The use of near infrared spectroscopy to examine the neural basis of affective responses during exercise at different intensities

Gavin D. Tempest BSc (Hons)

A thesis submitted for the degree of

Doctor of Philosophy (Health Sciences)

School of Health Sciences
Division of Health Sciences
University of South Australia

February 2014

Table of contents

List of figures vi
List of tables ix
Glossary ix
Summary xii
Declaration xiv
Acknowledgement xv

1 Introduction 1

1.1 Rationale 1

1.2 Overview of the thesis 3

2 The neural basis of affective responses to the intensity of exercise: background, theory, mechanisms and measurement 5

2.1 Exercise-induced affective responses 6

2.1.1 Definition 6

2.1.2 Exercise intensity-affect relationship 6

2.1.3 The dual-mode model (DMM) 11

2.2 Neural basis of affective responses 14

2.2.1 Amygdalaloid complex 14

2.2.2 Sensory (interoceptive) pathways to the brain 16

2.2.3 Prefrontal cortex 17

2.2.3.1 Dorsolateral frontal cortex 18

2.2.3.2 Ventromedial frontal cortex 18

2.2.3.3 Orbitofrontal Cortex 19

2.2.4 Anterior cingulate and Insula 19

2.3 Mechanisms of affective responses 20

2.3.1 Cognitive control processes 20

2.3.2 Frontal cortex asymmetry 22

2.3.3 Frontal cortex deregulation 24

2.4 Cognitive versus sensory processes 26

2.4.1 Individual differences 27

2.4.1.1 Tolerance of the intensity of exercise 27
2.5 Methods and techniques to investigate neural pathways during exercise 29
2.5.1 Near infrared spectroscopy 30
2.6 Summary 32
3 General Methods 33
3.1 Participants 34
3.2 Measures 34
3.2.1 Exercise-induced affective responses 34
3.2.2 Preference for and tolerance of the intensity of exercise questionnaire 35
3.2.3 Near infrared spectroscopy 36
3.2.3.1 Principles of near infrared spectroscopy 37
3.2.3.2 Cerebral haemodynamic response and neuronal activation 38
3.3 Procedures 39
3.3.1 Initial Assessments 39
3.3.2 Near infrared spectroscopy setup 39
3.3.2.1 Optode placement 39
3.3.2.2 Dual-channel near infrared spectroscopy (studies I, III, V) 40
3.3.2.3 Multi-channel near infrared spectroscopy (studies II, IV) 41
3.3.3 Initial incremental exercise test to exhaustion 42
3.4 Data Reduction 42
3.4.1 Identification of gas exchange thresholds 42
3.4.2 Determination of exercise intensities 46
3.4.3 Cerebral haemodynamic variables 46
3.5 Power analyses 46
3.5.1 Affective responses 46
3.5.2 Cerebral haemodynamic variables 47
3.5.3 Acoustic startle eye blink responses 47
3.6 Statistical analyses 47

4 Study I: Cerebral blood flow in the frontal cortex during incremental exercise: An examination of self-reported tolerance 49

4.1 Background 50

4.2 Methods 51

4.2 Results 53

4.2.1 Preliminary analyses 53

4.3.2 Cerebral haemodynamics (hypothesis 1) 55

4.3.3 Affective responses (hypothesis 2) 57

4.3.4 Exercise Duration (hypothesis 3) 58

4.4 Discussion 59

5 Study II: Cerebral blood flow in sub regions of the frontal cortex during incremental exercise: An examination of affective responses 64

5.1 Background 65

5.2 Methods 67

5.3 Results 69

5.3.1 Cerebral haemodynamics (hypothesis 1) 69

5.3.2 Affective responses (hypothesis 2) 73

5.3.3 Correlations (hypothesis 3) 73

5.4 Discussion 74

6 Study III: Cerebral blood flow in the frontal cortex during exercise: An examination of a cognitive strategy (imagery) 79

6.1. Background 80

6.2 Methods 82

6.3 Results 85

6.3.1 Preliminary Analyses 85

6.3.2 Cerebral haemodynamics (hypothesis 1) 86

6.3.3 Affective responses (hypothesis 2) 88

6.3.4 Correlations (hypothesis 3) 90

6.4 Discussion 91

7 Study IV: Cerebral blood flow in the frontal cortex during exercise: The influence of prior exhaustive activity 95

7.1 Background 96

7.2 Methods 98

7.3 Results 101

7.3.1 Preliminary Analyses 101

7.3.2 Cerebral haemodynamics (hypothesis 1) 102

7.3.3 Rating of perceived exertion and affective responses (hypothesis 2) 110

7.3.4 Exercise duration (hypothesis 3) 112

7.4 Discussion 112

8 Study V: Cerebral blood flow in the frontal cortex and activity in the amygdala during exercise: The influence of self-reported tolerance 118

8.1 Background 119

8.2 Methods 122

8.3 Results 126

8.3.1 Preliminary analyses 126

8.3.2 Cerebral haemodynamics (hypothesis 1) 127

8.3.3 Acoustic startle eye blink response (hypothesis 2) 129

8.3.4 Affective responses (hypothesis 3) 129

8.3.4 Correlations (hypothesis 4 and 5) 129

8.4 Discussion 130

9 Discussion and conclusions 136

9.1 Review of aims 137

9.2 Main findings 138

9.2.1 Affective responses 138

9.2.2 Cerebral haemodynamics 138

9.2.3 Acoustic startle eye blink response 140

9.3 The dual-mode model 140

9.4 Mechanisms of affective responses 141
9.4.1 Cognitive control processes 142
9.4.2 Frontal cortex asymmetry 143
9.4.3 Frontal cortex deregulation 145
9.4.4 Tolerance of the intensity of exercise 147
9.5 Limitations 148
9.6 Directions of future research 151
9.7 Conclusions 154
References 156
Appendices 181

List of figures

2.1. The circumplex model of affect 7

2.2. Exercise intensity domains 9

2.3. A representation of the respiratory response across exercise intensities 10

2.4. A representation of the pattern of affective responses across exercise intensities 11

2.5. The proposed dual-modes of input to the amygdala 12

2.6. A representation of the neural structures proposed to be involved in the dual-modes of input to the amygdala 14

2.7. A stylised representation of the approximate location of cortical regions of the brain according to Brodmann's areas 18

3.1. The Feeling Scale 34

3.2. The location of optodes using dual-channel NIRS 40

3.3. The location of optodes using multi-channel NIRS 41

3.4. Identification of the ventilatory threshold 44

3.5. Identification of the respiratory compensation point 45

4.1. Approximate locations of the dorsolateral regions of the frontal cortex 52

4.2. Cerebral oxygenation (ΔO_2Hb) in the frontal cortex at each time point for the high and low tolerance groups 56

4.3. Cerebral blood volume (ΔtHb) in the frontal cortex at each time point for the high and low tolerance groups 57

4.4. Affective responses reported at each time point for the high and low tolerance groups 58

5.1. Approximate locations of sub regions (1-8) of the frontal cortex 67

5.2. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex at each time point 70

5.3. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex across time 71

5.4. Cerebral oxygen saturation (ΔHbDiff) in sub regions of the frontal cortex at each time point 72

5.5. Affective responses reported at each time point 73

6.1. Approximate location of the dorsolateral regions of the frontal cortex 82

6.2. Affective responses reported at each time point in the imagery and control conditions 89

6.3. Affective responses reported at each time point during exercise at intensities above VT and at RCP to exhaustion 89

7.1. Approximate location of sub regions (1-8) of the frontal cortex 99

7.2. Cerebral oxygenation (ΔO_2Hb) in the experimental and control conditions at each time point 103

7.3. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex at each time point 104

7.4. Cerebral deoxygenation (ΔHHb) in the experimental and control conditions at each time point 105

7.5. Cerebral blood volume (ΔtHb) in the experimental and control conditions at each time point 107

7.6. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex at each time point 107

7.7. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex in the experimental and control conditions at each time point 108

7.8. Cerebral oxygen saturation (ΔHbDiff) in the experimental and control conditions at each time point 109

7.9. Cerebral oxygen saturation (ΔHbDiff) in sub regions of the frontal cortex at each time point 110

7.10. Rating of perceived exertion reported in the experimental and control conditions at each time point 111

7.11. Affective responses reported in the experimental and control conditions at each time point 112

8.1. Approximate location of the dorsolateral regions of the frontal cortex 123

8.2. Cerebral oxygenation (ΔO_2Hb) in regions of the frontal cortex at each intensity of exercise 127

8.3. Cerebral oxygen saturation (ΔHbDiff) in regions of the frontal cortex at each intensity of exercise 128

The image of the brain in Figures 2.5 and 2.6 was modified from Gibb, B. J. (2007). *The Rough Guide to the Brain*. London: Rough Guides. The image of the brain in Figures 2.7, 3.2, 3.3, 4.1, 5.1, 6.1, 7.1 and 8.1 was retrieved from http://www.csus.edu and annotated.

List of tables

4.1. Oxygen uptake ($ml \cdot kg^{-1} \cdot min^{-1}$) for males and females in the high and low tolerance group at each time point 54

5.1. Oxygen uptake ($ml \cdot kg^{-1} \cdot min^{-1}$) at each time point and duration of exercise to exhaustion 69

5.2. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex 70

5.3. Cerebral deoxygenation (ΔHHb) in sub regions of the frontal cortex 72

6.1. Cerebral haemodynamics (ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff) at each time point during exercise above VT 87

6.2. Cerebral haemodynamics (ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff) at each time point during exercise at RCP to exhaustion 88

7.1. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex 103

7.2. Cerebral deoxygenation (ΔHHb) in sub regions of the frontal cortex 105

7.3. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex 106

7.4. Cerebral oxygen saturation (ΔHbDiff) in sub regions of the frontal cortex 109

Glossary

Haemodynamic

Hb	haemoglobin
ΔO_2Hb	change in oxygenated haemoglobin (oxygenation)
ΔHHb	change in deoxygenated haemoglobin (deoxygenation)
ΔtHb	change in total haemoglobin (blood volume)
ΔHbDiff	change in haemoglobin difference (oxygen saturation)

Measures

PRETIE-Q	preference for and tolerance of the intensity of exercise questionnaire
FS	the feeling scale
RPE	rating of perceived exertion

Physiological

BMI	body mass index
GET	gas exchange threshold
VT	ventilatory threshold
RCP	respiratory compensation point
$\dot{V}O_2$	rate of oxygen uptake
$\dot{V}O_{2peak}$	rate of peak oxygen uptake
$\dot{V}CO_2$	rate of carbon dioxide output
$\dot{V}E/\dot{V}O_2$	ventilatory equivalent of oxygen uptake
$\dot{V}E/\dot{V}CO_2$	ventilatory equivalent of carbon dioxide output
$ExCO_2$	excess carbon dioxide output
$PETCO_2$	end-tidal carbon dioxide
HRR	heart rate reserve

Techniques

ASER	acoustic startle eye blink response
NIRS	near infrared spectroscopy

Theoretical

BA	Brodmann's areas
DMM	dual-mode model

Summary

How we feel (i.e. pleasure-displeasure) during exercise is important for future exercise participation and adherence. However, what happens in the brain that might explain these affective responses has not been confirmed. It has been proposed that the interplay between cognitive processes in the frontal cortex and sensory (interoceptive) inputs to the amygdala regulate affective responses. A predisposition of tolerance of the intensity of exercise may explain individual variability in affective responses. The aims of this thesis are to investigate activation in the brain (frontal cortex and amygdala) and tolerance upon affective responses during exercise at different intensities.

Five studies were undertaken to measure activation in the frontal cortex using near infrared spectroscopy (studies I-V), activity in the amygdala using acoustic startle methods (study V) and affective responses using the *Feeling Scale*. Exercise intensities were standardised to gas exchange thresholds (i.e. ventilatory threshold [VT] and respiratory compensation point [RCP]). Tolerance was measured using a questionnaire.

Study I examined frontal cortex activation and affective responses during incremental exercise to exhaustion between groups of individuals identified as having a high or low tolerance. The low group indicated greater activation in the left hemisphere at intensities above VT, whereas the high group indicated the same increase in activation at intensities above RCP. The high group indicated greater activation in the right hemisphere at exhaustion and reported less negative affective responses at intensities above VT. Differential activation between the two groups is presumably due to the cognitive regulation of negative affect. Study II describes activation in sub regions of the frontal cortex and affective responses during incremental exercise to exhaustion. Frontal cortex activation was greater in inferior than superior sub regions and was associated with affective responses above VT. Therefore, studies III and IV examined the influence of a cognitive strategy (imagery) and prior exhaustive activity (to heighten sensory [interoceptive] awareness) respectively, upon frontal cortex activation and affective responses during constant-load exercise at intensities above VT. Study III showed that imagery use influenced frontal cortex activation and maintained more positive affective responses at intensities above VT, but not at RCP. Study IV showed that prior exhaustive activity reduced frontal cortex activation and affective responses at intensities at VT, but

only frontal cortex activation at RCP. Studies III and IV indicate a limited capacity of the frontal cortex at intensities above VT which influence affective responses. Finally, study V examined activation in the frontal cortex and the amygdala during constant-load exercise at intensities below VT, at VT and RCP. Activation in the frontal cortex and amygdala were associated with tolerance and affective responses at intensities above VT highlighting the interplay between cognitive factors and sensory inputs.

The findings indicate *why* we might feel how we feel during exercise. Firstly, differential and asymmetrical activation in the frontal cortex and the amygdala occur during exercise at different intensities. Secondly, tolerance influences frontal cortex activation and is associated with activity in the amygdala. This understanding forms the basis to develop ways to help individuals feel good (or more importantly not feel bad) during exercise to potentially increase exercise participation and adherence.

Declaration

I declare that this thesis presents work carried out by myself and does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any university; to the best of my knowledge it does not contain any materials previously published or written by another person except where due reference is made in the text; and all substantive contributions by others to the work presented, including jointly authored publications, is clearly acknowledged.

Gavin D. Tempest

Acknowledgement

This thesis would not have been possible without the support of those around me.

First and foremost, Associate Professor Gaynor Parfitt and Professor Roger Eston, I would like to thank you for giving me not only the opportunity to embark upon this research journey, but on a journey that has literally taken me to the other side of the world. Your guidance, enthusiasm and encouragement have kept me motivated. Your compassion and support has helped me through this process. For all of those things I am very grateful.

