Effect of acclimation on performance and metabolism of female hockey players during intermittent running in the heat

by

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Abstract

The impact of heat acclimation on performance of high intensity intermittent exercise, that is representative of team sports has not been extensively studied. The performance of skills during team sports is also important in the outcome of a match or competition. The research presented in this thesis was undertaken to investigate the impact of heat acclimation on female performance of high intensity intermittent running and field hockey skill.

The first experimental study investigated the effect of the menstrual cycle and oral contraceptive use on performance of high intensity intermittent running in the heat. Seven normally menstruating women and 8 oral contraceptive users participated in the study. Two trials were undertaken near the predicted mid-point of the follicular (FT) and luteal (LT) phases of the menstrual cycle and the equivalent days for the oral contraceptive users. There were no differences in distance run between menstrual cycle phases or between the normally menstruating and oral contraceptive groups (follicular vs luteal phase; 5869 ± 2896 vs 6238 ± 2648 m). Plasma glucose concentrations were higher in the follicular phase than the luteal, (main effect phase $P<0.01$), while serum growth hormone concentrations were lower (main effect phase $P<0.05$). These results demonstrate that for unacclimatised games players the performance of intermittent, high intensity shuttle running in the heat is unaffected by menstrual cycle phase nor oral contraceptive use.

In the second experimental study the impact of 4 short heat acclimation sessions (30 – 45 min of the LIST) on high intensity intermittent running performance (LIST), in terms of distance run, was examined. Three groups were used, an acclimation group (30°C), a moderate training group (18°C) and a control group who did not complete any training between the main trials. The 4 acclimation or moderate training sessions were completed in a 10 day period prior to the post-acclimation trial. Exercise capacity was increased by 33% in the acclimation group (group x trial interaction $P<0.05$), but was unchanged in the moderate and control groups. The acclimation group had a lower rectal temperature (group x trial x time interaction $P<0.01$) and an increase in thermal comfort after acclimation (interaction group x trial $P<0.01$). Thus a lowering of deep body temperature and concomitant rise in thermal comfort may be responsible for the performance improvement.

The aim of the third study was to design a field hockey skill test that was both statistically reliable and valid for the use in the remaining research. The reliability and validity of the field hockey skill test was assessed in 20 men and 19 women. The mean difference ± limits of agreement was 0.03 ± 5.11s. Validity was determined by Spearman Rank correlations between coaches' subjective rankings and overall skill performance; values were 0.63 for the men and 0.85 for the women. It was concluded that the field hockey skill test was a reliable, valid and objective tool for the measure of specific field hockey skills.

In the fourth experimental study 9, well-trained, unacclimatised female hockey players performed the LIST interspersed with 3 field hockey skill tests in hot (30°C) and moderate (19°C) environmental conditions. Field hockey skill performance declined following 30 and 60 min of the LIST compared with pre-LIST (main effect time $P<0.01$). This decrement in performance was compounded in the hot

environment with a 6% poorer performance in the heat recorded for the 2nd skill test (main effect trial $P<0.05$, hot vs moderate, 101.7 ± 3.6 s vs 95.7 ± 2.9 s, interaction trial-time, $P<0.05$). However, no difference was found in the decision-making element of the skill test. In the hot environment, rectal temperatures (main effect trial $P<0.01$), perceived exertion (main effect trial $P<0.05$) perceived thirst (main effect trial, $P<0.01$) and blood glucose concentrations (main effect trial $P<0.05$) were higher. Fifteen metre sprint times were slower in the hot condition (main effect trial $P<0.01$). Estimated sweat rate was greater in the hot trial ($P<0.05$); however, body mass was well maintained in both trials. No difference in serum aldosterone and cortisol, lactate, plasma volume and plasma ammonia concentrations were found between the hot and moderate conditions. These results demonstrate that field hockey skill performance is decreased following intermittent, high intensity shuttle running and that this decrease is greater in hot environmental conditions. The exact mechanism for this decrement in performance remains to be elucidated, but is unlikely to be due to low glycogen concentration or dehydration as skill declined after only 30 min.

The final experimental study brought the previous studies together by examining the effect of 4 short heat acclimation sessions on high intensity intermittent running and field hockey skill performance. Eight well-trained unacclimatised, female, field hockey players completed 3 main trials. A main trial was completed before and after heat acclimation and a third main trial, which was randomly assigned, was completed as a control trial in which the 3 skills test were completed in the heat with passive recovery, rather than intermittent running between each skill test. Following 30 and 60 min of intermittent running skill performance declined prior to acclimation, but was maintained following acclimation and during the control trial (main effect trial $P<0.01$). Following heat acclimation, perceived thermal comfort was increased (main effect trial $P<0.01$). The results clearly show that field hockey skill performance, following high intensity intermittent running is improved after 4 short high intensity intermittent acclimation sessions. The mechanism for this improvement requires further investigation, but thermal comfort appears to be important for skill performance in the heat.

Heat acclimation, in the form of 4 short high intensity intermittent running sessions, improves exercise capacity and attenuates the decrement in field hockey skill performance observed in unacclimatised female hockey players. Following acclimation, deep body temperature is reduced and thermal comfort enhanced.

Acknowledgements

Preface

Some of the results of the studies presented in this thesis have been published as follows:

Sunderland, C. and Nevill, M.E. (2001). The effect of the menstrual cycle on performance of intermittent high-intensity shuttle running in a hot environment. J Sports Sci **19**: 63-64.

Sunderland, C., Nevill, M. E., Cooke, K., Lakomy, H. K. A., Milne, H. and Pout, M. (2000). The effect of intermittent, high intensity shuttle running and hot environmental conditions on field hockey skill performance. Proceedings of the 5th Annual Congress of the European College of Sport Science, Finland.

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1. Introduction

Women first participated in the Olympics in 1924, but only over short running distances, due to a concern that physiologically they would be unable to compete in endurance events. Until the 1960s, women were still prohibited from competing in any event over 800 m. By 1984 however, increasing physiological research and thus awareness, meant that after years of campaigning, women were finally allowed to compete in an Olympic marathon. In line with the development of female running in the Olympics and the mass media coverage accompanying the Games, over the last three decades there has been a rapid increase in female participation in recreation and sport. For example, women's soccer is the fastest growing sport in terms of participation at the current time. However, the research relating to women and sport, particularly with respect to participation in the 'games' based activities, is still limited. One of the major reasons for this lack of information on women is the reluctance of researchers to use female athletes as subjects due to the perceived need to control for phase of the menstrual cycle.

Many of the major championships for team sports are played in climates exceeding 30°C; the Olympic Games in Atlanta (1996), the Commonwealth Games in Kuala Lumpa (1998) and the World Club Championship for football in Rio de Janeiro (2000). Manchester United's poor performance in Rio de Janeiro was well publicised throughout the world's media, who had several explanations for the disappointing performance of Britain's best team. However, there is no doubt that the temperature and humidity will have affected the intensity of exercise that the players could maintain and thus affect the overall team play and performance. In an article entitled "Ferguson urges caution in heat of the moment," Winter (2000) outlined the difficulties facing Manchester United:

> "Manchester United will tonight need to curb their normal non-stop marauding inclinations in a concrete furnace called the Maracana. This is no high-speed Premiership collision in ideal conditions, it is a test of whether the champions of England and Europe know how to pace themselves in 35°C heat against opponents used to dwelling on the ball before the right run is made."

Without any prior heat acclimation, Sir Alex Ferguson (2000) had concerns about the match and adopted different tactics to the game,

> "The weather could be really sticky and it might make us have to change our game a bit. We have to concentrate on keeping possession and conserving energy."

Similarly, the England men's field hockey team arrived at the Shultan Azlan Shah Tournament (March, 1998) in Malaysia (35°C, 80% RH) without any prior heat acclimation,

> "The Australians, coming out of a hot summer of their own, have acclimatised by switching off the air conditioning in their hotel rooms. England, who arrived five days before the tournament, reckon they are just about coping now," (Middleton, 1998).

Nine to 12 consecutive days of heat acclimation (40-42°C) has been shown to improve cycling capacity (60% $\dot{V}O_2$ max) by 67% (Nielsen et al., 1993). Similarly, Pandolf and Young (1992) showed that following 7 days heat acclimation (49°C, 20% RH), 23 out of 24 men completed 100 min of walking (3.5 mph), whereas prior to acclimation none of them were able to finish (76 min was the greatest individual time). These studies required the subjects to exercise to exhaustion each day and they clearly demonstrate that exercise heat tolerance can be improved dramatically in less than 2 weeks. However, prior to competitions, it would not be appropriate for athletes to exercise to exhaustion on consecutive days. Like the research of Nielsen et al. (1993) and Pandolf and Young (1992) most research into acclimation has employed prolonged submaximal exercise for the acclimation protocol. However, games and similar intermittent exercise are characterised by variable speed running, bursts of maximal effort and walking (Williams, 1990) and have been shown to provide a greater thermal strain than continuous exercise at the same average power output (Kraning and Gonzalez, 1991; Nevill et al., 1995). Therefore, using intermittent running as the mode of exercise for acclimation and for the pre- and post-acclimation tests may result in different performance and metabolic responses to those reported in

the literature for submaximal continuous exercise. To date, only one study has examined the effect of heat acclimation on the performance of high intensity intermittent running, in a hot environment (35°C, 60% RH; Dawson and Pyke, 1990). The acclimation period required male hockey players to complete 14 days of normal training in moderate conditions (11-19°C) wearing sweat clothing. The responses of the acclimation group were compared with a group who had completed the same training sessions in light clothing (field hockey shirt, shorts and socks). Following acclimation, the responses to the high intensity intermittent treadmill run were similar between the acclimation and control groups. However, this research is questionable in its design, as the thermal responses (T_{rec}, ΔT_{rec}) during interval treadmill running wearing the sweat suit, were lower than the same running in shorts and t-shirt in a hot environment (35°C, 60% RH; Dawson and Pyke, 1988). Thus, the sweat suit may not have placed the acclimation group under any greater thermal strain than the light clothing group.

The key mechanisms underlying heat acclimation are a reduced cardiovascular and thermoregulatory strain (Wenger, 1988). The degree and rate of acclimation are related to the extent that repeated heat exposures raise deep body temperature above a critical temperature and the length of time above that temperature (Wenger, 1988; Armstrong and Maresh, 1991). The cardiovascular adaptations take place during the first few days of acclimation and are manifested by a decrease in heart rate. Several mechanisms have been proposed for alleviating cardiovascular strain include an increase in plasma volume and a decrease in skin blood flow (Mitchell et al., 1976; Fortney et al., 1979; Wenger, 1988). Thermoregulatory adaptations to heat acclimation occur centrally and peripherally. Deep body temperature is reduced and sweat sensitivity and sweat rate are increased (Wenger, 1988; Nielsen, 1994). While the adaptations to acclimation will vary with duration, intensity and training status, acclimation generally makes individuals more comfortable and increases the capacity for work (Wenger, 1988).

There is also a dearth of information into the acclimation responses of well-trained athletes and specifically female athletes. Alongside 4 men, Armstrong et al. (1987) studied 1 female athlete and Avellini et al. (1980) recruited 4 female athletes to

investigate the effect of heat acclimation on treadmill running or walking respectively. Only Avellini et al. (1980), therefore, were able to look at the responses to acclimation of female athletes specifically. Following acclimation (walking 5.6 km.h⁻¹; 10 days, 36°C, 65% RH), the women had a lower heart rate and rectal temperature at 90 min of the 3 h walk.

Thus overall, there is a dearth of information regarding the effect of acclimation on performance and on the physiological and metabolic responses to intermittent high intensity exercise, particularly for well-trained female athletes. Furthermore, there is no information on the effect of exercise in the heat on performance of hockey skills or on the effect of acclimation. Finally, the mechanism of adaptation to heat exposure is not fully understood. Therefore, the purpose of this research was to investigate the impact of heat acclimation on female performance of high intensity intermittent shuttle running. Two further aims were to devise a suitable acclimation protocol for implementation by teams prior to the onset of tournaments and to assess how skill performance in field hockey may be affected by intermittent running in the heat and heat acclimation.

The thesis is presented in 8 main Chapters:
- Chapter 2 presents a review of literature examining the physiological and metabolic consequences of high intensity intermittent exercise in the heat and heat acclimation. The physiological and performance responses of women (including the effect of oral contraceptives) during the menstrual cycle are discussed. The limited literature on hockey and games physiology and skill performance is also reviewed.
- In Chapter 3 the general procedures, equipment and methods of analysis used during the experimental studies are presented.
- In Chapter 4 the effect of the menstrual cycle and oral contraceptive use on performance of high intensity intermittent running in the heat are examined.
- Chapter 5 investigates the impact of 4 short heat acclimation sessions on high intensity intermittent running performance in terms of distance run.
- The design, reliability and validity of a field hockey skill test are examined in Chapter 6.

- In Chapter 7 the effect of hot and moderate environmental conditions and high intensity intermittent running on field hockey skill performance are examined.

- Chapter 8 brings the previous studies together by examining the effect of 4 short heat acclimation sessions on high intensity intermittent running and field hockey skill performance.

- The final Chapter (9) of the thesis summarises the findings from the experimental work and addresses some of the questions on which future research should focus.

2. Review of Literature

2.1. Introduction

This chapter collates the most pertinent findings from studies on the female athlete, intermittent exercise, heat acclimation and performance of motor skills. The review of literature has been divided into 5 main sections. The first section (2.2) examines the physiological demands of intermittent exercise. Section 2.3 reviews the literature on the menstrual cycle and its effect on exercise performance in eumennorheic and oral contraceptive women. Section 2.4 reviews appropriate research on heat acclimation. Field hockey physiology and skill tests are presented in section 2.5 and the possible mechanisms for decline in motor skill performance and cognition are reviewed in section 2.6.

2.2. Intermittent exercise

Activities such as field hockey, football and rugby, require participants to perform a pattern of activity that involves variable speed running, including bursts of maximal effort, and walking (Williams, 1990). Such an exercise pattern is not easy to model in a research environment, because in most actual games the physical demands are dictated by the competitive situation, which will vary from match to match. Nonetheless, using various forms of analysis (for example, video-recordings, film analysis, computer aided video analysis [Reilly, 1997]) it is possible to analyse, and reach some conclusions, regarding the physical demands of prolonged, intermittent, high-intensity exercise.

2.2.1. The characteristics of games type exercise

Much of the data investigating the total distance covered by games players in the course of a match has been produced from studies on competitors playing soccer. Outfield players have been estimated to cover anywhere from 8680 m (Reilly and Thomas, 1976), to between 10 and 11 km (Bangsbo et al.,1991; Bangsbo, 1994; Van Gool et al., 1988), up to 12 km (Reilly, 1997). The variation in these estimates

probably stems from differences in methodologies, and critically, the difficulties involved in accurately appraising the movement of players. In their assessment of the distances covered by female soccer players, Davies and Brewer (1993) concluded that the demands on females during a game were similar to those on males. With respect to other games, the distances covered by participants vary from the 11.5 km on average reported for Australian rules footballers in the course of a match (Withers et al., 1982), to the 5.61 km reported for field hockey players (Reilly and Borrie, 1992). However, this latter figure is based on data from the 1973 World Cup and field hockey has undergone enormous changes with respect to playing surfaces and playing styles since then. Similarly, rugby in the 21st Century is not the same game it was even toward the end of the 20th, but recorded data suggest players cover between 4.8 and 9.6 km (Reilly, 1990). Nonetheless, despite the variation between research studies in their estimates of the distances covered by players, it would not seem unreasonable to expect many games players to be performing something like 10 km of prolonged, intermittent high-intensity exercise in the course of a competitive encounter.

The playing position of an individual also seems to affect the distances that will be covered in a game, with 'midfielders' covering the greatest amounts (Reilly and Thomas, 1976; Van Gool et al., 1988). In addition, players cover a greater distance in the first half of a soccer game compared with the second (Reilly and Thomas, 1976; Van Gool et al., 1988).

In a soccer game, sprinting or faster running has been reported to account for between ~8 (Van Gool et al., 1988; Bangsbo, 1994) to ~11 % of the total distance covered (Reilly and Thomas, 1976; Mayhew and Wenger, 1985). All out sprints of ~2 s in duration and 15 m in length may be performed 19 times in a game (Bangsbo, 1994), and even up to 62 times (Reilly and Thomas, 1976). For Australian Rules the average sprint distance has been assessed at 22.4 m, with 18.8% of a game spent striding or sprinting (Withers et al., 1982), while in rugby up to 2000 m may be performed at a 'high-intensity' (McLean, 1992). It was noted that during the course of a field hockey or soccer match a sprint or high intensity run takes place every 30 s (Nevill, 1994; Reilly, 1997).

While the discussion above gives some idea of the physical pattern of activity in games such as field hockey, rugby and soccer, it should be remembered that players may also be required to turn, jump, tackle and perform skills such as dribbling, passing and shooting (Bangsbo, 1994). Such activities are an implicit part of all games and are likely to increase the physical demands on the individuals playing them.

2.2.2. The physiological demands of games or similar exercise activities

The majority of a soccer game is likely to be dependent on aerobic sources of energy supply (88-90%), with perhaps 12% relying on anaerobic systems (Mayhew and Wenger, 1985; Bangsbo, 1994). Using heart rate, it is possible to estimate that players work at ~70 to 75% of $\dot{V}O_2$ max during a game (Van Gool et al., 1988, Bangsbo, 1994; Reilly, 1997). However, blood lactate concentrations of almost 10 mmol.l^{-1} have been reported, although values do range from 3-9 mmol.l^{-1} in soccer players (Bangsbo et al., 1991; Davis and Brewer, 1993; Bangsbo, 1994), to 5.8-9.8 mmol.l^{-1} in rugby players (Reilly, 1990). With regard to the thermoregulatory strain imposed by games exercise, even in moderate environmental conditions, post-match rectal temperatures in rugby (39.4°C, Reilly, 1990) and soccer (>39.0°C, Bangsbo, 1994) players have been found to be above 39°C. Due to the prolonged, intermittent, high-intensity nature of games, endogenous carbohydrate stores are also likely to be heavily relied upon. Biopsy data obtained from soccer matches have shown significant reductions in the content of glycogen in the muscle of players as a result of a game (57%; Jacobs et al., 1982).

2.2.3. Research into the physiological demands of intermittent exercise

While it is not unusual for athletes to perform exercise of a relatively constant pace, most activities in the industrial, home and recreational environment are intermittent in nature: that is they intersperse bursts of physical effort with pauses (Astrand and Rodahl, 1986).

2.2.3.1.Early investigations

The early investigations in this area noted that the exercise capacity of individuals performing work of a set intensity on an intermittent basis could be considerably greater than when the same workload was performed on a continuous basis. For example, Astrand (1960) found that a subject cycling at 350 W in an intermittent 30 s work: 30 s rest format could exercise for an hour with only moderate exertion. In comparison the same workload performed continuously produced exhaustion after 9 min. The impression gained from such research, might be that the physiological demands associated with an intermittent pattern of exercise are lower than when the exercise is continuous. However, one needs to be aware of the importance of the work: rest ratio. Consequently in the same experiment when the subject cycled at 350 W in a 3 min work: rest format, the exercise could be continued for an hour only with great difficulty. Similarly, Margaria and colleagues (1969) found that during running on an inclined treadmill at a speed sufficient to cause exhaustion in 30-40 s, a work: rest ratio of 10: 30 could be continued almost indefinitely. When the rest periods were shortened to 20 and 10 s, subjects became exhausted after ~2.3 and ~1.6 min respectively.

Drawing conclusions from such investigations about the differences in the physiological demands between continuous and games exercise is also problematic because in the latter the sprints are all-out efforts. Thus, there may be substantial differences in not just the pattern of activity, but in its intensity. Karlsson and colleagues (1967) [op cit. Astrand and Rodahl, 1986] showed the importance of exercise intensity when they found that reducing the movement speed on a treadmill by 0.75 km.h^{-1}, when performing a 20 s run: 10 s rest exercise pattern, increased the exercise time from 25 to 60 min.

2.2.3.2.Maximal intermittent exercise

Following on from these early investigations researchers recognised that power outputs during maximal sprinting could be 2.5 to 3.5 times greater than those required

to elicit $\dot{V}O_2$ max (Wootton and Williams, 1983). Consequently, a number of studies

investigated the demands of maximal sprints interspersed with lower intensity activity or rest.

Wootton and Williams (1983) investigated the performance and physiological responses of 16 male subjects to five 6 s cycle ergometer sprints separated by either 30 or 60 s of passive recovery. When the recovery period was 60 s the decrease in peak power output (PPO) from the first to the fifth sprint was 3%; when the period was 30 s the performance decrement increased to 17.9%. Higher blood lactate concentrations in the shorter recovery trial suggested an increased reliance on glycolysis during the sprints and perhaps an acidosis, which may have impacted on ATP production or utilisation and reduced contractile force. The increased reliance on glycolysis may have also been a function of poorer PCr resynthesis when the rest periods were limited to 30 s. Similar responses have been shown for individuals performing ten 6 s maximal efforts on a sprint treadmill (Holmyard et al., 1988). Again the longer recovery period (60 s) resulted in a much smaller decrement in PPO from the first sprint to the last (3.0%), than the decrease (13.2%) associated with the shorter recovery period (30 s). Clearly, even for sprints of short duration, the length of the recovery interval can have substantial impacts on performance. Also, the work described above suggests that the rate of PCr resynthesis is an important physiological parameter in short sprints of this type.

The duration of an all-out sprint can also impact on performance. Balsom and co-workers (1992) found individuals could perform forty 15 m sprints with 30 s recovery essentially without any change in speed. However, 40 m sprints with the same recovery produced increases in plasma concentrations of hypoxanthine and uric acid, which were taken as markers of significant adenine nucleotide degradation. In addition, Gaitanos and colleagues (1993) found that 30 s was probably sufficient to allow significant PCr resynthesis during ten 6 s sprints on a cycle ergometer. Contrary to the assertions of Wootton and Williams (1983), the decrements in power output seemed to be associated with reductions in the rate of ATP production from anaerobic glycolysis. The fact that during the last sprint muscle lactate concentrations did not change suggested that the energy supporting exercise was mainly derived from PCr degradation and aerobic metabolism.

2.2.3.3.Intermittent exercise in the heat

The early comparisons of intermittent exercise with continuous exercise requiring the same energy expenditure suggested that the physiological responses, as assessed by rectal temperature, pulse rate and weight loss, were not different across a range of temperatures from 29.4 to 41.1°C (Lind, 1963). Again however, the intermittent exercise (walking at 3.5 mph up a 7.5% grade on a treadmill for 25 min interspersed with 35 min rest) was not maximal, even though it was maintained for 8 h.

More recent studies suggest that intermittent exercise performed under heat stress produces considerably more strain on the body than equivalent continuous exercise. Kraning and Gonzalez (1991) found that when evaporative heat loss was restricted during 2 h of either continuous or intermittent exercise in 30°C, rectal temperature responses were 0.4°C higher at 60 min in the intermittent trial, and endurance time was significantly less, compared to the continuous trial. In a recent study, where the intermittent exercise included all-out maximal sprints, it was concluded that intermittent exercise provided a greater thermal strain than continuous exercise (Nevill et al., 1995). Subjects performed 15 bouts each (90 s) of cycling at 40% of $\dot{V}$ O_2 max, a maximal sprint lasting ~6 s and ~15 s of passive recovery, in two temperature conditions (hot 35°C and cool 10°C) compared with 30 min of continuous exercise of the same total power output. It was found that rectal temperature, mean heart rates and blood lactate concentrations were greater in the hotter conditions than in the cooler, and were greater during intermittent exercise than continuous.

2.2.3.4.Prolonged, intermittent, high-intensity running: the LIST

Recently a different exercise model has been used to investigate the impact of various interventions on games type exercise. This model (LIST; Nicholas et al., 1995, 2000) has been used in our laboratory to investigate the impact of hot environmental conditions on prolonged, intermittent, high-intensity running. This exercise model requires subjects to sprint, jog, cruise, rest and turn during indoor shuttle running exercise.

Morris and colleagues (1998a, 1998b, 1999, 2000) using a modification of the LIST, found that hot environmental conditions (~30°C) reduced the distance run in both male and female subjects by between 21 and 49%. This reduction in exercise capacity occurred even though subjects were allowed to drink water ad libitum. In terms of repeated 15 m sprint performance, research does suggest that hot environmental conditions alone (Morris et al., 2000) or in combination with water restriction (Morris, 1998b) can be sufficient to bring about decrements in 15 m sprint performance. Interestingly, isokinetic knee flexion and extension peak force did not seem to be affected by exercising in hot environmental conditions (Morris et al., 1999).

2.2.4. What causes the earlier onset of exhaustion when prolonged, intermittent, high-intensity exercise is performed in hot environmental conditions?

In the studies mentioned above a very strong relationship (r>0.9) was always found between the distance subjects were able to run and the rate of rise in body temperature, as indicated by rectal temperature (Morris et al., 1998a, 1998b, 1999, 2000). In addition, exhaustion in the hot trials invariably coincided with a high deep body temperature (~39.5°C). Consequently, elevated deep body temperature would seem to be a key factor in the poorer exercise capacity elicited by hot environmental conditions. However, precisely how the elevated thermal stress produces these decrements is uncertain.

Exercising in the heat clearly produces an elevated cardiovascular strain. However, direct measurements of cardiac output and leg blood flow in humans exercising in hot environmental conditions do not suggest that muscle blood flow is compromised (Nielsen et al., 1990; Nielsen et al., 1993; Nielsen et al., 1997). Indeed even when cardiac output and muscle blood flow are reduced due to the imposition of dehydration and hyperthermia, muscle oxygen uptake is not compromised (Gonzalez-Alonso et al., 1998). Therefore, in most circumstances elevated cardiovascular strain would not seem to explain the poorer performance of prolonged, intermittent, high-intensity running.

Nicholas et al. (1995) showed that ingesting a carbohydrate-electrolyte beverage (~6.9%) during prolonged, intermittent, high-intensity running in moderate

environmental conditions increased running capacity by 33%. Such a finding emphasises the importance of endogenous carbohydrate stores and exogenous carbohydrate supply to the ability to perform prolonged, intermittent, high-intensity running. Combined with the fact that many studies have demonstrated an increase in muscle glycogen utilisation when the environmental conditions are hot (~40°C), one might argue that inadequate carbohydrate supply could explain why distance run is shorter in individuals performing prolonged, intermittent, high-intensity running in hot environmental conditions (Fink et al., 1975; Febbraio et al., 1994a; Febbraio et al., 1994b). Indeed, a number of studies have found that endurance performance in hot environmental conditions has been improved by drinking a carbohydrate solution (5-7%; Murray et al., 1987; Davis et al., 1988; Millard-Stafford et al., 1992). However, a carbohydrate-electrolyte drink (6.9%) had no effect on the capacity to perform prolonged, intermittent, high-intensity running in hot environmental conditions, and a number of studies have shown that at the termination of exercise in hot environmental conditions, glycogen concentrations are significantly higher than those seen at exhaustion when the same exercise is performed in moderate environmental conditions (Morris et al., 1998c; Morris et al., 1999; Parkin et al., 1999). Therefore, glycogen depletion or inadequate carbohydrate supply would not seem to explain why distance run, and in some circumstances repeated sprint performance, is poorer when prolonged, intermittent, high-intensity running takes place in hot environmental conditions,

It has been suggested that some factor associated with a high deep body temperature per se may explain the curtailment in capacity and performance usually seen in hot environmental conditions (Nielsen et al., 1990; Nielsen et al., 1993). Usually this is explained as 'a reduced drive to exercise'. Precisely how this occurs has yet to be clearly elucidated. However, a recent study found that elevations in deep body temperature initiated concomitant shifts in frontal brain cortical activity, suggesting that alterations in the electrical activity of the brain do occur as a result of exercise induced hyperthermia (Nielsen et al., 2001).

The muscle temperatures at the end of a bout of prolonged, intermittent, high-intensity running in hot environmental conditions are significantly higher than those produced by the same exercise performed in moderate conditions (Morris et al., 1999). It has

been suggested that high muscle temperatures could curtail skeletal muscle function by altering the structural and functional characteristics of proteins involved in activities such as the release and recovery of calcium in the sarcoplasmic reticulum, myosin and actin interactions and the mitochondrial respiratory chain (Hargreaves and Febbraio, 1998). There is also some suggestion that elevated muscle temperature may be associated with an elevated oxidative stress and damage: many mechanisms within the musculature may well be sensitive to changes in oxidative stress. However, such suggestions have still to be examined experimentally.

Clearly therefore, there are various mechanisms which may explain why hot environmental conditions reduce an individuals ability to perform prolonged, intermittent, high-intensity running.

2.3. Women

Despite the rapid increase in women's participation in exercise over the last thirty years, few studies have examined the physiological and metabolic responses of women to exercise, and particularly intermittent exercise in the heat. One of the reasons for this lack of information is the unwillingness of researchers to examine, and control for, any effects of the menstrual cycle on exercise performance and metabolism.

2.3.1. Menstrual cycle

2.3.1.1. Introduction

Numerous definitions exist in relation to the length of a normal menstrual cycle though 28 days is usually reported to be an average value. A normal menstrual cycle was defined by Shangold (1988) as lasting approximately 28 days and ranges from 23 to 35 days. The length varies both between women and between cycles (Vander et al., 1990). Day 1 of the menstrual cycle occurs at the start of menses (Figure 2.1).

FOLLICULAR PHASE			LUTEAL PHASE	

Bleeding starts

DAY	1	7	14	25	28

OVARIAN EVENTS	Multiple follicles develop	Dominant follicle matures	Corpus luteum is functioning	Corpus luteum degenerates

Dominant follicle selected   Ovulation occurs
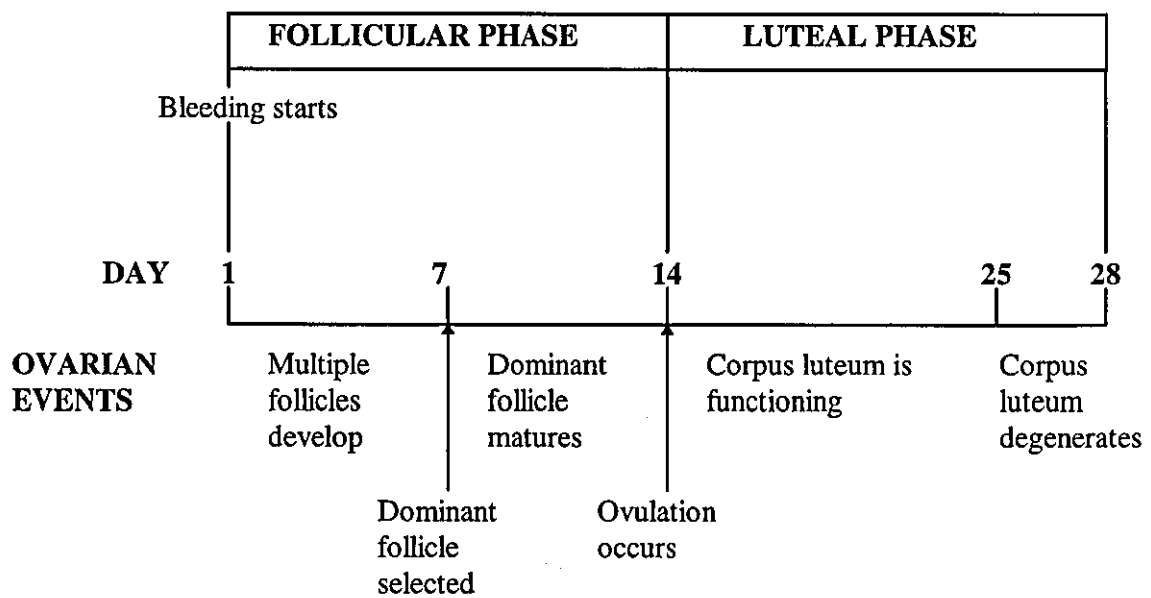
Figure 2.1 A diagrammatic representation of the ovarian events during a normal (28 day) menstrual cycle.

Hormonal fluctuations during the menstrual cycle may have an impact on a female's exercise response and performance. The hormonal events that dictate the ovarian events are controlled by the hypothalamus, which secretes many hormones including gonadotropin-releasing hormone (GnRH). GnRH controls the synthesis, storage, releasing ability and secretion of both pituitary gonadotropins, namely follicle stimulating hormone (FSH) and luteinising hormone (LH). Follicle stimulating hormone, as its name suggests, advances the growth of the follicle and promotes the synthesis of estrogen from androgen precursors. Luteinising hormone activates ovarian androgen production, maintaining a supply of androgens available for conversion to estrogens. Estrogen levels are low during the early follicular phase and high during the late follicular phase; it is this high level that triggers ovulation. During the luteal phase both estrogen and progesterone levels are high (Shangold, 1988). The high levels of progesterone have been associated with an elevated deep body temperature during the luteal phase of the menstrual cycle and this may have implications upon exercise thermoregulation. Thermoregulation may also be adjusted by changes in fluid retention during the menstrual cycle. Fluid retention is promoted during pre-menses, when progesterone concentrations are decreased, stimulating aldosterone secretion (Reilly, 2000).

2.3.1.2. Performance and physiological responses to exercise during the phases of the menstrual cycle in moderate conditions

The higher deep body temperature in the luteal phase of the cycle may have implications for the thermoregulatory responses of the body during this phase, and thus affect exercise performance. A higher deep body temperature threshold for the onset of heat loss mechanisms during exercise in the luteal phase has been well documented (Stephenson and Kolka, 1988a). The responses of women to exercise in moderate conditions during the phases of the menstrual cycle are presented in Table 2.1.

The investigations into performance and responses of women to exercise in moderate conditions during the menstrual cycle are equivocal. For example performance during the luteal phase has been reported to be enhanced (Jurkowski et al., 1981; Reilly and Whitley, 1994), unaltered (Schoene et al., 1981; Stephenson et al., 1982; Dombovy et al., 1987; De Souza et al., 1990; Lebrun et al., 1995) and attenuated compared with the follicular phase (Schoene et al., 1981; Williams and Krachenbuhl, 1997). An increase in exercise capacity during the luteal phase, when estrogen and progesterone concentrations are high, may be due to decreased glycogen utilisation (Reilly and Whitley, 1994 [see Table 2.3]; [Figure 2.2). This assertion is supported by a lower lactate concentration (Jurkowski et al., 1981) and lower respiratory exchange ratio during the luteal phase (RER; Reilly and Whitley, 1994). However, blood lactate and glucose concentrations were unaltered by menstrual phase during the treadmill running to exhaustion presented by Reilly and Whitley (1994), a finding in agreement with DeSouza et al. (1990) during similar exercise. While only a limited amount of research has been completed into the effect of menstrual cycle phases on substrate utilisation during endurance exercise, exercise intensity seems to be critical (Dombovy et al., 1987; Zderic et al., 2001). Zderic et al. (2001) compared cycling for 25 min at 42% $\dot{V}O_2$ max and then 25 min at 52% $\dot{V}O_2$ max during the follicular and luteal phases. At the lower exercise intensity, there were no discernable differences in substrate utilisation, however at 52% $\dot{V}O_2$ max, total carbohydrate oxidation was 13% lower and fat oxidation 23% higher in the luteal phase. Similarly, Dombovy et al. (1987; Table 2.1) reported a lower RER value during the luteal phase at exercise

intensities of 67, 75 and 100% $\dot{V}O_2$ max, whereas no apparent difference in substrate

utilisation was reported at 33 and 50% $\dot{V}O_2$ max. The mechanism for the suppression of carbohydrate metabolism remains to be elucidated but attenuation in gluconogenesis or hepatic glycogenolysis as a consequence of high estrogen concentrations during the luteal phase has been suggested (Zderic et al., 2001). In contrast to the research previously presented (Dombovy et al., 1987; Reilly and Whitley, 1994; Zderic et al., 2001), Bailey et al. (2000) reported that there were no differences between the follicular and luteal phases in RER or exercise time to

exhaustion when cycling at 70% $\dot{V}O_2$ max further complicating the picture.
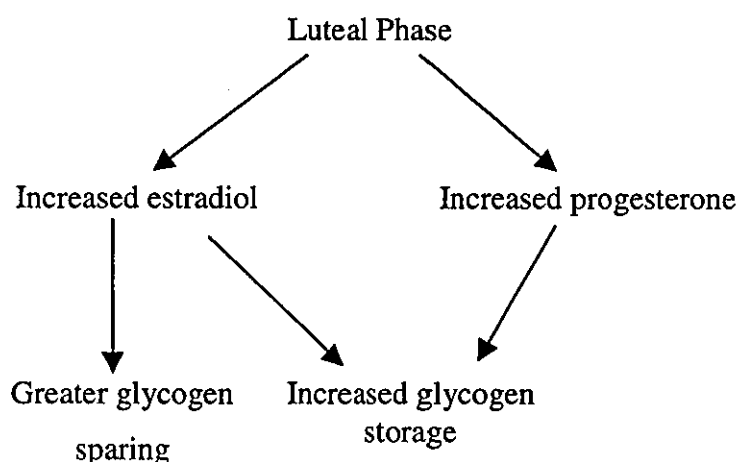
Figure 2.2 Effects of estrogen and progesterone on carbohydrate metabolism; (Jurkowski et al., 1981).

When performance was unaffected by menstrual phase the majority of studies have employed trained or well-trained women (Schoene et al., 1981; Stephenson et al., 1982; De Souza et al., 1990; Lebrun et al., 1995) suggesting a high training status may suppress any difference in performance due to menstrual cycle phase. Such a suggestion in supported by Schoene et al. (1981) who demonstrated a decrease in cycling time to exhaustion in untrained women, which was not observed in a trained group. However, this assertion is not supported by the investigation of Reilly and Whitley (1994) who employed female cross-country runners. The runners completed

approximately 50 km.week^{-1} and had a $\dot{V}O_2$ max of 56.4 ± 7.5 ml.kg^{-1}.min^{-1}. The time

to exhaustion at 70% $\dot{V}O_2$ max was 62.1 ± 7.4 and 51.8 ± 7.2 min during the luteal

and follicular phases, respectively. From the information presented, it was not

possible to ascertain whether the female runners were allowed to drink, the absence of

which would have reduced endurance time. Nonetheless, based on research in our

laboratory and others, the total exercise time is comparatively low (Fallowfield et al.,

1996; Tsintzas et al., 1996a, 1996b; Chryssanthopoulous and Williams, 1997). Using

a mixed group of male (n = 4) and female (n = 4) moderately trained subjects ($\dot{V}O_2$

max: 51.1 ± 1.8 ml.kg^{-1}.min^{-1}), the time to exhaustion was 77.7 ± 7.7 and 103.0 ± 12.4

min without and with fluid replacement, respectively. The discrepancy in exercise

capacity between these studies (Reilly and Whitley, 1994; Fallowfield et al., 1996;

Tsintzas et al., 1996a, 1996b; Chryssanthopoulous and Williams, 1997) may be due to

either the training status of the women employed by Reilly and Whitley (1994) and/or

the exercise intensity. This highlights the need for establishment of an accepted

criteria for training status and thus what is meant by trained, moderately trained and

well-trained as this appears to be clearly important in exercise performance of women

during the menstrual cycle.

Only one study has been completed investigating intermittent exercise and menstrual

cycle phase (Lynch and Nimmo, 1998). Ten recreationally active individuals

completed an intermittent protocol in moderate conditions that entailed repeated 20 s

sprints, separated by 100 s passive recovery until exhaustion. No difference in

exercise performance between phases was found as represented by total exercise time.

However, the starting speed of the treadmill was 14.3 km.h^{-1}, at a gradient of 10.5%

and increased progressively by 1.2 km.h^{-1}. This is an extremely fast speed, up a

difficult gradient, and therefore it is possible that the treadmill speed became too fast

for the subjects before they were exhausted.

Table 2.1 The responses of women, and the effects of the menstrual cycle, during exercise in moderate conditions

Study	Environmental conditions	Subjects	Exercise protocol	Cardiovascular responses	Metabolic responses	Thermoregulatory responses	Performance
Williams and Krahenbuhl (1997)	21-23°C 50-60%	8 moderately trained	Treadmill running 6 min 55% VO_2 max 6 min 80% VO_2 max	Higher VE luteal than early follicular 55% VO_2 max VO_2 unaffected 80% VO_2 max VO_2 higher luteal			Running economy unaffected at 55% VO_2 max Economy poorer luteal than follicular 80% VO_2 max
Lebrun et al., (1995)		16 well-trained	Treadmill running 90% VO_2 max & 8 mph 20% incline	No difference VO_2, VO_2 max, HR, RER, ventilation			No difference exercise time or performance
Pivarnik et al., (1992)	22°C 60%	8 recreational acclimatised	60 min cycling 65% VO_2 max	Higher HR luteal No difference VO_2		T_{rec} rest 0.3°C higher luteal T_{rec} end of exercise 0.6°C higher luteal T_{sk} no difference	RPE higher end of exercise luteal
De Souza et al., (1990)		8 well-trained	Treadmill running Maximal test 40 min 80% VO_2 max	No difference VO_2, HR, ventilation	No difference lactate		No difference VO_2 max, exercise time or performance

Study	Environmental conditions	Subjects	Exercise protocol	Cardiovascular responses	Metabolic changes	Thermoregulatory responses	Performance
Dombovy et al., (1987)		8 sedentary	Cycling Maximal test 4-min work loads different intensities	No difference $\dot{V}O_2$, HR, ventilation	No difference lactate		No difference $\dot{V}O_2$ max, exercise time or performance
Hessemer and Brück, (1985)	18°C	4 sedentary 4 recreational	15 min cycling 70% $\dot{V}O_2$ max	Higher HR, $\dot{V}O_2$ luteal No difference RER	No difference plasma lactate	T_{rec} 0.5°C and SR higher luteal	Net efficiency lower luteal
Stephenson et al., (1982)	23.3 ± 0.17°C	6 trained	5 min cycling, 15 min rest 4 submax intensities. Cycling to exhaustion maximal work load	No difference $\dot{V}O_2$, $\dot{V}O_2$ max, HR, RER, ventilation, respiratory rate, $\dot{V}CO_2$		T_{rec} higher luteal	No difference time to exhaustion
Jurkowski et al., (1981)		9 sedentary	Cycling 33, 66 & 90% PO max	No difference HR, CO, $\dot{V}O_2$, SV	Lower blood lactate luteal		90% time double luteal No difference PO max
Schoene et al., (1981)		6 untrained 6 well-trained	Progressive cycling to exhaustion	No difference $\dot{V}O_2$ max Ventilation equivalent higher luteal			No difference trained Exercise time lower luteal untrained

The impact of menstrual cycle phase on strength performance has received attention in recent years again with contradictory results. Birch and Reilly (1999) compared the maximal isometric lifting strength (MILS), strength endurance (45% MILS to exhaustion) and performance of a dynamic lift in the sagittal and asymmetric planes (maximal acceptable load 6 x min^{-1} for 10 min) during 5 phases of the menstrual cycle. There was no effect of menstrual phase on any of the performance measures, however heart rate was approximately 7 $beats.min^{-1}$ higher after ovulation during the endurance and dynamic lifts. Similarly, no difference in isokinetic strength of the quadriceps and hamstrings (Cybex II, $30^{\circ}.s^{-1}$) or the first dorsal interosseus has been reported with the fluctuations in estrogen associated with the menstrual cycle (Lebrun et al., 1995; Greeves et al., 1997). In contrast, quadriceps and handgrip maximal voluntary isometric force was observed to be approximately 11% higher at ovulatory phase compared to both the follicular and luteal phases (Sarwar et al., 1996). Further measurements were made in relation to relaxation time, force-frequency relationship and fatigue index of the quadriceps during percutaneous stimulation ranging from 1 to 100 Hz. Sarwar et al. (1996) observed that accompanying the increase in strength was a parallel significant slowing in relaxation and increase in fatigability at the mid-cycle point. Similarly, Phillips et al. (1996) compared the isometric maximal voluntary contraction of the adductor pollicis muscle in trained and untrained regularly menstruating women during the ovulatory and luteal phases. They showed a significant increase (10%) in maximal voluntary force during the follicular phase when the estrogen levels were rising and a concomitant decrease in force after ovulation. The timing during the menstrual cycle of the testing and/or the muscle groups concerned may explain the contradictory findings relating to the menstrual cycle and strength. The studies by Sarwar et al. (1996) and Phillips et al. (1996) were completed to coincide with the 'surge' in estrogen prior to ovulation, whereas the strength tests of Lebrun et al. (1995) and Birch and Reilly (1999) were undertaken after ovulation, when estrogen concentrations are lower. Thus, an increase in muscular force may be associated with the rise in estrogen prior to ovulation, however this improvement is short lived. This hypothesis requires further investigation.

Deep body temperature differences during the menstrual cycle phases make temperature responses prior to and during exercise of specific interest. The greatest differences in rectal temperature, between the follicular and luteal phases, are seen

when temperature is recorded during the night, approximately 03:00 – 04:00 hours. Thus, it is not surprising that during moderate cycling at this time rectal temperature during the luteal phase was 0.6°C higher than in the follicular phase (Hessemer and Brück, 1985). A similar magnitude of difference in rectal temperature was reported by Pivarnik et al. (1992) in heat acclimatised women. The protocols employed by these two research groups were similar in exercise type, intensity (70 and 65% $\dot{V}O_2$ max) and environment (18 and 22°C). During the day, the differences in rectal temperature are often not observed both prior to an experimental period and during exercise. Prior to testing no difference in rectal temperature was observed by Wells and Horvath; (1973) the authors suggested the finding was the result of individual variability within subjects due to the activity required to reach the laboratory and long preparation period before testing, increasing the stress levels of subjects. Thus, following a long rest and relaxation period the differences in rectal temperature between phases are more profound.

In summary, the literature is equivocal regarding the effect of the menstrual cycle on the physiological responses and performance of submaximal exercise in moderate conditions. While, the controversies in the literature may partially relate to differences in the exercise testing protocols employed, the methods used for menstrual cycle verification are questionable in some research. Williams and Krahenbuhl (1997) outlined that it was "possible to have a cycle which is normal in length but is anovulatory." Thus, where the timings of trials were determined via expected days and/or circamensal changes in temperature, and not verified by hormonal analysis, the confidence in the phases investigated is reduced. From the evidence presented, hormonal verification of menstrual cycle phase did not occur in the studies by Reilly and Whitley (1994), Stephenson et al. (1982) and Schoene et al. (1981). Also the timings of trials may also affect the responses to exercise; for example, there may be physiological and metabolic differences between the early and mid-follicular phases due to the differing levels of estrogen. Clearly though the literature is equivocal, the weight of evidence suggests that an increase in training status attenuates the effect of menstrual cycle phase on exercise performance.

2.3.1.3.Performance and physiological responses to exercise during the phases of the menstrual cycle in hot conditions

The limited research into menstrual phase and exercise in the heat is presented in Table 2.2. Thermoregulation may be altered by the fluctuating hormonal milieu of the menstrual cycle and this may have implication for exercise in the heat. Deep body temperature during exercise in the luteal phase has been reported to be higher than the follicular phase in 4 of the 6 studies presented in Table 2.2 (Carpenter and Nunneley, 1988; Stephenson and Kolka, 1988b; Kolka and Stephenson, 1997; Stachenfeld et al., 2000). Deep body temperature during exercise in the heat has also been shown to be lower during the pre-ovulatory phase of the cycle compared to the early follicular phase. Stephenson and Kolka (1999) compared the responses of 4 women to walking (1.43 $m.s^{-1}$, 2% incline) in the heat (30°C) during the pre-ovulatory and early follicular (days 2-6) phases of the menstrual cycle. The women wore chemical protective clothing to incite uncompensable heat stress and 'challenge' the human thermoregulatory system. For 3 of the women oesophageal temperature was higher during the early follicular phase than the pre-ovulatory phase and was not different between phases for the other. The deep body temperature threshold for sweating was lower in the pre-ovulatory phase than the early follicular phase (T_{es}: 36.64 ± 0.35 vs 36.88 ± 0.27°C); however, sweat rate was unaffected. The lower deep body temperature during the pre-ovulatory phase demonstrates that either the ratio of progesterone to estrogen is an important determinant of deep body temperature or estrogen may increase cutaneous vasodilation.

Skin temperature has only been reported to be higher during the luteal phase of the cycle in hot conditions (48°C) by Carpenter and Nunneley (1988) and Stephenson and Kolka (1988b), however sweat rate and sweat loss were unchanged between menstrual cycle phases. During exercise in the heat, the relationship between deep body temperature, skin temperature, skin blood flow and sweat rate determines the evaporative heat loss. During the luteal phase of the menstrual cycle, forearm blood flow (FBF) is higher than during the follicular phase when completing leg exercise at

80% $\dot{V}O_2$ max (14.6 ± 2.2 vs 10.9 ± 2.4 $ml.100ml^{-1}.min^{-1}$; 35°C, 22%; Kolka and Stephenson, 1997). The authors postulated that the increase in FBF would result in a redistribution of blood that would require an increase in cardiac output to maintain

muscle perfusion during the high intensity exercise in conjunction with the additional perfusion of the cutaneous circulation. A higher heart rate was recorded during the luteal phase, indicating a higher cardiac output than during the follicular phase; however, stoke volume was not measured. A higher cardiac output during exercise in the heat during the luteal phase of the menstrual cycle compared with the follicular phase may have implications for performance.

No research has been completed that investigates exercise capacity or performance per se during the phases of the menstrual cycle in a hot environment. However, the physiological responses to exercise in the heat allow for some inferences into performance to be made. Sweat rates and sweat loss seem to be unaffected by menstrual cycle phase during exercise in the heat (Wells and Horvath, 1974; Carpenter and Nunneley, 1988; Stephenson and Kolka, 1988b; Stephenson and Kolka, 1999; Stachenfeld et al., 2000). Similarly, in contrast to the finding of Kolka and Stephenson (1997), heart rate and perceived exertion appear not to be different between the follicular and luteal phases of the menstrual cycle, when exercising in the heat (Stachenfeld et al., 2000; Carpenter and Nunneley, 1988; Horvath and Drinkwater, 1982; Wells and Horvath, 1974). Clearly, further research is required into the effect of the menstrual cycle on exercise performance and capacity in the heat; however the literature available would suggest that menstrual phase doesn't impact on a woman's tolerance for exercise in the heat. Possibly, this is because the relatively small differences in thermal, physiological and metabolic responses between phases of the menstrual cycle are insignificant in comparison with the magnitude of the changes arising in response to exercise in the heat.

Table 2.2 The responses of women, and the effects of the menstrual cycle and oral contraceptives, during exercise in hot conditions

Study	Environmental conditions	Subjects	Oral contraceptive type	Exercise protocol	Cardiovascular responses	Metabolic responses	Thermoregulatory responses	Performance or metabolic rate
Stachenfeld et al., (2000)	35°C 30%	8 untrained	Norethidrone only Ethenyl estradiol and norethidrone	40 min cycling $\cdot$ 60% VO_2 max Luteal, follicular, progestin only and progestin and estrogen compared	No difference HR, SV, BP, cardiac output		T_{es} higher progestin only T^{es} sweating threshold higher luteal and progestin only No difference SR	
Kolka and Stephenson (1997)	35°C 22%	5	Not applicable	25-30 min leg exercise $\cdot$ 80% VO_2 max	HR, FBF higher luteal		T_{es} higher luteal T_{sk} no difference	No difference metabolic rate
Carpenter and Nunneley (1988)	48°C 10%	8 untrained acclimatised	Not applicable	2 hs cycling $\cdot$ 30% VO_2 max	No difference HR		T_{rec} higher luteal T_{sk} higher luteal No difference SR, sweat loss	No difference metabolic rate
Stephenson and Kolka (1988b)	48°C 10%	5	Not applicable	Cycling $\cdot$ 80% VO_2 max until $T_{es} \uparrow 0.8$°C		ΔPV higher follicular No difference end PV No difference protein, Na^+, K^+	T_{es} higher luteal T_{sk} higher luteal No difference SR	No difference in exercise time
Horvath and Drinkwater (1982)	28, 35, 48°C	4 untrained	Not applicable	50 min walking $\cdot$ 30% VO_2 max	No difference VO_2, HR, CO, SV	No difference Na^+, K^+	T_{rec} no difference T_{sk} no difference 38, 45°C T_{sk} lower luteal 28°C	
Wells and Horvath (1974)	48°C	7 untrained	Not applicable	40 min walking $\cdot$ 50% VO_2 max	No difference VO_2, HR, ventilation	Lactate higher luteal	T_{rec} no difference T_{sk} no difference No difference SR, evaporative heat loss	

2.3.2. Oral Contraceptives

2.3.2.1.Basal Metabolic Rate

The oral contraceptive pill is the most effective form of contraception available and therefore its use is widespread among the female population of child-bearing age, including many games players. Pill usage also reduces the pain and discomfort associated with menstruation, menses length, and improves the predictability of the cycle. A number of studies have investigated the physiological effects of using the oral contraceptive pill due to the synthetic hormones they contain, particularly progestins that increase deep body temperature. The basal metabolic rate (BMR) of women taking the contraceptive pill has been monitored throughout the period of the menstrual cycle. Curtis et al. (1996) showed a random pattern of response with an intra-individual coefficient of variation in the subjects ranging from 2.4 to 4.8%. This pattern varied from that observed in subjects who did not take oral contraception, who tended to have a much greater variation in BMR and thus Curtis and colleagues (1996) concluded BMR is a 'biological constant' in women using oral contraceptives.

2.3.2.2.Exercise performance of oral contraceptive users

While few studies have investigated submaximal and maximal exercise performance when taking oral contraceptives, they all demonstrate that performance is stable throughout the monthly oral contraceptive cycle (Table 2.3; Grucza et al., 1993; Reilly and Whitley, 1994; Reilly and Whitley, 1995; Lynch and Nimmo, 1998; Giacomoni et al., 2000). Only one study has been completed investigating intermittent running and oral contraceptive use (Lynch and Nimmo, 1998). Exercise performance within 1 week of resuming the oral contraceptive was compared with approximately one week later. The trials consisted of 20 s sprints (starting speed 14.3 km.h^{-1}, increased by 1.2 km.h^{-1} each sprint) separated by 100 s passive recoveries until exhaustion. No difference in exercise performance between the two trials was found. The subject group were only recreationally active and the initial speed of the treadmill was very fast, thus it is possible that the exercise was terminated when the subjects were unable to run at the required speeds rather than being at exhaustion, per se.

Table 2.3 The responses of women, and the effects of the menstrual cycle and oral contraceptives, during exercise in moderate conditions

Study	Environmental conditions	Subjects	Oral contraceptive type	Exercise protocol	Cardiovascular responses	Metabolic changes	Thermoregulatory responses	Performance
Giacomoni et al., (2000)		7 NM 10 OC Recreational	Low dose combined monophasic	4 x 8s cycle sprints 5 x maximal jumps Squatting jump				No difference between phases or groups
Lynch and Nimmo (1998)		10 NM 5 OC Recreational	Low dose combined monophasic	Progressive high intensity intermittent running to exhaustion	No difference HR	NM: No difference lactate and ammonia OC: Lactate and ammonia higher 1 week after taking pill compared to 1 week later. Ammonia higher NM than OC	T_{au} no difference	No difference exercise time between phases or groups
Reilly and Whitley (1995)		5 NM 4 OC Trained	Monophasic Week off vs OC use	Treadmill running 12.8 km.h^{-1} 20% incline to exhaustion				No difference
Reilly and Whitley (1994)		5 NM 4 OC Trained	Monophasic Week off vs OC use	Treadmill running $\dot{}$ 70% VO$_2$ max to exhaustion	No difference $\dot{V}O_2$, $\dot{V}E$	No difference glucose and lactate		NM: Exercise time higher luteal OC: No difference in time
Grucza et al., (1993)	24°C 50%	10 NM 10 OC	8 triphasic 2 combined monophasic	Cycling Maximal test $\dot{}$ 45 min 50% VO$_2$ max	OC: No difference HR, BP NM: ΔHR higher follicular		OC: T_{rec} higher luteal. No difference ΔT_{rec} T_{sk} no difference NM: T_{rec} no difference ΔT_{rec} higher follicular ΔT_{sk} higher follicular	No difference $\dot{V}$ O$_2$ max between phases or groups

Similarly, repeated 8 s cycle sprint exercise completed by eumennorheic and oral contraceptive users demonstrated that performance did not differ with menstrual phase or oral contraceptive use (Giacomoni et al., 2000; Table 2.3). Trials were completed during menses, the mid-follicular and mid-luteal phases. The 4 cycle sprints were undertaken against increasing braking forces to include an optimal workload, which was determined during preliminary testing. There was a 3 min recovery between each sprint. The eumennorheic and oral contraceptive users also completed 5 maximal vertical jumps keeping their legs as straight as possible and a maximal jump from a squat position (90° knee flexion). Similarly, menstrual phase and oral contraceptive use did not affect jumping performance. Reilly and Whitley

(1994; 1995) demonstrated that submaximal (70% $\dot{V}O_2$ max) and high intensity running (12.8 km.h^{-1}) to exhaustion was not different during the week when the women were not taking the oral contraceptive compared with contraceptive use. While all the recent literature shows that oral contraceptives do not affect exercise performance, there is a lot of scope for future research. The type of oral contraceptive used, the training status of the women and the environment may all impact upon performance and remain to be examined.

