield player.** Studies of English league players show that strikers and midfield players cover more distance sprinting than

defenders. Fullbacks show the greatest variability in distance covered, while centre-backs generally cover the greatest distances moving backwards (Reilly, 1994). Although work-rate profiles are relatively consistent from match to match, Bangsbo (1994) states that it is the high-intensity component that is the most constant feature.

Studies on Danish (Bangsbo, 1991) and Portuguese league players (Rebelo and Soares, 1992) have confirmed earlier observations that **soccer players cover 5-9% less distance in the second half compared to that of the first**. Tumilty (1993) and Reilly (1994a) suggest that the aerobically fit player may be spared this decrement in work-rate.

Goalkeepers cover a distance of around 4 km during a match. Their work-rate profile emphasises short duration anaerobic efforts when they are called into play. Therefore, a high aerobic fitness level is not regarded of being of prime importance to the goalkeeper (this thesis is devoted to the aerobic fitness / physiological demands of **outfield** soccer players).

(B) PHYSIOLOGICAL DEMANDS OF SOCCER

In addition to analysing the motion characteristics of soccer, more specific physiological parameters such as measurements of oxygen uptake and heart-rate can be collected to provide an indication of the physiological load imposed on soccer players during competitive match-play.

Soccer players perform many different activities during match-play, with the intensity alternating at any time. The intermittent nature of the game demands that players should have a well-developed ability to exercise with a high power output (anaerobic), whilst also being able to work for a long duration in a widely fluctuating tempo (intermittent aerobic endurance).

AEROBIC ENERGY PRODUCTION DURING SOCCER MATCH-PLAY

Some researchers have attempted to measure the aerobic contribution during soccer match-play by directly measuring oxygen uptake ($\dot{V}O_2$), using Douglas bags (Covell et al, 1965; Durnin and Passmore, 1967; Ogushi et al, 1991; Yamaoka, 1965). From these studies, oxygen uptake values of 1-2 $l.min^{-1}$ have been obtained, but these values should be treated with caution since the collecting procedures probably interfered with normal play. More recently, Kawakami and colleagues (1992) used a light-weight portable telemetry system (K2) to measure $\dot{V}O_2$ during various soccer drills. The $\dot{V}O_2$ values were between 2 and 4 $l.min^{-1}$ for small-sided games (1 v 1 and 3 v 3), with the highest $\dot{V}O_2$ recorded from dribbling drills (4 $l.min^{-1}$).

A less movement-restricting way of gathering information about the aerobic energy expenditure during soccer is by measuring heart-rate (HR) continuously during a match. The recent developments in HR monitoring equipment, along with the decreased costs over the last two decades have led to their adoption by exercise physiologists for testing athletes in field and laboratory conditions without restricting subject movement.

HR and oxygen uptake ($\dot{V}O_2$) are linearly related throughout a large proportion of the HR range (Astrand and Rodahl, 1986). Oxygen uptake can be estimated indirectly from the HR-$\dot{V}O_2$ relation determined in the sports laboratory. However, HR does not always reflect the actual $\dot{V}O_2$. HR may be elevated beyond the normal HR-$\dot{V}O_2$ relationship because of static contractions, emotional, and thermal stress (Astrand and Rodahl, 1986). Overestimation of $\dot{V}O_2$ due to these factors is probably minor in soccer, because of the high exercise intensity and the dominant use of large muscle groups (Bangsbo, 1994). A consideration when using the HR-$\dot{V}O_2$ relationship is the validity of using the relationship when it is determined from **continuous** sub-maximal exercise (usually running on a treadmill) and applying this relationship to estimate $\dot{V}O_2$ for soccer's **intermittent** exercise pattern. Research by Bansgbo (1994) suggests that the relationship obtained from sub-maximal treadmill running is valid for intermittent exercise

(alternating low and high speeds on a treadmill for 10 and 15s respectively) and probably for soccer as well.

When the HR determinations during match-play are related to $\dot{V}O_2$ using the HR-$\dot{V}O_2$ relationship described, mean values of 70 – 80% of $\dot{V}O_2$max have been estimated for soccer match-play (Reilly and Thomas 1979; Ekblom 1986; Bangsbo 1994).

Seliger (1968b) reported the mean HR during a competitive match for Czech players to be 165 beats per minute (bpm), or 80% of maximal HR, while Reilly (1986) found the average HR to be 157 bpm for English League players participating in friendly games. Van Gool (1988) recorded 169 and 165 bpm (for the first half and second half respectively) for University standard Belgian players. It is worth noting that positional role of the players monitored can influence the HR response. Van Gool (1987; cited by Bangsbo, 1994) observed that the mean HR for defenders was 155 bpm, while midfield and forward players exhibited higher average HR's (170 bpm). In competitive matches, Rhode and Espersen (1988) found that the HR for Danish players participating in 1st Division games was below 73% of HR-max for 11% of the total playing time, between 73% and 92% of HR-max for 63% of the total playing time, and greater than 92% HR-max for 26% of the total playing time. In another Danish study, Bangsbo (1992b), when monitoring six professional players, found that the mean HR was 164 bpm during the first half, with a subsequent reduction of about 10 bpm during the second half. In a more recent study Strudwick and Reilly (1999) monitored seventeen professional youth players of an English Premier League team using short-range radio telemetry during competitive match-play and training. The mean HR from competition was found to be 175 bpm. Distribution of the mean percentage of HR-max is shown in Table 2. It is of interest that the results of this study show how significantly more intense soccer match-play was compared to that of training.

ANAEROBIC ENERGY PRODUCTION

It is difficult to quantify the contribution made from the anaerobic energy sources during soccer match-play because the exercise intensity varies frequently, but the energy production from this system appears to account for only a minor part of the total energy

TABLE 2

Distribution of the mean % HR-max during soccer training and competitive match-play (Strudwick and Reilly, 1999)

% HR-max	Match % of time	Training % of time
>90%	**30.8 +/- 22.9**	**3.3 +/- 4.8**
>80-90%	**49.3 +/- 14.5**	**14.9 +/- 4.6**
>70-80%	16.7 +/- 9.0	21.0 +/- 4.7
>60-70%	3.2 +/- 4.2	23.4 +/- 3.8
>50-60%	0	23.5 +/- 5.9
>50%	0	14.3 +/- 10.8

cost of match-play. For elite Danish players, Bansgbo et al (1991) reported the total duration of high-intensity exercise during soccer match-play to be approximately 7 mins. This included about 19 sprints with an average duration of 2-3s. The sprints a soccer player makes during match-play are mostly 10-25m in length, or 3-5 s in duration (Apor, 1988), therefore it is assumed that the ATP-CP system is the anaerobic energy system of most importance for soccer. However, it has been demonstrated that the breakdown of intra-muscular phosphagen stores [creatine phosphate (CP) and ATP] **and** anaerobic glycolysis supplies approximately equal amounts of energy production during 6s of all-out exercise (Boobis, 1987), supporting the claim of Jacobs et al (1983) that anaerobic glycolysis begins almost immediately after the commencement of high intensity exercise.

To determine whether the energy supplied from anaerobic glycolysis is of significance during soccer match-play, several researchers have collected blood lactate samples during matches. Results from these studies have varied from values as low as 2 $mmol.l^{-1}$ (Tumilty et al, 1988) to 12 $mmol.l^{-1}$ (Ekblom, 1986). Most studies cite values in the 4-8 $mmol.l^{-1}$ range, which suggests that anaerobic glycolysis has a role (Bansgbo et al, 1991; Bangsbo, 1994; Gerisch et al, 1988; Rhode and Espersen, 1988; Smaros, 1980; Smith et al, 1993). Contrasts in the results are probably due to the varying standards of soccer in the different studies (college – elite level), the importance of the match (training games – competitive match-play), the time-point of sampling (i.e. during match-play, halftime/fulltime), and the blood media sampled (venous, capillary, whole blood, plasma lactate). Gerisch et al (1988) found that defensive tactics employed also affects the lactate response. Higher mean blood lactate levels were observed when teams used "man-to-man marking" compared to "zonal-coverage". The main reason for the contrast in results between studies is likely to be due to the actual time of sampling. It has been demonstrated **that blood lactate measurements are related to the incidence of high-intensity activities for the 5-minute duration immediately before sampling occurs** (Bangsbo et al, 1991). Bansgbo (1994) when sampling plasma lactate at various times during a competitive match found that there was a significantly lower blood lactate concentration in the second half than when compared to the first. This is similar to the observations of other studies (Bansgbo et al, 1991;Ekblom, 1986; Gerisch et al, 1988; Rhode and Espersen, 1988; Smaros, 1980). Ekblom (1986) reported that **the higher the level of play, the higher the lactate levels found**. Division 1 players were found to show blood lactate levels of 8-10 $mmol.l^{-1}$ decreasing to around 4 $mmol.l^{-1}$ for Division 4 players (see Table 3). These results led Ekblom to conclude that **as the playing standard increases, so may the contribution of anaerobic glycolysis**. Ekblom (1986) noted, "It seems that the main difference between players of different quality is not the distance covered during the game but the percentage of overall fast-speed distance during the game and the absolute values of maximal speed play during the game".

Despite its small contribution to the total energy turnover, anaerobic energy production is of vital importance as it supplies the soccer player with a high rate of energy provision during sustained intense periods of match-play. Intense anaerobic exercise is performed during the most decisive, interesting, and potential match-winning situations. Apor

(1988) suggests that soccer players do not need an exceptional anaerobic capacity, but should have a high anaerobic power, while Tumilty (1988) concluded that the contribution of anaerobic glycolysis remains unclear, but is probably of significant importance to elite soccer players.

TABLE 3

Blood Lactate concentrations for 1st – 4th Division Swedish players [halftime/fulltime] (from Ekblom, 1986)

Division	Half-time	Full-time
1st Division	**9.5** (6.9 – 14.3)	**7.2** (4.5 – 10.8)
2nd Division	**8.0** (5.1 – 11.5)	**6.6** (3.1 – 11.0)
3rd Division	**5.5** (3.0 – 12.6)	**4.2** (3.2 – 8.0)
4th Division	**4.0** (1.9 – 6.3)	**3.9** (1.0 – 8.5)

(C) PHYSIOLOGICAL PROFILES OF ELITE SOCCER PLAYERS

As well as examining motion characteristics and direct physiological measurements during soccer match-play, another way of obtaining information about the physiological demands of soccer is by determining the physical capacity of **elite** soccer players.

AEROBIC FITNESS PROFILES

Since 90% of energy provision during soccer match-play is estimated to come from aerobic sources (Bangsbo, 1994), it would be reasonable to expect high levels of aerobic fitness to have been reported in physiological profile studies of elite soccer players.

MAXIMAL AEROBIC POWER ($\dot{V}O_2$max) OF ELITE SOCCER PLAYERS

The average values of $\dot{V}O_2$max for elite soccer players tend to be high, supporting the belief that there is a large contribution from aerobic power when playing elite-level soccer. Mean $\dot{V}O_2$max of elite soccer players is normally reported between 55 and 65 $ml.kg^{-1}.min^{-1}$ (Astrand and Rodahl, 1986; Davis et al, 1992; Nowacki et al, 1988; Rhodes et al, 1986; Thomas and Reilly, 1979; White et al, 1988; Williams et al, 1973, Withers et al, 1977), the higher values being found when players are at peak fitness. **Some individual values higher than 70 $ml.kg^{-1}.min^{-1}$ have been recorded** (Wisloff et al, 1998). These high, but unremarkable values are similar to those found in other team sports, are higher than values reported for amateur soccer players (Ekblom, 1986), but are substantially lower than elite endurance performers where values close to 90 $ml.kg^{-1}.min^{-1}$ have been found (Wisloff et al, 1998). Nowacki et al (1988) when reviewing 26 studies of $\dot{V}O_2$max assessment on German soccer players, reported that over half of the studies were conducted using a cycle ergometer, which would underestimate the $\dot{V}O_2$max of the players. Nowacki and colleagues found the highest average $\dot{V}O_2$max value reported from treadmill running to be 69.2 $ml.kg^{-1}.min^{-1}$. Treadmill testing of 17 members of the 1978 German National Squad revealed a mean $\dot{V}O_2$max value of 62 $ml.kg^{-1}.min^{-1}$.

A couple of studies have indicated that a positive correlation may exist between aerobic power and team success. The importance of aerobic power has been reflected by rank-correlation (Apor, 1988) of the most successful teams in the Hungarian 1st Division Championship (see Table 4)

Wisloff et al (1998) supported this aerobic power-success relationship by demonstrating a clear difference in $\dot{V}O_2$max between the top team (Rosenborg, mean $\dot{V}O_2$max - 67.6 ml.kg^{-1}.min^{-1}), and a lower placed team (Strindheim, mean $\dot{V}O_2$max - 59.9 ml.kg^{-1}.min^{-1}) in the Norwegian elite division. Theoretically, this mean aerobic power difference would mean that Rosenborg would have one more player on the field with a $\dot{V}O_2$max of 77 ml.kg^{-1}.min^{-1} when compared to Strindheim.

TABLE 4

The relationship between mean $\dot{V}O_2$max and finishing position in the elite Hungarian league (from Apor, 1988)

	$\dot{V}O_2$max (ml.kg^{-1}.min^{-1})	League Position
Ujpesta Dozsa	66.6	1st
FTC	64.3	2nd
Vasas SC	63.3	3rd
Honved SE	58.1	5th

The observation of a high correlation between aerobic power ($\dot{V}O_2$max) and distance covered per game supports the adoption of training regimes that raise the aerobic fitness of soccer players to high levels (Reilly and Thomas, 1976; Smaros, 1980). Smaros (1980) found that, in addition to the strong correlation with the total distance covered in the game (r=0.89), $\dot{V}O_2$max also influenced the number of sprints attempted during a match. This is not suprising as players with a high $\dot{V}O_2$max have a faster recovery from intense exercise and have greater stores of muscle glycogen (Astrand and Rodahl, 1986; Bangsbo and Mizuno, 1988; Ekblom, 1986). Soccer players with high endurance capacities would be expected to spare glycogen during moderate intensity exercise due to an increased utilisation of free fatty acids (FFA's). This glycogen-sparing effect would help to reduce a possible decrement in work-rate during the second half, as the

fatigue that soccer players experience in the second-half of match-play may be due to glycogen depletion (Jacobs et al, 1982). Balsom (1994) has reported that the recovery between repeated short duration bouts of high-intensity exercise is dependent on oxidative processes and may be enhanced if aerobic fitness is increased.

THE "ANAEROBIC THRESHOLD" OF ELITE SOCCER PLAYERS

Although maximum aerobic power indicates the maximal ability of the exercising musculature to consume oxygen, it is not possible to sustain exercise for a long period of time at $\dot{V}O_2$max. The upper level at which exercise can be sustained for a prolonged period is thought to be indicated by the "anaerobic threshold" (Reilly, 1994b). Many studies have indicated that the "anaerobic threshold" is a better predictor of endurance performance than $\dot{V}O_2$max (Weltman, 1995). Most researchers express the "anaerobic threshold" as the work-rate corresponding to a blood lactate concentration of 4 mmol.l^{-1} determined from invasive incremental tests, or determined non-invasively by associated changes in respiratory gas exchange (Wasserman et al, 1973). The mean anaerobic threshold has been measured non-invasively at 77% of $\dot{V}O_2$max in English League 1st Division players (White et al, 1988), a value close to a work intensity associated with marathon running. In tests on elite Finnish soccer players, Rahkila and Luhtanen (1991) found that the anaerobic threshold (determined as the inflection point in the blood lactate response from incremental exercise), was 83.9% of $\dot{V}O_2$max on average for the 31 players tested. Using a fixed blood lactate concentration (FBLC) of 3 mmol.l^{-1}, Bangsbo and Lindquist (1992) reported that this FBLC corresponded to about 80% of $\dot{V}O_2$max for both continuous and interval testing on a treadmill.

The intermittent nature of soccer requires that players frequently perform match activities at an intensity above the "anaerobic threshold" intensity, although the average fractional utilisation of $\dot{V}O_2$max is deemed to be 70-80% of $\dot{V}O_2$max (Reilly, 1996).

MUSCLE FIBRE CHARACTERISTICS OF ELITE SOCCER PLAYERS

Investigations on the metabolic characteristics of elite soccer players' muscles can give an indication of the importance of the different energy systems. As the activity profile of soccer match-play is of a high-intensity intermittent nature, it would be expected that a balanced contribution of slow-twitch (ST) and fast-twitch (FT) fibres would be found in elite soccer players. The muscle fibre characteristics in the vastus lateralis of elite Swedish players was found to be about 60% FT, suggesting that the fibre types of elite soccer players are closer to that of a sprinter than an endurance athlete (Bansgbo, 1994). However, a large range of FT fibre percentage was found in the squad (40.8 - 79.1%). On the other hand, a smaller number of FTb fibres were found for the elite players compared to that of the non-elite players. Andersen et al (1993) [cited by Bangsbo, (1994)] revealed that in elite soccer players, a large component of the FTb fibres also had FTa myosin heavy chain expression. **Bansgbo concludes that the muscles of the elite soccer player can be characterised as having few FTb fibres, an observation found also in endurance athletes.** Smaros (1980) reported an average muscle fibre type distribution of 53% ST and 47% FT in the vastus lateralis muscles of Finnish soccer players. Importantly, the same study by Smaros details that analysis of muscle biopsies taken at the end of matches showed that **reduction in muscular glycogen stores occurred mainly in the ST fibres**, reflecting the aerobic demand for this muscle in particular.

When sampling for oxidative enzymes of the gastrocnemius muscle, Bansgbo and Mizuno (1988) found that the occurrence of ST, FTa and FTb fibres in four elite Danish soccer players was 55.9% (range 48-63.6), 39.8 (33-46.5) and 4.4 (3.0-5.5) respectively. The concentrations of mitochondrial enzyme 3-hydroacyl coenzyme A (HAD) found was similar to that of cross-country skiers, and values for citrate synthase were found to be between that reported in the literature for middle-distance runners and non-athletes.