Special thanks to Associate Professor Panteleimon 'Paddy' Ekkekakis, your guidance and support during this project have been invaluable.

To old and new colleagues and friends at the University of Exeter and the University of South Australia, thank you for sharing with me the good times and the bad. It has been a pleasure to work (and play) with you all.

Thank you to all the participants who took part in this research, your time and energy are greatly appreciated.

To my Mam, Dad and family, you have always supported me and let me follow my own path. I am grateful to have you every day.

Finally to Rach, for being by my side. It's just *you*, *me* and *the sea*.

Chapter 1

Introduction

1.1 Rationale

'Exercise is beneficial only if a person engages in it.'

Garber et al., 2011 (p1348)

American College of Sports Medicine Position Stand, 2011

Despite the well-known health benefits of exercise (Penedo & Dahn, 2005) a very high proportion of people continue to lead a sedentary lifestyle. On a global scale, physical inactivity is the fourth leading risk factor for mortality, with levels rising in many countries (World Health Organisation, 2010). In Australia, national physical activity guidelines recommend 30 minutes of moderate-intensity activity on most, preferably, all days (Australian Government, Department of Health and Ageing, 2005). However, based on the 2007-08 National Health Survey for Australia, only 28% of Australians aged 15 years and over were classified as exercising at this level of intensity which is associated with physiological benefit. The large proportion (72%) of people classified as sedentary or having low exercise levels has not changed in over 12 years (Australian Government, Department of Health and Ageing, 2008).

Reasons behind the low levels of public participation in physical activity remain poorly understood. This has led to a large research focus concerned with increasing the initial adoption of physical activity rather than the psychological (and physiological) processes underlying *why* individuals do not participate (Dishman et al., 2006). Therefore, a promising line of inquiry is the idea that physical activity should make individuals feel good to encourage continued participation, or more importantly, not make individuals feel bad and potentially discourage activity. As simple and attractive as this idea appears; it has not been

formally acknowledged in current physical activity recommendations (Haskell et al., 2007; Nelson et al., 2007). Only recently, have guidelines by the American College of Sports Medicine (2013) recognised the relevance of how an individual feels during exercise and the detrimental impact negative affect could have on future exercise participation.

Evidence so far has established a dose-response pattern of the intensity of exercise and the pleasure-displeasure individuals feel (Ekkekakis, Parfitt & Petruzzello, 2011). Furthermore, how an individual feels during exercise is linked to future exercise participation (Schneider, Dunn & Cooper, 2009; Williams et al., 2008, Williams, Dunsiger, Jennings & Marcus, 2012). However, the underlying neural mechanisms of why individuals feel how they feel during exercise have not been confirmed. A relatively new theoretical framework which explains the dose-response pattern of affective responses (pleasure-displeasure) during exercise is the dual mode model (DMM; Ekkekakis, 2003; Ekkekakis & Acevedo, 2006). According to the DMM, the pleasure-displeasure an individual may feel during exercise is regulated between the frontal cortex (involved in appraisal processes) and subcortical areas of the brain (which receive sensory input from the body). Grounded within the DMM is the suggestion of how an individual is able to maintain activation of the frontal cortex to override aversive states driven by sensory (i.e. interoceptive) input from the body during exercise at different intensities. Competition between the frontal cortex and sensory input from the body influences an individual's perception of exercise (i.e. how they feel and how hard they feel they are working). An individual's perception of exercise is hypothesised to influence their decision to continue or stop the activity (Acevedo, Gill, Goldfarb & Boyer, 1996). This is described as tolerance of the intensity of exercise and is a predisposed trait (Ekkekakis, Hall & Petruzzello, 2005a).

Until recently, what happens in the brain during exercise, for example the neural basis of how individuals may feel, has been difficult to investigate (Ekkekakis, 2009) and has consequently remained largely overlooked (Booth, Chakravarthy, Gordon & Spangenburg, 2002). An examination of brain areas during exercise and the factors that influence them (e.g. tolerance of the intensity), will help to determine why individuals feel the way they do during exercise. This understanding will help develop ways to encourage individuals to feel good, or more importantly not feel bad, and potentially increase physical activity participation and adherence.

Therefore, the primary aim of this thesis was to investigate activation in the brain (frontal cortex and amygdala) and affective (pleasure-displeasure) responses during exercise at different intensities. The secondary aim was to examine the influence of the predisposed trait of tolerance of the intensity of exercise upon activation in the brain and affective responses. The hypotheses for five experimental studies were generated essentially from the DMM and its proposed neural basis (i.e. the DMM) and mechanisms of affective responses in current literature.

1.2 Overview of the thesis

Chapter 2 provides a review of the literature of the neural basis of affective responses during exercise. Affective responses to exercise are defined and the development of a theoretical framework to explain the dose-response pattern of these responses is described. Neural anatomy and the proposed neural mechanisms of affective responses are identified. Tolerance of the intensity of exercise to explain individual differences in affective responses is discussed. Finally, methods used to investigate neural systems during exercise are presented.

Chapter 3 describes the self-report measures of affective responses and tolerance of the intensity of exercise. The theory and application of dual (studies I, III and V) and multi-channel (studies II and IV) near infrared spectroscopy methods used to measure activation in the frontal cortex are explained. The identification of individual gas exchange thresholds (i.e. ventilatory threshold [VT] and respiratory compensation point [RCP]) and the determination of the corresponding exercise intensities are described. Assessment and exercise protocols, data reduction, power analyses and statistical procedures are identified.

Chapter 4: Study I examines activation in the frontal cortex and affective responses during incremental exercise to exhaustion between groups of individuals identified as having high or low self-reported tolerance of the intensity of exercise. Cerebral activation and affective responses are compared between groups at intensities of exercise corresponding to 80% of VT (below VT), VT, RCP and end of exercise (exhaustion).

Chapter 5: Study II describes activation in sub regions of the frontal cortex and affective responses during incremental exercise to exhaustion at intensities of exercise corresponding to below VT, VT, RCP and the end of exercise (exhaustion).

Chapters 6 and 7: Studies III and IV examine the influence of a cognitive strategy (imagery) and prior exhaustive activity (to induce a state of heightened sensory [interoceptive] awareness) respectively, upon activation in the frontal cortex and affective responses during constant-load exercise at intensities above VT.

Chapter 8: Study V examines activation in the frontal cortex, activity of the amygdala and affective responses during exercise at intensities below VT, VT and RCP. The influence of tolerance of the intensity of exercise is examined.

Chapter 9 is a general discussion of the implications of the findings and directions for future research, concluding with a final summary of the contributions of the present studies.

Chapter 2

The neural basis of affective responses to the intensity of exercise: background, theory, mechanisms and measurement

This chapter will review and discuss current literature in the following key areas:

I. Affective responses and the intensity of exercise; the dual-mode model,

II. Neural basis of affective responses,

III. Mechanisms of affective responses,

IV. Cognitive versus sensory (interoceptive) processes,

V. Methods to investigate neural systems during exercise.

2.1 Exercise-induced affective responses

How a person feels during exercise is important in the decisions an individual may make to continue or terminate their behaviour (Hardy & Rejeski, 1989). In fact, 'affective states and the cognitive appraisal of exertion during exercise may determine whether or not an individual will persist at an activity' (p.286; Acevedo et al., 1996). Therefore, affect is considered a basis for 'decision-making' (Batson, Shaw & Oleson, 1992; Damasio, 1994; Kahneman, 1999) and has potentially important implications for ways to increase participation and adherence to physical activity.

Recent research has shown that affective responses (pleasure-displeasure) during exercise are important for future exercise participation and adherence (Schneider et al., 2009; Williams et al., 2008; Williams et al., 2012). However, the underlying neural basis of exercise induced affective responses has not been examined. This understanding would help to develop ways to promote pleasure (or importantly to avoid displeasure) and potentially increase exercise participation and adherence.

2.1.1 Definition

Affective responses are described as feelings along a continuum ranging from pleasure to displeasure, which are positive and negative constructs underlying core affect (Russell, 1980). Affect has been described as an evolutionarily adaptive response; in particular displeasure or negative affect is proposed to promote survival and maintain homeostasis in the presence of threat or danger (Damasio, 1994; Nesse, 1990, 1998). The pleasure-displeasure component of core affect has received the most attention in studies examining affective states during exercise (for reviews see Ekkekakis & Petruzzello, 1999; Ekkekakis et al., 2011). In this thesis, affective responses (positive or negative) will refer to the pleasure or displeasure individuals report during an acute bout of exercise.

2.1.2 Exercise intensity-affect relationship

Over a decade ago the relationship between affective responses during exercise, particularly at different exercise intensities was unclear, and findings were confounded by methodological limitations and inconsistencies as highlighted in a review by Ekkekakis and Petruzzello (1999). The authors addressed a number of methodological issues which prevented the comparison and replication of findings towards a dose-response relationship

between the intensity of exercise and affective responses (see also Ekkekakis & Petruzzello, 2000, 2002).

Firstly, the categorical nature of affect or rather *what* was and should be measured had not been determined (Gauvin & Brawley, 1993). Through much debate within the literature (see Russell & Feldman Barrett, 1999), a dimensional approach to the measurement of affect has been accepted to encompass a broad canvas of the construct, namely the circumplex model of affect (Larsen & Diener, 1992; Russell, 1980). The circumplex model provides a two-dimensional domain-general map of affective states with two orthogonal bipolar axes; valence (pleasure-displeasure) and perceived activation (high-low; as shown in Figure 2.1).

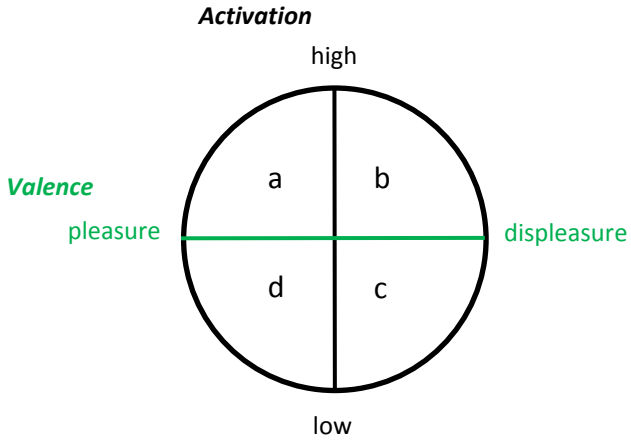


Figure 2.1. The circumplex model of affect (Larsen & Diener, 1992; Russell, 1980). Affective space (the circle) is divided into four quadrants by two intersecting dimensions (valence and activation). The four quadrants relate to a) high activation and pleasure, feelings of energy and excitability; b) high activation and displeasure, feelings of distress and tension; c) low activation displeasure, feelings of tiredness and boredom; and d) low activation and pleasure, feelings of calm, relaxation. The valence axis (shown in green) has received the most attention in exercise-affect literature.

The model offers a broad global affective space to measure variation in the dimensions (valence and activation) and intensity (along a continuum) of affective states and has a strong theoretical basis (Ekkekakis & Petruzzello, 2000, 2002). The circumplex model is argued to represent core affect, and not more complex feeling states or emotions (Russell

& Feldman Barrett, 1999). The valence (pleasure-displeasure) dimension has since become the most studied within the exercise-affect literature (Ekekkakis et al., 2011).

Secondly, the use of pre-post-test designs did not consider changes in affective responses during exercise. Negative affective responses reported during acute exercise have been shown to become more positive post-exercise (Bixby, Spalding & Hatfield, 2001; Hall, Ekkekakis & Petruzzello, 2002) indicating a rebound effect from negative responses during exercise (Bixby et al., 2001). Therefore, pre-post-test designs did not accurately represent the trajectory of affective responses to a bout of exercise. Consequently, measures of affective responses are now commonly taken during exercise to record the trajectory of affective responses over the full course of the exercise duration (Ekkekakis et al., 2011).

Finally, the intensity of exercise was not standardised between individuals within and between studies, which meant individual differences in physiological responses (i.e. metabolic state) were not accounted for. Early studies commonly defined exercise intensities as a percentage of maximal power (e.g. 50% of maximal oxygen uptake or 60% of maximal heart rate), based on the assumption that the activity stimulus would be the same for everyone. However, metabolic processes vary considerably between individuals, even between those with the same maximal oxygen uptake (Boutcher, 1990). Therefore, intensities are now most often selected based on transition markers such as thresholds of lactate or gas exchange variables to provide a fixed metabolic profile to establish comparable individual differences (Wells, Balke & Van Fossan, 1957). Ekkekakis and Petruzzello (1999) proposed a three-domain typology of intensity consisting of a moderate, heavy and severe domain (identified by Gaesser & Poole, 1996; see Figure 2.2).

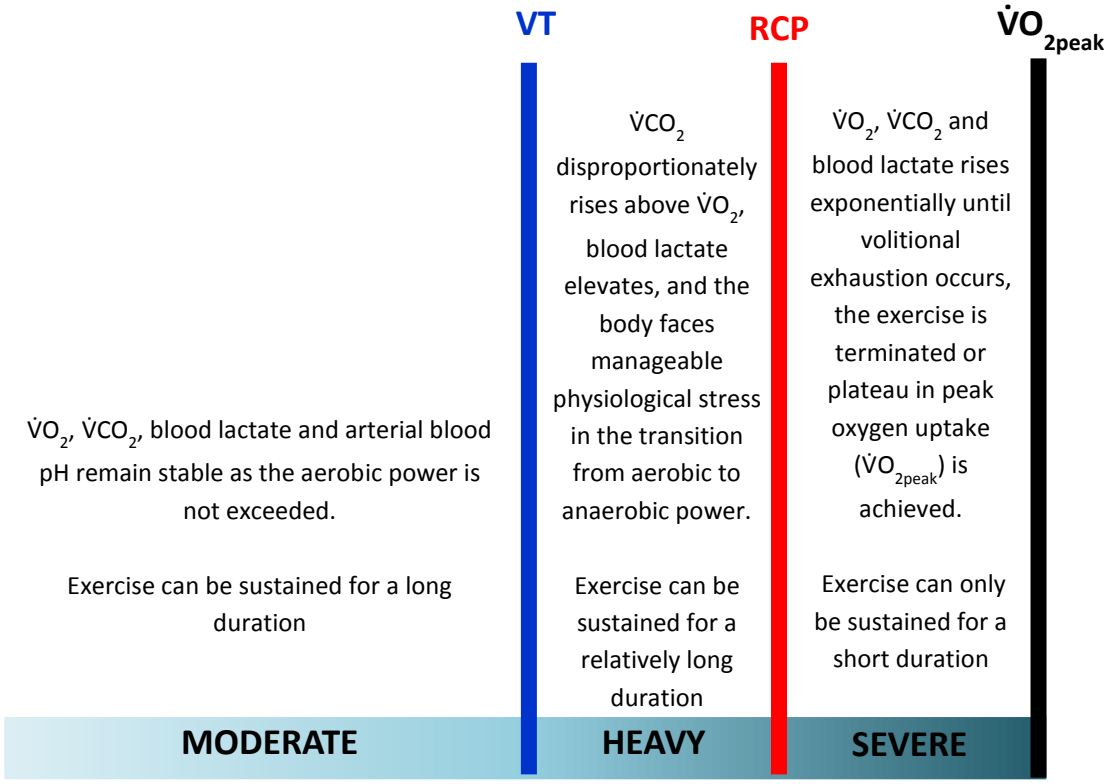


Figure 2.2. Exercise intensity domains (moderate, heavy and severe) identified by Gaesser and Poole (1996). The intensities of exercise are delineated by gas exchanges thresholds; ventilatory threshold (VT) and respiratory compensation threshold (RCP) up to peak oxygen uptake ($\dot{V}O_{2peak}$). The physiological (oxygen uptake [$\dot{V}O_2$] and carbon dioxide output [$\dot{V}CO_2$]) processes associated with each intensity domain are indicated.