2.3.2.3.Muscle strength in oral contraceptive users

Muscular strength and force has also been shown to remain constant in oral contraceptive users throughout the 28 day contraceptive month. Comparison of quadriceps and handgrip maximal voluntary isometric force (Sarwar et al., 1996) and isometric maximal voluntary contraction of the adductor pollicis (Phillips et al., 1996) was stable throughout the menstrual phases. Similarly peak torque during knee and hip extension and flexion at two different speeds (1.05 rad.s^{-1} and 4.20 rad.s^{-1}) is stable throughout the month in oral contraceptive users (Richardson and George, 1994). Clearly, muscular strength and force seem to be unaffected in oral contraceptive users during the different contraceptive phases.

2.3.2.4.Thermoregulation

The thermoregulatory changes observed during the menstrual cycle, which correspond to high progesterone concentrations, may be mirrored by the synthetic progestins contained in oral contraceptives.

In moderate environmental conditions, higher rectal temperatures both at rest and during exercise, have been reported in women taking oral contraceptives in comparison with the same women when not taking contraceptives, and compared with a eumennorheic group (Grucza et al., 1993; Rogers and Baker, 1997). In a walking study (60 min; 10% gradient; 4.8 km.h^{-1}) rectal temperature was 0.31°C higher at rest and remained higher during exercise in women taking oral contraceptives compared with a trial when the same women were not taking the oral contraceptive. Similarly, during the oral contraceptive phase heart rate was 6.5 beats.min^{-1} higher (Rogers and Baker, 1997). Thus Rogers and Baker (1997) concluded that the synthetic progestins found in the oral contraceptives caused an upward shift in the threshold for heat loss responses, thereby increasing both resting and exercise rectal temperatures. Grucza et al. (1993) compared the thermoregulatory responses to moderate intensity cycling (50% $\dot{V}O_2$ max) of triphasic oral contraceptive users and eumennorheic women during the follicular and luteal phases. The contraceptive group had a higher temperature threshold for sweating in the quasi-luteal phase (37.85°C) compared to the quasi-follicular phase (37.60°C; P<0.01). In the non-contraceptive group temperature sweating threshold showed a similar pattern, being at 37.7°C in the luteal phase and 37.47°C in the follicular. However, the contraceptive group showed no differences between phases in sweating dynamics and heart rate whereas the non contraceptive group had a higher heart rate increase and faster sweating dynamics (time for sweating to reach 63.1% steady state level) during the follicular phase compared with the luteal. A limitation of this study was the lack of hormone measurement to determine phase of the menstrual cycle and the use of change from rest for all statistics rather then employing an ANOVA incorporating all data points. The contrasting results from these studies (Grucza et al., 1993; Rogers and Baker, 1997) are related to the differences in protocols, particularly the types of contraceptives taken by the women and the timing of the trials. In summary, in a moderate environment, thermoregulatory responses seem to be lower during the non-contraceptive week compared with taking oral contraceptives, when the responses are more uniform.

During exercise in the heat, monopashic oral contraceptives have been shown to increase both rectal temperature and heart rate when compared to not taking contraceptives (Martin and Buono, 1997). Ten subjects performed 1 h of cycle exercise at 60% $\dot{V}O_2$ max on two occasions in the heat (30°C, RH 50%), once when they were taking an oral contraceptive and secondly when they were not. When exercise was completed during the contraceptive trial, rectal temperature and heart rate were 0.3°C and 8 beats.min^{-1} higher, respectively. Stachenfeld et al. (2000; Table 2.2) compared the exercise responses of 8 untrained women during the follicular and luteal phases of a normal menstrual cycle and when taking a progestin only or progestin and estrogen contraceptive. The women cycled in the heat (35°C; 60% $\dot{V}O_2$ max), with no discernible difference in cardiovascular responses during the 4 trials. However, deep body temperature was higher in the progestin only trial than the other 3 conditions and sweating threshold was higher in the luteal and progestin only trials. Sweat rates were not different between the follicular and luteal phases and when consuming oral contraceptives. The higher deep body temperature during the progestin only condition further emphasises that the relationship between estrogen and progesterone is clearly important, be it endogenous or exogenous, in determining deep body temperature. Oral contraceptives vary in the concentrations and proportions of synthetic progesterone and estrogen, with type (monphasic or triphasic) and with brand, and thus deep body temperatures will vary accordingly. This difference between contraceptive type may affect exercise performance in the heat and should be considered when completing research.

2.3.2.5.Hormonal and Metabolic Responses

The estrogen and progestin components of oral contraceptives have either similar or opposing effects on metabolism depending upon their concentrations, dose and the adrogenic nature of the progestin. Thus, the findings concerning oral contraceptives vary dramatically in terms of their effect on metabolites and hormones depending on the type of oral contraceptive being used (Bemben, 1993). The metabolic and hormonal responses to moderate and high intensity treadmill exercise (41, 50 and 85% $\dot{V}O_2$ max) have been compared in oral contraceptive users and eumennorheic women

(Bonen et al., 1991; Bemben et al., 1992). Bonen and coworkers (1991) employed both high and low monophasic contraceptive users, whereas Bemben et al. (1992) employed a mixed group of monophasic and triphasic users. In both studies higher growth hormone concentrations and lower glucose concentrations were recorded for the oral contraceptive users than the eumennorheic women. Due to the lower glucose concentration, Bemben et al. (1992) also recorded a lower carbohydrate utilisation in the contraceptive group, and suggested that this carbohydrate sparing effect may compensate for a decrease in hepatic glucose output. In contrast Bonen et al. (1991) did not observe any difference in substrate utilisation, even though free fatty acid concentrations were higher, as well as glucose concentrations being lower in the contraceptive group. The protocols employed by these two research groups were similar and yet in terms of substrate utilisation their findings differ. This emphasises the importance of the recognition that the types of contraceptives play a key role in the hormonal and metabolic responses to exercise.
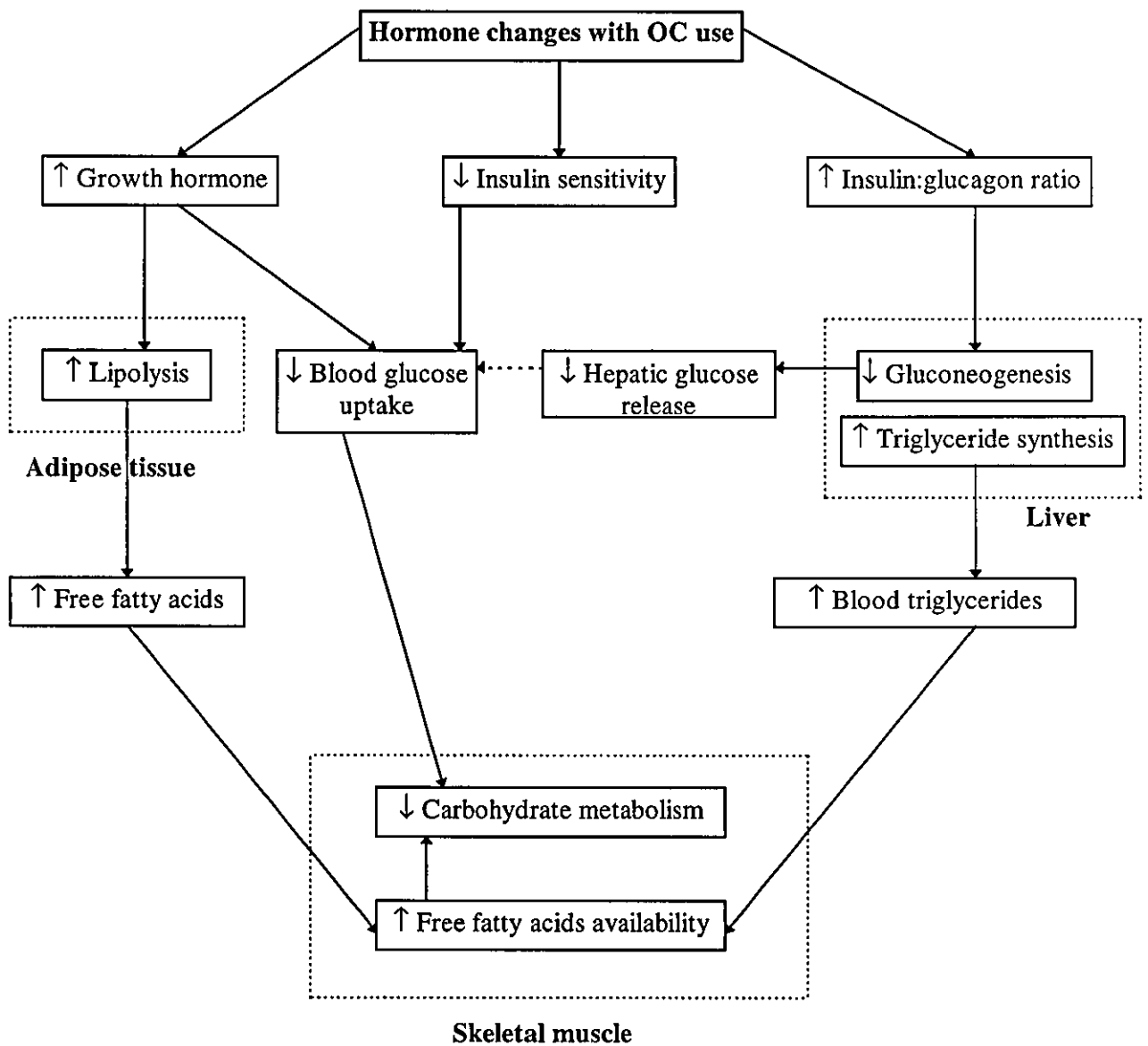
Figure 2.3 Theoretical model for the potential impact of oral contraceptives on hormonal responses and substrate utilisation during prolonged exercise (adapted form Bemben, 1993).

2.4. Heat acclimation

2.4.1. Introduction

Acclimatisation is the process by which a person adapts to repeated exposures to the heat in a natural environment; acclimation is the same process following exposures in an artificial environment, such as a climatic chamber. The early research work investigated acclimatisation, whereas with the development of technology and an increasing awareness of the importance of controlling as many variables as possible

during research, the most recent work has been completed in artificial environments. However, there are suggestions that acclimation takes longer to achieve and is less effective than exposure to the natural environment (Shephard, 1988). Throughout the rest of this thesis acclimation will be used to refer to the process of adapting to the heat.

Acclimation has been described as the result of combined stresses of internal metabolic heat production and high ambient temperature during exercise in the heat. The results of successive stresses are an improved heat transfer from the body's core to the skin and ultimately to the external environment (Armstrong and Maresh, 1991). The adaptations that occur in human systems during the acclimation process occur at different rates and the major benefits occur during the first few days (Parsons, 1993). Improved cardiovascular function is an early adaptation occurring between 3-5 days of acclimation, whereas adaptations such as an increase in sweat rate and increased blood flow require longer acclimation periods (Armstrong and Maresh, 1991). It should be emphasised that these periods of acclimation are based upon a mass of research that has predominantly investigated periods of 7 consecutive days of acclimation that entails training of an endurance nature. There is very little research into short periods of acclimation, high intensity and intermittent protocols. Thus, the time courses of adaptations for these types of acclimation protocols are still to be elucidated.

2.4.2. Early Studies on acclimation

The effect of heat acclimation has been studied widely during the last 60 years. Early research focussed upon desert type environments for military preparation (Eichna et al., 1950; Duncan, 1964). For example, Eichna et al. (1950) investigated 10 days heat acclimation in 27°C and relative humidity 15%. Three men completed the intermittent protocol of 5 bouts of 10 min walking up an inclined treadmill (2.5%) at 2.5 mph. Prior to the onset of the acclimation, all the men completed 14 days of treadmill walking and marching for conditioning and then completed 9 days of the exercise protocol outlined in a moderate environment (17°C, RH 39%). Two days after acclimation, the protocol was again completed in the moderate condition. The results showed that from day 1 of acclimation to day 10 there was a 44 beats.min^{-1} decrease

in heart rate and a 1.1°C decrease in rectal temperature at the end of the 5th bout of exercise. The lower heart rate and deep body temperature were attributed to an increase in sweat production, which resulted in a greater evaporative cooling and a higher internal gradient. However, it should be emphasised that this and other early studies had several flaws in their design regarding the training effect of the acclimation period itself. For example, Eicha et al. (1950) required their subjects to complete a further 2 h training each day on top of the acclimation period to maintain the conditioning status. However, when the data are examined with regard to the moderate trials, prior to and after the acclimation period, a slight decline in heart rate and a lower deep body temperature were observed at the end of exercise. Thus, it is difficult to determine the extent of the acclimation benefits on top of the training benefits alone. Thus, more recent studies have included a training group so comparisons between acclimation and training could be completed.

Performance following two weeks of acclimation compared to training in moderate conditions was improved (Duncan, 1964). Physiological adaptations included lower oral temperatures, pulse rates and an increase in sweat loss following acclimation compared to equivalent training in moderate conditions. Thus, from the early research it is clear that acclimation is essential for performance improvements in hot environments for both military personnel and for athletes. The research in recent years has focussed upon the adaptations that occur during acclimation, particularly the intensity, duration and type of exercise used for acclimation and the training status of the athletes being acclimatised.

2.4.3. Muscle metabolism adaptations to acclimation

A large proportion of the research into muscle metabolism and acclimation has employed a protocol of 7 or 8 days cycling (50-55% $\dot{V}O_2$ max) for 90 min.day^{-1} (40°C, 20-30% RH; King et al., 1985; Kirwan et al., 1987; Febbraio et al., 1994b). The studies employing this acclimation protocol have differed in the type, duration and intensity of exercise undertaken, before and after the acclimation protocol and thus their findings. Kirwan et al. (1987) and King et al. (1985) both observed a decrease in muscle glycogenolysis following acclimation. During the latter study,

subjects completed two 45 s sprints separated by 6 h of 30 min cycling (50% $\dot{V}O_2$ max) and 30 min rest before and after acclimation. Muscle biopsy samples were taken before and immediately after each of the sprints. The acclimation period resulted in a decrease in heart rate and rectal temperature change during the exercise and an increase in resting plasma volume of 9.2 ± 1.7%. Following acclimation there was a 42% decrease in muscle glycogen utilisation during the 6 h intermittent exercise period. Furthermore sprint performance, in terms of work done, was maintained, whereas prior to acclimation there had been a significant decrement in performance between sprint 1 and 2. King and co-workers (1985) found that during the 6 h period there were no differences in respiratory exchange ratio or blood lactate concentration and thus the rate of glycolysis remained unchanged following acclimation. Thus, King et al. (1985) suggested that the decrease in glycogen utilisation might have been a result of an increased perfusion of the active skeletal muscles after acclimation and thus increasing the delivery of both glucose and fatty acids. Furthermore, following acclimation there may have been a decrease in catecholamine secretion, as rectal and skin temperatures were lower, and thereby an increased hepatic delivery of glucose by improving splanchnic blood flow.

The decrease in sprint performance prior to acclimation is unlikely to be due to changes in body fluids, as plasma volume changes and body mass changes were similar both prior to and after acclimation, even though plasma volume expansion had occurred. Furthermore, no differences in the electrolyte (Na^+, K^+, Cl^-) concentrations of the muscle were recorded, and thus the excitability of the skeletal muscle would not have been different. Prior to acclimation, a lower muscle pH and higher lactate concentration was recorded during sprint 1, when total work was higher. Jacobs (1981) postulated that sprint and high intensity exercise performance was decreased when muscle glycogen concentration was <40 mmol.kg^{-1}, due to a lack of substrate for the flux generating step of glycogenolysis. King et al. (1985) recorded a glycogen concentration of only 46 mmol.kg^{-1} prior to sprint 2 and thus concluded that the decrement in performance prior to acclimation was a direct result of a decrease in the capacity for anaerobic energy release. The participants in this study were untrained and a control group was not employed, thus it is difficult to distinguish, following 10 days acclimation, between the effects of the acclimation and training. It is possible

that following acclimation sprint performance was maintained due to the training per se (Hamilton et al., 1991).

Kirwan et al. (1987) employed an exercise trial that consisted of 60 min cycling at 50% $\dot{V}O_2$ max and also recorded a lower glycogen utilisation. However, in contrast to King et al. (1985) lower concentrations of blood glucose and lactate were recorded. There was a slight increase in free fatty acid uptake and blood flow following acclimation, though these increases were not large enough to account fully for the decrease in muscle glycogen metabolism. In contrast, glycogenolysis during prolonged cycling (40 min at 70% $\dot{V}O_2$ max) in endurance trained athletes was unchanged following acclimation (Febbraio et al., 1994b). However, the reduction in muscle glycogen was lower in the type 1 fibres after acclimation. A lower heart rate, muscle and rectal temperature and respiratory exchange ratio were observed. Muscle lactate, blood lactate and glucose and plasma epinephrine concentrations were also lower. The lower muscle lactate concentrations may have resulted from a lower glycolytic rate in the type 1 fibres (Febbraio et al., 1994). The mechanism for this lower utilisation of glycogen in the type 1 fibres remains in question however, as the authors did not measure plasma volume (an increase in which may decrease catecholamine secretion) and did not employ a control group who undertook training in moderate conditions. Thus they were unable to suggest whether a change in plasma volume or other training adaptation was responsible for the lower epinephrine concentrations and concomitant decrease in muscle glycogen utilisation. Muscle temperature increased more following acclimation, because of a lower resting value, and thus it is unlikely that the slightly lower end temperature (40.0 vs 40.4°C) value would have had a significant effect on metabolism. In summary, even though the acclimation protocols were similar, the findings of the research groups are inconsistent. This emphasises the specificity of acclimation findings to type, duration and intensity of exercise being investigated and the training status of the subjects.

Metabolism and acclimation has also been investigated with regard to season of the year, sex and environmental temperature (20 and 40°C; Sawka et al., 1983). The acclimation protocol entailed standardised treadmill walking (1.56 m.s^{-1} or 1.34 m.s^{-1})

in temperatures of 40°C (RH 30%) or 49°C (RH 20%) for two 50 min periods daily separated by a rest of 10 min. Heat acclimation was shown to lower oxygen cost of exercise, irrespective of season of the year and environmental temperature for both men and women (Sawka et al., 1983). Overall, heat acclimation was shown to decrease metabolism further in moderate conditions (-5%) when compared to a hot environment (-3%). The study control did not include any form of training/control group and therefore the training adaptations could not be distinguished from acclimation. Also, no control or record of the menstrual cycle was undertaken and therefore acclimation effects may have been affected by menstrual cycle status.

Young et al. (1985) also observed a decrease in the aerobic metabolic rate after acclimation during 30 min cycling at a work-rate set to elicit 70% $\dot{V}O_2$ max in moderate (21°C, RH 30%) and hot (49°C, RH 20%) conditions. The acclimation period was 9 days consisting of a daily 2 h bout of treadmill walking at a temperature of 49°C (RH 20%). There were no differences in muscle glycogen utilisation both between environments or pre- and post-acclimation. In contrast to Sawka et al. (1983) accumulation of muscle and blood lactate during exercise were lower following acclimation.

2.4.4. Thermoregulatory adaptations

One of the 'classic' responses to heat acclimation is a decrease in deep body temperature (Mitchell et al., 1976; King et al., 1985; Nielsen et al., 1993). For example, 4 trained men cycled for 4 h.day^{-1} (40-50% $\dot{V}O_2$ max) for 3 days at 18°C (RH 51%), followed by a heat acclimation period of 10 days at 32°C (RH 42%; Mitchell et al., 1976). Little effect on body temperature occurred within the first 4 days of acclimation. By the tenth day skin temperature was maintained at a level to achieve thermal balance, via sweat evaporation, whilst the body surface was wet. Initially, a significant decrease in body temperature did not occur until the sweat and evaporation rate increased; a 30% increase in sweat rate was accompanied by a 10% increase in evaporation rate. However, there was also a 200% increase in unevaporated sweat emphasising the wasteful overproduction of sweat and thus the efficiency of heat dissipation within the body did not change following the

acclimation period. Overall, an increased sweating and evaporation rate and a decrease in body temperature were recorded, though individual differences were marked (Mitchell et al., 1976). Clearly acclimation to heat, as with many physiological responses, shows large inter-individual variations in the thermal adaptations. However, the small subject group (n = 4) used in these studies may have emphasised these differences.

A decrease in deep body temperature following acclimation has been shown to be associated with an increase in exercise capacity (Nielsen et al., 1993). A 78% increase in endurance time when cycling at 60% $\dot{V}O_2$ max, was recorded following 9-12 days of similar exercise at 40°C (RH 10%). A control group completed a similar protocol of training in moderate conditions (20°C); however only 3 control subjects undertook a trial in the heat before and after training. There were no reductions in muscle blood flow, cardiac output, substrate utilisation or accumulation of metabolites. Thus, Nielsen et al. (1993) concluded that high deep body temperature and not circulatory failure was the critical factor for exhaustion, which occurred when oesophageal temperature reached 39.7 ± 0.15°C. It should be highlighted however, that blood samples were not taken from the control subjects so no comparisons could have been made.

The sweating adaptations that occur may be peripheral or central in nature. Peripheral adaptations would include an increase in sweat sensitivity or mean sweating rate, whereas central changes would include a decrease in sweat threshold for example. Whether a change in sweat rate occurs is not only dependent upon the acclimation protocol and training status of subjects prior to the exposure period, but also has a relationship with the humidity in which the acclimation is taking place. In dry heat sweat rates may remain unchanged, whereas in hot and humid conditions, a much greater sweating response would be recorded following acclimation (Armstrong and Maresh, 1991). For example, Fox et al. (1967) compared the acclimation of individuals to hot-dry (45-55°C, RH 15-20%) or hot-wet conditions (49°C; subjects wore vapour-barrier suits) and a control group who undertook no acclimation. The intermittent stepping exercise was undertaken for 12 days, 2 h daily maintaining aural temperatures of 38.2°C. The hot-wet condition decreased the rate of sweat

suppression, which was not observed in the hot-dry environment. This difference could be important in determining an individual's subsequent tolerance to heat, with varying humidity level being an essential criterion to the success of the acclimatisation strategy. Also, partially linked to the environmental conditions as well as the exercise intensity, if during the acclimation sessions a sweat rate greater than 600 ml.h^{-1} is not attained, an increase in whole body sweat rate may not be achieved (Armstrong and Maresh, 1991).

A study using a short acclimation protocol (5 days cycling; 39.5°C, 59% RH) showed that sweat threshold temperature was reduced; however, there were no changes in sweat sensitivities or mean sweating with skin region or across regions (Cotter et al., 1997). A lower aural temperature was recorded at rest and was the explanation for the decrease in sweat threshold, because the onset of sweating was at the same time into the exercise period. The protocol for the acclimation and heat stress tests was for a rise in auditory canal temperature of 1.4°C to be maintained for 70 min by cycling intermittently. Overall whole body sweat loss was unaffected and the subjects felt similar both before and after acclimation. A limitation in this study was that the subjects were not allowed to drink during the two heat stress tests and thus any sweating adaptations may have been minimised by dehydration. Furthermore, the total work completed in both trials was not different and thus no evidence of acclimation was recorded. Thermal sensation, comfort and Borg scale all showed no differences between the two trials. Perhaps if the subjects had been allowed to drink water ad libitum, significant improvements in performance may have been recorded. Cotter et al. (1997) suggested that the 5 day acclimation period was too brief to permit complete a change in sweat redistribution and that the subjects who were described as "habitually active" would have an increased sweat sensitivity. However, no maximal oxygen uptake data were presented in the paper. It is worth noting that there was uniformity for the onset of sweating at all 8 body sites recorded.

In summary, thermoregulatory adjustments following acclimation may occur, but only after a prolonged period of acclimation. The timescale and extent of changes will not only be dependant upon the acclimation protocol employed, but also on the environmental conditions, particularly the humidity and the training status of the individuals (discussed in section 2.4.8).

2.4.5. Cardiovascular adaptations

An early adaptation to acclimation is an improved control of cardiovascular function, to maintain cardiac output (Wyndham et al., 1976; Mitchell et al., 1976; Fortney et al., 1979). Wyndham et al. (1976) described four specific phases of cardiovascular adaptations to the acclimation protocol used by Mitchell et al. (1976; section 2.4.4). Similarly, during prolonged low intensity acclimation (45-50°C) in untrained men and women these phases could be distinguished (Fortney et al., 1979 [cycling 30% $\dot{V}O_2$ max]; Rowell et al., 1967 [treadmill walking]). On day 1 of acclimation, there was an increase in heart rate to maintain cardiac output as stoke volume had decreased (Wyndham et al., 1976). Phase II was opposite to phase I in that during days 2-3 there was an increase in stroke volume, with a reciprocal fall in heart rate, thereby resulting in little change in cardiac output (Rowell et al., 1967; Wyndham et al., 1976; Fortney et al., 1979). Phase III, during days 4-8 of acclimation there was an increase in cardiac output, ensuing from an increase in stroke volume, due to a transient expansion of plasma volume (Fortney et al., 1979). Finally, phase IV (after days 6-8) indicated a decrease in both rectal and skin temperature towards control levels, whilst stroke volume and heart rate slightly declined toward control levels.

When analysing the thermoregulatory responses in relation to the central circulatory responses, heart rate was shown to be independently associated with an increase in stroke volume and a decrease in rectal temperature. Also, the main overall adaptations of central circulation were mostly over by day 4, whereas adjustments in rectal and skin temperature were only fractionally complete at this point. Over the final 6 days the major decreases in skin and rectal temperature occurred. Thus, it was deduced that central circulatory and temperature regulating events are not linked during acclimatisation (Wyndham et al., 1976).

It has been suggested that an increase in plasma volume may be the most critical event in heat acclimation (Mitchell et al., 1976). Plasma volume was shown to be independently correlated with heart rate, stroke volume and cardiac output. This increase in volume was accredited to a transfer of interstitial protein and water into

the vascular compartment and an increase in aldosterone (Mitchell et al., 1976; Nielsen et al., 1993). In agreement a 13% increase in plasma volume resulted in a parallel increase in stroke volume and cardiac output. In contrast, Harrison et al. (1981) suggested that it is unlikely that haemodilution is primarily responsible for the cardiovascular adjustments following acclimation. The acclimation required the auditory canal temperature of the subjects to be maintained at 38.3 ± 0.3°C for 70 min.day^{-1} over an 11 day period (except weekends). This was achieved by 30 min of intermittent immersion in a hot bath, 30 min of intermittent exercise on a cycle ergometer and 10 min of continuous exercise (environmental temperature 28°C). Following acclimation there was a dilution of the blood and an increase in haemoconcentration during exercise in the heat. However, there was no change in concentrations of plasma protein, but a net rise in total intravascular protein content.

Maximal oxygen uptake during cycling in the heat (49°C) is lower than that in moderate conditions (21°C) and this is unaffected by acclimation (Sawka et al., 1985; acclimation protocol see Young et al., 1985, section 2.4.3). Following 9 days of acclimation $\overset{\bullet}{V}O_2$ max had increased by 4% (P<0.01) for both the moderate (21°C) and hot environments (49°C; Sawka et al., 1985). A lower $\overset{\bullet}{V}O_2$ max was observed in the hot temperature compared to the moderate temperature, which was not related to differences in rectal temperature in the two environments nor related to aerobic fitness, defined in a rather limited way in this study as $\overset{\bullet}{V}O_2$ max (ml.kg^{-1}.min^{-1}) for the subjects in moderate conditions. This suggests that heat stress was the primary cause of the reduction in $\overset{\bullet}{V}O_2$ max and this was not altered via acclimation status.

The cardiovascular adaptations that occur during acclimation appear to precede any adjustments in thermoregulation. However, this research has focussed on untrained individuals and often did not employ a control group. The cardiovascular changes following acclimation mirror those associated with training and therefore it is difficult to distinguish between the two. For example, Rowell et al. (1967) employed sedentary subjects whom they familiarised in 23-25°C whilst undertaking the same protocol, until heart rate was reproducible, but this may not have prevented a further increase in

training status following the acclimation. Clearly, there is scope for research employing control groups and subjects of differing levels of training status.

2.4.6. Hormonal adaptations

It has been well documented that aldosterone may play a key role in the maintenance of fluid homeostasis following acclimation and cause a decrease in sweat sodium concentration. The renin-angiotensin-aldosterone system is the biochemical pathway that controls sodium chloride concentrations in both urine and sweat (Wenger, 1988). A conservation of sodium chloride allows the body to maintain blood and extracellular fluid volumes at an optimum. Following acclimation, there may be a decrease in stimulation for the release of aldosterone and renin as an improvement in cardiovascular stability occurs (Armstrong et al., 1989).

Finberg and Berlyne (1977) investigated the effects of acclimation and natural acclimatisation on the renin, cortisol and aldosterone response during cycling exercise (50°C, 12% RH; $\dot{V}O_2$ 1.2 l.min^{-1}). They found that there was no difference in the aldosterone or cortisol response with acclimation, but a decrease in plasma renin activity was recorded. This decrease may have been due to an increase in plasma volume and/or a decrease in splanchnic and renal vasoconstriction. This study however, has major limitations in that both the natural and artificial acclimation groups only consisted of 4 subjects each. Also, there were no control groups for comparison, the training status of the subjects was unknown and the natural acclimation testing was done over a year period during which time the training status of the group may have changed considerably. There is no evidence of any control over training and other variables during this period. A more comprehensive study was completed by Armstrong et al., (1989) to investigate plasma cortisol, renin and aldosterone response to an 8 day intermittent running acclimation period (41°C, 39% RH; Table 2.5). The intermittent running involved 9 run (68% $\dot{V}O_2$ max): rest periods of 5, 8 or 10 min. Following acclimation, the heart rate, rectal temperature, skin temperature and plasma volume changes were all lowered. However, there were no differences in either aldosterone or renin concentration. These findings differ from

those of Finberg and Berlyne (1977) but this difference could be due to the shorter protocol employed and the better study design. Armstrong et al. (1989) also reported a lower cortisol concentration following acclimation. The authors suggested that the increase in body water, physical fitness, metabolic efficiency, decrease in thermoregulatory strain or electrolyte conservation may be responsible for the decrease in cardiovascular strain and cortisol concentrations. Again, this study did not employ a control group and the subjects were not trained, thus training adaptations and acclimation can not be distinguished from one another, thus making it impossible to postulate the mechanisms of acclimation.

Vasopressin or antidiuretic hormone (ADH) has also been shown to decrease following acclimation (Shvartz et al., 1977; Table 2.4). An increase in vasopressin is observed during an initial bout of exercise in the heat decreasing urinary water loss, thereby conserving body fluid and electrolytes. The secretion of vasopressin during exercise in the heat may be stimulated by an increase in osmolality, changes in plasma volume, blood pressure and renal and hepatic blood flow (Wade 1984; Armstrong and Maresh, 1991).

2.4.7. Men vs women

The previous subsections have outlined the adaptations that may occur following heat acclimation and the discrepancies within the research. The final subsections will draw these adaptations together and particularly focus upon the possible acclimation responses of well-trained female games players.

Men and women show similar adaptations to acclimation (Horstman and Christensen, 1982; Avellini et al., 1980). However it should be recognised that the humidity of the environment will impact upon sweating responses. In humid conditions men and women have similar sweat rates, but during exercise in a dry environment men have higher sweat rates (Horstman and Christensen, 1982). The responses of men and

women to acclimation in a hot dry environment (45°C, 14% RH; $\leq$ 2 h.day^{-1}, 40% $\dot{V}$ O$_2$ max) are similar in magnitude. For example, sweat rate is increased by a similar amount in men and women, but remains higher in men, due to the initial higher rate

during exercise in the heat, prior to acclimation. A decrease in heart rate and rectal temperature and an increase in exercise time following acclimation also occurred in both men and women (Horstman and Christensen, 1982). A study comparing 4 men and women showed that the acclimation adaptations were similar during prolonged low intensity walking (36°C, 65% RH; Avellini et al., 1980; Table 2.1). Sweat rate was higher for men than women both before and after acclimation. Following the acclimation heart rate and rectal temperature were lowered in both the men and women, but no difference was recorded for the skin temperature. Sweat rate increased in the men to a much greater extent than women. During the 1st 90 min of the 3 h walk after acclimation, the men and women had similar rectal temperatures, heart rates and skin temperatures. However, by the end of the 3 h, men had a 0.3°C higher rectal temperature and a higher heart rate by 15 beats.min⁻¹. Avellini and colleagues (1980) concluded that a greater increase in sweat rate in the men did not result in any additional thermoregulatory benefits and that women were able to dissipate heat at the same rate as men, with less sweat production. Thus, women were more efficient regulators of body temperature. The reason for the lower sweat rate in women was that there was a more efficient suppression of non-evaporative sweat output, evidenced by the women demonstrating sweat suppression at a lower sweat rate than men. The authors suggested that this may be due to a more sensitive feedback from the wet skin surface to prevent excessive dripping of nonevaporative sweat.

2.4.8. Training in the cool and training status

Intense training in a cool environment cannot serve as a substitute for exercise in the heat if acclimation is desired within a 2 week period. However a 50% improvement in heat tolerance can occur as a result of 8 weeks of interval training under moderate conditions (Gisolfi and Robinson, 1969). The heat tolerance test that was completed pre- and post- training involved walking at 5.6 km.h⁻¹ for up to 90 min in an environmental temperature of 50°C. Training resulted in an increase in exercise time, accompanied by a decrease in rectal temperature, skin temperature, tissue heat storage and heart rate. Gisolfi and Cohen (1979) compared the responses of a group of endurance runners with the acclimated group of subjects. The endurance runners were shown to be in thermal equilibrium, having lower heart rates and rectal temperatures

than the acclimated group. Clearly training status impacts on the responses to exercise in the heat, however, the endurance runners and acclimated groups were working at the same absolute work intensity. Thus, they would be expected to have a lower heart rate and rectal temperature to the walking exercise irrespective of environmental condition. For such a comparison to be undertaken both groups should be working at the same relative exercise intensity, to allow inferences to be made about training status per se.

Cohen and Gisolfi (1982) employed a similar protocol study using female subjects. The interval training period was 11 weeks and required the subjects to run on a treadmill at 10-13 km.h^{-1} for 90 s followed by a 30 s recovery period. This pattern was repeated for 50-60 min.day^{-1} for 4 day.week^{-1}. A heat stress test (45°C, 17% RH) of walking at 30% $\overset{\bullet}{V}O_2$ max for 4 h was completed before and after the training and acclimation periods. Acclimation took place after the 11 weeks of interval training and required the heat stress test to be repeated on 8 consecutive days. Following training, sweat rate was increased and rectal, skin temperatures and heart rates were lowered. Thus following training, there was an improved thermal tolerance and cardiovascular stability. The heat acclimation further improved the heat tolerance, but did not result in any increase in sweat rate. However, one of the subjects showed no further improvements with acclimation than training. The exercise intensity of the heat stress test was very low, and thus may not have placed the subject under enough physiological strain, as rectal temperature did not rise over 37.6°C. A heat stress test involving higher intensity exercise may have shown further adaptations with acclimation. Sedentary females who completed a 4 week training programme of moderate exercise followed by a 4 week heat acclimation programme (30% $\overset{\bullet}{V}O_2$max) showed similar responses to exercise following training and acclimation (Fortney et al., 1979). Resting plasma volume, sodium and chloride ion concentrations were higher following training and acclimation than sedentary status. The increase after training was not enhanced by the acclimation period. However, acclimation did result in a decrease of total circulating potassium during exercise in the heat (45°C, 30% RH) that was not observed following training alone. Rectal temperatures were not altered during exercise following either training or acclimation. In contrast skin

temperatures and heart rates during exercise in the heat were lower following training and further lowered to significant levels following acclimation.

Similarly, an early training study confirmed that endurance training could improve performance in hot conditions; nonetheless it cannot replace acclimation (Strydom et al., 1966). The acclimation period involved 5 h daily of stepping up a 30 cm step at a rate of 12 steps.min^{-1}, (33.9°C), whereas a training group exercised in cool conditions (27.3°C). The acclimation group consisted of five mine labourers, who were used to working underground (hot conditions) whereas the training group was not used to such conditions. Following training in the cool condition, partial acclimation occurred, so that the training group reached the same state of tolerance to heat stress as the miners on their first exposure to the heat condition. However, the miners' heart rates and rectal temperatures following acclimation were much lower than their training counterparts. Thus, Strydom et al. (1966) concluded that training may improve performance during stepping exercise in the heat, but it cannot replace acclimation.

Roberts et al. (1977) compared the effects of a 10 day training programme followed by a 10 day heat acclimation programme. Training comprised of 1 h.day^{-1} at 75% $\dot{V}O_2$ max in an environmental temperature of 13°C, whereas the acclimation consisted of 1 h.day^{-1} at 50% $\dot{V}O_2$ max in temperatures of 32°C. Following training, there was a shift in vasodilation and sweating thresholds toward lower internal temperatures, which were further lowered with acclimation ($P<0.05$). In relation to blood flow there was no change in the linear blood flow relationship (relationship between deep body temperature and blood flow) which was higher at any given internal temperature after acclimation.

The weight of available evidence clearly suggests training in a cool environment is beneficial for thermal tolerance and thus exercise performance in the heat. Training results in adaptations of the metabolic, biochemical, haematological and cardiovascular systems and may or may not improve exercise economy in the heat, depending on intensity, volume and type of training (Daniels, 1985; [Exercise

economy is the relationship between $\dot{V}O_2$ and work rate, for example exercise economy would be improved if an athlete was running at 12 km.h^{-1} and the $\dot{V}O_2$ had decreased.]) However acclimation always results in an improved exercise economy (Armstrong and Maresh, 1991). In summary, over a short period, acclimation has greater benefits than training in a cool environment, when exercise in the heat is imminent, but it is clear that training in the cool has an important part to play in an athlete's long-term preparation for exercise in a hot environment.

From the research presented above, it is clear that training in cool conditions has a beneficial effect for exercise in the heat. Therefore, the acclimation adaptations and acclimation rate of well-trained athletes may differ markedly from sedentary or recreationally active individuals. Table 2.1 presents some of the acclimation studies that have employed well-trained athletes and their responses to acclimation.

Comparisons between trained and moderately trained athletes have shown that the responses of these groups to acclimation differ (Table 2.4; Shvartz et al., 1977; Cheung and McLellan, 1998). In the study by Shvartz et al. (1977) both heart rate and rectal temperature decreased during prolonged stepping exercise ($\leq$3 h, 39°C), following the acclimation with the smallest decreases being observed in the trained group. However the increase in sweat rates, and decrease in oxygen consumption and skin temperature were similar after acclimation in the two groups. In contrast, Cheung and McLellan (1998) showed there was a higher sweat rate during walking (3.5 km.h^{-1}; 40°C) for both the trained and moderately trained groups, but there were no such improvements in time to exhaustion, evaporation rate, metabolic rate, respiratory exchange ratio, initial or end point rectal temperatures. Cheung and McLellan (1998) employed an uncompensable heat stress acclimation protocol, which was not used by Shvartz et al. (1977); thus the contrasting findings of these two research groups may be related to the different heat stress levels of the acclimation.

The performance of well-trained athletes has been shown to be both improved and unchanged by heat acclimation (Dawson and Pyke, 1990; Nielsen et al., 1993; Cheung and McLellan, 1998). Nielsen et al. (1993) recorded nearly a doubling in time to

exhaustion during cycling (50% $\dot{V}O_2$ max; ~41°C) following 7-10 days of similar

exercise. However, the group had a wide range of $\dot{V}O_2$ max levels which would have

impacted upon the performance and other responses, as there would have been a large

interindividual variation.

Hormones and metabolites are largely unaffected by acclimation in well-trained

athletes. Aldosterone was unchanged in the studies by Nielsen et al. (1993) and

Houmard et al. (1990), because salt balance is already maximised by training (Davies

et al., 1981). Similarly plasma volume, any increase in which, has been suggested to

be the critical event in acclimation (Mitchell et al., 1976) is often stable in this group

(Armstrong et al., 1987; Houmard et al., 1990). However, thermoregulatory changes

seem to be less affected by training status. A decrease in deep body temperature has

been shown to occur in all acute acclimation protocols that have taken place in a hot

environment (Shvartz et al., 1977; Avellini et al., 1980; Houmard et al., 1990; Nielsen

et al., 1993; Febbraio et al., 1994; Cheung and McLellan, 1998). However, during a

chronic acclimation of the summer (Armstrong et al., 1987) and acclimation using

sweat clothing in normal environmental conditions (Dawson and Pyke, 1990; Table

2.4), no decrease in deep body temperature during treadmill running was recorded

compared with pre-acclimation or in comparison with a control group. In contrast,

sweat rates did not increase in all the groups where a decrease in deep body

temperature was recorded (Shvartz et al., 1977; Houmard et al., 1990) and an increase

in sweat rate in women was only recorded at one time point (Avellini et al., 1980).

Thus, a decrease in deep body temperature may occur following acute heat

acclimation, however, an increase in sweat rate, which is often observed in

recreational or sedentary groups, does not seem to be a mechanism for this

occurrence.

The cardiovascular adaptations following acclimation in a well-trained group seem to

be inconsistent and influenced by the type, duration and intensity of exercise. Of the

studies presented in Table 2.4 that measured heart rate and oxygen uptake, half

showed a decrease in these variables and the other half found no change. The

contrasting findings for well-trained athletes, emphasise that they seem to respond

differently to acclimation when compared with recreationally active or sedentary individuals who generally do show a decrease in heart rate following heat acclimation.

Table 2.4 Acclimation studies using 'well-trained' subjects

Study	n	$\dot{V}O_2$ max	Environment conditions	Acclimation protocol	Before and after acclimation trial	Thermoregulatory changes	Cardiovascular changes	Metabolic changes	Performance
Cheung & McLellan (1998)	8 7	59.8 ± 1.0 46.1 ± 2.9	40°C 30%	2 wk 5day.week^{-1} Walk 4.8 km.h^{-1} 3-7% incline Combat clothing ↑T_{rec} >1.5°C	Walking 3.5 km.h^{-1} 0% incline	↑ SR ↓ T_{rec} = T_{rec} resting ↓ T_{sk}	= HR = RER		= Time
Febbraio et al. (1994)	13	64.8 ± 4.8	40°C 20%	7 days Cycling ~50% $\dot{V}$O$_2$ max 90 min	Cycling 70% $\dot{V}O_2$ max 40 min	↓ T_{rec} ↓ T_{mus} = ΔT_{mus}	= $\dot{V}O_2$ ↓ HR ↓ RER	↓ Lactate ↓ Glucose ↓ Epinephrine = Norepineph	
Nielsen et al. (1993)	13	~59 (49-74)	40-42°C	7-10 days Cycling ~60% $\dot{V}$O$_2$ max ≤ 90 min	Cycling ~60% $\dot{V}O_2$ max ≤ 90 min	= T_{oes} end ↑ SR ↓ rate of rise T_{oes}	= $\dot{V}O_2$ = HR ↑ SV ↑ Q end pt.	↑ PV = GH = Aldosterone = Catechols. = Vasopressin	↑ Time
Dawson & Pyke (1990)	8exp 7con	63.6 ± 1.9 63.6 ± 2.4	34.5 ± 0.1 °C 60%	14 days Normal hockey training Sweat clothes 11-19°C 64-100%	Interval treadmill running 60 min	= SR ↓ T_{rec} same as con ↓ T_{sk} same as con	= HR	= Lactate	= Control

Study	n	$\dot{V}O_2$max	Environment conditions	Acclimation protocol	Before and after acclimation trial	Thermoregulatory changes	Cardiovascular changes	Metabolic changes	Performance
Houmard et al. (1990)	9	~58	40°C 27%	8 days Treadmill run 60 min 50% or 30-35 min 75% $\dot{V}O_2$ max	90 min Treadmill running 50% $\dot{V}O_2$ max	= T_{sk} = SR = S Na$^+$, K$^+$ ↓ T_{rec} 50% day 4 ↓ T_{rec} 75% day 8	↓ $\dot{V}O_2$ ↓ HR 50% day 4 ↓ HR 75% day 8	= Aldosterone = Protein = Osmolality = ΔPV	
Armstrong et al. (1987)	5	~69 ± 5	30.3 ± 0.1°C 34.9 ± 0.5%	14.5 weeks Summer training	10 min 80 m.min^{-1} 10 min 120 m.min^{-1} 10 min 160 m.min^{-1} 60 min 200 m.min^{-1}	= SR = S Na$^+$, K$^+$ = T_{rec} = T_{sk}	↓ $\dot{V}O_2$ = HR	= PV = Na$^+$, K$^+$	
Avellini et al. (1980)	4♀ 4♂	52.5 ± 3.3 57.0 ± 1.5	36°C 65%	10 days Walking 2 h 5.6 km.h^{-1} 2% gradient	Walking 3 h 5.6 km.h^{-1} 2% gradient	= T_{sk} = T_{rec} rest ↓ T_{rec} 90 min only ↑ SR men ↑ SR women 60 min only	= $\dot{V}O_2$ ↓ HR 60 and 90 min only		
Shvartz et al. (1977)	7	60.1 ± 3.7	39.4°C 51.9%	8 days Stepping ≤ 3 h 12 steps.min^{-1}	Stepping ≤ 3 h 12 steps.min^{-1}	↓ T_{rec} ↓ T_{sk} = SR	↓ $\dot{V}O_2$ ↓ HR		

2.4.9. Exercise intensity and intermittent exercise

Acclimation has been suggested to occur only when there is maintenance above a specific threshold value of both peripheral and deep body temperatures for a certain duration (Houmard et al., 1990; Armstrong and Maresh, 1991). If these criteria are achieved, a lower metabolic rate and thus metabolic heat production may occur (Houmard et al., 1990). Thus during acclimation, an exercise intensity must be used elicit these criteria. Limited research has been undertaken into either exercise intensity or intermittent exercise and acclimation (Table 2.5).

Houmard et al. (1990) compared treadmill running at 50 and 75% $\dot{V}O_2$ max on the acclimation responses to 90 min walking or jogging at 50% $\dot{V}O_2$ max. The two protocols resulted in similar responses of a lower heart rate, rectal temperature, oxygen uptake and energy expenditure. However, the decrease in heart rate and rectal temperature occurred at a faster rate following the lower intensity protocol (4 compared to 8 days), and may suggest a specificity of the acclimation. A major flaw in this study, however, was the energy expenditure of the two protocols, which was significantly higher for the 50% $\dot{V}O_2$max protocol.

Few studies have been completed with regard to acclimation and intermittent exercise, with most published studies involving only submaximal exercise both for the testing and acclimation period (Eichna et al., 1950; King et al., 1985; Armstrong et al., 1989; Cotter et al., 1997). This limited research is surprising since it has been claimed that improvements in exercise-heat tolerance are more likely to be achieved during interval training (Gisolfi and Cohen, 1979).

The only study that has incorporated games type intermittent exercise, involving high intensity bouts as well as low and moderate intensity exercise, was completed by Dawson and Pyke (1990; Table 2.5). They found no change in performance, cardiovascular, thermoregulatory or metabolic adaptations compared with a control group. However, the acclimation was undertaken in moderate environmental conditions wearing a sweat suit, rather than in the heat, which had previously been

outlined not to match the thermal responses recorded in a hot humid climate (Dawson and Pyke, 1988). Thus, there remains a dearth of information regarding acclimation protocols using high intensity intermittent running and the responses of well-trained games players to this protocol. Thus, the research presented in this thesis will focus upon this area.

Table 2.5 Acclimation studies incorporating high intensity or intermittent acclimation protocols

Study n	$\dot{V}O_2$max	Environmental conditions	Acclimation protocol	Before and after acclimation trial	Thermoregulatory changes	Cardiovascular changes	Metabolic changes	Performance
Cotter et al. 8 (1997)	Habitually active	$39.5 \pm 0.2°C$ $59.2 \pm 0.8\%$	4 days 70 min Intermittent cycling to maintain $T_{au} > 1.4°C$ above resting	As acclimation	$= T_{sk}$ $= S$ sensitivity $= SR$			$=$ Total work $=$ RPE $=$ Thermal comfort
Dawson & 8exp Pyke (1990)7con	63.6 ± 1.9 63.6 ± 2.4	$34.5 \pm 0.1°C$ 60%	14 days Normal hockey training Sweat clothes $11-19°C$ $64-100\%$	60 min Interval treadmill running	$= SR$ $\downarrow T_{rec}$ same as con $\downarrow T_{sk}$ same as con	$= HR$	$=$ Lactate	$=$ Control
Houmard et 9 al. (1990)	~58	$40°C$ 27%	8 days Treadmill run 60 min 50% or 30-35 min 75% $\dot{V}O_2$ max	90 min Treadmill running 50% $\dot{V}O_2$ max	$= T_{sk}$ $= SR$ $= S$ Na$^+$, K$^+$ $\downarrow T_{rec}$ 50% day 4 $\downarrow T_{rec}$ 75% day 8	$\downarrow \dot{V}O_2$ $\downarrow$ HR 50% day 4 $\downarrow$ HR 75% day 8	$=$ Aldosterone $=$ Protein $=$ Osmolality $= \Delta PV$	
Armstrong 12 et al. (1989)	46.9 ± 2.1	$41.2 \pm 0.5°C$ $39.0 \pm 1.7\%$	6 days 100 min 9 treadmill run – rest periods Run at $68 \pm 1\%$ $\dot{V}O_2$ max	100 min Treadmill 4 walk – rest 5 run – rest periods	$\downarrow T_{rec}$ $\downarrow T_{sk}$	$\downarrow$ HR	$\downarrow \Delta PV$ $\uparrow$ Resting PV $=$ Aldosterone $=$ Renin $\downarrow$ Cortisol	

2.5. Field hockey

2.5.1. Field hockey physiology

The aerobic and anaerobic contribution to hockey has been investigated during the 1980s and has been shown to be widely variable. Fox (1984) estimated that 30% of the energy was derived from aerobic and 70% from anaerobic metabolism, whereas Sharkey (1986) described hockey as demanding 60% of energy from aerobic metabolism and only 40% from anaerobic sources. The disparity between these two researchers may well be due to differences in the performance level of the games that they evaluated. Furthermore, there is divergence in physiological demand in relation to playing position. For example (Wein, 1981) outlined that forwards/strikers had the highest number of strenuous movements, such as hitting in the game, whereas defenders used light movements, such as pushing 70% of the time. However, in a more recent study, Lothian and Farrally (1994) outlined that there was no difference in the % of playing time spent at a high intensity between playing positions. The energy demands of hockey have similarly varied between researchers; however most estimates fall within the range of 30-50 kJ.min^{-1} (Skubic and Hodgkins, 1967; Reilly and Secher, 1990; Lothian and Farrally, 1992).

In recent years, there have been several developments in field hockey that have possibly made previous physiological research data inapplicable to the current game. Rule changes were introduced to increase the speed and attraction of the game such as the abolishment of offside and obstruction. Also the playing of the game on artificial pitches instead of the traditional grass has increased the demands on the player (Malhotra et al., 1983). The game played on an artificial pitch differs from that on grass in that it allows players to run with the ball more and has been described as 'total hockey' in that it is a team game that requires players to cover greater distance and interchange position. Players that traditionally did less, such as defenders are required to work much harder in channelling back and positioning. Malhotra et al. (1983) compared the aerobic demands of the game on both the artificial and grass pitches and outlined that playing on an artificial pitch was 18% more demanding ($\dot{V}O_2$ artificial 2.26 l.min^{-1} vs grass 1.91 l.min^{-1}). However, this was a half pitch 6-side game and differences in demand maybe greater or lesser during a full 11-side match.

2.5.2. The physiological profile of female field hockey players

Studies during the eighties, prior to the developments in the game outlined above, have shown variations in physiological profiles of hockey players with regard to playing position, though these differences have not all been consistent between researchers (Table 2.6). Bale and McNaught-Davis (1983) reported that for centre of excellence, college and club champions' players that halves/midfield players had highest cardiovascular fitness, explosive strength and were the lightest, had lower percentage fat and consequently lower somatotype ratings, particularly endomorphy in comparison to attackers/strikers, defence and goal keepers. The investigators then compared the attackers, which comprised the strikers and halves, with the defenders, which comprised backs and goalkeepers. The significant differences recorded were lower total skinfolds and absolute fat and a higher step test index (used by the researchers to measure cardiovascular fitness) for the attacking players. Thus Bale and McNaught-Davis (1983) concluded that attackers were slimmer and fitter than defenders.

Reilly and Bretherton (1986) completed an investigation that not only compared positional differences, but also playing standard. The two standards were county and centres of excellence. Numerous measurements were carried out to compare the players, which included 'anthropometric, muscular strength and power, lung function, aerobic fitness (included estimated $\dot{V}O_2$ max, maximal heart rate and physical working capcity [PWC_{170}]) and field tests. The results showed that there were no differences between playing positions in any of these measurements. Similarly for South Australian players, where even though goalkeepers had a lower $\dot{V}O_2$ max than their outfield counterparts, no significant positional physiological differences were found (Withers and Roberts, 1981). County players had a lower $\dot{V}O_2$ max, physical working capacity (PWC_{170} 13% less) and dribbled the ball slower than their centre of excellence counterparts (Reilly and Bretherton, 1986). The authors concluded from these differences that the better fitness profile of centres of excellence players suggests the importance of aerobic status (higher $\dot{V}O_2$ max) for higher standards of play.

Table 2.6 Kinanthropometric measurements of female field hockey players

Study	Player standard	n	$\dot{V}O_2$ max $(ml.kg^{-1}.min^{-1})$	Somatotype	Height (cm)	Mass (kg)	Body fat (%)	Fat free mass (kg)
Sparling et al., (1998)	US Olympic	12	57.1 ± 2.7		165 ± 6	59.6 ± 3.6	16.9 ± 2.6	49.9 ± 2.8
Withers et al., (1987)	South Australian	17			167 ± 8	62.3 ± 7.3	20.2 ± 6.0	49.6 ± 5.9
Cheetham et al., (1987)	County	12			163 ± 6	61.0 ± 5.6		
Reilly & Bretherton (1986)	Centre excellence	12	45.7 ± 8.9*	3.3, 3.6, 2.7	164 ± 6	60.6 ± 3.8	23.0 ± 1.9	
Reilly et al., (1985)	Wales international	10	54.5 ± 5.3			59.0 ± 3.2	25.8 ± 2.1	
Bale & McNaught – Davis (1983)	Centre excellence / College champs	43		3.5, 4, 2.5	165 ± 5	60.2 ± 5.2	22.7 ± 2.9	46.6 ± 3.0
Withers & Roberts (1981)	South Australian	11	50.2 ± 4.2		165 ± 7	62.9 ± 9.2	25.3 ± 6.6	

* Estimated $\dot{V}O_2$ max

2.5.3. Field hockey physiology compared to other games

It is not surprising that the dribbling position for field hockey, which has been described as 'ergonomically unsound' for fast locomotion (Fox, 1981) places an increased physiological strain upon the player when dribbling, which compounds the demands of the game (Reilly and Seaton, 1990). Seven male hockey players were asked to dribble a hockey ball or run conventionally on a treadmill for 5 min at 8 and 10 $km.h^{-1}$. Dribbling increased energy expenditure by 15-16 $kJ.min^{-1}$, which was equivalent to a 37% increase in energy expenditure at 8 $km.h^{-1}$ and a 27% increase at 10 $km.h^{-1}$. Similarly heart rate was 15% greater and rating of perceived exertion was higher when dribbling the ball in comparison to running (Reilly and Seaton, 1990). This increase in heart rate is greater than that observed in relation to dribbling a soccer ball (Reilly and Ball, 1984) possibly due to postural factors, as soccer dribbling is a lot more upright, and also due to the use of the arms for dribbling the hockey ball (Reilly and Seaton, 1990). It should be emphasised that in the modern game, this semi-crouched dribbling position is adopted not only when dribbling, but also when channelling, tackling, and running to receive the ball. Thus, the increase in work rate due to the increased physiological strain of this position on top of the 'background running' is considerable.

Withers & Roberts (1981) compared the physiological profiles of female South Australian representative hockey, softball and netball players. Measurements of $\dot{V}O_2$ max on a treadmill, power output and % body fat were taken to compare the profiles of the three groups of games players. Netball players had a lower $\dot{V}O_2$ max than the hockey and softball players. No other differences were recorded though. Thus in a later study, Withers et al. (1987) undertook a more detailed study comparing a wider variety of sports. They investigated body density and body composition of 182 South Australia representative sportswomen, which included 107 games players and covered sports such as badminton, basketball, hockey, lacrosse, squash, gymnastics, orienteering, powerlifting and rowing. Not surprisingly, the lowest % body fat was recorded for the long distance runners (13.5 ± 3.6%), with the highest % fat for the heavyweight rowers (24.2 ± 4.2%) and soccer players (22.0 ± 6.8%). In comparison the field hockey players had a body fat of 20.2 ± 6.0%, which did not differ from any

of the games players, though it was significantly higher than for the track and field athletes. Similarly all games players had a similar body density, fat mass and fat free mass, regardless of sport.