Muscle fibre capilliarity of elite soccer players has been found to be higher than that of untrained individuals, but not as high as that found in elite endurance athletes (Bansgbo, 1994)

It is difficult to draw conclusions on the importance of the aerobic/anaerobic energy systems from the findings on the skeletal muscle characteristics of soccer players. Different muscles studied, the sample number, positional role of the players studied and training status of the players when sampled must be taken into consideration, but it seems that in top level soccer, aerobic and anaerobic fitness components are of importance.

SUMMARY

Due to its acyclical nature and intensity, soccer is classified as a high-intensity intermittent team sport (Bangsbo, 1994). During competitive soccer match-play, elite players cover a distance of about 10 km (Bansgbo et al, 1991, Balsom, 1991) at an average intensity close to the "anaerobic threshold", being 80-90% of HR-max, or 70-80% of $V0_2max$ (Reilly, 1994a; Van Gool, 1988)". **It is estimated that aerobic metabolism provides 90% of the energy cost during soccer match-play** (Bangsbo, 1994). In order to be successful, professional soccer players require a high aerobic fitness level as well as the game skills to create match-winning situations, and to sustain runs for defending or attacking purposes.

Although the majority of the time spent exercising during soccer match-play is of a sub-maximal intensity, match-play has some periods of high intensity that result in the accumulation of blood lactate. It is important that the soccer player has the appropriate anaerobic fitness to participate to the best of his ability in these intense periods of match-play and also has the aerobic fitness for quick recovery and elimination of muscle/blood lactate. Bangsbo (1994) states that a player's physical capacity can be divided into the following categories: a) the ability to perform prolonged intermittent exercise (endurance); b) the ability to exercise at high intensity; c) the ability to sprint; and d) the ability to develop a high power output in single match situations.

From the discussion of the several studies applied to soccer science in this chapter, most of the studies indicate the importance of aerobic fitness in soccer. It is reasonable to expect that the obtainment of a high aerobic fitness level and subsequent maintenance of a high aerobic fitness level throughout the competitive season is beneficial to the

professional soccer player. Indeed, Apor (1988) suggests that soccer players should aim to train their aerobic power to above 65 $ml.kg^{-1}.min^{-1}$. This suggestion is supported by Wisloff and colleagues (1998), who advocate that soccer trainers should aim to elevate the aerobic fitness levels of their players, to values around 70 $ml.kg^{-1}.min^{-1}$.

It seems beneficial for the professional soccer player to increase their aerobic fitness levels to as high a level as possible and maintain a high aerobic fitness level throughout the competitive season. However, the importance of the anaerobic energy system, especially in top-level soccer players should not be underestimated. It should also be remembered that top-level soccer players require to specifically train for other important aspects of soccer, e.g. technical skills, tactical awareness, strength, speed and power throughout the soccer season.

Although the importance of anaerobic fitness parameters in soccer is recognized, the main aim of this study was to monitor the **aerobic fitness levels** of professional youth soccer players belonging to a Scottish Premier League Club throughout a full soccer season. Aerobic fitness levels in this study were obtained by measurement of the "anaerobic threshold" by sub-maximal blood lactate assessment - running velocities at the lactate threshold (vLT) and 4mM marker (vLac4) were collected at strategic times throughout the soccer season.

CHAPTER 2

THE BLOOD LACTATE RESPONSE TO EXERCISE

CHAPTER 2 - THE BLOOD LACTATE RESPONSE TO EXERCISE

INTRODUCTION

Traditionally, maximal oxygen uptake ($\dot{V}O_2$max) has been viewed as the "gold standard" measurement of aerobic fitness. However, $\dot{V}O_2$max assessment has a number of limitations (Weltman, 1995). It has been suggested that the blood lactate response to incremental sub-maximal exercise may be a better marker of endurance performance and a more sensitive indicator of changes in training status than $\dot{V}O_2$max.

The aim of this chapter is to introduce the theory and mechanisms of the blood lactate response to the reader, and by presenting results and conclusions from a select few of the multitude of research studies conducted in this area of exercise science, demonstrate to the reader why sub-maximal blood lactate assessment may a useful tool for the assessment of aerobic fitness in professional soccer players.

(A) HISTORICAL REVIEW

The presence of lactic acid in skeletal muscle was first discovered by Berzelius in 1812 (cited by Jordfelt, 1970). Fletcher and Hopkins (1907) (cited by Jordfelt, 1970) were the first to provide evidence that a relationship existed between the production of lactic acid and muscular activity. In 1909 Douglas and Haldane (cited by Jordfelt, 1970) suggested that lactate stimulated respiration during high-intensity exercise. Hill (1911) building on the work of Fletcher and Hopkins, who had shown that exercise could take place without the availability of oxygen, first identified the phases of aerobic and anaerobic skeletal muscle metabolism, and considered lactic acid to be the trigger for muscle contraction. In 1924, Hill and colleagues described a close correspondence between the blood lactate concentration ($[Lac]^b$) and $\dot{V}O_2$ determined before and after muscular exercise. Soon after this finding Hewlett et al (1926) observed that $[Lac]^b$ was decreased on inspiration of hyperoxic gas during exercise. This finding led Douglas (1927) to believe that muscle lactate production was influenced by oxygen availability. Owles (1930) put forward the

notion that during exercise, a unique threshold intensity existed, "a critical metabolic level", above which lactate production was accelerated. Owles recognised that increases in CO_2 excretion and ventilation (VE), along with a reduction of plasma bicarbonate accompanied the accumulation of lactate in the blood. In 1933, Margaria showed that $[Lac]^b$ could rise during sub-maximal exercise, while Bang (1936) showed that lactate only rose at work-rates of at least 50% of $\dot{V}O_2$max. Importantly, Bang (1936) also showed that the $[Lac]^b$ of trained individuals was lower than that of untrained individuals for any absolute workload.

During the late 1950's and early 60's, Hollman and colleagues, by measuring arterial and venous lactate and pyruvate concentrations during cycle exercise, concluded that the determination of arterial $[Lac]^b$ allowed the identification of the highest exercise intensity which could be performed exclusively by aerobic means. Venous $[Lac]^b$ was found to be lower than that of arterial blood, a finding that the researchers attributed to removal of lactate from the venous blood by less metabolically active arm muscles. Hollman and co-workers also observed that during incremental exercise, a point is reached where VE increases by a greater degree than does oxygen uptake. Changes in VE and blood lactate were thought to coincide and this breakpoint was defined as the "point of optimal ventilatory efficiency", a point at which a maximum amount of oxygen could be taken up with a minimum of ventilation (Weltman, 1995).

Carrying on from the research of Hollman, Wasserman and McIlroy (1964) first introduced the concept of the "anaerobic threshold" (AT), or as they described, "the threshold of anaerobic metabolism". According to Wasserman (1984) the AT is defined as "the level of exercise $\dot{V}O_2$ above which aerobic energy production is supplemented by anaerobic mechanisms". Wasserman suggested that pulmonary gas exchange could be used to estimate the lactate breakpoint observed during incremental exercise. By measuring the respiratory exchange ratio (RER) in exercise testing of cardiac patients (non-invasive technique), Wasserman and McIlroy were able to avoid repeated skin punctures (invasive measurement) for the obtainment of arterial blood lactate samples. This non-invasive technique was seen to significantly improve the usefulness of AT determination in exercise test diagnostics.

Wasserman's "anaerobic threshold" hypothesis inferred that a point is reached during progressive exercise where the oxygen required by the working muscles exceeds the oxygen supply. This imbalance between oxygen demand and supply causes an increase in the anaerobic conversion of pyruvate to lactate. Lactic acid is almost completely dissociated on formation due to its low pK, and liberated protons are buffered predominantly by intracellular and plasma bicarbonates, resulting in the production of "non-metabolic" CO_2. This additional CO_2 adds to what is normally produced as a result of metabolism, and as such, at the "anaerobic threshold" CO_2 levels begin to increase nonlinearly. In 1973, Wasserman and colleagues were the first to suggest that changes in these ventilatory variables could be used to accurately, and non invasively, determine the point of accumulation of lactate in the blood, i.e. the lactate threshold.

In the last thirty years, most of the literature in this area of exercise physiology has been directed towards the theoretical and mechanistical considerations of the blood lactate response to exercise with the AT hypothesis being challenged and defended. Davis (1985a, 1985b) supports the idea that the initial increase in blood lactate is due to the onset of lactate production in the muscle. Davis argues that the term "anaerobic threshold" is deceiving and should be referred to using other less mechanistically descriptive terms such as the lactate threshold (LT).

Brooks (1985a, 1985b) has challenged the fundamental concept of the AT hypothesis. Brooks suggests that the first major failing of the AT hypothesis is the issue of the muscle tissue becoming hypoxic during sub-maximal exercise. He cites research that suggests that during sub-maximal and even maximal exercise, critical levels of mitochondrial oxygen are not reached, and hence muscle tissue hypoxia does not occur. Brooks suggests that blood lactate levels are influenced not only by muscle lactate production but also blood lactate removal by skeletal muscle and other organs.

In recent years, evidence has accumulated that dissociates lactate production from anaerobiosis. Studies performed over the last 15 years using isotopic tracer technology have suggested that muscles can release lactate when their oxygen supply is more than

adequate (Brooks, 1991). The formation of blood lactate appears to be dependent on several factors, including but not limited to the availability of oxygen.

Apart from the controversies regarding the theoretical and mechanistic considerations of the blood lactate response to exercise over the years, this area of exercise science has become even more complicated through the use of many different terms and definitions in the literature, making comparisons between different studies difficult.

In recent years, the development of reliable rapid-response blood lactate analysers has made blood lactate assessment a quicker and easier physiological assessment method for exercise physiologists and coaches by doing away with the need for complicated and time-consuming methods of blood lactate analysis. The invention of these rapid-response blood lactate analysers in the last twenty years has had a huge influence on the amount of research data collected.

Today, despite the current controversies regarding the mechanistic characteristics of the blood lactate response to exercise, it is still widely accepted that the blood lactate response to exercise is a useful tool for predicting endurance performance and monitoring training status. Indeed, numerous studies have shown that the blood lactate response to incremental exercise appears to be highly correlated to various types of endurance performance (Allen et al, 1985; Coyle et al, 1991; Fay et al, 1989; Fohrenbach et al, 1987; Heck et al, 1985; Sjodin et al, 1981; Weltman et al, 1992) and because of these findings, blood lactate assessment is now used regularly on a worldwide basis by exercise physiologists as a predictor of endurance performance, but also as a tool for exercise prescription and the monitoring of aerobic fitness in professional athletes in the exercise laboratory or in field conditions.

(B) THEORY AND MECHANISMS OF BLOOD LACTATE ACCUMULATION

1) LACTATE FORMATION

Glycolysis is the term given to the breakdown of glucose into pyruvate, and high intensity, short duration exercise is fuelled predominantly via this anaerobic pathway since it is this energy pathway that supplies ATP at the highest rate. Glycolysis involves pyruvate being reduced by lactate dehydrogenase (LDH) to form lactic acid (McArdle and Katch, 1994). Once formed in the muscle, lactic acid rapidly dissociates to form a proton (H^+) and a lactate anion (Lac^-). The dissociation is buffered predominantly by the bicarbonate system – the lactate anion forms a salt with either Na^+ or K^+, while the proton joins with bicarbonate to form carbonic acid:

Lactic acid + sodium bicarbonate $\rightarrow$ sodium lactate + carbonic acid (H_2CO_3)

The carbonic acid produced is then converted into CO_2 and H_20 by the enzyme carbonic anhydrase:

$$H_2CO_3 \leftrightarrow CO_2 + H_2O$$

This conversion occurs both intracellularly and also when the H_2CO_3 enters the muscle vasculature (Brooks, 1985a). It has been contended that the terms 'lactic acid' and 'lactate' can be used interchangeably, due to the almost instantaneous dissociation of lactic acid to lactate (Brooks, 1991).

Once lactate is produced, it has two main effects (Spurway, 1992). Firstly, it can be carried in the bloodstream (blood lactate) to sites where it is further oxidized or resynthesised back to glucose (Brooks, 1985a, 1991; Spurway, 1992). Secondly, the lactate anion and proton may accumulate in the source fibres (intra-muscular lactate) where the liberated protons strongly contribute to the fatigue process. Levels of intra-muscular H^+ arising from lactic acid dissociation that lowers the intramuscular

environment pH to a value of 6.4 or lower, causes inhibition of glycolytic enzymes, with phosphofructokinase (PFK) in particular being affected (Spurway, 1992). The increased acidity of the intramuscular environment also affects the mechanics of force generation, as troponin C's affinity to bind to Ca^{2+} is affected (Brooks, 1985a).

2) GENERATION OF BLOOD LACTATE ACCUMULATION

During low levels of incremental exercise there is a minimal increase, no change, or sometimes a decrease in blood lactate concentration (Figure 1). However, as the intensity of the exercise increases, a work load is reached above which the blood lactate response to increasing exercise intensity is curvi-linear (Weltman, 1995). Several potential mechanisms that cause this response have been suggested and will now be discussed.

FIGURE 1

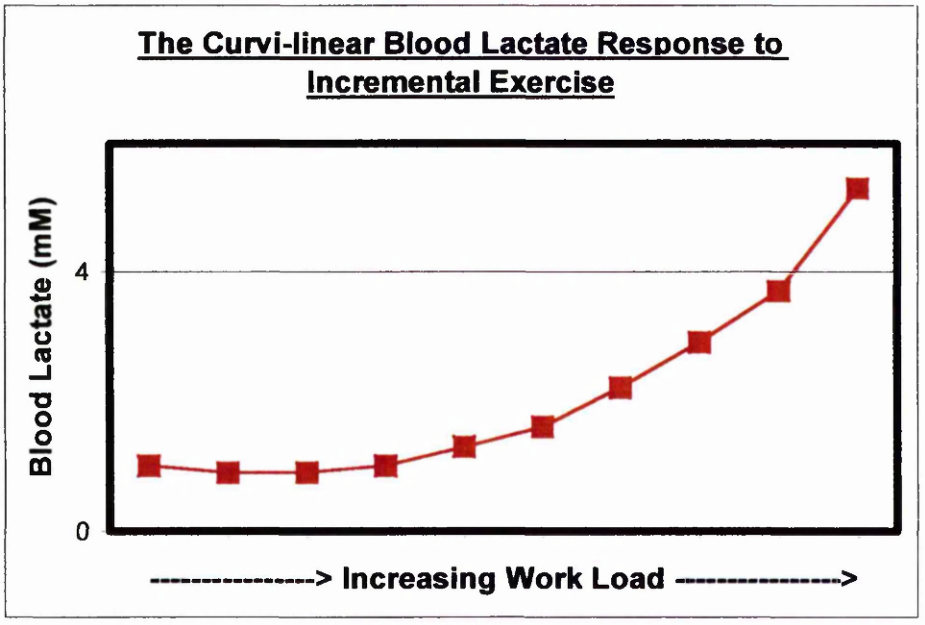

Figure 1 - The Blood Lactate Response to Exercise. With increasing work load, baseline blood lactate values typically begin to increase in a curvi-linear fashion after reaching a specific work-load, traditionally referred to as the "lactate threshold".

The amount of lactate present in the blood at any given time is due to lactate **production**, but also lactate **clearance**.

(a) MECHANISMS OF LACTATE PRODUCTION

OXYGEN AVAILABILITY

Muscle oxygen availability has been linked with lactate production for almost a century. In 1923, Hill and Lupton determined that hypoxia increased lactate production when studying isolated muscles in vitro. Hogan et al (1983) have shown that breathing hypoxic gas ($FIO_2 = 0.17$) increases $[Lac]^b$ and breathing hyperoxic gas ($FIO_2 = 0.60$) reduces $[Lac]^b$ in comparison to the $[Lac]^b$ elicited by breathing room air during incremental exercise. Wasserman (1984) cites these results as evidence that hyperoxic gas mixtures increase oxygen delivery to the muscles thereby alleviating muscle hypoxia and reducing lactic acid production, while Brooks (1985b) argues that hyperoxia may lower $[Lac]^b$ due to an increased perfusion of organs capable of lactate clearance. While muscular hypoxia can stimulate increased lactate production at the onset of exercise and during maximal exercise, evidence now exists that muscle lactate production during sub-maximal exercise need not imply the existence of tissue hypoxia. For example, Connett et al (1984) when using auto-perfused preparations of canine gracilis (red muscle) contracting in situ confirmed muscle lactate production in the absence of hypoxia.

CATECHOLAMINERGIC STIMULATION

Researchers have reported that blood catecholamines demonstrate a curvi-linear increase during progressive exercise, and this response has been speculated to be a possible cause of the curvi-linear blood lactate response to exercise (Peronnet et al, 1981; Lehmann et al, 1981; Jevoza et al, 1985; Jansson et al, 1986; Lehmann et al, 1986; Mazzeo and Marshall, 1989; Podolin et al, 1991; Mazzeo et al, 1994). Mazzeo and Marshall (1989) reported simultaneous threshold increases in plasma epinephrine and blood lactate concentration at identical workloads ($r = 0.97$), and hypothesised that increased

epinephrine levels stimulate muscle glycogenolysis therefore increasing muscle lactate production.

MOTOR UNIT RECRUITMENT

Some researchers have suggested that the increase in $[Lac]^b$ seen during incremental exercise is due to the progressive recruitment of motor units with greater glycolytic capacity, and report that the recruitment of FT fibres and LT are closely associated (Jones and Ehrsam, 1982; Costill et al, 1973; Vollestad et al, 1984; Vollestad et al, 1985). Nagata and co-workers (1981) demonstrated a strong correlation between integrated electromyography (iEMG) and LT ($r = 0.92$), and suggested that the increased iEMG found was due to the recruitment of FT fibres and increased firing of motor units already recruited. The physiological and metabolic characteristics of FT fibres relative to ST fibres supports the notion of the influence of FT fibre recruitment on blood lactate production. Davis (1985a) however, argues that FT motor unit recruitment may occur in response to muscle lactate production.