The intensity domains are defined in relation to thresholds of respiratory gas exchange; the ventilatory threshold (VT) and respiratory compensation point (RCP). Exercise below the VT poses little challenge to homeostasis, the regulation of the internal state. However, above VT the physiological steady-state becomes difficult to maintain. A representation of the respiratory response during exercise is provided in Figure 2.3.

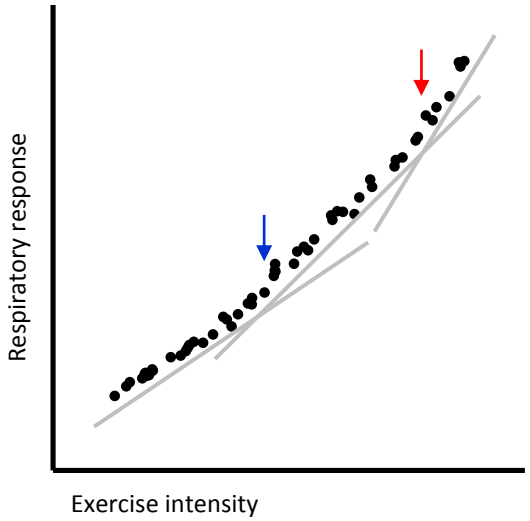


Figure 2.3. A representation of the respiratory response (pulmonary oxygen uptake) across intensities of exercise. The first rise indicates the ventilatory threshold (VT [blue arrow]) and the second exponential rise to exhaustion (peak oxygen uptake) indicates the respiratory compensation point (RCP [red arrow]).

The relationship between affective responses and the intensity of exercise has recently been revisited. Following the recommendations in the 1999 review, Ekkekakis et al. (2011) were successful in providing evidence towards a dose-response relationship of the intensity of exercise and affective responses. The authors reviewed 33 studies from 1999 to 2009 and established that (a) physical activity performed at intensities substantially below VT induce positive self-reported affective responses (pleasure) among most individuals (Ekkekakis, Hall & Petruzzello, 2005b; Ekkekakis, Hall, Van Landuyt & Petruzzello, 2000); (b) physical activity performed at an intensity substantially above VT (i.e. proximal to RCP) induce negative affective responses (displeasure) in nearly all individuals (Ekkekakis, Hall & Petruzzello, 2004, Ekkekakis, Hall & Petruzzello, 2008; Parfitt, Rose & Burgess, 2006; Rose & Parfitt, 2007); and (c) physical activity at intensities around VT induces marked inter-individual variability, with some individuals reporting more positive affective and others negative affective responses (Ekkekakis et al., 2005b, 2008; Parfitt et al., 2006; Rose & Parfitt, 2007; Sheppard & Parfitt, 2008). A representation of the pattern of affective responses across exercise intensity domains is provided in Figure 2.4.

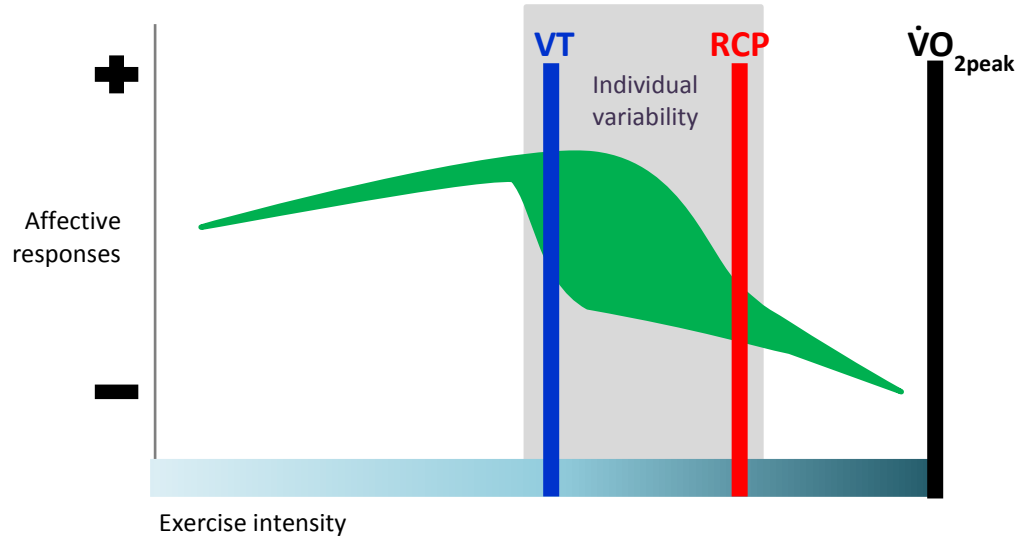


Figure 2.4. A representation of the pattern of affective responses to the intensity of exercise. The intensity domains moderate, heavy and severe are distinguished by respiratory gas exchange thresholds; the ventilatory threshold (VT) and the respiratory compensation point (RCP) up to peak oxygen uptake ($\dot{V}O_{2peak}$). Individual variability in affective responses (shaded area) at intensities around and above the VT, proximal to the RCP is highlighted.

The emergence of the dose-response relationship between affective responses and the intensity of exercise is due not only to resolution of the methodological limitations outlined above, but also to the development of a new theoretical framework, namely the dual-mode model (DMM; Ekkekakis, 2003; Ekkekakis & Acevedo, 2006). The DMM explains the dose-response relationship which has received a large body of support for over a decade (Ekkekakis et al. 2011). In addition, the DMM provides a putative neural basis of affective responses to exercise based on neuroscientific data (see Ekkekakis & Acevedo, 2006). The model is an evolutionarily adaptive approach and describes the interaction between psychological and physiological processes in the brain which influence how individuals feel (pleasure-displeasure) during exercise. The DMM is the only framework which has been developed to account for a neural basis of affective responses to exercise at different intensities; however tenets of this neural basis remain to be examined.

2.1.3 The dual-mode model (DMM)

The DMM was conceived from the notion that affective responses should be measured during exercise, the intensity of exercise should be standardised to a fixed metabolic profile

and that individual variability of affective responses (around and above VT) could be accounted for (Ekkekakis, 2003). The DMM proposes that affective responses are determined by the interplay between cognitive processes (i.e. appraisal processes, predisposed traits of sensory modulation, attentional techniques) and sensory inputs from the body (i.e. interoceptive cues).

Cognitive processes and sensory (interoceptive) input from the body are defined as modes (Ekkekakis, 2003). These modes are theorised in the DMM to reflect two types of inputs to the amygdala; a subcortical structure located within the anterior portion of the medial temporal lobes of the brain. The amygdala is associated with emotional reactions; particularly negative (aversive) states (see Armony, 2013). Of the two main types of input to the amygdala, one is from the frontal cortex and the other subcortical areas of the brain which relay sensory input from the body. The dual-modes are shown in Figure 2.5.

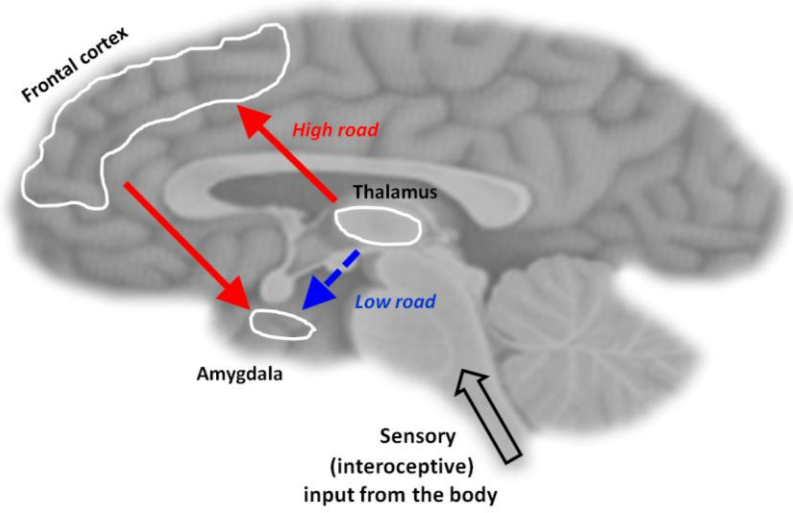


Figure 2.5. The proposed dual-modes of input to the amygdala. The DMM proposes that sensory (interoceptive) input from the body reaches the amygdala via two pathways (or modes) from the thalamus; the *high* road and the *low road* (LeDoux, 1996; see 2.2.1). The *high* road describes how stimuli reach the amygdala indirectly via the frontal cortex (i.e. thalamo-cortico-amygdala pathway). Stimuli along the *high road* undergo appraisal or cognitive processing. The *low* road describes stimuli that reach the amygdala directly (via the thalamo-amygdala pathway). Stimuli along the *low road* undergo no appraisal or cognitive processing.

The interaction between the two modes of input to the amygdala change as the intensity of exercise is increased. At intensities below VT, the frontal cortex has dominance over the amygdala. However, at intensities above VT, sensory input from the body is increased due to physiological adaptations in the body associated with the increased intensity of exercise.

It is proposed that the frontal cortex has to work harder in order to compete with sensory input from the body to maintain control over the amygdala. Once a physiological steady-state can no longer be maintained (at intensities above VT and RCP), the frontal cortex cannot compete with the intense sensory input to the amygdala as homeostasis becomes challenged.

Therefore, the DMM explains that affective responses should be mostly positive when the intensity of physical activity does not challenge homeostasis (i.e. at intensities below and proximal to VT). It is at these intensities that frontal cortex activation has uncontested dominance over the amygdala as sensory input from the body is manageable. However, affective responses become mostly negative when the intensity of exercise prevents the maintenance of a physiological steady state (i.e. at and above RCP). It is at these intensities that sensory input from the body is relayed through subcortical homeostatic afferents to the amygdala and the frontal cortex has less control over the amygdala.

At intensities around and above VT, affective responses can be variable; some individuals report positive affective responses (pleasure) and others report a decline or negative affective response (displeasure), when sensory (interoceptive) inputs begin to rise exponentially. The DMM proposes that the competition between the frontal cortex and the intensified sensory (interoceptive) input from the body to the amygdala drives these affective responses. Therefore, the amygdala and the dual-modes of input from subcortical (i.e. sensory processes) and cortical (frontal cortex) areas of the brain are a proposed neural basis of affective responses. The anatomy and function of these areas are outlined (see 2.2) and the possible mechanisms by which these areas may influence affective responses are discussed (see 2.3).

2.2 Neural basis of affective responses

The DMM provides a framework of the neural basis of affective responses in which the amygdala is central to the model. There are two modes of input to the amygdala, one from subcortical areas of the brain which receive homeostatic afferents from the body, and another from areas of the frontal cortex related to cognitive processes (for a full review see Ekkekakis & Acevedo, 2006). The neural structures associated with the two modes are briefly discussed below and are shown in Figure 2.6.

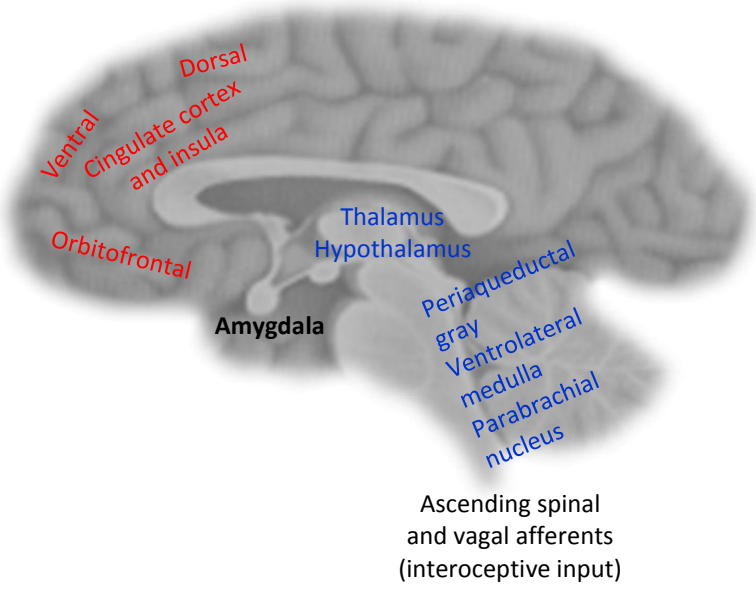


Figure 2.6. A representation of the neural structures proposed to be involved in the dual-modes of inputs to the amygdala (Ekkekakis & Acevedo, 2006). The subcortical structures in blue are primarily involved in the sensory (interoceptive) mode and the structures in red are primarily involved in the cognitive mode of input to the amygdala.

2.2.1 Amygdalaloid complex

The neural basis of the DMM is supported by evidence examining fear and anxiety of which the amygdala plays a significant role (LeDoux, 1996). The amygdala is part of an evolutionarily defined 'survival circuit' which regulates emotional feelings in order to reach a specific goal, provide motivation, or process arousing stimuli, important for normal physiological functioning (LeDoux, 1996, 2012).

