Master of Science Thesis

Investigation of the Relationships that Exist between Athletic Training, Hormones and Sleep in Young Healthy Male Athletes

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Abstract

Background. Many people engage in exercise for recreation, to promote personal health and as a profession. Accordingly there is wide ranging interest in the factors that affect a person's performance during exercise and in how that performance can both be assessed and enhanced. The physiological basis of exercise performance and its enhancement have been investigated for many years. Such investigations in people are impeded by the understandable reluctance of participants to provide significant numbers of blood samples by venepuncture. The recent development of an ultrasound method for non-invasive sampling of extracellular fluid, called transdermal electrosonophoresis (ESP), offers tremendous opportunities for benign monitoring of physiological responses involving changes in blood/extracellular fluid composition associated with exercise and indeed in clinical settings. Sleep quality/quantity is considered to have significant impact on training effectiveness and performance, with poor sleep correlated with poor athletic outcome. The link here is considered to involve growth hormone, as poor sleep quality/quantity diminishes growth hormone concentrations and reduced growth hormone concentrations impede training induced muscle development. Training effectiveness and recovery have been monitored in past research through measurement of blood hormone profiles, in particular the testosterone: cortisol ratio. The overall objective of this study was to validate the use of ESP as a non-invasive blood sampling technique through the study of the relationships that exist between exercise, fatigability, fitness, and hormone levels in blood, saliva and extracellular fluid and the investigation of the impact of spontaneous sleep disturbances on these relationships in young healthy male athletes. ***Methods.*** Plasma, ESP and saliva samples were taken regularly from 14 male rugby players during a four-week study. The plasma and ESP samples were analysed for testosterone, cortisol and growth hormone concentration. The saliva samples were analysed only for testosterone and cortisol levels. Fitness was assessed each week using a maximal treadmill test and fatigability was also investigated. Sleep quantity/quality was investigated using personal sleep logs which the participants filled out daily. In addition the participants' alcohol consumption was reported in the sleep log. ***Results.*** Correlations between hormone concentrations measured in plasma and ESP were higher than the correlations for plasma and saliva. The results here were highly significant. An equation was derived to estimate plasma concentrations of

testosterone, cortisol and growth hormone using the concentrations of the hormones measured in ESP samples. Few statistically significant relationships between hormone profiles, sleep quality/quantity and athletic training were revealed in the analysis of the results of this study. A negative correlation was found between the mean plasma cortisol concentration measured in the morning and the estimated VO_{2max}, and also between the cortisol concentration measured in the ESP sample and estimated VO_{2max}, but this did not quite reach significance. A negative correlation between estimated VO_{2max} and the mean total time asleep for the previous three or four nights was revealed. ***Conclusions.*** ESP sample analysis provides a more accurate estimation of testosterone, cortisol and growth hormone concentrations in plasma than saliva sample analysis does.

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1.1 INTRODUCTION

New Zealand is a country of generally quite active people. The Hillary Commission (now Sport and Recreation New Zealand) defines the minimum level of physical activity required to improve a person's health and well-being as 2.5 hours per week and such person is called 'active' (Hillary Commission, 1999). In New Zealand, two out of three people over the age of five are active. In this study being 'active' will be referred to as partaking in some form of exercise. We can define exercise as the contraction of skeletal muscle for the purposeful movement of the body. These movements are controlled by the electrical conduction system of the central nervous system and include both reflex and voluntary contractions. The acute adaptations to exercise are mediated through the integration of the nervous and the endocrine systems. The above definition allows many activities to be considered as a form of exercise, from walking to sprinting, soccer to gymnastics and also other activities of daily life including work related physical activity. It is not only of interest to investigate the processes that occur during exercise but also to examine how other processes in the body essential for life, such as sleep, may impact on adaptations to exercise.