(b) MECHANISMS OF LACTATE CLEARANCE

Traditionally it was accepted that the liver was the major site of lactate removal from the blood during exercise, but it is now clear that the liver is not the only site of lactate clearance, being found to not even be a major site of lactate clearance due mainly to the fact that blood flow is redistributed away from tissues such as the liver and directed towards the exercising musculature and skin at the onset of exercise (Brooks, 1985a). Skeletal muscle and cardiac muscle are now accepted to be the major sites of blood lactate clearance, using lactate as a substrate (Stainsby and Brooks, 1990).

The mechanisms of lactate efflux are complex, and do not always involve simultaneous efflux of H^+. Studies have shown that H^+ efflux from contracting musculature greatly exceeds that of Lac^-. Research by Brooks has stressed the importance of mechanisms of lactate clearance in the determination of blood lactate accumulation. Brooks (1986) argues that lactate is an important gluconeogenic pre-cursor during exercise. Skeletal muscle is probably the most prominant site of lactate removal, being able to extract

lactate from the blood for gluconeogenesis even when it exhibits a net lactate release (Stanley et al, 1986). Lactate produced in neighbouring muscle fibres may be metabolized in less active neighbouring muscle fibres (Chirtell et al, 1984).

Jones (1994) concluded that the following mechanisms are the most important for the generation of the blood lactate response to incremental exercise –

- transient increases in blood lactate concentration at the onset of exercise by mass action
- recruitment of FT fibres at higher exercise intensities being primarily responsible for the LT phenomena
- increased lactate production in all fibre types by catecholaminergic stimulation of muscle glycogenolysis at high exercise intensities
- further increased lactate production beyond LT due to recruitment of FT fibres, and development of anoxic loci in muscle when close to maximal work loads
- temperature effects on metabolism and ventilation

(C) FACTORS AFFECTING THE INTERPRETATION OF THE BLOOD LACTATE RESPONSE TO EXERCISE

There are numerous factors that may affect the researcher's interpretation of the blood lactate response to exercise. It is these factors that make it extremely difficult to make comparisons between studies:

TERMINOLOGY

As mentioned previously, the plethora of terminology used to describe the blood lactate response to exercise confuses the issue of comparing the results from different studies. Terms such as lactate threshold (Weltman, 1995), maximal lactate steady state (MLSS) (Tegtbur, 1993), anaerobic threshold (Wasserman, 1973), aerobic threshold (Kindermann et al (1979), aerobic-anaerobic threshold (Mader et al, 1976), individual anaerobic threshold (Keul et al, 1979), individual aerobic threshold (Stegmann and

Kindermann, 1982), lactate breaking/inflection point (Weltman, 1995), and lactate minimum running speed (Romer et al, 1998) have all been used over the last thirty years to describe the blood lactate response to exercise (Figure 2). Furthermore, the lactate threshold has been defined by some researchers as the work-rate (or $\dot{V}O_2$) associated with a blood lactate inflection/breakpoint, while others have set the lactate threshold as a fixed blood lactate concentration (FBLC), e.g. 1 mmol.l^{-1} above baseline, or the work-rate/ $\dot{V}O_2$ corresponding to a FBLC of 2.5, 3, or more commonly 4 mmol.l^{-1} (Heck et al, 1985). To complicate matters further, some investigators have labeled the FBLC of 4 mmol.l^{-1} as the AT or onset of blood lactate accumulation (OBLA) (Sjodin and Jacobs, 1981).

FIGURE 2

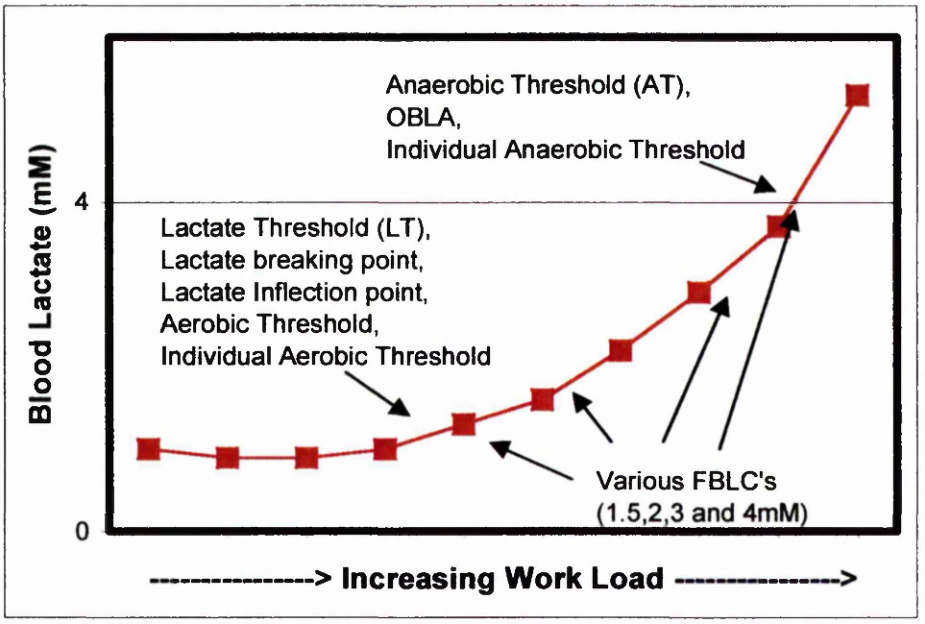

Figure 2 - Blood lactate marker terms used by researchers. Over the years, various researchers have used many different terms and definitions to describe the blood lactate response to exercise, causing confusion in a convoluted area of exercise science.

TESTING PROTOCOL

One factor that affects the measurement of the blood lactate response to exercise is the testing protocol used. Some researchers have used continuous incremental protocols

(Sjodin and Jacobs, 1981; Tanaka, 1983; Weltman et al, 1990) whilst others have used discontinuous exercise bouts (Allen et al, 1985; Coyle et al 1983; Farell et al, 1979; Hagberg et al, 1982). Stage duration of incremental tests has been shown to influence the interpretation of the blood lactate response (Weltman, 1995). Numerous studies have demonstrated that the longer the stage duration, the lower the AT values (Foxdal et al, 1996; Freund et al, 1989; Heck et al, 1985; McLellan, 1987; Rusko et al, 1986; Yoshida, 1984).

BLOOD MEDIA

The blood media collected for analysis influences the blood lactate concentration measured (Foxdal et al, 1990, 1991) For example, Foxdal (1991) and his researchers reported that measuring lactate in the plasma, venous blood, and capillary blood in the same subjects during an incremental sub-maximal test resulted in the power output at the 4mmol.l^{-1} marker to be 180, 204, 216 Watts (W) for venous plasma, capillary blood, and venous blood respectively.

(D) FACTORS AFFECTING THE BLOOD LACTATE RESPONSE TO EXERCISE

Investigators have identified several factors that can alter the blood lactate response to exercise:

MUSCLE FIBRE TYPE/METABOLIC PROFILE

Ivy et al (1980) reported that the percentage of ST fibres of the vastus lateralis muscles of cyclists was significantly related to both absolute ($r = 0.74$) and relative ($r = 0.70$) LT. A strong relationship was also found between the muscle's respiratory capacity and LT, suggesting that the mitochondrial content of the muscle is an important determinant of LT. Ivy and researchers (1980) concluded that the ratio of ST to FT fibres may have a genetic influence upon LT, ultimately affecting the ability to improve LT. Komi (1981) found that OBLA was significantly related to the percentage of ST fibres in runners ($r = 0.78$), while Sjodin and Jacobs (1981) found that OBLA was positively correlated to

%ST fibres and %ST fibre area. Tesch et al (1981) also found that 92% of the variance in OBLA was explained by the %ST area and capillary density. During incremental exercise, motor unit recruitment is thought to progress from an initial recruitment of ST fibres to FT fibres, therefore it is apparent that athletes with high percentages of ST (and FTa) fibres should be able to attain greater absolute work rates before observation of LT.

An important study by Aunola et al (1988) investigated the power of muscle metabolic profile and oxygen transport capacity on the measurement of the aerobic and anaerobic threshold. Aunola and co-workers constructed a factor type model to estimate to what extent the thresholds could be explained by muscle metabolic profile and oxygen transport capacity. A factor model using four factors ($\dot{V}O_2$max, sub-maximal endurance [measured by aerobic and anaerobic threshold], muscle metabolic profile, and oxygen transport capacity) was constructed. Sub-maximal endurance correlated strongly with $\dot{V}O_2$max ($r = 0.92$), but correlated even more strongly with muscle metabolic profile ($r = 0.83$) than with oxygen transport capacity ($r = 0.41$). Muscle metabolic profile was also found to correlate more strongly with sub-maximal endurance ($r = 0.83$) than with $\dot{V}O_2$max ($r = 0.70$). The authors concluded that the aerobic and anaerobic thresholds are to be considered as better indicators of sub-maximal endurance than $\dot{V}O_2$max, due largely to the influence of muscle metabolic profile. Weltman (1995) concluded that muscle metabolic characteristics are of major importance in determining the blood lactate response to exercise.

SUBSTRATE AVAILABILITY

Results from two studies by Yoshida (Yoshida, 1984; Yoshida, 1986) and a study by Ivy (Ivy et al, 1981) has shown that alterations in substrate availability can affect endurance performance, findings that led several researchers to speculate that altering substrate availability might affect the blood lactate response to exercise.

Hughes et al (1982) compared LT and AT in subjects who were in glycogen depleted and normal glycogen states. The glycogen depleted state was found to cause a dissociation between LT and AT, with LT occurring at a greater work load and AT

occurring at a lower work-load when compared to a normal glycogen state. Yoshida (1984) observed that a carbohydrate (CHO) rich diet, a low CHO diet, and a mixed diet (3-4 days on each diet) had no effect on LT. However, because the high CHO diet was associated with an increased $[Lac]^b$ at each sub-maximal exercise stage, the work load and $\dot{V}O_2$ at 4 $mmol.l^{-1}$ were significantly reduced after the high compared to the low CHO diet.

CAFFEINE USE

Studies that have investigated the effect of caffeine ingestion on the blood lactate response to exercise have produced mixed results, with some studies reporting that caffeine ingestion before exercise increases $[Lac]^b$, while others state that caffeine has no effect (Bangsbo et al, 1992a; Dodd et al, 1991; Falk et al, 1989; Gaesser and Rich, 1985; Graham and Spriet, 1991; Sasaki et al, 1987; Tarnopolsky et al, 1989; [cited by Weltman, 1995]).

(E) THE EFFECT OF AEROBIC FITNESS TRAINING ON THE BLOOD LACTATE RESPONSE TO EXERCISE

Aerobic fitness training results in numerous adaptations to the neuromuscular, metabolic, cardiovascular, respiratory and endocrine systems. These adaptations are manifested by a graphical shift in the blood lactate response curve. For example, a typical graphical change in the blood lactate response curve of an individual who had undertaken 6 weeks of endurance training is illustrated in Figure 3.

1) PHYSIOLOGICAL ADAPTATIONS TO AEROBIC FITNESS TRAINING

It is widely reported in the literature that aerobic fitness training reduces blood lactate levels, and it has been suggested that training affects the blood lactate response to exercise by decreasing the rate of lactate production (caused by lowering of the rate of muscle glycogen utilization, or by faster oxygen uptake kinetics that may increase initial O_2 availability), or increasing the rate of lactate clearance from the exercising

musculature, or both (Bonen et al, 1997; Donovan and Brooks, 1983; Donovan and Pagliassotti, 1990; Favier et al, 1986; Freund et al, 1992; MacRae et al, 1992; Stanley et al, 1985).

FIGURE 3

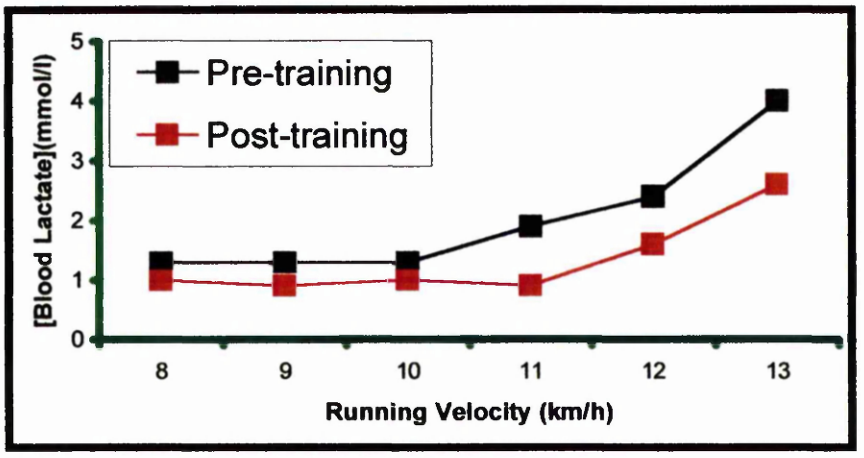

Figure 3- The effect of 6 weeks of endurance training on the blood lactate response to exercise [modified from Carter et al (1999)]. After endurance training, the blood lactate curve has shifted downwards and to the right.

Physiological adaptations to aerobic training that cause graphical shifts in the blood lactate response curve are now discussed.

SKELETAL MUSCLE ADAPTATIONS

Successful aerobic fitness training results in numerous beneficial adaptations within skeletal muscle, including an increase in sodium-potassium pump concentration (Green et al, 1993) an increased lactate transport capacity (Pilegraard et al, 1994; McCullagh et al 1996), and possibly an increased myoglobin concentration (Harms and Hickson, 1983). Endurance training markedly increases the oxidative capacity of skeletal muscle, due to an increase in size and number of mitochondria per unit area, and an increase in the concentration of specific enzymes belonging to the Krebs cycle, electron transport chain and malate-aspartate shuttle (Spina et al, 1996; Schantz et al, 1986; Suter et al, 1995). These adaptations to aerobic training maintain cellular phosphorylation potential,

improve the sensitivity of respiratory control and also increases the capacity for aerobic ATP resynthesis during exercise in both ST and FT muscle fibres. Higher concentrations of oxidative enzymes in ST muscle fibres might delay the point at which FT fibres are recruited during exercise (Moritani et al, 1993), while an increase in the oxidative potential of FT fibres may reduce their reliance on anaerobic glycolysis for ATP production (Gollnick and Saltin, 1982), therefore reducing lactate production. The greater capacity of the Krebs cycle to accept pyruvate following endurance training may be important in reducing the production of lactate by mass action at the onset of exercise and during high-intensity exercise (Graham and Saltin, 1989).

A strong relationship exists between the percentage of ST fibres and the LT (Ivy et al, 1980; Weston et al, 1999; Aunola and Rusko, 1992). Aerobic training causes a selective hypertrophy of ST fibres, and can cause a shift in muscular enzymatic profile, e.g. FTb to FTa (Gaesser et al, 1984; Anderson and Henriksson, 1977) and perhaps FTa to ST (Simoneau et al, 1985; Sale et al, 1990) over a long period of endurance training.

SKELETAL MUSCLE CAPILLARISATION

Aerobic training increases the capillarity of skeletal muscle (Andersson and Henriksson, 1977; Ingjer, 1979) ultimately having the effect of increasing the maximal muscle blood flow capacity, and increasing the surface area available for the exchange of gases, substrates and metabolites between the muscle-blood barrier. This greater capillarity of aerobically trained muscle allows for a greater uptake of FFA's from the blood, and, along with an increased activity of enzymes involved in lipid metabolism, the capacity for mitochondrial B-oxidation is increased by aerobic training, lowering the rate of glycolysis and production of lactate of the exercising musculature.

HORMONAL RESPONSE ADAPTATIONS

The catecholamine response to exercise has been found to be significantly blunted by aerobic training after only a few training sessions (Mendenhall et al, 1994; Green et al, 1989). A lower secretion rate of epinephrine (a major effector of lactate production through modulation of muscle glycogenolysis) at the onset of exercise will result in a

positive shift in the blood lactate response curve. The overall effect of aerobic training of reducing sympathetic nervous system activity will also contribute to changes seen in the blood lactate response curve.

OXYGEN UPTAKE KINETICS

Some studies have evaluated the effects of endurance training on $\dot{V}O_2$ kinetics, and although the steady state $\dot{V}O_2$ for the same moderate intensity exercise has not been found to change following a period of endurance training (Davis et al, 1979; Hagberg et al, 1980), the primary exponential increase in $\dot{V}O_2$ at the onset of exercise may be accelerated. Faster $\dot{V}O_2$ kinetics at the onset of exercise which results in a faster attainment of the steady state oxygen uptake requisite may decrease the early increase in lactate production of the exercising muscles, causing a positive change in the blood lactate response curve.

(F) INTERPRETATION OF TRAINING INDUCED SHIFTS IN BLOOD LACTATE RESPONSE CURVES

Graphical overlays of the individual athlete test-retest blood lactate profiles, with a subjective assessment of any curve shifts, are the most commonly used methods of determining the extent of adaptation to aerobic fitness training (Bourdon, 2000).

The most common changes in the blood lactate-intensity curve and their interpretations according to Madsen and Lohberg (1987), Pyne (1989), Weltman (1995) and Bourdon (2000) are as follows –

- A shift in the blood lactate response curve down and/or to the right is indicative of an increase in aerobic fitness of the subject, expressed by the subject's increased ability to exercise at a greater intensity for a given blood lactate level or to express lower blood lactate levels for the same intensity (Figure 4)

- A graphical shift upwards and/or to the left indicates deterioration in the aerobic fitness of the subject. This is expressed by the subject exercising at a lesser intensity for a given blood lactate level or expressing a higher blood lactate level for the same intensity (Figure 5)

FIGURE 4

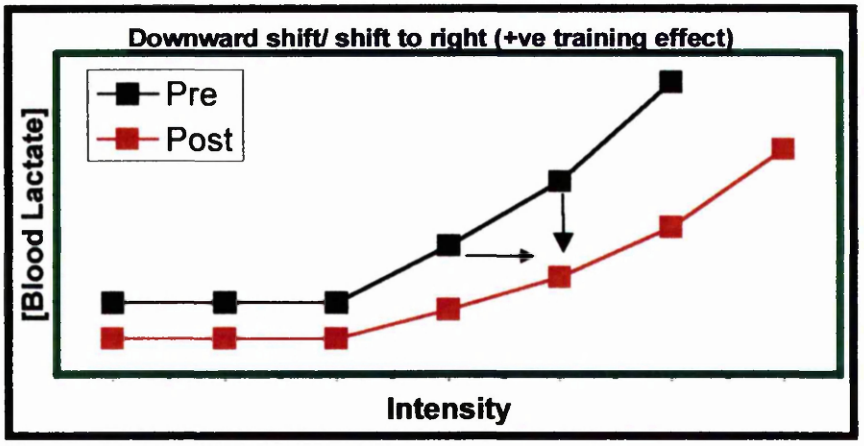

Figure 4 - Graphical shifts in the blood lactate response curve due to a positive training effect. A positive training effect results in a downward shift and shift to the right of the blood lactate curve.