The amygdala and the thalamus (a subcortical structure involved in relaying sensory input from the body to other parts of the brain) detect impending harm or danger and prevent disruption to homeostasis. Intense sensory input or stress would cause the amygdala to enter a state of hyper vigilance in the anticipation of a threat (typically an aversive state), which would require an action (i.e. fight or flight response; LeDoux, 1996). The amygdala shows automaticity to incoming sensory input and attaches affective significance which occur independent of attention (Dolan & Vuilleumier, 2003). If the amygdala was allowed to remain in a state of hyper vigilance, the central nervous system would become preoccupied and overloaded with affective distractions (predominantly fearful, aversive or anxious states; LeDoux, 1996). Over activity of the amygdala is associated with anxiety (Davidson, 2002; Tye et al., 2011) and post-traumatic stress disorders (Pitman, Shalev & Orr, 2000). Inhibition from cortical areas (i.e. the frontal cortex) provides control over the amygdala so it can execute and focus relevant behaviours, tasks and general functioning without interference (Botvinick, Braver, Barch, Carter & Cohen, 2001).

LeDoux (1986, 1996) identified two distinct input pathways (hence the dual-modes of the DMM) from the thalamus to the amygdala (see Figure 2.5.). Sensory stimuli (input from the body) are predominantly processed through an indirect thalamo-cortical-amygdala pathway (which LeDoux deemed the 'high road'). Stimuli via the 'high road' undergo complex cognitive interpretation and evaluation in the frontal cortex. The frontal cortex has the ability to override the response of the amygdala. However, as an evolutionarily adaptive system, potentially dangerous (intense) stimuli (which may threaten homeostasis) are processed quickly through a direct thalamo-amygdala pathway (which LeDoux deemed the 'low road') as immediate action may be required.

In the case of exercise, at intensities below VT, sensory information from the body is not perceived as threatening; therefore the main input to the amygdala is via the 'high road'. At intensities of exercise above VT, sensory information from the body is intensified which may be perceived as threatening and have the potential to disrupt the body's internal state. Therefore, exercise at intensities above VT would trigger an aversive response via the 'low road'. It is for this reason that the amygdala is a central structure to the DMM. The ability of the frontal cortex to maintain control of the amygdala through the thalamo-cortical-amygdala pathway is proposed to influence affective responses (Ekkekakis, 2003; Ekkekakis & Acevedo, 2006).

2.2.2 Sensory (interoceptive) pathways to the brain

Interoceptive factors refer to internal stimuli (sensory input from the body) such as respiratory or muscular cues, whereas exteroceptive factors refer to external stimuli (input from outside of the body) such as social and environmental cues. Sensory inputs from the body or interoceptive cues feed information from the peripheral to the central nervous system. This allows the physiological integrity of the body to be maintained through adaptations of the nervous system (Craig, 2002). Sensory input travels along discrete pathways from the peripheral nervous system to spinal regions which terminate in supra-spinal sites in cortical and subcortical areas in the brain (Craig, 1996). The neural pathways allow the integration of a number of sensory modalities from the periphery (homeostatic afferents) to enter the central nervous system (for a detailed account see Craig, 1996, 2005).

Primary afferent fibres activate somato-autonomic reflexes (musculoskeletal stimulation) at spinal and supra-spinal levels from lamina I neurons; the first site of the central nervous system to receive afferent signals from the peripheral nervous system. The first area in the brain to receive supra-spinal input from lamina I neurons is the nucleus of the solitary tract; a bundle of sensory fibres located within the medulla. Parasympathetic (associated with maintaining rest) small diameter fibres topographically terminate in the nucleus of the solitary tract which provides visceral (bodily) sensory information via cranial nerve branches. Baro- and mechanosensory receptors (i.e. sensitive to touch, pressure, tension) relay information along ascending afferent fibres which terminate within the lateral nucleus of the solitary tract. Parasympathetic nerve afferents converge with sympathetic (associated with initiating action) incoming lamina I input in the nucleus of the solitary tract, with further projections to the reticular formation and the ventrolateral medulla (Craig, 1996).

Within the Pons, the parabrachial nucleus extends anteriorly from the nucleus of the solitary tract and is the main integrative site for lamina I afferent projections. Particularly, lamina I input of noxious (pain), thermal (temperature) and metaboreceptive (chemical) activity of the entire body are integrated within the parabrachial nucleus (Craig, 1996). The parabrachial nucleus relays information to the hypothalamus and amygdala which is an important pathway providing the link between sensory input from the body and affect, motivation and emotional memory (Bernard, Bester & Besson, 1996).

Sensory input termination sites in the pons also include the ventrolateral and dorsolateral tegmentum and locus coerulus. These areas are essential for the maintenance of cardiovascular, respiratory, energy and fluid activity through connections to forebrain limbic sites such as the thalamus, hypothalamus and amygdala, and to the periaqueductal gray in the midbrain (Craig, 1996).

The nucleus of the solitary tract, ventrolateral medulla and parabrachial nucleus are the major termination sites for convergence of spinal and vagal (cranial nerves) afferent fibres. Within the forebrain spinal afferent fibres make up an extension of the vagal fibres to the thalamus and cortex (Saper, 2000). The inputs arise from sensory fibres which relay information regarding nociceptive tissue integrity and mechanosensory input from spinal afferent fibres via lamina I dorsal pathways (Craig, 1996).

Sensory input through these pathways determines action by the autonomic nervous system which consists of two-opposing systems or branches. The parasympathetic branch is involved in the management of nourishment, safety, positive affect and approach-related (appetitive) behaviours. The sympathetic branch is involved in the management of arousal, danger, negative affect and withdrawal-related (aversive) behaviours. The dynamics of these two-opposing branches of the autonomic nervous system maintains balance and equilibrium of the bodily state (i.e. homeostasis). Afferent interoceptive input to the autonomic nervous system provides the most basic drive for motivational behaviours such as hunger, thirst, comfort or discomfort and the components of core affective responses (see Craig, 1996, 2006; Critchley, 2005).

The sensory (interoceptive) mode of input to the amygdala is described by the DMM to influence affective responses at exercise intensities around and above VT. Affective responses are driven by increased activity in the amygdala which produces an aversive state through the discrete pathways outlined above. It is proposed that higher order structures (i.e. the frontal cortex) have to compete to override the response from areas of the brain (beyond the preservation of life) to consciously perceive and determine behaviour.

2.2.3 Prefrontal cortex

The prefrontal cortex is involved in working memory, executive function and decision-making processes, which are highly connected to areas of the brain associated with feeling

states (Miller & Cohen, 2001). The approximate locations of sub regions of the frontal cortex according to the map of Brodmann's areas (BA) are provided in Figure 2.7. Regions of the prefrontal cortex are primarily divided into three parts; the dorsolateral, ventromedial and orbitofrontal cortices. These parts will be briefly defined and their main connections outlined with relevance to their potential role in the regulation of affective responses.

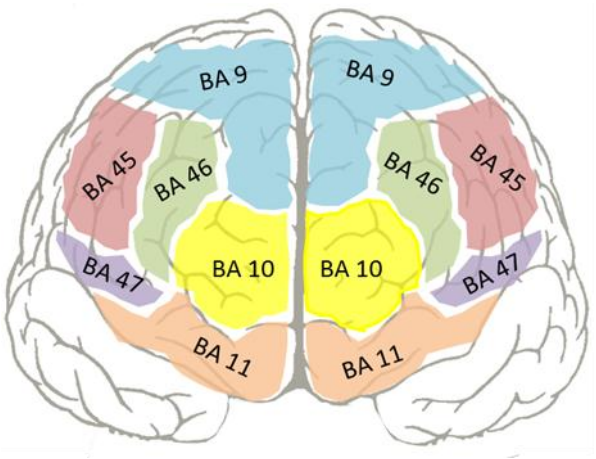


Figure 2.7. A stylised representation of the approximate location of cortical regions according to Brodmann's areas (BA).

2.2.3.1 Dorsolateral frontal cortex

The mid-dorsal and lateral PFC (BA 9 and 46) are highly connected with sensory and motor systems and are involved in the control of behaviour (Miller & Cohen, 2001). The dorsolateral area functions to maintain the contents of working memory to aid goal-directed behaviours (Petrides, 2005). The dorsolateral cortex is involved in effortful (conscious) and attentional processing, executive function and task-switching abilities (Duncan & Owen, 2000; Wager, Jonides & Reading, 2004).

2.2.3.2 Ventromedial frontal cortex

The ventromedial (located on the medial surface of the frontal cortex not shown in Figure 2.7) and frontopolar areas (BA 10) are primarily involved in top-down inhibitory processes of the amygdala in the regulation of mood and affective states (Myers-Schulz & Koenigs, 2012) via direct connections (Rempel-Clower & Barbas, 1998). The ventromedial cortex is

also involved in rational thinking and decision-making processes (Damasio, 1994), and is therefore important in the link between emotions and behaviours (Saper, 2002). The ventrolateral area (BA 45 and 47) serves to maintain task-relevant information (Petrides, 2005) and supress task-irrelevant information during cognitive control (Cohen, Dunbar & McClelland, 1990).

2.2.3.3 Orbitofrontal Cortex

The orbitofrontal cortex (BA 11) is a highly active region in affect representation (Kringelbach, 2005; Kringelbach, O'Docherty, Rolls & Andrews, 2003). The amygdala, orbitofrontal cortex and the cingulate gyrus (collectively named the rostral limbic system), is a proposed affective loop (Thayer & Lane, 2000). The orbitofrontal cortex is a multi-modal associative learning centre of positive and negative reinforcers (stimuli which will either increase or decrease a behaviour; Rolls, 1999, 2004), and is correlated with the subjective experience of affective valence (Kringelbach et al., 2003). Highly processed sensory and cognitive inputs from other areas of the prefrontal cortex are indirectly relayed to the orbitofrontal cortex via the anterior cingulate (Van Hoesen, Morecraft & Vogt, 1993). The orbitofrontal cortex has direct and indirect (via the anterior cingulate cortex) connections with the amygdala (Amaral & Price, 1984; Carmichael & Price, 1995). Evidence suggests anterior and posterior portions of the orbitofrontal cortex are associated with the representation of affect (Rempel-Clower & Barbas, 1998). Anterior areas are involved in complex sensory processing and consciousness due to their close proximity to higher cortical structures. Posterior areas are involved in the basic processing of visceral (bodily sensations) input due to heavy connections to the posterior hypothalamus and its close proximity to subcortical areas.

2.2.4 Anterior cingulate and Insula

Located on the medial surface of the prefrontal cortex overlaying the corpus callosum are the cingulate cortices and insulae. Both the anterior cingulate cortices and insulae are involved in autonomic function representation (i.e. heart rate, breathing) which forms the basis of subjective emotional awareness. The cingulate cortex has direct connections to the amygdala (Amaral & Price, 1984; Carmichael & Price, 1995) and is associated with attentional (Posner, 1995) and affective pain processing (Beckmann, Johansen-Berg & Rushworth, 2009). The insulae are highly connected to the cingulate (Beckmann et al.,

2009), the central nucleus of the amygdala (Reynolds & Zahm, 2005), and orbitofrontal and dorsolateral prefrontal areas (Ongur & Price, 2000). The insula receives afferent input and projects efferent signals in the regulation of autonomic responses. The role of the insula is the representation of the experience of bodily emotions, especially related to visceral afference (Cechetto & Saper, 1990), and autonomic (cardiovascular) arousal (Critchley, 2005; Critchley, Corfield, Chandler, Mathias & Dolan 2000; Williamson et al., 1997; Williamson et al., 1999).

The connections and role of the neuroanatomical areas identified thus far are certainly by no means definitive or exhaustive. It is difficult to determine discreet functions and roles of specific areas of the frontal cortex, or any (both cortical and subcortical) areas of the brain due to the complexity and highly integrated nature of the structures. However, meta-analyses of imaging data are starting to convincingly match anatomy and function, particularly between areas of the frontal cortex and their role in affective and emotional processing (see Barrett & Wager, 2006; Ochsner, Silvers & Buhle, 2012; Phan, Wager, Taylor & Liberzon, 2002; Wager, Phan, Liberzon & Taylor, 2003).

2.3 Mechanisms of affective responses

The DMM provides a detailed theoretical framework of how affective responses are proposed to be regulated at different intensities of exercise: specifically by the frontal cortex and the amygdala. The main proposal is that the frontal cortex must maintain control of the amygdala through the cognitive mode (via the 'high road') in order to retain a positive affective (i.e. pleasurable) state, and to avoid a negative (i.e. aversive) affective state. Therefore individual variability in affective responses is presumed to be dependent on maintenance of frontal cortex activation through cognitive control processes (see 2.3.1). These processes may be influenced by theories of frontal cortex asymmetry (see 2.3.2) associated with approach-withdrawal motivation. Furthermore, a proposed deregulation of frontal cortex function may be a prevailing mechanism for affect regulation (see 2.3.3).

2.3.1 Cognitive control processes

A significant role of the prefrontal cortex is to provide cognitive control which can be defined as 'the ability to coordinate thoughts and actions in relation with internal goals'

(p1181; Koechlin, Ody & Kouneiher, 2003). Cognitive control mechanisms explain an individual's ability to facilitate or inhibit emotional stimuli at a neural level which may influence behavioural responses (Ochsner & Gross, 2005). Two main outcomes of affective and emotional regulation are (1) inhibition or suppression of stimuli through attentional processes which serve to focus towards or distract away from stimuli and limit the expressive action, and (2) cognitive reappraisal (defined by Lazarus, 1991) which involves manipulating the meaning of the stimuli in relation to current goals which neutralise negative experiences and therefore influence the on-going emotional response (Ochsner & Gross, 2005).

Evidence from studies examining cognitive control processes (e.g. inhibition, reappraisal) have indicated top-down regulation of subcortical structures (i.e. the amygdala) in bilateral areas of the frontal cortex (Ochsner & Gross, 2005, 2007). Neuroimaging studies have shown that activity in areas of the frontal cortex induced by attempts of active cognitive coping with a negative affective stimulus, are associated with reduced activity in the amygdala (Banks, Eddy, Angstadt, Nathan & Phan, 2007; Goldin, McRae, Ramel & Gross, 2008; Kim & Hamann, 2007; Ochsner, Bunge, Gross & Gabrieli, 2002; Ochsner & Gross, 2008; Phan et al., 2005; Schaefer et al., 2002). This work has shown greater activity in left-lateralised areas of the frontal cortex in the presence of positive stimuli (Kim & Hamann, 2007; Ochsner & Gross, 2004), and greater activity in right-lateralised areas of the frontal cortex in the presence of negative stimuli (Ochsner & Gross, 2004) during cognitive control. This is in line with a meta-analysis of neuroimaging studies which indicated region-specific activation of emotional processes in these and other areas of the brain (Wager et al., 2003). However, these findings were only observed when structures were compared within hemisphere, highlighting the complexity of lateralised emotional processes (Wager et al., 2003).