There were three objectives in this study:

1. With particular reference to testosterone, cortisol and growth hormone, to compare their concentrations in plasma and saliva with those derived by transdermal electrosonophoresis (ESP), a novel methodology. The aim here is to further validate the use of ESP as a non-invasive sampling methodology.
2. To explore relationships between exercise, fatigability (as a measure of fitness), fitness increases as assessed by maximum oxygen uptake (VO_{2max}) and the ventilatory anaerobic threshold, and the hormone levels in plasma, ESP and saliva samples.
3. Using spontaneous disturbances in sleep quality/quantity, to explore their impact on hormone profiles and athletic performance.

The physiological basis of human exercise performance and its enhancement have been investigated for many years. Such investigations are sometimes impeded by the reluctance of participants to provide significant numbers of blood samples by

venepuncture. The recent development of an ultrasound method for non-invasive sampling of extracellular fluid (transdermal electrosonophoresis) offers tremendous opportunities for benign monitoring of physiological responses involving changes associated with exercise (Cook, 2002). The opportunities in clinical settings are even wider. As this method is still in it infancy the extracellular fluid composition determined by electrosonophoresis must be correlated with the composition of plasma and/or saliva samples, in order to validate its development for widespread use. Cook (2002) investigated the best method of collecting transdermal exudate in sheep and humans. The result of this study was that high correlations between plasma and ESP sample concentrations were seen both at rest and during exercise and recovery in both species. This was unlike salivary concentrations where post exercise correlations with plasma decrease; this may be due to a time lag in partitioning of constituents between plasma and saliva which is complex and poorly understood (Quissell, 1993).

Sleep quality and quantity are considered to have significant impact on training effectiveness and performance, with poor sleep correlated with poor athletic outcome (Mougin et al, 1991), however, there is apparently no agreement in the literature about the effect of sleep deprivation on physiological responses to exercise. This will be discussed further below. Poor sleep is likely to have a negative impact on the effectiveness of athletic training and the link here is considered to involve growth hormone, as poor sleep quality/quantity diminishes nocturnal growth hormone secretion (Van Cauter et al, 1992). Reduced growth hormone concentrations impede training-induced muscle development (reviewed in Jenkins, 1999). Training effectiveness and recovery have been monitored in past research through measurement of plasma hormone profiles, in particular the testosterone:cortisol ratio (Hakkinen et al, 1987).

What was proposed for the present thesis research was a pilot study to investigate these phenomena. Before providing details of the present study, however, literature relevant to different aspects of the topic will be considered.

1.2 PHYSIOLOGICAL RESPONSES TO EXERCISE

1.2.1 Metabolic Demands of Exercise

1.2.1.1 Bioenergetic systems to support muscle contraction

The following details are well established and are reviewed extensively by Poortmans (1993). Energy in the form of ATP is synthesised in the body from energy-rich nutrients in food. When these nutrients (carbohydrates, fats, proteins) are metabolised they release ATP to fuel muscular contraction and other biological processes. Three bioenergetic systems exist to produce ATP, two of which are anaerobic and the other is aerobic. The phosphagen system comprises the stored ATP and phosphocreatine within cells; enough to maintain vigorous muscular activity for between three and 15 seconds. This anaerobic system is utilised during short duration, high intensity activities such as the 100m sprint or lifting a box. The other anaerobic system, the glycogen-lactic acid system, involves glucose mobilisation and anaerobic glycolysis producing pyruvate which in the absence of oxygen, is converted into lactic acid. It is able to produce ATP for longer than the phosphagen system but as the lactic acid accumulates in skeletal muscle, the decline in pH impedes the further breakdown of glucose. This system is primarily used in high intensity, short duration events such as a 400m sprint.

In the presence of oxygen, the pyruvate produced during glycolysis is converted into acetyl CoA. This then enters the tricarboxylic acid (TCA) cycle, producing 38 ATP molecules for every one acetyl CoA molecule compared with only 2 ATP molecules from anaerobic glycolysis. This third system, termed the oxidative system, is predominantly used during endurance type activities and yields far more ATP than either of the anaerobic systems. Fats can also be used to produce energy via the oxidative system, one 18-carbon chain fatty acid producing 147 ATP molecules. Most exercise activities require a blend of both anaerobic and aerobic metabolism. For example, the energy required for the beginning sprint and end sprint in a 1500m race will be derived from the anaerobic systems while the middle of the race will rely predominantly on the aerobic system. Table 1.1 compares the capacities of each of the bioenergetic systems in a 70kg male.