FIGURE 5

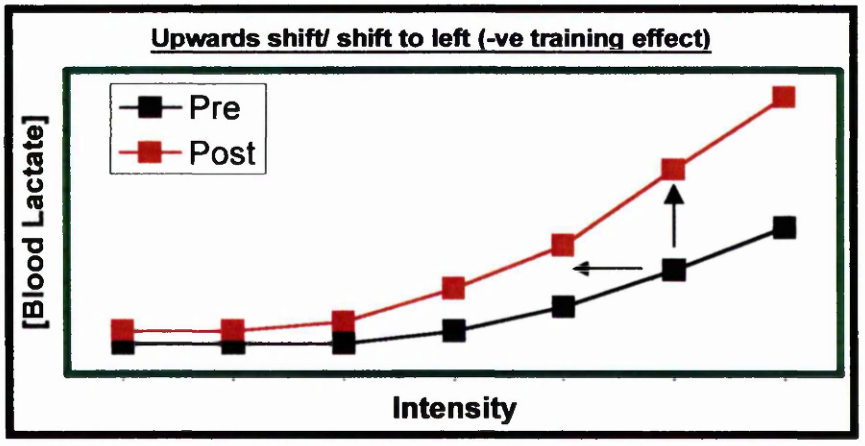

Figure 5 - Graphical shifts in the blood lactate response curve due to a negative training effect. A negative training effect results in an upwards shift and shift to the left of the blood lactate curve.

(G) SUB-MAXIMAL BLOOD LACTATE ASSESSMENT OF SOCCER PLAYERS – PREVIOUS RESEARCH

It has been discussed that improved endurance performance as a result of aerobic training is associated with lowered blood lactate concentrations during sub-maximal continuous exercise and specific graphical shifts of the blood lactate response curve. This makes blood lactate measurements a potentially useful tool for evaluating aerobic fitness levels in soccer players, and un-surprisingly, such measures have been previously obtained by various researchers (Bangsbo and Lindquist, 1992; Bangsbo, 1994; Brady et al, 1995; Dunbar, 1999; Jensen and Larsson, 1993; Nowacki et al, 1988; White et al 1988).

It is difficult to compare the results of different studies dealing with the blood lactate response to exercise as the results and conclusions reported are dependent on several factors that affect the interpretation of the results of the study, such as exercise mode, duration of the sub-maximal bouts, and the blood media sampled. Due to these difficulties, the results and conclusions of this study have been compared to five previous studies only. These studies have been selected primarily due to the fact that all five studies:

1) used soccer players for subjects,
2) utilised an incremental sub-maximal blood lactate assessment protocol,
3) used running on treadmills as the exercise mode,
4) measured aerobic fitness of the soccer players across either a pre-season training period , a soccer season, or a longer period of time.

The results of this study compared to that of the five studies selected will be discussed in Chapter 5.

CHAPTER 3

METHODS

CHAPTER 3 - METHODS

SUBJECTS

Thirty-seven male professional soccer players aged 18.9 $\pm$ 1.8 years (mean $\pm$ sd) participated in this study. All the players were in good health and free of injury at each testing time-point.

Each player completed a medical and physical activity questionnaire and were required to sign a consent form after being informed of the procedures and risks involved before each assessment. The study was approved by the University of Glasgow Interim Ethics Committee for Non-Clinical Research Involving Human Subjects. All testing was completed in the University of Glasgow exercise physiology laboratory (BASES accredited).

TESTING TIME-POINTS

The study involved the players being assessed by sub-maximal blood lactate assessment on a treadmill at six strategic time-points throughout a soccer season -

1) July (Beginning of pre-season training)
2) October
3) December
4) January
5) April
6) June (end of season)

TIME CONSTRAINTS OF SUBJECTS

Since the subjects in this study were professional youth soccer players, all testing was completed as quickly as possible so that there was minimal disruption to the players training schedule. All of the players were tested within 2 to 3 days at each testing time-

point. All efforts were made to ensure that each player was tested at approximately the same time of day (± 1 hour) at each of the testing time-points.

SUBJECT FAMILIARISATION

All subjects were highly experienced in treadmill running and wearing a HR monitor. All subjects were accustomed to blood collection procedures, but not necessarily blood collection whilst treadmill running. For these particular subjects, blood was extracted from their thumb during the warm-up on their first visit to the laboratory to familiarise them to the blood collection procedure.

TESTING PROCEDURES

On arriving in the laboratory, each player was given an information sheet, a consent form and an activity diary. All subjects completed and signed a medical questionnaire (Appendix 1). The subjects were instructed to participate only in light activities (or preferably no activity at all) during the day preceding the assessment day. The players were instructed not to consume any food or caffeine for the 3 hours before each test. These guidelines were given to the players (and their coaches) in the hope that the players would arrive for each assessment throughout the season in comparable physiological states.

ANTHROPOMETRIC DATA

Before the warm-up procedure, the players were weighed in sports kit and socks and had skinfold measurements taken for estimation of % body-fat using Harpenden skin-fold calipers (Durnin and Womersley, 1974) Three skin-fold measurements were taken from four sites: sub-scapula, supra-iliac, biceps and triceps. The average skin-fold measurement from each site was recorded and from the total of the four sites, % body-fat was calculated (Durnin and Wormsley, 1974). These measures were taken in order to describe the population and also to provide extra physiological data for the players, coaches, and medical staff.

WARM-UP PROCEDURE

Before the lactate profile assessment, the subject was fitted with a Polar HR monitor (Polar Accurex Plus, Kempele, Finland) and performed a 5-minute warm-up at a HR of approx 120 to 130 bpm (an exercise intensity that is typically below a soccer player's vLT) on the test treadmill. All testing was carried out on a Woodway ERGO ES2 (Cranlea, Birmingham, UK), a sophisticated treadmill specially designed for exercise testing. Before the warm-up procedure the subject was instructed on the safety features of the treadmill. On cessation of the warm-up the subject rehearsed emergency termination of the test that involved the subject raising his legs away from the treadmill belt by supporting the body on the treadmill hand-rails. Finally, the subject carried out static and ballistic stretching exercises for approximately 3-5 minutes, or until the subject felt that he was adequately prepared to commence the test.

INCREMENTAL LACTATE PROFILE PROTOCOL

Each test started at a treadmill speed that was estimated to elicit a HR of approximately 60% of maximum HR. The incremental lactate profile test used in this study consisted of individual 4-minute exercise stages, with a 0.5 $km.h^{-1}$ increase in treadmill speed until termination of the test. The test was terminated when the blood lactate concentration of the exercising subject exceeded 4 $mmol.l^{-1}$ or by the subject's own request. HR was recorded at 3 minutes and 45 seconds. The subject was not aware of his HR throughout the test. Blood samples were withdrawn from the subjects thumb after 4 minutes by pin-prick. After successful attainment of the capillary blood sample (the procedure time taking approximately 10 - 15 seconds), the treadmill speed was increased. During all tests a fan was directed at the subjects' head and upper torso to aid cooling.

TREADMILL SPEED MONITORING

During the second minute of each exercise test stage, an odometer was used to obtain an accurate treadmill speed. In all cases, the value obtained from the odometer reading was taken to represent the true speed of the treadmill.

BLOOD LACTATE ANALYSIS

All blood sampling was carried out by the same researcher for all of the tests, adhering to the guidelines set down by the British Association of Sports and Exercise Science (BASES). Blood samples were taken from the subject's thumb while the subject was running. The thumb was first cleaned with an alcohol Mediswab, and then a small puncture in the skin was made using an Autoclix lancet. The first drop of blood after puncture was discarded to prevent sweat contaminating the collected blood sample. Approximately 20 – 25 μl of arterialised capillary blood was then collected in a capillary tube containing a fluoride/heparin/nitrite mixture. The blood samples were thoroughly mixed for 3 minutes and were then analysed for whole blood lactate concentration using an Analox GM7 Multi-assay rapid response analyser. The assay is a simple one step procedure that involves lactate reacting with oxygen to form pyruvate:

$$\text{L-Lactate} + O_2 \rightarrow \text{Pyruvate} + H_2O_2$$

The reaction is catalysed by the enzyme L-Lactate oxygen oxidoreductase (LOD) at pH 6.5. Buffered reagent is entrained into the reaction chamber of the analyser with the reaction being initiated by injection of the blood sample. The maximum rate of oxygen consumption measured by an electrical change across the membrane of the electrode is directly related to lactate concentration.

All blood samples were measured in duplicate. If the concentration of the two blood samples differed by no more than 0.2 $mmol.l^{-1}$, the average value was recorded. If the concentration of the two original blood samples differed by more than 0.2 $mmol.l^{-1}$, a third blood sample was analysed with the average of the two closest results taken.

Before all tests, and at regular intervals throughout each test, the Analox GM7 analyser was calibrated with an aqueous 8.0 $mmol.l^{-1}$ lactate standard supplied by the manufacturers.

AEROBIC FITNESS MARKER DETERMINATION

Each individual data set was analysed for two important aerobic fitness markers:

(1) the running velocity at the estimated lactate threshold (vLT) and,
(2) the running velocity at a fixed blood lactate concentration (FBLC) of 4 $mmol.l^{-1}$ (vLac4).

vLT DETERMINATION

Lactate threshold was identified in this study as the first significant elevation of blood lactate above resting levels (Kindermann et al. 1979). In order to achieve objectivity, a mathematical regression analysis was applied to model the relationship between $[Lac]^b$ and running velocity (Orr et al. 1982). vLT was determined using a specially designed Minitab macro devised by a member of staff of the University of Glasgow Statistics Department. When $[Lac]^b$ is plotted against running velocity the inflection point of the lactate profile "curve" (corresponding to the lactate threshold) is identified by the computer programme. This inflection point is identified by the interception of two regression lines that the Minitab macro has estimated as the best fits of the data points.

vLac4 DETERMINATION

With blood lactate concentration being plotted against running velocity, a straight line was extrapolated between the two points corresponding to the running velocities directly before and after the 4 $mmol.l^{-1}$ concentration using Microsoft Excel Version 7. The exact running velocity at the 4 $mmol.l^{-1}$ concentration was calculated by linear extrapolation, where $y = mx + c$, and $y = 4$.

TEST DATA ELIMINATION

Test data from a player who was not involved in full soccer practice at a specific testing time-point was not included in the statistical analysis, i.e. players that were undergoing a period of injury rehabilitation. Data collected from players who missed 6 weeks or more

of soccer training due to illness or injury were excluded from the study. All data from goalkeepers were also excluded.

STATISTICAL ANALYSIS

All statistical analyses were performed using Minitab and Statistica computer applications.

Due to the fact that professional soccer players were used as the subjects in this study, the players were not always available at every testing time-point, sometimes because of illness or international youth team commitments, but mostly due to injury. Therefore, considerable imbalance in the data in terms of missing values was anticipated.

The missing data in this study were assumed to be missing at random. Appropriate statistical tests were selected to analyse the data in order to include as much 'useful' information as possible at each testing time point.

There were four possible approaches available to deal with the problem of missing data in this study. The first approach involved excluding all players that have any missing data. Use of this approach however would have left only 9 players for inclusion in the analysis, discarding 28 players test data (i.e. 75% of the players). This strategy was considered far from ideal as a large percentage of potentially useful data are discarded.

The second possibility involved replacing the missing data with imputed values (i.e. the mean value for all players with complete data at that time point). Given the large proportion of missing data in this study this approach was not used either as a large degree of variability will be incorrectly reduced by substituting missing values with the mean.

The third possible approach was to use a General Linear Model where all the data available at each time point are included. For example, if a player has complete data for October but has data missing for all subsequent time points, his data **are** included when calculating the mean for October, but contributes no information at all other times. This

approach is sensible in that all the available data **are** used in the comparison but it has the drawback of allowing players with missing data having an overly strong 'influence'. Consider a 'fictitious' player with high aerobic fitness marker measurements across all the testing time-points that was available for testing in October only. The mean value will increase at the first time point due to this player's contribution but will drop (incorrectly) at the times when this player was unavailable for testing. This drop is purely due to the player being missing but may be interpreted as a 'real' effect. Therefore, this statistical approach was not used to analyse the data set in this study.

The following statistical approach that was used in this study was to apply Bonferroni adjusted paired t-tests for all the 10 possible time point comparisons. The confidence level for each test is set at a higher level than 95% so that collectively all the tests simultaneously have a confidence level of 95% (in this case with 10 comparisons the confidence level is set at 0.05/10*2 = 99.75%). This approach had the advantage that all comparisons involved only those players that had complete 'paired' data available for analysis. The fictitious player introduced above would not be involved in any such comparisons as he contributes information at one testing time point only. However, a player who has measurements recorded at the start of the competitive season (October) and at the end of the season (June), for example, would be included in the 'October-June' comparison.

Summary statistics, paired t-tests, a Repeated Measures Analysis of Variance (ANOVA), and Bonferroni multiple comparisons procedures were also carried out to complete the statistical analysis of the study.

CHAPTER 4

RESULTS

CHAPTER 4 - RESULTS

(A) RESUME

Two blood lactate markers of aerobic fitness, namely the running velocity at the lactate threshold (vLT), and the running velocity eliciting a blood lactate concentration of 4mmol.l^{-1} (vLac4) were measured in 37 male professional youth soccer players at six distinct time points during a soccer season. The main aim of the study was to investigate formally whether there is a change in aerobic fitness across the testing time points as evidenced by changes in each of the two aerobic fitness markers.

There are two hypothesis of interest in this study. The first concerns a comparison of the mean blood lactate measurements at pre-season and October in order to determine if pre-season training produces an aerobic training effect, while the second hypothesis concerns a comparison of the aerobic fitness markers over the course of the competitive playing season (from October to June).

The null hypotheses were –

1) Aerobic fitness levels would not change significantly from pre-season to October.

2) There would be no significant fluctuations in aerobic fitness over the course of the competitive season.

(B) SUMMARY STATISTICS AND SUBJECTIVE IMPRESSION

A visual impression of the data is presented below in Tables 5 and 6.

TABLE 5

vLT ($km.h^{-1}$) Descriptive Statistics

Time-Point when Tested	Players Tested (n)	Missing Players (n)	Mean vLT ($km.h^{-1}$)	Minimum vLT ($km.h^{-1}$)	Maximum vLT ($km.h^{-1}$)	Standard Deviation
Pre-Season	16	21	11.39	9.22	13.30	1.05
October	37	0	12.62	10.93	14.08	0.73
December	30	7	12.69	11.08	14.62	0.82
January	20	17	12.35	10.65	14.16	0.94
April	20	17	12.37	10.72	14.21	0.84
June	21	16	12.39	10.62	13.89	0.84

TABLE 6.

vLac4 ($km.h^{-1}$) Descriptive Statistics

Time-Point when Tested	Players Tested (n)	Missing Players (n)	Mean vLac4 ($km.h^{-1}$)	Minimum vLac4 ($km.h^{-1}$)	Maximum vLac4 ($km.h^{-1}$)	Standard Deviation
Pre-Season	14	23	13.02	11.95	13.96	0.70
October	34	3	14.17	12.20	15.76	0.82
December	29	8	14.14	12.70	15.97	0.82
January	17	20	13.97	11.60	15.75	1.11
April	18	19	14.27	12.29	16.50	1.02
June	18	19	14.37	12.83	15.67	0.80

There is an imbalance in the number of players tested at each time point. The largest frequency of vLT and vLac4 missing values occurred at the pre-season testing (21 and

23 players respectively). When considering the vLT data only, 17 players (46%) were not tested in January or April. If the players not tested are missing at random (i.e. they represent a random sample of players and do not represent a specific group with predominately low or high lactate levels) then we can make reliable inference otherwise the conclusions may not be valid. It is plausible to assume that missing data from players who contributed measurements for some time points only, but not others, were missing at random. Unfortunately, this assumption of 'randomness' is difficult to verify formally.

In order to investigate the agreement between vLT and vLac4, scatterplots of these measurements for each time point are given in Figure 6 below. The line of best fit (with 95% confidence intervals) and the line of equality are shown.