Sparling et al. (1998) investigated the bone mineral content and bone mineral density of international field hockey players. It has previously been suggested that high impact exercise tends to have a positive effect upon bone mineral density in eumenorrheic individuals (Dook et al., 1997). Thus it is not surprising that the findings of Sparling et al. (1998) were that the hockey players had a higher bone mineral density than the age and weight adjusted norms. However, it is surprising that the value (1.253 ± 0.0475 $g.cm^{-2}$) was so much higher than not only the norm, but also notably higher (approximately 8%; 1.164 ± 0.06) that other elite games players namely volleyball and soccer players, gymnasts and athletes (Madsen et al., 1998). Sparling et al. (1998) suggested that the high bone mineral density in the US Olympic hockey team was probably a combination of long term training and genetic disposition. However, as the value is so much higher than other sports, it seems possible that the extra strain placed upon the vertebrae by hockey may further impact upon bone mineral density compared to other sports.

2.5.4. Field hockey skill tests

Table 2.7 shows the validity and reliability of the field hockey skill tests that have been published in the literature. To our knowledge, no field hockey skill tests have been formulated in the last decade since the advent of the rule changes and the introduction of artificial pitches at all levels of the game in the UK and abroad. The previous skill tests will be reviewed here in terms of not only their reliability and validity, but also how appropriate they are to today's game and their suitability to be termed 'skill' tests and not 'technique' tests. For this purpose the definition of technique and skill used by Knapp (1963) will be applied. Knapp (1963) referred to technique as "the production of some pattern of movements which are technically sound" and skill as "the learned ability to bring about predetermined results with maximum certainty, often with the minimum outlay of energy, or of time and energy."

Table 2.7 The validity and reliability of field hockey skill tests

Publication	Skill test name and sport	n	Protocol	Performance measurement	Reliability	Validity
Reilly & Bretherton (1986)	"T" – run dribbling test	24	Dribbling round 4 cones 5 yards a part in a T – shape. No reverse stick	As many circuits as possible in 2 min. Best of 3 trials.		Differentiated between county and Centre of Excellence.
Reilly & Bretherton (1986)	Distance and accuracy test	24	Dribbling and hitting at a target. A set sequence repeated.	2 min. Distance covered to nearest 2.5 yards. Accuracy = no. of accurate shots as % of total shots taken.		
Champan (1982)	Chapman ball control test	23	Movement of a hockey ball across a 9.5" circle.	Total points from 3x15 s trials. 1 point each time the ball crosses the circle.	Intraclass correlation R = 0.89	Varsity players vs junior varsity. r = 0.63 (Pearson, ranked by players)
Wessel and Koening (1971)	Stewart Backboard Test	228	Hitting a ball at 2 36" targets alternately, 12" apart. From 8' away. 6 x 30s trials.	Trial score is no. of accurate hits. Total score is sum of 3 best trials.	r = 0.81	ANOVA indicated test could distinguish between poor, average & good.

Publication	Skill test name and sport	n	Protocol	Performance measurement	Reliability	Validity
Wessel and Koening (1971)	Field, control and drive (Friedel)		Pick up, controlling and hitting the ball whilst on the move.		r = 0.81 from the left r = 0.62 from the right	r = 0.87 (correlated with Schmithals & French Ball Control Test)
Schmithals & French (1940)	Combined goal shooting	51	Shooting at a numbered target backboard. 12' long and 9" high. Target in front, left and right.	Best 2 even scores + best 2 odd scores from 10 trials.	r = 0.92 (Spearman-Brown prophecy formula)	
Schmithals & French (1940)	Fielding and drive	51	Pick up ball on the run and hit it after only 1 touch out of circle. Repeated 16 times.	Time taken from picking up ball until it leaves the circle. Sum of average of 3 best odd and even times.	r = 0.90 (Spearman-Brown prophecy formula)	
Schmithals & French (1940)	Dribble, dodge, circular tackle, and drive	51	Dribbling the ball for 30' Passing the ball round a cone and dribbling round a cone. Hitting over a line.	Total time to complete 6 repetitions. Each repetition is time taken from start line to ball crossing start line.	r = 0.92 (Spearman-Brown prophecy formula)	

In 1940, Schmithals and French attempted to produce some objective tests of field hockey that could be used to determine the status of college students. Validity was determined by comparing the subjective ratings of the 51 college and club players by three nationally rated umpires with the scores generated on the tests. Six tests were designed and tested, namely 1) Dribble, dodge, circular tackle, and drive (hit); 2) Combined goal shooting; 3) Fielding and drive (pick up and hit); 4) Push pass; 5) Drive (hit) for distance; 6) Receiving ball from team mate. The authors concluded that the 'Fielding and drive' and 'Combined Goal Shooting' tests were statistically the best tests, whereas the easiest test to administer is the 'Dribble, dodge, circular tackle, and drive' test. Thus, the authors recommended the use of these three tests and suggested that further research and design needed to be completed to produce a test that incorporated changes in direction of running and improvements in the passing test.

The 'Dribble, dodge, circular tackle, and drive' test is shown in Figure 2.4. The time is recorded from the start until the ball crosses the starting line again. This was completed 6 times, with players allowed to rest as long as is required, and the final score being the average of these 6 trials.
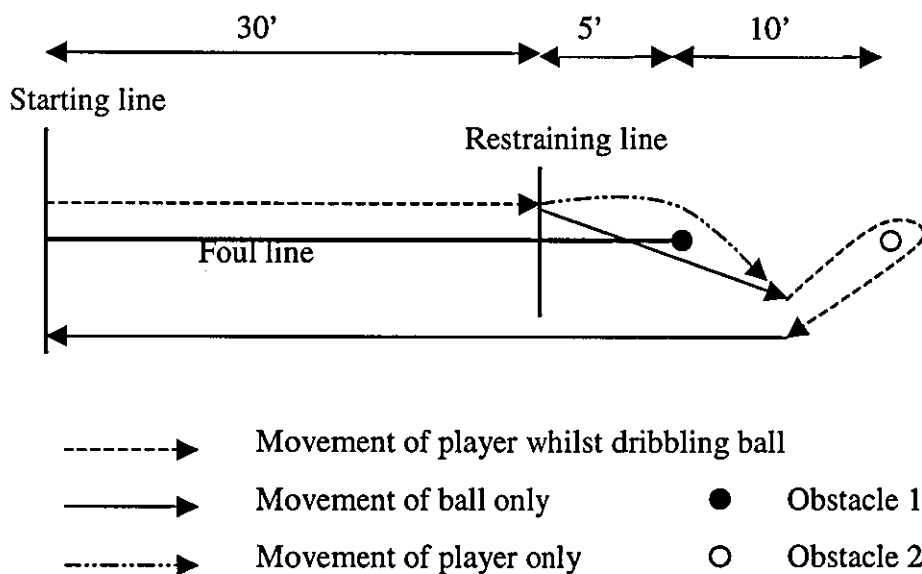
Figure 2.4 Field markings for the Dribble, Dodge, Circular tackle and Drive test; (Redrawn from Schmithals and French, 1940).

Figure 2.5 shows the Combined Goal Shooting test, which requires the players to dribble the ball from the starting line into the rectangle and then shoot at a target goal backboard. The players completed 10 repetitions with the target in each of the three positions. The score for each trial was the time taken from the start line until the ball hits the target. The final score is the 2 best odd and 2 best even scores from the 30 trials.
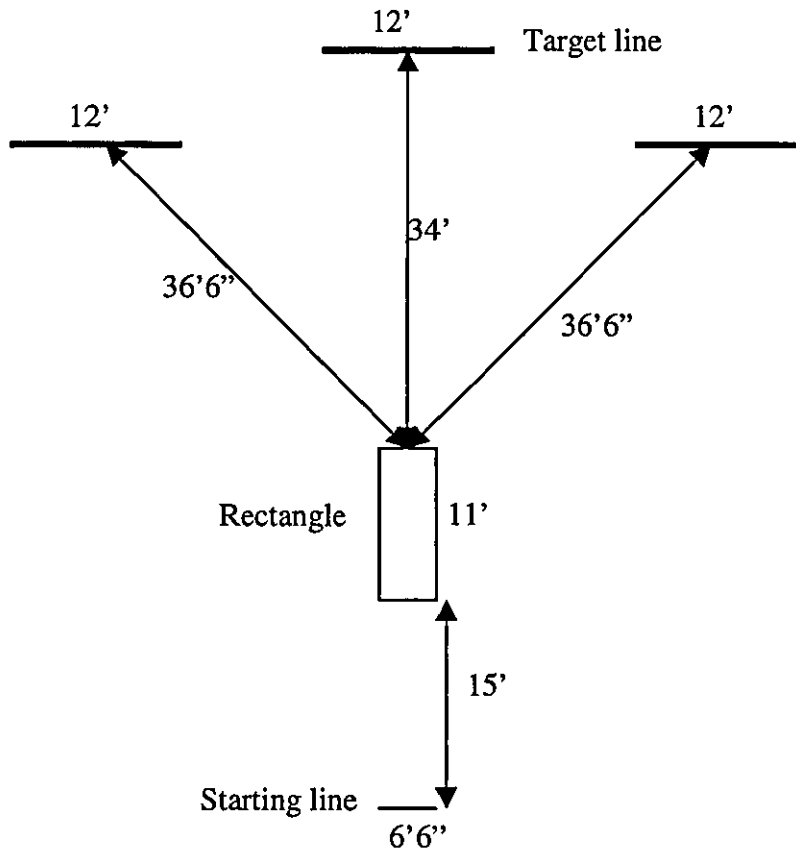
Figure 2.5 Field markings for Combined Goal Shooting test; (Redrawn from Schmithals and French, 1940).

The third test that Schmithals and French (1940) advocated was the 'Fielding and Drive' test (Figure 2.6). The tester says "go" and rolls the ball towards the goal at a speed so that it travels 45' in 1.7 s. Simultaneously, the player runs forward and attempts to pick up the ball before it reaches the foul line, takes 1 touch and hits it out of the circle between the restraining line and the foul line. This is repeated until 16 trials have been completed. The time taken from the start until the ball leaves the circle is recorded and the final score is the sum of the average of the 3 best even and 3 best odd numbered scores.
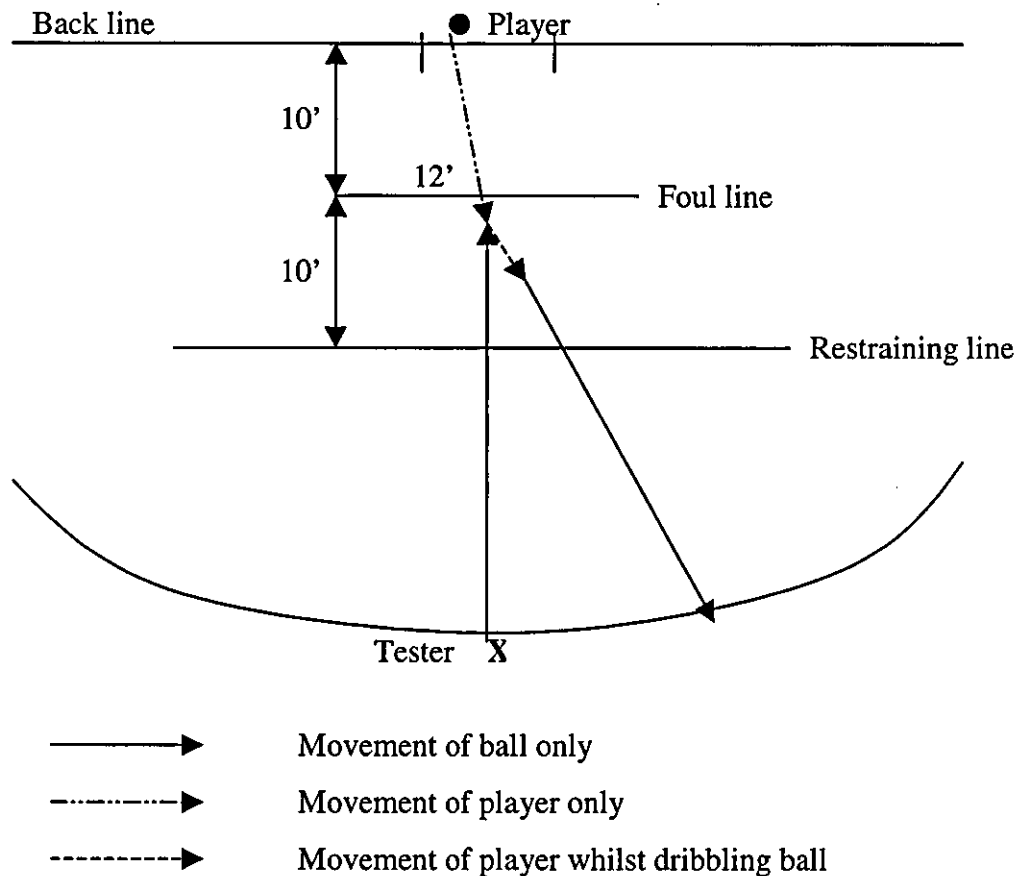
Figure 2.6 Layout of the Fielding and Drive test; (Redrawn from Schmithals and French, 1940).

The tests designed by Schmithals and French (1940) showed good reliability, but poor validity. The fielding and drive test required the rolling of a ball, which would incorporate error as it is impossible for a human to reproduce the exact ball speed and direction on each rolling. None of the tests incorporated any backward movement, passing, receiving or decision making. With this in mind, the field, control and drive test was developed which was a test with a decision making element that integrated receiving and hitting on the run (Wessel and Koening, 1971). The validity and reliability scores were <0.90 and the validity was determined in relation to one of the tests of Schmithals and French (1940) and thus was dependent on the validity of that test, which was poor. Again the test was dependent upon another human interaction.

The Stewart Backboard Test and Chapman Ball Control Test were designed with ease of administration and transference between sites, players and investigators in mind.

The Stewart Backboard Test required players to hit a hockey ball alternately at 3' targets that were separated by 12" from 8' away. Each trial was 30 s in duration and the number of accurate hits was recorded. The total score was the sum of the 3 best trials. The Chapman Ball Control Test (Figure 2.7) required the movement of a hockey ball across the circle, each time into a different third. The tests could be administered easily by anyone and did not require an external player adding to the variance. However, both these tests measure technique, rather than skill and do not require the movement of the player off the spot, which bears no resemblance to the game. Furthermore, the measure of performance was primarily related to speed of actions, with no penalties for inaccuracy.

120°

9.5 in

4.5 in
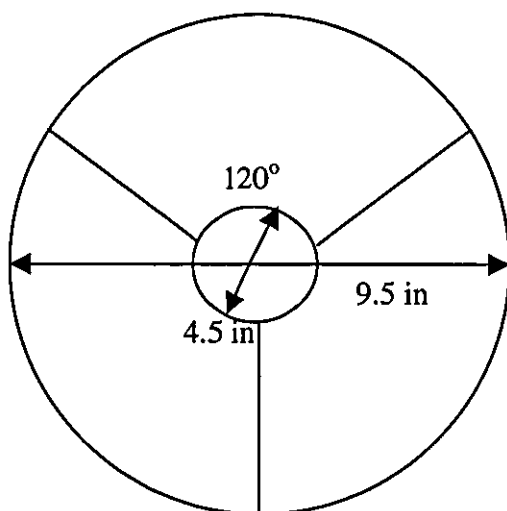
Figure 2.7 Schematic representation of the target used in the Chapman Ball Control Test.

Reilly and Bretherton (1986) designed field based tests, with performance being measured in terms of distance covered. Again the tests were easily administered and could be repeated from year to year and group to group. However, the "T" run dribbling test was controlled so that players were unable to use reverse stick, which takes away its realism from the game and also makes it a technique test rather than skill test. The distance and accuracy test, allowed a score for error to be determined in terms of percentage accuracy, but was again technique oriented in that as with many of the previous tests, there was no passing, receiving or backward movement. In summary, though the previous tests may show good reliability, the focus is upon

technique rather than skill and validity in terms of their resemblance to the demands and skills of today's game is weak.

2.6. Motor skill performance and cognition

2.6.1. Central Fatigue

Central fatigue has been defined as a negative central influence that exists despite the subject's full motivation or more objectively, a force generated by voluntary muscular effort that is less than that produced by electrical stimulation (Davis and Bailey, 1997). There is a lot of evidence regarding the role of peripheral fatigue during games activity (section 2.2.3). However, recently there has been an increasing amount of research completed with regard to central fatigue or central nervous system (CNS) fatigue. Clearly, there is a role in sports particularly, for motivational and psychological factors. Failure in central processes, also appears to play an important role in intense activity (Green, 1997). However, measuring central fatigue is very difficult and thus there is still limited research in this area.

In a review by Davis and Bailey (1997) of the possible mechanisms of central nervous fatigue during exercise, roles for the neurotransmitters serotonin, dopamine and acetylcholine and the neuromodulators cytokines and ammonia were suggested. The majority of research, however, focuses on a role for serotonin (5-hydroxytryptamine or 5-HT) and thus this will be discussed here. Brain 5-HT affects arousal, lethargy and mood and thus during prolonged exercise increased concentrations of brain 5-HT can impair CNS function, thereby causing a deterioration in exercise performance (Davis and Bailey, 1997). An increase in brain 5-HT synthesis occurs due to an increase in delivery of blood-borne tryptophan (TRP), which is a precursor amino acid to 5-HT. Tryptophan in the plasma circulates loosely bound to albumin, but free tryptophan (f-TRP) is transported across the blood-brain barrier by a specific mechanism that it shares with other large neutral amino acids, particularly the branched chain amino acids (BCAA). Thus during prolonged exercise there will be an increase in the ratio of f-TRP to BCAA and thus an increase in brain 5-HT or brain serotonergic activity. The increase in ratio is a result of BCAA being taken up from the blood and oxidised for energy in contracting skeletal muscle and an increase in f-TRP due to an increase in

free fatty acids with the onset of exercise which displaces TRP from its binding site on albumin (Davis and Bailey, 1997).

The mechanisms by which 5-HT or an increased serotonergic activity may induce fatigue, include an inhibition of the dopaminergic system, a decrease in arousal or motivation and/or an effect on the hypothalmic-pituitary-adrenal axis, which is of particular interest as this will have implications for exercise in the heat and thermoregulation. In order to assess serotonergic activity, prolactin has been measured as a marker of serotonergic activity.

In our laboratory serum prolactin concentrations have been shown to be unaffected by the ingestion of carbohydrate compared with placebo or no fluid during 90 min of the LIST and a soccer skill test before and after the intermittent running. Performance in a soccer skill test was worse when the subjects did not take on any fluids, and thus there was no evidence of 5-HT being the cause of the poorer performance (McGregor, 1999). Following rehydration and 2 h recovery serum prolactin concentrations returned to resting values, but soccer skill performance did not return to pre-exercise values. Thus McGregor (1999) concluded that it was unlikely that an increase in serotonergic activity was the cause of the decrement in soccer skill following exercise and particularly following the recovery period. This research (McGregor, 1999) was conducted in moderate environmental conditions (13-15°C, 57% RH). It is possible that prolactin and central fatigue may be more of an issue in the heat as several authors have established a relationship between prolactin concentration and deep body temperature (Brisson et al., 1989; Brisson et al., 1991; Marvin et al., 1998; Strachan et al., 1999).

The relationship between deep body temperature, exercise and central fatigue has been investigated both by undertaking exercise in different environmental conditions (Bridge et al., 1991; Pitsalidis et al., 1998) and by pre-warming and pre-cooling prior to the onset of exercise (Strachan et al., 1999). The findings have been equivocal, with prolactin concentrations reported to be both related (Pitsalidis et al., 1998; Strachan et al., 1999) and unrelated to deep body temperature (Bridge et al., 1999). Following cycling at 70% $\dot{V}O_2$ max in 4 environmental conditions (4, 11, 21, 31°C) prolactin

concentrations were higher in the heat and were shown to be correlated to rectal temperature in the hot trial only (r_s=0.50). Heart rate and skin temperature were higher throughout the hot trial than the other 3 trials (Galloway and Maughan, 1995). During cycling at 75% $\dot{V}O_2$ max in hot (31°C) and moderate (20°C) conditions, prolactin concentrations were higher in the heat, even when rectal temperatures were similar (Bridge et al., 1991). This finding suggests that skin temperature may play a key role in serotonergic activity, rather than deep body temperature. Following pre-warming and cooling by water immersion (30 min), 1 h cycling in the heat (60% $\dot{V}O_2$ max, 34°C) resulted in a greater prolactin concentration when rectal temperature was higher (Strachan et al., 1999). Thus, the authors concluded that deep body temperature was associated with central fatigue. However, they did not measure skin temperature, which could have been correlated with prolactin concentration as the higher prolactin response was reported in the pre-warming condition. Central fatigue is clearly an important mechanism when exercising in the heat and may affect skill performance. However, the initiation for an increase in serotonergic activity may be deep body temperature or skin temperature.

To distinguish between the role of deep body and skin temperature, the impact of facial cooling on prolactin concentrations has been well researched. Brisson and colleagues (1989) investigated 30 min of cycling in hot environmental conditions (27 ± 0.5°C) using trained cyclists (Table 2.8).

Table 2.8 The three trials employed by Brisson et al. (1989).

Trial	Face cooling?	Work rate (W)	% $\dot{V}O_2$ max	% HR max
1	No	222 ± 9	66 ± 0.5	78 ± 4
2	Yes	222 ± 9	65 ± 0.5	74 ± 3
3	Yes	242 ± 10	70 ± 2	78 ± 4

The 30 min exercise period resulted in a similar increase in rectal temperature to approximately 38.5°C and thus produced a similar thermal strain. The prolactin concentration was decreased by 31% with facial cooling and there was a bradycardia at a similar work rate, however the prolactin concentration were restored to the control levels when the subjects exercised at a higher work rate. There was a strong

relationship between rectal temperature and prolactin concentration (r = 0.92) during the control trial, although with facial cooling this relationship was diminished. It was suggested that the bradycardia may be the result of a vagal reflex generated from cold-stimulated skin receptors and thus the blunting of the prolactin response may be derived from afferent nerve impulses originating from face skin receptors (Brisson et al., 1989). There was no relationship between the B-endorphin response and the prolactin concentrations, and thus the authors concluded that exercise induced hyperprolactinemia does not derive from an endorphinergic stimulation. Exercise capacity has been shown to increase by 51% with facial cooling, with a concomitant decrease in prolactin concentration, during cycling (75% $\dot{V}O_2$ max; 29 ± 1.0°C, 50% RH; Marvin et al., 1998). Rectal temperature, heart rate and blood metabolites were unaltered, suggesting that fanning attenuated the central fatigue response and may be the mechanism for the increase in capacity. However, this study had final rectal temperatures that were below 39°C, which is low compared to the temperatures that have previously been associated with the exhaustion point during exercise in the heat (Nielsen et al., 1993; Gonzalez-Alonso et al.1999; Morris, 1999). Thus, it seems likely that the subjects in this study did not stop due to a high deep body temperature (Nielsen et al., 1993). Furthermore, perceived exertion was not significantly lowered with fan cooling.

Endogenous (45 min cycling 65%) and exogenous heat loads (30 min in a water tank) increase prolactin concentration (Brisson, 1991). The prolactin concentration was unchanged by fanning with air at ambient temperature, but was decreased by cold air face fanning, perhaps due to the cooling of arterial blood that supplies the thermoregulatory centre. Exercise in the cold increased rectal temperature, but not to the level of the other trials. Thus, the authors suggested that there is a body temperature threshold that must be achieved before there is a significant prolactin response. This finding could also be interpreted that the exercise in the cold did not increase skin temperature to the threshold required.

Central serotonergic activity is clearly suppressed by head cooling and suggests that central thermoreceptors, probably in the hypothalamus, play a key role in the mechanism of central fatigue. The temperature of the skin, and more specifically the

arterial blood entering the brain, clearly has a much greater association with prolactin concentration than deep body temperature (Marvin et al., 1998; Bridge et al., 1999). Thus, an increased skin temperature appears to be the trigger for the release of prolactin and probably affects both the perception of effort and exercise tolerance. The next section will further investigate how thermal comfort may affect skill performance.

2.6.2. Thermal Comfort

Studies into thermal comfort and performance have been limited, primarily due to the amount of interpretation that is required as brain activity and feelings are not only difficult to determine, but also difficult to control (Table 2.9). Also the number and variations in mechanisms controlling thermal comfort are widespread, making the pinpointing of any specific mechanism very difficult. At low deep body temperatures, though thermal comfort was related to both aural ($P<0.05$) and skin temperature ($P<0.01$), performances in a motor skill (rotor pursuit) and cognitive (manikin) task were unaffected by heating and cooling (Gibson and Allan, 1979). These findings suggest that thermal comfort is not associated with performance. Conversely, at higher deep body temperatures, performance of the pursuit task was poorer, whilst the manikin task was unaffected by heating (Allan and Gibson, 1979; Nunneley et al., 1982). While Allan and Gibson (1979) related aural and skin temperatures to thermal comfort in the study, this relationship was weaker than the association between heating and cooling the skin and thermal comfort. There was a tendency for aural temperature to affect performance ($P<0.10$), but this relationship was small compared to that of skin temperature. Overall 4 of the 5 subjects showed a negative linear relationship between thermal comfort and rotary task performance. Thus, Allan and Gibson (1979) concluded that the substantial difference in rotary task performance between heating and cooling was due to differences in skin temperature and/or the differences in direction of change of both aural and skin temperatures and thermal comfort. The manikin task was unaffected by thermal comfort, though there was a tendency for reaction time to be shortened and errors increased by heating (Nunneley et al., 1982). However, when different skin temperatures were compared at similar deep body temperatures, no performance differences were established. In summary, thermal comfort, like serotonergic activity is associated with skin temperature and

perhaps more specifically head temperature rather than deep body temperature. There appears to be a critical level of thermal discomfort beyond which decrements in motor skill performance occur.

In recent years the relationship between head and facial cooling and thermal comfort has been more widely studied as the temperature of blood going into the brain could have a significant effect on thermal perception (Brown and Williams, 1982; Nunnely et al., 1982; Boutcher et al., 1995). Loss of heat from the head is not a new concept and has been anecdotally acknowledged for centuries. The head lends itself to the removal of heat because of its large blood supply and lack of vasoconstrictive innervation. Head cooling may be particularly effective in decreasing the temperature of blood entering the brain by countercurrent heat exchange between venous blood draining the head and arterial blood ascending to the brain. There may also be local heat exchange between arterial blood and cooled venous blood from facial skin. These two heat exchange methods may thereby increase thermal comfort by cooling arterial blood supply to the brain even when oesophageal temperature is still high (Brown and Williams, 1982). Head or facial cooling has increased thermal comfort, decreased perceived exertion and increased exercise capacity (Brown and Williams, 1982; Brisson et al., 1989; Boutcher et al., 1995; Marvin et al., 1998).

Head cooling of 6 men during a 2 h period in a hot environment (40°C) was investigated using a liquid conditioned hood (Brown and Williams, 1982). Two trials were completed, with either head cooling in the first or second hour. During the first hour auditory canal and oesophageal temperatures increased similarly irrespective of head cooling. However, during the second hour, deep body temperature stabilised at approximately 38°C without cooling and 37.4°C with head cooling. Skin temperature rose similarly in both trials, but scalp temperature was considerably lower after head cooling. Thermal comfort was greater for the head than the body during cooling and greater overall than no cooling. When head cooling occurred during the second hour, thermal comfort improved to the levels seen at the beginning of the experiment even though auditory canal and oesophageal temperatures were higher by approximately 0.8°C. Similarly, Nunneley et al. (1982) found that head cooling, even with a heated body, increased the head, body and overall thermal comfort levels and lowered heart rate. With body suit cooling, a milder response was seen. Thus, clearly thermal

comfort is more associated with skin and head temperature than deep body temperature and as suggested by Cabanac et al. (1979) cooling of the arterial blood ascending to the brain may play a crucial role in thermal comfort. Studies into performance and thermal comfort are limited, but clearly where motor skills are involved thermal comfort above a critical level is associated with performance. Further research is required into more complex skills to determine if thermal comfort also plays a key role.

Table 2.9 Studies of thermal comfort and motor skill and/or cognitive functioning

Study	Temperature ($^\circ$C)	Method for altering skin temperature	Motor skill and/or cognitive task	Performance measurement	Findings
Gibson and Allan (1979)	T_{au} 37.0, 37.3, 37.6	Liquid conditioned suit 28 and 52°C	Rotary pursuit task Follow light for 1 min	Total time on target	No difference
			Manikin task Holding square or circle Facing any direction Shown square or circle 2.6s to say which hand respective shape is in	No. of correct responses Reaction time	No difference
Allan and Gibson (1979)	T_{au} 37.9, 38.2, 38.5	Liquid conditioned suit	Rotary pursuit task Follow light for 1 min	Total time on target	Performance poorer during heating by 13.6, 16.0 and 18%
Nunneley et al. (1982)	T_{oes} 38.5 to 39.0	Liquid conditioned body suit and hood. Independently controlled	Manikin task (see above)	No. of correct responses Reaction time	No difference

2.6.3. Muscle glycogen degradation and hypoglycaemia

Research into muscle glycogen degradation, hypoglycaemia and skill performance has concentrated on soccer and tennis performance and thus will be discussed here. Muscle glycogen degradation following soccer matches has been documented to range from 40 to 90% (Saltin, 1973; Jacobs, 1988) and results in a concomitant decrease in blood glucose concentration. Hypoglycaemia may affect soccer skill performance, as the central nervous system is dependent upon glucose for its metabolism (Shephard and Leatt, 1987). Carbohydrate ingestion prior to (1775-1895 kJ glucose syrup) and during (6.4% carbohydrate – electrolyte solution) simulated and real soccer matches has shown that blood glucose concentrations are maintained (Muckle, 1973; McGregor et al., 1999; McGregor, 1999). However, glycogen degradation may be decreased or remain the same (Kirkendall et al., 1988; Leatt and Jacobs, 1988).

The findings for carbohydrate supplementation and soccer performance are equivocal, as performance may be improved (Muckle, 1973), unaffected (Zeederberg et al., 1996, Strudwick, 1998) or decreased (Zeederberg et al., 1996). Team and individual performance in terms of goals scored and conceded, scoring efforts and ball contact, is improved with glucose syrup ingestion (Muckle, 1973). However, successful tackles were decreased and the success rate of heading, dribbling and shooting also tended to be lower after glucose polymer ingestion. In our laboratory, soccer skill was shown to be maintained following 90 min of the LIST in moderate conditions, but the performance was not different between a placebo and a 6.4% carbohydrate-electrolyte solution (Strudwick, 1998). The equivocal findings may be due to the measurements used in the determination of skill performance as during match-play (Muckle, 1973) many external variables will affect performance in comparison with the laboratory based test (Strudwick, 1998).

Carbohydrate supplementation has also been shown to improve stroke performance in well-trained tennis players (Vergauwen et al., 1998). A skill test was completed before and after 2 h of controlled training, which diminished defensive rally performance in the carbohydrate and placebo condition. However, carbohydrate

supplementation ($0.7g.kg^{-1}BW.h^{-1}$) decreased the number of errors and unreachable balls compared with the placebo.

The effect of glucose ingestion and glycogen degradation on motor skill performance following intermittent exercise in the heat has not yet been investigated. In such environmental conditions glycogen utilisation may be increased (Fink et al., 1975; Febbraio et al., 1994a; Febbraio et al., 1994b, Morris 1999) or unaffected compared with a moderate environment (Young et al., 1985; Nielsen et al., 1990; Yaspelkis et al., 1993; Maxwell et al., 1999). Therefore, skill performance may be detrimentally affected to a greater extent in the heat than in a moderate condition.

2.6.4. Dehydration and intermittent exercise

During intermittent exercise in the heat, the evaporation of sweat is often the only means of exchange of body heat to the environment. Prolonged sweating results in a net loss of body water and electrolytes and thus dehydration (Wade and Freund, 1990). Soccer players can lose 4 to 5 kg in body mass during the course of a match in the heat (Mustafa and Mahmoud, 1979). This figure will vary widely between players and with the environment. Such levels of dehydration can impair endurance performance (Armstrong et al., 1985; Sawka and Pandolf, 1990; Fallowfield et al., 1996) cognitive functioning (Gopinathan et al., 1988) and soccer skill performance (McGregor et al., 1999).

McGregor et al. (1999) compared soccer skill performance with no fluid and fluid replacement after 90 min of intermittent running (LIST) in moderate conditions. Dehydration resulted in a decrease in soccer skill performance that was attenuated with fluid replacement. Cognitive functioning, in terms of a simple mental test, however was unaffected by dehydration. The performance of basketball players has also been compared with and without fluid replacement (Hoffman et al., 1995). Again skill performance was declined in terms of field goal percentage, when dehydrated. McGregor (1999) further investigated the relationship between dehydration and soccer skill performance. Players were rehydrated during a 2 h recovery period before completing a further soccer skill test. Following rehydration, soccer performance

remained adversely affected, thus suggesting that dehydration was not the mechanism behind the decrement in performance following intermittent running.

Soccer skill performance following a match in the heat (27°C) has also been examined in acclimatised national players (Rico-Sanz et al., 1996). The players were either hyperhydrated or voluntary hydrated prior to the start of the match. Hyperhydration was associated with increased body water reserves and a lower rectal temperature. However, soccer skill (Zelenka et al., 1967), declined from pre-match scores in both hydration conditions. Thus, though hydration decreases thermal strain during games activity in the heat (Morris, 1999; Rico-Sanz et al., 1996), it does not result in a maintenance of soccer skill following intermittent running.

The impact of a hot environment (35°C, 65% RH) on tennis skill performance as measured by service, groundstroke and volley accuracy and power has been investigated (Dawson et al., 1985). The players completed a tennis match on court in moderate environmental conditions (23°C, 64% RH) for 1 h and then completed the skill test. At the change of ends in the match rectal and skin temperatures were recorded. Work – rest patterns were timed so that they could be replicated on a treadmill in a heat chamber. The players completed intervalised treadmill running in the heat, which mimicked the activity pattern of the match on court and included a racket simulator. Immediately following the 1 h period in the heat, the players went on court and completed the skill test, after warming up as they required. Following the intervalised treadmill running in the heat, there was a 19%, 26% and 14% poorer performance than the match in moderate conditions, for service, groundstrokes and volleys respectively. This decrement in performance was linked to the greater thermoregulatory and cardiovascular strain experienced by the players in the heat. However, this study is limited in its design, as the treadmill running would not provide a good comparison to the tennis match. The match would have resulted in numerous turns, postural changes, different running directions and muscle fibre recruitment, which would not have occurred on the treadmill. Also, following the treadmill run, rectal temperature was not recorded during the warm up phase on court and could have decreased considerably before the onset of the skill test, thus not allowing for comparison in thermoregulatory strain during the skill test itself. Dehydration and intermittent exercise in the heat and moderate conditions have both

been associated with a decrease in motor skill performance, though the findings are equivocal.

2.6.5. Summary

No research to date has been completed in regard to field hockey skill performance, which has been shown to be unique in its physiological demands, following intermittent running. Thus, the research presented in this thesis will try to investigate the effect of intermittent running, in hot and moderate conditions, on field hockey skill.

3. General Methods

3.1. Introduction

This chapter explains the methodological procedures that were used in the experimental studies described in the thesis. All the studies were carried out in the laboratories and gymnasium facilities of the Department of Physical Education, Sports Science and Recreation Management at Loughborough University. The Loughborough University Ethical Advisory Committee approved the methods described here prior to the commencement of data collection. All procedures were carried out in accordance with the 'Code of Practice for Workers having Contact with Body Fluids'.

The chapter is organized into 4 sections. The first section outlines how the subjects were recruited and the information they received is explained. The second section describes the preliminary tests and procedures completed by the individuals who participated in the various studies. In the third section, the protocol and procedures used and measurements made in the main trials are described. The final section outlines the methods adopted for the collection, treatment, storage and analysis of blood samples.

3.2. Subject recruitment

The subjects who participated in the studies described in this thesis were all volunteers recruited by general advertising or 'word of mouth'. A potential subject was given in writing information that detailed the rationale for the study, the specific requirements of the study design, the procedures and techniques involved, and any possible risks and discomforts (Appendix A1). In addition to the written information, the procedures and time commitment were also verbally explained. Every effort was made to ensure an individual had a complete understanding of what their participation in a particular study would require. Subsequently, if an individual indicated she still wished to involve themselves in the particular study she signed a statement of informed consent (Appendix A2). However, prior to actual involvement an individual was required to complete a health history questionnaire in the presence of an

experimenter for clarification purposes (Appendix A3). All subjects were female hockey players who trained at least 3 times a week and played 2 matches per week during the competitive season.

3.3. Preliminary tests

3.3.1. Measurement of height

Height was evaluated to the nearest 0.1 cm using a stadiometer (Holtain Ltd., Crymych, U.K.). Subjects were instructed to stand with their heels together against a metal plate with their toes separated by an angle of approximately 60°. With the subject in a relaxed, but erect, stance the experimenter ensured the buttocks and the back of the head were in contact with the vertical board of the stadiometer. Subjects were then asked to breathe in and the stadiometer headboard was brought down to the superior aspect of the head with sufficient pressure to compress the hair. In order to compensate for any shrinkage in the intervertebral discs gentle traction was applied to the mastoid processes. The height was then read off the stadiometer scale.

3.3.2. Estimation of maximal oxygen uptake

In the studies described in Chapters 4, 5, 7 and 8 maximal oxygen uptake ($\dot{V}O_2$ max) was estimated using the progressive multistage fitness test (Ramsbottom et al., 1988), which was based on previous work (Leger and Lambert, 1982). The test required subjects to run over a marked 20 m distance, in time to an audible signal ('bleep') dictated by a pre-recorded tape (The National Coaching Foundation, Leeds, U.K.), turning at each end of the 20 m. The running speed of the tape was verified by checking that two pre-recorded 'bleeps' on the cassette were within 60 ± 0.5 s of each other. The 20 m distance was also checked with a 30 m measuring tape. Subjects started running at 2.22 m.s^{-1} and the speed increased by 0.14 m.s^{-1} approximately every 60 s thereafter. They were required to place a foot on or over the taped line that delimited the 20 m distance and to keep running until they were unable to keep pace with the audible signal. Subjects were verbally encouraged throughout the test and the numbers of shuttles completed by subjects were recorded (Appendix A4). A test was

regarded as having ended, and $\dot{V}O_2$ max attained, when a subject withdrew herself or was unable to reach the taped line in time with the 'bleep' on three consecutive occasions.

The value obtained from this test was then used to set the submaximal 'cruise' and 'jog' running speeds used in the main trial protocol using the formula and calculations presented in Appendix A5.

3.3.3. Familiarization with the protocol for the main trial

In the studies described in Chapters 4, 5, 7 and 8, prior to their participation in the main trials, subjects were familiarized with a modified version of the Loughborough Intermittent Shuttle Test (subsequently referred to as the 'LIST' in this thesis; Nicholas et al., 2000), in hot environmental conditions (30-31°C). The familiarization sessions required subjects to perform 2 sets of the LIST or 30 min of intermittent running. Subjects were also fully familiarised with the field hockey skill test on at least 3 occasions prior to participating in the studies described in Chapters 6, 7 and 8.

3.4. Protocol, procedures and measurements for the main trial

3.4.1. Protocol

3.4.1.1.The LIST

In the main trials in the studies described in Chapters 4, 5, and 8 subjects performed the LIST in hot (30-31°C) environmental conditions and in Chapter 7 in hot and moderate (20°C) conditions. The test was a modified version of the Loughborough Intermittent Shuttle Test (LIST) developed by Nicholas et al. (1995) and reprinted by Nicholas et al. (2000). The reliability of the LIST in hot and moderate conditions is shown in Appendix A6. The mean difference ± 95% limits of agreement are –0.7 ± 5.6 (n = 7) and 0.3 ± 5.0 min (n = 8) in hot and moderate (Nicholas, 1996) conditions respectively. The LIST requires subjects to walk, sprint, cruise and jog until 11 sprints have been completed taking approximately 15 min (Figure 3.1). This series of activities constitutes 1 set of the LIST. Subjects were allowed 13 s to complete each

of the three 20 m walks, the sprint was maximal, and the intensity of the cruise and jog phases of the test was set relative to an individual's maximal aerobic power as estimated on the progressive multistage shuttle run test (Appendix A5). The average intensities of the cruise and jog phases in the studies were 95/55% in Chapter 4 and

85/50% in Chapters 5, 7 and 8 of estimated $\dot{V}O_2$ max. Each set of exercise was followed by a 3 min rest period. The intensity of the cruise and jog was reduced after the study presented in Chapter 4 to try to increase the distance completed by the subjects to allow a plateau in deep body temperature to be recorded. In the studies in Chapters 4 and 5, subjects repeated sets of the LIST until exhaustion or rectal temperature reached a pre-determined level (39.5 or 40°C). An increase in rectal temperature after Chapter 4 to 40°C was the result of an alteration in this criterion by the Loughborough University Ethical Committee and again allowed the subjects to exercise longer. In the studies described in Chapters 7 and 8 subjects completed 4 sets of the LIST interspersed with 3 field hockey skill tests. A skill test was completed prior to the onset of the LIST and after 2 and 4 sets of the LIST.
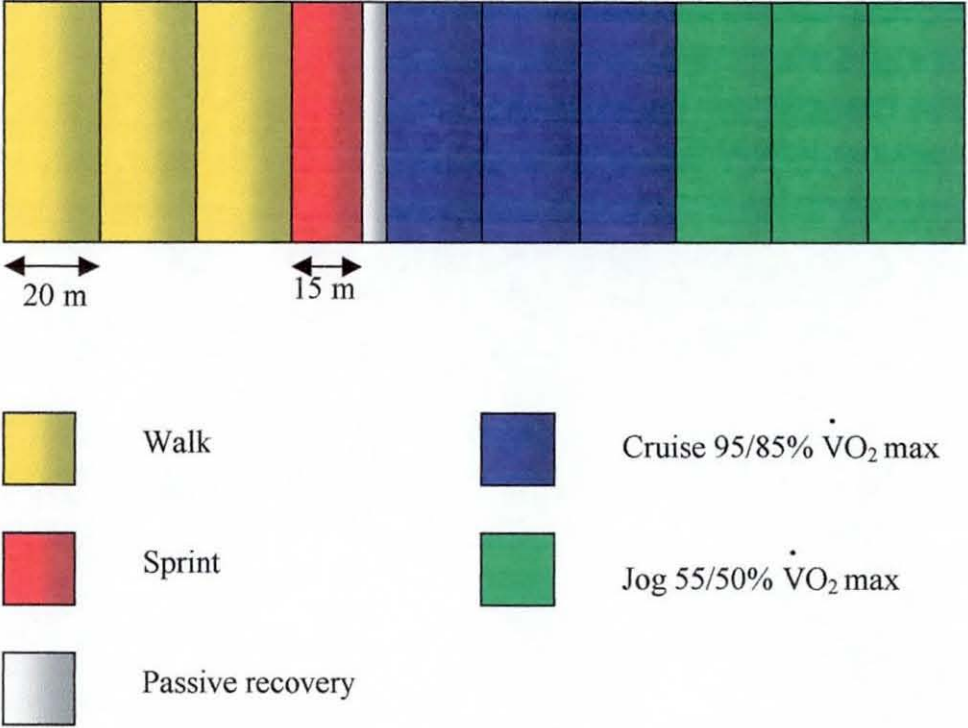
Figure 3.1 Schematic representation of 1 repetition of the LIST. (11 sprints equates to 1 set of the LIST).

3.4.1.2. The field hockey skill test

The field hockey skill test was designed and used during the studies described in Chapters 6, 7 and 8. (A detailed description of the test is given in Chapter 6). The skill test took place in the gymnasium on a section of nylon, fully synthetic water based turf (DD Action Turf, Desso DLW Sports Systems, Oxfordshire, England). The players regularly train and play on this and similar surfaces. Players initially started on a line marked on the gymnasium floor, which was 16 yards from the goal, which will be referred to as the starting line. The test required the subjects to dribble a hockey ball round 3 cones; they then broke an infra-red beam (RS Components Ltd., Corby, U.K.) which was at pitch level, which triggered a light on either the left or right hand side of a goal. A pass was then played against a hockey specific rebound board (Exportise Ltd. UK), the ball was received by the player from the board and the player then shot towards a target area (36 x 18" [0.92 x 0.46 m]) at the opposite side of the goal to where the light was illuminated (Figure 3.2). The time taken between the player breaking the beam to trigger the light and the ball hitting the backboard (144 x 18" [3.66 x 0.46 m]) was recorded using a computerised timing system (BBC Microcomputer). The timing was triggered when the infra-red beam was broken and was stopped by a signal from a microphone placed on the reverse side of the backboard. This time period was referred to as the 'decision making' time as it required players to decide on the strength of pass to be made against the rebound board, the direction in which they had to shoot and the type of shot they would take. On completion of the shot players ran immediately back to the starting line, on which they had to place at least 1 foot and then repeated the test. In total each skill test comprised of 6 series of dribbling, passing, shooting and running back to the starting line. The total time for the 6 repetitions was recorded. If a player missed the appropriate target area or touched one of the cones, a 2 s penalty was added to their total time and this was regarded as the overall performance time or performance on the field hockey skill test. The reliability and validity of the field hockey skill test are examined in Chapter 6.
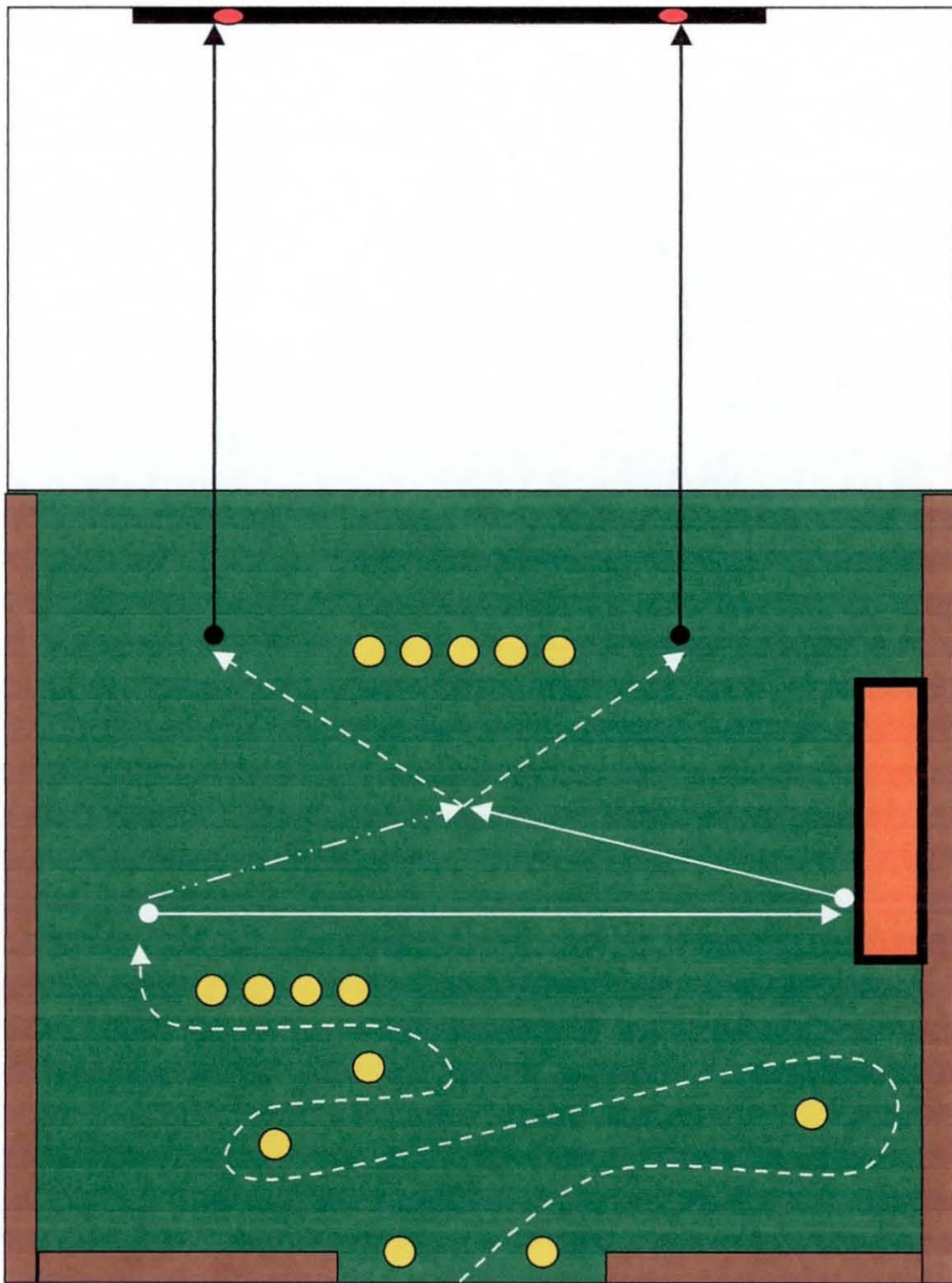
Backboard with light

Movement of ball only (pass or shot)

Movement of player whilst dribbling ball

Movement of player only

Disc cone

Indoor boards

Rebound board

Sports Turf

Figure 3.2 Diagram of the field hockey skill test

3.4.2. Procedures

In all studies (and familiarization sessions) the intermittent exercise and/or the field hockey skill test took place in a gymnasium (~25 x 15 x 10 m) on a flat, non-slippery, wooden floor. Two taped lines delimited the 20 m distance that formed the basis of the LIST. Another tape-strip set 10 m from these lines indicated the half way distance. The distances were verified using a 30 m measuring tape. The distance between the photoelectric light cells (RS Components Ltd., Corby, U.K.) used to measure the 15 m sprint times during the LIST and all the field hockey skill test dimensions were confirmed in the same way. A diagrammatic representation of the layout of the gymnasium is given in Figure 3.3.

For the studies in Chapter 4, 5, 7 and 8 subjects reported to the laboratory adjacent to the gymnasium on the morning of a main trial 12 h after their last meal. In the 2 days preceding their first trial subjects followed their normal diet. This was recorded in terms of average food portions and repeated prior to the remaining trial. Subjects refrained from alcohol and intense physical activity for 24 h before each trial. For all studies, trials for a particular individual were carried out at the same time of day to control for circadian influences.

For the studies in Chapters 4, 5, 7 and 8 an 18 gauge/45 mm cannula (Venflon 2, BOC Ohemeda AB, Helsingborg, Sweden) was then inserted into an antecubital or forearm vein of a subject under local anaesthetic (Lignocaine hydrochloride 1% w/v, Antigen pharmaceuticals Ltd., Roscrea, Ireland). The cannula was kept patent with sterile saline solution (Sodium chloride 0.9% w/v, Antigen pharmaceuticals Ltd., Roscrea, Ireland). After insertion of the cannula subjects were allowed to rest for 10 min and then stood up when they felt capable of doing so. Fifteen minutes after cannulation, during which time subjects remained standing, a resting blood sample was collected. Having voided, nude body mass was measured (Model 3306ABV, Avery Industrial Ltd., Leicester, U.K.).
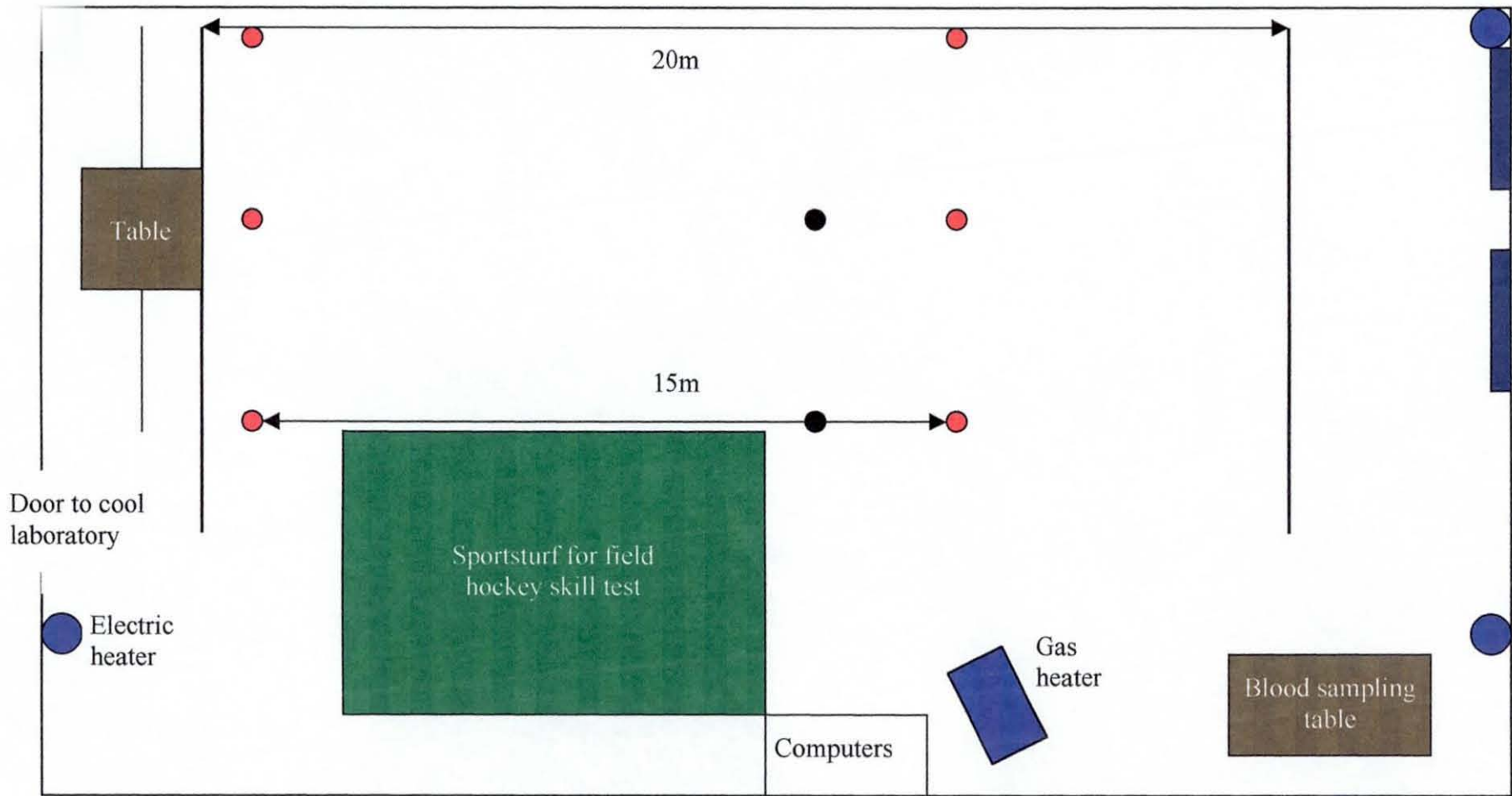
Figure 3.3 Diagrammatic representation of the layout of the gymnasium.

20m

15m

Table

Door to cool laboratory

Electric heater

Sportsturf for field hockey skill test

Gas heater

Computers

Blood sampling table

● Infra-red beam   ● Stools for water   Crash mat

Following insertion of a rectal probe (Edale Instruments Ltd., Cambridge, U.K.) to a depth of 10 cm beyond the anal sphincter, subjects moved into the gymnasium. After a resting rectal temperature was recorded, subjects performed a standard warm-up of jogging, stretching and faster pace running for 15 min.

During this period subjects were encouraged to drink water thereby ensuring an initial volume of water was placed in the stomach to facilitate gastric emptying. During the main trials subjects drank water ad libitum.

Subjects then began the LIST (Chapters 4 and 5) or completed the field hockey skill test (Chapters 7 and 8). The same experimenters were responsible for conducting the trials in a particular study. During each trial investigators ensured that subjects placed at least one foot on or over the lines delimiting the 20 m distance at each end of the gymnasium and on the starting line of the field hockey skill test. Subjects were able to gauge their required running speeds and exercise intensity by following the amplified audio signals generated from a microcomputer (BBC model B). Experimenters ensured subjects kept pace with the audio signals from the computer and reminded the subjects immediately prior to each sprint to perform it maximally and thereafter encouraged them to do so. The experimenters gave the same verbal cues to all subjects. Sprint times over 15 m were measured using 2 infra-red photoelectric cells (RS Components Ltd., Corby, U.K.) connected to the microcomputer.

3.4.2.1.Subject supervision and chaperoning and reasons for withdrawal

Throughout the preliminary and main trials, study participants were supervised by at least two investigators, one of whom was female. The subjects weighed themselves node, and inserted the rectal probe in the strictest privacy. The participants were instructed and encouraged to stop exercising at any time when the demands of the test become intolerable. In addition, subjects were continually monitored and if an individual appeared unduly distressed, or if rectal temperatures rose above 39.5 (Chapter 4) or 40°C (Chapter 5, 7 and 8) during the intermittent running, exercise was terminated.

3.4.2.2.Raising the environmental temperature in the gymnasium

During the hot trials in the studies, the temperature in the gymnasium was raised to the appropriate level by means of four electric fan heaters (Andrews DE65, Andrews Industrial Equipment Ltd., Nottingham, U.K.) placed in the corners of the gymnasium, and by means of an externally vented gas heater (Andrews IG175, Andrews Industrial Equipment Ltd., Nottingham, U.K.) placed at a mid-point in the hall (Figure 3.3).