Table 1.1. Maximum rates of active phosphate production from different substrates and amounts available in a 70 kg man (muscle mass estimated to 28 kg) (Greenhaff et al, 1993).

System	Conversion	Rate (mol/min)	Amount Available (mol)
Phosphogen	ATP, PCr → ADP, Cr	4.4	0.67
Glycogen* – lactic acid	Glycogen* → lactate	2.35	1.6
Oxidative	Glycogen → CO_2	0.85 - 1.14	84
	Liver glycogen → CO_2	0.37	19
	Fatty acids → CO_2	0.40	4000

* Glycogen stored in muscle

1.2.1.2 Influence of intensity, duration and type of exercise

During rest the dominant bioenergetic system being used is the oxidative system. About two-thirds of the fuel being metabolised is fat, the remainder is glucose, as the contribution of protein is negligible (Greenhaff et al, 1993). During exercise both the anaerobic and aerobic systems contribute ATP, with their relative roles depending on the intensity of exercise, the state of training, and the diet of the athlete.

At exercise intensities below maximal oxygen consumption (VO_{2max}) the aerobic system dominates the supply of ATP for muscular contraction. As the exercise intensity increases there is a higher demand for ATP, but the aerobic system is limited in supplying adequate ATP for two reasons. Firstly, each person has an upper limit to the rate at which they can consume oxygen and secondly, it takes two to three minutes for ATP production to increase to a new higher level using this system. At low rates of oxygen consumption, ATP is predominately supplied by the oxidation of free fatty acids. However, during incremental exercise, as exercise intensity increases, a shift in substrate utilisation occurs with an increase in carbohydrate metabolism and a decrease in lipid metabolism – this is known as the "crossover concept" (Brooks and Mercier, 1994). Once the maximal rate of oxygen consumption by the aerobic system is reached, anaerobic systems contribute to ATP production at an increasing rate while the aerobic system continues to produce ATP.

Training, depending on the type, has several biochemical effects that improve the capacity of the bioenergetic pathways. Anaerobic training results in an increase in the

muscular stores of ATP and phosphocreatine (Eriksson et al, 1973), an increase in the activities and concentrations of enzymes involved in the phosphogen system (Thorstenson et al, 1975) and an increase in the activities and concentration of enzymes involved in the glycogen-lactic acid system (Costill et al, 1976; Eriksson et al, 1973; Gollnick et al, 1973).

Endurance (aerobic) training results in an increase in the myoglobin content of muscle (Mole et al, 1971), which is important in the delivery of oxygen from the cell membrane to the mitochondria where it is consumed. There is also an increase in the size and number of mitochondria present in skeletal muscle cells (Costill et al, 1971), the amount of glycogen stored (Gollnick et al, 1972), and an increase in the level of activity and concentration of enzymes involved in the tricarboxyclic acid (TCA) cycle and the electron transport chain (Gollnick et al, 1973). Additionally there is an increase in intramuscular stores of triacylglycerides (TAG), an increase in the mobilisation of free fatty acids from adipose tissue and an increase in the concentration of enzymes involved in the oxidation of fatty acids in the skeletal muscle cell (Gollnick, 1977). There is also a decrease the sympathetic nervous system responses to given submaximal exercise levels with aerobic training (Brooks and Mercier, 1994). These biochemical adaptations enhance lipid oxidation during mild- to moderate-training intensity so that trained individuals oxidise a greater proportion of fat at similar workloads than untrained individuals (Gollnick, 1977). Aerobic training therefore results in adaptations that conserve glycogen stores by utilising the body's stores of free fatty acids in adipose tissue. This prolongs the time to exhaustion as glycogen depletion results in exhaustion (Brooks and Mercier, 1994).