FIGURE 6

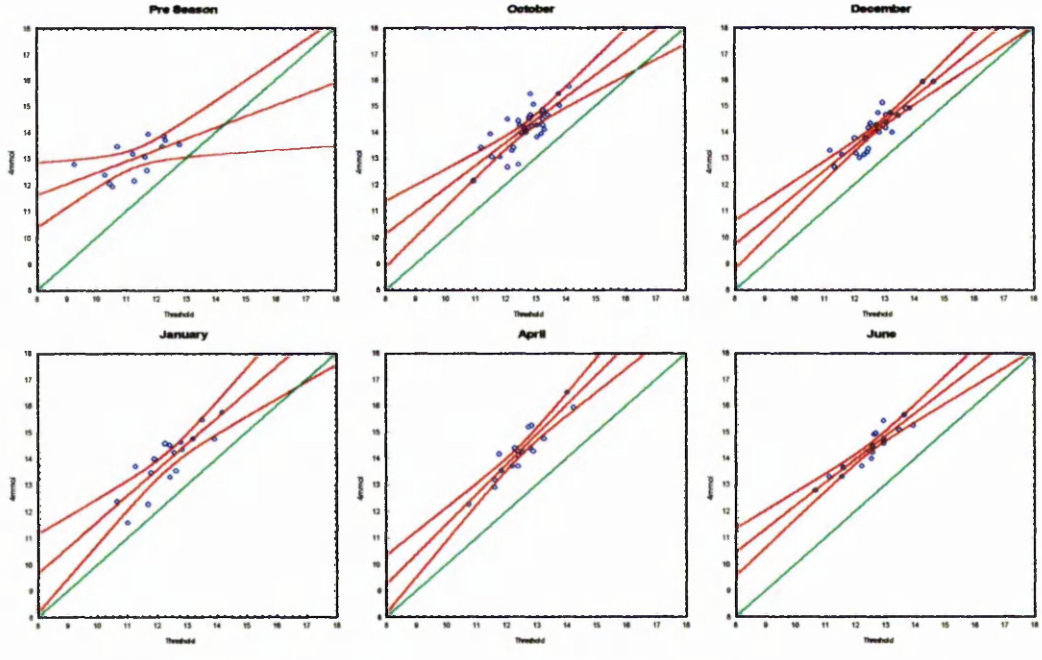

Figure 6 - Scatterplots of vLT and vLac4 for each testing time-point.
(x-axis - vLT, y-axis – vLac4)

There is a strong positive correlation between both markers at each testing time-point and, ignoring the pre-season data there is a consistent 'additive' bias where vLac4 tends to be on average 1.5 mmol.l^{-1} greater than vLT.

Case-profile plots for all tested players at each time point are given below in Figures 7 and 8.

FIGURE 7

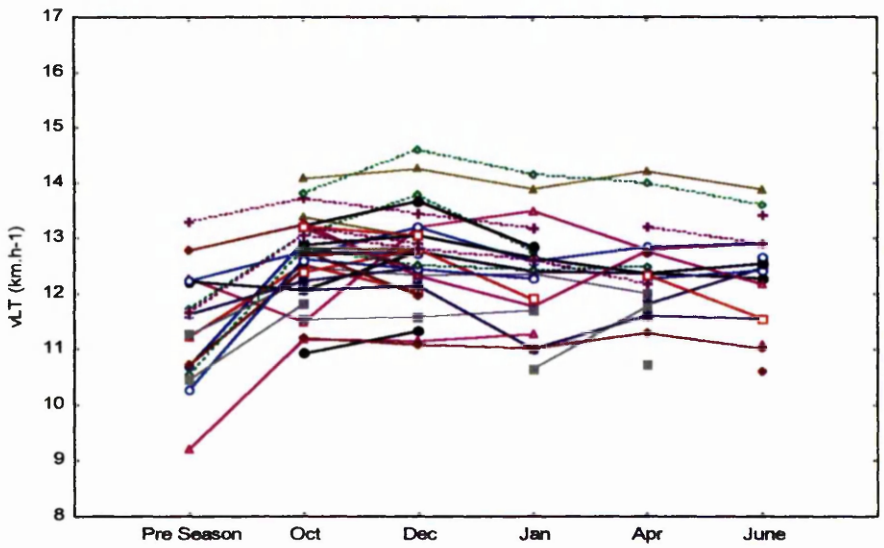

Figure 7 - Case Profile Plot of vLT across the testing time-points, showing individual variations over the full soccer season.

FIGURE 8

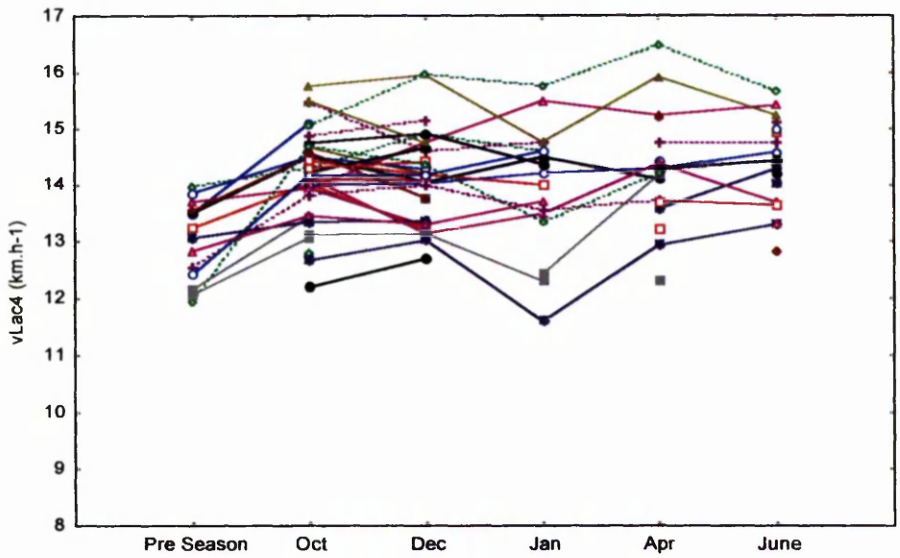

Figure 8 - Case Profile Plot of vLac4 across the testing time-points, showing individual variations over the full soccer season.

There is considerable (and comparable) variability at each testing time-point for both markers and a suggestion of an increase in general from pre-season to October. Boxplots of distributions for both markers with connected medians across time are given in Figures 9 and 10.

There is a strong suggestion of an increase in both the vLT and vLac4 from pre-season to October. When considering the vLT data only, there appears to be a slight suggestion that vLT decreases from its value in December and stays lower until the end of the season (June), but still above the pre-season value.

The vLac4 measurements strongly suggest an increase on average from pre-season to October. There seems no strong suggestion that vLac4 fluctuates markedly during the rest of the season.

In order to investigate if any of these suggested trends were indeed significant, a formal analysis of the mean lactate level over time for the two markers was carried out.

FIGURE 9

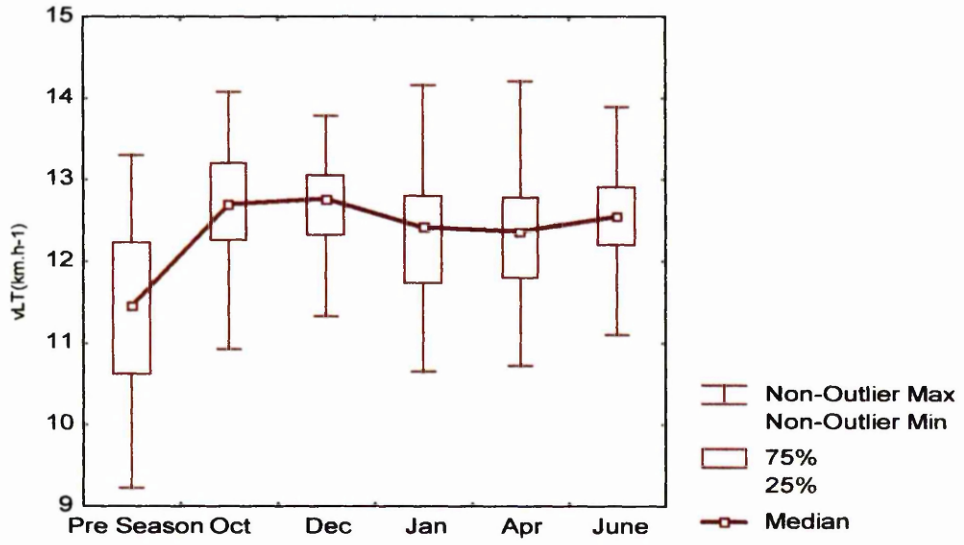

Figure 9 - Boxplot of vLT across each of the testing time-points.

FIGURE 10

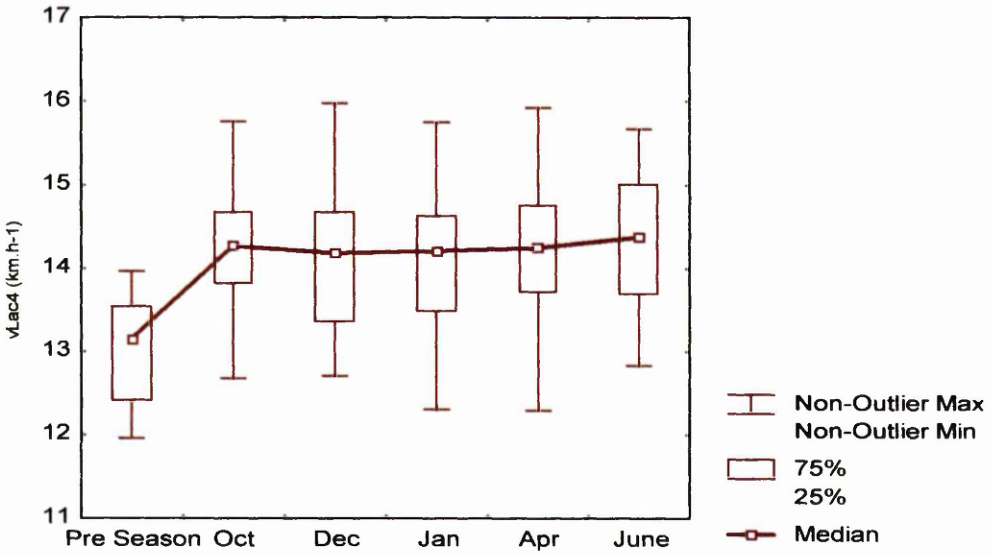

Figure 10 - Boxplot of vLac4 across each of the testing time-points.

(C) FORMAL ANALYSIS

TESTING FOR A PRE-SEASON TRAINING EFFECT

The first analysis undertaken was to test formally for any significant fluctuations in vLT and vLac4 from pre-season values to October. Boxplots of pre-season and October lactate marker scores are given in Figures 11 and 12.

FIGURE 11

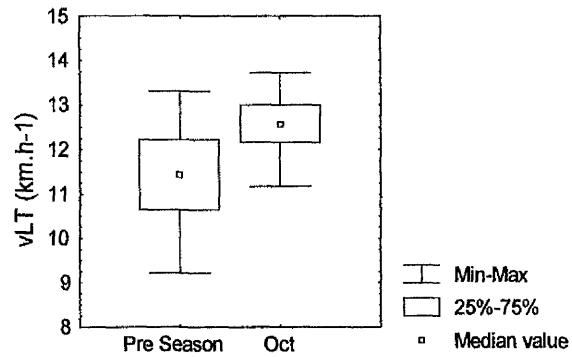

Figure 11 - Boxplot of Pre-season and October vLT. The October vLT is significantly greater than the Pre-season vLT ($p < 0.001$).

Results from a paired t-test showed a significant increase ($p < 0.001$) in mean vLT in October compared to pre-season of 1.12 $km.h^{-1}$ (95% Confidence Interval [0.64, 1.60]).

FIGURE 12

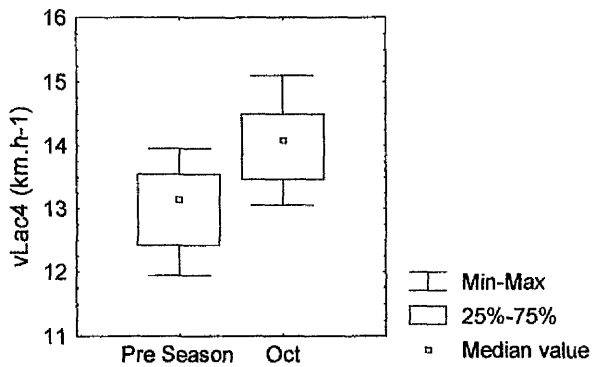

Figure 12 - Boxplot of Pre-season and October vLac4. The October vLac4 is significantly greater than the Pre-season vLac4 ($p < 0.001$).

The results of a paired t-test revealed a significant increase ($p < 0.001$) in mean vLac4 in October compared to pre-season of 1.04 $km.h^{-1}$ (95% Confidence Interval [0.65, 1.43])

Regardless of which lactate marker is used, a highly significant increase in aerobic fitness from pre-season to October was found ($p<0.001$).

ANALYSIS OF AEROBIC FITNESS VALUES FROM OCTOBER TO JUNE

A Repeated Measures Analysis of Variance (ANOVA) was carried out (using a Generalised Linear Model) with a null hypothesis that the mean blood lactate marker values would not change significantly over the course of the playing season. There was one within factor, time, with 5 levels (i.e. October, December, January, April and June). This analysis was carried out separately for both of the aerobic fitness markers.

CHANGES IN vLT DURING THE COMPETITIVE SEASON

The Repeated Measures ANOVA analysis showed that there was a significant difference in mean vLT somewhere across the season ($p= 0.001$). In order to identify where this potential mean difference might have occurred a Bonferroni Multiple Comparisons procedure was carried out. The results from this analysis showed that the mean vLT is significantly higher in December when compared to January and June and when comparing October to June. The corresponding estimated mean vLT difference and 95% confidence interval for actual vLT difference are displayed below in Table 7.

TABLE 7

Repeated Measures ANOVA - Bonferroni Multiple Comparisons Results (vLT)

Time Point Comparison	Estimated Mean Difference ($km.h^{-1}$)	95% Confidence Interval for the actual mean Difference ($km.h^{-1}$)
Dec – Jan	0.36	(0.02, 0.73)
Dec – June	0.39	(0.03, 0.75)
Oct – June	0.39	(0.04, 0.74)

In addition to the Repeated Measures ANOVA, Bonferroni adjusted paired t-tests for all pair-wise time comparisons were carried out. The comparisons where there was a suggestion of a significant difference are displayed in Table 8.

TABLE 8

Bonferroni adjusted paired t-test results on pair-wise comparisons where a significant difference in vLT was suggested

Time Point Comparison	Estimated Mean Difference ($km.h^{-1}$)	95% Confidence Interval for the actual mean Difference ($km.h^{-1}$)
Dec – Jan	0.36	(0.04, 0.69)
Dec – June	0.39	(0.04, 0.79)

Results from the Bonferroni adjusted paired t-tests analysis showed that the mean vLT is significantly higher in December when compared to January and June. No difference in mean vLT in October and June was found as suggested by the Repeated Measures ANOVA.

When the results from both statistical approaches are combined, the overall conclusion was that vLT was highest on average in December, and was significantly lower in January (the lowest competitive season aerobic fitness time-point) and June (end of the season).

A plot of mean vLT across time is given in Figure 13 below where the difference in mean vLT in October and December when compared to the later testing time-points is clear.

FIGURE 13

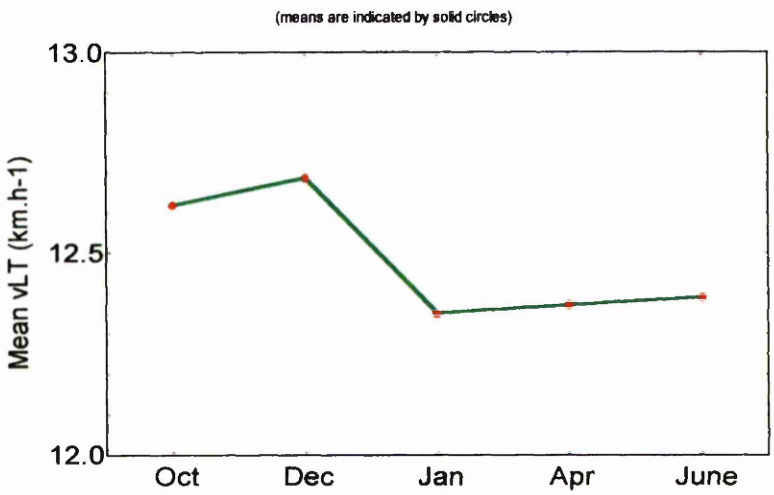

Figure 13 - Plot of mean vLT at each testing time-point during the competitive season (means are indicated by solid circles). Note the lower vLT values after the winter intermission (January, April, and June vLT) when compared to October and December.

CHANGES IN vLAC4 DURING THE COMPETITIVE SEASON

A Repeated Measures ANOVA was carried out (using a Generalised Linear Model) with a null hypothesis that the mean vLac4 would not change over the course of the playing season. Results indicated that there was a possible suggestion of a difference in mean vLac4 across the competitive season time-points ($p = 0.07$). A Bonferroni Multiple Comparisons procedure showed that mean vLac4 was possibly higher on average in October when compared to January (Table 9) only.

TABLE 9

Repeated Measures ANOVA - Bonferroni Multiple Comparisons Results

Time Point Comparison	Estimated Mean Difference ($km.h^{-1}$)	95% Confidence Interval for the actual mean Difference ($km.h^{-1}$)
Oct – Jan	0.38	(-0.03, 0.79)

When Bonferroni adjusted paired t-tests for all pair-wise time comparisons were carried out, no significant mean differences between the testing time-points were found.

Given the results of both analyses, in particular the p-value resulting from the Repeated Measures ANOVA, there was no strong evidence that mean vLac4 changed significantly on average across the competitive season testing time-points.

This is further illustrated in a plot of mean vLac4 across the testing time points (Figure 14).

FIGURE 14

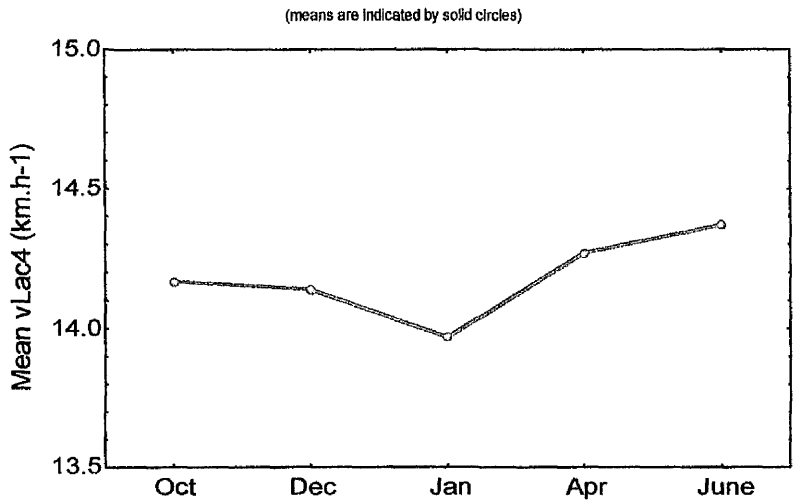

Figure 14 - Plot of mean vLT at each testing time-point during the competitive season (means are indicated by solid circles).