3.4.3. Main trial measurements (Chapters 4, 5, 7 and 8)

3.4.3.1.Measurement of environmental conditions

A whirling hygrometer (Brannan Thermometers Ltd., Cumberland, U.K.) was used to measure atmospheric dry and wet bulb temperatures. This was done at three set positions along the length of the gymnasium at the mid-point of each exercise set and following the rest period, of the LIST. From these measurements relative humidity was calculated using a formula from Parsons (1993) and a spreadsheet (Appendix A7).

3.4.3.2.Measurement of rectal temperature

Rectal temperatures were measured using a probe and logger (Edale Instruments Ltd., Cambridge, U.K.) during the 4th, 8th cycles of each set of the LIST and the 3 min rest period between sets (Chapters 4, 5, 7 and 8), before and after the field hockey skill test (Chapters 7 and 8) and at exhaustion (Chapters 4 and 5). When rectal temperatures were measured during exercise subjects remained stationary and only walked for 20 m in that cycle (compared with 60 m when temperature was not being measured).

The accuracy of the readings given by the rectal probes and thermometers employed during the studies was ensured prior to their use by cross-checking the values given by them in a water bath (temperature range 37 to 40°C) with those given by a thermometer.

Just prior to each main trial, and at 15 min intervals during experiments, the logger (into which the connecting plug from the rectal probe was placed) was calibrated. Prior to testing individual probes and loggers were labelled and, wherever possible, the subject's rectal temperature during a series of trials was always taken using the same probe and logger.

3.4.3.3. Heart rate and rating of perceived exertion, thirst and thermal comfort

Heart rate was continuously monitored throughout all main trials using short range telemetry (Sport Tester or Vantage NV, Polar Electro Oy, Kempele, Finland; sampling frequency 15s [Chapter 4 and 5] and 5s [Chapter 7 and 8]). Rating of perceived exertion was recorded prior to the 11th sprint in each exercise set using the Borg scale (1962). At the same time point ratings of perceived thirst (Appendix A9) and thermal comfort (Appendix A10) were recorded.

3.4.3.4. Estimation of sweat rate

Sweat rates were estimated from pre- and post-exercise nude body mass measurements correcting for fluid intake. It was assumed that 1 litre of fluid was equivalent to 1 kg.

3.5.   Blood treatment, storage and analysis (Chapters 4, 5, 7 and 8)

3.5.1.   Blood sample collection, treatment and storage

In the studies described in Chapters 4, 5, 7 and 8, venous blood samples were obtained via an indwelling cannula at pre-determined time points during the main trials. The cannula was connected to a three-way stopcock (Connecta, BOC Ohmeda AB, Helsingborg, Sweden). A sterile, non-heparinized saline solution (B. Braun Medical Ltd., Buckinghamshire, U.K.) was used to keep the cannula patent. Twelve ml samples of venous blood were drawn into appropriately sized syringes (Becton-Dickinson, Oxford, U.K.) and the blood was immediately dispensed into collection tubes (Sarstedt Ltd., Leicester, U.K.). All samples were taken in the standing position because changes in posture may influence plasma volume (Harrison, 1985).

Each 12 ml of venous blood was divided into 3 (Chapter 4 only) or 4 aliquots (Chapters 5, 7 and 8): 1.5 ml was placed into a Ca^{2+} heparinized (20 $U.ml^{-1}$) microcentrifuge tube (Sarstedt Ltd., Leicester, U.K.), 1.5 ml into a microcentrifuge tube (Sarstedt Ltd., Leicester, U.K.; Chapters 5, 7 and 8 only) 4.5 ml was dispensed into a potassium ethylenediamine tetraacetic acid (anticoagulant) coated (EDTA) tube and 4.5 ml into a serum tube. The heparinized microcentrifuge tube was then immediately centrifuged at 13000 rev for 3 min (Eppendorf-Anderman Centrifuge 5414, Germany), the plasma then drawn off and snap frozen in liquid nitrogen for later determination of plasma ammonia concentration. The 1.5 ml of blood in the remaining microcentrifuge tube was immediately analysed for blood glucose and lactate using a Yellow Springs Instruments automated analyser (YSI Incorporated, 2300 Stat Plus, U.S.A.; Chapters 5, 7 and 8 only). The EDTA tube was gently mixed and duplicate 20 μl volumes of blood were collected and stored, at -20°C, for analysis of lactate and of glucose should the automated analyser fail. Further small aliquots from the EDTA tube were used for determination of haematocrit and haemoglobin concentration. The remaining blood from the EDTA tube was then immediately centrifuged for 15 min at 6000 $rev.min^{-1}$ at ~3°C (Koolspin, Burkard Scientific Ltd., Uxbridge, U.K.). The plasma was then dispensed into microcentrifuge tubes (Sarstedt Ltd., Leicester, U.K.) and stored at -20°C for later analysis. The blood in the serum tube was left to coagulate for 60 min before it was centrifuged as described above. The serum was then stored at -70°C for later analysis. The plasma and serum were stored in aliquots of 0.7 to 1 ml to minimize any effects of freezing.

3.5.2. Blood sample analysis

Unless otherwise stated the analyses described here were carried out in the Biochemistry Laboratory of the Department of Physical Education, Sports Science and Recreation Management at Loughborough University. The hormone measurements were made in either the Genetics Laboratory of the Human Sciences Department (Chapters 4 and 5) or the Radiochemistry Department (Chapters 7 and 8) at Loughborough University.

In all studies haematocrit was determined in triplicate. Venous blood was collected in small bore tubes and these were then centrifuged for 15 min (micro-haematocrit centrifuge, Hawksely and Sons Ltd, Lancing, U.K.). The haematocrit portion of the blood sample was subsequently established using a micro-haematocrit reader (Hawksely and Sons Ltd., Lancing, U.K.; Appendix B) In addition, haemoglobin concentration was established spectrophotometrically (Digital Grating Spectrophotometer Series 2, Model CE393, Cecil Instruments Ltd, Cambridge, U.K.), in duplicate 20 μl volumes of blood using the cyanmethaemoglobin method (Appendix B). Changes in plasma volume (%) were estimated using the method of Dill and Costill (1974).

In the studies described in Chapters 4, 5 and 7, plasma ammonia concentration was determined spectrophotometrically (Digital Grating Spectrophotometer Series 2, Model CE393, Cecil Instruments Ltd, Cambridge, U.K.) using a commercially available kit (Sigma Diagnostics, Poole, U.K.; Appendix B). Analysis was carried out within 24 h of collection of the sample. Previous work (Tsintzas and Wilson, unpublished observations) showed that plasma ammonia concentrations in a sample frozen at -70°C were stable for at least 48 h after collection.

In the study described in Chapter 4, plasma lactate and glucose were determined by automated analyser (Cobas Mira, Roche Diagnostic Systems, Welwyn Garden City, U.K.). Commercially available kits were used for the analysis of both lactate and glucose (Boehringer Mannheim U.K. Ltd., Lewes, U.K.; Appendix B).

In Chapters 4, 5, 7 and 8 serum progesterone concentration was determined using a solid-phase [125]Iodine radioimmunoassay (Coat-A-Count 'Insulin', EURO / DPC Ltd., Caernarfon, U.K.)[*Appendix B*]. An automated gamma counter (Cobra II, Packard Instrument Company Inc., U.S.A.) was used to quantify the radioactivity.

In Chapter 4, serum growth hormone concentration, in Chapters 5, 7 and 8 serum cortisol and aldosterone concentration and in Chapter 8 serum prolactin concentration were measured in the same way (Coat-A-Count, EURO/DPC Ltd., Caernarfon, U.K.).

3.5.2.1. Coefficient of variations for blood sample assays

The coefficients of variation for the various blood sample assays used in this thesis were calculated using the following formula and are presented in Table 3.1. The coefficients of variation were calculated from 10 repeated measurements on a human blood, plasma or serum sample.

Coefficient of Variation (CV) = [(100*standard deviation)/sample mean] %

(Cohen and Holliday, 1982)

Table 3.1 Coefficients of variation for blood sample assays.

Metabolite	n	Mean	± Standard Deviation	Coefficient of Variation (%)
Plasma Ammonia	10	54.1 μmol.l^{-1}	0.03	3.3
Plasma Glucose	10	5.22 mmol.l^{-1}	0.05	0.9
Plasma Lactate	10	2.36 mmol.l^{-1}	0.01	0.4
Blood Glucose	10	4.74 mmol.l^{-1}	0.04	0.8
Blood Lactate	10	1.23 mmol.l^{-1}	0.01	0.8
Serum progesterone	10	8.3 nmol.l^{-1}	0.3	3.3
Serum growth hormone	10	18.9 mIU.l^{-1}	0.6	3.1
Serum aldosterone	10	354.0 pmol.l^{-1}	22.5	6.4
Serum cortisol	10	1240.2 nmol.l^{-1}	63.2	5.1
Serum prolactin	10	2142.7 nmol.l^{-1}	52.4	2.4

3.5.3. Pipette 'Calibration'

Prior to undertaking assays the pipettes used to dispense reagent and sample volumes (Gilson Pipetman [Gilson Medical Electronics, France], Gilson Microman [Gilson Medical Electronics, France] and Microlab Plus 1000 [Hamilton Bonaduz AG, Switzerland]) were 'calibrated' using the gravimetric method. Ten samples of distilled water were dispensed and measured using the particular pipettes. The mean, standard deviation and coefficient of variation were calculated, and corrections were made for the temperature of the water, which had been measured during the dispensing

procedure using a thermometer. Coefficients of variation were always less than 2.0 and 1.5 % for the reagent and sample volumes respectively.

3.6.   Statistical analyses

A variety of statistical procedures were used to analyse the results presented in the studies. One, two and three way analysis of variance, post-hoc Tukey test, correlated t-test and Pearson product moment correlation coefficient were among the procedures used to establish if any significant differences existed between subject response (in terms of a variety of physiological and metabolic parameters) to the performance of the LIST and/or field hockey skill test under the various experimental conditions. A more detailed description of the procedures used in each study is given in the appropriate chapter. Data are presented as means and standard error of the mean (SEM), and are based on the subject populations stated.

4. Effect of the menstrual cycle on performance of intermittent, high intensity shuttle running in a hot environment

4.1. Introduction

Increasing interest in female sport performance at both recreational and elite levels has resulted in a proliferation of studies examining the effects of the menstrual cycle and oral contraceptives upon exercise performance. Cyclic variations in deep body temperature during the menstrual cycle may alter thermoregulatory responses when exercising in the heat, whilst increasing oral contraceptive usage makes this an area where increasing research is required. However, no previous study has investigated the effect of the menstrual cycle and oral contraceptive usage on high intensity intermittent exercise in the heat.

Previous studies examining the effect of the menstrual cycle performance and thermoregulatory and cardiovascular strain have shown differing results. Furthermore, there have been variable levels of control in relation to hormonal determination of the cycle phase. During the mid-luteal phase, cycling for 15 min at 70% $\dot{V}O_2$ max or 60 min at 65% $\dot{V}O_2$ max has been shown to provide a greater thermoregulatory and cardiovascular strain, as well as an increased perception of exercise stress, in comparison with the follicular phase (Hessemer and Bruck, 1985; Pivarnik et al., 1992). In contrast, during high intensity cycling (90% $\dot{V}O_2$ max), and prolonged running (70% $\dot{V}O_2$ max) time to exhaustion has been shown to be increased during the luteal phase compared with the follicular (Jurkowski et al., 1981; Reilly and Whitley, 1994). Furthermore performance of continuous (90% $\dot{V}O_2$ max) and progressive high intensity running was unaffected by menstrual cycle phase (Lebrun et al., 1995; Lynch and Nimmo, 1998).

Few studies have been completed in hot environments, with none undertaken using high intensity intermittent cycling or running as the exercise mode. The research that has been completed has focused upon prolonged submaximal cycling and walking in both unacclimatised (Horvath and Drinkwater, 1982; Stephenson and Kolka, 1988b; Stachenfeld et al., 2000) and acclimatised women (Carpenter and Nunneley, 1988). Deep body temperature has been shown to be higher during the luteal phase compared with the follicular (Carpenter and Nunneley, 1988; Stephenson and Kolka, 1988b) or unaffected by menstrual phase (Horvath and Drinkwater, 1982; Stachenfeld et al., 2000). However, performance per se has not been investigated during exercise in the heat.

The literature regarding the metabolic and hormonal responses to exercise during the phases of the menstrual cycle is also equivocal. During submaximal treadmill exercise (70% $\dot{V}O_2$ max), glucose concentrations have been shown to be higher during the luteal compared with the ovulatory phase, but not different to the follicular phase (Galliven et al., 1997). However, during cycling (42% $\dot{V}O_2$ max) and treadmill exercise (40, 60 and 80% $\dot{V}O_2$ max) the metabolic and hormonal responses were unaffected by the menstrual cycle (Bonen et al., 1983; Kanaley et al., 1992; Zderic et al., 2001). Glucose concentrations have also been reported to be lower during cycling (52 and 63% $\dot{V}O_2$ max) in the luteal phase (Lavoie et al., 1987; Zderic et al., 2001). The research into hormonal responses to exercise appears less confusing. Growth hormone responses to cycling (52%) and treadmill walking (40 and 80% $\dot{V}O_2$ max) have been reported to be higher during the luteal and ovulatory phases of the cycle compared with the follicular phase (Bonen et al., 1983; Hornum et al., 1997; Zderic et al., 2001). High growth hormone concentrations have been associated with the high estrogen concentrations during the luteal and ovulatory phases (Hornum et al., 1997).

The responses of oral contraceptive users to prolonged cycling (60% $\dot{V}O_2$ max) in a hot environment has been investigated (Martin and Buono, 1997; Stachenfeld et al., 2000;). Martin and Buono (1997) found that women had a higher heart rate and rectal

temperature during exercise when taking oral contraceptives compared with exercise during the week when the women were not taking oral contraceptives. In contrast, Stachenfeld et al. (2000) compared the responses to taking 2 different types of oral contraceptives (progestin only and progestin and estrogen). During exercise in the progestin only trial, oesophageal temperature and oesophasgeal sweating threshold were higher than when the women were taking the combined estrogen and progestin contraceptive. There were no differences in heart rate, stroke volume or cardiac output between the two contraceptive types. This limited research, suggests that oral contraceptives may alter thermoregulatory responses to exercise, which could affect performance, the extent of which is dependent upon the type of oral contraceptive.

The metabolic and hormonal responses of oral contraceptive users to exercise have only been studied in moderate environmental conditions. This research has compared the metabolic responses of oral contraceptive users with eumennorheic women.

During moderate and high intensity treadmill exercise (41, 50 and 85% $\dot{V}O_2$ max), oral contraceptive users had higher growth hormone and lower glucose concentrations than eumennorheic women (Bonen et al., 1991; Bemben et al., 1992). The lower glucose concentration has also been associated with a decrease in carbohydrate utilisation, which may impact on exercise capacity during endurance exercise (Bemben et al., 1992). There remains a dearth of literature regarding the metabolic and hormonal responses of oral contraceptive users across menstrual cycle phase.

The weight of available evidence suggests that exercise performance in the heat is unaffected by menstrual cycle phase, whereas in moderate environmental conditions the research is equivocal. However, the growth hormone responses are expected to be higher in the luteal phase. The impact of oral contraceptive use on performance in phases of the cycle is unknown. Therefore, the purpose of the present study was to test the hypothesis that the performance of high intensity intermittent exercise in the heat is unaffected by menstrual cycle and oral contraceptive use.

4.2. Methods

4.2.1. Subjects

Fifteen well-trained female games players volunteered for the study, seven of whom had normal menstrual cycles (NM) lasting between 24 and 30 days, and eight who had been taking monophasic oral contraceptives (OC) for an average of 22 ± 6 months. The physical characteristics of the NM and OC groups are presented in Table 4.1. All subjects gave their written informed consent and the Loughborough University Ethical Committee approved the study. The oral contraceptives taken by the subjects are outlined in Table 4.2.

Table 4.1 Physical characteristics of the normal menstruating group (NM) and oral contraceptive users (OC) who participated in the study (mean ± SEM).

	NM	OC
Age (years)	20.3 ± 0.3	20.2 ± 0.4
Mass (kg)	62.1 ± 2.3	59.8 ± 1.1
Height (cm)	168.4 ± 2.0	165.6 ± 1.9
Estimated $\dot{V}O_2$ max ($ml.kg^{-1}.min^{-1}$)	51.1 ± 0.7	50.3 ± 1.6

Table 4.2 The type and active ingredients of the oral contraceptives.

Oral contraceptive	No.	Synthetic estrogens	Synthetic progesterones
Microgynon® 30	5	30 µg ethinylestradiol	150 µg levonorgestrel
Ovranette®	1	30 µg ethinylestradiol	250 µg levonorgestrel
Loestrin®	1	20 µg ethinylestradiol	1 mg norethisterone acetate
Dianette®	1	35 µg ethinylestradiol	2 mg cyproterone acetate

4.2.2. Experimental design

Subjects performed the Loughborough Intermittent Shuttle Test (LIST; Nicholas et al., 1995, 2000) in hot environmental conditions (~30°C), during the follicular phase, ~7 days after the onset of menstruation and in the luteal phase, ~ day 21. The order of trials was randomly assigned and 14 days elapsed between the follicular and luteal trials. Subjects exercised over a 20 m distance and repeated a walk, sprint, cruise

(~95% $\dot{V}O_2$ max) and jog (~55% $\dot{V}O_2$ max) pattern of exercise until 11 sprints had been completed. The 11 sprints took approximately 15 min and were followed by a 3 min rest period. This series of activities constituted 1 Set of the LIST. This pattern was repeated until exhaustion, or rectal temperature reached 39.5°C.

4.2.3. Preliminary measurements

Maximal oxygen uptake ($\dot{V}O_2$ max) was estimated using the progressive multistage fitness test (Ramsbottom et al., 1988). From this estimate of $\dot{V}O_2$ max, running speeds to elicit 95 and 55% $\dot{V}O_2$ max were determined using the tables for predicted $\dot{V}O_2$ max values (Ramsbottom et al., 1988). Subjects were then familiarised with the LIST at ~30°C for 2 Sets or ~30 min.

4.2.4. Main trials

Subjects reported to the laboratory at least 12 h after their last meal. In the 2 days prior to each trial subjects were encouraged to consume the same diet. All experiments were arranged so that each individual ran at the same time of day for both the follicular and luteal trials to control for circadian influences. Nude body mass was recorded and a 45 mm cannula (Venflon 2, BOC Ohemeda AB) was then inserted into a forearm vein of the subject under local anaesthetic (Lignocaine hydrochloride 1% w/v, Antigen pharmaceuticals Ltd.). The cannula was kept patent with saline solution (Sodium choloride 0.9% w/v, Antigen pharmaceuticals Ltd.). A rectal probe (Edale Instruments Ltd.) was inserted to a depth of 10 cm beyond the anal sphincter.

Fifteen minutes after cannulation, during which time subjects remained standing, a 'resting' blood sample was collected. Subjects then moved into the gymnasium and a resting rectal temperature was recorded. A standardised warm-up of ~15 min was performed which consisted of jogging, stretching and faster pace running. During the warm up and throughout the exercise period subjects were encouraged to drink water to ensure adequate hydration levels.

During each trial investigators ensured that subjects placed at least one foot on or over the lines delimiting the 20 m distance at each end of the gymnasium. Subjects were able to gauge their required running speeds by following an amplified audio signal generated from a microcomputer (BBC). During the sprints subjects were verbally encouraged to perform maximally. Sprint times over 15 m were measured using 2 infra-red photo electric cells (RS Components Ltd.) connected to the microcomputer.

A whirling hygrometer (Brannan Thermometers Ltd.) was used to measure atmospheric dry and wet bulb temperatures. Heart rate was continuously monitored throughout each test using short range telemetry (Sport Tester™, PE3000, Polar Electro Fitness Technology, sampling frequency 15 s). Rating of perceived exertion, thirst and thermal comfort were recorded prior to the 11th sprint in each exercise set using the Borg scale (1962). A 12 ml blood sample was collected from each subject between the sets of exercise and at exhaustion. Rectal temperatures were measured during the 4th and 8th cycle of each set and in the 3 min blood sampling period between sets of LIST. When rectal temperatures were measured subjects were stationary for 40 m of the 60 m walk in that cycle.

4.2.5. Blood sampling and analysis

Five ml of blood was dispensed into an EDTA tube and aliquots from the venous sample were used for determination of haematocrit and haemoglobin concentration (by microcentrifugation and the cyanmethaemoglobin method respectively). Changes in plasma volume (%) were estimated using the method of Dill and Costill (1974). One ml of blood was dispensed immediately into a calcium-heparin tube, centrifuged for 3 min at 13,000 rev.min^{-1} and the plasma frozen at -70°C. Ammonia concentration was determined within 24 h using a commercially available kit (Sigma Diagnostics). The remaining blood was centrifuged for 15 min at 6000 rev.min^{-1} at ~3°C. The resulting plasma was then stored at -20°C for subsequent determination of lactate and glucose using fully automated colorimetric instrumentation (Cobas Mira, Roche Products Ltd.).

Five ml of blood was also dispensed into a serum tube for determination of progesterone and growth hormone concentration by using a commercially available radio immunoassay kits (Diagnostic Products Corporation).

4.2.6. Statistical analyses

The physiological and blood responses to the performance of the LIST were analysed using a three-way analysis of variance (ANOVA; group x trial x time) with repeated measures on two factors (trial x time). Environmental temperatures, distance covered during the LIST, body mass and plasma volume responses during the main trials were analysed using a two-way ANOVA (group x trial) with repeated measures on one factor (trial). Significant differences between means were identified using a Scheffè post-hoc test. Data are presented as means ± standard error of the mean (SEM) and are based on a subject population of 7 NM and 8 OC unless otherwise stated. The follicular trial and luteal trial are abbreviated in the results with FT and LT respectively.

4.3. Results

4.3.1. Environmental conditions

Dry bulb temperatures averaged ~ 31°C throughout the trials and were controlled so that there was no difference between the trials or groups (NM: FT vs LT, 31.0 ± 0.3 vs 30.7 ± 0.7°C; interaction group - trial, n.s.; OC: FT vs LT, 31.1 ± 0.2 vs 31.0 ± 0.2°C; interaction group - trial, n.s.). Relative humidity was not different between the groups and was well maintained during the trials (NM: FT vs LT, 21.7 ± 2.2 vs 24.7 ± 1.6%; main effect trial, n.s.; OC: FT vs LT, 23.0 ± 1.6 vs 23.0 ± 2.2%; main effect trial, n.s.).

4.3.2. Performance

The distance completed by the NM and OC was not significantly different between phases (Figure 4.1; FT vs LT, 5869 ± 2896 vs 6238 ± 2648 m). The overall distance completed did not differ between groups (NM vs OC, 6059 ± 896 vs 6048 ± 543 m). Similarly, the exercise time showed no difference between phases of the menstrual cycle or group. Maximal 15 m sprint performance did not differ between groups and

cycle or group. Maximal 15 m sprint performance did not differ between groups and the decline in performance was similar both between groups and between trials (main effect time $P<0.01$, $F_{2, 26} = 27.3$; Figure 4.2; NM: FT vs LT, 2.68 ± 0.03 to 2.80 ± 0.04 vs 2.73 ± 0.04 to 2.83 ± 0.06 s; OC: FT vs LT, 2.78 ± 0.08 to 2.92 ± 0.07 vs 2.79 ± 0.07 to 2.98 ± 0.07 s).
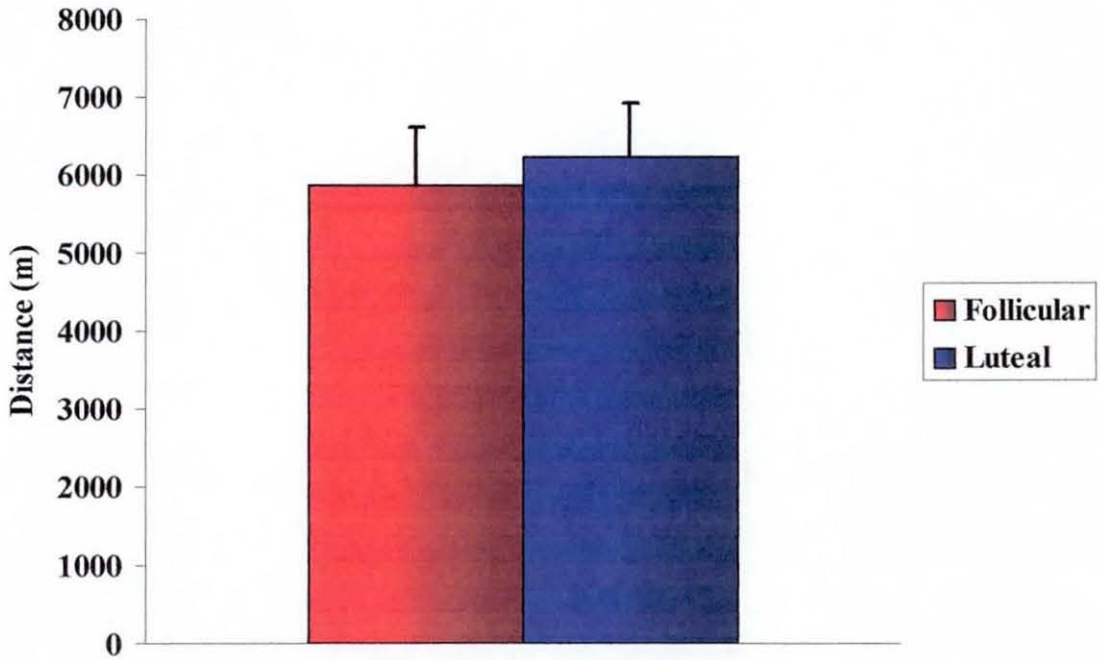
Figure 4.1 Distance completed during the FT and LT for the NM and OC together.
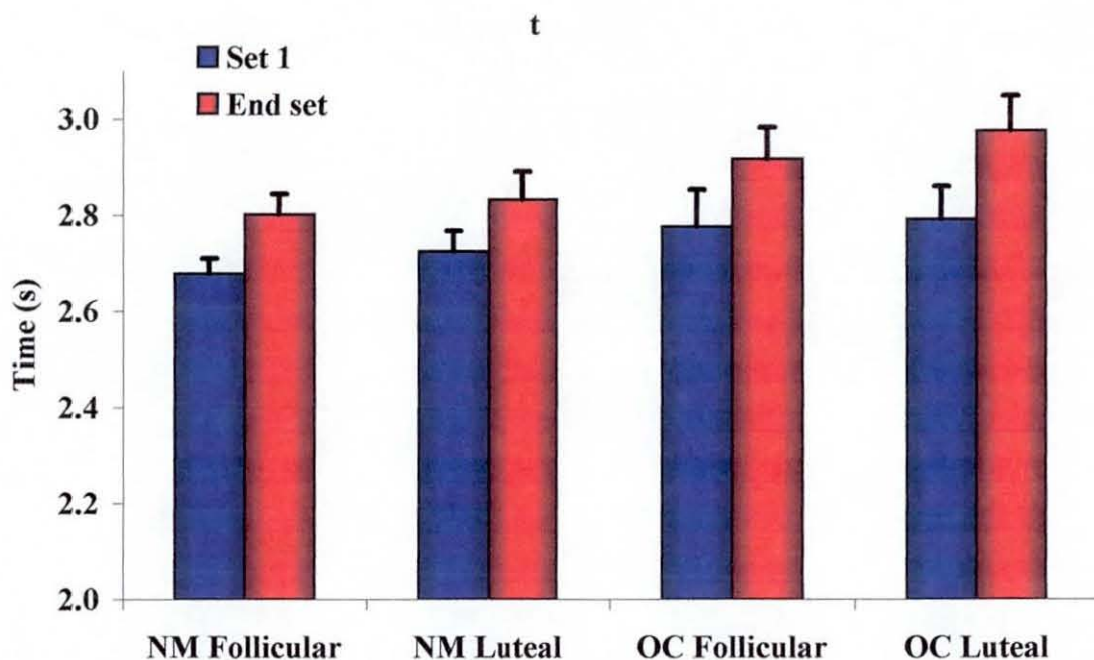
Figure 4.2 Maximal 15m sprint times during the first and exhaustion sets of the LIST; t = main effect time P<0.05, $F_{2, 26}$ = 27.3.

4.3.3. Rectal temperature

Resting rectal temperature was higher for the OC than the NM being $37.36 \pm 0.05°C$ and $37.16 \pm 0.07°C$ respectively (main effect group P<0.05, $F_{1, 13}$ = 8.3). The NM resting temperatures were higher for five of the seven subjects prior to the LT, though this was not significant for the group as a whole (Rest, FT vs LT, 37.1 ± 0.1 vs $37.2 \pm 0.1°C$, n.s.). The difference between the OC and NM was not maintained after the onset of exercise, as rectal temperatures were similar during all four trials. The rate of rise of rectal temperature showed no differences between the two groups (main effect group n.s., $F_{1, 13}$ = 1.8; NM vs OC, 3.4 ± 0.4 vs $2.6 \pm 0.2 °C.h^{-1}$).

4.3.4. Heart rate and rating of perceived exertion

Heart rate (Table 4.3) and rating of perceived exertion (Table 4.4) increased with exercise time and was not different between menstrual phase or oral contraceptive use (main effect time P<0.01, heart rate $F_{2, 14}$ = 11.1, RPE $F_{1, 13}$ = 42.6).

Table 4.3 Mean heart rate (beats.min^{-1}) of the NM and OC during the first set and exhaustion sets of the LIST; t = main effect time P<0.01, $F_{2, 14}$ = 11.1.

	NM		OC	
	Set 1	End set	Set 1	End set
FT	169 ± 5	171 ± 4	171 ± 1	178 ± 3
LT	171 ± 6	180 ± 3	173 ± 1	178 ± 2
		t		

Table 4.4 Mean rating of perceived exertion (RPE) of the NM and OC during the first set and exhaustion sets of the LIST; t = main effect time P<0.01, $F_{1, 13}$ = 42.6.

	NM		OC	
	Set 1	End set	Set 1	End set
FT	13 ± 1	17 ± 1	14 ± 1	17 ± 1
LT	13 ± 1	18 ± 1	13 ± 1	18 ± 1
		t		

4.3.5. Body mass, fluid consumption and estimated sweat rate

Body mass was well maintained by both groups during the FT and LT and was not different with oral contraceptive usage. The change in body mass as a percentage of basal body mass for the NM was –0.13 ± 0.24 and 0.06 ± 0.16% for the FT and LT respectively and for the OC was –0.51 ± 0.10 (FT) and –0.25 ± 0.22% (LT). Ad libitum fluid consumption was not different between the groups or between the menstrual cycle phases (NM: FT vs LT 19.6 ± 4.4 vs 17.1 ± 2.6 ml.kg^{-1}.h^{-1}; OC: FT vs LT, 16.1 ± 1.65 vs 16.8 ± 2.6 ml.kg^{-1}.h^{-1}). However, estimated sweat rate was higher during the follicular phase of the menstrual cycle being 1.4 ± 0.1 l.h^{-1}, whereas during the luteal phase it was 1.1 ± 0.1 l.h^{-1} (main effect phase P<0.05, $F_{1, 13}$ = 5.7). There was no difference between the two groups.

4.3.6. Hormonal responses

Figure 4.3 indicates that the resting serum progesterone concentrations for the NM were greater for the LT than the FT (interaction group x phase $P<0.05$, $F_{1, 11} = 5.0$). Similar resting progesterone concentrations were seen during the two trials for the OC. Serum growth hormone concentrations were not different between the groups but were higher for the luteal phase than the follicular phase (main effect phase $P<0.05$, $F_{1, 11} = 8.5$; Figure 4.4). However, the concentrations were not different between menstrual phases for the NM and OC groups separately.
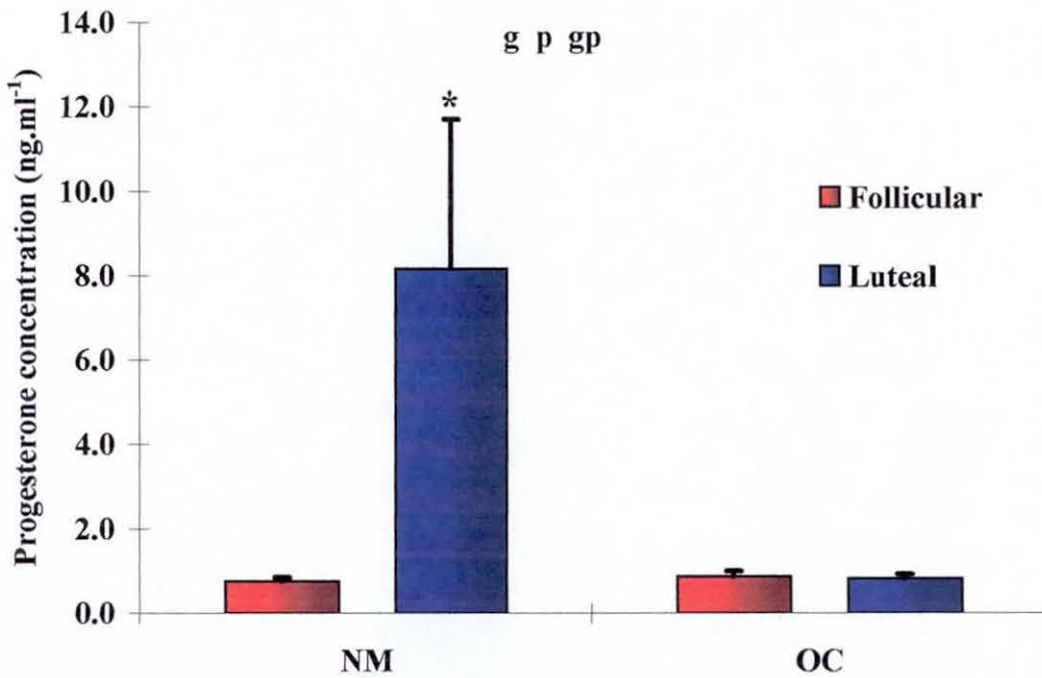
Figure 4.3 Resting progesterone concentrations for the NM and OC; g = main effect group $P<0.05$, $F_{1, 11} = 5.1$; p = main effect phase $P<0.05$, $F_{1, 11} = 4.9$; gp = interaction group x phase $P<0.05$, $F_{1, 11} = 5.0$, * post –hoc $P<0.05$ from NM FT.
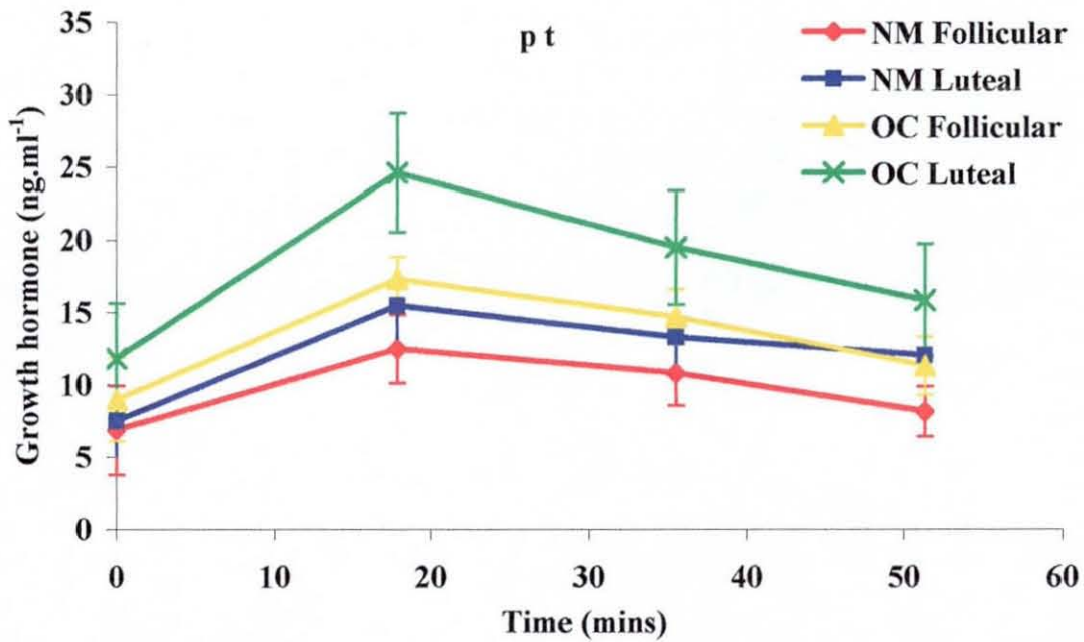
Figure 4.4 Growth hormone concentrations for the NM and OC during the follicular and luteal trials; p = main effect phase $P<0.05$, $F_{1, 11} = 8.5$; t = main effect time $P<0.01$, $F_{3, 33} = 11.4$.

4.3.7. Metabolic Responses

Both plasma lactate and ammonia responses did not differ between groups, with a similar response in both trials (main effect time $P<0.01$, lactate $F_{3, 36} = 78.1$, ammonia $F_{3, 36} = 24.3$; Table 4.5).

Figure 4.5 discloses that during the exercise period plasma glucose concentrations were greater during the follicular trial than the luteal trial, but did not differ between groups (main effect phase $P<0.01$, $F_{1, 12} = 9.5$; main effect time $P<0.01$, $F_{3, 36} = 34.2$). Thus end concentrations for FT were 9.56 ± 1.19 mmol.l^{-1} compared to 8.04 ± 0.65 mmol.l^{-1} for the LT (interaction phase x time, $P<0.01$, $F_{3, 36} = 4.5$).

Table 4.5 Plasma lactate and ammonia concentrations for NM and OC at rest and exhaustion; t = main effect time P<0.05, lactate $F_{3, 36}$ = 78.1, ammonia $F_{3, 36}$ = 24.3.

	NM		OC	
	Rest	End	Rest	End
Plasma lactate concentration (mmol.l^{-1})				
FT	1.14 ± 0.12	5.26 ± 0.72	1.16 ± 0.15	5.74 ± 0.82
LT	0.92 ± 0.08	5.40 ± 0.92	1.13 ± 0.12	5.83 ± 0.97
		t		
Plasma ammonia concentration (µmol.l^{-1})				
FT	16.0 ± 4.0	45.6 ± 8.0	20.7 ± 5.1	42.0 ± 10.2
LT	13.5 ± 2.2	56.5 ± 10.8	17.1 ± 5.9	49.9 ± 6.5
		t		
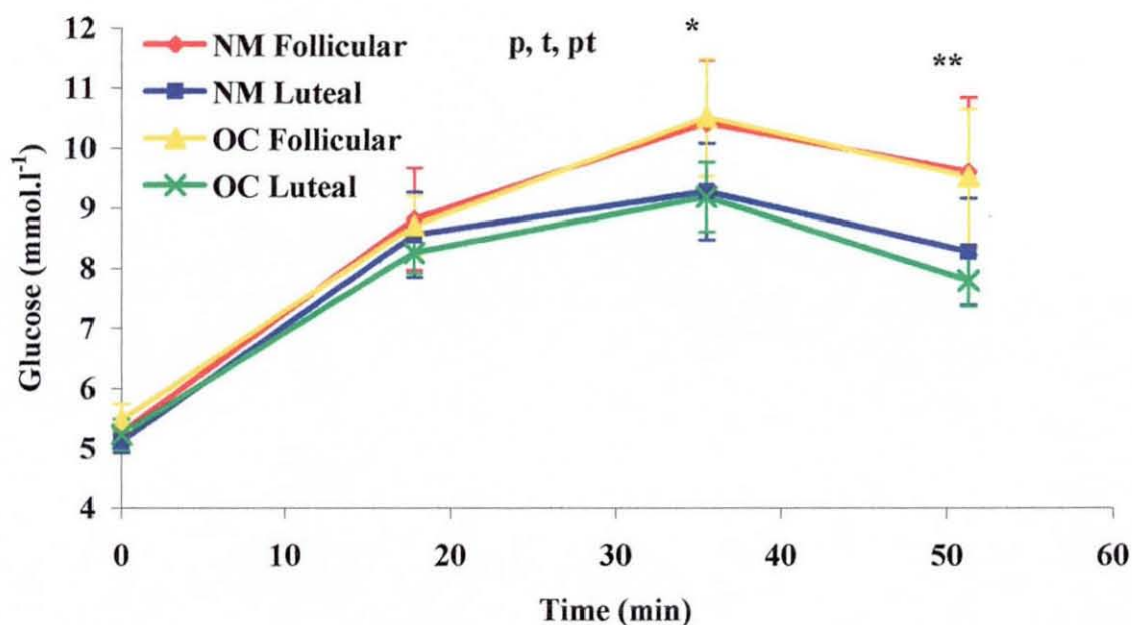
Figure 4.5 Plasma glucose concentrations for the NM and OC during the follicular and luteal trials; p = main effect phase $P<0.01$, $F_{1, 12} = 9.5$; t = main effect time $P<0.01$, $F_{3, 36} = 34.2$; pt = interaction phase x time $P<0.01$, $F_{3, 36} = 4.5$, * post–hoc $P<0.05$ FT from LT, ** post–hoc $P<0.01$ FT from LT.

4.3.8. Plasma volume

Estimated resting plasma volume was not different between the OC and NM groups. However resting plasma volume was higher during the luteal phase, (FT vs LT, 61.6 ± 1.1 vs 62.7 ± 1.3 ml.100ml^{-1}, main effect phase $P<0.05$, $F_{1, 12} = 5.9$) and higher for the OC in the luteal phase (FT vs LT, 61.0 ± 1.1 vs 63.4 ± 1.3 ml, interaction group x phase $P<0.01$, $F_{1, 12} = 11.1$). The estimated change in plasma volume for the NM was not different between trials (FT vs LT, -1.7 ± 1.5 vs $-1.04 \pm 1.9\%$; n.s.). Similarly the OC showed no difference in plasma volume changes between trials (FT vs LT, -3.3 ± 1.6 vs $-5.2 \pm 1.5\%$; n.s.). The decreases in plasma volume were not different between the two groups.

4.4. Discussion

The main finding in the present study was that in unacclimatised women, who were eumenorrheic or monophasic oral contraceptives users, performance, in terms of distance run, of prolonged, intermittent, high-intensity running was not significantly affected by the cyclic variations in hormones associated with normal menstrual

function or exogenous synthetic hormones. Maximal sprint performance over 15 m, heart rate, plasma lactate and ammonia responses were also not different between the luteal and follicular phases of the menstrual cycle or with oral contraceptive use. However, plasma glucose concentration was higher and serum growth hormone concentrations were lower in the follicular in comparison with the luteal phase.

It has been found previously in a number of studies (Wells and Horvath, 1974; Horvath and Drinkwater, 1982) that the performance and responses to exercise in the heat in unacclimatised eumenorrheic women who were not using oral contraceptives is unaffected by the different phases of the menstrual cycle, which is consistent with the findings in the present study. Horvath and Drinkwater (1982) reported that during walking in three different environments (28, 35 and 48°C) during the flow (follicular), ovulatory and luteal phases no differences in heart rate, core temperature, sweat rate or performance of the 50 min walk were observed between the three phases. Previous research investigating combined monophasic oral contraceptive use in women exercising in hot environmental conditions has found contradictory thermoregulatory and cardiovascular responses. Martin and Buono (1997) found that the strain on the

thermoregulatory and cardiovascular systems during cycling for 1 h at 60% $\dot{V}O_2$ max (as indicated by rectal temperature and heart rate) was greater when subjects were taking oral contraceptives. It was suggested that this might result in reduced capacity or performance in oral contraceptive users, although this was not investigated in the study. In agreement with the current study, Stachenfeld and co-wokers (2000) found no difference in oesophageal temperature and heart rate responses between the follicular phase, luteal phase and with combined contraceptive use during cycling (40

min 60% $\dot{V}O_2$ max). A possible explanation for the different findings in these studies is the time of the testing in relation to taking the oral contraceptive. In the current study, to allow for group comparisons, subjects were tested between days 6-10 after the onset of menses, so that subjects would have been taking the oral contraceptive for 1-5 days, whereas in the study of Martin and Buono (1997) testing was undertaken in the no oral contraceptive week. However, Stachenfeld et al. (2000) compared acute pill usage, rather than chronic usage, with the normal menstrual cycle.

In acclimatised subjects, however, responses of eumennorheic women to exercise in the heat (48°C, 10% RH) have been shown to differ, with a higher rectal temperature recorded in the luteal phase compared with the follicular. However, this difference was not of a magnitude to alter the performance of prolonged cycling (30% $\dot{V}O_2$ max; Carpenter and Nunneley, 1988). A higher deep body temperature during the luteal phase of the menstrual cycle was also apparent when exercising at night (03:00 – 04:00 hours), when the largest differences in deep body temperature between the luteal and follicular phase are observed. Prior to exercise a 4 h rest period was completed in the heat (30 - 32°C). During the cycling, which was in moderate conditions, rectal temperature, heart rate, sweating thresholds and oxygen uptake were all greater during the luteal phase (Hessemer and Brück, 1985). In the current study, the overall response of the unacclimatised subjects to the exercise in a hot environment, suggests that the cyclic variations that are observed via a higher deep body temperature during the luteal phase of the menstrual cycle and with monophasic oral contraceptive use at rest and in acclimatised subjects are negated by the heat stress and the intensity of the exercise.

In the current study, there were no differences in rectal temperature at rest, prior to the warm up or during exercise for the eumennorheic group. However, at rest rectal temperature was higher for the oral contraceptive users, than their eumennorheic counterparts. The progestin concentration in the oral contraceptives was high (>150 µg) and therefore the increase in basal body temperature, in line with progestin concentration, was not of a gradual nature as seen in the menstrual cycle or in multiphasic oral contraceptive preparations. Furthermore the potency of exogenous progestins has been shown to be greater than their endogenous counterparts (Bemben, 1993). Thus exogenous progestins have a significant effect upon thermoregulation (Grucza et al., 1993; Rogers and Baker, 1997). Both the high progestin concentration and potency of the oral contraceptives seem the likely explanation for the higher resting rectal temperatures observed. For the eumennorheic women, the lack of a difference in deep body temperature at rest, prior to the warm up, is surprising as the progesterone levels confirmed that subjects were in the luteal phase and thus an increase in deep body temperature might have been expected. However, Wells and Horvath (1973) concluded that intervening variables associated with walking to the

laboratory and long preparation period prior to testing resulted in high individual variability and no difference in rectal temperature prior to testing. In the current study a long preparation time before testing was undertaken and therefore it seems clear that the lack of difference prior to the warm up period was due to this preparation period and its effect upon the subjects.

During the exercise period in the present study, in agreement with Stachenfeld et al. (2000), Horvath and Drinkwater (1982) and Wells and Horvath (1974), no differences in deep body temperature were seen between the phases of the menstrual cycle for the eumenorrheic women. This finding is in contrast to a study conducted at night (Hessemer and Brück, 1985) and studies using acclimatised individuals (Carpenter and Nunneley, 1988; Pivarnik et al., 1992). The study at night was primarily conducted to show the large differences in deep body temperature between the luteal and follicular phases and the cycling exercise period only lasted 15 min at a moderate intensity (70% $\dot{V}O_2$ max). This may not have placed the subjects under enough thermal strain to increase the follicular deep body temperature to a similar level as the luteal temperature. In the acclimatised individuals, exercise in the heat was of a low intensity (30% $\dot{V}O_2$ max) and moderate exercise (65% $\dot{V}O_2$ max) was completed in moderate conditions and thus would not have stressed the subjects to an extent where the rectal temperature differences between phases were overcome (Carpenter and Nunneley, 1988; Pivarnik et al., 1992). However in the present study using unacclimatised female subjects partaking in high intensity intermittent exercise, rectal temperature was not different throughout the exercise period. Furthermore, rectal temperature changes during the menstrual cycle for oral contraceptive users are more uniform (Grucza et al., 1993). Though no differences were seen at rest for the eumenorrheic individuals, it is suggested that the heat and exercise stress combined to negate any differences related to the menstrual cycle phase or oral contraceptive use during exercise. This suggestion is supported by Horvath and Drinkwater (1982) who did find a difference in deep body temperature at rest that was not maintained during low intensity exercise due to the heat stress on the unacclimatised subjects.

There were no differences in plasma lactate and plasma ammonia responses between the normal menstruating group and oral contraceptive group and no difference during the menstrual phases. Plasma lactate and ammonia responses of the subjects during the menstrual cycle for eumenorrheic and oral contraceptive users has been shown not to vary with menstrual cycle phase in numerous studies (De Souza et al., 1990; Bonen et al., 1991; Bemben et al., 1992; Galliven et al., 1997). The findings in the current study are in agreement with these previous investigations.

There was a higher plasma glucose concentration during the high intensity intermittent running in the follicular phase in the present study, although there was no difference between groups. Previous studies examining glucose concentration, menstrual cycle phase and oral contraceptive use have yielded equivocal findings with increases, decreases and no change being recorded (Bonen et al., 1991; Kanaley et al., 1992; Bemben, 1993; Galliven et al., 1997; Bailey et al., 2000). These equivocal findings might be related to the intensity and type of exercise, the type of oral contraceptive and whether the subjects are fasted. When the metabolic contribution required from gluconeogenesis is high there tends to be a higher glucose concentration eumennorheic and oral contraceptive users during the luteal phase of the menstrual cycle (Lavoie et al., 1987; Bemben, 1993). Thus in the current study, the differences in glucose concentrations became significant towards the end of the exercise period. The lower glucose concentration during the luteal phase may be related to the higher endogenous and exogenous ovarian hormones that may impair hepatic gluconeogenesis (Lavoie et al., 1987). The higher growth hormone concentration during the luteal phase may also contribute to the lower glucose concentration during this phase. High estrogen concentrations stimulate growth hormone release, which in turn decreases peripheral glucose uptake and utilisation, and thereby decreases the gluconeogenic contribution to blood glucose concentrations (Bemben 1993). These metabolic and hormonal differences between the two phases did not result in any significant differences in performance between phases or groups.

In summary, menstrual cycle phase and/or oral contraceptive use did not affect performance in terms of distance run or 15 m maximal sprint time during prolonged intermittent high intensity shuttle running. However, plasma glucose was higher and

serum growth hormone lower in the follicular phase in comparison with the luteal phase and these differences need to be considered in future research.

5. Effect of heat acclimation on performance of intermittent, high intensity shuttle running in a hot environment

5.1. Introduction

Heat acclimation has been well documented to prolong the time to exhaustion during endurance exercise in the heat (Nielsen et al., 1993). However, limited research has been completed regarding intermittent activity and specifically prolonged high intensity intermittent running. Furthermore, many acclimation studies have not included an equivalent training group in moderate conditions and/or a control group which makes it difficult to determine the extent of the acclimation adaptations compared with the training adaptations per se. The acclimation vs training issue is further compounded by the training status of the subjects. The adaptations following acclimation vary depending upon the training status of subjects. Well-trained individuals may show a lower deep body temperature and heart rate following acclimation, whereas untrained individuals show a greater range of adaptations including a lower deep body temperature, skin temperature, heart rate and higher aldosterone concentrations, sweat rate and plasma volume (Shvartz et al., 1977; Fortney et al., 1979 and Houmard et al., 1990). Thus, the acclimation adaptations are very specific to the training status of the group.

The majority of previous studies have investigated 7 or more consecutive days of acclimation. Nielsen et al. (1993) reported an increase in exercise capacity of 67% following 7-10 days of cycling at 60% $\dot{V}O_2$ max (40-42°C) to exhaustion each day. This increase in exercise capacity was attributed to a decrease in deep body temperature after acclimation (Nielsen et al., 1993). Febbraio et al. (1994b) also used cycling as the mode for both acclimation (~50% $\dot{V}O_2$ max, 90 min) and the pre- and post- acclimation tests (70% $\dot{V}O_2$ max, 40 min). Following acclimation, there was a lower rectal temperature, muscle temperature and heart rate. Clearly, a prolonged period of acclimation, both in terms of duration of sessions and number of consecutive days can attenuate thermoregulatory responses to exercise and heat stress. However, there appears to be very little literature, if any, that has used less than 45

min per acclimation session or less than 5 total sessions. The long acclimation periods may be difficult to adopt for games players who will need to practice on the pitch as well as acclimate before competitions in hot climates. They may also have match commitments that would not allow a long consecutive period of acclimation.

The two studies outlined above, provide an example of the majority of research that has been completed into heat acclimation. All testing is undertaken in a climatic chamber and incorporates continuous cycling or treadmill running. Though some acclimation protocols have incorporated intermittent exercise, this has not been of the nature that characterises the multiple sprint sports. High intensity intermittent exercise in the heat (35°C) has previously been demonstrated to provide a greater thermal strain than continuous exercise and may therefore be a more powerful stimulus for acclimation (Nevill et al., 1995).

Though numerous acclimation studies have incorporated women, from the information presented in papers, it appears that only 3 studies have recruited well-trained women as subjects (Avellini et al., 1980; Frye and Kamon, 1983; Armstrong et al., 1987). Armstrong et al. (1987) used 1 female in a group of 5, to investigate the effects of chronic heat acclimation on performance of treadmill running (30°C, 35% RH). The group were discussed as a whole, so no inferences can be made about the responses of the female athlete. Frye and Kamon (1983) compared the sweating responses to low intensity walking (25-30% $\dot{V}O_2$ max) after acclimation of 4 women with 4 men in hot dry (37°C, 30°C) and hot humid (48°C, 25°C) conditions. The women had lower sweat rates than the men in humid heat, but there were no sweat differences in the hot dry condition. The women also had a greater sweating efficiency, in the humid environment than the men. Similarly, Avellini et al. (1980) compared the responses to 3 h walking (~30% $\dot{V}O_2$ max) of 4 men and women to 10 days acclimation (36°C, 65%). The heart rate and rectal temperature responses after acclimation were similar between sexes until 90 min, but by the end of exercise the men had a higher heart rate and rectal temperature than the women. However, the decrease in rectal temperature and heart rate post-acclimation from pre-acclimation was similar between groups. Sweat rate was increased more by acclimation in the men than the women. As with research that has compared the responses to acclimation of

untrained women and men (Horstman and Christensen, 1982), Avellini et al. (1980) concluded that men and women respond similarly to acclimation.

Overall it seems that men and women respond in a similar way to acclimation and that intermittent exercise provides a greater thermal and physiological strain than continuous exercise. Thus, the purpose of the present study was to test the hypothesis that 4 high intensity intermittent acclimation sessions improve the performance of well-trained female games players during intermittent, high intensity shuttle running in a hot environment and the mechanism of adaptation is a decrease in body temperature.

5.2. Methods

5.2.1. Subjects

Seventeen well-trained female games players volunteered for the study, thirteen of whom had normal menstrual cycles, and four who were taking monophasic oral contraceptives. The subjects were divided into 3 groups, (Acclimation, n = 6, trained in a hot environment; Training, n = 6, trained in a moderate environment; Control, n = 5, pre- and post-acclimation trials only, no training). The physical characteristics of the acclimation, moderate training and control groups are presented in Table 5.1. All subjects gave their written informed consent and Loughborough University Ethical Committee approved the study.

Table 5.1 Physical characteristics of the acclimation, training and control groups who participated in the study (mean ± SEM).

	Acclimation	Training	Control
Age (years)	20.1 ± 0.6	20.3 ± 0.8	21.3 ± 0.9
Mass (kg)	68.5 ± 3.1	63.2 ± 4.4	66.1 ± 4.3
Height (cm)	169.4 ± 3.0	165.3 ± 3.8	165.2 ± 1.4
Estimated $\dot{V}O_2$ max (ml.kg^{-1}.min^{-1})	49.7 ± 2.2	49.3 ± 1.8	49.1 ± 1.8

5.2.2. Experimental design

Subjects performed the Loughborough Intermittent Shuttle Test (LIST; Nicholas et al., 1995, 2000) in hot environmental conditions (~30°C), on two occasions approximately one month apart. Subjects exercised over a 20 m distance and repeated a walk, sprint, cruise (~85% $\dot{V}O_2$ max) and jog (~50% $\dot{V}O_2$ max) pattern of exercise until 11 sprints had been completed. The 11 sprints took approximately 15 min and were followed by a 3 min rest period. This series of activities constituted 1 Set of LIST. This pattern was repeated until exhaustion, or until rectal temperature reached 40°C. The acclimation group and moderate training group also completed 4 training sessions, in hot and moderate conditions respectively, of 2 (30 min) or 3 (45 min) sets of the LIST. All training sessions (hot or moderate conditions) were completed in the ten days prior to the 2nd main trial (Figure 5.1).

Figure 5.1 Protocol of the 28 days of the acclimation study.

5.2.3. Preliminary measurements

Maximal oxygen uptake ($\dot{V}O_2$ max) was estimated using the multistage fitness test (Ramsbottom et al., 1988). From this estimate of $\dot{V}O_2$ max, running speeds to elicit 85 and 50% $\dot{V}O_2$ max were determined using the tables for predicted $\dot{V}O_2$ max values

(Ramsbottom et al., 1988). Subjects were then familiarised with the LIST at ~30°C for 2 Sets or ~30 min.