Likewise, diet can have an effect on athletic performance. In particular a high carbohydrate diet prior to competition (carbohydrate loading) increases the concentration of stored glycogen in muscle and prolongs time to exhaustion in endurance activities (Bergstrom et al, 1967). Consumption of a high fat meal prior to exercise can also improve performance by increasing the amount of fat available for use and therefore may prolong the time to exhaustion by glycogen sparing (Costill et al, 1977).

1.2.1.3 Specifics of rugby training and the game

The energy demands during a rugby union football match can be estimated by a combination of notational analysis and metabolic measurement (Nicholas, 1997). Notational analysis involves the observation and recording of game play. At this point it should be noted that, due to different player position requirements in a game, the physiological demands of particular positions may not be reflected correctly by studies that present mean values.

While a match is made up of two 40-minute halves plus the time that the referee adds at the end of a game, it has been estimated that the ball is in play for an average of 29 minutes (McLean, 1992). Morton (1978) estimated that the distance covered by players during a game is approximately 5.8km and that this is made up of 37% walking, 29% jogging and 34% striding or sprinting. In a study on Canadian players it was concluded that 85% of the time is spent in low-intensity activity, likely due to the need to resume formations in order for play to continue following a quick switch from one end of the field to the other as a result of tactical kicking and running with the ball (Docherty et al, 1988). The remaining 15% of the time is spent on intense exercise, 6% being related to running and 9% to tackling, pushing and competing for the ball.

Treadwell (1988) splits the analysis of time spent in movement modes into the two distinct playing units in a rugby union team, forwards[1] and backs[2]; the results are presented in Table 1.2 and Table 1.3 (In: Nicholas, 1997). Non-purposive running refers to all types of movement and stationary periods when a player is engaged in game play. Purposive running includes jogging and running up to three-quarters of maximal pace.

[1] Forwards – eight players located in three rows in the scrum; their role is to win and retain possession in set pieces (scrums and line-outs) and in open play (rucks and mauls).

[2] Backs – seven players including a half back (coordinates play between forwards and backs), a first five (coordinates the back line), two centres and two wings and a full back (the last defensive player); the role of the backs is to run into spaces in order to score tries.

Table 1.2. Combined time and motion analysis for rugby union forwards (Nicholas, 1997).

	Movement Category [min.sec (no.)]			
	Nonpurposeful Running	**Purposeful Running**	**Scrums**	**Rucks/ mauls**
Playing position				
Front five	47.18	17.12	9.14 (52)	6.32 (53)
Back row	55.10	18.00	6.28 (48)	4.02 (56)
Mean time	51.14	17.36	7.51 (50)	5.17 (54.5)
Proportion of total time (%)	63.80	21.50	9.19	6.26

Table 1.3. Combined time motion analysis for rugby union backs (Nicholas, 1997).

	Movement category (min.sec)			
	Nonpurposeful Running	**Purposeful Running**	**Sprinting**	**Time With Ball**
Playing position				
Half-back	51.04	21.28	2.16	1.52
Centre	55.16	14.42	2.14	1.20
Wing/full back	69.24	9.54	0.56	2.16
Mean time	58.35	15.21	1.49	1.29
Proportion of total time (%)	73.20	19.18	2.25	1.85

McLean (1992) investigated the density of work during a rugby union football match by timing work:rest ratios (W:RRs) and concluded that most work periods were 19-seconds in length with the most frequent W:RRs being in the range of 1:1 to 1:19. He also concluded that a scrum, line-out, ruck or maul occurred on average every 33-seconds. Notational analysis of a rugby union game may underestimate total match demands due to any additional energy expenditure required for accelerations, decelerations, running with the ball, running sideways, jumping in the lineouts, pushing in the scrums, rucks and mauls, tackling and other game related activities. For these activities the energy would be provided primarily by the phosphagen system.

Metabolic measurements, such as blood lactate levels, may provide additional information about what energy supplying systems are in use. Most studies report a blood lactate concentration of between 2.8-9.8mmol^{-1} (Reilly, 1997). Measurements of

blood lactate are taken during stoppages, at half time or at the end of the match. This may grossly underestimate the importance of the glycolytic pathway in the production of energy during a rugby match as lactate concentration at any one time is the balance between the rates of production and removal and is a measure of only the preceding few minutes of physical activity and metabolism (Brooks, 1985).