(D) CONCLUSIONS

1. A strong positive correlation between vLT and vLac4 was found, and, when ignoring pre-season data, there is a consistent 'additive' bias where vLac4 tends to be on average 1.5 $mmol.l^{-1}$ greater than vLT.

2. There was a highly significant increase in aerobic fitness from pre-season to October, evidenced by significant increases in both the mean vLT and mean vLac4 values ($p<0.001$).

3. Mean vLT was found to significantly decrease from its highest in-season value in December ($p<0.001$) to its lowest in-season value in January, and at the end of the season (June).

4. Mean vLac4 was not found to change significantly throughout the competitive season.

CHAPTER 5

DISCUSSION

CHAPTER 5 - DISCUSSION

(A) CONCLUSIONS

HYPOTHESIS ONE CONCLUSION

The first hypothesis of this study was that there would be no significant change in aerobic fitness levels from the start of pre-season training (July) to October.

This null hypothesis was rejected as firm evidence was found to show that aerobic fitness levels were higher in October compared to the start of the pre-season. This increase in aerobic fitness levels was evidenced by highly significant changes in mean vLT ($p<0.001$) and mean vLac4 ($p<0.001$).

Therefore, there is strong evidence that there is a highly significant increase in aerobic fitness during the pre-season to October testing time-point period.

HYPOTHESIS TWO CONCLUSION

The second hypothesis of this study was that aerobic fitness levels would not significantly fluctuate throughout the competitive season, because of the aerobic training stimulus of competitive match-play.

It was found that the mean vLT decreased significantly in value from December to January, and from December to June, but no significant changes in mean vLac4 were evident over these two time-periods to compliment the decrease in mean vLT. In fact, the statistical analysis found that there were no significant changes in mean vLac4 across any competitive season testing time-points.

On evidence of the strong relationship found between vLT and vLac4 in this study, it is reasonable to state that, for an aerobic fitness fluctuation between two testing time-points to be truly conclusive, **a significant change in BOTH vLT and vLac4 is required.**

It is concluded that, when applying this "all or none" rule, there is no clear evidence that aerobic fitness fluctuates significantly across any of the competitive season testing time-point periods.

(B) CONCLUSIONS DISCUSSION

AEROBIC FITNESS DURING THE PRE-SEASON – OCTOBER PERIOD

An increase in aerobic fitness, evidenced by a highly significant increase in both the mean vLT and mean vLac4 ($p<0.001$) over the pre-season-October testing time-point was evident in this study, and may be due to the following reasons.

Firstly, the players were returning to pre-season training in a detrained state, due to a summer intermission break of approximately 4 to 5 weeks. Amigo and co-workers (1998) investigated the effects of the summer intermission on 37 amateur adolescent Spanish soccer players who had been training for 11 months prior to the intermission. By taking biopsies of m. Vastus Lateralis before and after the intermission, they found significant decrements in cross-sectional area of ST and FT fibres, and a significant decrement in the activities of creatine kinase, citrate synthase, phosphofructokinase (PFK), lactate dehydrogenase and aspartate aminotransferase.

Secondly, the pre-season training period in Britain traditionally consists mainly of aerobic fitness work, soccer training, and friendly matches (Mercer et al, 1995). As an example, when investigating fitness profiles of English professional soccer players before and after the pre-season period, the training records kept by Mercer and his researchers during this 6-week pre-season period revealed the conditioning emphasis of sessions in weeks 1 to 3 to be 85% aerobic/endurance conditioning and 15% game-related activity. The aerobic conditioning was based almost exclusively on variants of distance running such as aerobic interval-training, hill-running, and cross-country running. Time allocated to aerobic conditioning in weeks 4 to 5 was 60% of the total training time (Mercer et al, 1995). It was expected that the training activities of the

players in this study over the four-week pre-season training period would have been of sufficient frequency, duration, and intensity to increase aerobic fitness levels.

A further increase in aerobic fitness may have evident in this study because of the timing of the second testing time-point (October). Due to the time constraints of the soccer players (see Limitations of the Study below) the proposed scheduling of a testing time-point directly after the pre-season training period was not possible. It can be speculated that further gains in aerobic fitness on top of that gained during the pre-season training period may have been possible due to the high aerobic energy demands of competitive soccer match-play (see Table 2). For example, Rhode and Espersen (1988) found that the HR response for Danish players participating in 1st Division games was below 73% of HR-max for 11% of the total playing time, between 73% and 92% of HR-max for 63% of the total playing time, and greater than 92% HR-max for 26% of the total playing time.

AEROBIC FITNESS THROUGHOUT THE COMPETITIVE SEASON

One of the main conclusions of this study was that aerobic fitness levels of the soccer players tested was not found to change significantly across the competitive season testing time-points. This conclusion was mainly (a) logistical, but may also be due to (b) other factors -

a) Logistical factor - Although significant changes were found in mean vLT throughout the competitive season in this study (over the time periods of December to January, and December to June), the significant changes in mean vLT were not paralleled with similar significant changes in mean vLac4. This led to the conclusion that there was no strong evidence to state that aerobic fitness levels of the soccer players changed significantly throughout the competitive season. For example, a decrease in aerobic fitness from December to January would only be construed as a decrement in aerobic fitness if there was evidence of a significant negative change in **BOTH** mean vLT and mean vLac4.

b) Other factors – There are various other factors that may have contributed to the conclusion of there being no aerobic fitness fluctuation of significance throughout the

competitive season. The soccer training regimes and pattern of regular competitive match-play of the soccer players assessed in this study may have been of sufficient frequency, duration, and intensity to maintain the aerobic fitness increase gained during the pre-season to October period. Another reason is probably due to highly individual changes in mean vLT and mean vLac4 throughout the competitive season. For example, a player whose aerobic fitness may have gradually increased throughout the competitive season (perhaps due to the player partaking in extra aerobic conditioning), may be counteracted by a player whose aerobic fitness decreased gradually throughout the season (perhaps because of a lack of competitive match-play throughout the season). It can be expected that there is highly individualised aerobic fitness gain/loss throughout the competitive season, due to factors such as regularity of competitive match-play (team selection), number of training sessions attended throughout the season, and number of individual training sessions partaken throughout the season. Individual changes in vLT and vLac4 across the season for each player are illustrated in figures 8 and 9. Although results obtained from sub-maximal blood lactate assessment can be used effectively to demonstrate to coaches how their training regimes are affecting **whole squad** aerobic fitness levels during the season (the pre-season to October training effect being an example in this study), the collation of results from each **individual player** of the squad is also of great, if not greater relevance.

WHY A SIGNIFICANT CHANGE IN ONE AEROBIC FITNESS MARKER BUT NOT THE OTHER OVER A SPECIFIC TIME PERIOD?

There may be two main reasons why mean vLT and mean vLac4 were found to not always change in unison and/or change in similar magnitude -

1) Physical and nutritional status of players:

It is possible that the players in this study may have reported for the assessment sessions in differing physical and nutritional states across the testing time-points. Although all attempts were made to standardise all aspects of the testing procedures, it was possible that, due to the frequency of competitive matches and training sessions throughout the

season, some players may have attended testing sessions in a glycogen depleted or fatigued state.

Several researchers have speculated that alterations in substrate availability might affect LT and/or AT (Hughes et al, 1982; Yoshida, 1984; Ivy et al 1981). Hughes et al (1982) compared LT and AT in subjects who were in glycogen depleted and normal glycogen states. The glycogen depleted state was found to cause a dissociation between LT and AT, with LT occurring at a greater work-load and AT occurring at a lesser work-load when compared to a normal glycogen state. Yoshida (1984) observed that a carbohydrate (CHO) rich diet, a low CHO diet, and a mixed diet (3-4 days on each diet) had no effect on LT. However, because the high CHO diet was associated with an increased $[Lac]^b$ at each sub-maximal exercise stage, the work-load and $\dot{V}O_2$ at 4 $mmol.l^{-1}$ were significantly reduced after the high compared to the low CHO diet. Yoshida's study indicates that the use of FBLC's to monitor aerobic fitness levels may be more susceptible to changes in dietary status than when compared to using LT, which is usually estimated by mathematical or visual means. It can be speculated that, because of the sensitivity of FBLC markers to changes in dietary status, vLT might have been the more reproducible and valid marker of aerobic fitness in this study. Bourdon (2000) has emphasised that FBLC's are strongly influenced by an athletes nutritional and training/recovery state, and care must be taken to control for such factors when testing. It should be noted that determination of vLT can be difficult if a "lactate baseline dip" occurs in the early stages of an incremental test. The determination of vLT may be flawed due to the fact that vLT determination occurs at low basal lactate concentrations. In practice, it is certainly a lot easier to describe sub-maximal blood lactate assessment results to players using FBLC's, rather than describing assessment results that are based on vLT.

In an attempt to counteract the glycogen depleting effects of soccer training and match-play (Jacobs et al, 1982) in this study, most of the testing days were scheduled either two days after competitive match-play (with a day of rest prior to the test day), or on days after light training sessions. Jacobs and colleagues (1982) reported that soccer players, who after match-play consumed a less than optimum percentage of carbohydrate (CHO),

took up to three to four days to replenish muscle glycogen stores to their pre-match level. Therefore, it is possible that players in this study who were practising poor nutritional strategies for replenishing glycogen stores prior to assessment might have been tested in a glycogen depleted state, causing a dissociation between vLT and vLac4 (Hughes et al, 1982).

It is also reasonable to assume that when the players reported for testing sessions that were scheduled after an intermission from training/match-play (e.g. pre-season and January in this study), they may have been assessed in a more "standardised" state for sub-maximal blood lactate assessment, being of a non-glycogen depleted/non-fatigued status. This is in comparison to testing time-points that were scheduled between competitive matches (e.g. October, December, April and June), being time periods where players were more susceptible to fatigue and glycogen depletion. Again, this discrepancy in the players nutritional status might have affected the results of this study. Consider the following fictitious example of a player who when tested in December, was of a partially glycogen depleted state because of his participation in three competitive matches over the course of ten days prior to testing. After a two-week winter break, with the player not physically training at all over this period, he attends the laboratory for assessment in a (presumably) non-fatigued/non-glycogen depleted state. Although the player may have lost some of his aerobic fitness over the two-week break, his vLac4 result may look artificially lower in January than that anticipated (and of that recorded in December) due to his vLac4 result being artificially lowered in January (due to his non-glycogen depleted state), but also because of an artificially raised vLac4 result in December (due to muscle glycogen depletion).

It is of interest to note that the highest squad average vLac4 value in this study was found in June, a month in Scottish football where a lot of matches are played in a short space of time because of a build-up of matches caused by previously abandoned matches and perhaps the latter stages of cup competitions. Mean vLac4 may have been found to be the highest value at this time due to glycogen depletion of the players because of a busy fixture list, or because of an actual improvement in squad aerobic fitness levels due to the regular conditioning stimulus of regular competitive match-play.

2) vLT and vLac4 may be detecting different specific training adaptations -

According to Bourdon (2000), changes in specific threshold values may serve as distinct indicators of specific changes in an athletes training status. Increases in the LT (vLT in this study) may reflect an improvement in base aerobic condition, an adaptation that manifests itself as a shift in the blood lactate response curve to the right and/or downwards. This change in the blood lactate response curve is thought to be the result of delaying blood lactate production due to increased fat oxidation and enhanced aerobic mechanisms. Bourdon (2000) also speculates that increases in exercise intensity at AT (vLac4 in this study), represented by a downwards and/or shift to the right of the blood lactate response curve to be indicative of an improvement in "higher-level" aerobic endurance, with possible causes being an improved lactate clearance and acid buffering ability. Since AT intensity is in the approximate intensity range of 89-93% of maximum HR (Bourdon, 2000), being of an intensity that is reached for a significant amount of time during soccer match-play (Strudwick and Reilly, 1999), it is possible that vLac4 may be construed as a more "soccer-specific" blood lactate marker of aerobic fitness. The upward swing in mean vLac4 towards the end of the season, along with the fact that the mean vLac4 was of the highest value in June compared to the other testing time-points, may lend some support to the notion that vLac4 may be a better indicator of "soccer-specific fitness" than vLT. It has been previously reported that blood lactate concentrations of 2-10 $mmol.l^{-1}$ have been reported in professional soccer players during competitive match-play (Ekblom, 1986). An important "soccer specific fitness" adaptation of the soccer player to regular competitive match-play might be an enhanced lactate clearance ability of the involved musculature, which may manifest itself as an increase in vLac4 (or AT).

(C) RESULTS OF THIS STUDY IN RELATION TO SIMILIAR STUDIES

There have been some previous studies, mentioned earlier in Chapter 2 that are similar to the design of this study – they involve the assessment of aerobic fitness in soccer players across time by sub-maximal blood lactate assessment. The following two examples highlight the fact that sub-maximal blood lactate assessment is a useful tool for quantifying changes in squad aerobic fitness levels over a specific time-period.

1) Studies by Bangsbo (1994)

Fourteen elite soccer players were tested before and after a five week pre-season training period where the players trained five times a week and played one match per week (Bangsbo, 1994). Blood lactate and $\dot{V}O_2$ were determined during an incremental protocol on a treadmill. In addition, $\dot{V}O_2$max and peak blood lactate were measured during and after exhaustive exercise, respectively. At the end of the pre-season period, $\dot{V}O_2$max and peak blood lactate were only slightly higher than before the pre-season. However, the blood lactate concentrations during sub-maximal running were significantly lower after the pre-season training, resulting in a 25% higher mean speed at a FBLC of 3 mmol.l^{-1}.

2) Another study by Bangsbo (1994) again indicated the usefulness of sub-maximal blood lactate assessment for the monitoring of aerobic fitness changes following an intense period of soccer training. Bangsbos' study involved quantifying aerobic fitness changes in a Danish soccer team over a seven week **preparation period** for two European Cup quarter-final matches. Twenty players were tested in the middle of January and again in early March just before the first quarter-final match. In addition to an average increase in $\dot{V}O_2$max from 58.6 to 60.3 ml.min^{-1}.kg^{-1} over the preparation period, the mean blood lactate concentration during sub-maximal treadmill running (9 to 13 km.h^{-1}) was found to be significantly lower at all of the treadmill velocities.

The following three studies dealt with sub-maximal blood lactate assessment of soccer players over a longer time period.

3) Study by Jensen and Larsson (1993)

Jensen and Larsson (1993) tested Danish female national soccer players at different sub-maximal treadmill velocities over a period of three years. $\dot{V}O_2$ and [Lac]^b were determined at each running velocity. The running velocity resulting in a FBLC of 3

mmol.l^{-1} was 11.1 km.h^{-1} with a corresponding $\dot{V}O_2$ of 43.1 ml.min^{-1}.kg^{-1}. These values were significantly lower than the corresponding values for elite male soccer players, but the relative work rate at the FBLC of 3 mmol.l^{-1} (82% of $\dot{V}O_2$max) was similar to that of the male players. Over a period of three years, with an increased soccer training volume, the running velocity corresponding to a FBLC of 3 mmol.l^{-1} for 17 of the players increased from 12.1 (range 11.0 – 13.3) to 13.4 (11.5 – 15.6) km.h^{-1}.

4) Study by Brady et al (1995)

Brady and colleagues (1995) assessed the aerobic fitness levels of twenty-four players belonging to an elite Scottish soccer team over the course of two seasons. Players were tested by sub-maximal blood lactate assessment, using an incremental treadmill protocol. Exercise stage duration was 4 minutes, with an exercise intensity increase of 1 km.h^{-1}. The players were tested in June and December 1992; April, June, July and December 1993; and March 1994. The lactate threshold (LT) was determined by linear regression (see Table 10 below). Differences in LT throughout the season were analysed using one-way ANOVA.

TABLE 10 –

Lactate Threshold (LT) running velocity across 2 soccer seasons

[modified from Brady et al (1995)]

Test Date	LT (km.h^{-1})
June '92	13.36
Dec. '92	13.68
April '93	13.36
June '93	12.77
July '93	13.75
Dec. '93	14.14
March '94	13.78

The researchers found the mean LT in December '92, July '93, December '93 and March '94 were significantly higher ($p<0.05$) than in June '93. Brady and his co-workers concluded, in contrast to the results of this study, that the fitness of soccer players fluctuates throughout a season, with fitness levels appearing to deteriorate over the course of the season following the achievement of peak fitness at the end of the pre-season. The finding by Brady of a significant decrease in mean LT at the end of the season (June '93) was not evidenced in this study. The authors suggested that the deterioration of aerobic fitness levels towards the end of the season may have been a consequence of coaches 'scaling down' training in order to save the players' efforts for competition.

5) Study by Dunbar (1999)

In contrast to the conclusions of the four studies discussed above are the results from a study conducted by Dunbar (1999), who found no significant fluctuations in aerobic fitness throughout a soccer season. Dunbar examined the changes in aerobic fitness throughout a soccer season in a squad of elite English professional soccer players using sub-maximal blood lactate assessment. Players were tested in July, August, January and May; which corresponded to the start of pre-season training, early, mid, and end of season, respectively. The running speed at FBLC's of 2 and 3 $mmol.l^{-1}$ was determined by linear extrapolation (Table 11).