Theories of emotional regulation have particularly focused on the prefrontal cortex, anterior cingulate and insular cortices for their role in the modulation and control of emotional responses (Bush et al., 2000; Ochsner & Gross, 2005). During cognitive control tasks, activation in dorsolateral and ventromedial frontal areas has been shown to increase during reappraisal and is associated with reduced self-reported negative affect (Ochsner et al., 2002). Activation in the right dorsolateral areas of the frontal cortex is increased in attempts to inhibit (Beauregard, Levesque & Bourgouin, 2001) and suppress (Duncan &

Owen 2000; Miller & Cohen, 2001) emotional responses. In response to emotional judgements made during fMRI, individuals with depression indicate greater activity in the right dorsolateral compared to the left dorsolateral areas of the frontal cortex. However, greater activity in the right dorsolateral area was only present in expectation of the emotional stimuli, indicating the role of attentional modulation (Grimm et al., 2008).

These findings indicate that activation in the frontal cortex is associated with reduced activity in subcortical areas of the brain (i.e. the amygdala) through cognitive control processes. The DMM proposes that these processes are dominant during exercise at intensities whereby sensory input from the body is manageable (at intensities proximal to VT). However, at higher intensities of exercise (above VT) the frontal cortex must work harder (i.e. increase cognitive control) to supress activity in the amygdala (driven by increased sensory inputs from the body).

Cognitive control processes indicate lateralised activation in the frontal cortex during affective processing; activity in left lateralised areas is associated with positive stimuli and activity in right lateralised areas with negative stimuli. This fontal cortex asymmetry has been linked to the approach-withdrawal motivational system.

2.3.2 Frontal cortex asymmetry

Functional asymmetry of the frontal cortex in affective processing and its association with approach-withdrawal motivation and behaviours has been a highly studied area (for a full review see Alves, Fukusima & Aznar-Casanova, 2008). One of the most prominent theories is the valence asymmetry hypothesis (Davidson, 1993, 2004). This theory suggests that the left hemisphere is associated with processing positive affective stimuli, and the right hemisphere is involved in processing negative affective stimuli. Affective valence is related to two fundamental motivational systems; approach and withdrawal. The approach system is associated with positive affect, which drives goal-orientation and behaviours towards something (Lazarus, 1991). In opposition, the withdrawal system is associated with negative affect and aversion, which therefore drives behaviours away from something. The motivational system is proposed to influence an individual's dispositional trait affect, described as *affective style* (Davidson & Irwin, 1999). The approach-withdrawal motivation constructs reflecting functional asymmetry are identified as underlying behavioural inhibition and activation systems (Coan & Allen, 2003; Sutton & Davidson, 1997).

Studies have provided neurophysiological evidence of increased left anterior activation and approach-related positive affect and increased right anterior activation and withdrawal-related negative affect within the frontal cortex using EEG electroencephalography (EEG) (Davidson, Ekman, Saron, Senulis & Freisen, 1990; Davidson, Schaffer & Saron, 1985; Harmon-Jones, 2003), functional magnetic resonance imaging (fMRI; Canli, Desmond, Zhao, Glover & Gabrieli, 1998) and near infrared spectroscopy (NIRS; Tuscan et al., 2013). In exercise studies, an association between frontal cortex asymmetry and affective responses reported due to exercise has been shown (Petruzzello, Ekkekakis & Hall, 2006; Petruzzello, Hall & Ekkekakis, 2001; Petruzzello & Tate, 1997).

Conversely, some evidence failed to support the lateralisation of affective processing (see Hagemann et al., 1998). Such conflicts were attributed to methodological differences in study designs, analytical procedures and whether automatic or voluntary regulatory processes are examined (Davidson & Irwin, 1999). Individual differences, presumably due to cognitive control processes and 'affective style' is likely to create variability in findings of frontal cortex asymmetry and associated affective states (Jackson et al., 2003). However, more recent evidence suggests that motivational constructs, rather than affective stimuli, primarily relate to frontal asymmetry (Berkman & Lieberman, 2010; Harmon-Jones, 2004). Despite these recent advances, a large body of converging evidence indicates support for the association between motivational and affective constructs and frontal cortex asymmetry.

From a neuroanatomical perspective Craig (2005) describes a model of emotional asymmetry with similar affective-motivational components as the valence asymmetry hypothesis. Craig proposes that the management of bodily processes have an organised autonomic opponancy in the forebrain which link to the discrete pathways providing sensory input to the brain from the body (see 2.2.2). Similar to Davidson's valence asymmetry hypothesis (1993), but with a defined anatomical basis, Craig indicates that the left forebrain is associated with positive affect and approach (appetitive) behaviours directed by parasympathetic activity related to nourishment and safety. The right forebrain is associated with negative affect and withdrawal (aversive) behaviours directed by sympathetic activity related to arousal and danger. During homeostatic regulation, the left forebrain is involved in energy enrichment and the right forebrain energy expenditure. Specifically the left and right insula and anterior cingulate cortices are the main

representative sites of homeostatic emotions such as affect, which Craig suggests are interoceptively driven and their purpose is to influence motivations and behaviours. This theory is potentially important in understanding affective responses during exercise due to the defined anatomical basis of how sensory input from the body may influence affective states.

2.3.3 Frontal cortex deregulation

The DMM proposes that the competition between cognitive and sensory inputs to the amygdala influence the regulation of affective responses during exercise, particularly at intensities above VT. The frontal cortex should remain active through cognitive control and motivational processes in order to promote a more positive affective response. If activation in the frontal cortex is not maintained, and sensory input from the body increases, the amygdala may become uninhibited by the frontal cortex which would promote a more negative (aversive) affective response.

During exercise cerebral blood flow and metabolism remains constant, however there is a shift in resources (oxygen delivery, metabolite removal) between regions required for cognitive, emotional, sensory and motor control (Ide & Secher, 2000). Activation in some regions in the brain comes at the expense of other regions in the brain (Miller & Cohen, 2001). The shift in metabolic resources causes a deregulation in regions not directly essential to the activity (Dietrich, 2003, 2006).

A deregulation in the brain is described by the transient hypofrontality hypothesis and is theorised to be an evolutionarily adaptive mechanism (Dietrich, 2003). The transient hypofrontality hypothesis describes a hierarchical deregulation of brain areas (Dietrich, 2003, 2006). This deregulation is related to higher states of consciousness and occurs during exercise. Dietrich (2003, 2006) proposes that during exercise, metabolic resources are re-allocated within the brain to meet the intensified demands of the exercise stimulus. Deregulation occurs in a five-stage hierarchy of consciousness of which the first are higher-order processes (in the dorsolateral frontal cortex); the second, self-representation and emotional processes (in the ventromedial prefrontal cortex); the third, memory, learning and basic emotions (the limbic system including the amygdala); the fourth, sensory processing (in the thalamus); and finally, arousal processes (in the brainstem).

The transient hypofrontality hypothesis is proposed to explain the anxiolytic effects of acute exercise due to a reduction in over-activity in frontal areas. Evidence from EEG studies has shown an enhancement of alpha and theta activity in the frontal cortex following exercise which indicates decreased frontal activation (Boutcher & Landers, 1988; Nybo & Nielsen, 2001; Petruzzello & Landers, 1994).

Recent studies have provided indirect evidence in support of the transient hypofrontaility hypothesis by examining executive function during exercise. Dietrich and Sparling (2004) found that tasks dependent on executive function (i.e. the Wisconsin Card Sorting Task and Paced Auditory Serial Addition Task) were impaired during cycling and running (70-80% heart rate max) exercise. Intelligence tests (Kaufman Brief Intelligence test and the Peabody Picture Vocabulary test) showed no cognitive impairment during exercise compared to a control. The authors reported that exercise reduced frontal capacity (specifically executive function) and proposed the onset of the first stage of the hypofrontality response (i.e. deregulation in the dorsolateral frontal cortex). In a similar study, Del Giorno, Hall, O'Leary, Bixby and Miller (2010) examined executive function (using the modified Wisconsin Card Sorting Task) during and following exercise at intensities standardised to VT (75% of VT and VT). The findings indicated that executive function was impaired during exercise at both intensities, but the impairment remained following exercise at VT potentially due to hypofrontality effects in the frontal cortex. However, using the same task, no impairment was replicated during exercise at intensities 30% and 50% heart rate reserve (HRR; intensities presumably around VT [approximately 60% aerobic power]) and an impairment was shown at during exercise at intensities at 80% HRR (Wang, Chu, Chu, Chan & Chang, 2013). Wang et al. (2003) propose that inconsistencies in the data are due to differences in exercise modes, durations and intensities.

There is limited direct evidence of the hypofrontality hypothesis. Direct evidence of the transient hypofrontality hypothesis has been provided by measuring event related brain potentials during a flankers task during exercise (60% HRR; Pontifex & Hillman, 2007). These results showed a slower processing speed in frontal and lateral cortical areas indexed by P3 (related to decision-making processes) indicating limited cognitive processing.

According to the DMM, the hypofrontality hypothesis may explain the mechanism by which affective responses are regulated during exercise. This deregulation or hypofrontality

response of the frontal cortex during exercise is proposed to reflect the possible competition of blood flow between cognitive and interoceptive processes at intensities of exercise above VT (Ekkekakis, 2003), which impairs the capacity of the fontal cortex. This would explain the interplay between the cognitive and sensory (interoceptive) mode of inputs to the amygdala. Reduced activation in the frontal cortex would leave the amygdala uninhibited (Davidson 2001, 2002; Davidson, Jackson & Kalin, 2000; Ekkekakis, 2009). Therefore, cognitively-driven control and motivational processes would have little or no influence on affective responses at higher exercise intensities (i.e. above RCP).

The transient hypofrontality hypothesis may be an overarching mechanism by which changes in affective responses during exercise are regulated (Dietrich, 2006). A subtle deregulation in activation in the frontal cortex may be enough to impact more positive affective responses. However, greater deregulation may limit the capacity of the frontal cortex to maintain activation and override the amygdala and induce negative affective responses. In fact, deregulation may induce the anxiolytic effects of exercise at low intensities, but becomes detrimental as the intensity increases. This would potentially explain some inconsistencies within the literature (Wang et al., 2013; Dietrich & Sparling, 2004), but this remains untested.

The DMM is supported by evidence of cognitive control and motivational processes and the hypofrontality hypothesis in its proposal that the frontal cortex and the amygdala provide the main neural basis of affective responses to exercise. It is proposed that affective responses are largely influenced by engagement of the frontal cortex and the capacity to maintain activation through cognitive processes in competition with sensory (interoceptive) inputs during exercise at different intensities.

2.4 Cognitive versus sensory processes

The competition between cognitive and sensory (interoceptive) processes during exercise at intensities around VT and proximal to RCP is thought to account for individual variability in affective responses (Rose & Parfitt, 2007). The observations that some individuals appear to feel better than others at these intensities could potentially be due to activation (reflecting the proposed mechanisms of affective responses outlined) in the frontal cortex, which influence how individuals respond to exercise and how they feel at different intensities.

2.4.1 Individual differences

In accordance with the DMM and the associated mechanisms, individual differences in affective responses may be directly mediated by the way individuals respond to the intensity of exercise. This could impact on the interaction between the frontal cortex and amygdala. Of particular relevance to the DMM are traits associated with somatosensory modulation, the perception of physiological changes, and the notion of individual tolerance of the intensity of exercise (Ekkekakis et al., 2005a; Ekkekakis et al., 2007). Tolerance of the intensity of exercise may impact upon the interplay between cognitive and sensory processes (inputs to the amygdala) and therefore the maintenance of frontal cortex activation to maintain inhibition of the amygdala.

2.4.1.1 Tolerance of the intensity of exercise

Personality traits, such as extraversion, have been shown to modulate mood and are associated with positive affect (Larsen & Ketelaar, 1989, 1991). Yeung (1996) has previously called for personality traits to be considered to help explain variability in responses to exercise and Morgan (1997) stated that 'future work should take both the psychological and physiological profiles into account' (p11). To explain the large individual variability in affective responses, Van Landyut, Ekkekakis, Hall and Petruzzello (2000), argued the need for research to investigate the role of traits. The authors suggested a theoretically relevant self-report measure be developed to assess psychophysiological indices of an approach versus avoidance disposition to exercise behaviours. Already available measures such as the Behaviour Inhibition System-Behavioural Activation System (BAS/BIS; Carver & White, 1994) have been criticised as not fully acknowledging interoceptive factors (Van Landuyt et al., 2000). Instead previous measures (i.e. the Revised Reducer-Augmenter Scale, Clapper, 1990; the Reactivity Scale, Kohn, 1985; and the Eysenck Personality Questionnaire, Eysenck, 1989) focussed too heavily on exteroceptive stimuli (i.e. social cues), whereas affective responses to exercise for example, are proposed to be influenced by predominantly interoceptive factors (see Ekkekakis et al., 2005a for a detailed discussion on current personality measures and exercise related stimuli).