5.2.4. Main trials

Subjects reported to the laboratory at least 12 h after their last meal. In the 2 days prior to each trial subjects were encouraged to consume the same diet. All experiments were arranged so that each individual ran at the same time of day for both main trials to control for circadian influences. Nude body mass was recorded and a 45 mm cannula (Venflon 2, BOC Ohemeda AB) was then inserted into a forearm vein of the subject under local anaesthetic (Lignocaine hydrochloride 1% w/v, Antigen pharmaceuticals Ltd.). The cannula was kept patent with saline solution (Sodium choloride 0.9% w/v, Antigen pharmaceuticals Ltd.). A rectal probe (Edale Instruments Ltd.) was inserted to a depth of 10 cm beyond the anal sphincter.

Fifteen minutes after cannulation, during which time subjects remained standing, a 'resting' blood sample was collected. Subjects then moved into the gymnasium and a resting rectal temperature was recorded. A standardised warm-up of ~15 min was performed which consisted of jogging, stretching and faster pace running. During the warm up and throughout the exercise period subjects were encouraged to drink water to ensure adequate hydration levels.

A whirling hygrometer (Brannan Thermometers Ltd.) was used to measure atmospheric dry and wet bulb temperatures during the main trials and training sessions. Heart rate was monitored continuously throughout each main trial using short range telemetry (Sport Tester[TM] or Vantage NV, Polar Electro Fitness Technology, sampling frequency 15s). Rating of perceived exertion, thirst and thermal comfort were recorded prior to the 11th sprint in each exercise set. At the same time perceived heat and thirst were recorded using an integer scale. A 12 ml blood sample was collected from each subject between the sets of exercise and at exhaustion. Rectal temperatures were measured during the 4th and 8th cycle of each set and in the 3 min blood sampling period between sets of LIST. When rectal temperatures were measured subjects were stationary for 40 m of the 60 m walk in that cycle.

5.2.5. Blood sampling and analysis

Five ml of blood was dispensed into an EDTA tube and aliquots from the venous sample were used for determination of haematocrit and haemoglobin concentration. Changes in plasma volume (%) were estimated using the method of Dill and Costill (1974). One ml of blood was dispensed immediately into a calcium-heparin tube, centrifuged for 3 min at 13000 rev.min^{-1} and the plasma frozen at -70°C. Ammonia concentration was determined within 24 h using a commercially available kit (Sigma Diagnostics). A further 1.5 ml of blood was dispensed into an eppendorf tube for immediate determination of blood glucose and lactate using an automated analyser (Yellow Springs Instruments).

Five ml of blood was also dispensed into a serum tube for determination of progesterone, aldosterone and cortisol concentration by using commercially available radio immunoassay kits (Diagnostic Products Corporation).

5.2.6. Statistical analyses

The sprint times, physiological and blood responses to the performance of the LIST were analysed using a three-way analysis of variance (group x trial x time) with repeated measures on two factors (trial x time). Environmental temperatures, distance covered during the LIST, body mass and plasma volume responses during the main trials were analysed using a two-way ANOVA (group x trial) with repeated measures on one factor (trial). Data are presented as means ± standard error (SEM).

5.3. Results

5.3.1. Environmental conditions

Dry bulb temperatures averaged ~ 30°C throughout the main trials and were controlled so that there was no difference between the trials or groups (Hot: A vs B, 30.2 ± 0.5 vs 30.1 ± 0.6°C; Moderate: A vs B, 30.8 ± 0.1 vs 30.8 ± 0.1°C; Control: A vs B, 30.7 ± 0.2 vs 30.3 ± 0.3°C; main effect group P=0.49, main effect trial P=0.10). Relative humidity was not different between the groups and was well maintained during the main trials (Hot: A vs B, 25.5 ± 2.0 vs 24.6 ± 2.3%; Moderate: A vs B, 27.2 ± 2.4 vs 25.8 ± 1.9°C; Control: A vs B, 28.9 ± 3.5 vs 31.2 ± 4.1%; main effect

group P=0.36, main effect trial P=0.99). The dry bulb temperature during the training sessions was higher for the acclimation group than the moderate training group being 30.6 ± 0.2 and $18.1 \pm 1.1^\circ$C respectively (P<0.01). However, the relative humidity was higher during the moderate training sessions (hot vs moderate, 23.9 ± 1.3 vs $40.9 \pm 2.4\%$, P<0.01).

5.3.2. Performance

Heat acclimation resulted in a 33% improvement in distance run from trial A to B (7703 ± 1401 to 10215 ± 1746 m), with a very similar performance in trials A and B for the moderate training and control group (Figure 5.2; group x trial interaction P<0.05, $F_{2, 14} = 4.8$). The overall distance completed during trial A did not differ between the three groups. The exercise time during trial A was 68 ± 13, 76 ± 11 and 66 ± 7 min respectively for the acclimation, training and control groups. The time increased for the acclimation group only to 90 ± 15 min (interaction group x trial P<0.05, $F_{2, 14} = 5.0$). Maximal 15 m sprint performance did not differ between the two main trials or between the groups. Decline in sprint performance was similar in all trials (main effect time P<0.01, $F_{2, 28} = 23.5$; Table 5.2).
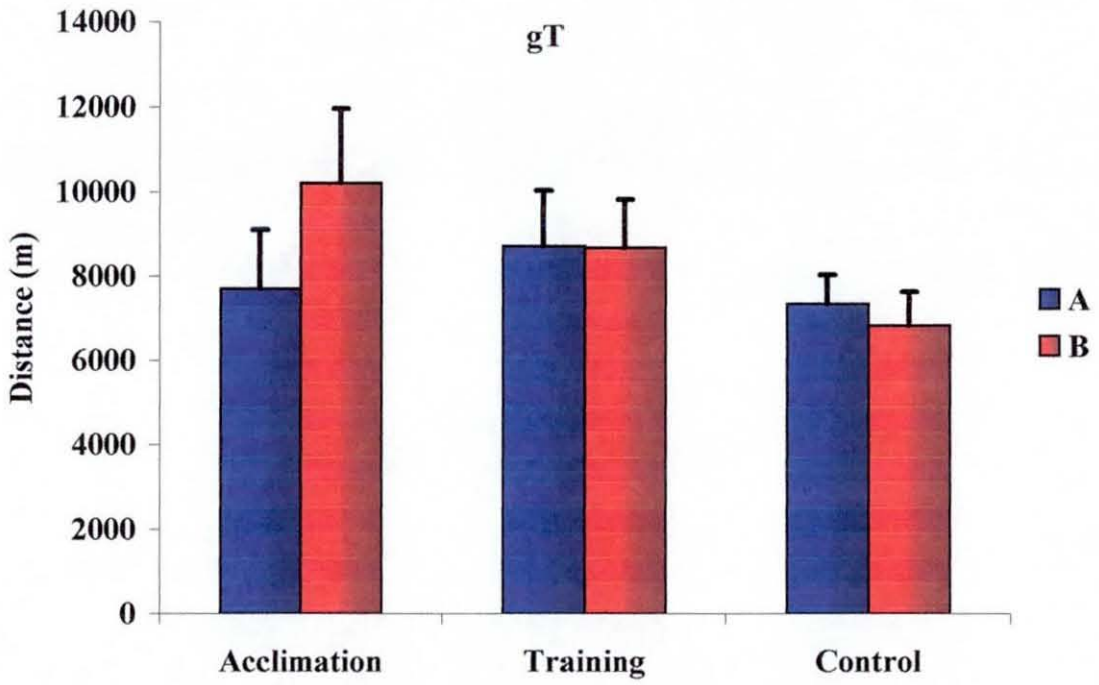
Figure 5.2 Distance completed during the main trials by the acclimation, training and control groups; gT = group x trial interaction $P<0.05$, $F_{2, 14} = 4.8$.

Table 5.2 Maximal 15m sprint times (s) during the first set and exhaustion sets of the LIST; t = main effect time P<0.01, $F_{2, 28}$ = 23.5.

	Acclimation		Training		Control	
	Set 1	End set	Set 1	End set	Set 1	End set
A	2.73 ± 0.08	2.87 ± 0.05	2.73 ± 0.05	2.85 ± 0.08	2.77 ± 0.09	3.00 ± 0.16
B	2.74 ± 0.08	2.90 ± 0.06	2.73 ± 0.06	2.92 ± 0.10	2.78 ± 0.11	3.01 ± 0.15
			t			

5.3.3. Rectal temperature

Resting rectal temperature was similar between groups and trials. With the onset of exercise rectal temperature increased in all trials (main effect time P<0.01, $F_{8, 112}$ = 175.7). Three-way ANOVA with repeated measures showed that there was a group x trial x time interaction (P<0.01, $F_{16, 112}$ = 2.7), resulting from the decrease in deep body temperature in early exercise and increase toward the end of exercise in the acclimation group. There was no difference in rectal temperature response during trial A, between the groups (Figure 5.3A). The acclimation group had a lower rectal temperature than the training and control groups during trial B and the acclimation group were able to continue exercising to a higher end rectal temperature (Figure 5.3B).
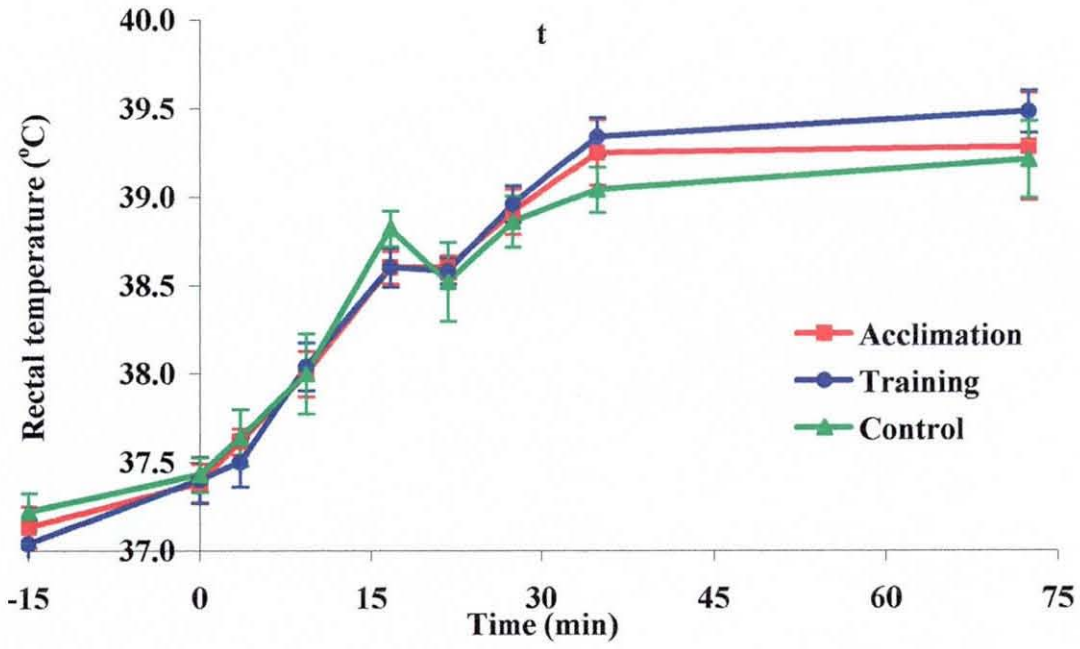
Figure 5.3A Rectal temperature response during trial A.
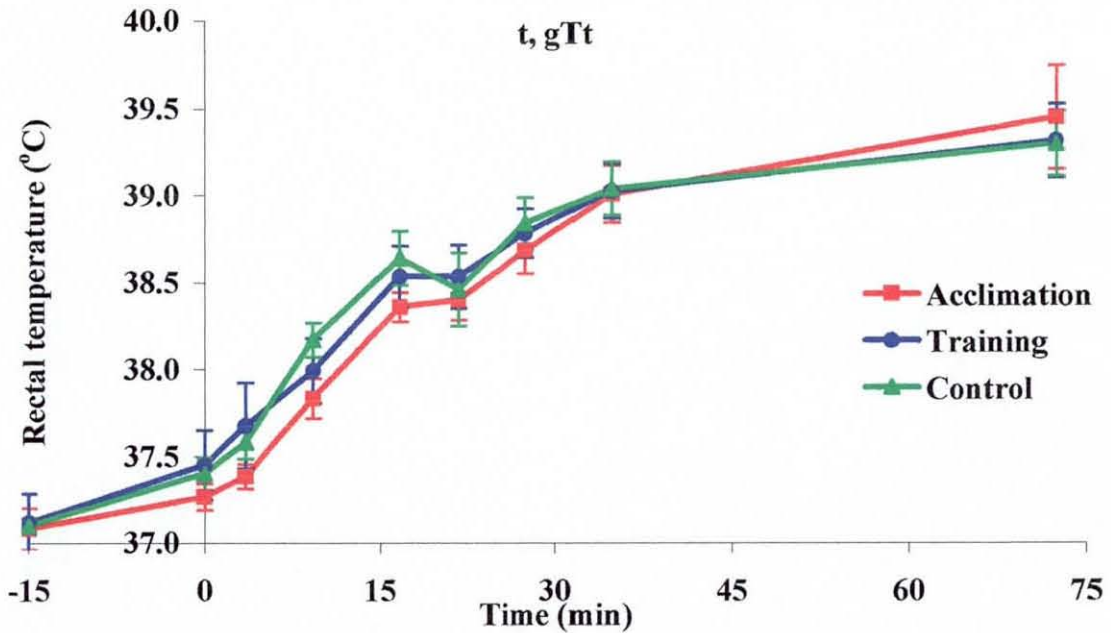
Figure 5.3B Rectal temperature response during trial B.

Figure 5.3A & B Rectal temperature response for the acclimation, training and control groups; t = main effect time $P<0.01$, $F_{8, 112} = 175.7$; gTt = group x trial x time interaction $P<0.01$, $F_{16, 112} = 2.7$.

5.3.4. Heart rate and rating of perceived exertion, thirst and thermal comfort

Rating of perceived exertion and perceived thirst increased throughout the exercise duration, but was not different between the two main trials (Table 5.3; main effect time P<0.01, RPE $F_{2, 28}$ = 120.2, thirst $F_{2, 28}$ = 38.1). However, subjects felt cooler after acclimation in the heat, with no differences in perceived heat found for the training or control groups (Table 5.4; interaction group x trial P<0.01, $F_{2, 14}$ = 6.9).

Table 5.3 Mean perceived exertion (RPE) and perceived thirst during the first 3 sets of the LIST; t = main effect time P<0.01, RPE $F_{2, 28}$ = 120.2, thirst $F_{2, 28}$ = 38.1.

	RPE			Thirst		
	Set 1	Set 2	Set 3	Set 1	Set 2	Set 3
Acclimation A	14 ± 1	17 ± 1	19 ± 1	6 ± 0	7 ± 1	9 ± 1
Acclimation B	13 ± 1	15 ± 1	17 ± 1	5 ± 0	6 ± 0	7 ± 1
Training A	13 ± 0	16 ± 1	17 ± 1	5 ± 0	7 ± 1	8 ± 1
Training B	12 ± 1	15 ± 1	16 ± 1	6 ± 1	7 ± 1	7 ± 1
Control A	13 ± 1	16 ± 1	18 ± 1	5 ± 0	7 ± 0	8 ± 1
Control B	14 ± 1	17 ± 1	19 ± 1	5 ± 0	7 ± 1	8 ± 1
		t			t	

Table 5.4 Mean perceived heat during the 1st 3 sets of the LIST; t = main effect time P<0.01, $F_{2, 28}$ = 22.8; gT = interaction group x trial P<0.01, $F_{2, 14}$ = 6.9.

	Set 1	Set 2	Set 3
Acclimation A	5 ± 0	7 ± 1	8 ± 1
Acclimation B	3 ± 1	5 ± 1	6 ± 2
Training A	4 ± 1	6 ± 1	7 ± 1
Training B	4 ± 1	6 ± 1	6 ± 1
Control A	5 ± 1	6 ± 0	8 ± 1
Control B	6 ± 1	8 ± 1	9 ± 1
		t, gT	

Heart rate increased throughout the exercise period during all the trials (main effect time P<0.01, $F_{2, 28}$ = 34.3). Figure 5.4 shows that during the first 2 sets of the LIST the heart rate was lower during trial B (main effect trial P<0.01, $F_{1, 14}$ = 11.1). The lower heart rate during trial B was due to lower heart rates for both the acclimation and

training groups, whereas there was a slight increase in the control group (interaction group x trial $P<0.05$, $F_{2, 14} = 8.9$).
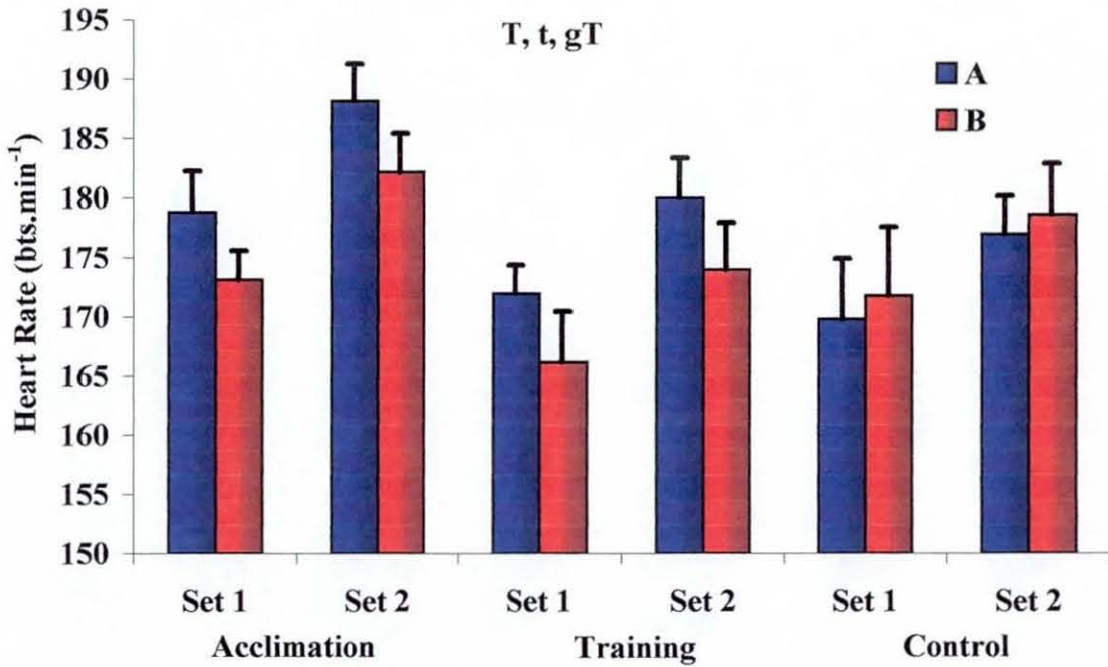
Figure 5.4 Heart rate during the first 2 sets of the LIST for the acclimation, training and control groups; T = main effect trial $P<0.01$, $F_{1, 14} = 11.1$; t = main effect time $P<0.01$, $F_{2, 28} = 34.3$; gT = group x trial interaction $P<0.05$, $F_{2, 14} = 8.9$.

5.3.5. Body mass, fluid consumption and estimated sweat rate

Body mass was well maintained by the acclimation, training and control groups decreasing by less than 0.7% of basal body mass during all the trials. Ad libitum fluid consumption was not different between the groups or between the trials. However there was a tendency for a lower water consumption after acclimation with all the acclimation subjects consuming less fluid during trial B than A, (acclimation A vs B, 18.6 ± 2.6 vs 14.5 ± 2.4 ml.kg^{-1}.h^{-1}; training A vs B, 17.9 ± 3.3 vs 17.9 ± 3.7 ml.kg^{-1}.h^{-1}; control A vs B, 12.0 ± 1.8 vs 12.0 ± 2.3 ml.kg^{-1}.h^{-1}). Estimated sweat rate was similar in all trials, (acclimation A vs B, 1.33 ± 0.24 vs 1.09 ± 0.14 l.h^{-1}; training A vs B, 1.18 ± 0.11 vs 1.19 ± 0.16 l.h^{-1}; control A vs B, 1.18 ± 0.06 vs 1.23 ± 0.05 l.h^{-1}).

5.3.6. Hormonal responses

Serum progesterone concentrations were similar between groups and between trials being 6.0 ± 2.3 and 6.3 ± 3.1 nmol.l^{-1} during trial A and trial B respectively. Similarly, serum aldosterone and serum cortisol concentrations were not different between trials (Table 5.5). However, resting serum cortisol was lower for the acclimation group than the training and control groups, but this difference was not present after the onset of exercise (interaction group x time $P<0.01$, $F_{6, 42} = 3.5$).

Table 5.5 Serum aldosterone and cortisol concentrations at rest and during the exhaustion set of the LIST; t = main effect time $P<0.01$, aldosterone $F_{3, 42} = 83.5$, cortisol $F_{3, 42} = 24.5$; gt = interaction group x time $P<0.01$, $F_{6, 42} = 3.5$.

	Acclimation		Training		Control	
	Rest	End set	Rest	End set	Rest	End set
Serum aldosterone concentration (pmol.l^{-1})						
A	551 ± 84	1877 ± 221	433 ± 108	1678 ± 298	443 ± 84	1567 ± 206
B	450 ± 62	2054 ± 332	495 ± 73	1815 ± 199	509 ± 35	1554 ± 106
t						
Serum cortisol concentration (nmol.l^{-1})						
A	538 ± 86	968 ± 62	702 ± 131	848 ± 106	791 ± 118	798 ± 58
B	561 ± 61	862 ± 72	811 ± 182	937 ± 190	806 ± 166	819 ± 115
t, gt						

5.3.7. Metabolic Responses

Table 5.6 shows that blood lactate concentrations were lower following training in the moderate condition (interaction group x trial $P<0.05$, $F_{2, 14} = 6.4$). There was no difference in the blood lactate response of the acclimation and control groups between trials. Plasma ammonia concentrations were lower during trial B at the end of exercise, with the greatest decreases being for the moderate training group (interaction trial x time $P<0.05$, $F_{3, 42} = 3.3$; Table 5.6). However, there was no difference between the trials for any of the three groups. Blood glucose concentrations did not differ between the groups or the trials (Table 5.6).

Table 5.6 Various blood metabolite concentrations at rest and during the exhaustion set of the LIST; t = main effect time P<0.01, lactate $F_{3, 42}$ = 55.8, ammonia $F_{3, 42}$ = 41.3, glucose $F_{3, 42}$ = 38.2; gT = group x trial interaction P<0.05, $F_{2, 14}$ = 6.4; gTt = group x trial x time interaction P<0.05, $F_{6, 42}$ = 5.6; Tt = interaction trial x time P<0.05, $F_{3, 42}$ = 3.3.

	Acclimation		Training		Control	
	Rest	End set	Rest	End set	Rest	End set
	Blood lactate concentration (mmol.l^{-1})					
A	0.8 ± 0.1	4.6 ± 1.1	0.7 ± 0.1	5.5 ± 0.9	0.9 ± 0.1	4.1 ± 0.6
B	0.8 ± 0.1	5.1 ± 1.1	0.8 ± 0.1	4.2 ± 0.9	0.8 ± 0.1	4.2 ± 0.3
	t, gT, gTt					
	Blood glucose concentration (mmol.l^{-1})					
A	4.5 ± 0.1	6.2 ± 0.8	4.4 ± 0.1	6.3 ± 0.6	4.8 ± 0.2	7.4 ± 1.0
B	4.6 ± 0.2	6.7 ± 0.9	4.4 ± 0.2	5.2 ± 0.5	4.8 ± 0.2	7.2 ± 0.6
	t					
	Plasma ammonia concentration (μmol.l^{-1})					
A	18.5 ± 6.5	48.2 ± 8.3	17.7 ± 3.5	62.0 ± 10.4	8.3 ± 2.0	39.8 ± 8.2
B	17.3 ± 4.9	45.6 ± 9.3	13.4 ± 1.5	35.5 ± 7.3	8.2 ± 2.3	38.9 ± 10.0
	t, Tt					

5.3.8. Plasma volume

There were no differences in resting plasma volume between the groups or between trial A and trial B, (acclimation A vs B, 61.9 ± 1.1 vs 60.7 ± 1.0 ml.100 ml^{-1}; training A vs B, 60.8 ± 0.7 vs 62.2 ± 0.4 ml.100 ml^{-1}; control A vs B, 61.6 ± 1.3 vs 61.1 ± 1.1 ml.100 ml^{-1}). The estimated change in plasma volume response was lower following acclimation (acclimation end A vs B –3.5 ± 1.4 vs –0.6 ± 2.2%, interaction group x trial P<0.05, $F_{2, 14}$ = 4.2). In contrast, the change in plasma volume response was greater following training in the moderate condition (training end A vs B –0.1 ± 2.0 vs –3.7 ± 1.2%, interaction group x trial P<0.05, $F_{2, 14}$ = 4.2). The estimated change in

plasma volume response was not different for the control group between trials (control end A vs B –3.3 ± 1.2 vs –4.0 ± 1.4%).

5.4. Discussion

The main finding of the present study is that the performance of prolonged intermittent, high intensity shuttle running, in terms of distance run was improved following heat acclimation. There were no improvements in performance in a group who completed the same amount of training in moderate conditions. A lower rectal temperature in early exercise and a higher temperature at exhaustion was recorded in the post-acclimation trial for the acclimation group in comparison with the training and control groups.

High intensity intermittent exercise performance, in terms of distance run was increased by 33% following 4 short sessions of acclimation, in a 10 day period. Similarly, performance following 7-10 days of acclimation has been reported to be improved by 5-15% using walking acclimation protocols or by as much as 67%, when subjects exercised to exhaustion each day (Nielsen et al., 1993; Cheung and McLellan, 1998; Aoyagi et al., 1998). The large improvement in performance in the present study, following only 4 sessions, may be related to the high intensity intermittent nature of the acclimation protocol. Gisolfi and Cohen (1979) stated that interval or intermittent training resulted in a rapid increase in deep body temperature and thus was a powerful stimulator of thermoregulatory responses. Thus, it is likely that the intermittent high intensity protocol played a key role in the performance improvement. The response and extent of capacity improvements are dependent upon the training status, exercise intensity and duration, environmental conditions and length of the acclimation protocol used. Therefore, comparisons with previous research are difficult. For example, a 5% increase in walking (3.5 km.h^{-1} 40°C, 30% RH) capacity has been reported in moderately fit men following acclimation, whereas there was no change in well trained men (Cheung and McLellan, 1998).

Serum aldosterone concentrations were unaltered by the acclimation or training intervention. This finding is in agreement with previous research using well-trained subjects. It has been previously reported that acute acclimation and chronic acclimatisation do not alter the aldosterone response to exercise in well-trained

subjects (Finberg and Berlyne, 1977; Houmard et al., 1990; Nielsen et al., 1993; Nielsen 1994). This may be the result of a maximised salt balance in this group as a result of their training status (Davies et al., 1981). However, Nielsen et al. (1993) did observe a lower resting aldosterone concentration following acclimation, though this was not maintained with the onset of exercise. The finding by Nielsen et al. (1993) may have been the result of the wide range of maximal oxygen uptake values in the group that were classed as well-trained (49 – 74 ml.kg^{-1}.min^{-1}). However, the authors concluded that this lower resting aldosterone concentration would not have contributed to the enhanced performance after acclimation. Similarly, in the present study, serum cortisol concentrations were not different following acclimation between the groups. Only a limited number of studies have investigated serum cortisol concentrations and responses following acclimation. Studies by Finberg and Berlyne (1977) and Franseconi et al. (1984) showed that there were no differences in cortisol responses in both males and females (low intensity cycling and walking); however Armstrong et al. (1989) found a lower cortisol concentration. As the authors did not employ a control group and the subjects were not well-trained, this lower level may have been partially related to an increase in physical fitness with the training undertaken. The acclimation protocol employed by Armstrong and co-workers (1989) was high intensity intermittent running (8 days of 9 bouts of ≤ 10 min running [68%

$\dot{V}O_2$ max]: rest periods), which can induce training in a short period of time.

In the present study, resting plasma volume was not different between the groups or the trials. In previous research, which has shown an expansion of plasma volume, this has occurred during days 3-6 of the consecutive acclimation period and was short-lived (Wyndham et al., 1976; Houmard et al., 1990). Thus, the lack of change in plasma volume in the present study, may be due to only 4 sessions over a 10 day period. Furthermore, well-trained individuals have a higher than normal plasma volume and thus this may prevent the necessity for plasma volume expansion as part of acclimation (Horstman and Christensen, 1982). Fortney et al. (1979) completed a study that required subjects to undertake a prolonged training period before acclimation. Plasma volume expansion occurred following the training period, but was not increased by the acclimation. Thus, the training status of the games players

(well-trained) could explain why there were no changes in resting plasma volumes in the current study.

Sweat rate was unaffected by the four acclimation sessions. This finding is contrary to some previous research that has shown in absolute sweat rate and sweat sensitivity during prolonged cycling and treadmill running (Horstman and Christensen, 1982; Nielsen et al., 1993; Cheung and McLellan, 1998). Nielsen suggested that the increase in cycling capacity (60% $\dot{V}O_2$ max) that was observed following the 9-12 day acclimation (40°C, 15% RH) was due to an increase in evaporative rate and increase in sweating sensitivity. The increase in sweating sensitivity may be due to an increase in receptor density. The humidity of the environment for the acclimation and training will also alter the sudomotor response, as sweat rates tend to be unchanged in a dry environment but may increase following acclimation in a humid conditions (Armstrong and Maresh, 1991). The humidity in the current study was less than 32% and thus the sweating adaptations would be diminished. The sex of the subjects will have also impacted upon the sweating response following acclimation as men have been shown to have a greater sweat rate both before and after acclimation and a greater increase following acclimation (Avellini et al., 1980). Therefore, it has been suggested that women are more efficient regulators of body temperature.

Furthermore, studies that have used trained subjects have shown that sweat rate is unaffected by acclimation (Gisolfi and Cohen, 1979; Houmard et al., 1990). The study by Gisolfi and Cohen (1979) investigated the effects of 11 weeks of interval training and then acclimation on the physiological responses of women to a 4 h walk at 30% $\dot{V}O_2$ max (45°C, 17% RH). The interval training was of a high intensity requiring that the subjects' heart rate be maintained between 90-95% of maximum. They ran on a treadmill at 10-13 km.h^{-1} for 90 s and then rested for 30 s; this pattern was repeated for 50-60 min, four times per week. Following the training period sweat rate was increased, but there was no further increase after acclimation. The findings of the current study are in agreement with those of Gisolfi and Cohen (1979) and suggest that well-trained games players do not show an increase in sweat rate during exercise in a hot dry environment following acclimation. Training has been suggested to cause

glandular or neuro-glandular sweat adaptations or peripheral changes, such as an increase in sweat sensitivity, whereas acclimation may cause a central adaptation such as a decrease sweat threshold (Cotter et al., 1997). While sweat sensitivity was not measured in the present study, it is therefore likely to have been heightened by the chronic interval training undertaken by the games players. This may provide a possible explanation for the finding that sweat rate was unaffected by acclimation in the current study.

A decrease in heart rate has been outlined as one of the first adaptations to occur during days 3-5 of acclimation, as an indicator of cardiovascular adjustments (Armstrong and Maresh, 1991). An expanded plasma volume and redistribution of blood volume via a modification to the autonomic nervous system may partially explain this decrease (Fortney et al., 1979; Armstrong and Maresh, 1991). Decreases have been shown in both men and women (Avellini et al., 1980; Horstman and Christensen, 1982) and training status may affect the degree of the decrease observed (Shvartz et al., 1977). In the current study, heart rate was lower during trial B and this was attributed to lower heart rates for the acclimation and moderate training groups. This decrease in heart rate, though not significant for the acclimation and moderate groups separately (P=0.05), was similar between these groups, thus suggesting that heart rate was not affected by acclimation per se in the current study, nor was serum aldosterone, plasma volume or sweat rate. Thus, there is little evidence of a redistribution or increase in blood volume and no improvement in cardiovascular function. Furthermore, subjects perceived the exertion in both trials to be similar. Rating of perceived exertion is usually proportional to central cardiorespiratory stress (Armstrong and Maresh, 1991). The demands of the intermittent protocol required maximal 15 m sprints, which ensured that high heart rates were attained throughout both trials. The previous limited research examining the responses to intermittent or high intensity exercise following acclimation is in agreement with the current study.

In a study by Houmard and colleagues, low intensity (60 min at ~50% $\dot{V}O_2$ max) and high (30-35 min at ~75% $\dot{V}O_2$ max) intensity acclimation protocols were compared. The acclimation period was 7 days and showed that from day 4 onward there was a lower heart rate recorded in comparison with day 1 for the low intensity condition.

However, for the high intensity protocol heart rate was lower only on the final acclimation day. An interval training regimen in the heat was employed by Gisolfi and Cohen (1979) using females and heart rate was unchanged during a prolonged walk. Thus, the intensity of the exercise and intermittent nature may partially explain why lower heart rates were not recorded in the current study after only 4 short acclimation sessions.

The acclimation group had a lower rectal temperature than the moderate training and control groups during the first 30 min of trial B. It has been suggested that the key determinant for exercising and resting rectal temperature is acclimation status (Shvartz et al., 1977). Thus, a lower rectal temperature is often used as an indicator that acclimation has occurred. Lower deep body temperatures have been recorded in well-trained groups for cycling, running and walking and for men and women (Shvartz et al., 1977; Fortney et al., 1979; Avellini at al., 1980; Horstman and Christensen, 1982; Nielsen et al., 1993). The subjects also perceived that they were cooler following the heat acclimation period. The reasons for a lower rectal temperature following acclimation have been attributed to increased heat dissipation (Nielsen, 1994) and a decrease in metabolic heat production (Houmard et al., 1990). There was no increase in the sweat rate and thus heat dissipation may not have been increased but without any measures of blood flow, this can not be ruled out.

Blood lactate concentrations were unaltered by acclimation suggesting that metabolism was unaffected. Previous research that has investigated metabolism adaptations to acclimation and have shown a decrease in lactate (Febbraio et al., 1994b) and no change in lactate (King et al., 1985). Both these research groups employed acclimation protocols of 90 min cycling at 50% $\dot{V}O_2$ max (40°C), but the heat stress tests differed markedly. Febbraio et al. (1994b) recorded lower muscle and blood lactate concentrations during 40 min cycling at 70% $\dot{V}O_2$ max and attributed this to decrease in glycogenolysis in the type 1 fibres. The heat stress test employed by King at el. (1985) involved the completion of two 45 s sprints separated by 6 h of 30 min cycling (50% $\dot{V}O_2$ max) and 30 min rest in the heat. Following acclimation, there was a 42% decrease in glycogen utilisation during the 6 h period, but no change

in respiratory exchange ratio or blood lactate concentration. Therefore, the authors suggested that there might have been an increase in perfusion of the active skeletal muscle, thus increasing the delivery of blood glucose and free fatty acids. Furthermore, a decrease in catecholamine concentration as a result of a lower deep body temperature may have also contributed to the lower glycogen utilisation. These differing findings between studies are related to the type, intensity and duration of the acclimation protocols employed.

In contrast to the acclimation group, in the present study the moderate training group had lower blood lactate concentrations after acclimation. However, this metabolic difference had no effect on performance in terms of distance run or 15 m maximal sprint time. A possible explanation for this lower lactate concentration in the moderate training group is that these subjects were able to work at a higher intensity during the sprints. The LIST, through its very design to mimic games type activity, requires the completion of 15 m maximal sprints. Previous research using female games players has shown that sprint performance is not only faster in moderate conditions than hot, but does not decline (Morris et al., 2000). The moderate group therefore may have had a greater sprint training effect than the acclimation group, thereby lowering their blood lactate concentrations.

In summary the main finding of the present study is that the high intensity intermittent running capacity in the heat was improved by 33%, in well-trained female games players, following 4 short acclimation sessions, but sprint performance was unaffected. The underlying mechanisms for the adaptive changes seen following acclimation are dependent upon not only the protocol employed, but also the training status of the subjects. The unique protocol in the current study resulted in a lower rectal temperature following acclimation than a moderate training or control group. Thus a lowering of deep body temperature and an increase in thermal comfort may be responsible for the performance improvement. The magnitude of improvement in performance in the acclimation group suggests that a short-term high intensity intermittent protocol could improve performance for games players competing in championships in the heat.

6. The reliability and validity of a field hockey skill test

6.1. Introduction

To undertake research into field hockey in a controlled setting, it is necessary to employ a skill test that can be completed in the laboratory environment. However, there are only a limited number of field hockey tests and very little has been done scientifically to formulate tests that measure playing ability (Wessel and Koening, 1971). Two decades later, further developments of hockey tests had not advanced. Reilly and Borrie (1992) noted that it was surprising that even though field hockey had been part of the Physical Education curriculum in Europe and North America since the beginning of the 20th Century, there had been little attention given to the design of field tests for the game.

Thus, at present the number of published tests of field hockey skill is limited. Table 6.1 outlines previous tests that have been published, none of which were published during the last fifteen years. With the advent of synthetic sportsturfs as the major playing surface over that period, it is apparent that the skills have changed significantly and thus there is a need to develop a skill test that is appropriate to modern hockey. Furthermore, the skill tests were designed to determine differences in skill performance between players, rather than to monitor improvements or changes for a particular player, and thus were not stringently tested for reliability.

In the formulation of a skill test, it is important that technique is differentiated from skill. Technique is the production of some pattern of movements which are technically sound (Knapp, 1963). The following definition of skill will be used for the purpose of the design of this study: "Skill is the learned ability to bring about predetermined results with the maximum certainty, often with the minimum outlay of energy, or of time and energy," (Knapp, 1963). This encompasses the idea that a skilled athlete must take an action that is appropriate and therefore the skill involves interpreting the needs of the situation and making the correct decision as well as carrying out the necessary movements. The main point here is that the cognitive component in the form of decision making is a fundamental element of the skill.

Over the past decade there has been an increase in the literature regarding the importance of reliability and validity studies and the statistics that should be employed and interpreted. In terms of reliability, it has been advocated that a number of statistical methods be cited and interpreted (Atkinson and Nevill, 1998). Reliability has been partially defined to include the "consistency of an individual's performance on a test" (Atkinson and Nevill, 1998). It should be recognised that tests will always include some form of measurement error and therefore reliability needs to be considered as the amount of measurement error that has been deemed acceptable for the effective practical use of a measurement tool. When the tool is to be used for scientific research, the acceptable level is of paramount importance. To conclude that a measuring tool is valid, it must show logical, construct and criterion validity (Strand and Wilson, 1993). Logical validity means that the tool is appropriate to want you want to measure, construct validity refers to a measuring tool that can discriminate between standards and criterion validity refers to how well the measuring tool correlates to previous tools used to measure the same variable (Strand and Wilson, 1993).

Thus, the aim of the present study was to design a field hockey skill test which was both reliable and valid for the modern game of hockey and determine the acceptable levels which would make it a suitable tool to use for research in a laboratory environment.

Table 6.1 Published tests of field hockey skill or technique.

Publication	Skill test	n	Reliability	Validity
Reilly & Bretherton (1986)	"T" – run dribbling test	24		Differentiated between County & Centres
Chapman (1982)	Chapman ball control test	23	R = 0.89	r = 0.63 (score with subjective rank)
Wessel and Koening (1971)	Stewart backboard test	228	r = 0.81	ANOVA differentiated between poor, average & good
Wessel and Koening (1971)	Field, control and drive (Friedel)		r = 0.81 (left) r = 0.62 (right)	r = 0.87 (with Schmithals & French ball control test)
Schmithals & French (1940)	Dribble, dodge, circular tackle and drive	51	r = 0.92	
Schmithals & French (1940)	Combined goal shooting	51	r = 0.90	
Schmithals & French (1940)	Fielding and drive	51	r = 0.90	

6.2. Methods

6.2.1. Subjects

Thirty-nine university hockey players volunteered to take part in the study. Twenty males and 19 females completed the validity study, whereas only 14 males and 17 females completed the reliability of the skill test.

6.2.2. Skill test design

As outlined, to produce a skill test, it must incorporate an element of decision making to define it from a technique assessment. With this in mind, the test was also designed to include numerous elements of the game of hockey, whilst controlling as many variables as possible. For example a rebound board was used to pass off, as it will

react in the same manner each time, whereas a player making this pass, would add a further uncontrolled variable. The surface for the test was a water based sportsturf (Desso), the type of surface all the players regularly play and train on, thus allowing the players to perform their skills as normally as possible as well as controlling the environment. The goal is the width of a normal field hockey goal and the target area for the skill test is 18 in high, which is the height of a backboard in hockey. The skill test design incorporates dribbling, passing and shooting.

The objectivity of the skill test was paramount in the design and therefore subjects were only given instructions about the penalty timing system and completing the test as quickly and as accurately as possible. No information on how to approach the test was provided. This allowed the subjects to use any techniques they would normally use in the game, allowing them to decide and react to the different elements as they would in a game situation.

Figure 6.1 shows the layout of the skill test. The numbers represent each of the dimensions of the test which are below:

1. 12 Ft (width of a hockey goal)
2. Distance from the end of the playing area to the target
3. 7.55 m
4. 7.35 m
5. 4.22 m
6. 2.82 m
7. 5.50 m
8. 1.98 m
9. 1.17 m
10. 3.25 m
11. 1.77 m
12. 1.5 m
13. 1.55 m
14. 2.75 m
15. 2.88 m
16. 0.25 m
17. 1.00 m
18. 1.55 m
19. 0.70 m
20. 3.37 m
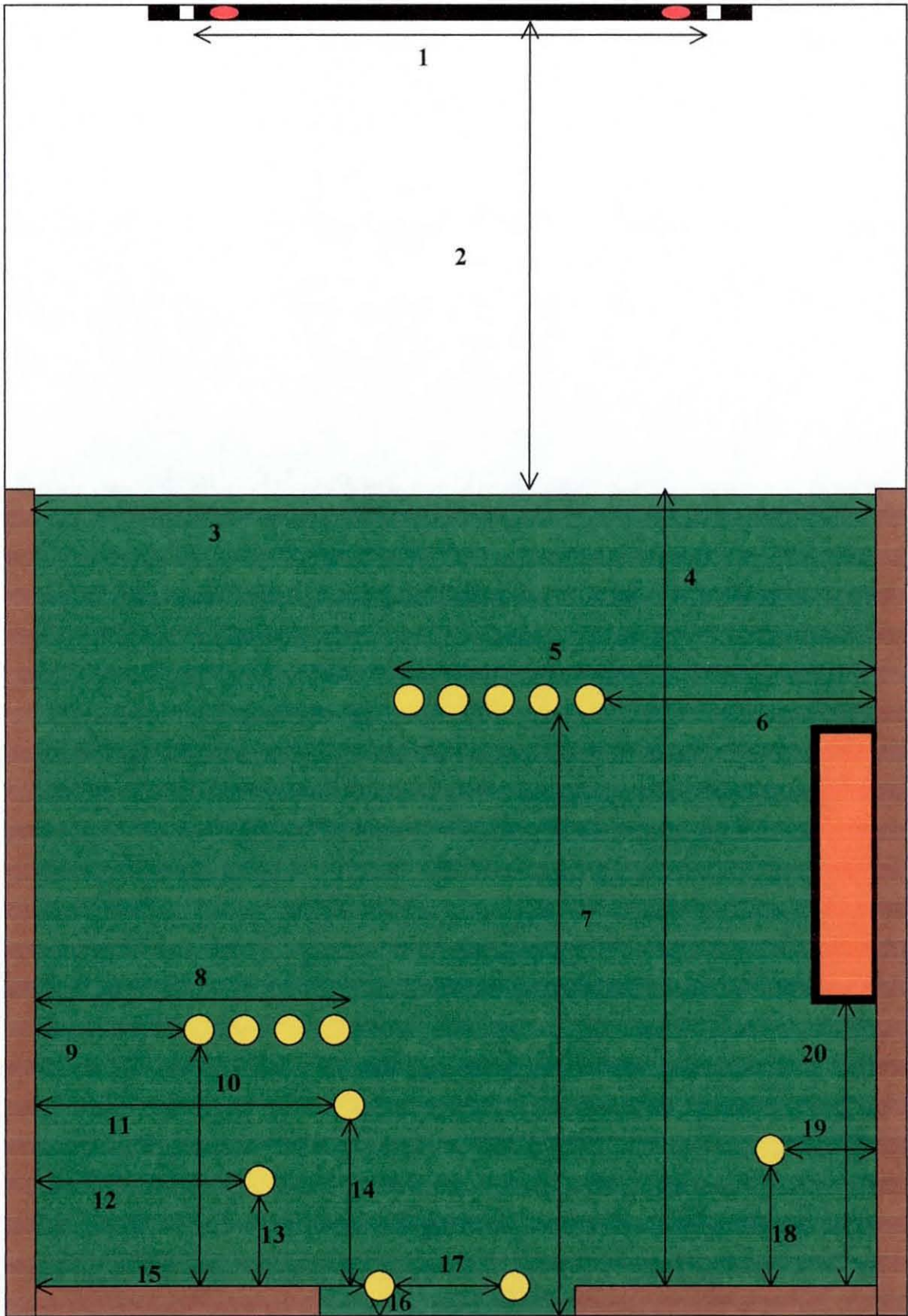
Figure 6.1 Plan of the skill test. See previous page for the dimensions.

The skill test requires the subjects to start from a yellow line, permanently on the sports hall floor (Figure 6.2). The player then runs to pick up a hockey ball and then dribbles round the cones in a specific sequence (Figure 6.4). The completion of the dribbling phase requires the player's foot or ball to break an infra-red beam (Figure 6.3) which triggers a light on either side of the goal and starts a computer timing system (BBC microcomputer). The player then plays a pass against the rebound board (Figure 6.2) and shoots at either the red and white or blue and white target on the goal. The player must shoot at the opposite target to where the light is on, for example if the light is on above the red and white area (right side of the goal), the player must shoot at the blue and white target area (left side of the goal). The player must always shoot straight at the target and not diagonally, for the previous example to shoot at the blue target the player must bring the ball round the left hand side of the five cones to shoot (Chapter 3, Figure 3.2). When the player has shot, the ball will hit the goal and stop the timing system, which is triggered by the sound of the ball. The time taken between crossing the infra-red beam and the ball hitting the backboard was termed the 'decision making' time as it incorporates the decision making elements of how and when to pass against the rebound board or shoot and determining which side of the goal to shoot. After the completion of the shot the player then runs back to the yellow line.

Yellow start line

Rebound board

Figure 6.2 Photograph of the skill test layout.

Infra-red beam   Reflector for beam
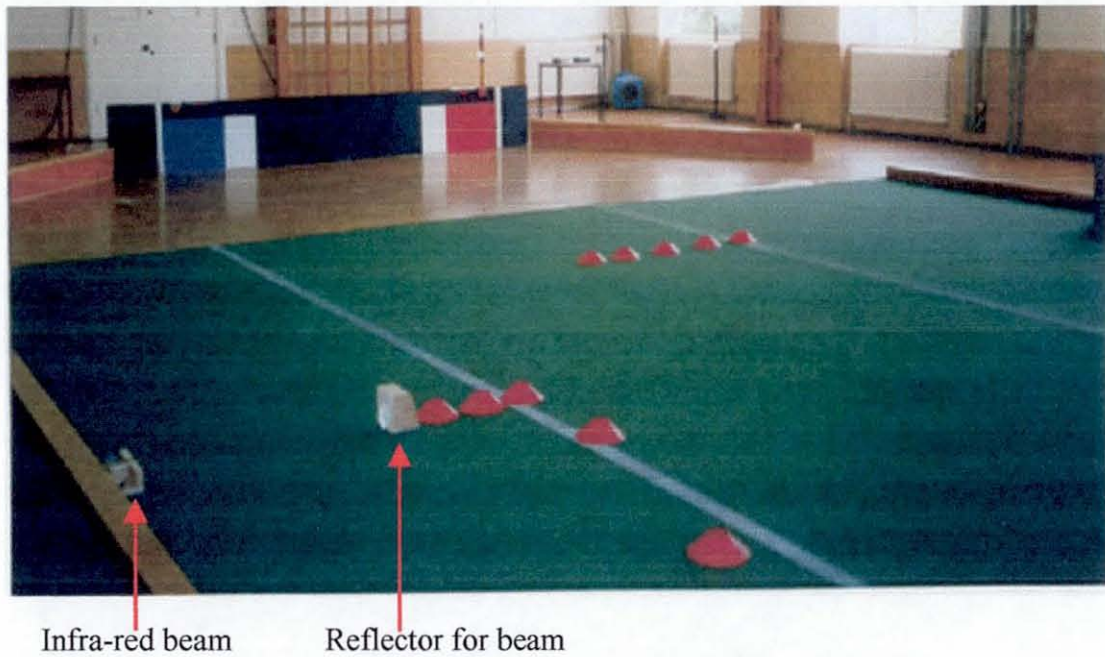
Figure 6.3 Photograph of the infra-red beam placement on the skill test.

The player repeats the dribble, pass and shot pattern 6 times; each time the player has to touch the yellow line with their foot. The total time is recorded for the 6 continuous runs. In addition, a penalty time of 2 s per error is added, if the player misses the target area on the goal, touches a cone with the ball or the ball touches the player's feet. The total time for the 6 runs and any error time is termed the 'overall time' and is used as the measure of performance for the field hockey skill test. The 'decision time' is taken as the average of the 6 decision timings, which incorporates 3 shots at the red and white target and 3 shots as the blue and white target, in a randomised order. Three shots at each target controls for the different distance that is covered by the player depending on the side of the goal that he/she is shooting at.

The players were verbally encouraged to perform maximally and informed about the number of repetitions remaining. If the players lost control of the ball, they had to continue from wherever the ball went.

6.2.3. Familiarisation

Subjects were familiarised with the skill test on two occasions. During the first session they were instructed about how to complete the skill test and the timing and penalty system. They then completed 10 repetitions of the test, resting between each repetition. The pattern was randomised, but 5 shots were completed to each side of the goal. The second familiarisation session required the subjects to perform the skill test in its entirety. Thus, they completed the 6 repetitions as fast as they could, and the overall time and decision time were recorded.

6.2.4. Reliability trials

After being fully familiarised, 31 subjects completed the skill test on two occasions on separate days, at least 3 days apart. The subjects were asked to refrain from vigorous exercise on the day of the skill test. To account for circadian rhythms, the skill tests were completed at the same time of day.

6.2.5. Validity trials

Thirty-nine subjects completed the skill test, after refraining from vigorous exercise on that day, but were not informed about their performance. The male players who completed the test, were then ranked for performance and skill on their normal game play, by one International standard coach (coach 1) and one National League coach (coach 2). Similarly, an International standard coach (coach 3) and one National League coach (coach 4) ranked the female players' who completed the test on their normal game play. The coaches were provided with a definition of skill and performance, which they could use to rank the players, so that all the coaches were working to the same criteria. Performance was defined as overall match performance and contribution to a match and skill defined as "the learned ability to bring about predetermined results with the maximum certainty, often with the minimum outlay of energy, or of time and energy," (Knapp, 1963). The coaches were provided with the names of the players, but were not given any information about the performance of the players on the field hockey skill test. All the coaches regularly coached and watched the players who they ranked, so were fully aware of their abilities. The

performance ranks were compared with the overall time for the skill test, whereas the skill ranks were compared with the decision time.

6.2.6. Statistical analyses

The reproducibility of the skill test was determined using numerous statistical techniques. These were mean difference, Bland and Altman limits of agreement, correlations and typical error (Bland and Altman, 1986; Atkinson & Nevill, 1998; Hopkins, 2000). The coaches' ranks and skill test scores were compared using a Spearman Rank correlation. Data were checked for heteroscedasticity, so that the appropriate statistical techniques could be employed.

6.3. Results

6.3.1. Heteroscedasticity

The results showed some herosedasticity, therefore statistics were employed on both the raw data and the log transformed data and all the results are presented to allow comparison and suitable inferences about the reliability to be made.

6.3.2. Reliability

The mean (±SE) for trial 1 and trial 2 was 90.85 ± 1.65 and 90.89 ± 1.65 s for overall performance time and 4.17 ± 0.09 and 4.17 ± 0.10 s for the decision time. Table 6.2 and 6.3 shows a variety of statistical results used for comparing the overall performance and 'decision making' time reliability of the skill test respectively. Figure 6.5 and 6.7 shows a scatter diagram displaying the relationship between the overall skill test performance and decision time respectively in trials 1 and 2 with the line of equality shown on the figure. There is a strong relationship for overall skill test performance as indicated by a Pearson and intraclass correlation above 0.85 (Table 6.2 and 6.3). The relationship for decision time was also good, being above 0.70 (Table 6.4 and 6.5). Figure 6.6 shows the Bland and Altman plot for overall performance time for trial 2-1, and gives a mean difference and limits of agreement of 0.03 ± 5.11 s. The Bland and Altman plot for decision time shows a mean difference and limits of agreement of 0.01 ± 0.52 s (Figure 6.8).
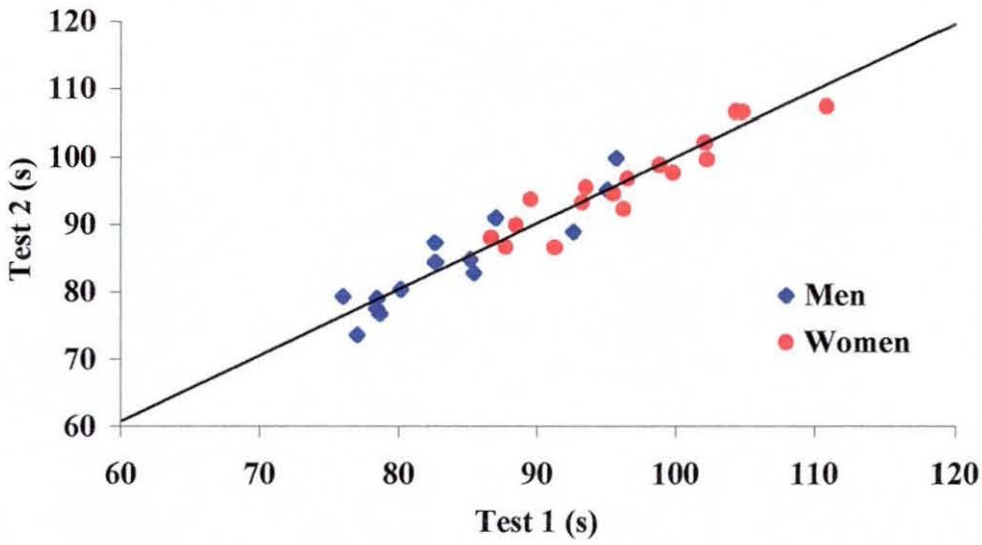
Figure 6.4 Relationship between the two test scores for the overall performance time raw data; (r = 0.96, P<0.0001).
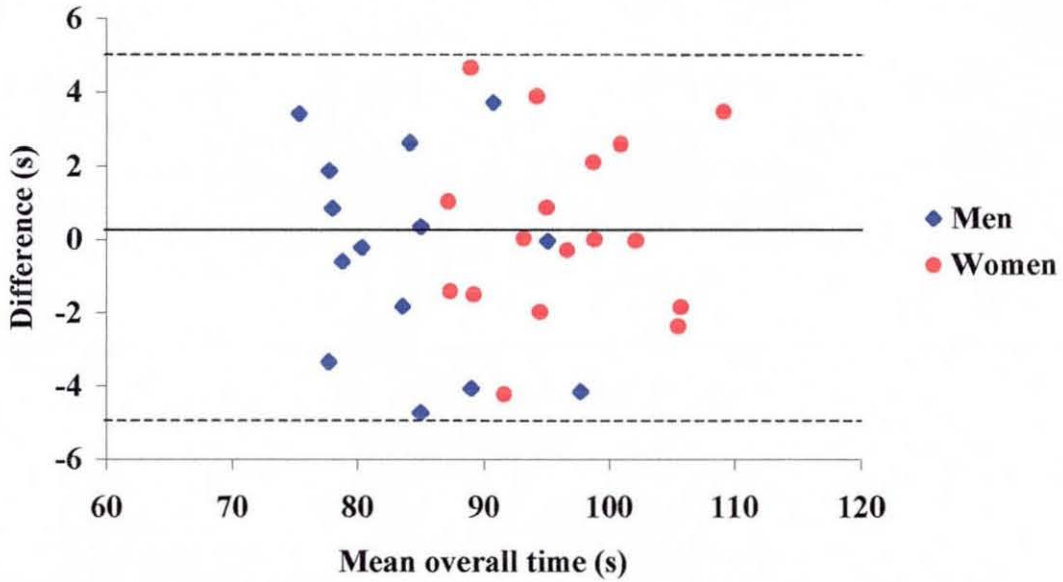
Figure 6.5 Bland-Altman plot for the overall time raw data.

Table 6.2 Statistical summary of the reproducibility of the raw data for the overall time of the skill test.