This information supports the classification of rugby union as an intermittent high intensity sport. Thus emphasis in training should be placed not only on the power of the anaerobic energy system in order to perform brief periods of maximal exercise, but also on improving the capacity of the aerobic system to enhance recovery between bursts of maximal exercise (Maud, 1984).

1.2.2 Physiological Support System Changes and Their Control

1.2.2.1 Cardiorespiratory adaptations to exercise

Exercise imposes stress upon many physiological systems throughout the body. In order to maintain optimal performance it is essential that a complex series of responses is coordinated and controlled. There are three regulatory roles of the cardiovascular system during exercise in the human, (1) the proper control of arterial blood pressure, (2) augmentation of blood flow to active muscle, and (3) the maintenance of internal body temperature (Seals et al, 1994). Turner (1991) describes the 'exercise reflex', which is initiated within the active muscle mass by a build up of metabolites due to a mismatch between perfusion and muscle metabolism. Chemoreceptors within the muscle register an imbalance and increase their firing rate in chemosensitive afferent nerves linked to the central nervous system (CNS). The efferent arm of the reflex arc then activates increases in ventilation, central and peripheral components of blood flow and blood pressure. Two neural mechanisms, central command and a reflex originating in contracting muscles, are known to play a large role in exercise-associated adjustments in cardiovascular and respiratory activity (Kramer and Waldrop, 1998). Relevant features of the nervous system's involvement in the changes that occur during exercise will be outlined here. Nervous control of the cardiorespiratory system is paramount to the overall functional efficiency of this system. The task of receiving information relative to pulmonary ventilation and blood flow, integrating it and then initiating a response to match mechanical and metabolic demands occurs within the

cardiovascular and respiratory control areas of the brain. These control areas are located specifically within the medulla oblongata of the brain stem (Kramer and Waldrop, 1998). The cardiovascular and respiratory areas are neurally interconnected, so that stimulation of one area will, via its connection with the other, generally affect both ventilation and blood flow (Harper, 1996).

Turner (1991) described three phases of moderate exercise. In phase 1 (0-15s) the cardiorespiratory responses are rapid. In fact they are so rapid that purely neural control mechanisms are thought to be responsible for the initial actions of the various physiological systems. The two most important neural control systems responding during phase one are (a) mechanical feedback reflexes originating from the active muscle mass and (b) a centrally generated feedforward motor pattern. The primary mechanism causing the increase in heart rate at this point is considered to be the decrease in efferent cardiac parasympathetic nerve activity to the sinoatrial node (Seals et al, 1994). This allows cardiac output and therefore blood pressure to rise. Additionally, it is likely that there is some sympathetic vasoconstrictor activity to the viscera so that blood can begin to be diverted to the metabolically more active skeletal muscle (Turner, 1991). At the same time, the operating point for the carotid artery and aortic baroreceptors is quickly reset to a higher level, which is likely to contribute to a further decrease in parasympathetic tone (DiCarlo and Bishop, 1992). As exercise proceeds to phase two (15s to two or three min), slower increases in cardiorespiratory variables occur until a new steady state is reached (phase three, three min onwards) during which neural and humoral mechanisms combine to bring about an appropriate response (Turner, 1991). During phase two, the cardiovascular and respiratory systems begin to attain a steady-state response profile and each physiological system comes increasingly under the influence of neural afferent inputs originating in the lungs, the heart, the carotid bodies, muscle chemoreceptors, the arterial baroreceptors and thermoreceptors (Turner, 1991). In addition, feedback control mechanisms, act directly on the central nervous system or indirectly via peripheral receptor systems (Turner, 1991). As the intensity of work effort increases, stimulation by efferent cardiac sympathetic neurons of the sinoatrial node and ventricular muscle causes release of noradrenaline which in turn binds to β-adrenergic receptors resulting in further increases in heart rate and myocardial contractility (Seals et al, 1994). The increase in sympathetic tone is caused by activation of the muscle metaboreceptor, by further