Consequently, Ekkekakis et al. (2005a) developed the *Preference for and Tolerance of Exercise Intensity Questionnaire* (PRETIE-Q) to provide a method to assess sensory modulation to exercise with a focus on interoceptive rather than exteroceptive stimuli. The authors define

preference for exercise intensity as 'a predisposition to select a particular level of exercise intensity when given the opportunity' (p354), and tolerance of exercise intensity as 'a trait that influences one's ability to continue exercising at an imposed level of intensity even when the activity becomes uncomfortable or unpleasant' (p354). The two constructs are related (r = .40 and .50). During the development of the PRETIE-Q, the preference and tolerance subscales indicated weak correlations with the BIS/BAS (r = .38 and .41, respectively) and the Revised Reducer-Augmenter Scale (r = .31 and .34, respectively). In addition, the tolerance subscale indicated weak correlations with the Reactivity Scale (r = .35) and the Eysenck Personality Questionnaire (r = .30). These findings tentatively support independence of the subscales for arousability and sensory modulation-related traits for interoceptive (as opposed to exteroceptive) stimuli (Ekkekakis et al., 2005a). Following conceptual validation of the self-report questionnaire (PRETIE-Q; Ekkekakis et al., 2005a), the authors investigated the relationships between the preference and tolerance scales and affective responses at different intensities of exercise. Participants completed a 15 minute bout of treadmill exercise at an intensity corresponding to 80% of aerobic power below VT, at VT and 10% aerobic power above VT. Neither the preference nor the tolerance scale was associated with affective responses at exercise intensities below the VT. However both scales were positively associated with affective responses at VT, and only the tolerance scale above the VT. Therefore, a higher self-reported tolerance was associated with a more positive affective response at intensities of exercise above the VT which is predominantly aversive or negative. It is unlikely that these intensities would be self-selected and maintained for a long duration, forcing the individual to challenge their perceived, potentially predisposed tolerance. An individual's predisposition to tolerate the intensity of exercise is proposed to underscore individual differences in the responses to exercise beyond somatometric, physiological and exteroceptive differences, which influence affective responses to intensities at (Ekkekakis et al., 2005a) and above the VT (Ekkekakis et al., 2005a; Ekkekakis et al., 2007). It is proposed that tolerance influences affective responses through different cognitive processes via the cognitive mode proposed by the DMM. Interoceptive input to the body during exercise is more biologically hard-wired; however the perception and modulation of the interoceptive input via the cognitive mode may be influenced by predisposed traits.

Afferent cues from the body during strenuous physical activity (around VT and proximal to RCP) represent mostly aversive stimuli (Ekkekakis et al., 2004, 2005b; Hall et al., 2002;

Parfitt et al., 2006; Rose & Parfitt, 2007; Sheppard & Parfitt, 2008; Welch, Hulley, Ferguson & Beauchamp, 2007). Thus, an individual's predisposed exercise intensity tolerance may directly impact the ability of the frontal cortex to maintain activation of the amygdala through the neural mechanisms identified (see 2.3), which may explain some of the variability in affective responses to the intensity of exercise.

Hence, the primary aim of this thesis is to investigate activation in the brain (frontal cortex and amygdala) and affective (pleasure-displeasure) responses during exercise at different intensities. The secondary aim is to examine the influence of the predisposed trait of tolerance of the intensity of exercise upon activation in the brain and affective responses.

This thesis will identify the capacity of the frontal cortex to maintain function during exercise at different intensities of exercise. This thesis will not identify the specific cognitive control or motivational processes employed, but their potential as mechanisms to explain activation of the frontal cortex will be theorised. Activation of the frontal cortex and activity in the amygdala (study V) are investigated to examine their influence upon affective responses towards a neural basis of affective response to exercise. An introduction to the methods and techniques required to examine these aims is provided below.

2.5 Methods and techniques to investigate neural pathways during exercise

There have been few attempts to explore the network of brain areas that are engaged during exercise beyond those involved in basic homeostatic regulation (Delp et al., 2001; Iwamoto et al., 1996). Large bodily movement during exercise severely limits the methods available for researchers to measure activity within the brain during exercise (Ekkekakis, 2009). Modern neuroimaging techniques within the field of psychology and neuroscience such as positron emission tomography (PET), fMRI and EEG are limited in suitability for use in studies involving such dynamic movements. Motion artefact is generally conceived as a potential barrier in this line of research, especially for the use of fMRI. Although research is advancing in this area (Fontes et al., 2013), methodological concerns i.e. head motion and physiological effects (increased blood flow, respiration etc.) associated with exercise during MRI remain (Mehta, Verber, Wieser, Schmit & Schindler-Ivens, 2009, 2012). For example, Mehta et al. (2009, 2012) investigated pedalling during functional MRI

to identify areas of the brain associated with motor function. However, the authors reported artefact in the signal intensity of the blood-oxygenated level dependent (BOLD) response during pedalling, potentially due to the increased physiological arousal caused by exercise. Other researchers have conducted fMRI following exercise (Janse Van Rensburg, Taylor, Hodgson & Benattayallah, 2009), but the exact temporal changes in brain function during exercise cannot be identified by this method. Herein lays the problem associated with determining activation of the brain during exercise, especially to examine affective responses.

To investigate the neural basis of affective responses to exercise the cognitive mode needs to be challenged by stimuli (i.e. exercise) which drives sensory (interoceptive) processes. The changes are transient, and temporal measurements are required. Therefore to examine the aims of this thesis, novel techniques to measure activation in bilateral areas of the frontal cortex and activity in the amygdala during exercise were chosen.

To measure frontal cortex activation, haemodynamics were measured using NIRS. Although this technique is quickly becoming popular in exercise science, its use to understand affective processes during exercise is novel. To measure the amygdala, acoustic startle response methods were employed. Consequently, this is a traditional method used to examine amygdala activity and affective states, but its use in combination with NIRS during exercise at different intensities is novel and exploratory (acoustic startle methodology is described in chapter 8 [study V]).

2.5.1 Near infrared spectroscopy (NIRS)

NIRS is a non-invasive optical neuroimaging technique. It is an effective tool for the examination of cerebral haemodynamics in regions near the surface of the frontal cortex. NIRS can be used during exercise to provide direct temporal (Huppert, Hoge, Diamond, Franceschini & Boas, 2006) and acceptable spatial resolution (approximately 1cm; Rooks, Thom, McCully & Dishman, 2010). NIRS involves the transmission of light in the near infrared light range (700-1000nm), which when passed through tissue is either absorbed by chromophores or scattered throughout the medium. The returned light is detected at multiple wavelengths and provides quantitative measures of the changes in cerebral oxygenation and deoxygenation which reflects functional neuronal activation (Bhambani,

Malik & Mookerjee, 2007; Ekkekakis, 2009; Ferrari, Mottola & Quaresima, 2004; Ferrari & Quaresima, 2012; Perrey, 2012; Obrig et al., 1996).

A recent meta-analysis by Rooks et al. (2010) describes the cerebral (prefrontal) haemodynamic response across exercise intensity domains defined by percentages of peak oxygen uptake (%$\dot{V}O_{2peak}$); low (<30% $\dot{V}O_{2peak}$), moderate (≥30% to <60% $\dot{V}O_{2peak}$), hard (≥60% to <100% $\dot{V}O_{2peak}$) and very hard (≥ 100% $\dot{V}O_{2peak}$). In brief, the data indicated an increase in oxygenation, deoxygenation and blood volume at hard intensities, but a decline in oxygenation and blood volume at maximal and supramaximal intensities as deoxygenation levels continued to increase. Therefore, at volitional exhaustion the supply of oxygen in cerebral areas is reduced compared to the relative demand and activation in the frontal cortex is limited (Ide & Secher, 2000; Shibuya et al., 2004; Subudhi 2007; Rupp & Perrey, 2008). Rooks et al. (2010) observed a quadratic trend of cerebral oxygenation as the exercise increased; oxygenation increased from low to moderate, was stable between moderate and hard, but declined at very hard intensities. These findings provide some support for the transient hypofrontality hypothesis (Dietrich, 2006) as this indicates a deregulation or decline in cerebral activation at maximal intensities. However, hypofrontality did not appear to occur during sub-maximal intensities (potentially above VT and proximal to RCP or hard intensities as described by Rooks et al., 2010) as is theorised by the DMM. Hypofrontality effects may be explained by individual differences. Rooks et al. (2010) described differences between trained and untrained individuals by which training status was determined with an above average (M = 62.7, SD = 7.9) rather than average (M = 40.3, SD = 10.4) $\dot{V}O_{2peak}$, respectively. Cerebral oxygenation and blood flow appeared to decline at hard intensities of exercise in untrained, but not trained individuals. The level of deoxygenation was increased in trained individuals, indicating greater oxygen demand relative to supply. Timinkul et al. (2008) reported high inter-individual variability in prefrontal oxygenation at moderate intensities of exercise. Investigations into the sources of variability to fully understand the haemodynamic response and what it means in terms of neural activation are warranted. Individual differences observed in the haemodynamic response may contribute and explain variability of affective responses to exercise. However, there are currently no NIRS studies examining the neural basis of affective responses and the intensity of exercise. NIRS appears to offer

a practical method for application to examine activation in the frontal cortex during exercise in line with the aims of this thesis.

2.6 Summary

How an individual feels (i.e. pleasure-displeasure) during exercise is important for future exercise participation and adherence. The DMM is a theoretical framework which explains the dose-response pattern of affective responses during exercise at intensities standardised to gas exchange thresholds (i.e. VT and RCP). In addition, the DMM proposes a neural basis for affective responses which remains to be tested. In line with the DMM and support from relevant mechanisms (cognitive control and motivational processes and the transient hypofrontality hypothesis), the frontal cortex is proposed to maintain activation (through cognitive control and motivational processes) to prevent potential hypoactivation in order to override activity in the amygdala driven by sensory (interoceptive) input from the body during exercise. The ability to maintain frontal cortex activation would be associated with a more positive affective response to exercise. Activation of the frontal cortex may be influenced by tolerance of the intensity of exercise, which could explain individual variability in affective responses to exercise. Activation in regions of the frontal cortex can be measured using NIRS, amygdala activity can potentially be measured using acoustic startle methods and affective responses can be recorded using a self-report measure.

Chapter 3

General Methods

This chapter describes the general methods that are used for the five studies within this thesis. In summary, the following aspects of the methodology are described: participant recruitment and screening; self-report measures of affective responses and tolerance of the intensity of exercise; the principles, theory and application of dual (studies I, III and V) and multi-channel (studies II and IV) near infrared spectroscopy techniques; initial assessments and exercise testing, identification of individual respiratory gas exchange thresholds (i.e. ventilatory threshold [VT] and respiratory compensation point [RCP]) and the determination of the corresponding exercise intensities. Data reduction, power analyses and statistical procedures are also explained.

3.1 Participants

The studies were approved by the University Ethics Committee. Studies I and V (project code A11 24/2/10) and study III (project code A11 23/02/11) were conducted at the University of Exeter, U.K. Studies II and IV (project code 290422) were conducted at the University of South Australia, Australia. Participants were given an information sheet and provided their informed consent prior to participation (see Appendix 1). Upon arrival to the laboratory, all participants completed a pre-exercise testing health status questionnaire to ensure their suitability to exercise; the Physical Activity Readiness Questionnaire (Canadian Society for Exercise Physiology, 2002; studies I, II and III) and the Adult Pre-exercise Screening System (Exercise and Sports Science Australia 2011; studies II and IV). All participants were physically active and reported participating in at least 150 minutes of moderate (55 to < 70% maximal heart rate) intensity exercise per week.

3.2 Measures

3.2.1 Exercise-induced affective responses

Affective valence (pleasure-displeasure) was measured using the Feeling Scale (Hardy & Rejeski, 1989). The scale is a uni-dimensional 11-point scale ranging from -5 to +5 with verbal anchors at all odd integers, and at the zero point (see Figure 3.1).

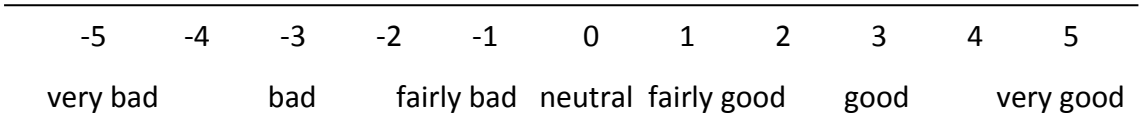


Figure 3.1. The Feeling Scale. Individuals report their affective response corresponding to a number on the scale ranging from *very bad* to *very good*.

The simplicity and brevity of the Feeling Scale allows multiple assessments to be made during exercise (i.e. pre-, during and post-exercise) to capture any acute fluctuations in

responses. Van Landuyt et al. (2000) have previously shown Feeling Scale correlations ranging from 0.51 to 0.88 with the valence scale of the Self-Assessment Manikin (Lang, 1980). The Feeling Scale corresponds to one of the two dimensions of the circumplex model (Russell, 1980; Figure 2.1) recommended for measuring basic affect (pleasure-displeasure; see Ekkekakis & Petruzzello, 1999). The Feeling Scale has been successfully used in studies with adolescents (Schneider et al., 2009; Sheppard & Parfitt, 2008), and both active (Ekkekakis et al., 2004, 2008) and sedentary (Lind, Joens-Matre, & Ekkekakis, 2005; Parfitt et al., 2006) adults.

3.2.2 Preference for and tolerance of the intensity of exercise questionnaire

The preference for and tolerance of the intensity of exercise questionnaire (PRETIE-Q; Ekkekakis et al., 2005a) was used to assess individual self-reported tolerance of the intensity of exercise. The 16-item questionnaire contains eight-items for 'preference' and similarly eight-items for 'tolerance'. The preference scale consists of four-items related to preference for high intensity exercise (e.g., 'I would rather have a short, intense workout than a long, low intensity workout') and four-items related to low intensity exercise which are reverse scored (e.g., 'When I exercise, I usually prefer a slow, steady pace'). The tolerance scale consists of four-items related to tolerance of high intensity exercise (e.g., 'While exercising, I try to keep going even after I feel exhausted') and four-items related to low intensity exercise which are reverse scored (e.g., 'Feeling tired during exercise is my signal to slow down or stop'). Each item is rated along a 5-point response scale (1= 'I totally disagree', 2= 'I disagree', 3= 'Neither agree nor disagree', 4= 'I agree' and 5= 'I strongly agree'). Alpha coefficients of internal consistency ranged from 0.81 to 0.85 for the preference scale, and 0.82 to 0.87 for the tolerance scale. Test-retest reliability coefficients of 0.67 for the preference scale and 0.85 for the tolerance scale after a 3- month interval, and 0.80 for the preference scale and 0.72 for the tolerance scale after a 4-month interval have been shown (Ekkekakis et al., 2005a, for full details). Studies (I and V) focus only on the tolerance scale. Previous research has found significant associations between both the preference and tolerance scales and affective responses reported during exercise at intensities corresponding to the VT (r = .40, and r = .52, respectively). However only the tolerance scale is associated with affective responses (r = .48; Ekkekakis et al., 2005a) and the duration of exercise at intensities above the VT (r = .43; Ekkekakis et al., 2007).

Ekkekakis et al. (2007) reported that the relationship between tolerance and exercise duration in healthy participants remained significant after age, body mass index (BMI) and self-report measures of physical activity status (frequency and duration) and age, body mass index and fitness ($\dot{V}O_{2peak}$) indicating conceptual validity of the subscale.