	Men	Women	All
Mean difference (s)	0.44	-0.30	0.03
95% confidence interval bias (s)-	-1.20	-1.55	-0.92
95% confidence interval bias (s)+	2.08	0.96	0.99
Typical error (s)	2.01	1.73	1.89
Pearson correlation (r)	0.93 P <0.0001	0.94 P <0.0001	0.96 P<0.0001
Intraclass correlation (r)	0.92	0.94	0.96
T-test (t)	0.57 P = 0.58	0.50 P = 0.62	-0.02 P = 0.98

Table 6.3 Statistical summary of the reproducibility of the log transformed data for the overall time of the skill test.

	Men	Women	All
Mean difference (%)	0.4	-0.3	0.0
95% confidence interval bias (%)-	-1.5	-1.6	-1.0
95% confidence interval bias (%)+	2.4	1.0	1.1
Typical error as CV (%)	2.4	1.8	2.1
Pearson correlation (r)	0.92 P <0.0001	0.93 P <0.0001	0.96 P<0.0001
Intraclass correlation (r)	0.92	0.93	0.96
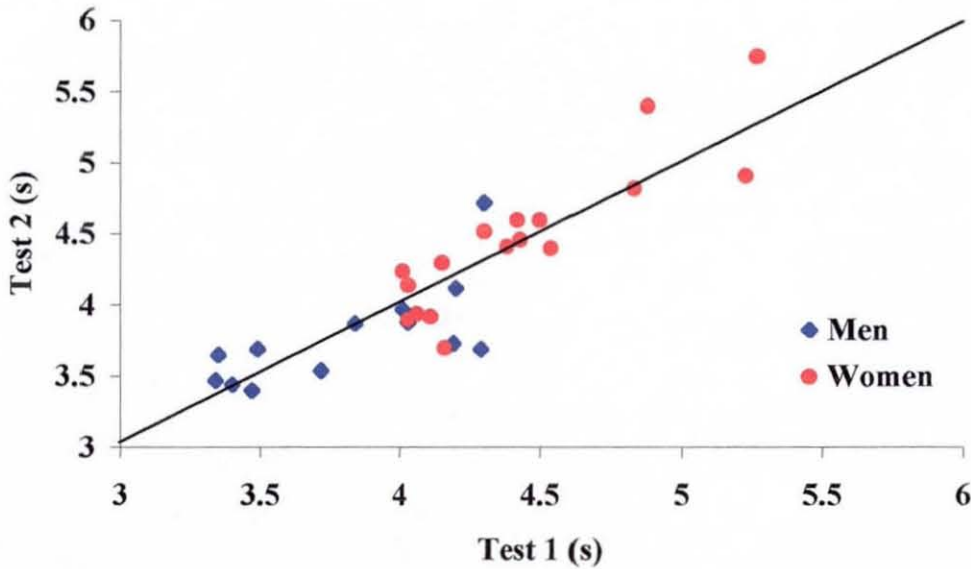
Figure 6.6 Relationship between the two test scores for the 'decision making' time raw data; (r = 0.89, P<0.0001)
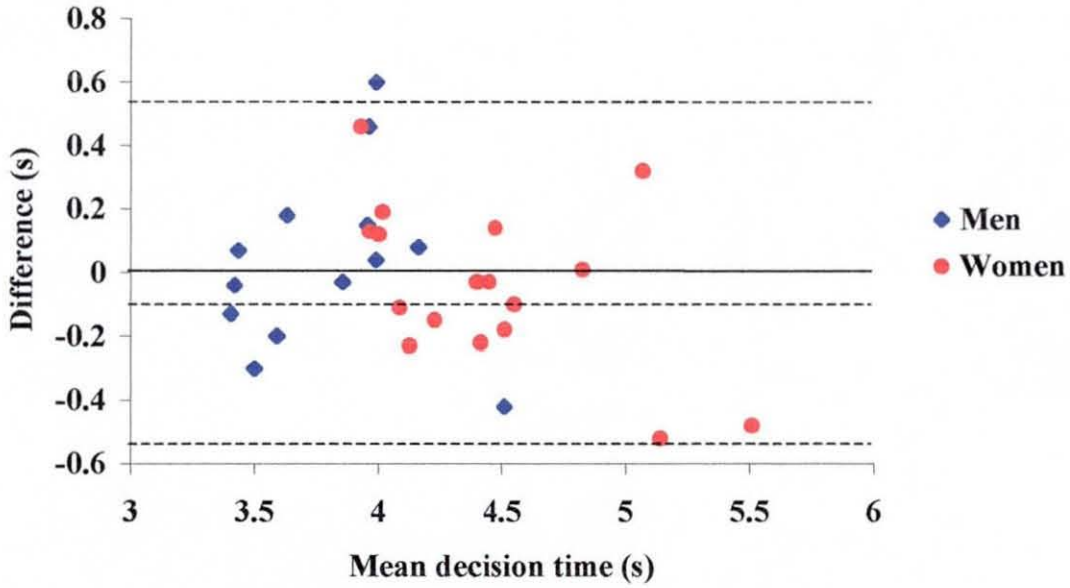
Figure 6.7 Bland-Altman plot for the 'decision making' time raw data.

Table 6.4 Statistical summary of the reproducibility of the raw data for the 'decision making' time of the skill test.

	Men	Women	All
Mean difference (s)	-0.04	0.04	0.01
95% confidence interval bias (s)-	-0.21	-0.09	-0.09
95% confidence interval bias (s)+	0.13	0.17	0.11
Typical error (s)	0.20	0.18	0.19
Pearson correlation (r)	0.70 P <0.01	0.89 P <0.0001	0.89 P <0.0001
Intraclass correlation (r)	0.70	0.85	0.88
T-test (t)	-0.45 P = 0.66	0.64 P = 0.53	0.15 P = 0.88

Table 6.5 Statistical summary of the reproducibility of the log transformed data for the 'decision making' time of the skill test.

	Men	Women	All
Mean difference (%)	-0.9	0.6	0.0
95% confidence interval bias (%)-	-5.0	-2.2	-2.3
95% confidence interval bias (%)+	3.5	3.6	2.3
Typical error as CV (%)	5.2	4.0	4.5
Pearson correlation (r)	0.72 P <0.01	0.88 P <0.0001	0.88 P <0.0001
Intraclass correlation (r)	0.70	0.85	0.87

6.3.3. Validity

The Spearman rank values for the men and women are presented in table 6.3 and 6.4 respectively. The validity values were much greater for both the decision time and the overall time for the women than the men.

Table 6.6 The Spearman Rank values for the validity of the skill test for the men.

	Decision time	P value	Overall time	P value
Coach 1	0.70	P<0.01	0.64	P<0.01
Coach 2	0.65	P<0.01	0.54	P<0.05
Average	**0.70**	**P<0.01**	**0.63**	**P<0.01**

Table 6.7 The Spearman Rank values for the validity of the skill test for the women.

	Decision time	P value	Overall time	P value
Coach 3	0.73	P<0.001	0.85	P<0.00001
Coach 4	0.73	P<0.001	0.80	P<0.0001
Average	**0.74**	**P<0.001**	**0.85**	**P<0.00001**

6.4. Discussion

The main finding from the present study was that the reproducibility of the skill test was good. The mean ($\pm$SE) differences in overall and 'decision making' time between the 2 trials was 0.03 ± 0.47 and 0.01 ± 0.05 s respectively. Correlations between the two trials were high to very high for the raw and log transformed data (Cohen and Holliday, 1982). The correlations between coaches rankings and player performance was also high, with Spearman rank order correlations for the overall performance of 0.63 and 0.85 for the men and women respectively.

The statistical approach to reliability is not straightforward. Comparing means between the two trials is one indicator of reliability but does not provide any information about the range of values. Thus, the responses to two tests could differ considerably without being deemed significant. However, comparing means is useful as that is what is tested for, compared and reported during scientific research. The means in the current study show good similarity and are almost identical. Another approach to measure association between two tests is to use correlations. However, a high correlation does not necessarily imply good agreement. This problem can be overcome by examining how close the points on a plot of trial 1 against trial 2 lie to the line of equality. In the current study, data points all lie close to the line of equality, implying good agreement as well as good association between trials. It should be noted that the data for overall skill test performance lie closer to the line of equality that the data for 'decision making' time.

Hopkins (2000) has advocated the use of typical error rather than the limits of agreement approach that is recommended by Atkinson and Nevill (1998). Hopkins (2000) suggested that the value of the limits of agreement approach is dependent upon the sample size of the reliability study. The bias of the limits of agreement are <5% if there are >25 subjects; however if there is only 8 subjects this bias is 21%. In the current study, there are over 30 subjects for the data for men and women combined and therefore the bias will be low. The Bland and Altman (1986) limits of agreement provides a confidence interval for the differences between two trials and it is up to the experimenter to determine whether this range is acceptable. Hopkins (2000) suggested that a 95% confidence interval is too stringent a measure if used for looking at an athlete's improvement in performance and that half the limits of agreement would still leave approximate odds of 5-1 that performance had actually improved. Thus, the 95% confidence interval allows for an underestimation of the reliability of the protocol as it takes into account 2 standard deviations rather than the usual 1 that is used as an indicator of variation. The mean difference and confidence intervals for overall skill test performance was 0.03 ± 5.11 s and for 'decision making' was 0.01 ± 0.52 s. In contrast the typical error of overall performance, as advocated by Hopkins (2000) of the test was 1.89 s and 2.1% for the raw and log transformed data respectively. For the 'decision making' raw and log transformed data, the typical error was 0.20 s and 5.2% respectively. The typical error is the within-subject standard deviation and represents the variation we could expect to see from trial to trial for each subject (Hopkins, 2000). The limits of agreement and typical error indicate the reliability of the skill test is very good and that changes in overall performance of greater than 2.1% could be attributed to the intervention.

These data show that the reliability of the skill test is considerably better than any previously published data for field hockey (Table 6.1). The tests that have been formulated during the last 20 years have been designed as field tests to determine differences between players rather than for scientific research and thus would not be transferable. The Chapman Ball Control Test (1982) isolates the ability of an individual to control the ball manipulatively by arm, wrist and hand action within a 9.5" (24 cm) diameter circle. This could be described as measuring dribbling technique rather than field hockey skill per se. Thus, it can not be a measure of

playing ability since this is not what constitutes the entire domain of field hockey skill. The testing took place on a gymnasium floor, which is a considerably different surface from the outdoor game. While the results from the Chapman test correlate well with subjective opinions of playing ability, it does not attempt to measure any other characteristics. Testing of ball control is obviously important, but analyses of match play highlight how little time players spend with the ball during a match and the very short duration of each period with the ball. The validity of the Chapman test would be reasonable if the test scores were compared with subjective ratings of ball control and not overall playing ability. Reilly and Bretherton (1986) developed a field-based skill test, namely the "T"-dribbling test and a dribbling and accuracy test, to help determine the fitness of female hockey players. The T-dribbling test was

shown to be correlated with aerobic fitness ($r = 0.48$; estimated $\dot{V}O_2$ max and physical working capacity) and anaerobic power ($r = 0.6$; stair run test). The accuracy was correlated with ectomorphy ($r = -0.63$). The skill tests provide useful field tests, but do not provide us with a test that includes a passing aspect and 'decision making' element. Furthermore, the "T"-dribbling test, is restrictive in that the players were unable to use reverse sticks, which is an integral part of the game and therefore would not be a suitable measure of hockey performance per se.

In our laboratory, previous soccer skill tests have been developed for use in researching the effects of fatigue on skill performance. The reliability in terms of mean difference ($\pm$ limits of agreement) Loughborough Soccer Passing Test was $-0.1 \pm 11.2\%$ (McGregor, 1999). The limits of agreement are much greater than those in the current study ($0.0 \pm 5.6\%$), suggesting that the reliability of the field hockey skill test is good and acceptable for scientific research.

The validity of the field hockey skill test is good. The term 'validity' used in the current study, refers to both logical validity and construct validity. Logical validity means that the test is appropriate to what you want to measure, whereas construct validity refers to a test which can discriminate between groups of performers (Strand and Wilson, 1993). A further type of validity, should be tested for, namely criterion validity, which means that the test needs to be compared with an established test. However, as there does not seem to be a previous field hockey skill test that is

regarded as 'established', this is inapplicable. The skill test provides slightly better validity for female hockey players than male. This may be due to the different demands and styles of play adopted by men and women. In field hockey there are "physical and physiological differences between the sexes" that means that the game of hockey will be played differently by men and women (Lakomy et al., 2000). For elite hockey players, men were found to have a higher $\dot{V}O_2$max and haemoglobin content and were faster, taller and heavier than the women (Lakomy et al., 2000).

Skill tests need to be objective as well as valid and reliable. Though the objectivity of the test has not been statistically determined, the test should exhibit good objectivity. The test performance is determined by timings, which are completed by a computer and stopwatch and penalty time. The players are only instructed in what order to complete the test and the penalty system, and thus the inferences of the testers are minimal. The tester is only responsible for timing and counting the number of penalties so the results should be similar, if not identical between all testers.

The test was performed on a typical sportsturf and is thus easily transferable between pitches. The field hockey skill test could be easily transferred to the pitch, using the goal and could be made as realistic as is required. The movement of a goalie could determine the side for shooting, with another attacker playing the pass and a defender taking the place of the five cones to shoot around. Thus, the test could be as scientific or match like as is required, and could range from a coaching aid to a selection aid.

In summary, the field hockey skill test provides a reliable, objective and valid tool for testing the skills of good to elite field hockey players. The high reliability and validity allows it to be used for scientific research as well as determining how the skills of individual players are developing.

7. The effect of intermittent, high intensity shuttle running and hot environmental conditions on field hockey skill performance

7.1. Introduction

Team sports, which are characterised by intermittent high intensity exercise bouts, also require a contribution of motor skill performance and cognitive functioning (Burke, 1997). In terms of the assessment of the physiological demands of team sports, soccer has been most extensively investigated (Shi and Gisolfi, 1998). There has been little research investigating the demands of field hockey and the few studies completed (Wein, 1981, Fox, 1984; Lothian and Farrally, 1992; Lothian and Farrally, 1994) are of limited value due to the new surfaces and rule changes that have altered considerably the characteristics of the game in recent years.

While the maintenance of physiological function is clearly important in team sports such as field hockey, soccer and rugby, during such activities, the production of the required skills is at least as important in determining success in the particular sport. However, very few studies have investigated how fatiguing exercise may influence motor skill performance and cognitive functioning. In this context, fatigue is not simply meant as the termination of a particular form of exercise, it is meant to recognise that in the course of an activity, such as soccer or field hockey, distance run and exercise intensity will be reduced, but not terminated during a match. Research into the performance of field hockey during National League matches has shown that during the second half of a normal match there is a decrease in the level of high intensity activity performed (Lothian and Farrally, 1994). There was, however, no reference made to the extent to which field hockey specific activities were affected by the duration of a match.

Studies which have assessed changes in some measure of 'skill' performance, are scarce and have concentrated on the effects of exercise in moderate environmental conditions on skill performance, particularly soccer skill. This is partially due to the difficulties of undertaking field-testing. McGregor et al. (1999) have found that

following prolonged intermittent high intensity running in moderate conditions skill performance is decreased when subjects refrained from fluid replacement. Skill performance was maintained when water was ingested ad libitum. However, following 2 h of tennis training, fluid ingestion did not prevent a decline in skill performance, during defensive rallies (Vergauwen et al., 1998). Suggestions for these decrements in performance following either match play or prolonged intermittent high intensity running are low glycogen concentrations, hypoglycaemia or dehydration (Shephard and Leatt, 1987; Jacobs, 1988; Leatt and Jacobs, 1988; Rico-Sanz et al., 1996; McGregor et al., 1999).

Many important World Championship events, Olympic and Commonwealth Games are held in hot environmental conditions, where the challenge to the homeostatic mechanisms in the body is probably substantially greater than that presented in moderate conditions. However, research into the effects of exercising in hot environmental conditions compared with moderate conditions on skill performance in general is limited (Dawson et al., 1985) and hockey skill performance in particular has not been investigated. Dawson et al. (1985) compared the performance of tennis players by the measurement of serving, groundstroke and volley power and accuracy. Players completed a 1 h tennis match in moderate conditions (23°C, 65% RH), followed by a tennis skills test (serve, volley, groundstroke). To compare performance in a hot environment (35°C, 65% RH), the players undertook intervalised treadmill running for 1 h which attempted to simulate the work performed by each player during the match in moderate conditions. Skill performance was poorer by 19, 26 and 14% respectively for serving, groundstrokes and volleys. Dawson et al. (1985) attributed the decrement in performance to the greater thermoregulatory and cardiovascular strain during the treadmill running in the hot environment, compared with match play in moderate environmental conditions.

Research into intermittent exercise and skill performance has also been completed in the heat, to compare hydration strategies, rather than the impact of environmental stress per se on skill performance (Rico-Sanz et al., 1996). Irrespective of hydration status (hyperhydration or voluntary hydration) soccer skill test performance was shown to decline in elite players (Rico-Sanz et al., 1996). The majority of field hockey matches are played on an artificial playing surface. When exposed to solar radiation,

the sportsturf is heated and reradiates thermal energy to the hockey players on the pitch. The temperature on the pitch can be raised by 1-5°C in the presence of solar radiation (Buskirk et al., 1977). Thus the performance of field hockey in hot environments and the thermal strain on the players is compounded by the sun on the pitch surface.

In light of the previous research, the current study tested the hypothesis that performance of a field hockey skill test would be decreased following intermittent running, and that the performance decrease would be greater in hot conditions.

7.2. Methods

7.2.1. Subjects

Nine, well-trained, unacclimatised female university hockey players volunteered to participate in the study. Six of the nine had normal menstrual cycles, and three had been taking oral contraceptives for over one year. The physical characteristics of the subjects are presented in Table 7.1.

Table 7.1 Physical characteristics of the female hockey players who participated in the study.

	Mean ± SE
Age (years)	21.7 ± 0.4
Mass (kg)	68.5 ± 3.6
Height (cm)	167.3 ± 2.2
Estimated $\dot{V}O_2$ max (ml.kg^{-1}.min^{-1})	50.3 ± 1.1

7.2.2. Experimental design

Subjects performed the Loughborough Intermittent Shuttle Test (LIST; Nicholas et al., 1995, 2000) and field hockey skill tests in hot (30°C) and moderate (20°C) environmental conditions.

During the LIST subjects exercised over a 20 m distance and repeated a walk, sprint,

run (~85% $\dot{V}O_2$ max) and jog (~50% $\dot{V}O_2$ max) pattern of exercise for 15 min,

followed by a 3 min rest period. This pattern of activities constituted 1 Set of the

LIST. Subjects performed 4 sets of the LIST in all. The protocol is shown

schematically in Figure 7.1.
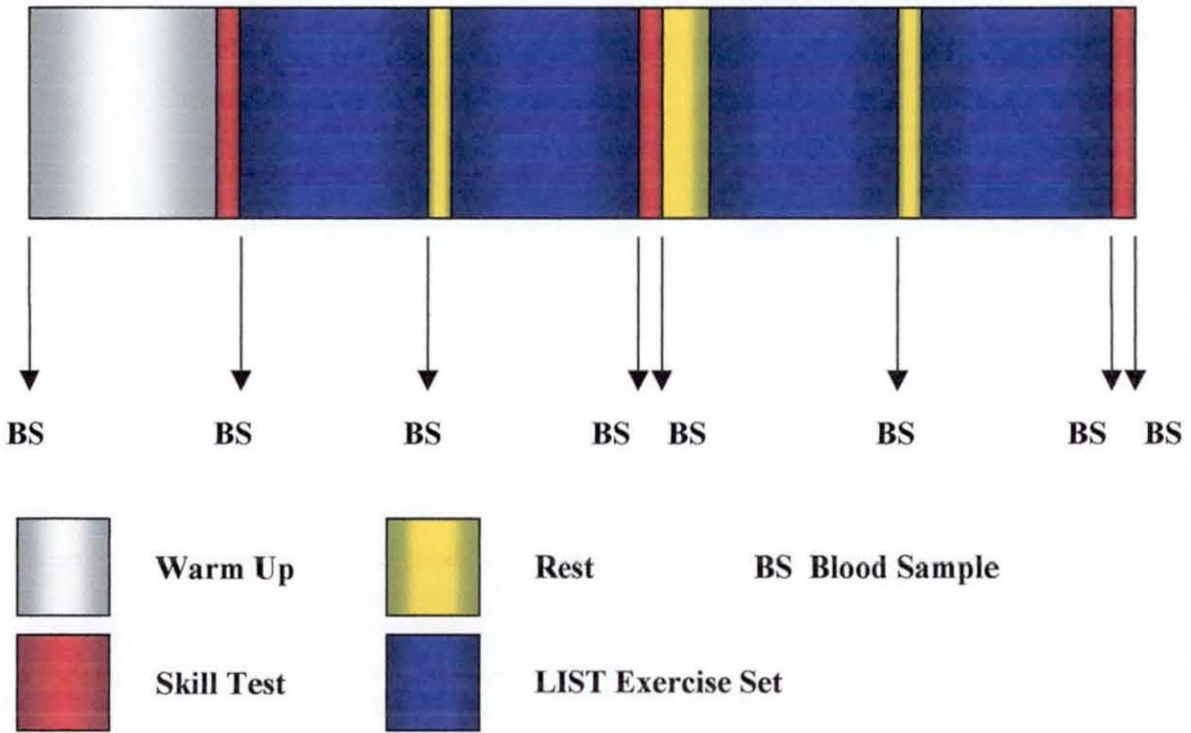
Figure 7.1 Schematic representation of the protocol.

The field hockey skill test was performed prior to the LIST and after the 2nd and 4th

sets of exercise. The skill test was started from a line 16 yards (14.56 m) from a goal

and was undertaken on a sporturf, which would be used for field hockey. The field

hockey skill test required the subjects to dribble round cones in a specified order,

before breaking an infra-red beam (RS Components Ltd.) which randomly turned on

a light at either the left or right hand side of the goal. The subjects then passed the

hockey ball off a rebound board (Exportise Ltd) before shooting at a target on the

opposite side of the goal to the light (Chapter 3, Figure 3.2). This was repeated 6

times to complete the field hockey skill test, with the total time being recorded. Any

errors (for example missing the target on the goal, touching cones or the ball touching

time was recorded automatically (BBC microcomputer), which will be referred to as the 'decision time'. This was the time taken between breaking the infra-red beam and hitting the backboard with the shot, which was determined by a microphone attached to the backboard. (Full description of the skill test can be found in Chapter 6).

Each trial consisted of three skill tests and four sets of the LIST; one skill test was completed prior to the commencement of the LIST and then repeated after two sets and again after four sets of the LIST. The subjects rested for a total of 10 minutes after the first two sets to mimic half-time in a match situation. If subjects were unable to complete all four sets of the LIST (volitional exhaustion or rectal temperature >40°C), the final skill test was undertaken immediately after the subject had stopped running. The order of trials was randomly assigned and 28 days elapsed between the moderate and hot trials. The trials were conducted one month apart to take account of menstrual cycle phase, which was verified by measurement of serum progesterone concentration.

7.2.3. Preliminary measurements

Maximal oxygen uptake ($\dot{V}O_2$ max) was estimated using a progressive multistage fitness test (Ramsbottom et al., 1988) prior to the main trials. Subjects were also familiarised with the LIST at 30°C for 2 sets or 33 min and fully familiarised with the field hockey skill test.

7.2.4. Main trials

Subjects reported to the laboratory at least 12 hours after their last meal. In the 2 days prior to their first main trial each subject recorded food consumed as average food portion consumed on dietary sheets provided for them. They were then asked to repeat the same diet prior to the second trial. All experiments were arranged so that each individual ran at the same time of day for both the moderate and hot trials to control for circadian influences. Having reported to the laboratory, a 45 mm cannula (Venflon 2, BOC Ohemeda AB) was then inserted into a forearm vein of the subject under local anaesthetic (Lignocaine hydrochloride 1 % w/v, Antigen pharmaceuticals Ltd.). The cannula was kept patent with saline solution (Sodium choloride 0.9% w/v, Antigen pharmaceuticals Ltd.). Subjects' nude body mass was recorded and a rectal probe (Edale Instruments Ltd.) was inserted to a depth of 10 cm beyond the anal sphincter.

Fifteen minutes after cannulation, during which time subjects remained standing to ensure that changes in posture would not effect the estimated changes in plasma volume, a 'resting' blood sample was collected. A resting rectal temperature was recorded immediately after entering the hot or moderate environment. A standardised warm-up of 15 min was then performed which consisted of jogging, stretching and faster pace running. During the warm-up and throughout the exercise period subjects were allowed to drink water ad libitum.

During each trial investigators ensured that subjects performed the exercise correctly by placing at least one foot on or over the lines marking the 20 m distance. The same careful procedures were used during the field hockey skill test. Subjects were able to gauge their required running speeds by following an amplified audio signal generated from a microcomputer (BBC). During the sprints and field hockey skill test, subjects were verbally encouraged to perform maximally. Sprint times over 15 m were

measured using 2 infra-red photo electric cells (RS Components Ltd.) connected to the microcomputer.

A whirling hygrometer (Brannan Thermometers Ltd.) was used to measure atmospheric dry and wet bulb temperatures every 5 min. Heart rate was continuously monitored throughout each trial using short-range telemetry (Vantage NV, Polar Electro Fitness Technology, sampling frequency 15 s). Rating of perceived exertion, using the Borg scale (1962) and perceived thirst and thermal comfort were recorded prior to the 11th sprint in each exercise set. A 10 ml blood sample was collected from each subject between the sets of exercise and before and after each skill test. Rectal temperatures were measured during the 4th and 8th cycle of each set and prior to, and post each skill test. When rectal temperatures were measured subjects were stationary for the equivalent time of 40 m of the 60 m walk in that cycle.

7.2.5. Blood sampling and analysis

Three ml of blood was dispensed into an EDTA tube and aliquots were used for determination of haematocrit and haemoglobin concentration (by microcentifugation and the cyanmethaemoglobin method respectively). Changes in plasma volume (%) were estimated using the method of Dill and Costill (1974). One ml of blood was immediately analysed for blood glucose and lactate concentrations using a fully automated machine (Yellow Springs Instruments Ltd. Stat 2300 Plus). One ml of blood was dispensed immediately into a calcium-heparin tube, centrifuged for 3 min at 12,000 rev.min^{-1} and the plasma frozen at -70°C. Ammonia concentration was determined within 24 h using a commercially available kit (Sigma Diagnostics). The remaining blood was centrifuged for 15 min at 6000 rev.min^{-1} at ~3°C. The resulting plasma was then stored at -20°C.

Five ml of blood was also dispensed into a serum tube for determination of progesterone, aldosterone and cortisol concentration by using commercially available radioimmunoassay kits (Diagnostic Products Corporation).

7.2.6. Statistical analyses

A two-way analysis of variance with repeated measures was used to establish if any significant differences existed between subject response in terms of physiological and

metabolic parameters to the performance of the LIST and the field hockey skill test in the two different environmental conditions. Where necessary, significant differences in the way subjects responded to the LIST and the skill test in the 2 conditions were located using Post-hoc Tukey tests. A students t-test was also used where appropriate. Data are presented as means ± standard error of the mean (SEM) and are based on a subject population of 9 unless otherwise stated.

7.3. Results

7.3.1. Environmental conditions

Dry bulb and wet bulb temperatures were higher in the hot trial than in the moderate (T_{db}: HT 30.2 ± 0.5 vs MT 19.1 ± 1.3°C, $P<0.01$; T_{wb}: HT 18.6 ± 0.8 vs MT 14.0 ± 1.3°C, $P<0.05$). Relative humidity was not different between trials (HT 37.9 ± 4.6 vs MT 50.8 ± 4.2%).

7.3.2. Field hockey skill performance

When subjects performed the field hockey skill test following a warm up, but prior to any intermittent running, the time taken to complete the test was not different when performance in hot and moderate conditions were compared. Field hockey skill performance declined following 30 and 60 min of the LIST compared with pre-LIST (main effect time $P<0.01$, $F_{2,\ 16} = 7.5$). This decrement in performance was compounded in the hot environment with a 6% poorer performance in the heat recorded for the 2nd skill test (MT: 93.37 ± 2.35, 95.75 ± 2.96, 97.50 ± 2.28 s; HT: 93.71 ± 2.74, 101.73 ± 3.68, 101.35 ± 3.68 s; main effect trial $P<0.05$, $F_{1,\ 8} = 8.0$; Figure 7.2). However, no difference was found in the 'decision making' element of the skill test (Table 7.2).
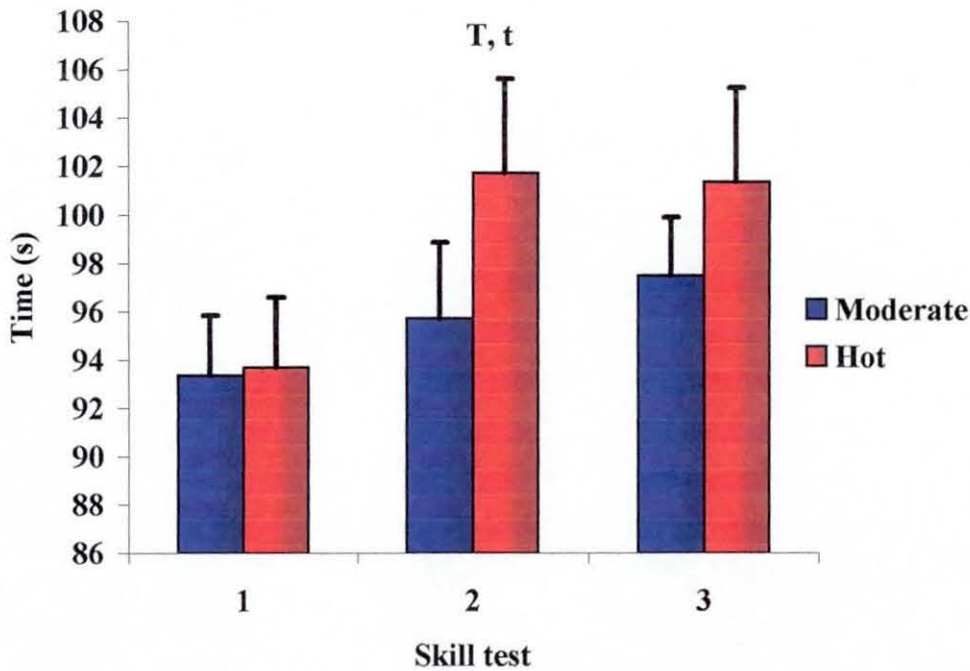
Figure 7.2 Skill test performance during the hot and moderate trials; T = main effect trial P<0.05, $F_{1,8}$ = 8.0; t = main effect time P<0.01, $F_{2,16}$ = 7.5.

Table 7.2 'Decision making' time for the skill test during the hot and moderate trials.

Time (s)	Skill test		
	1	2	2
Moderate	4.37 ± 0.09	4.59 ± 0.20	4.47 ± 0.10
Hot	4.39 ± 0.11	4.67 ± 0.22	4.68 ± 0.21

7.3.3. Distances run and 15 m sprint performance

Three subjects were unable to complete the full distance in the hot environmental condition, whereas only 1 subject failed to complete the moderate trial. However, there was no significant difference in the total distance completed which was 7442 ± 414 and 7881 ± 196 m in the hot and moderate trials. Table 7.3 shows that subjects sprinted faster in the moderate trial than the hot trial (main effect trial P<0.01, $F_{1,9}$ = 11.3). The average time taken to complete the 15 m sprints increased during the first 3 sets of exercise in the hot and moderate trials (main effect time P<0.01, $F_{3,27}$ = 14.0).

Table 7.3 Fifteen metre sprint times during the LIST in the hot and moderate environmental conditions; T = main effect trial P<0.01, $F_{2, 16}$ = 7.5; t = main effect time P<0.01, $F_{3, 27}$ = 14.0; Tt = interaction trial x time P<0.05, $F_{3, 27}$ = 8.3.

	Set 1	Set 2	Set 3	Set 4
Moderate	2.78 ± 0.04	2.82 ± 0.05	2.87 ± 0.05	2.85 ± 0.04
Hot	2.81 ± 0.03	2.95 ± 0.06	3.08 ± 0.07	3.09 ± 0.08
		T, t, Tt		

7.3.4. Rectal temperature

Figure 7.3 shows that rectal temperature was higher in the hot trial in comparison with the moderate trial (main effect trial P<0.01, $F_{1, 8}$ = 12.6) and increased throughout the exercise period in both trials (main effect time P<0.01, $F_{15, 120}$ = 89.6; Figure 7.3).
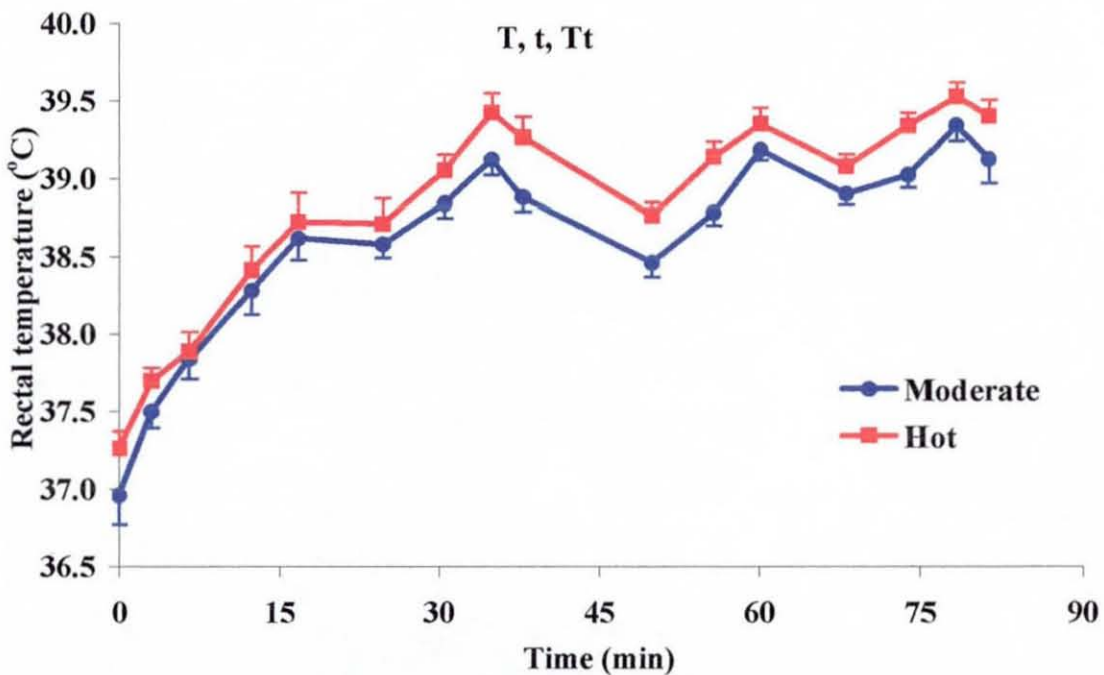
Figure 7.3 Rectal temperature during the hot and moderate trials; T = main effect trial P<0.01, $F_{1, 8}$ = 12.6; t = main effect time P<0.01, $F_{15, 120}$ = 89.6; Tt = interaction trial x time P<0.01, $F_{15, 120}$ = 3.4.

7.3.5. Body mass, fluid consumption and estimated sweat rate

Body mass, as a percentage of resting body mass was well maintained in both environmental conditions (HT -0.57 ± 0.23 vs MT -0.39 ± 0.25%). However, estimated sweat rate was 20% higher in the heat (HT 1.27 ± 0.10 vs MT 1.05 ± 0.12

l.h^{-1} P<0.05) and concurrently water consumption during the hot trial was also greater (HT 14.6 ± 1.7 vs MT 12.3 ± 1.6 ml.kg^{-1}.h^{-1} P<0.05).

7.3.6. Heart rate and perceived ratings

As figure 7.4 shows, average heart rates were higher throughout exercise when the environmental conditions were hot (main effect trial P<0.05, $F_{1, 8}$ = 8.0). Perceived exertion and perceived thirst were higher during the hot trial than the moderate trial (main effect trial P<0.05, RPE $F_{1, 8}$ = 5.7, thirst $F_{1, 8}$ = 21.3; Table 7.4). Similarly, there was a tendency for a greater feeling of thermal stress during the hot trial (main effect trial P=0.05, $F_{1, 8}$ = 5.3; Table 7.4).
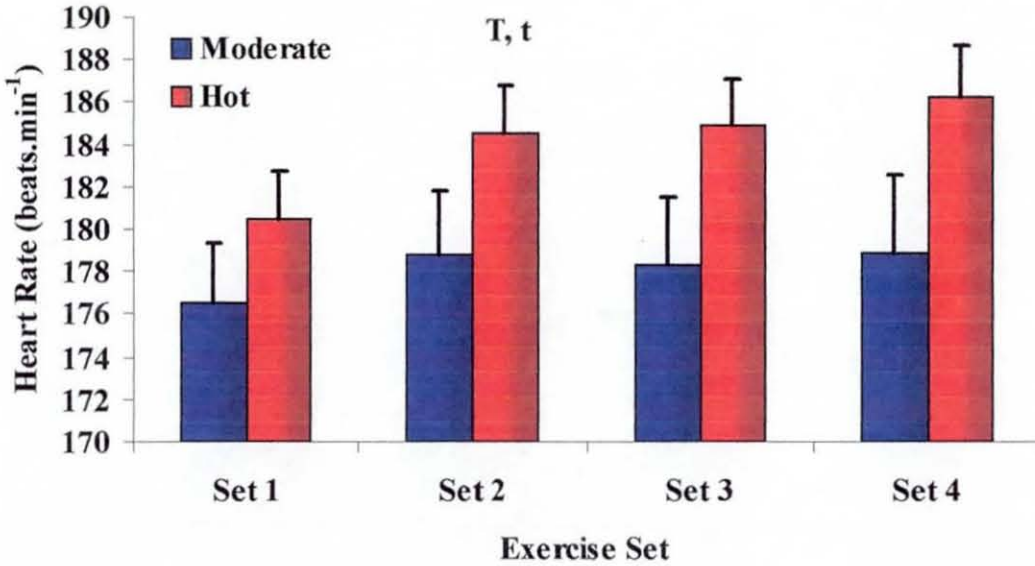
Figure 7.4 Mean heart rate during the hot and moderate trials; T = main effect trial P<0.05, $F_{1, 8}$ = 8.0; t = main effect time P<0.01, $F_{3, 24}$ = 5.1.

Table 7.4 Perceived ratings of exertion, thirst and thermal comfort during the hot and moderate trials; T = main effect trial P<0.05, $F_{1, 8}$ = 5.7; T* = main effect trial P<0.01, $F_{1, 8}$ = 21.3; t = main effect time P<0.01, RPE $F_{3, 24}$ = 40.8, thirst $F_{3, 24}$ = 9.7; Tt = interaction trial x time P<0.01, $F_{3, 24}$ = 5.9.

	Set 1	Set 2	Set 3	Set 4
Rate of perceived exertion				
Moderate	13 ± 1	14 ± 1	15 ± 1	16 ± 1
Hot	13 ± 1	16 ± 1	17 ± 1	18 ±1
		T, t, Tt		
Perceived thirst				
Moderate	4 ± 0	5 ± 0	5 ± 0	6 ± 0
Hot	6 ± 0	7 ± 0	7 ± 1	8 ±1
		T*, t		
Perceived thermal comfort				
Moderate	3 ± 1	3 ± 1	3 ± 1	1 ± 2
Hot	4 ± 1	4 ± 1	4 ± 1	2 ± 2

7.3.7. Metabolic responses

Blood glucose concentrations were similar at rest and following the first skill test: however with the onset of intermittent running glucose concentrations increased to a higher concentration in the heat (interaction trial x time P<0.01, $F_{7, 56}$ = 3.7; Figure 7.5). Blood lactate concentrations were not different between trials but were elevated above resting values throughout exercise and were further elevated following the performance of the field hockey skill test (main effect time P<0.01, $F_{7, 56}$ = 45.8; Figure 7.6).
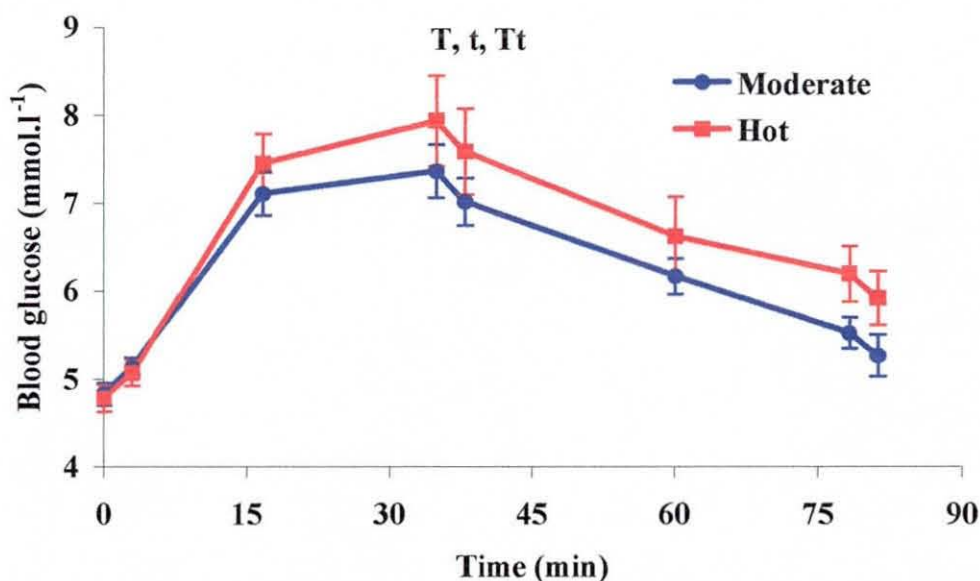
Figure 7.5 Blood glucose concentrations during the hot and moderate trials; T = main effect trial $P<0.05$, $F_{1, 8} = 8.1$; t = main effect time $P<0.01$, $F_{7, 56} = 22.7$; Tt = interaction trial x time $P<0.01$, $F_{7, 56} = 3.7$.
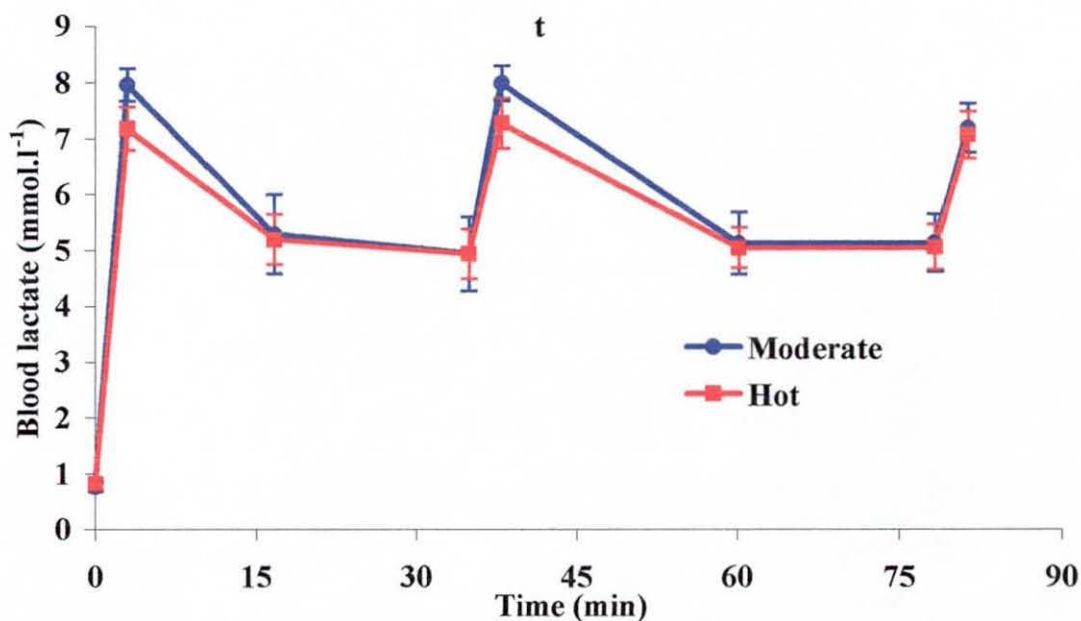
Figure 7.6 Blood lactate concentrations during the hot and moderate trials; t = main effect time $P<0.01$, $F_{7, 56} = 45.8$.

Plasma ammonia concentrations were similar at rest, but increased so that at the end of the first set of LIST, the concentration was significantly higher in the moderate environment (interaction trial x time $P<0.01$, $F_{7, 56} = 2.6$). Throughout the remaining

exercise period the plasma ammonia concentration was similar for both trials, attaining a plateau at approximately 50 µmol.l^{-1} (Figure 7.7).
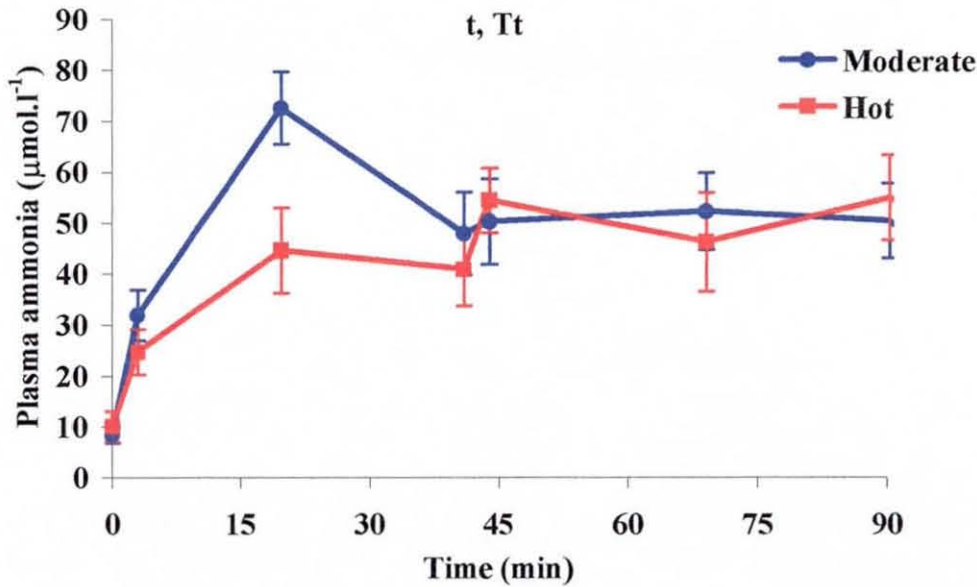
Figure 7.7 Plasma ammonia concentrations during the hot and moderate trials; t = main effect time P<0.01, F$_{7, 56}$ = 14.9; Tt = interaction trial x time P<0.05, F$_{7, 56}$ = 2.6.

Plasma volume changes were not different between the hot and moderate trials. The change in plasma volume was −10.3 ± 3.1 and −7.1 ± 1.9% at the end of the hot and moderate trials respectively. Plasma volume decreased significantly following each of the skill tests (main effect time P<0.01).

7.3.8. Hormonal responses

Serum progesterone concentrations were not different between trials confirming similar menstrual status in both trials (HT 10.58 ± 4.70 vs MT 11.00 ± 4.94 nmol.l^{-1}).

Serum cortisol concentrations and increases in concentration from rest were similar in both environmental conditions and remained above rest throughout exercise (main effect time P<0.01, $F_{7, 56}$ = 8.6). Figure 7.8 shows that serum aldosterone concentrations were similar at rest and increased during the exercise period (main effect time P<0.01, $F_{7, 56}$ = 38.7). The rate of rise was faster in the hot condition such that there was a higher aldosterone concentration at the end of exercise in the hot trial (interaction trial x time P<0.01, $F_{7, 56}$ = 3.4).
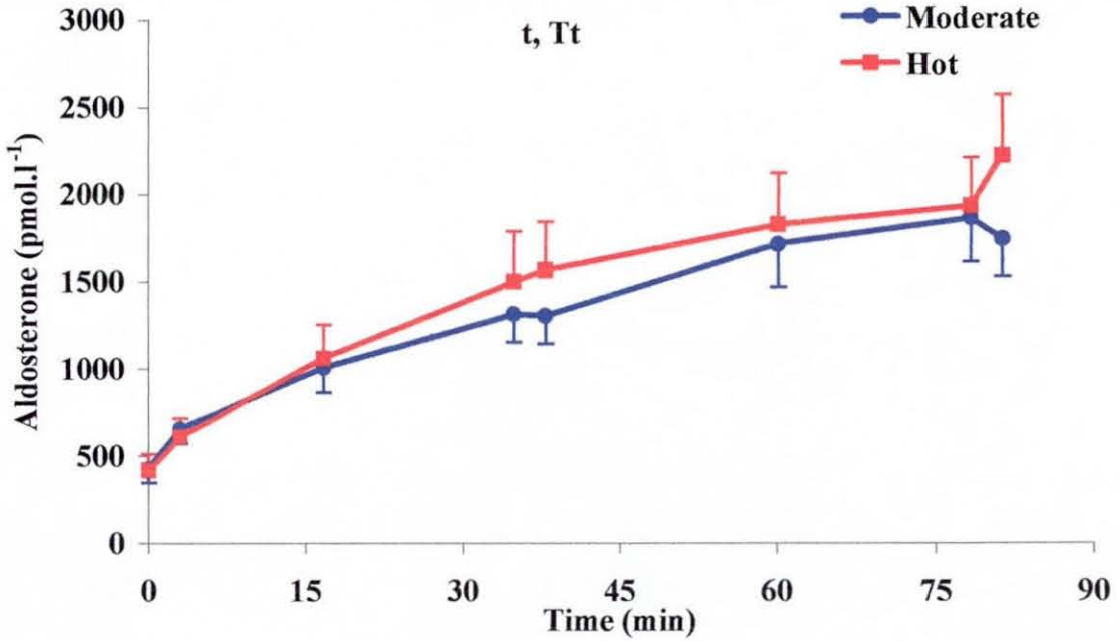
Figure 7.8 Serum aldosterone concentration during the hot and moderate trials; t = main effect time P<0.01, $F_{7, 56}$ = 38.7; Tt = interaction trial x time P<0.01, $F_{7, 56}$ = 3.4.

7.4. Discussion

The main findings of the present study were that hockey skill test performance was decreased following prolonged intermittent high intensity running and that the decrement in performance was greater in the hot environment. However, 'decision making' times were not different following prolonged intermittent high intensity running and were not different between environmental conditions. Deep body temperature, heart rate, rating of perceived exertion, perceived thirst and blood glucose concentrations were significantly higher in the hot trial compared to the response seen in the moderate trial. Repeated 15 m sprint performance was slower

when the environmental conditions were hot. Blood lactate, serum cortisol and aldosterone concentrations did not differ between the hot and moderate trials.

In the present study subjects were dehydrated by 0.57 and 0.39% in the hot and moderate trials, respectively. Previous studies have suggested that decrements in skill performance or cognitive functioning following exercise in hot or moderate conditions are due to dehydration (Gopinathan et al., 1988; McGregor et al., 1999;). McGregor et al. (1999) performed a soccer skill test prior to and following 90 min of prolonged intermittent high intensity running or 6 sets of LIST and showed that when subjects were dehydrated by 2.4% of body mass, there was a decrement in skill performance of 5%. In contrast, following a soccer match in a hot environment, skill test performance has been shown to decline similarly in hyperhydrated and euhydrated subjects even though their total body water content was higher and their thermoregulation was improved in comparison to the euhydrated players (Rico-Sanz et al., 1996). It could be suggested that in the study of Rico-Sanz (1996) the subjects in both hydration states may have been sufficiently dehydrated to reduce skill performance as body mass decreases of >2.5% were recorded and it has been suggested that decreases of only 2% can effect exercise performance and capacity (Armstrong et al., 1985; Sawka et al., 1992).

In the current study, it seems unlikely that the subjects were dehydrated. This was due to the ingestion of 1.00 ± 0.12 and 0.84 ± 0.11 $l.h^{-1}$ of water during exercise in the hot and moderate trials respectively. Furthermore, it has been suggested that in team sports for the prevention of dehydration water intake should be one half of the individual's sweat rate (Gisolfi and Duchman, 1992). For both the hot and moderate trials fluid intakes of 75% of sweat rates were recorded. Furthermore serum aldosterone concentrations were only different between the two environmental conditions at the end of exercise, suggesting that dehydration levels did not differ (Montain et al., 1997). In previous studies in this laboratory, performance of the LIST has been investigated in hot (~30°C, 26% RH) and moderate (~15°C, 54% RH) conditions with and without fluid replacement (Morris, 1999). In comparison to the moderate fluid trial, performance in terms of distance run were 2, 23 and 35% lower in the moderate, no fluid, hot fluid and hot no fluid trials respectively. Osmolality and sodium concentrations were higher and rectal temperatures and heart rates lower in

the 'no water' trials, clearly demonstrating that in the water trials, the water was being absorbed from the stomach, helping to attenuate the thermoregulatory strain of exercising in the heat. Thus it is likely that dehydration did not affect hockey skill performance in the current investigation.

During intermittent sports such as hockey, glycogenolysis is the major metabolic pathway by which energy is transferred to exercising muscles, thus performance is partially dependant upon glycogen stores. Not surprisingly therefore, depletion of muscle glycogen has also been suggested to decrease skill test performance (Rico-Sanz et al., 1996; McGregor et al., 1999) with low muscle glycogen concentrations being recorded after soccer matches (Jacobs et al., 1982; Leatt & Jacobs, 1988). In previous studies in this laboratory muscle glycogen concentrations that have still been high (180 - 190 mmol.kg dry wt^{-1}) following 100 min of the LIST in a hot environment (Morris, 1999) and after 90 min (6 sets) in moderate conditions (Nicholas et al., 1999). As the total exercise time in the present study was approximately 70 min, it is unlikely that the subjects were glycogen depleted. This suggestion is supported by the significant decline in skill after only the first 2 sets of the LIST (30 min). Furthermore, blood lactate concentrations have been suggested to be representative measures of whether or not the rate of energy transferred from glycogen breakdown has decreased as a result of low glycogen concentrations (Jacobs, 1988). In the present study blood lactate concentrations did not differ between trials during the LIST or following the performance of the field hockey skill test. However, it should be recognised that blood lactate concentrations only represent a balance between the appearance and disappearance of blood lactate rather than the actual concentration, but the similar concentrations recorded after the field hockey skill test suggest that there was no difference in rate of glycogenolysis rate during the skill tests or between the two environmental conditions. Therefore, while glycogen concentrations were not measured, it seems unlikely that glycogen depletion occurred in the current study and thus does not seem to be the reason for the decline in skill performance observed.

The similar increase in blood lactate concentrations during the three hockey skill tests and across both environmental conditions suggests that the further decline in skill in the heat and within trials was not due to the build up of acid metabolites. Impaired

skill performance may be due to an accumulation of acid metabolites (Carron, 1972), though the results in the present study can not be attributed to this.

Hypoglycaemia has been associated with a decrease in soccer skill performance as the central nervous system is dependent upon glucose for its metabolism (Shephard and Leatt, 1987). Carbohydrate supplementation has been shown to reduce the deterioration in stroke performance in tennis following a 2 h training session. Although the exact mechanism for this attenuation was unclear, blood glucose concentrations may have been better maintained (Vergauwen et al., 1998). In the present study, blood glucose concentrations were higher in the hot environment compared to the moderate and were maintained above rest throughout. Thus, it seems clear that hypoglycaemia is not the source of the poorer skill performance in the current study in either the hot or the moderate trial.

'Decision making' times in the three skill tests did not differ during the time course of each trial and were not affected by the two environmental conditions. Very few studies have examined the effect of exercise on cognition or psychomotor ability and of these, the relevance of the findings in these studies with respect to the present study which investigated skill performance during 60 min of prolonged intermittent high intensity running is questionable. Nevertheless, Hammerton (1971) suggested that exercise did not affect cognition, but he only used 400 s of submaximal exercise. Sjoberg (1980) investigated training status and found no difference in psychomotor ability or cognition between the two groups at various exercise intensities. Also, it has been suggested by Reilly and Smith (1986) that an inverted-U relationship exists between exercise intensity and cognitive task ability. Whilst cycling at intensities ranging from 25-85% $\dot{V}O_2$ max, individuals completed an arithmetic adding task (Reilly and Smith, 1986). Cognitive function was attenuated at the lowest and highest intensities and was estimated to be optimised at 44% $\dot{V}O_2$ max. However, in the present study the exercise intensity during the skill test did not change. No difference in 'decision making' time is therefore consistent with the findings of Hammerton (1971), Sjoberg (1980) and Reilly and Smith (1986).

Hockey skill performance was poorer in the heat than in moderate environmental conditions. Sport-specific skill performance has previously been shown to be significantly impaired due to heat stress in a study on tennis players (Dawson et al., 1985). Dawson et al. (1985) completed a comparison of tennis skill following a 1 h match in moderate conditions (23°C 64% RH) and following 1 h intervalised treadmill running (which was supposed to mimic the tennis game in moderate conditions) in hot conditions (35°C 65% RH). Skill performance was measured in terms of service, groundstroke and volley power and accuracy. Heat stress resulted in a 19, 26 and 14% decrement in service, groundstroke and volley performance. This decrement in performance was attributed partially to the greater cardiovascular and thermoregulatory strain experienced by the subjects. The decrements in performance were similar to those in the present study, but the tennis study has numerous limitations. Though the treadmill running was designed to represent the work – rest patterns of the match (by heart rate matching), it did not include turning, moving backwards or sidewards or postural changes that are seen in the match. Thus the total work and muscle fibre type recruitment will have differed markedly between the match and the intervalised treadmill running. Furthermore, players were allowed to 'knock up' after the treadmill run in the heat, during which time rectal temperatures would have been decreasing. Thus, the thermal strain prior to the skill tests was unknown and could not be compared.