involvement of the baroreceptors, and possibly by activation of the muscle mechanoreceptors (O'Leary, 1993). During the steady state (phase three), prolonged exercise may be compromised by thermoregulatory and fluid homeostatic imbalances as well as limiting changes in substrate utilisation and delivery (Turner, 1991). At near maximal effort, parasympathetic activity is very low, sympathetic activity exists at greatly increased levels, and the release of adrenaline into the circulation will lead to further activation of the cardiac β-adrenergic system and greater stimulation of heart rate and ventricular contractility (Seals et al, 1994). As a result, heart rate, stroke volume and cardiac output all operate maximally. Following maximal exercise, heart rate decreases sharply due to a decrease in the enhanced sympathetic nerve activity; activation of parasympathetic activity plays a greater role once heart rate has partially recovered (Savin et al, 1982).

Foss and Keteyian (1998) classified the incoming stimuli into four functional groups: central command, humoral, physical and peripheral neural. These are summarised in Table 1.4.

Central motor command

Input into the cardiorespiratory centre occurs mostly via neurons that originate in the motor cortex and pass through the cardiorespiratory centre on their way to initiate a skeletal muscle action. The central command involves a parallel, simultaneous excitation of neuronal circuits controlling both the locomotor and cardiorespiratory systems. Actions initiated by the cardiorespiratory centres influence neural activity to the heart and blood vessels as well as neural outflow to the motor neurons innervating respiratory muscles. In addition central command can be initiated by mental conditions integrated by the hypothalamic region of the lymbic system.

Humoral stimuli

Humoral stimuli originate from changes in the chemical properties of blood or cerebro-spinal fluid, which ultimately influence receptors located elsewhere in the body. Once activated, these receptors provide afferent neural input to the cardiorespiratory area, which then evokes an appropriate response. The receptors are primarily chemoreceptors that are sensitive to changes in fluid chemistry, such as PO_2, PCO_2, K^+ concentration and/or pH. These chemoreceptors are located in the medulla (central chemoreceptors) or

elsewhere in the body, such as the aortic bodies or in the carotid bodies found at the bifurcation of the carotid arteries.

Physical stimuli

Changes in the physical characteristics of blood (e.g. pressure and volume) are classified as physical stimuli. There are pressure sensitive mechanoreceptors (the baroreceptors) located in the aortic arch and carotid arteries. Low pressure baroreceptors located in the atria, ventricles, pulmonary artery and pulmonary vein are also involved.

Peripheral neural stimuli

These stimuli originate from changes that take place in the lungs, muscles, joints, tendons and skin, and result in an afferent neural response being generated toward the cardiorespiratory areas in the brain. The feedback information they provide is concerned with (1) changes in the local chemistry (and possibly temperature) in and around the skeletal muscle; (2) muscle contraction and limb movement or tension development; and (3) intense pain, general discomfort and/or the presence of respiratory irritants.

During exercise, the respiratory rate increases to maintain optimal concentrations of O_2, CO_2 and H^+ in the tissues. There are similarities between control of the ventilatory system and cardiovascular regulation. Both the respiratory and the cardiovascular areas of the medulla receive impulses from the descending higher motor regions of the cerebrum and the ascending afferent impulses of the periphery (Weissman et al, 1979). The simultaneous increases in cardiac output and ventilation involves the direct activation of ventilation by a signal from the heart itself or from within the blood flowing from it – this is described as "cardiodynamic coupling' (Turner, 1991).

Table 1.4. Action of various stimuli on the cardiorespiratory areas of the brain stem and their effects on respiration and circulation (Foss and Keteyian, 1998).