3.2.3 Near infrared spectroscopy

NIRS is a non-invasive optical neuroimaging technique. Jobsis (1977) first established the use of NIRS techniques to measure biological tissues by showing the transparency of brain tissue to photons in the range of near infrared light which allows chromophores to be detected (for a historical overview see Ferrari & Quaresima, 2012). The optimal range of near infrared light to detect chromophores; specifically haemoglobin (Hb), is between 700 and 1000 nm. Below 700 nm, light cannot be transmitted due to increased light scattering and high absorption by Hb, yet above 1000 nm, photons of light are readily absorbed by water. Therefore light transmittance is highly dependent on tissue composition. Near infrared light in the range of 700 and 1000 nm can pass through most biological tissues with relatively low scatter and absorption. Wavelengths of light within this range are emitted from a light source (transmitter optode) placed on the skin surface. Oxy-haemoglobin (O_2Hb) and deoxy-haemoglobin (HHb), exhibit distinct optical characteristics, so changes in their concentrations can be detected and quantified by measuring the returned light (through a receiver optode) from a location adjacent to the light source. When measuring cerebral areas, the reflection mode is used whereby the light enters the tissue and follows a curved-shape pathway through the tissue which exits near to the point of entry (as opposed to 'transmission' mode where the light passes through the entire tissue, from one side to the other). The depth the light travels (the apex of the curve) is proportional to the distance between the light source and exit point. The light travels through heterogeneous tissue (composed of skin, bone, and fluid) which causes light scattering due to the various absorption and reflectance properties of the different medium the photons pass through. Due to light scattering, which is increased by the depth the light travels (proportional to the area between the transmitter and receiver), the path length of the light is unknown making absolute quantification of haemoglobin impossible. Using the distance between the light source and detector, a differential path length factor can be calculated to allow the quantification of the change (Δ) in concentration of O_2Hb and HHb.

3.2.3.1 Principles of near infrared spectroscopy

The principle of NIRS is based on a modified Beer-Lambert equation for the quantification of changes in chromophores (i.e. Hb);

$$O\,D_\lambda = \mathrm{Log}\left(\frac{I_0}{I}\right) = \varepsilon_\lambda \cdot c \cdot L$$

The optical density (O Dλ) of a medium is a dimensionless factor, I_0 is the incident radiation, I is the transmitted radiation, ε_λ is the extinction coefficient of the chromophore (Hb; $mM^{-1} \cdot cm^{-1}$), c is the concentration of the chromophore (mM^{-1}), L is the distance (cm) between the light source and detector and λ is the wavelength (nm) used. The Beer-Lambert equation applies to wavelengths traveling through non-scattering medium, therefore a modification is required to account for the increased optical path length when light travels through a scattering medium (e.g. biological tissue). The modified Beer-Lambert equation includes a dimensionless 'differential path length factor' B, to account for the increased distance the light travels;

$$O\,D_\lambda = \varepsilon_\lambda \cdot c \cdot L \cdot B + O\,D_{R,\lambda}$$

The oxygen independent loss of light ($O\,D_{R,\lambda}$) is due to scattering in biological tissue. It is assumed that the loss of light due to scattering is constant during measurement. The optical density of each chromophore is measured and the concentration change can be calculated;

$$\Delta c = \frac{\Delta\, O\,D_\lambda}{\varepsilon_\lambda \cdot L \cdot B}$$

The number of chromophores measured (i.e. two; ΔO_2Hb and ΔHHb) must be at least equal to the number of wavelengths transmitted, which results in a set of linear equations to include each chromophore.

3.2.3.2 Cerebral haemodynamic response and neuronal activation

Cerebral ΔO_2Hb and ΔHHb represent the oxygenation and deoxygenation status of tissue, respectively. Total haemoglobin (ΔtHb) can be calculated (ΔO_2Hb + ΔHHb) and provides a measure of local blood volume. The difference between ΔO_2Hb and ΔHHb can be calculated (ΔO_2Hb - ΔHHb) and provides a measure of local oxygen saturation. Oxygen metabolism is required for regional cerebral activation. Therefore, quantification of the concentration changes: ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff when measured over the brain reflect functional cerebral (i.e neuronal) activation (Bhambhani et al., 2007; Ekkekakis, 2009; Ferrari et al., 2004; Perrey, 2012).

If ΔtHb remains constant, ΔO_2Hb and ΔHHb exist in equilibrium. When the tissue is activated, oxygen is extracted causing an increase in ΔHHb. This is followed by an increase in ΔO_2Hb which occurs in parallel with an increase in arterial blood flow, as arterial blood is mostly oxygenated. The increase in ΔO_2Hb is two-fold that of ΔHHb, and leads to hyper-oxygenation and increased ΔtHb, and is defined as 'activation' (Perrey, 2012). Therefore, reduced ΔO_2Hb reflects a reduction in cerebral cortex activation (Shibuya & Tachi, 2006; Subudhi et al., 2007). Cerebral ΔHHb is sensitive to changes in venous oxygen content and consequently less sensitive to changes in ΔtHb than ΔO_2Hb. ΔHHb reflects local changes in deoxygenation status through oxygen extraction. Changes in ΔtHb represent local tissue blood volume and reflect neuronal activity, but ΔtHb is also influenced by systemic effects such as cerebral autoregulation; vasoconstriction of the arteries due to increased end-tidal carbon dioxide ($PetCO_2$; Ogoh, et al., 2005). The difference between ΔO_2Hb and ΔHHb, indicated by ΔHbDiff, represents the relationship between oxygen supply (ΔO_2Hb) and demand (ΔHHb) independent of ΔtHb. If ΔO_2Hb and ΔHHb increase, ΔHbDiff increases and represents adequate oxygen relative to the demand. A concurrent rise in ΔtHb would be driven by the increase in ΔO_2Hb indicating cerebral activation. If the concentration change in ΔO_2Hb remains stable or declines, and ΔHHb increases, ΔHbDiff declines and represents a reduced or inadequate supply of oxygen relative to the demand. A concurrent rise in ΔtHb would be driven by the increase in ΔHHb indicating cerebral deoxygenation. Therefore, ΔHbDiff indicates activation status (i.e. up or down-regulation) of tissue in relation to supply and demand.

In this thesis, for conceptual purposes, ΔO_2Hb and ΔHHb were examined as the primary cerebral haemodynamic variables of interest as they are directly measured using NIRS.

Consequently, the change in ΔtHb and ΔHbDiff were examined as secondary cerebral haemodynamic variables as they are indirectly measured (i.e. calculated from ΔO_2Hb and ΔHHb). The secondary variables help to explain the relationship between ΔO_2Hb and ΔHHb (in relation to oxygen supply and demand).

3.3 Procedures

3.3.1 Initial Assessments

Participants visited the lab on one (studies I and II), three (studies III and IV) or four (study V) occasions with a minimum of 48 hours between sessions (studies III - V). All participants (studies I-V) were instructed to avoid the consumption of alcohol and partaking in any vigorous exercise the day before testing. Participants were also instructed to avoid caffeine for at least 3 hours prior to testing.

Upon arrival to the laboratory for the first time, participants completed an informed consent form and the exercise screening questionnaire (see 3.1). Participant's height and weight were recorded.

3.3.2 Near infrared spectroscopy setup

To measure cerebral haemodynamics, two systems were employed based on the number of independent tissue areas the systems could measure. The dual-channel system (NIRO 200, Hamamatsu Photonics K.K., Hamamatsu, Japan) was capable of measuring up to two independent tissue areas and the multi-channel system (Oxymon Mk II, Artinis Medical Systems, Zetten, the Netherlands) up to eight independent tissue areas.

3.3.2.1 Optode placement

Due to the variability in head size between participants and to ensure consistency and accuracy of optode placement, the location of the optodes were standardised to fixed landmark points on the head in line with the 10/20 international system for electrode placement (see Appendix 2). Measurements were made from (a) the nasion (the dent at the top of the nose) to the inion (the bony part of the skull above the neck); and (b) head circumference, from the supraorbital ridge to the inion around the head. For dual-channel

NIRS, the percentage distances for the placement of optodes to specific electrode positions were calculated for each participant. For multi-channel NIRS, percentage distances for the placement of optodes were calculated for two standard head caps (56 and 58 cm).

3.3.2.2 Dual-channel near infrared spectroscopy (studies I, III, V)

In studies I, III and V (conducted at the University of Exeter, U.K.), two transmitter and two receiver optodes were encased in one of two rubber holders consisting of one transmitter and one receiver placed 40 mm apart. A differential path length factor of 5.93 for the adult forehead was used (van der Zee et al., 1992) to provide a measure (sample rate = 2 Hz) of the concentration changes (μM) in ΔO_2Hb, ΔHHb ΔtHb, and ΔHbDiff.

The optode holders were placed over the left and right dorsolateral prefrontal regions (approximately BA 46) located between Fp1-F3 and Fp2-F4 respectively, of the international 10-20 system. The receiver and transmitter optodes were arranged as shown in Figure 3.2. The optodes were secured to the skin using a double adhesive sticker. Elastic surgical tape and a dark bandage were placed over the optodes and around the participants head to shield from any extraneous light.

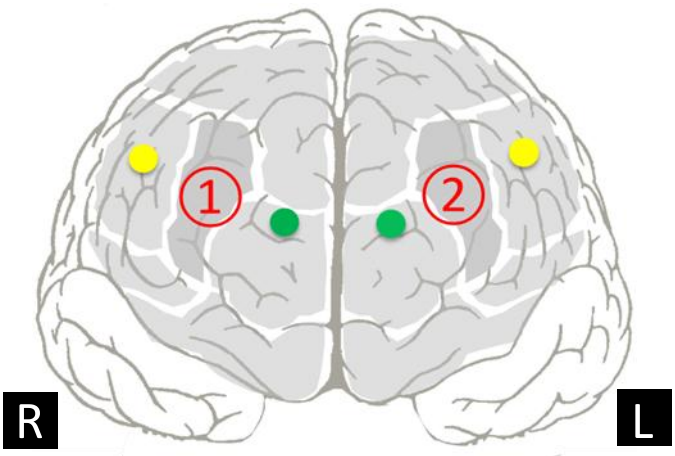


Figure 3.2. The location of optodes using dual-channel NIRS. A light transmitter (green circles) and light receiver (yellow circles) were placed over the approximate locations of the right (1) and left (2) dorsolateral regions of the frontal cortex.

3.3.2.3 Multi-channel near infrared spectroscopy (studies II, IV)

In studies II and IV (conducted at the University of South Australia), four transmitter and two receiver (split-fibre) optodes were secured into the eight fixed optode holders on the head cap. The optode holders were arranged in two squares (four holders 38 mm equidistance apart). The four optodes; two receivers and two detectors, of each square measured four regions (placed bilaterally over the left and right frontal cortex). The receiver and transmitter optodes were arranged as shown in Figure 3.3. The most inferior optodes, were placed in line with Fpz (10% of the distance from the nasion to the inion, above the nasion). As two standard head caps were used, once the cap was fitted to each participant the distance between the optodes was checked to ensure the optodes were 38 mm equidistance apart. Age-specific differential path-length factors (4.99 + 0.0067*age0.814) were calculated (Duncan et al., 1995) to provide a measure (sample rate = 2 Hz) of the concentration changes (μM) in ΔO_2Hb, ΔHHb ΔtHb and ΔHbDiff.

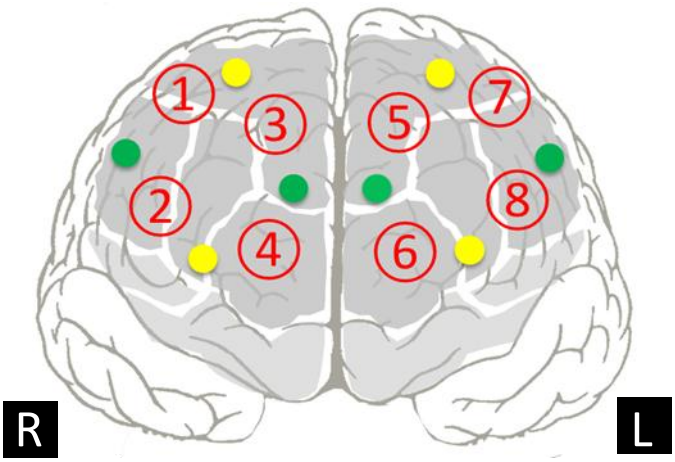


Figure 3.3. The location of optodes using multi-channel NIRS. Light transmitters (green circles) and light receivers (yellow circles) were placed over (eight) sub regions (1-8) of right and left hemispheres of the frontal cortex. Regions with an odd number identify superior regions and areas with an even number identify inferior regions of the frontal cortex.

The approximate locations of the eight regions measured correspond to the right and left frontopolar (BA 10), ventrolateral (BA 47) and posterior (BA 9) and mid (BA 46) dorsolateral regions of the prefrontal cortex. The optodes were adjusted and secured in the holders to ensure good contact was made with the participant's skin and that the area

beneath the optodes was free from obstruction (i.e. hair) as much as possible. A dark bandage was placed over the optodes around the participants head to shield from any extraneous light.

3.3.3 Initial incremental exercise test to exhaustion

In the first session of each study (I-V), participants completed an incremental (20W/min) exercise test to volitional exhaustion (a pedal frequency < 60 rpm despite strong verbal encouragement) on a recumbent cycle ergometer (studies I, III and V, Lode Angio, Groningen, the Netherlands; studies II and IV, Lode Corival, Groningen, the Netherlands) to establish participants peak oxygen uptake ($\dot{V}O_{2peak}$). Expired gases were collected continuously using a computerised metabolic gas analysis system (Cortex Metaylzer 3B, Biophysik, Leipzig, Germany) connected to a oro-nasal (full face) mask (7450 Series V2, Hans Rudolph Inc, Kansas City, USA). Before each test, the system was calibrated using certified gases of known concentration (15% O_2 and 5% CO_2) and an ambient air measure. Volume calibration was conducted using a 3.0 l syringe (5530 series, Hans Rudolph Inc, Kansas City, USA). Heart rate using a chest strap (RS400, Polar Electro, Kempele, Finland) was collected via telemetry by the gas analysis system.

3.4 Data Reduction

3.4.1 Identification of gas exchange thresholds

The achievement of $\dot{V}O_{2peak}$ was verified by, a) a peak or plateau in oxygen consumption (changes < 2 $ml \cdot kg^{-1} \cdot min^{-1}$) with increasing workload; and b) a respiratory exchange ratio of at least 1.10. $\dot{V}O_{2peak}$ was determined by the highest 30 second average.