The responses in the current study of a higher rectal temperature and a decline in sprint performance in the hot trial compared to the moderate are consistent with previous research relating to prolonged intermittent high intensity running in the heat (Morris, 1999). The greater thermal strain on the subjects may partially explain the poorer skill performance in the heat. A critically high deep body temperature has been suggested to induce reductions in the motivation or 'drive' to exercise (Nielsen et al., 1993). Motivation, however, is difficult to define and therefore to test for. The subjects in the current study perceived the exercise to be harder in the heat and tended to feel less comfortable, which may have impacted on their motivation. Field hockey relies heavily on motivation, as individuals self-determine the pace adopted and distances covered in a match. Furthermore a high deep body temperature has been suggested to be the key factor inducing the earlier onset of exhaustion in hot environmental conditions (Nielsen et al., 1993). The high body temperature may have

a physiological impact on brain serotonergic activity (Marvin et al., 1998), muscle function or metabolism (Gonzalez-Alonso et al., 1999), cardiovascular strain and/ or some other mechanism that remains to be elucidated. It seems likely that a higher rectal temperature and thus greater thermoregulatory strain in the heat is associated with the poorer skill performance and 15 m sprint performance. The high rectal temperature ($>39^{\circ}$C) during the moderate trial may also partially explain the decline in skill performance following the LIST.

Heart rate was higher in the heat than in the moderate condition during the LIST. Several studies have found an increase in cardiovascular strain in the heat compared with moderate environmental conditions (Dawson et al., 1985; Morris et al., 1998; Morris et al., 1999). It has been suggested that the increased heart rate in the heat was caused by a fall in central blood volume, and hence cardiac filling pressure and stroke volume, due to the increased cutaneous blood flow required to facilitate heat dissipation. An increased heart rate would thus have been required to maintain cardiac output.

Blood glucose concentrations were also higher in the heat. While hyperglycaemia is often associated with heat stress (Yaspelkis et al., 1993; Morris 1999) the elevated blood glucose concentrations would not appear to be due to a reduced uptake of glucose by muscle, or due to any change in whole body utilisation. Rather it seems it is caused by an increase in glucose output by the liver (Hargreaves et al., 1996). This may be as a result of increased sympathetic stimulation, which has been previously recorded during the LIST (Morris et al., 1999).

Performance of the hockey skill test resulted in a rapid rise in blood lactate concentration to ~9-10 mmol.l^{-1}. The lactate concentration immediately after the skill test was higher than that found after a match or training in Indian senior players, but the concentration during the LIST was representative of that during a hockey match (Ghosh et al., 1991). Furthermore the heart rate ranges during the trials were representative of those seen in field hockey matches suggesting that the LIST was fairly representative of the physiological demands of field hockey (Lothian and Farrally, 1992). The higher blood lactate concentration following the skill test may be due to the long period dribbling the ball (~8 s). Lothian and Farally (1992) found the

majority of hockey related activity (time spent directly involved with the ball) to take place within 2 s and Wein (1981) indicated that 61% of the time on the ball lasted from 0.5 to 2.0 s with only 5% lasting longer than 7 s. However, during competitive soccer matches blood lactate concentrations above 12 mmol.l^{-1} have been recorded. Thus, the blood lactate concentrations in the current study do not seem unduly high. There is a dearth of information regarding the demands of field hockey since the development of sportsturfs and rule changes and thus further research would be beneficial.

A reduction in plasma volume was found to accompany the sharp rise in blood lactate concentration. The movement of plasma volume into muscle cells may occur during intense exercise as the breakdown products of glycolysis result in strong osmotic forces within the muscle cells (Wade and Freund, 1990). Commencement of the LIST, reduced the large negative change in plasma volume; however resting levels were never regained and haemoconcentration was present throughout both the hot and moderate trials.

The results of the present study show that field hockey skill performance is decreased following 30 and 60 min of intermittent, high intensity shuttle running and that this decrease is greater in hot environmental conditions. A greater thermoregulatory strain, reflected by a higher deep body temperature and heart rate, may partially explain the poorer performance in the heat and following the LIST. However, the exact mechanism for this decrement in performance remains to be elucidated, but is unlikely to be due to low glycogen concentration or dehydration as skill declined after only 30 min.

8. Effect of heat acclimation on field hockey skill performance

8.1. Introduction

Previous research presented in Chapter 5 shows that for female hockey players following four 30-45 min heat (~30°C) acclimation sessions, performance of the LIST, in terms of distance run is increased by 33%. It was suggested that this increase in intermittent running capacity was due to a decrease in rectal temperature and a concurrent increase in thermal comfort. In Chapter 7 hockey skill performance was poorer in hot conditions compared with moderate. During field hockey and other games, the performance of skills is as important, if not more important than the maintenance of physiological function. Therefore, if the decrement in skills seen when exercising in the heat can be diminished, it may make the difference in the result of the match and the tournament.

Only 2 previous heat acclimation studies have been completed using both high intensity intermittent running for the acclimation and for the pre- and post-acclimation trials. The study by Dawson and Pyke (1990) was reviewed in Chapter 5, but will be discussed again here as it is the only study that has employed well-trained games players (male field hockey). A 60 min interval treadmill run (34.5°C, 60% RH) was completed prior to and after 2 weeks of normal field hockey training wearing sweat clothing. The responses of the acclimation group were compared with those of players who had completed the same training sessions wearing field hockey kit. After acclimation, both the acclimation group and the normal clothing group responded similarly to the interval treadmill running (Dawson and Pyke, 1990), suggesting there were no thermoregulatory benefits to artificial acclimation wearing a sweat suit. This finding is unsurprising, as Dawson and Pyke (1988) had previously outlined that the suit did not provide as larger thermal strain as exercising in normal clothing in the heat (34.5°C, 60% RH). In a laboratory based study, Armstrong et al. (1989) examined the responses to 8 days of 100 min of intermittent running (2, 5, 8 and 10 min walk, run [68% $\dot{V}O_2$ max] and rest periods; 41.2°C, 39.0% RH). After the acclimation period, there was a decrease in cardiovascular and thermoregulatory

strain, evidenced by lower heart rates, rectal temperatures and skin temperatures decreased. However, there were no differences in hormonal activity of aldosterone, cortisol or renin. While, the intermittent running was a clear stimulator of heat acclimation, the subjects employed by Armstrong et al. (1989) were untrained.

Research shows that motor skill performance is decreased following high intensity intermittent exercise in a hot environment (Rico-Sanz et al., 1996 and Dawson et al. 1985; Chapter 7). Soccer skill test performance was shown to decline in elite players following a soccer match in the heat (Rico-Sanz et al., 1996). Also, performance of tennis skills were 19, 26 and 14% poorer respectively for serving, groundstrokes and volleys after 1 h of interval treadmill running in the heat (35°C, 65% RH) compared with 1 h of game play in moderate conditions (23°C, 65% RH; Dawson et al., 1985). While the scientific control of the studies by Rico-Sanz et al. (1996) and Dawson et al. (1985) is questionable, clearly this field-based testing suggests that skill performance is decreased when thermoregulatory strain is high.

In the recent literature, there has been an increase in the use of prolactin as a marker of serotonergic activity (Brisson et al., 1989; Brisson et al., 1991; Marvin et al., 1998; Pitsalidis et al., 1998; McGregor, 1999). An increase in serotonergic activity or central fatigue may be implicated in poorer skill performance following prolonged exercise by altering the arousal, lethargy and mood of the player (Davis and Bailey, 1997). Thus, serum prolactin was measured in the current study to try to identify a relationship, if any between central fatigue and skill performance following prolonged intermittent shuttle running in the heat.

No previous studies have examined the effect of heat acclimation on performance of motor skills. However, skill performance is decreased in hot conditions and high intensity intermittent heat acclimation has been shown to decrease thermoregulatory strain. Thus, this study tests the hypothesis that 4 short heat acclimation sessions, which have been shown to increase intermittent running capacity, improve field hockey skill performance.

8.2. Methods

8.2.1. Subjects

Eight, well-trained, unacclimatised female university hockey players volunteered to participate in this study. Six of the eight had normal menstrual cycles, and two had been taking oral contraceptives for over two years. The physical characteristics of the subjects are presented in Table 8.1.

Table 8.1 Physical characteristics of the female hockey players who participated in the study.

	Mean ± SEM
Age (years)	21.8 ± 0.4
Mass (kg)	63.1 ± 2.5
Height (cm)	167.3 ± 1.6
Estimated $\dot{V}O_2$ max ($ml.kg^{-1}.min^{-1}$)	50.7 ± 1.4

8.2.2. Experimental design

Subjects performed 3 main trials in hot environmental conditions (~30°C), 28 days apart. Two main trials were completed before and after acclimation and required the subjects to complete 4 sets of the Loughborough Intermittent Shuttle Test (LIST; Nicholas et al., 1995, 2000) and 3 field hockey skill tests. During the LIST subjects exercised over a 20 m distance and repeated a walk, sprint, cruise (~85% $\dot{V}O_2$ max) and jog (~50% $\dot{V}O_2$ max) pattern of exercise until 11 sprints had been completed. The 11 sprints took approximately 15 min and were followed by a 3 min rest period. This series of activities constituted 1 Set of LIST. A field hockey skill test was completed prior to and after 2 and 4 Sets of the LIST. The field hockey skill test required the players to complete 6 repetitions of dribbling, a pass and a shot (Figure 8.2; Full description Chapter 6). Following the 2nd set of the LIST the rest was 10 min to mimic the half time period of a hockey match. Exercise was terminated prior to the

completion of the trial if rectal temperature reached 40°C or exhaustion. In the ten days prior to the post acclimation trial, all the subjects completed 4 acclimation sessions (30°C) on separate days of 2 sets of the LIST (Figure 8.1). A third main trial or control trial was also completed which required the subjects to complete the 3 skill tests at identical times to the other main trials, but with passive recovery between skill tests rather than intermittent running. The timing of this trial was randomised so that half the subjects completed the control trial before the acclimation.

1   18   28

■ Main trial

□ Normal training

■ Acclimation session

■ Rest

Figure 8.1 Protocol of the 28 days of the acclimation study.

8.2.3. Preliminary measurements

Maximal oxygen uptake ($\dot{V}O_2$ max) was estimated using the multistage fitness test (Ramsbottom et al., 1988). From this estimate of $\dot{V}O_2$ max, running speeds to elicit 85 and 50% $\dot{V}O_2$ max were determined using the tables for predicted $\dot{V}O_2$ max values (Ramsbottom et al., 1988). Subjects were then familiarised with the LIST at ~30°C for 2 Sets or ~30 min and were fully familiarised with the field hockey skill test on 2 separate days.

8.2.4. Main trials

Subjects reported to the laboratory, following a 12 h overnight fast. In the 2 days prior to each main trial subjects were encouraged to consume the same diet. All experiments were arranged so that each individual ran at the same time of day for both main trials to control for circadian influences. A 45 mm cannula (Venflon 2, BOC Ohemeda AB) was then inserted into a forearm vein of the subject under local anaesthetic (Lignocaine hydrochloride 1% w/v, Antigen pharmaceuticals Ltd.). The cannula was kept patent with saline solution (Sodium choloride 0.9% w/v, Antigen pharmaceuticals Ltd.). Nude body mass was recorded and a rectal probe (Edale Instruments Ltd.) was inserted to a depth of 10 cm beyond the anal sphincter.

Figure 8.2 shows the main trial protocol. Fifteen minutes after cannulation, during which time subjects remained standing, a 'resting' blood sample was collected. Subjects then moved into the gymnasium and a resting rectal temperature was recorded. A standardised warm-up of ~15 min was performed which consisted of jogging, stretching and faster pace running.
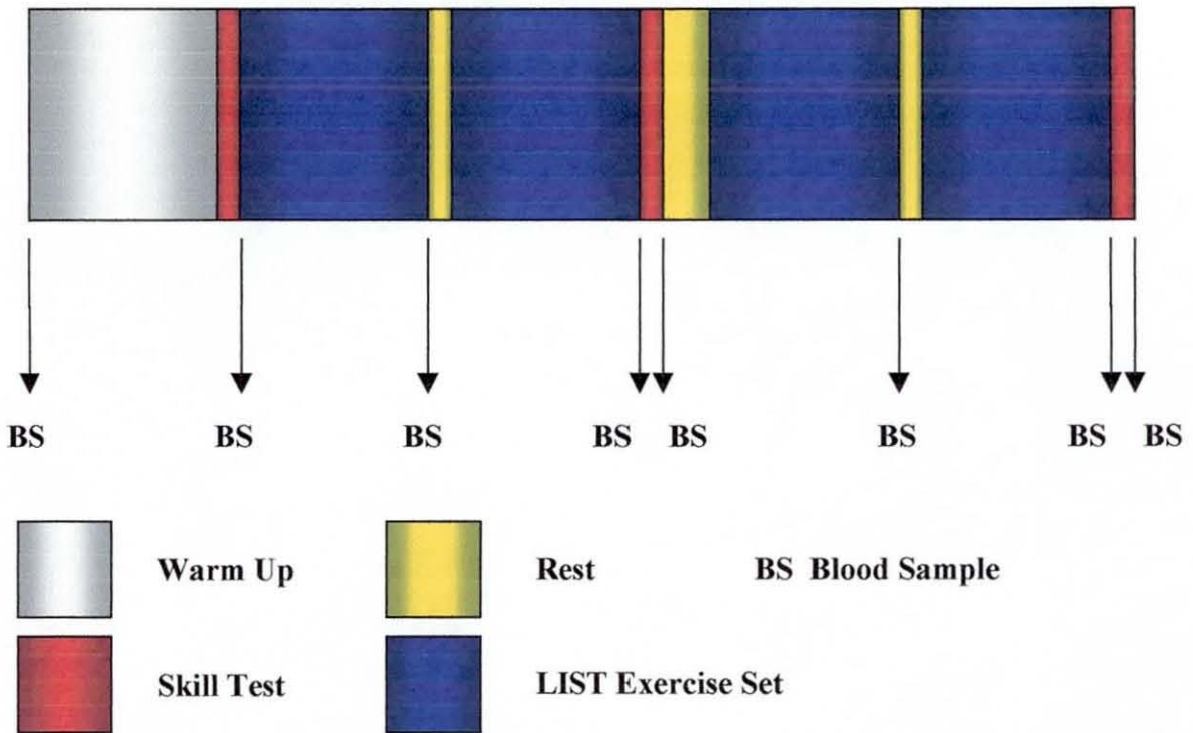
Figure 8.2 Protocol diagram of the main trials.

Sprint times over 15 m were measured using 2 infra-red photo electric cells (RS Components Ltd.) connected to the microcomputer. Skill test time was recorded using a stopwatch and 'decision making' time initiated by infra-red photo electric cells and terminated by the sound of the ball hitting the backboard using a microphone connected to a computer.

A whirling hygrometer (Brannan Thermometers Ltd.) was used to measure atmospheric dry and wet bulb temperatures during the main trials and acclimation sessions. Heart rate was continuously monitored throughout each main trial using short range telemetry (Vantage NV, Polar Electro Fitness Technology, sampling frequency 5s). Rating of perceived exertion, thirst and thermal comfort were recorded prior to the 11th sprint in each LIST exercise set. At the same time perceived heat and thirst were recorded using an integer scale. A 12 ml blood sample was collected from each subject before and after each field hockey skill test and after the 1st and 3rd Sets of the LIST. Rectal temperatures were measured during the 4th and 8th cycle of each set and concurrently with each blood sample. When rectal temperatures were

measured during the LIST exercise Set, subjects were stationary for 40 m of the 60 m walk in that cycle.

8.2.5. Blood sampling and analysis

Five ml of blood was dispensed into an EDTA tube and aliquots from the venous sample were used for determination of haematocrit and haemoglobin concentration. Changes in plasma volume (%) were estimated using the method of Dill and Costill (1974). A 1.5 ml aliquot of blood was dispensed into an eppendorf tube for immediate analysis of blood lactate and glucose using a fully automated analyser (Yellow Springs Instruments).

Five ml of blood was also dispensed into a serum tube for determination of progesterone, aldosterone, prolactin and cortisol concentration by using commercially available radio immunoassay kits (Diagnostic Products Corporation).

8.2.6. Statistical analyses

A two-way analysis of variance with repeated measures was used to establish if any significant differences existed between subject response in terms of physiological and metabolic parameters to the performance of the LIST and the field hockey skill test. Where necessary, significant differences in the way subjects responded to the LIST and the skill test were located using Post-hoc Tukey tests. A students t-test was also used where appropriate. Data are presented as means ± standard error (SEM). The mean results are for an n of 8, except for blood sample results which are for an n of 7.

8.3.   Results

8.3.1. Environmental conditions

Dry bulb (T_{db}) and wet bulb (T_{wb}) temperatures were not different in the pre, post and control trials (T_{db}: Pre 30.7 ± 0.3 vs Post 30.9 ± 0.1 vs Control 31.0 ± 0.3°C; T_{wb}: Pre 18.5 ± 0.5 vs Post 17.9 ± 0.3 vs Control 17.9 ± 0.2°C). Relative humidity (RH) was

not different between the trials (Pre 29.7 ± 1.9 vs Post 26.2 ± 1.4 vs Control 26.1 ± 1.6%).

8.3.2. Field hockey skill performance

Figure 8.3 shows that field hockey skill performance was not different between trials prior to the commencement of the LIST. Skill performance was poorer before acclimation than after acclimation or during the control trial (pre: 93.81 ± 1.48, 95.85 ± 1.26 and 96.64 ± 1.20 s; post: 93.22 ± 1.19, 93.49 ± 1.40 and 92.86 1.63 s; main effect trial $P<0.01$, $F_{2, 14} = 15.2$). Following 30 and 60 minutes of intermittent running skill test performance declined prior to acclimation, but was maintained following acclimation and during the control trial (interaction trial x time $P<0.01$, $F_{4, 28} = 5.2$). Skill performance during the control trial at 30 and 60 min was greater than the pre- and post-acclimation trials (interaction trial x time $P<0.01$, $F_{4, 28} = 5.2$, post hoc $P<0.05$). There was no difference is 'decision making' time following the LIST or between the trials (Table 8.2).
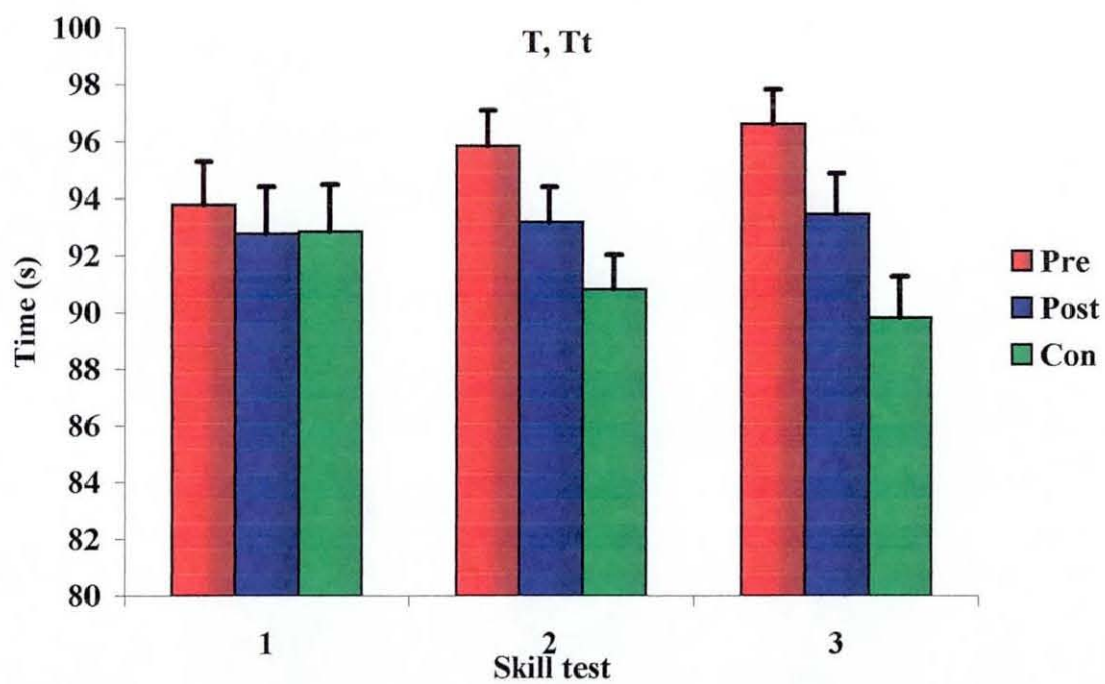
Figure 8.3 Overall skill test performance before and after acclimation and during the control trial; T = main effect trial P<0.01, $F_{2, 14}$ = 15.2; Tt = interaction trial x time P<0.01, $F_{4, 28}$ = 5.2.

Table 8.2 'Decision making' time before and after acclimation and during the control trial.

Time (s)	Skill test no		
	1	2	3
Pre acclimation	4.23 ± 0.19	4.20 ± 0.12	4.33 ± 0.11
Post acclimation	4.21 ± 0.14	4.17 ± 0.12	4.25 ± 0.15
Control	4.39 ± 0.15	4.31 ± 0.06	4.24 ± 0.24

8.3.3. Fifteen metre maximal sprint performance

Maximal sprint performance over 15 m declined during the LIST but did not differ before and after acclimation (main effect time $P<0.01$, $F_{3, 21} = 23.9$; Table 8.3).

Table 8.3 Fifteen metre maximal sprint time before and after acclimation; $t =$ main effect time $P<0.01$, $F_{3, 21} = 23.9$.

	Set 1	Set 2	Set 3	Set 4
Pre acclimation	2.77 ± 0.03	2.91 ± 0.07	2.99 ± 0.09	2.97 ± 0.07
Post acclimation	2.81 ± 0.06	2.91 ± 0.08	3.02 ± 0.07	3.00 ± 0.07
			t	

8.3.4. Rectal temperature

Figure 8.4 and Figure 8.5 show the rectal temperature response over time and the increase in rectal temperature before and after acclimation and during the control trial. Rectal temperature was higher in the pre- and post-acclimation trials than the control trial (main effect trial $P<0.01$, $F_{2, 12} = 104.8$) and continued to increase throughout the pre- and post-acclimation trials (main effect time $P<0.01$, $F_{15, 90} = 96.8$). There was a tendency for rectal temperature to be lower after acclimation towards the end of exercise, however there was only a significantly lower temperature at 73 min (interaction trial x time, $P<0.01$, $F_{30, 180} = 37.5$, post hoc $P<0.05$).
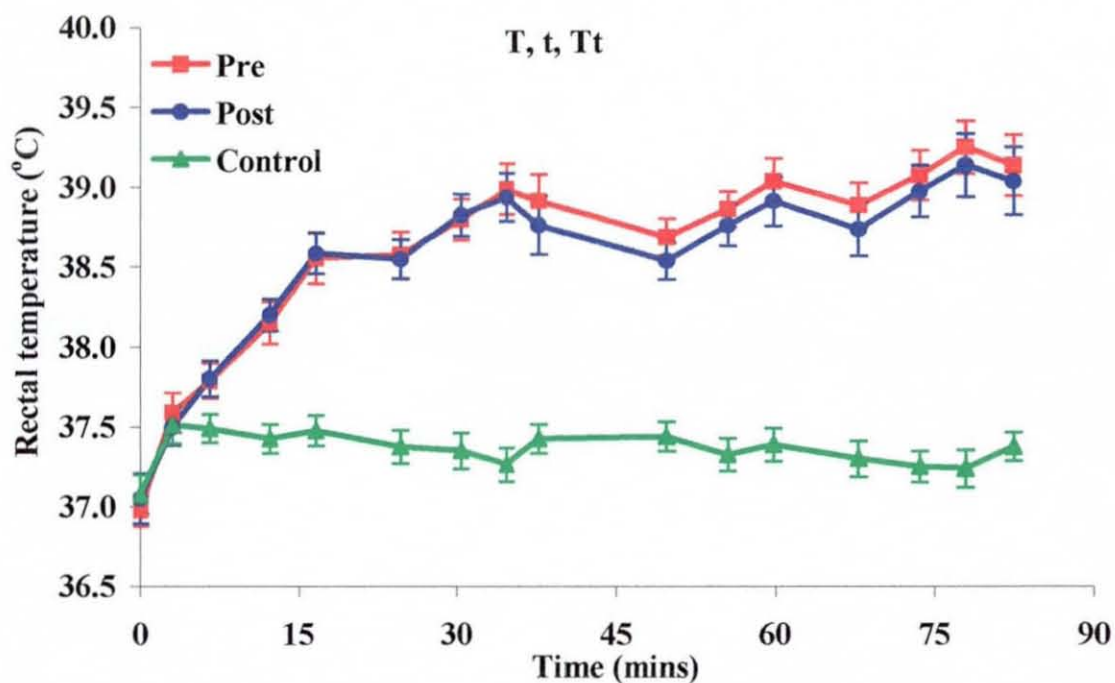
Figure 8.4 Rectal temperature before and after acclimation and during the control trial; T = main effect trial P<0.01, $F_{2, 12}$ = 104.8; t = main effect time P<0.01, $F_{15, 90}$ = 96.8; Tt = trial x time interaction P<0.01, $F_{30, 180}$ = 37.5.
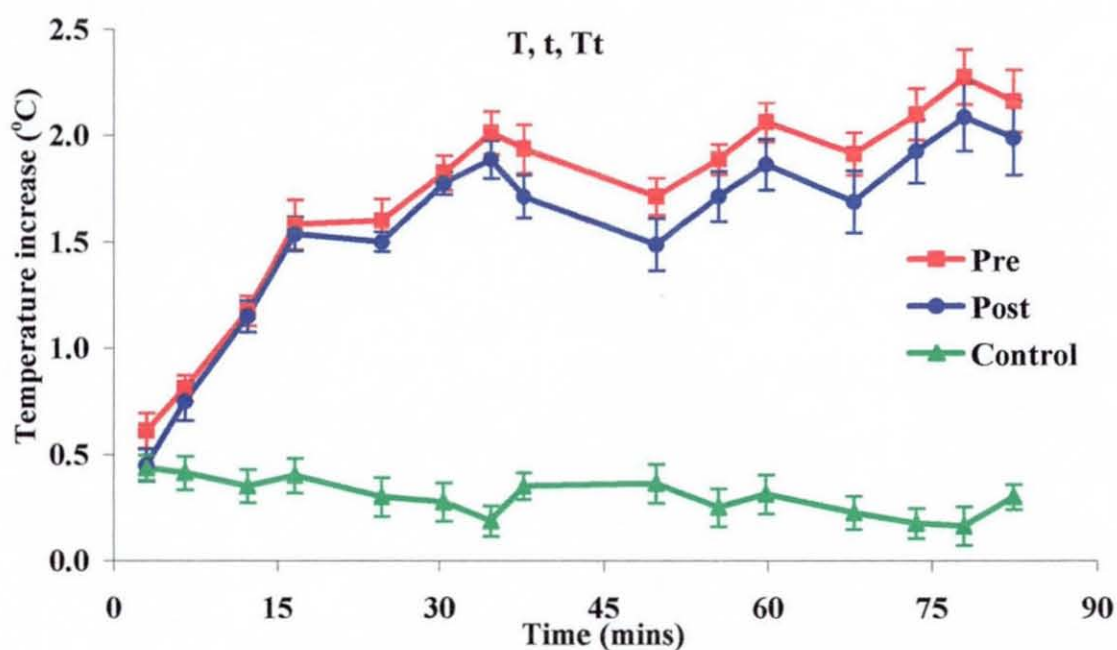
Figure 8.5 Rectal temperature increase before and after acclimation and during the control trial; T = main effect trial P<0.01, $F_{2, 14}$ = 126.8; t = main effect time P<0.01, $F_{14, 98}$ = 62.5; Tt = trial x time interaction P<0.01, $F_{28, 196}$ = 32.6.

8.3.5. Heart rate

As Table 8.4 shows, average heart rate was considerably higher during the pre- and post-acclimation trials than the control trial (main effect trial $P<0.01$, $F_{2, 14} = 521.2$). Average heart rate increased throughout the LIST during the exercise trials, but did not differ before and after acclimation (main effect time $P<0.01$, $F_{3, 21} = 5.7$). Average maximum heart rates during the skill tests were higher during the pre- and post-acclimation trials than the control trial (main effect trial $P<0.01$, $F_{2, 14} = 16.0$; Figure 8.6). Maximum heart rate did not differ between trials during the first skill test (interaction trial x time $P<0.01$, $F_{4, 28} = 23.5$). Maximum heart rate was higher during the post-acclimation test compared with the pre-acclimation test for the second skill test (interaction trial x time, $F_{4, 28} = 23.5$, post hoc $P<0.05$), there were no differences during the final skill test.

Table 8.4 Heart rate during the LIST before and after acclimation and during the equivalent time period during the control trial; T = main effect trial $P<0.01$, $F_{2, 14} = 521.2$; t = main effect time $P<0.01$, $F_{3, 21} = 5.7$; Tt = trial x time interaction $P<0.01$, $F_{6, 42} = 13.8$.

	Set 1	Set 2	Set 3	Set 4
Pre acclimation	176 ± 3	179 ± 3	180 ± 3	183 ± 3
Post acclimation	177 ± 2	182 ± 2	182 ± 2	184 ± 3
Control	107 ± 3	100 ± 2	105 ± 3	101 ± 3
		T, t, Tt		
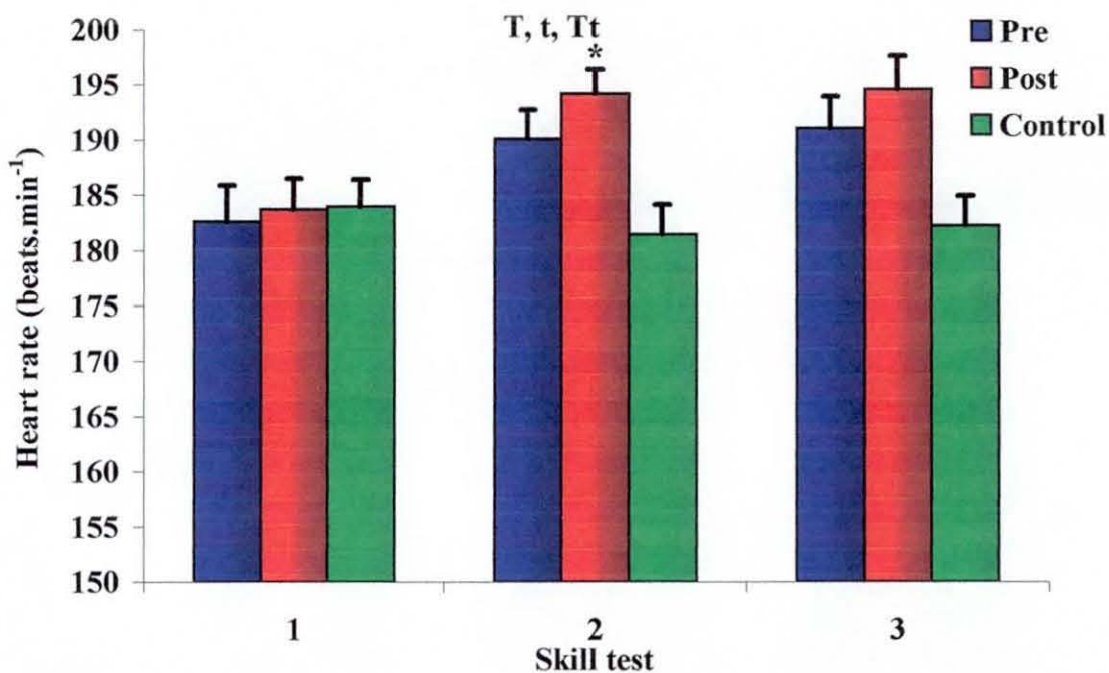
Figure 8.6 Average maximum heart rate during the field hockey skill tests during the pre, post and control trials; T = main effect trial P<0.01, $F_{2, 14}$ = 16.0; t = main effect time P<0.01, $F_{2, 14}$ = 13.3; Tt = interaction trial x time P<0.01, $F_{4, 28}$ = 23.5; * post-hoc P<0.05 from pre-acclimation trial during skill test 2.

8.3.6. Rating of perceived exertion, thirst and thermal comfort

Rating of perceived exertion, thirst and thermal comfort were higher during the pre- and post-acclimation trials than the control trial (main effect trial P<0.01, RPE $F_{2, 14}$ = 385.1, thirst $F_{2, 14}$ = 67.2, $F_{2, 14}$ = 25.1; Table 8.5). There was a tendency for perceived thermal comfort to be lower following acclimation compared with before acclimation (P = 0.07). If sets 3 and 4 alone were considered (post-hoc test) perceived heat was lower (P<0.01) after acclimation.

Table 8.5 Perceived ratings of exertion, thirst and thermal comfort during the pre, post and control trials; T = main effect trial P<0.01, RPE $F_{2, 14} = 385.1$, thirst $F_{2, 14} = 67.2$, thermal comfort $F_{2, 14} = 25.1$; t = main effect time P<0.01, RPE $F_{3, 21} = 52.3$, thirst $F_{3, 21} = 39.2$, thermal comfort $F_{3, 21} = 9.6$; Tt = interaction trial x time P<0.01, RPE $F_{6, 42} = 13.1$, thirst $F_{6, 42} = 7.0$, thermal comfort $F_{6, 42} = 6.4$.

	Set 1	Set 2	Set 3	Set 4
Rate of perceived exertion				
Pre acclimation	14 ± 1	16 ± 0	17 ± 0	18 ± 1
Post acclimation	14 ± 0	16 ± 0	17 ± 0	17 ± 1
Control	7 ± 0	6 ± 0	6 ± 0	6 ± 0
		T, t, Tt		
Perceived thirst				
Pre acclimation	6 ± 1	7 ± 0	7 ± 0	8 ± 1
Post acclimation	6 ± 0	7 ± 0	7 ± 0	7 ± 0
Control	3 ± 0	3 ± 0	3 ± 0	2 ± 0
		T, t, Tt		
Perceived thermal comfort				
Pre acclimation	5 ± 0	6 ± 1	8 ± 1	8 ± 1
Post acclimation	4 ± 0	6 ± 0	5 ± 1	5 ± 1
Control	2 ± 1	2 ± 1	2 ± 1	2 ± 1
		T, t, Tt		

8.3.7. Body mass, fluid consumption and estimated sweat rate

Water consumption did not differ between the pre- and post-acclimation trials, but was approximately double that ingested during the control trial (Pre 0.90 ± 0.12 vs Post 0.86 ± 0.11 vs Control 0.45 ± 0.05 l.h^{-1}; main effect trial P<0.01, $F_{2, 14} = 19.0$). Estimated sweat rate was almost three times as great during the pre- and post-trials trials than the control trial (Pre 1.25 ± 0.11 vs Post 1.25 ± 0.09 vs Control 0.42 ± 0.04 l.h^{-1}; main effect trial P<0.01, $F_{2, 14} = 79.5$). Thus, water consumption replaced 75 ± 12, 69 ± 9 and $118 \pm 19\%$ of the sweat loss during the pre-acclimation, post-acclimation and control trials respectively (main effect trial P<0.01, $F_{2, 14} = 9.4$). Actual body mass loss was not different before and after acclimation, but on average mass increased during the control trial (Pre 0.49 ± 0.23 vs Post 0.54 ± 0.14 vs Control

-0.04 ± 0.10 kg; main effect trial P<0.01, $F_{2, 14} = 13.4$). These mass changes represented on average 0.74 ± 0.35, 0.84 ± 0.23 and $-0.08 \pm 0.17\%$ of pre exercise body mass in the pre, post and control trials respectively (main effect trial P<0.01, $F_{2, 14} = 13.4$).

8.3.8. Hormonal responses

Resting serum progesterone concentrations were not different between trials (Pre 4.5 ± 1.8 vs Post 3.1 ± 0.4 vs Control 6.3 ± 2.3 nmol.l^{-1}) confirming that menstrual cycle phase was the same in all 3 trials. Serum cortisol concentration did not differ between the three trials as a whole. However, cortisol concentration was lower during the control trial both before and after the final skill test (interaction trial x time P<0.01, $F_{14, 84} = 6.8$, post-hoc P<0.05; Table 8.6). Serum aldosterone concentration during the post-acclimation trial was not different from the pre-acclimation trial but was higher than the control trial (main effect trial P<0.05, $F_{2, 12} = 4.0$; Table 8.6). Serum prolactin concentration was lower in the control trial than the pre- and post-acclimation trials (main effect trial P<0.01, $F_{2, 12} = 13.4$; Table 8.6).

Table 8.6 Serum cortisol, aldosterone and prolactin concentrations during the pre, post and control trials; T = main effect trial $P<0.05$, cortisol $F_{2, 12} = 3.7$, aldosterone $F_{2, 12} = 4.0$; T* = main effect trial $P<0.01$, $F_{2, 12} = 13.4$; t = main effect time $P<0.01$, cortisol $F_{7, 42} = 4.1$, aldosterone $F_{7, 42} = 28.0$, prolactin $F_{7, 42} = 9.6$; Tt = trial x time interaction $P<0.01$, cortisol $F_{14, 84} = 6.8$, aldosterone $F_{14, 84} = 6.3$, prolactin $F_{14, 84} = 8.5$.

	Rest	End skill 1	End skill 2	End skill 3
Serum cortisol (nmol.l^{-1})				
Pre acclimation	700 ± 91	666 ± 113	961 ± 113	1134 ± 131
Post acclimation	846 ± 155	806 ± 186	1091 ± 170	1421 ± 237
Control	1002 ± 208	851 ± 183	762 ± 214	553 ± 151
		t, Tt		
Serum aldosterone (pmol.l^{-1})				
Pre acclimation	283 ± 44	591 ± 84	1233 ± 235	1915 ± 249
Post acclimation	390 ± 57	684 ± 92	1498 ± 228	2177 ± 274
Control	528 ± 143	726 ± 159	911 ± 193	1062 ± 246
		T, t, Tt		
Serum prolactin (mIU.l^{-1})				
Pre acclimation	300 ± 45	256 ± 35	988 ± 304	1206 ± 254
Post acclimation	308 ± 24	257 ± 15	1148 ± 250	1321 ± 197
Control	302 ± 32	267 ± 25	264 ± 51	246 ± 56
		T*, t, Tt		

8.3.9. Metabolic Responses

Figure 8.7 shows that blood glucose concentrations remained relatively stable during the control trial and increased similarly during the pre- and post-acclimation trials until the second skill test (main effect trial $P<0.01$, $F_{2, 12} = 33.7$; main effect time $P<0.01$, $F_{7, 42} = 15.2$). Blood lactate concentration showed a hierarchical response and was highest post-acclimation and lowest during the control trial (main effect trial $P<0.01$, $F_{2, 12} = 10.4$; Figure 8.8). Blood lactate increased rapidly following each field hockey skill test (main effect time $P<0.01$, $F_{7, 42} = 80.5$).
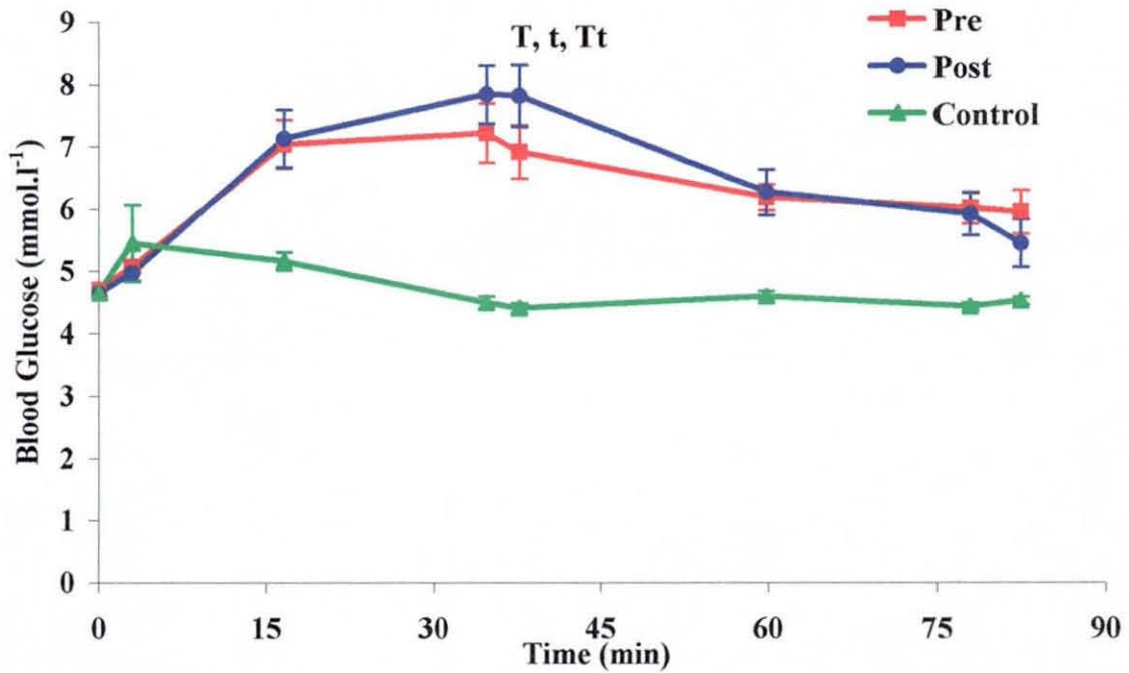
Figure 8.7 Blood glucose concentration during the pre, post and control trials; T = main effect trial P<0.01, $F_{2, 12} = 33.7$; t = main effect time P<0.01, $F_{7, 42} = 15.2$; interaction trial x time P<0.01, $F_{14, 84} = 8.4$.
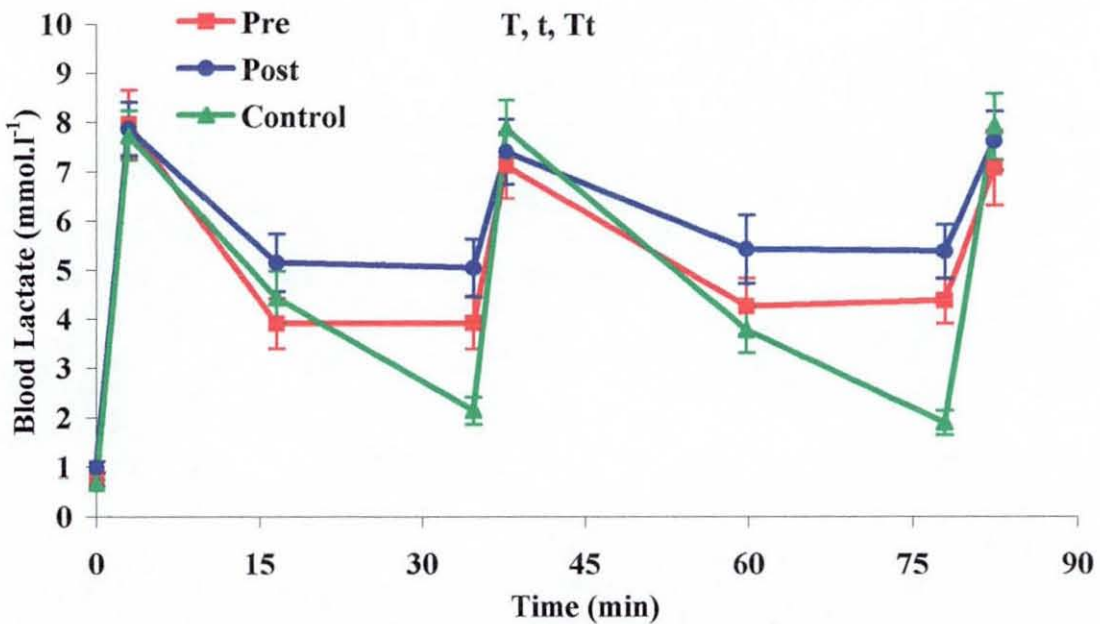
Figure 8.8 Blood lactate concentration during the pre, post and control trials; T = main effect trial P<0.01, $F_{2, 12} = 10.4$; main effect time P<0.01, $F_{7, 42} = 80.5$; interaction trial x time P<0.01, $F_{14, 84} = 7.4$.

8.3.10. Plasma volume

Resting plasma volume was not different between the trials (Pre 61.2 ± 0.4 vs Post 61.1 ± 0.4 vs Control 61.3 ± 0.5 ml). Estimated change in plasma volume throughout the trial was greater during the pre- and post-acclimation trials than the control trial (main effect trial $P<0.01$, $F_{2, 12} = 20.2$; Figure 8.9). The completion of the field hockey skill test resulted in haemoconcentration, which did not differ between the trials (main effect time $P<0.01$, $F_{6, 36} = 24.2$).
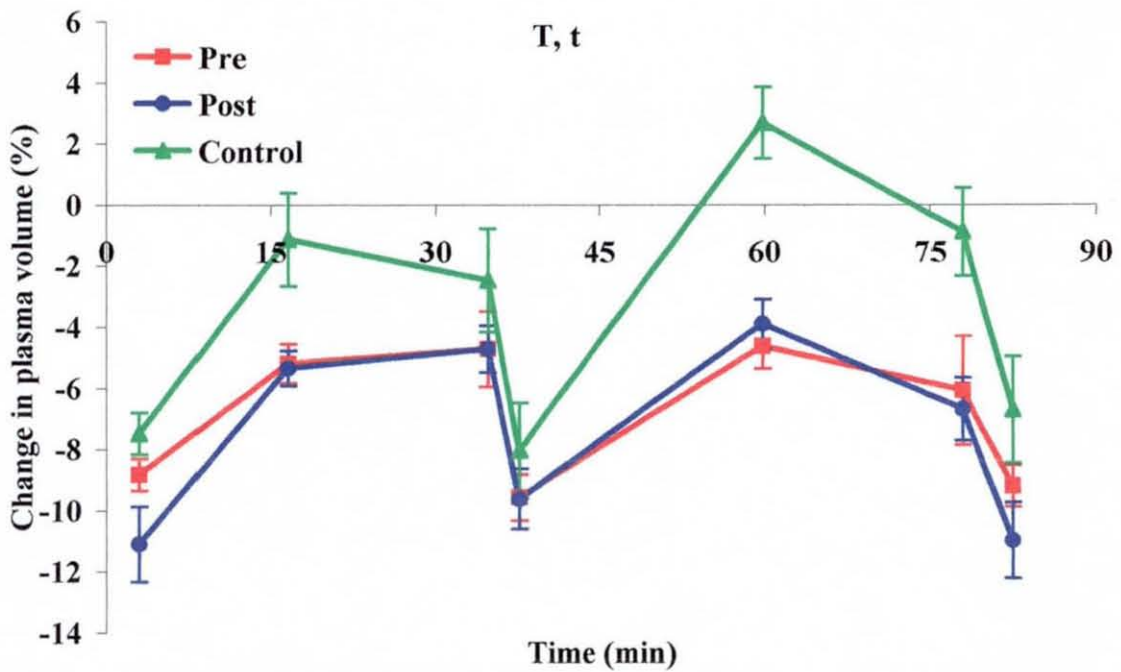
Figure 8.9 Estimated change in plasma volume during the pre, post and control trials; T = main effect trial $P<0.01$, $F_{2, 12} = 20.2$; t = main effect time $P<0.01$, $F_{6, 36} = 24.2$.

8.4. Discussion

The main finding of the present study was that following acclimation, performance of a field hockey skill test following 30 and 60 min of intermittent running was improved. However, there were no differences in decision times recorded in the skill tests. Furthermore, following acclimation there was greater thermal comfort and a tendency for a decrease in rectal temperature. There were no differences in serum aldosterone, cortisol and prolactin concentrations during the intermittent running or the skill tests before and after acclimation. Similarly, no differences were found in plasma volume, estimated sweat rate or change in body mass between these two trials. The present study also showed that the field hockey skill test was reproducible when performed 3 times in succession in one day, and did not have a detrimental or improvement effect on the performance of the next skill test, since overall time and decision time were the same in each of the field hockey skill tests during the control trial. Similarly the metabolic and hormonal responses did not differ between the skill tests in the control trial. However, the study clearly demonstrated a decrease in hockey skill performance during exercise and heat stress compared to passive rest in the same conditions.

There are clearly numerous possible explanations for the decrements in skill seen in the field hockey skill test following intermittent running. Previous explanations for skill decrement has included low glycogen concentrations, hypoglycaemia, dehydration and a critically high deep body temperature and were all discussed in Chapter 7. It has also been suggested that eccentric muscle damage may play a role in the decline in soccer skill performance following 90 min of the LIST (McGregor, 1999). Following exercise-induced muscle damage, isometric muscle strength and motor skill in the form of tracking ability have been shown to decline (Pearce et al., 1998). Thompson et al. (1999) investigated the effects of 90 min or 6 sets of the LIST on muscle soreness and markers of muscle damage in habitually active non-soccer players. The LIST requires subjects to sprint maximally, accelerate and decelerate and change direction, and thus has a large and extensive eccentric component (Thompson et al., 1999). Muscle soreness was determined using an arbitrary scale both at rest and during concentric contractions (75% 1RM) during a hamstring curl, squat, shoulder press, lateral pull and bicep curl. Twenty-four hours following the LIST, subjects'

general and active soreness was considerably greater than that in a control group who did not complete any exercise. In addition, markers of muscle damage were elevated. These findings clearly show that the LIST does cause muscle damage, probably as a result of the large number of eccentric muscle actions. However, the suggestion by McGregor (1999) that the eccentric muscle damage may be a cause of the decrement in soccer skill is questionable. The subjects used in the study by Thompson et al. (1999) were chosen specifically as they were not games players and were thus not used to the type of exercise in the LIST and were not familiarised with the protocol. Following only a single bout of eccentric exercise rapid adaptation has been shown to occur (Clarkson et al., 1987). Therefore you would expect that a group of soccer or hockey players who are used to the demands of interspersing bouts of sprinting and direction changes in both training and matches would not suffer the same extent of muscle damage as the group employed by Thomson and colleagues. Following the completion of the LIST to exhaustion by games players, in environmental conditions similar to those employed in the current study, concentric knee extension and flexion and peak torque values were not different to pre-exercise values (Morris, 1999). Furthermore, the subjects are fully familiarised one week prior to the commencement of the main trial. Subjective opinions from the subjects were that the day following the test they were not stiff or sore. Also following acclimation there was an improvement in skill test performance even though the number of eccentric muscle actions will have been the same in both main trials. As the acclimation period was in the 10 days prior to the second trial, it could be suggested that the acclimation sessions may have caused muscle damage and therefore would have been expected to have a detrimental effect on the second main trial. This was not observed and thus suggests that eccentric muscle damage is not responsible for the decrement in skill performance observed before acclimation in the current study and in Chapter 7.

Serum aldosterone and prolactin concentrations were not different before and after acclimation, but were lower during the control trial. Aldosterone has been well documented not to differ following acclimation in well-trained subjects, such as those employed in the present study (Houmard et al., 1990; Nielsen et al., 1993). The lack of change in aldosterone with acclimation is consistent with the results presented in Chapter 5. Serum prolactin concentration has been suggested to be determined by deep body temperature and has been used as a marker of central serotonergic activity

(Brisson et al., 1989; Marvin et al., 1998). Thus, as there was no significant difference in rectal temperature, this partially explains the similar prolactin responses recorded in the present study. Central fatigue has been suggested to be a possible mechanism for both decrements in skill and the early onset of fatigue in the heat (Nielsen et al., 1993; Marvin et al., 1998; McGregor, 1999; Morris, 1999). Central fatigue has been defined as a negative central influence that exists despite the subjects full motivation or more objectively, a force generated by voluntary muscular effort that is less than that produced by electrical stimulation (Davis and Bailey, 1997). Increases in brain serotonin have effects on the arousal, lethargy and mood of a person and thus an increase in serotonin will impair central nervous system function and thus decrease performance. However, as serum prolactin concentrations did not differ between the two trials, there seems to be no difference in the levels of central fatigue which seems to be unaffected by heat acclimation. This finding is in agreement with that of McGregor (1999) who recorded that there was no evidence of serotonin being a cause of the difference observed in soccer skill performance during fluid and no fluid trials. In contrast, Marvin et al. (1998) recorded a much lower prolactin concentration following head cooling, even though rectal temperature and metabolic parameters were similar, which resulted in more than a 50% improvement in cycling capacity (75% $\dot{V}O_2$ max). The external facial cooling will not have differed in the current study before and after acclimation and therefore would not impact on prolactin concentrations and serotonergic activity. However, sweating rates in specific areas of the body and redistribution of blood flow were not measured and therefore may have altered following acclimation. If any alterations did occur, they did not impact upon serotonergic activity, as this was similar both before and after acclimation. Thus, it seems unlikely that central fatigue is decreased following the acclimation period.

Serum cortisol concentrations were only higher during the exercise trials at the end of 60 min of the LIST. Several authors have outlined how serum cortisol concentrations are related to exercise intensity (Farrel et al., 1983; Vanhelder et al., 1985; Buono et al., 1986). However, this does not seem to be the case in the present study as similar cortisol concentrations were recorded during the LIST and during passive rest. This suggests that cortisol concentrations may not be a good indicator of stress during high intensity intermittent exercise as clearly exercise intensity was considerably different

between the pre- and post-acclimation trials and the control trial. Pollard (1995) outlined how intense positive and negative moods may have similar effects upon the cortisol response and thus concluded that it was not possible to use cortisol as a stress marker with any confidence. From the current data it is clear that serum cortisol concentrations differ markedly between subjects, perhaps more so than between the three trials.

The decrement in skill performance may be due to selective fatigue in the type II fibres. Soderlund et al. (1992) showed twice the rate of glycogen depletion in the type II fibres than the type I. Nicholas et al. (1999) recorded glycogen concentrations of 31 and 27 mmol.kg^{-1}ww for type I and type 2 fibres respectively following 90 min of the LIST. However, as previously outlined in Chapter 7, the glycogen concentrations at the end of exercise in the heat, are much higher than those recorded in moderate conditions (Morris, 1999). Muscle glycogen concentration has been associated with fatigue during prolonged intermittent running and thus any decrease in utilisation rate would attenuate a decrement in motor performance. Though, in Chapter 7 it was previously outlined that glycogen concentrations are unlikely to be low enough to be the mechanism responsible for the decrement in skill performance seen after only 30 min, a partial role at the end of exercise should not be ruled out before acclimation. Febbraio et al. (1994b) outlined that there was a decrease in glycogen utilisation in the type II fibres following acclimation, but this finding was not shown by King et al. (1985) and Young et al. (1985). Thus, glycogen levels may have been higher following acclimation at the end of exercise, than the pre-acclimation trial. However, performance following acclimation was improved after only 30 min, when glycogen values would have been very high and it seems unlikely that a decrease in glycogen utilisation following acclimation or glycogen depletion before acclimation is responsible for the differences in skill performance recorded in the present study.

The fluid replacement during the trials showed that the subjects replaced 75, 69 and 118% of estimated sweat loss during the pre-acclimation, post-acclimation and control trials respectively. Thus, there was no difference in the body mass change before and after acclimation and body mass was well maintained in both trials. The players would not seem to be dehydrated to a level that has been suggested to be detrimental to motor skill performance (Armstrong et al., 1985). Though, the sweat rates did not

differ, there may have been a redistribution of sweating following the acclimation period. An increase in the sweat rate from the head may result in the decrease in perceived heat recorded by the subjects. Sweat has been observed to be redistributed towards the limbs from the trunk (Shvartz et al., 1979); however in well-trained subjects this phenomenon has not been observed (Cotter et al., 1997). Thus it seems likely, in the current subject group, that there was not such a redistribution of sweating, but as sweat rates in certain areas of the body were not recorded, this can not be ruled out.

There were no differences in the rating of perceived exertion or perceived thirst before and after acclimation. However, thermal comfort was improved following acclimation. The greater thermal comfort could be attributed to a tendency for a decrease in deep body temperature or a decrease in skin temperature, which was not measured in the current study (Allnutt and Allan, 1973; Allan and Gibson, 1979). Thermal comfort has also been shown to have a direct relationship with head cooling (Nunneley et al., 1982; Boutcher et al., 1985). The airflow in the gymnasium will have been identical during all the trials and thus there will have been no extrinsic difference in head cooling before and after acclimation. However, as previously outlined, a difference in sweat rate from the head, though unlikely, can not be discounted. An increase in thermal comfort has been previously associated with an increase in accuracy of tracking tasks (Griffiths and Boyce, 1971) and an increase in cognitive performance (Nunneley et al., 1982). Thus, it seems likely that the improvement in skill performance following acclimation may be a direct result of the increased thermal comfort experienced by the subjects following acclimation.