TABLE 11

2 and 3 $mmol.l^{-1}$ running velocities (mean + sd) of professional soccer players throughout a season [Modified from Dunbar (1999)]

	July	August	January	May
2 $mmol.l^{-1}$ vel. ($km.h^{-1}$)	14.3 (1.4)	14.5 (1.2)	14.8 (1.5)	13.9 (1.7)
3 $mmol.l^{-1}$ vel. ($km.h^{-1}$)	15.4 (1.2)	15.4 (1.1)	15.7 (1.6)	15.0 (1.5)

Although the running velocity of the FBLC's was of the highest value in January compared to any other times during the season, ANOVA revealed no significant differences for these variables throughout the year ($p<0.05$). Although this is in agreement with the results of this study for aerobic fitness changes throughout the competitive season, Dunbar's study failed to detect any aerobic fitness conditioning effect of the pre-season period. It is noted that in Dunbar's study, out of the thirty-three players who were tested throughout the season, only eleven of the players participated at all four of the testing time-points. The data from these eleven players only were used for the statistical analysis, which is of direct contrast to this study, where all available test data was used in the statistical analysis. Dunbar concluded that the finding of no mean aerobic fitness fluctuations throughout the year was most likely due to individual variations in aerobic fitness throughout the season. The aerobic fitness values reported in Dunbar's study seem extra-ordinarily high compared to the values reported in this study. For example, in Dunbar's study, mean pre-season running velocity at a FBLC of 3 $mmol.l^{-1}$ was found to be 15.4 $km.h^{-1}$, compared to a pre-season vLac4 mean pre-season running velocity of 13.02 $km.h^{-1}$ for this study. Dunbar used a 3-minute exercise stage in comparison to the 4-minute exercise stage used in this study. Since exercise stage duration has been reported as one factor that can affect the blood lactate response to incremental exercise (Weltman, 1995), this difference in exercise stage duration may partly explain the high FBLC running velocities reported by Dunbar. A number of recent studies have indicated that exercise stage duration of at least 5-7 minutes may be required to attain steady-state blood lactate concentrations, allowing accurate determination of blood lactate markers (Foxdal et al, 1996; Foxdal et al, 1994; Heck et al, 1985; LaFontaine et al, 1981; Oyono-Enguelle et al, 1990; Rieu et al, 1989; Stegmann and Kindermann, 1982).

(D) LIMITATIONS OF THE STUDY

There were various limitations of the present study, which may have had an influence on the test data. Most of the limitations were due to the fact that the subjects in this study were professional, full-time soccer players.

TEST FAMILIARISATION

Although all players in this study were familiar with treadmill running prior to this study, the pre-season testing time-point (or October) was the first time that they had exercised on a Woodway treadmill. For many of the players, it was also their first visit to an exercise laboratory, therefore anxiety might have affected the test results. Every effort was made to familiarise the subject during their warm-up prior to their first assessment, especially the blood lactate sampling procedure. A separate familiarisation session with all of the players before commencement of the study was impossible due to the time constraints of the players. It is possible that a lack of familiarisation may have helped add to the significance of the increase in mean vLT and mean vLac4 between the pre-season and October testing time-points.

TEST STANDARDISATION

All attempts were made to standardise the testing as much as possible. Due to time constraints, it was in-evitable that some players were tested $\pm$ 1 hour outwith their specified testing time across the different testing time-points.

NO POST PRE-SEASON TRAINING TESTING TIME-POINT

Before the study commenced, it was proposed that the players would be assessed at the start and end of the pre-season training period. However, the proposed testing time-point directly after the pre-season training period (August) was not possible due to a busy fixture list of youth team matches. Although it might have been possible to test all the players, or even a limited number of players, the youth team coaches involved felt that available time was better spent on preparation for the forthcoming matches. Test data directly after the pre-season training period would have been a welcome addition to this study so that the effectiveness of the pre-season training period **alone** on raising aerobic fitness could have been elucidated. A testing time-point in August would also have served as a "true" start of competitive season marker rather than the October testing date that was used. A pair-wise comparison between the proposed post pre-season training testing time-point (August) and October would have supplied useful information to

investigate the notion that competitive match-play does indeed add to the expected increase in aerobic fitness levels gained throughout the pre-season training period.

LACK OF TRAINING RECORDS

It was beyond the scope of this study to record in detail each player's training schedule throughout the season. This information would be useful for coaches to help them assess the effectiveness of their training for increasing/maintaining aerobic fitness levels.

COLLATION OF THE TEST DATA

Test data were collected from players of Glasgow Celtics Under-18's and Under 21's squads. It should be noted that these two squads train separately, which would result in the players of one squad being subjected to a different seasonal soccer training/match-play routine than the other. The data were analysed as a whole group because some of the players of the Under 18's squad were required to train and play with the Under 21's squad periodically.

MISSING TEST DATA

Missing data were assumed to be at random in this study. Appropriate statistical tests (detailed in the methods section) were selected to best deal with this problem.

REPRODUCIBILITY OF vLT AND vLAC4 IN SOCCER PLAYERS

This study would have benefited from the addition of a reproducibility study of vLT and vLac4 using professional soccer players as subjects. This was impossible due to the time constraints and lack of availability of professional soccer players. To the author's knowledge there have been no studies conducted on the reproducibility of blood lactate markers with soccer players. Since vLT and vLac4 have been reported to be highly reproducible markers of aerobic fitness when testing endurance-trained subjects on a treadmill ergometer (Weltman et al, 1989; Pfitzinger and Freedson, 1998), it is assumed that the protocol used in this study was highly reproducible. Unpublished data from a

reproducibility study of vLT and vLac4 from the University of Glasgow exercise laboratory (involving the author of this thesis and also using the same equipment and incremental protocol as this study) using moderately trained males for subjects indicated a high reproducibility ($r = 0.86$ for vLT, and $r = 0.93$ for vLac4). Importantly, results from this study indicated that the more "aerobically fit" the subject, the higher the reproducibility of vLT and vLac4 (see Appendix 2).

DETERMINATION OF THE vLT INFLECTION POINT

For the purposes of this study, vLT was estimated through use of a specially designed Minitab macro. The vLT (corresponding to the first inflection point on the lactate curve) was estimated from the point where two regression lines crossed each other. The two regression lines were estimated as the best fits of the data points. This method of estimating the vLT was selected because it is objective, therefore avoiding the possibility of experimenter bias (Weltman et al, 1995). However, the application of this computer programme may have been limited due to the following assumptions:

(a) The Minitab macro assumes that the early blood lactate values of the assessment are of a similar magnitude, hence falling roughly in a straight line. However, this is not always the case. Some players exhibited a gradual increase in blood lactate levels during the early stages of the lactate profile, while some exhibited a dip in blood lactate levels during the early exercise stages.

(b) Once lactate levels begin to rise above the vLT, the Minitab macro fits a regression line through the rising data-points, which tend to increase in a curvi-linear fashion.

These two limitations may produce a distortion of the estimation of vLT. Log-log plotting of blood lactate concentration against running velocity (i.e. making the lactate curve less curvi-linear) may make the fitting of regression lines by the Minitab macro more appropriate. This application of using log-log transformation of the data to aid determination of lactate threshold has also been previously advocated by Beaver (1985).

(E) THE PRACTICALITY OF SUB-MAXIMAL BLOOD LACTATE ASSESSMENT IN PROFESSIONAL SOCCER PLAYERS

There are a number of positive and negative aspects associated with using sub-maximal blood lactate assessment to assess the aerobic fitness levels of professional soccer players -

PROS

- The results of this study, as well as those from similar previous studies (Bangsbo et al, 1994; Brady et al, 1995; Dunbar et al, 1999; Jensen and Larsson, 1993) indicate that sub-maximal blood lactate assessment of soccer players can be used as sensitive indicators of change in aerobic fitness over a specified time-period, therefore providing soccer coaches with useful information on the efficacy of their training regimes.

- Individual results from sub-maximal blood lactate assessment can be used for designing **individualised** training programmes to increase (or maintain) a player's aerobic fitness (Weltman, 1995).

- Sub-maximal blood lactate assessment serves as a good educational tool for educating soccer players (and coaches) on the importance of aerobic fitness in professional soccer. The time of assessment also proves to be a useful time for educating players on the merits of using a HR monitor.

- Results from sub-maximal blood lactate assessment can serve as a motivational tool for players to help them increase (or maintain) their aerobic fitness level.

- Regular sub-maximal blood lactate assessment can prove useful during the monitoring of soccer players undergoing a period of injury rehabilitation. This important application of sub-maximal blood lactate assessment in professional soccer is demonstrated in more detail later in this discussion.

CONS

Although there are a lot of good reasons to advocate the use of sub-maximal blood lactate testing for the assessment of aerobic fitness in soccer players, there are also certain drawbacks –

- Soccer is a high-intensity, intermittent team sport (Bangsbo, 1994), and is multi-directional in nature. However, the blood lactate testing procedure used in this study was conducted on a treadmill and was therefore only uni-directional. Therefore, some specific aerobic fitness adaptations that arise from soccer-specific activities may not be fully detected by sub-maximal blood lactate assessment. Muscle fibres that are recruited during soccer-specific movements such as turning and decelerating will not be recruited to the same magnitude during treadmill running. FT fibres recruited preferentially during intermittent, explosive bouts of exercise will probably not be recruited during the sub-maximal blood lactate assessment. Sub-maximal blood lactate assessment performed on a treadmill may not be specific enough for detecting aerobic fitness changes in FT muscle fibres that are routinely recruited during soccer match-play.

- Sub-maximal blood lactate assessment usually consists of an incremental test with exercise periods of 3-5 minutes (Weltman, 1995), and evaluates mainly continuous (or cyclical) endurance performance. The sensitivity of sub-maximal blood lactate testing may be limited when trying to detect changes in aerobic fitness resulting from high-intensity, intermittent, multi-directional exercise.

- Poor face validity - with sub-maximal blood lactate assessment being traditionally uni-directional, non-intermittent in nature, and generally carried out indoors on a treadmill (or other exercise ergometer), the assessment of aerobic fitness by this method may seen alien and of little relevance to the professional soccer player.

- Sub-maximal blood lactate assessment is time consuming, with only one player being tested at a time (if only one Exercise Physiologist available). A sub-maximal blood lactate test may last for approximately 24 to 36 minutes. Therefore, assessment of a whole professional soccer squad (20 – 30 players) requires a considerable amount of available time. The soccer coach may decide that time allocated for assessment purposes would be better spent on other aspects, such as technical training, or preparing for a forthcoming game.

- Sub-maximal lactate testing is expensive, requiring expensive equipment such as a blood lactate analyzer and pricey consumables (blood lactate reagent being an example when using the Analox GM7). The cost of hiring qualified members of staff, such as an Exercise Physiologist to perform the testing also adds to the expense. However, the cost of sub-maximal blood lactate assessments is easily within the budget of Scottish and English Premier clubs.

- For the obtainment of "true" sub-maximal blood lactate assessment results, it is of vital importance that the test conditions are standardised as much as possible. Due to the congested fixture list of Scottish football, it may prove difficult to schedule sub-maximal blood lactate testing of a soccer squad during the competitive season, with the players being assessed in a suitable physiological state.

- Due to the scientific nature of the testing, and also because of the previous lack of sports science support work in most soccer clubs (Bangsbo, 1994), some players (and coaches) may not fully understand the benefits of blood lactate assessment, and may be confused by the test results. Time is required to educate the players and coaches to facilitate their understanding of sub-maximal blood lactate assessment (see Appendix 3). It is important to educate the player's on the benefits of fitness assessment at an early an age as possible/relevant.

(F) SUB-MAXIMAL BLOOD LACTATE ASSESSMENT DURING INJURY REHABILITATION OF SOCCER PLAYERS

In addition to the benefits of sub-maximal blood lactate assessment discussed previously, sub-maximal blood lactate assessment can also prove to be a very useful aid when monitoring the aerobic fitness of individual players, such as injured soccer players during their rehabilitation (see Appendix 3). The example shown below (in Figure 15 and Table 12) illustrates how a player's aerobic fitness level can be monitored effectively throughout the rehabilitation period. Data in this example were taken from an Under-18 youth team player who was forced to withdraw from this study because of knee ligament injury two weeks after the October testing time-point. Knowledge of the player's vLT and vLac4 before injury provided some helpful information for the physiotherapists when deciding when it was appropriate for the player to resume full soccer training.

FIGURE 15

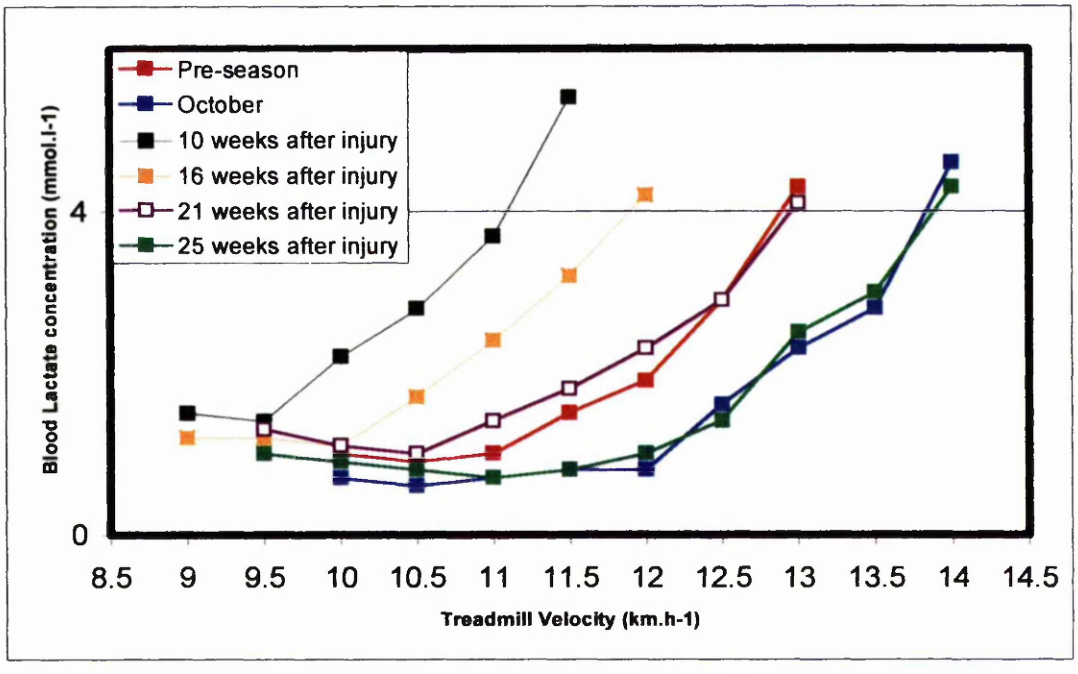

Figure 15 - Sub-maximal blood lactate assessment is an excellent tool for monitoring aerobic fitness changes across the rehabilitation period. This example shows a players aerobic fitness fluctuations when recovering from significant knee ligament injury. It can be seen that 25 weeks after injury, vLac4 has returned to a pre-injury Lac4 level (October).

TABLE 12

Rehabilitation example – changes in vLT and vLac4

Testing Time-point	vLT ($km.h^{-1}$)	vLac4 ($km.h^{-1}$)
Pre-season	11.2	12.8
October	12.1	13.8
10 weeks post injury	9.6	11.0
16 weeks post injury	10	11.8
21 weeks post injury	11.2	13.0
25 weeks post injury	12.2	13.9

(G) RECOMMENDATIONS FOR FUTURE RESEARCH

The following points are recommended for future research projects dealing with the application of sub-maximal blood lactate assessment in professional soccer–

1) All efforts should be made to ensure that all players being assessed have had at least one comprehensive familiarisation session.
2) Each player should be not be tested $\pm$ 1 hour outwith their specified testing time across each of the testing time-points.
3) All players should be educated before assessment on the importance of standardising their nutritional and physical state prior to each testing bout.
4) A large emphasis should be made to test as many players as possible at each testing time-point, so that the statistical power of the study is increased.
5) If possible, all testing procedures should take place at a site which is of easy access for the soccer players, e.g. at the training ground.
6) It is of benefit to schedule a testing time-point immediately before and after a specific preparation period to assess aerobic fitness changes over this period.

7) Testing time-points scheduled before and after intermission periods (such as summer and winter intermissions) would help to quantify the detraining effect during these times.
8) A reproducibility study using elite male soccer players for subjects would provide invaluable information for quantifying individual changes in vLT and vLac4. For example, application of Bland and Altman's "Limits of Agreement" (Atkinson and Nevill, 1998) statistical procedure would deduce what magnitude of change in vLT or vLac4 is indicative of a "true" aerobic fitness change, i.e. a change that is outwith the change associated with day-to-day variability.
9) If time permits, collection of max data would provide additional useful information on the aerobic fitness levels of soccer players. Collection of fitness assessment data measuring other components of fitness that are of importance to the professional soccer player other than aerobic fitness, such as muscle strength, flexibility, sprinting ability and jumping ability (Reilly, 1996) would provide even more useful information, and provide data to investigate seasonal fluctuations in these additional fitness parameters. It would seem important to also assess "anaerobic recovery" fitness since soccer is a high-intensity **intermittent** sport. An anaerobic recovery test could consist of a repeated sprint test or a repeat Wingate cycle test for example.
10) Detailed training records that classify the type of training, and its frequency, duration, and intensity, as well as a record of competitive matches played should be collected throughout the season. Analysis of these training records can be used to explain any findings of fitness fluctuations across a specific time-period, and may also be used to improve future training plans. For example, Mercer et al (1995) concluded in their study when investigating the effects of the pre-season training period on the fitness profiles of an English professional soccer squad, that no training time during the pre-season period was allocated to strength development, indicating that the players may have carried potential deficiencies in leg strength performance into the competitive season. Training records that monitor resting heart-rate in the morning could also prove useful in highlighting possible sub-clinical illness, although monitoring every player each morning over time would be extremely time-consuming.

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APPENDICES

APPENDIX 1 –

PRE-TESTING MEDICAL QUESTIONAIRRE

UNIVERSITY OF GLASGOW
INSTITUTE OF BIOMEDICAL AND LIFE SCIENCES

SUBJECT'S QUESTIONNAIRE AND ASSENT FORM FOR HIGH-INTENSITY EXERCISE TESTING

If you feel unwell on the day of a proposed test, or have been feeling poorly over the preceding day or two, DO NOT TAKE PART in a high intensity exercise test
The considerations which follow apply to people who are feeling well at the time.

NAME .....