	Action on Cardiorespiratory Areas				Effects		
			Receptor			Circulation	
Stimuli	**Feed-forward**	**Feed-back**	**Type**	**Location**	**Respiration Rate**	**Heart Rate**	**Arteriolar Tone**
Central command	✔				↑↑↑	↑↑↑	↑↑↑
Humoral							
Increased PCO_2, H^+, K^+	✔ ?	✔	Chemo	Brainstem, carotid arteries or aortic arch	↑↑	↑	↑
Decreased PO_2		✔	Chemo		↑	↑	↑
Increased (nor)adrenaline		✔	Chemo		↑	↑	↑
Physical							
Increased pressure		✔	Baro	Aortic arch, carotid arteries	↓	↓	↓
Increased volume		✔	Pressure or stretch	Right atrium/ pulmonary artery	↓	↓	↓
Peripheral neural							
Respiratory muscles		✔	Stretch or mechano	Muscle, joints, tendons	↑	↑?	?
Other skeletal muscle							
Mechanical		✔	Mechano	Muscle	↑?	↑	↑
Metabolic		✔	Metabo		↑?	↑↑	↑↑

1.2.2.2 Thermoregulation

Normally body temperature is regulated around a set point, and, generally, heat production is equal to heat loss (Gleeson, 1998). As heat production increases with exercise, heat is stored, increasing body temperature (Gleeson, 1998). Such a deviation from a set point is detected by sensory input from the body triggering reflexes that help to eliminate or in the case of core temperature being below the set point, to conserve heat. The thermoregulatory system of the body uses a thermal regulatory centre, thermal receptors and thermal effectors to carry out its function.

1.2.2.2.1 Thermal regulatory centre

The raphe nuclei located in the midbrain are key sites involved in integration of peripheral and central thermal stimuli and in processing of information ascending to the preoptic/anterior hypothalamic area (Bruck and Hinckle, 1980). Studies have shown that thermoafferent inputs from the skin converge in the hypothalamus (Cabanac, 1975; Ivanov et al, 1981) and it is at this site that incoming sensory information is coordinated with outgoing regulatory action (Gordon, 1986). The internal temperature is measured by receptors and compared with a set point and if the measured temperature deviates from the set point, the information is relayed to thermal effectors (see below), which correct the body temperature (McEwen and Heath, 1974).

1.2.2.2.2 Thermal receptors

Thermal receptors or sensors are sensitive to heat and cold stimuli and provide neural input to the co-ordinating centre. Central receptors are located in the anterior hypothalamus, peripheral receptors are located in the skin, thermoreceptors also exist in the spinal cord, abdominal viscera and deep body veins (Hensel, 1974). Peripheral receptors sense changes in ambient temperature while the internal receptors monitor changes in core temperature (Hensel, 1973). The heat receptors increase their rate of firing as the temperature of the blood perfusing the area increases, while the cold receptors increase their rate of firing when the temperature of the blood perfusing the area decreases (Hensel, 1974). There are three times as many heat receptors as cold receptors in the hypothalamus; these are sensitive to small temperature fluctuations within the arterial blood perfusing the area (Hensel, 1973). The heat, cold and pain receptors located just below the skin consist mainly of free nerve endings; the pain

receptors are sensitive to extremes of heat and cold (Hensel, 1973). In contrast to the hypothalamic receptors, there are three to ten times more cold than warm receptors in the peripheral and core body areas (Hensel, 1973).

1.2.2.2.3 Thermal effectors

Thermal effectors or organs are directed by the hypothalamic co-ordinating centre to produce regulatory or corrective changes these include skeletal muscle, smooth muscle encircling the arterioles that supply blood to the skin, the sweat glands, and certain endocrine glands (Benzinger, 1969). In warm conditions arterioles supplying blood to the skin dilate, facilitating heat dissipation and sweating occurs (Brengelmann, 1983). In the cold, cutaneous vasoconstriction occurs, decreasing the amount of heat lost to the environment through the skin, the metabolic rate is also increased by shivering and there may be an increase in the secretion of adrenaline from the adrenal medulla (Lossec et al, 1998). In addition to non-voluntary reflexes that help to eliminate or conserve body heat, cortical connections provide a means for voluntary (behavioural) regulation, for example seeking or avoiding shade, removing or adding clothing (Benzinger, 1969).