Head cooling has been suggested not only to be play a key role in controlling thermal comfort due to the head's ability to lose heat but also to transfer heat between the venous blood leaving the brain and the arterial blood supplying the brain (Brown and Williams, 1982; Nunneley et al., 1982; Boutcher et al., 1985). Thus, the cooling of the arterial supply to the brain may increase thermal comfort. Head cooling in a hot environment (40°C) has shown that thermal comfort of both the head and body are improved even though deep body temperatures are high (Brown and Williams, 1982). Cognition at similar oesophageal temperatures (38.5 to 39.0°C) has been shown to be unaffected by heating and cooling, even though there was a tendency for a faster

reaction time during heating; however, this was also associated with an increase in errors (Nunneley et al., 1982). The effect of thermal sensation during exercise has also been studied during 30 min of cycling in cool (8°C), moderate (24°C) and hot (40°C) environmental conditions (Boutcher et al., 1985). Boutcher and coworkers (1985) found that thermal sensation or comfort was worse during the hot and moderate trials than the cool trial, despite rectal temperatures being similar. Skin temperatures were found to be correlated with thermal sensation and thus the authors concluded that thermal comfort is determined by skin temperature. During the exercise in the cool, forehead skin temperature was correlated with thermal comfort. Possible mechanisms for the improved thermal comfort are that scalp temperature determines thermal comfort, serotonergic activity is decreased by facial cooling and arterial blood temperature may be decreased (Brown and Williams, 1982; Boutcher et al., 1985; Marvin et al. 1998). The mechanisms of improved thermal comfort in the brain will involve numerous complex processes, which remain to be elucidated and will require in depth research. Whatever, the mechanisms, it seems likely that the increase in thermal comfort observed in the final 30 min of intermittent running after acclimation resulted in an improvement in hockey performance. The improved thermal comfort, following acclimation may be a result of the tendency for a lower rectal temperature, a decrease in skin temperature, specifically the head, or some other mechanism that resulted in cooler arterial blood ascending to the brain. As skin temperature was not measured, the reason for the increased thermal comfort is difficult to conclude with any certainty.

In summary, hockey skill performance was improved following 4 short acclimation sessions after 30 and 60 min of intermittent running. This increase in performance may be in part due to an enhancement in thermal comfort. Furthermore, the present study provides further evidence that exercise and heat stress detrimentally affect skill performance compared to passive rest in the same conditions (~31°C). The mechanism for the decrement in skill performance observed in the current study and study presented in Chapter 7 during intermittent running is unlikely to be associated with eccentric muscle damage, glycogen depletion or central fatigue. A possible mechanism involving thermal comfort, the temperature of blood entering the brain and deep body temperature requires further investigation.

9. General Discussion

9.1. Introduction and key findings

Team sports, such as field hockey are characterised not only by periods of walking, variable pace running and short repeated bursts of maximal sprinting (Williams, 1990), but also by numerous skills. The impact of heat acclimation and hot environments, such as the climate for the next Olympic games, on intermittent high intensity running performance in females has not been extensively studied. The aim of the research studies presented in this thesis was to examine the effect of acclimation on performance and metabolism of female hockey players during intermittent running in the heat. Two further aims were to devise a suitable acclimation protocol for implementation by teams prior to the onset of tournaments and to assess how field hockey skill performance may be affected by intermittent running in the heat and heat acclimation. The main findings are summarised below:

- High intensity intermittent exercise capacity and sprint performance in the heat was reliable (Apendix A6).

- The field hockey skill test developed for the work in the thesis was both reliable and valid (Chapter 6).

- Menstrual cycle phase and oral contraceptive use do not impact on exercise time to exhaustion or sprint times when performing prolonged intermittent high intensity shuttle running. However, blood glucose was higher and serum growth hormone lower during the follicular phase compared with the luteal phase of the menstrual cycle (Chapter 4). Thus in subsequent studies in the thesis, trials were performed one month apart.

- In regard to the aim of investigating whether skill performance was affected by intermittent running in the heat, field hockey skill performance was shown to decline by up to 8% in the heat and by 4% in moderate environmental conditions following 60 min of intermittent running (Chapter 7 and 8). In Chapter 7 skill performance was decreased by 6% in the heat after 30 minutes of intermittent running compared with similar exercise in moderate conditions. The 'decision making' element of the skill test was unaffected by both intermittent running and a hot environment.

- Fifteen metre sprint performance was slower in the heat in Chapter 7 than in the moderate trial and declined at a greater rate (HT vs MT 10 vs 3% decline, P<0.05). In hot environmental conditions, sprint performance was shown to decline in all studies incorporating the LIST by the end of exercise (Chapter 4, 5%; Chapter 5, 6%; Chapter 7, 10%; Chapter 8, 7%, P<0.01).

- The major aim of the research was to investigate the impact of heat acclimation on performance of high intensity intermittent shuttle running. A further aim of the research was to design an acclimation protocol that could be incorporated by teams prior to the onset of tournaments, that was both specific to team sports and suitable to be undertaken in the tapering period. The protocol that was designed of four 30-45 min intermittent heat acclimation sessions in a 10 day period was shown to improve exercise capacity by 33%, but did not affect sprint performance (Chapter 5).

- Field hockey skill performance, but not 'decision making', was improved by 3% by acclimation (Chapter 8).

- Thermal comfort was enhanced by heat acclimation (P<0.01; Chapters 5 and 8).

- Body mass was well maintained with ad libitum fluid ingestion (Chapter 4, 5, 7 and 8). Body mass decreased by <0.9% of pre-exercise body mass in all studies and >69% of estimated sweat loss was replaced during the studies presented in Chapters 7 and 8. This finding suggests that dehydration was not the reason for exhaustion during intermittent running in the heat or a decrease in hockey skill performance following intermittent running.

The following discussion will try to analyse these findings in respect to other research and evaluate mechanisms for the beneficial effect of heat acclimation on intermittent running and field hockey skill performance.

9.2. Subject characteristics

Table 9.1 shows the physical characteristics of the female hockey players used in the 4 main experimental studies in this thesis and characteristics of female hockey players presented in the literature to date. The players in the current thesis are of a similar height and mass to those presented in previous literature. Estimated $\dot{V}O_2$ max was

similar to that for South Australian representative players (Withers and Roberts, 1981), was greater than that for centres of excellence players (Reilly and Bretherton, 1986) but not as high as that for international players (Sparling et al., 1998; Reilly et al., 1985). This descriptive data suggests that the hockey players in the current study are representative in stature and maximal aerobic power to high level territorial or national league standard players.

Table 9.1 Physical characteristics of the female hockey players used in this thesis and previous literature; (* estimated $\dot{V}O_2$ max).

Study	n	Age (yrs)	Mass (kg)	Height (cm)	$\dot{V}O_2$ max (ml.kg^{-1}.min^{-1})
Chapter 4	15	20.3 ± 0.4	60.9 ± 1.2	166.9 ± 1.4	50.6 ± 0.9*
Chapter 5	17	20.5 ± 0.4	65.9 ± 2.2	166.7 ± 1.7	49.4 ± 1.1*
Chapter 7	15	21.7 ± 0.4	68.5 ± 3.6	167.3 ± 2.2	50.3 ± 1.1*
Chapter 8	15	21.8 ± 0.4	63.1 ± 2.5	167.3 ± 1.6	50.7 ± 1.4*
Sparling et al., (1998)	15		59.6 ± 3.6	165 ± 6	57.1 ± 2.7
Withers et al., (1987)	15		62.3 ± 7.3	167 ± 8	
Cheetham et al., (1987)	15		61.0 ± 5.6	163 ± 6	
Reilly & Bretherton (1986)	15		63.1 ± 2.5	167.3 ± 1.6	45.7 ± 8.9*
Reilly et al., (1985)	15		59.0 ± 3.2		54.5 ± 5.3
Bale & McNaught – Davis (1983)	15		60.2 ± 5.2	165 ± 5	
Withers & Roberts (1981)	15		62.9 ± 9.2	165 ± 7	50.2 ± 4.2

9.3. Acclimation

9.3.1. High intensity intermittent running

The results from Chapter 5, which showed that exercise capacity was increased by 33% following acclimation, suggests that intermittent running is a good form of exercise for fast and effective acclimation. The 4 short acclimation sessions in a 10 day period was chosen to provide a realistic regimen that could be employed by teams prior to championships in hot climates. For example, in preparation for tournaments field hockey teams would be completing training sessions on and off the pitch, and would be busy with other such activities as meetings, video analysis, health assessments and so on. The short sessions interspersed with rest days would allow players not only time to complete the other sessions, but also long recovery periods so that tapering prior to the tournament could be maintained. This protocol, because of its unique nature, makes comparison with previous research, which used longer duration, submaximal exercise over a continuous daily period, problematic. Only one previous study using trained subjects has shown such a high increase in exercise capacity (cycling 60% $\dot{V}O_2$ max); however this was after 10 continuous days of acclimation to exhaustion each day (60% $\dot{V}O_2$ max ; Nielsen et al., 1993).

For acclimation to occur, there must be maintenance above a specific threshold of both peripheral and deep body temperature (Armstrong and Maresh, 1991). Intermittent running or interval training causes a rapid increase in deep body temperature and is a powerful stimulator of thermoregulatory responses compared with continuous exercise (Gisolfi and Cohen, 1979; Kraning and Gonzalez, 1991; Nevill et al., 1995). This type of exercise therefore will increase deep body temperature to the critical level for acclimation at a faster rate than continuous exercise at the same average power output (Nevill et al., 1995) and thus shorter acclimation sessions can be effective.

In summary, the high intensity intermittent acclimation protocol used in this thesis has been shown to increase both exercise capacity and hockey skill performance in the heat. The rapid rise in deep body temperature that accompanies this type of exercise

(Chapter 4,5, 7 and 8) was a critical factor in the success of the acclimation. The findings in this thesis and previous research suggest that the type, duration and intensity of exercise are crucial if acclimation is to be achieved in a short time period.

9.3.2. Well-trained hockey players

Following acclimation, a lower deep body temperature (Chapter 5) and an increase in thermal comfort was documented (Chapter 5, 8). However, no changes in plasma volume, hormones, metabolites or estimated sweat rate were recorded (Chapter 5 and 8). Early research regularly reported alterations in the above physiological measures, specifically aldosterone, plasma volume and sweat rate, following acclimation. Much of this early research however, used sedentary or recreational subjects and did not incorporate a control group and thus training and acclimation adaptations could not be distinguished. In more recent research, where either well-trained subjects were employed or a prolonged training period was embarked on prior to acclimation, the findings are more consistent with those in the current study. Well-trained athletes have a higher than normal plasma volume (Horstman and Christensen, 1982), a maximised salt balance (Davies et al., 1981) and glandular and/or neuroglandular sweat adaptations such as an increase in sweat sensitivity (Wenger, 1988). Sweat rate is also lower in women than men, and does not increase to the same extent as in men following acclimation (Horstman and Christensen, 1982). Thus, plasma volume, aldosterone concentrations and sweat rate are generally unchanged in well-trained athletes following acclimation (Finberg and Berlyne, 1977; Gisolfi and Robinson, 1979; Avellini et al., 1980; Houmard et al., 1990; Nielsen et al., 1993; Nielsen 1994). The female hockey players in the present study, who were all regularly training and participating in matches, responded similarly to acclimation to the well-trained athletes in previous studies.

The hockey players had a lower rectal temperature and an increase in thermal comfort following acclimation. Deep body temperature is reported to be decreased in the majority of research using well-trained athletes (Shvartz et al., 1980; Houmard et al., 1990; Nielsen et al., 1993; Febbraio et al., 1994b; Cheung and McLellan, 1998). However, thermal comfort has not been reported previously in relation to heat acclimation. Thus it has been shown for the first time acclimation of well-trained

athletes will result in both a decrease in deep body temperature and an increase in thermal comfort, if the stimulus is high enough. However, metabolic, hormonal and sweat rate modifications may not be observed in this group who have optimised these physiological responses through their training regimens.

9.3.3. Deep body temperature and thermal comfort

In chapter 5, deep body temperature was decreased following acclimation in comparison with an equivalent training group in moderate conditions and a control group. A tendency for a lower rectal temperature and rate of rise of rectal temperature was also reported in Chapter 8 (6 of the 8 subjects). Thermal comfort, which is related to skin temperature, was improved following acclimation (Chapter 5 and 8; Allan and Gibson, 1979; Nunneley et al., 1982; Brown and Williams, 1982).

The rectal temperatures at exhaustion were not only high, but also similar in the studies presented in Chapters 4 and 5 (Chapter 4, 39.4 ± 0.1°C; Chapter 5 39.4 ± 0.1°C) and previously in our laboratory using female games players (Morris, 1999; 39.3 ± 0.1°C). The increase in exercise capacity, following acclimation which reduced deep body temperature, provides evidence for the hypothesis that high deep body temperature is the key factor inducing the earlier onset of exhaustion in hot environmental conditions (Nielsen et al., 1993; Gonzalez-Alonso et al., 1999). Possible mechanisms for this earlier onset of exhaustion are an elevation in brain serotonin (Marvin et al., 1998; Mittleman et al., 1998; Strachen et al., 1999), or a high muscle temperature. There is very little information regarding muscle temperature at the end of exhaustive exercise in the heat (Gonzalez-Alonso et al., 1999). In male subjects, at exhaustion in the LIST, the temperature of the vastus lateralis was 40.2 ± 0.3°C (Morris, 1999), and at exhaustion following pre-cooling and pre-heating a similar temperature was reported (40.7-40.9°C; Gonzalez-Alonso et al., 1999). High muscle temperature may reduce skeletal muscle function (Hargreaves and Febbraio, 1998), perhaps by a reduction in the efficiency of mitochondrial function (Brooks et al., 1971). The present research did not measure muscle temperature, but a lower deep body temperature and an increase in thermal comfort, would indicate that muscle temperature was also lower. A lower muscle temperature would thereby maintain muscle function for a longer period, which is not only important for exercise capacity

but also for motor skills. Following acclimation, absolute muscle temperature has been publicised to be lower, however the change in muscle temperature from rest was unaffected following acclimation (Febbraio et al., 1994b). Clearly, further research is required in this area, but it provides a possible explanation for the findings reported in this research.

A key determinant for acclimation status is rectal temperature (Shvartz et al., 1977). The reasons for the lower rectal temperature following acclimation have been attributed to increased heat dissipation (Nielsen, 1994) and a decrease in metabolic heat production (Houmard et al., 1990). There was no increase in the sweat rate and thus heat dissipation may not have been increased, but without any measures of blood flow, this can not be ruled out. Further, the increase in thermal comfort would suggest that facial skin temperature was decreased and thus there may have been a redistribution of sweating or an increase in sweating sensitivity in this region.

9.4. Field hockey skill performance

In Chapters 7 and 8, field hockey skill performance following high intensity intermittent running was decreased in both hot and moderate environmental conditions. However, the 'decision making' element of the skill test was unaffected. Several mechanisms have been discussed in the thesis that may explain the decrement in performance after the LIST. Eccentric muscle damage, dehydration, glycogen depletion and a central fatigue mechanism due to an increase in serotonin and hypoglycaemia are unlikely to be individually responsible for the decrement in performance. However, a combination of these mechanisms and those that will be discussed in this section may provide an explanation for the decline in skill performance.

The study presented in Chapter 7 showed a higher rectal temperature and decline in sprint performance when exercising in the heat compared with similar exercise in moderate conditions. The greater thermal strain may partially explain the difference in performance in these environmental conditions. Thermal strain and discomfort have been associated with a decrease in motivation or drive to exercise and a decrease in accuracy of tracking tasks (Griffiths and Boyce, 1971; Allan and Gibson, 1979;

Nielsen et al., 1993). The studies in Chapter 7 and 8 reported an increase in thermal comfort and a concomitant improvement in field hockey skill performance. Skin temperatures, specifically head and facial skin temperatures were not measured in the present research, but have been suggested to be the determining factor in thermal comfort. However, the relationship between deep body temperature and heating and cooling the skin also seem to impact upon thermal comfort. Thus, the exact mechanism for the increase in thermal comfort is difficult to stipulate in physiological terms from the measurements made in this thesis. Its importance in motor skill performance is clearly apparent, be it a physiological, psychological or a combined physiological/ psychological effect.

Low glycogen concentrations have been reported following 90 min of the LIST in moderate conditions (Nicholas et al., 1999); however at exhaustion in hot conditions glycogen concentrations were not depleted (Morris, 1999). Type II fibres have been shown to have twice the rate of glycogen depletion, which would decrease hockey skill performance. Skill performance was decreased after only 30 min when glycogen stores would not be depleted suggesting another perhaps thermoregulatory mechanism. However, there is greater glycogen utilisation in the heat during intermittent running (Morris, 1999) and acclimation has been shown to decrease this rate in type II fibres (Febbraio et al., 1994b). Thus, by the end of the intermittent running, glycogen depletion may play a part in the decrease in skill performance.

Field hockey skill performance is poorer following intermittent running in the heat compared with similar exercise in moderate conditions (Chapter 7). Estimated sweat rate was higher during intermittent running in the heat and this would result in higher dehydration levels in the heat without fluid replacement. Research into fluid replacement and soccer skill following 90 min of the LIST, has shown that skill performance is detrimentally effected by dehydration (McGregor, 1999). However, following a 2 h recovery and rehydration period skill performance was still poorer than pre-exercise levels. With ad libitum fluid ingestion in the current research, dehydration expressed as body mass loss was 0.57 and 0.39% in the hot and moderate trials respectively. Furthermore, fluid intake was over 50% of the estimated sweat rate which has been suggested to be the level required to prevent dehydration in team

sports (Gisolfi and Duchman, 1992). Thus, the subjects were not dehydrated and thus dehydration was not responsible for the decline in skill performance.

Muscle damage due to the number of eccentric actions has been discussed in Chapter 8. However an increase in muscle temperature may further lead to an increase in muscle damage (Armstrong et al., 1991) and reduction in motor unit recruitment potential. During the field hockey skill test the complex movements require a certain pattern of fibre type recruitment. If altered, performance may be decreased. Following muscle damage, an alteration in neurone recruitment pattern may occur and cause a decrease in performance of motor skill tasks (Saxton et al., 1995). Even a modest rise in muscle temperature has been shown to increase the number of large motor neurones recruited following eccentric exercise, which can decrease fine control (Saxton et al., 1995). As outlined in the previous section high muscle temperatures have been reported in this type of exercise, and thus a decrease of fine control, by altering muscle recruitment, would impact on hockey skill performance.

In summary, field hockey skill performance is poorer in the heat than in moderate conditions, but is improved following acclimation. The mechanism for the poorer performance in the heat and prior to acclimation is related to a greater thermal strain. The mechanism for the decline in skill performance following intermittent running in the heat may be related to muscle temperature, muscle damage, temperature of arterial blood entering the brain, a diminished motivation of the player and/or other mechanisms that require further investigation.

9.5. Directions for future research

- Compare high intensity intermittent running and continuous running of the same average exercise intensity as acclimation protocols.
- Establish whether the acclimation protocol is specific to the trial pre- and post-acclimation.
- Try to determine if continuous daily exercise or exercise interspersed with rest days is the superlative acclimation method.

- Establish what duration and critical deep body temperature is required for acclimation to occur in well-trained athletes. Try to establish if there is a relationship between the temperature maintained and any increase in exercise capacity following acclimation.

- Establish whether muscle temperature is a key mechanism in this type of acclimation and its effect on skill performance.

- Examine the responses of men to the acclimation protocol employed in this thesis.

- Establish if glycogen utilisation is critical for hockey skill performance. Repeat the study presented in Chapter 8, with muscle biopsies before and after each trial and acclimation to measure type I and II glycogen concentrations.

- Establish whether facial cooling increases thermal comfort during intermittent running in the heat and whether this impacts on hockey skill performance.

9.6. Practical advice

The research in this thesis clearly shows that heat acclimation increases exercise capacity and improves hockey performance in the heat. The precise mechanisms for the improvement are not fully understood, but are likely to be linked to deep body temperature and thermal comfort. The findings in the thesis suggest that short high intensity intermittent acclimation sessions should be employed in the acclimation and holding camps prior to major championships in the heat. Acclimation will still occur if sessions are interspersed with rest, pitch and tactical sessions. The intermittent sessions should be of a high intensity to increase deep body temperature rapidly.

Perhaps if the GB hockey squads use acclimation of the type outlined above, prior to the Olympics in Athens, though a medal cannot be guaranteed, skill performance will be improved. It will, however, help to put us on a 'level sportsturf' with those teams who live in hot environments even if the advantage of natural acclimatisation cannot be exactly matched.

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Appendices

Appendix A1

Subject Information and Statement of Informed Consent

Effect of acclimation upon performance of prolonged intermittent, high-intensity running and field hockey skill performance in a hot environment.

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SUPERVISORS :   Dr. Mary E. Nevill

INVESTIGATOR :   Caroline Sunderland (Ph.D.student)
                             Simon Marwood (M.Sc. student)
                             Claire Spooner (B.Sc. student)

INTRODUCTION

Many of the major hockey competitions take place in hot and humid climates, for example the 1998 Commonwealth Games in Kuala Lumpa. Thus it is of major interest to investigate the effects of heat and how we can improve hockey skill performance in these conditions.

Although there has been some research that has investigated the effect of heat and the performance of games based sports, the studies have utilised predominantly male subjects. Furthermore, no studies have investigated the effects of heat acclimation upon hockey skill performance in a hot environment or the changes in performance during the time period of a game. Thus, in view of the limited previous research, the aim of the present study is to determine the effect of acclimation upon performance of prolonged intermittent, high-intensity running and field hockey skill performance in a hot environment. This type of exercise provides a realistic representation of the physiological demands of the 'multiple sprint sports.' Thus, this study will provide female hockey players with information relating to both their hockey skill performance pre and post heat acclimation.

Subjects will be asked to perform a number of preliminary tests, which gives an opportunity to practice and become acquainted with the experiment, as well as the main trials.

METHODS

Familiarisation and Preliminary Tests

Subjects will be required to attend the laboratory prior to the first main trial.

Visit 1: Approximately 40 min.
        Medical and menstrual cycle questionnaires completed.

Maximal oxygen uptake estimated measured using the Multi-Stage Fitness ('bleep') test.
Familiarisation with the hockey skill test.

Visit 2: Approximately 80 min.
Performance of the LIST (Loughborough Intermittent Shuttle Test) in hot environmental conditions (30 °C) in anticipation of the main trials.
Familiarisation with the hockey skill test.

Further short visits (approximately 20 min) may be required to fully familiarise the subjects to the hockey skill test.

Main Tests

All subjects will be required to perform the LIST on six occasions in hot (~30°C) environmental conditions. During the two main trials a hockey skill test will be completed prior to, during and after the completion of approximately 60 min of the LIST. The two main trials will be separated by approximately 1 month. Subjects will need to set aside approximately 2.5 hours to complete each trial. In the ten days prior to the second main trial, subjects will be asked to complete approximately 30 min of the LIST on four separate days. Subjects will need to set aside approximately 1 hour to complete each of the acclimation/ training trials.

The LIST (Loughborough Intermittent Shuttle Test) involves repeated bouts of:

	PACE	DISTANCE	INTENSITY
1	Walking	3 x 20 metres	~ 13 s / 20 metres
2	Maximum sprint	1 x 15 metres	Maximum speed
3	Recovery walk	~ 3 metres	4 second duration
4	Running (Cruise)	3 x 20 metres	~ 85% VO_2max
5	Jogging	3 x 20 metres	~ 50% VO_2max

This pattern of exercise is repeated until approximately 15 min has elapsed followed by a 3 min rest period. This 15 min exercise set: 3 min rest period pattern will be repeated four times. The hockey skill test will take place before the commencement of exercise, after 2 sets (approximately 30 min) and then again after a further 2 min.

Venous blood samples (10 ml) will be taken at rest, post the hockey skill tests and during each 3 min rest period, and at the end of the intermittent shuttle test during the two main trials.

Core temperature (measured via a rectal probe) and heart rate will be continuously monitored throughout. Rectal probe will be inserted by the subject in the strictest privacy approximately 10cm passed the anal sphincter.

Each individual's perceived exertion during the exercise will be noted toward the end of each 15 min period.

SUBJECT REQUIREMENTS: In the 2 days prior to the main trials subjects should refrain from undertaking intense training. You will need to monitor your food intake so that you can repeat as near as possible the same diet before both main trials.

Subjects should not consume food in the 12 hours before a main trial. Please refrain from consuming alcohol on the day before the main trials. Subjects, immediately prior to and following each main test, will be weighed nude in the strictest privacy.

LOCATION OF THE STUDY

All testing will take place in the Sports Science Research Laboratories and Sports Hall Gymnasium in the Department of Physical Education, Sports Science and Recreation Management at Loughborough University of Technology. Testing will be arranged at times mutually convenient to subjects and the research team.

POSSIBLE RISKS AND DISCOMFORTS

Exercise is maximal and therefore the trials are of a demanding nature. Therefore, those involved in the study should be accustomed to performing demanding exercise and will be thoroughly familiarised with experimental procedures, including exercising in hot conditions. If the test becomes too demanding, or core body temperature rises above 40 degrees centigrade exercise will be terminated. Equipment will be available for rapid body cooling in the unlikely case that it should be required.

The cannula placement will be carried out by Dr Mary E. Nevill or Dr Ceri Nicholas under local anaesthetic, and therefore discomfort to the subjects should be minimal. There is an extremely small risk that cannulation could result in an air or plastic embolism, but good practice minimises this risk. All blood samples will be taken by individuals trained to do so and experienced. All procedures will be carried out in accordance with the Code of Practice for Workers having Contact with Body Fluids.

Because of the intense nature of the exercise, subjects are asked to inform the investigators if they have a cold or feel unwell immediately prior to any trial in the study. Your safety is of paramount importance and you should not perform a trial unless you are in the best of health. Subjects are fully entitled to withdraw from the study at any time without giving any reason.

CONFIDENTIALITY

Even though the data collected may be presented in various forms (journal articles, papers, etc.), personal information will be treated in confidence. That is, names will not be used, nor will subject's initials be associated with the data in the presentations.

FURTHER INFORMATION

If you require any further information please contact Caroline Sunderland, either in the Sports Hall Building (RR105; tel. 01509 228183), at William Morris Hall (Sub-Warden, tel 01509 219439) or by e-mail C.D. Sunderland@lboro.ac.uk.

ONE PAGE LAY STATEMENT

Title: Effect of acclimation upon performance of prolonged intermittent, high-intensity running and field hockey skill performance in a hot environment.

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Supervisor: Dr. Mary E. Nevill
Department of Physical Education, Sports Science and Recreation Management, Loughborough University, Loughborough, Leics. LE11 3TU.

Hot environmental conditions decrease the capacity of individuals to exercise, resulting from increased thermal strain. Similarly, in a previous study a decrease in field hockey skill performance has been shown to occur following intermittent shuttle running in the heat. Thus, the aim of this study is to determine whether heat acclimation/ training will prevent or decrease the decline in hockey skill performance during the approximate time length of a game in a hot environment.

The intermittent shuttle run test was developed at Loughborough University and provides a realistic representation of the physiological demands of 'games-type' activities, such as field hockey. The test comprises of 20m shuttles at walking, jogging, cruise and all-out pace. Subjects will perform the intermittent shuttle run test on six occasions, after being fully familiarised with the procedures involved. For the two main trials, subjects will complete a hockey skill test prior to undertaking the intermittent shuttle run test. After approximately 30 min of the shuttle run test, subjects will again complete the hockey skill test. The 30 min exercise period and the skill test are then repeated to try and mimic the two halves of a hockey game. Two trials are completed in hot (~30 °C) conditions using this hockey skill test and shuttle running pattern. The two main trials will be completed approximately 1 month apart, and will require the subjects to be available for 2.5 hours on each occasion. In the ten days prior to the second trial, subjects will perform approximately 30 min of the shuttle run test on four separate days as heat acclimation/training. The acclimation/training sessions will require the subjects to be available for approximately 1 hour.

Preliminary trials and familiarisation will involve the subjects completing a submaximal and a maximal running test, the Multistage fitness test, which requires the participant to run 20m shuttles at progressively increasing exercise intensities.

To fully understand the physiological responses in this situation, subjects will be required to give blood samples throughout both main trials. Monitoring of inner body temperature will be undertaken using rectal thermometers.

Subjects should be female hockey players who participate regularly in vigorous activity of this nature. Personal performance results as well as the overall results of the study will be provided.

Appendix A2

STATEMENT OF INFORMED CONSENT

I have read the Subject Information and am fully aware of the procedures and requirements involved in the " Effect of acclimation upon performance of prolonged intermittent, high-intensity running and field hockey skill performance in a hot environment" study. I understand what will be required of me as a subject. I am aware that I have the right to withdraw from the study at any time with no obligation to provide reasons for my decision and have had opportunity to ask for further information or discuss any concerns.

I agree to take part in the Prolonged, Intermittent, High-Intensity Running and Field Hockey Skill Performance in a Hot Environment Study

Signed _____   Date _____

Witnessed by _____

Appendix A3

HEALTH SCREEN FOR STUDY VOLUNTEERS Name or Number ....

It is important that volunteers participating in research studies are currently in good health and have had no significant medical problems in the past. This is to ensure (i) their own continuing well-being and (ii) to avoid the possibility of individual health issues confounding study outcomes.

Please complete this brief questionnaire to confirm fitness to participate:

1. **At present,** do you have any health problem for which you are:

(a) on medication, prescribed or otherwise   Yes ☐ No ☐

(b) attending your general practitioner   Yes ☐ No ☐

(c) on a hospital waiting list   Yes ☐ No ☐

2. **In the past two years,** have you had any illness which require you to:

(a) consult your GP   Yes ☐ No ☐

(b) attend a hospital outpatient department   Yes ☐ No ☐

(c) be admitted to hospital   Yes ☐ No ☐

3. **Have you ever** had any of the following:

(a) Convulsions/epilepsy   Yes ☐ No ☐

(b) Asthma   Yes ☐ No ☐

(c) Eczema   Yes ☐ No ☐

(d) Diabetes   Yes ☐ No ☐

(e) A blood disorder   Yes ☐ No ☐

(f) Head injury   Yes ☐ No ☐

(g) Digestive problems   Yes ☐ No ☐

(h) Heart problems   Yes ☐ No ☐

(i) Problems with bones or joints   Yes ☐ No ☐

(j) Disturbance of balance/coordination   Yes ☐ No ☐

(k) Numbness in hands or feet   Yes ☐ No ☐

(l) Disturbance of vision   Yes ☐ No ☐

(m) Ear / hearing problems   Yes ☐ No ☐

(n) Thyroid problems   Yes ☐ No ☐

(o) Kidney or liver problems   Yes ☐ No ☐

(p) Allergy to nuts   Yes ☐ No ☐

4. **Has any,** otherwise healthy, member of your family under the age of 35 died suddenly during or soon after exercise?   Yes ☐ No ☐

If YES to any question, please describe briefly if you wish (eg to confirm problem was/is short-lived, insignificant or well controlled.)

....

....

Appendix A4

MUTLISTAGE SHUTTLE RUN TEST

Name: _____ D.O.B. _____ Date: _____

Weight (kg): _____ Height (cm): _____

SHUTTLE RUN TEST

Level 1	1	2	3	4	5	6	7								
Level 2	1	2	3	4	5	6	7	8							
Level 3	1	2	3	4	5	6	7	8							
Level 4	1	2	3	4	5	6	7	8	9						
Level 5	1	2	3	4	5	6	7	8	9						
Level 6	1	2	3	4	5	6	7	8	9	10					
Level 7	1	2	3	4	5	6	7	8	9	10					
Level 8	1	2	3	4	5	6	7	8	9	10	11				
Level 9	1	2	3	4	5	6	7	8	9	10	11				
Level 10	1	2	3	4	5	6	7	8	9	10	11				
Level 11	1	2	3	4	5	6	7	8	9	10	11	12			
Level 12	1	2	3	4	5	6	7	8	9	10	11	12			
Level 13	1	2	3	4	5	6	7	8	9	10	11	12	13		
Level 14	1	2	3	4	5	6	7	8	9	10	11	12	13		
Level 15	1	2	3	4	5	6	7	8	9	10	11	12	13		
Level 16	1	2	3	4	5	6	7	8	9	10	11	12	13	14	
Level 17	1	2	3	4	5	6	7	8	9	10	11	12	13	14	
Level 18	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15

RESULTS

Level and shuttle attained: _____/_____

VO_2max equivalent [$ml.kg^{-1}.min.^{-1}$] (Ramsbottom et al., 1988):_____

	VO_2 $ml.kg^{-1}.min.^{-1}$	Equivalent level and shut. in the shuttle run test	20 m speed which VO_2 equates with
50% VO_2max			
85% VO_2max			

Appendix A5

	A	B	C	D	E	F	G
1	LEVEL	Shuttle	PredM-VO2	85 per cen	y=0.258*%PredVO2	Speed	Time for 20m
2		Number	ml/kg/min.	Pred VO2	-1.876	2.08+0.14*y	20/speed
3							
4	11	2	50.8	43.2	9.26	3.38	5.92
5	11	4	51.4	43.7	9.40	3.40	5.89
6	11	6	51.9	44.1	9.51	3.41	5.86
7	11	8	52.5	44.6	9.64	3.43	5.83
8	11	10	53.1	45.1	9.77	3.45	5.80
9	11	12	53.7	45.6	9.90	3.47	5.77
10	12	2	54.3	46.2	10.03	3.48	5.74
11	12	4	54.8	46.6	10.14	3.50	5.71
12	12	6	55.4	47.1	10.27	3.52	5.68
13	12	8	56	47.6	10.40	3.54	5.66
14	12	10	56.5	48.0	10.51	3.55	5.63
15	12	12	57.1	48.5	10.65	3.57	5.60
16	13	2	57.6	49.0	10.76	3.59	5.58
17	13	4	58.2	49.5	10.89	3.60	5.55
18	13	6	58.7	49.9	11.00	3.62	5.53
19	13	8	59.3	50.4	11.13	3.64	5.50
20	13	10	59.8	50.8	11.24	3.65	5.47
21	13	13	60.6	51.5	11.41	3.68	5.44
22	14	2	61.1	51.9	11.52	3.69	5.42
23	14	4	61.7	52.4	11.65	3.71	5.39
24	14	6	62.2	52.9	11.76	3.73	5.37
25	14	8	62.7	53.3	11.87	3.74	5.34
26	14	10	63.2	53.7	11.98	3.76	5.32
27	14	13	64	54.4	12.16	3.78	5.29
28							

	A	B	C	D	E	F	G
30	LEVEL	Shuttle	PredM-VO2	50 per cen	y=0.258*%PredVO2	Speed	Time for 20m
31		Number	ml/kg/min.	Pred VO2	-1.876	2.08+0.14*y	20/speed
32							
33	11	2	50.8	25.4	4.68	2.73	7.31
34	11	4	51.4	25.7	4.75	2.75	7.28
35	11	6	51.9	26.0	4.82	2.75	7.26
36	11	8	52.5	26.3	4.90	2.77	7.23
37	11	10	53.1	26.6	4.97	2.78	7.20
38	11	12	53.7	26.9	5.05	2.79	7.18
39	12	2	54.3	27.2	5.13	2.80	7.15
40	12	4	54.8	27.4	5.19	2.81	7.12
41	12	6	55.4	27.7	5.27	2.82	7.10
42	12	8	56	28.0	5.35	2.83	7.07
43	12	10	56.5	28.3	5.41	2.84	7.05
44	12	12	57.1	28.6	5.49	2.85	7.02
45	13	2	57.6	28.8	5.55	2.86	7.00
46	13	4	58.2	29.1	5.63	2.87	6.97
47	13	6	58.7	29.4	5.70	2.88	6.95
48	13	8	59.3	29.7	5.77	2.89	6.92
49	13	10	59.8	29.9	5.84	2.90	6.90
50	13	13	60.6	30.3	5.94	2.91	6.87
51	14	2	61.1	30.6	6.01	2.92	6.85
52	14	4	61.7	30.9	6.08	2.93	6.82
53	14	6	62.2	31.1	6.15	2.94	6.80
54	14	8	62.7	31.4	6.21	2.95	6.78
55	14	10	63.2	31.6	6.28	2.96	6.76
56	14	13	64	32.0	6.38	2.97	6.73
57							

Appendix A6

The reliability for the LIST in hot conditions (Part A to exhaustion):

The mean difference ± limits of agreement is –0.7 ± 5.6 min.

Raw Data

Subject	Trial A (min)	Trial B (min)	Difference (min)
1	20.3	23.2	-2.9
2	32.2	27.9	4.3
3	35.7	35.7	0.0
4	21.7	23.2	-1.4
5	44.5	44.5	0.0
6	30.5	30.5	0.0
7	30.3	35.2	-4.9
Mean	30.7	31.4	-0.7
SD	8.2	7.7	2.9

	Trial A	Trial B
Mean	30.7	31.4
Variance	67.7	58.6
Observations	7	7
Pearson Correlation	0.94	
Hypothesized Mean Difference	0	
df	6	
t Stat	-0.65	
P(T<=t) one-tail	0.27	
t Critical one-tail	1.94	
P(T<=t) two-tail	0.54	
t Critical two-tail	2.45	

The reliability for the LIST in moderate conditions (Part A and B):

From C. W. Nicholas (1996) "Influence of Nutrition on Muscle Metabolism and Performance during Prolonged, Intermittent, High-Intensity Running in Man" Ph.D. Thesis.

See also Nicholas et al., (2000).

The mean difference ± limits of agreement is 0.3 ± 5.0 min.

Raw Data

Subject	Trial A (min)	Trial B (min)	Difference (min)
1	110	104.3	5.7
2	92.1	94.4	-2.3
3	95.2	96.1	-0.9
4	96.2	97.5	-1.3
5	93.4	93.4	0.0
6	93.8	92.4	1.4
7	93.2	94.7	-1.5
8	96.7	95.7	1.0
Mean	96.3	96.1	0.3
SD	5.7	3.7	2.5

	Trial A	Trial B
Mean	96.3	96.1
Variance	33.0	13.6
Observations	8	8
Pearson Correlation	0.95	
Hypothesized Mean Difference	0	
Df	7	
t Stat	0.29	
P(T<=t) one-tail	0.39	
t Critical one-tail	1.89	
P(T<=t) two-tail	0.78	
t Critical two-tail	2.36	

Appendix A7

	A	B	C	D	E	F	G
1		Dry Bulb	Wet bulb	Psa	Pswb	Pa	Relative
2		temp	temp	assume t =DB	assume t =WBT		Humidity
3							
4	Equations			**EXP(18.956-4030.18/(B10+235))**			
5	Equations			**EXP(18.956-4030.18/(C10+235))**			
6	Equations				**E10-0.667*(B10-C10)**		
7	Equations					**F10/D10*100**	
8							
9							
10	Mid S1-1	33.3	18.9	51.15	21.82	12.22	23.9
11	End S1	33.8	19	52.60	21.96	12.09	23.0
12	Mid S2	33.9	19	52.90	21.96	12.02	22.7
13	End S2	34	19.2	53.19	22.23	12.36	23.2
14	Mid S3	33.8	19.4	52.60	22.51	12.91	24.5
15	End S3	34	19.5	53.19	22.65	12.98	24.4
16	Mid S4	33.9	19.7	52.90	22.94	13.47	25.5
17	End S4	33.8	19.6	52.60	22.80	13.32	25.3
18	Mid S5	34	19.7	53.19	22.94	13.40	25.2
19	End S5	33.4	19.6	51.44	22.80	13.59	26.4
20	Mid S6	33.4	19.6	51.44	22.80	13.59	26.4
21	Mid S1-2	33.9	18.9	52.90	21.82	11.82	22.3
22	End S1	33.2	18.9	50.87	21.82	12.28	24.1
23	Mid S2	33.8	19	52.60	21.96	12.09	23.0
24	End S2	34	19.2	53.19	22.23	12.36	23.2
25	Mid S3	34.3	19.8	54.09	23.08	13.41	24.8
26	End S3	33.9	19.4	52.90	22.51	12.84	24.3
27	Mid S4	34	19.7	53.19	22.94	13.40	25.2
28	End S4	33.8	19.6	52.60	22.80	13.32	25.3
29	Mid S5	34	19.6	53.19	22.80	13.19	24.8
30	End S5	33.8	19.8	52.60	23.08	13.74	26.1
31	Mid S6	33.3	19.8	51.15	23.08	14.08	27.5
32	Mid S1-3	33.7	19	52.31	21.96	12.15	23.2
33	End S1	33.9	19	52.90	21.96	12.02	22.7
34	Mid S2	33.9	19.1	52.90	22.10	12.22	23.1
35	End S2	34	19.2	53.19	22.23	12.36	23.2
36	Mid S3	33.9	19.4	52.90	22.51	12.84	24.3
37	End S3	34.1	19.7	53.49	22.94	13.33	24.9
38	Mid S4	34	20	53.19	23.37	14.03	26.4
39	End S4	34.1	19.8	53.49	23.08	13.54	25.3
40	Mid S5	33.8	19.7	52.60	22.94	13.53	25.7
41	End S5	33.4	19.8	51.44	23.08	14.01	27.2
42	Mid S6	33.4	19.9	51.44	23.22	14.22	27.6

Appendix A8

LOUGHBOROUGH UNIVERSITY
ETHICAL ADVISORY COMMITTEE
RESEARCH PROPOSAL FOR HUMAN BIOLOGICAL INVESTIGATIONS

This application should be completed after reading the Code of Practice paying particular attention to the advice given in Section 6.3.

(i)   Applicants:

Dr. Mary E. Nevill, Ms. Caroline Sunderland, Mr. Simon Marwood, Ms. Claire Spooner

(ii)   Project Title:

Effect of acclimation upon performance of prolonged intermittent, high-intensity running and field hockey skill performance in a hot environment.

(iii)   Aims and Outline of the Project:

The increased physical and thermal strain and the decreased capacity of men to perform intermittent exercise in a hot environment is well documented (Kraning and Gonzalez, 1991; Nevill et al., 1995). Similarly, following acclimation to a hot environment endurance capacity is known to increase (Nielsen et al., 1993; Wyndham et al., 1976). However, few studies have investigated acclimation for intermittent or games type activity in female subjects. Furthermore, no studies have investigated the effects of acclimation upon the performance of intermittent exercise and field hockey skill performance.

Thus, in view of the previous research, the aim of the present study is to determine the acclimation effect of 4 days training in the heat upon performance of prolonged, intermittent, high intensity shuttle running and field hockey skill performance in a hot   (~30 °C) environment. This type of exercise provides a realistic representation of the physiological demands of the 'multiple sprint sports.'

It is proposed that female subjects will complete two trials, approximately one month apart, separated by four acclimation training trials.

(iv)   Names and status of investigators:

Dr. Mary E. Nevill - Senior Lecturer, Dept. of P.E., S.S. & R.M., Loughborough University
Ms Caroline Sunderland-PhD. student, Dept. of P.E., S.S. & R.M., Loughborough University
Mr Simon Marwood - MSc. Student, Dept. of P.E., S.S. & R.M., Loughborough University
Ms Claire Spooner - BSc. Student, Dept. of P.E., S.S. & R.M., Loughborough University

(v)   Subjects (see Section 6.3e):

The subjects will be physically active female volunteers, recruited by general notice, aged 18-30 years, although it is anticipated that nearly all will be either students or research staff, within the Dept. of Physical Education, Sports Science and Recreation Management.

The protocol will include both oral contraceptive users and normal menstruating women.

Subjects will also be required to complete a health history questionnaire and menstrual cycle questionnaire in the presence of an experimenter (to provide clarification and assistance) prior to any test.

(vi)   Location (any special facilities to be used):

The work will be carried out in the Sports Science laboratories and in a gymnasium of the Department of Physical Education, Sports Science and Recreation Management at Loughborough University.

(vii)   Duration (including demand on subject's time):

Prior to trials, each subject will be asked to complete practice sessions, lasting approximately 1 hour, so that the purpose of the study can be fully explained to them and in order to become familiarised with the intermittent shuttle test and the hockey skills test. During at least one of the practice sessions subjects will be exposed to the 30 $^{\circ}$C conditions.

The main trials will require the subjects to visit the laboratory on six separate occasions, within approximately one month. On two of the testing days the subjects will need to set aside approximately 2.5 hours, the remaining four training days will require approximately 1 hour.

(viii)   Reasons for undertaking the study (e.g. contract, student research):

This is a staff and Ph.D. research project that examines the effect of acclimation upon performance of prolonged intermittent, high-intensity running and field hockey skill performance in a hot environment.

(ix)   Methodology (a brief outline of research design):

After completing the preliminary tests and familiarisation, subjects will report to the laboratory on six separate occasions. Subjects will complete a hockey skill test and then perform 2 bouts lasting approximately 30 minutes, of intermittent shuttle exercise, separated by the hockey skill test. A final hockey skill test will then take place. This protocol will be repeated twice pre and post a 4 day acclimation period in hot conditions (~30 °C). These two main trials will be seperated by approximately one month to allow for control of the menstrual cycle. Thus the four acclimation sessions will be undertaken in the 10 days prior to the second trial. All trials will take place in the heat (~30 °C). All running takes place over a 20 metre distance and comprises walking, jogging, running and sprinting. (See attached experimental protocol which has generic approval). Heating of the gymnasium in which the intermittent shuttle test will be carried out will be achieved by using four electrically powered fan heaters (Andrews DE 65, Andrews Industrial Equipment Ltd.) installed by the University. Previous use of this heat source has shown it to be a safe and suitable means of raising the temperature of the gymnasium. A thermostatically controlled gas heater (Andrews ID 175, Andrews Industrial Equipment Ltd.) will be used to provide additional heat if required. The combustion unit is entirely closed and exhaust fumes are ducted to outside the gymnasium. Consequently, the heated ambient air will contain no products of propane combustion. The heater and gymnasium air quality will be approved by the University Health and Safety Officer before any subjects are exposed to the hot environment.

(iv)   Procedures and measurements

Subjects will be asked questions relating to their health status prior to all exercise bouts and will be asked to complete a small health questionnaire prior to undertaking any such exercise. Please see attached questionnaire.

Heart rate will be monitored at rest and throughout exercise by means of short-range telemetry (Sport Tester PE 3000). Ten ml venous blood samples, obtained by means of an indwelling cannula (venflon, 16 - 18 gauge) inserted into a forearm vein, will be taken at rest, post hockey skill test, during and post the intermittent shuttle test, and after the final hockey skill test during the main trials only. The cannula will be inserted while the subject is seated and kept patent by a single delivery of sterile isotonic saline immediately after the withdrawal of each blood sample. No more than 100 ml of blood will be collected from each subject during each trial (consequently no more than 200 ml will be taken over the whole study). Blood samples will be analysed for haematocrit, haemoglobin, glucose, progesterone, aldosterone, cortisol, lactate and ammonia.

Rectal temperature will be measured using a rectal thermometer, which will be washed in soap and water and sterilised in Decon solution. Handling of rectal thermometers and handling of blood samples will be carried out in accordance with the University's Code of Practice for Person's Having Contact with Human Body Fluids. Temperature measurements will be made approximately every 5 minutes.

(xi)   Possible risks, discomforts and/or distress (see Section 6.3k):

This study requires volunteers to exercise maximally, intermittently, for the duration of the intermittent shuttle test and is therefore of a demanding nature. However, all subjects involved in the study will be well-trained individuals accustomed to vigorous exercise. Participants will be thoroughly familiarised with experimental procedures, including exercising in hot conditions, and will be instructed and encouraged to stop exercising at any time that the demands of the test become intolerable. In addition, subjects will be continually monitored and if an individual appears unduly distressed, or if rectal temperature rises above 40 $^{\circ}$C exercise will be terminated. (In previous treadmill running studies in this laboratory mean rectal temperatures have been found to be consistently above 39.0 $^{\circ}$C during the last 30 minutes of an exercise bout even in cool conditions). Equipment will be available for rapid body cooling in the unlikely case that it should be required. The cannula placement will only be carried out by trained persons (Dr Mary E. Nevill, Dr Ceri Nicholas). There is an extremely small risk that cannulation could result in an air or plastic embolism, but good practice minimises this risk. The blood samples will only be drawn by individuals approved by the ethical committee. All procedures will be carried out in accordance with the Code of Practice for Workers having Contact with Body Fluids. All data will be stored in line with the Data Control Act.

(xii)   Procedures for taking measurements and for chaperoning and supervision of
        subjects during investigations:

Throughout the preliminary and main trials study participants will be supervised by at least two investigators.

(xii)   Names of investigators and personal experience of proposed procedures   and/or methodologies:

Dr. Mary E. Nevill - Qualified to perform vein cannulations and has over 10 years experience of similar investigations in this laboratory.
Ms Caroline Sunderland-Trained to undertake exercise testing of the type described.
Mr Simon Marwood-Trained to undertake exercise testing of the type described.
Ms Claire Spooner-Trained to undertake exercise testing of the type described.

(xiv)   Details of any payments to be made to the subjects

None.

(xv)   Do any investigators stand to gain from a particular conclusion of the   research project:

No.

(xvi)   Whether the University's Insurers have indicated that they are content for   the University's Public Liability Policy to apply to the proposed Investigation   (Committee use only):

**(xvii) Whether the insurance cover additional to (xv) has been arranged
by the   Investigator (see Section 6.3o):**

No.

**(xviii) In the case of studies involving new drugs or radioisotopes, written
approval   for the study must be obtained from the appropriate
national body and   submitted with the protocol. State if applicable:**

Not applicable.

(xix)   Declaration

I have read the University's Code of Practice on Investigations on Human   Subjects and completed this application.

Signature of applicant:

....

Signature of Head of Department:

....

Date: ....

Not thirsty	**0**
	1
Very Slight thirst	**2**
	3
Slight thirst	**4**
	5
Thirsty	**6**
	7
Very Thirsty	**8**
	9
Very Very Thirsty	**10**

Too Cold	**-10**
	-9
	-8
Cold	**-7**
	-6
Very Cool	**-5**
	-4
Cool	**-3**
	-2
	-1
Comfortable	**0**
	1
	2
Warm	**3**
	4
Very Warm	**5**
	6
Hot	**7**
	8
	9
Too Hot	**10**

Appendix B

BLOOD ASSAYS

Haematocrit (% Cell Volume)

Procedure:

1. Three small bore haematocrit tubes (Scientific Products Ltd., Baxter Healthcare Corporation, U.S.A.) were filled with venous blood using capillary action.

2. Plasticine was used to seal the tubes at one end, and tubes were then centrifuged for 15 minutes (micro- haematocrit centrifuge, Hawksely and Sons Ltd, Lancing, U.K.).

3. The haematocrit portion of the blood sample was subsequently established using a micro-haematocrit reader (Hawksely and Sons Ltd, Lancing, U.K.).

Spectrophotometric determination of Haemoglobin (The Cyanmethemoglobin Method) [546 nm]

Principle:

Haemoglobin is oxidised to methemoglobin by ferricyanide. The addition of potassium cyanide converts the methemoglobin to the stable cyanmethemoglobin.

Haemoglobin + Cyanide + Ferricyanide ⇒ Cyanmethemoglobin.

Reagents:

A commercially available kit was used to establish haemoglobin concentration (Test-Combination Haemoglobin, Boehringer Mannheim U.K. Ltd., Lewes, U.K.). Drabkin's solution was prepared in a 1 litre volumetric flask by diluting 25 ml of potassium hexacyanoferrate (III) (0.6 mmol.l^{-1}) and 25 ml of potassium cyanide (0.75 mmol.l^{-1}) with distilled water. The one litre of solution was then stored in an amber bottle.

Procedure:

1. Duplicate samples of 20 μl of venous blood were dispensed into 5 ml of Drabkin's solution in a glass or plastic tube. The samples were thoroughly mixed.

2. Within 6 hours of collection the absorbance of each sample was established spectrophotometrically (Digital Grating Spectrophotometer Series 2, Model CE393, Cecil Instruments Ltd., Cambridge, U.K.) in a cuvette at 546 nm, against a Drabkin's blank.

3. The relative absorbance (A) was calculated (absorbance of sample - absorbance of Drabkin's blank). The concentration of haemoglobin was given by the following equation:

$$\text{haemoglobin concentration (g.dl}^{-1}) = (37.2 * A) + 0.06$$

Spectrophotometric determination of plasma ammonia (340 nm)

Principle:

$$\text{2-Oxoglutarate} + NH_3 + NADPH \xrightarrow{\boxed{\text{GLDH}}} \text{Glutamate} + NADP$$

Reagents: The Sigma Diagnostics Ammonia kit was used

Initial concentrations of solutions:

Reagent solution: NADPH 0.23 mmol·l⁻¹
2-Oxoglutarate 3.4 mmol·l⁻¹

Enzyme: Glutamate dehydrogenase (GLDH) 1200 U·ml⁻¹
Phosphate buffer pH 7.4
Glycerol 50%

Procedure:

1. 500 µl of the reagent solution was added to 50 µl aliquots of samples or standards in a 1 ml disposable cuvette (light path: 1 cm). 2 reagent blanks were used for every assay.
2. The contents of each cuvette were mixed well upon addition of the reagent solution and were incubated at room temperature for 10 min.
3. After the 10 min of incubation the initial absorbance (A_1) of samples, blanks and standards was read at 340 nm (N.B. absorbance decrease).
4. After the absorbance was read, 5 µl of enzyme (GLDH) was added to each cuvette with a positive displacement pipette, the contents of each cuvette were mixed well and incubated for 10 min.
5. After the 10 min of incubation the absorbance of samples, blanks and standards was read again (A_2).
6. Steps 4 and 5 were repeated, and a final absorbance of samples, blanks and standards was read (A_3).

Cuvettes were sealed during incubations with a plastic cap.

The concentration of ammonia in the sample was calculated using the extinction coefficient for NADPH, as follows:
$(A_1-A_2) - (A_2-A_3) = DA_{BLANK}$ or $DA_{SAMPLE / STANDARD}$

Concentration (µmol·l⁻¹): 701.5 x ($DA_{SAMPLE / STANDARD} - DA_{BLANK}$)
Standards were used only to check the asay.

^{125}I radioimmunoassay for the determination of serum progesterone, cortisol, aldosterone, prolactin, growth hormone concentration

Principle:

^{125}I-labelled hormone (eg progesterone) and the hormone within the subjects serum sample compete for antibody sites on the wall of the polypropylene tube. Once incubated, simply removing the supernatant allows isolation of the antibody-bound fraction. The radioactivity in the tube is then established using a gamma counter, and the progesterone concentration present in the subject sample is inversely related to the counts. The exact concentration is determined using a standard curve.

Reagents:

A commercially available kit (Coat-A-Count, EURO / DPC Ltd., Caernarfon, U.K.) was used to perform the assay. It contained the required tubes, calibrators, and a concentrate of iodinated hormone. An automated gamma counter (Cobra II, Packard Instrument Company Inc., U.S.A.) was used to quantify the radioactivity.

Standards:

Lyophilized calibrators in processed human serum were provided with the commercial kit.

Quality Control:

A human serum-based quality control (CON6, EURO/DPC Ltd., Caernarfon, U.K.) was used at three concentrations.

Procedure:

1. Serum samples and quality controls were thawed at room temperature for 60 minutes, and mixed thoroughly.

2. Four plain (uncoated) polypropylene tubes were marked with a T or USB. Fourteen antibody-coated tubes were labelled A-G in duplicate for the kit calibrators, and the required number of antibody-coated tubes for serum samples were labelled appropriately.

3. A specified amount of calibrator was placed into the bottom of the NSB marked tubes.

4. A similar specified amount of each calibrator, quality control and serum sample was pipetted into the bottom of an antibody-coated tube.

5.   A specified quantity of iodinated hormone was then added to each tube (T, NSB, calibrators, quality controls and samples).

6.   All tubes were thoroughly vortexed and incubated for a specific length of time at a specific temperature for each hormone assay.

7.   At the end of the incubation period, the iodine solution was poured from each tube (except the 'T' tubes). Every effort was made to remove all the moisture from a tube by striking it sharply on absorbent paper for 2-3 minutes.

8.   The radioactivity present in each tube was then established using an automated gamma counter.

9.   A logit-log representation of the calibration curve, and a computer programme within the gamma counting system, was used to determine the actual insulin concentration of the samples and quality controls.
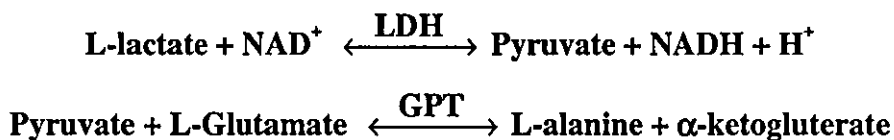
Cobas Mira Analysis

Glucose

Principle:

$$\text{Glucose} + O_2 + H_2O \xrightarrow{\quad \textbf{GOD} \quad} \text{Gluconate} + H_2O_2$$

Reagents: The Boehringer Mannheim GOD-PAP method and Test-Combination kit was used

Lactate

Principle:

$$\text{L-lactate} + NAD^+ \xrightarrow{\quad \textbf{LDH} \quad} \text{Pyruvate} + NADH + H^+$$

$$\text{Pyruvate} + \text{L-Glutamate} \xrightarrow{\quad \textbf{GPT} \quad} \text{L-alanine} + \alpha\text{-ketogluterate}$$

Reagents: The Boehringer Mannheim method and Test-Combination kit was used