SEX ..... AGE ..... (yrs) HEIGHT .....(m) WEIGHT .....(kg)

Exercise lifestyle
a) What kind(s) of exercise do you regularly do (20+ min/session)? (Please circle)

	Number of times per average week				
Walking	1	2	3	4	5
Running	1	2	3	4	5
Cycling	1	2	3	4	5
Swimming	1	2	3	4	5
Skiing	1	2	3	4	5
Rowing	1	2	3	4	5
Gymnastics	1	2	3	4	5
Martial Arts	1	2	3	4	5
Tune Up	1	2	3	4	5
Popmobility	1	2	3	4	5
Sweat Session	1	2	3	4	5
Weight Training	1	2	3	4	5
Field Athletics	1	2	3	4	5
Racket Sports	1	2	3	4	5
Rugby/soccer/hockey	1	2	3	4	5
Others*	1	2	3	4	5

"Specify .....

b) How long have you been exercising at least twice/week for at least 20 min/session?

.....

Smoking (Please tick one)

Never smoked .....
Not for > 6 months .....
Smoke < 10 per day .....
Smoke> 10 per day .....

Illnesses
Have you ever had .....? (Please circle Yes or No)

Asthma	NO/YES
Diabetes	NO/YES
Epilepsy	NO/YES
Heart Disease	NO/YES
High Blood Pressure	NO/YES

Any other illness that could affect your safety in performing maximal exercise NO/YES"

"(Please specify) .....

Symptoms

Have you ever had any of the following symptoms to a significant degree?
i.e. have you had to consult a physician relating to any of the following?

Breathlessness	NO/YES
Chest Pain	NO/YES
Dizzy Fits/Fainting	NO/YES
Heart Murmurs	NO/YES
Palpitations	NO/YES
Anaemia	NO/YES

Muscle or joint injury

Do you have/or have had any muscle or joint injury which could affect your safety in performing maximal exercise or strength testing or strength training?

NO/YES*

*(Please specify) .....

Medication
Are you currently taking any medication? NO/YES*

*(Please specify) .....

Signature .....

Date .....

APPENDIX 2 –

SUB-MAXIMAL BLOOD LACTATE ASSESSMENT REPRODUCIBILITY

ABSTRACT

(Submitted for BASES conference Book of Abstracts, Newport, September 2001)

Reproducibility of the lactate threshold, 4mmol.l^{-1} marker, heart rate and ratings of perceived exertion during incremental treadmill exercise.

There are only limited data on the reproducibility of blood lactate variables, heart rates and ratings of perceived exertion (RPE) during sub-maximal exercise (Weltman, 1995, *The blood lactate response to exercise*. Human Kinetics, Illinois). The aim of this study was to investigate the reproducibility of speed at lactate threshold (vLT), speed at a blood lactate concentration of 4mmol.l^{-1}(vLac4), and heart rate (HR) and RPE (at vLT and vLac4), during a continuous, incremental treadmill test. Possible differences in reproducibility related to fitness levels were also investigated.

Twenty males (mean $\pm$ s) {20.5 $\pm$ 1.4 years} and sixteen females {21.2 $\pm$ 0.9 years} gave consent to participate in the study which received ethical approval. Subjects performed a familiarisation run and two identical incremental tests (tests were one week apart). At the end of each 4-minute stage, the subjects provided a RPE, HR was recorded and a blood lactate sample was taken. After the blood sample was taken, the speed of the treadmill was increased by 0.5 km.h^{-1}. Subjects with a vLT of <10.5 km. h^{-1} were arbitrarily categorised as "unfit" and those with a vLT of >10.5 km. h^{-1} were classified as "moderately fit". Using Bland and Altman's Limits of Agreement, correlation coefficients and 95% confidence intervals for the mean difference between tests, the present study investigated the level of agreement and reproducibility of vLT and vLac4, and HR and RPE at vLT and vLac4.

For the group as a whole, the test-retest correlation coefficient for vLT was r = 0.88, and r = 0.92 for the vLac4. At vLT, the correlation coefficients for the "moderately fit" and "unfit" groups were r = 0.94 and r = 0.36 respectively and at vLac4, r = 0.93 and r = 0.68, for the "moderately fit" and "unfit" groups respectively. There was evidence of a systematic bias for the unfit group v-Tlac, with test 2 being on average 0.29 km. h^{-1} higher than test 1 (95% confidence interval (C.I.) [0.06, 0.51]). There was no evidence of a bias for the "moderately fit" group where the width of the Limits of Agreement indicated that a change of 1.62 $km.h^{-1}$ in vLT is necessary to be considered a change in training status. For the "unfit" group there was a systematic bias of 0.33 km. h^{-1} (95% C.I. [0.01, 0.67]) for vLac4. The width of the Limits of Agreement for the 'moderately fit group' indicated a change of 1.34 $km.h^{-1}$ in vLac4 is necessary to be considered a change in training status. For all subjects, test-retest correlation coefficients for HR at the vLT were 0.77 and 0.81 for HR at the vLac4. For HR at the vLT, the 95% C.I. for the population mean bias is completely negative and indicates a significantly lower mean HR between the two tests where HR's at test 2 are likely to be between 0.3 $beats.min^{-1}$ to 5.8 $beats.min^{-1}$ lower than the corresponding HR's measured at test 1. There is no systematic reduction in HR at vLac4 from test 1 to 2. For all subjects, correlation coefficients for RPE at vLT and vLac4 were 0.69 and 0.76 respectively. Limits of agreement for RPE at vLT and vLac4 were 3.05 and 2.8 Borg scale units respectively (no significant bias).

While the correlations between tests 1 and 2 are generally good, Limits of Agreement suggest that changes in scores must be fairly large before they can be deemed to be outside the range of day-to-day variability. These findings cast doubt on the sensitivity to change of blood lactate testing, heart rate and RPE in this population.

APPENDIX 3 -

THE ROLE OF SUB-MAXIMAL BLOOD LACTATE TESTING IN THE MONITORING OF AEROBIC FITNESS OF SOCCER PLAYERS

(Soccer coach/player information on the relevance of blood lactate testing in soccer)

Published in FA Insight magazine, Spring 2001

The role of sub-maximal blood lactate testing in the monitoring of aerobic fitness of soccer players.

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A number of fitness components have been identified as being important for the soccer player including aerobic fitness, anaerobic fitness components (such as jumping ability and acceleration), strength and flexibility (Reilly, 1996). The importance of aerobic fitness in soccer is highlighted by the fact that elite players cover around 10 –12 km during a game at an average intensity of around 75% of their maximal oxygen uptake ($\dot{V}O_2max$) and that the aerobic system contributes around 90% of the total energy cost of the game (Bangsbo, 1994). In addition, high aerobic fitness has the potential to optimise performance by enhancing recovery during the game and contributing to the ability to sustain quality endurance runs.

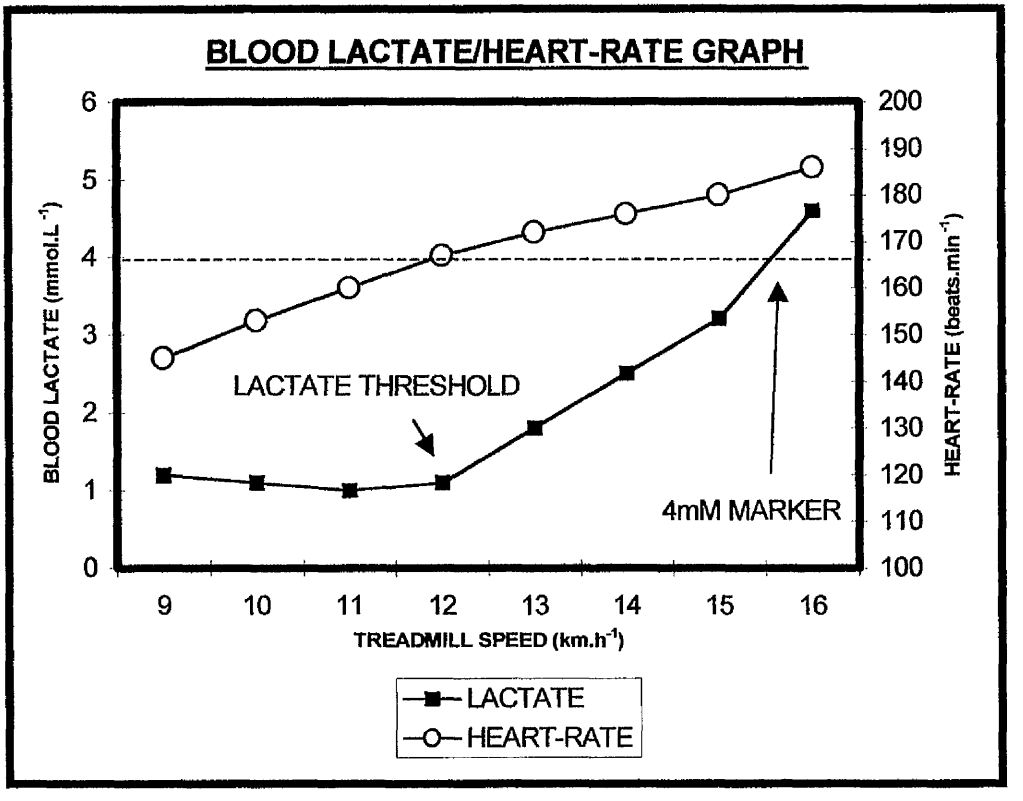
While a high maximal oxygen uptake ($\dot{V}O_2max$) has been considered to be important for endurance running success, sub-maximal blood lactate variables have been shown to be better predictors of endurance running performance than $\dot{V}O_2max$ (Weltman, 1995). It has been shown that $\dot{V}O_2$ max may not change despite an improvement in endurance performance. The changes in the skeletal muscles after training are associated with adaptations in the skeletal muscles, resulting in sub-maximal blood lactate variables being sensitive barometers of training status. Therefore, it is not surprising that sub-maximal blood lactate tests are a regular feature of the fitness test battery at Celtic Football Club.

Blood lactate is measured during an incremental test during which small drops of blood are obtained from the player and analysed almost immediately for lactate content. The test begins at an easy level for the player (at approximately 60% of maximum heart rate {HR}) and has gradual progressions to high exercise intensities (at approximately 90-95% maximum HR). Plotting of the results on a graph (Figure 1) shows that the blood

lactate levels remain fairly constant until a breakpoint is reached. This breakpoint is called the lactate threshold and is described as the first significant elevation of blood lactate above resting levels. The lactate threshold is important as it is considered to reflect the interaction of the aerobic and anaerobic energy systems. Testers wish to identify the speed at which the lactate threshold is reached and may also wish to determine the speed at which a specific blood lactate level is attained. For example, the speed at a blood lactate level of 4 mmol l^{-1} is often measured as it has been shown to be a very good predictor of endurance performance.

FIGURE 1.

BLOOD LACTATE/HEART-RATE GRAPH

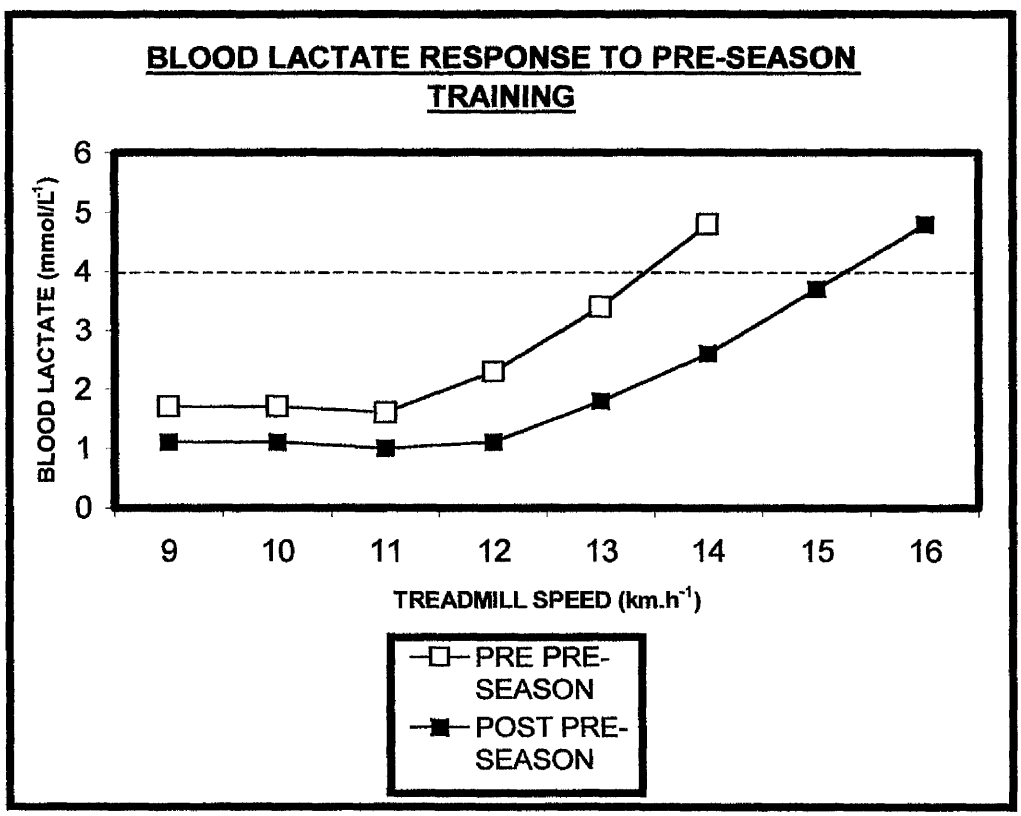


It is advantageous to be able to carry out sustained running at high speeds without a marked increase in lactate, as a build-up of lactate within the muscles will contribute to fatigue. The effect of aerobic training is to delay the accumulation of lactate until higher speeds have been attained. Thus after training the speed at lactate threshold and at a

blood lactate level of 4 mmol l^{-1} is higher, which indicates that the player is more aerobically fit. The effects of soccer training (or rehabilitation) can be assessed by comparing the changes in speeds at lactate threshold and 4 mmol l^{-1} (Figure 2). In addition, a marked reduction in HR at a given speed is a typical response to a period of soccer training.

FIGURE 2.

PRE TO POST PRE-SEASON CHANGE IN BLOOD LACTATE RESPONSE

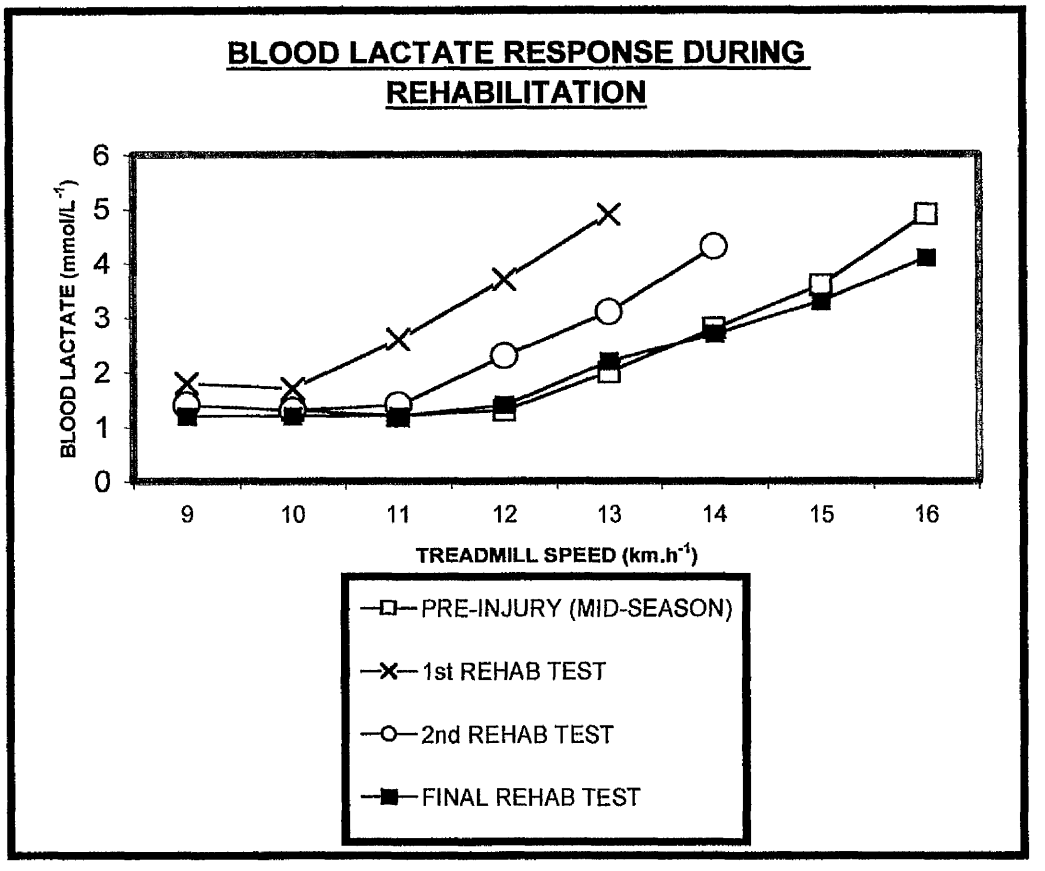


There are some limitations associated with sub-maximal blood lactate testing. It is time consuming and relatively expensive to carry out. However, lactate testing of soccer players at strategic times throughout the season will provide information on the effectiveness of the club training programmes. Test results provide the coach with objective evidence on the endurance status of the players and identify players who are in need of improvement. In addition, the use of blood lactate testing has proved to be a very useful aid for the monitoring of progress during rehabilitation of injured players

(Figure 3), as well as a useful tool for the design of specific running drills. Knowledge of what the player was capable of before injury provides some guidance on when it is appropriate for the player to resume playing.

FIGURE 3

BLOOD LACTATE TESTING IN REHABILITATION OF SOCCER PLAYERS



In summary, despite the limitations of blood lactate testing, blood lactate variables (lactate threshold or a fixed lactate concentration) are powerful predictors of a soccer player's endurance capability. These variables are sensitive indicators of seasonal fluctuations in aerobic fitness and of the detraining effect/training effect during rehabilitation programmes. These are good reasons for a soccer test battery to include blood lactate testing on a regular basis if time and money are available.