An individual's physiological profile during exercise can be defined by respiratory gas exchange thresholds and/or changes in blood lactate concentration (see Figure 2.2.). Although there have been reports of a disassociation of respiratory and lactate thresholds for determining the transition from aerobic to anaerobic metabolism, both thresholds indicate the onset of significant physiological demand during exercise (Svedahl & MacIntosh, 2003). Respiratory gas exchange thresholds can be determined from the continuous collection of expired gases taken during exercise. However, to determine blood

lactate concentration, invasive measures (multiple fingerpick blood samples) are required to be taken during exercise. Therefore in the studies within this thesis, exercise intensities were standardised to respiratory gas exchange thresholds; the VT and RCP.

The ventilatory threshold (VT) was identified using the three-method procedure proposed by Gaskill et al. (2001). This involved agreement in the visual identification of VT using; a) modified V-slope method; b) ventilatory equivalent (VE) method; and c) excess carbon dioxide ($ExCO_2$) method, which were checked by an independent investigator. A representation of each of the plots is shown in Figure 3.4.

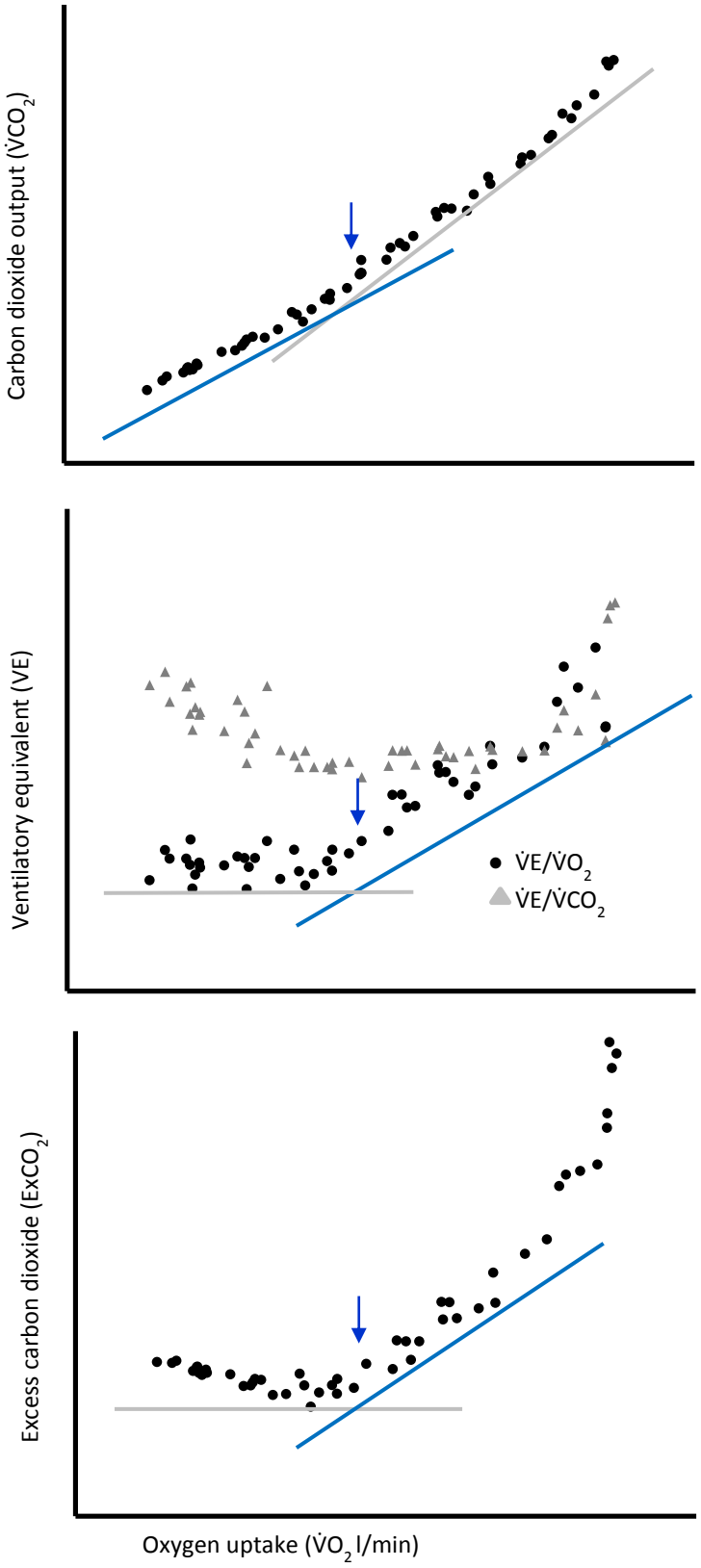
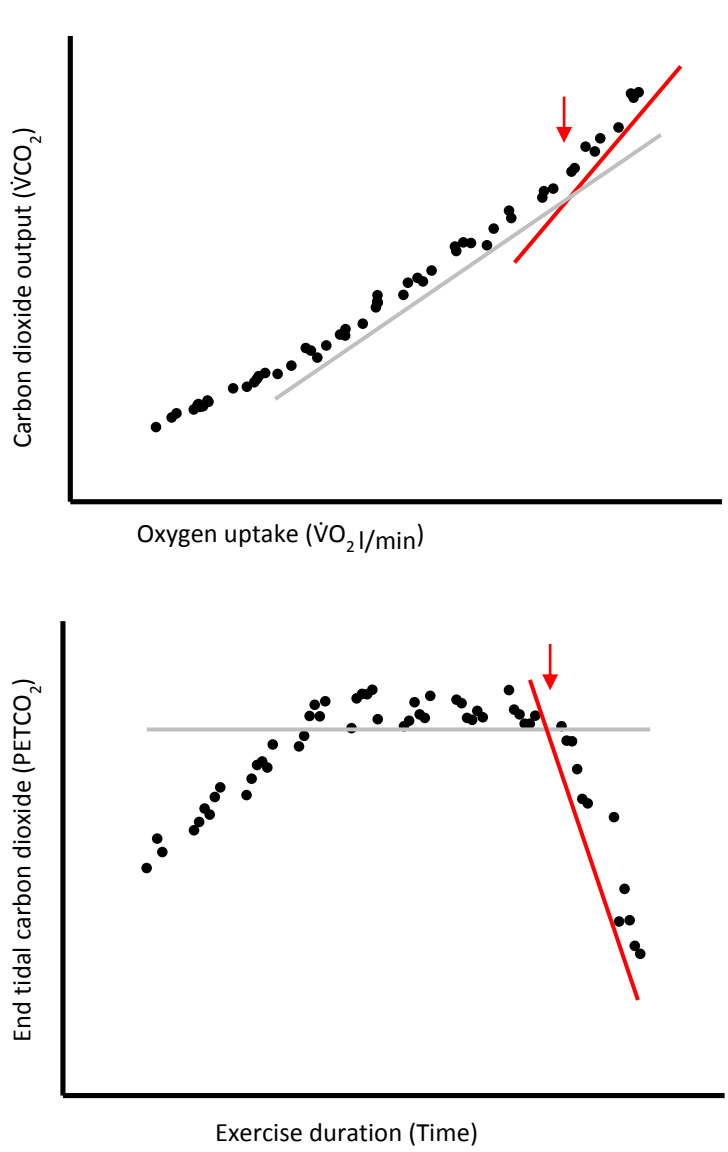


Figure 3.4. Identification of the ventilatory threshold.

The respiratory compensation point (RCP) was determined by the visual agreement in the determination of plots using a) the modified v-slope method (Beaver et al., 1986); and b) $PetCO_2$ over time (Takano, 2000; Wasserman, 1986). A representation of each of the plots is shown in Figure 3.5.



The modified v-slope method (Beaver et al., 1986) involves plotting the production of $\dot{V}CO_2$ over utilisation of $\dot{V}O_2$ to identify the second breakpoint at which there is a disproportionate increase in $\dot{V}CO_2$. The breakpoint between the two slopes indicates RCP.

$PetCO_2$ is an indirect measure of arterial carbon dioxide (Ogoh et al., 2005) is plot over time (Takano, 2000; Wasserman, 1986) to determine the point at which there is a systematic decline in $PetCO_2$ until the attainment of $\dot{V}O_{2peak}$.

Figure 3.5. Identification of the respiratory compensation point.

3.4.2 Determination of exercise intensities

Following the identification of the gas exchange thresholds the corresponding workloads were determined for exercise intensities in subsequent sessions (studies III -V). Individual plots of VO_2 over workloads (W) were created, and a linear regression equation ($y = bx + c$) was used to calculate the workload corresponding to VO_2 (l/min) at, a) 80% of VT (study V); b) VT (study VI); c) 5% above VT (study III); and d) 5% below RCP (studies III - V). The mean response time of the VO_2 adaptation to ramp exercise was accounted for by subtracting two-thirds of the ramp rate (i.e. 20W/min = 13.33W) from the calculated workloads (Whipp, Davis, Torres & Wasserman, 1981).

3.4.3 Cerebral haemodynamic variables

A 30 second baseline measure of cerebral haemodynamics (ΔO_2Hb, ΔHHb, ΔtHb and $\Delta HbDiff$) was recorded in each session immediately prior to exercise. Data were averaged every 10 seconds and normalised to express the magnitude of changes from the baseline period (arbitrarily defined as 0 μM) at the start of exercise to control for within- (studies III and IV) and between- (studies III, IV and V) session effects (Subudhi et al., 2007).

3.5 Power analyses

To calculate sample sizes, power calculations from previous studies were reported or appropriate effect sizes from previous studies and the proposed research design were entered into a statistical power analysis computer program (G*power 3.1.2; Faul et al., 2007). The highest estimate (n) for the combined variables (affective responses, cerebral haemodynamic variables and acoustic startle eye blink responses) for each study was applied.

3.5.1 Affective responses

Affective responses measured during incremental exercise for within-between subject main and interaction effects, expected moderate effect size (Cohen's $d = 1.3$; Welch et al., 2007), and a-priori power of 0.90 at $\alpha = 0.05$, indicate a minimum $n = 8$ (studies I - V).

3.5.2 Cerebral haemodynamic variables

In a recent quantitative analysis of the use of NIRS during incremental exercise critical sample sizes for cerebral haemodynamic variables were calculated (Rooks et al., 2010). The primary haemodynamic variables (ΔO_2Hb and ΔHHb) measured during exercise intensities up to exhaustion with an optode (transmitter-detector) distance of 4 cm (assuming statistical power = .80 and α = .05) indicate a minimum n = 12 (studies I - V).

3.5.3 Acoustic startle eye blink responses

The acoustic startle eye blink response is measured using a photo-electric cell (see 8.2 for detailed methods). However, common methods involve the use of electromyography and no studies have used the photo-electric cell during exercise. Therefore the sample size is based on measurements of the eye blink response using electromyography from one of a few studies to examine the eye blink response following exercise (Tieman, Peacock, Cureton & Dishman, 2001). Flaten (1993) has previously shown that the two methods yield similar recordings of startle eye blink probability and magnitude. Therefore, within subject main and interaction effects, expected moderate effect size (Cohen's d = .28; Tieman et al., 2001), average correlations (r = .60), and a-priori power of .90 at α = .05 indicate a minimum n = 20 (study V).

3.6 Statistical analyses

Data were organised and managed in Microsoft Excel and all statistical procedures were conducted using in statistical software IBM SPSS (Version 19).

Data were explored for normality and homogeneity of variance. Preliminary (physiological data) and main (affective responses and acoustic startle eye blink responses) analyses were conducted using two or three factor analyses of variance (ANOVAs) with either a mixed model (study I) or repeated measures (studies II - V) design.

A multivariate ANOVA for the dependent cerebral haemodynamic variables (ΔO_2Hb, ΔHHb, ΔtHb, $\Delta HbDiff$) was not appropriate due to multicollinearity effects (due to composite variables; ΔtHb and $\Delta HbDiff$). Therefore, separate two or three factor ANOVAs were conducted for each of the primary (ΔO_2Hb, ΔHHb) and secondary (ΔtHb,

ΔHbDiff) cerebral haemodynamic variables. The use of separate ANOVAs to analyse individual cerebral haemodynamic variables is common place within the literature. A bonferroni (or similar) correction has not been reported in previous studies, presumably due to the risk of a Type II error. Therefore, no correction to the analyses (in studies I - V) of the primary or secondary variables was applied.

Greenhouse Geisser corrections were applied to the degrees of freedom if the assumption of sphericity was not met. Significant ($p < .05$) main and interaction effects were followed up using pairwise comparisons and/or partial ANOVAs as appropriate. Effect sizes associated with *F* statistics were expressed as partial eta squared (η_p^2). A description of the dependent and independent variables and specific analyses in each of the five studies are detailed within each experimental chapter. All results are reported as means (*M*) and standard deviations (*SD*) or standard error of the mean (SEM; figures only).

Chapter 4

Study I: Cerebral blood flow in the frontal cortex during incremental exercise: An examination of self-reported tolerance

This chapter (study I) investigates the influence of the predisposed trait of tolerance of the intensity of exercise upon activation in the frontal cortex and affective responses during incremental exercise to exhaustion.

This study formed the basis of oral presentations at the 16th Annual European College of Sports Science congress, Liverpool, UK, July 2011. Tempest, G., Ekkekakis, P., Broomfield, C., Drewell, L., Parfitt, G. *Cerebral asymmetry during exercise using Near Infrared Spectroscopy: An examination of self-reported tolerance* and the Asics conference of Science and Medicine in Sport, Phuket, Thailand, October 2013. Tempest, G., Ekkekakis, P., Parfitt, G. *Self-reported tolerance influences cerebral blood flow during exercise.*

4.1 Background

The influence of the predisposed trait of tolerance of the intensity of exercise has been proposed by Ekkekakis et al. (2005a; see 2.3.1.1). The authors developed this measure to identify specific traits dependent upon the intensity of exercise and associated sensory (interoceptive) processes, which may influence exercise behaviours. Tolerance of the intensity of exercise is a self-reported measure using the preference for and tolerance of the intensity of exercise questionnaire (PRETIE-Q; see 3.2.2). The PRETIE-Q is proposed to provide a psychophysiological index that accounts for interoceptive (as opposed to exteroceptive) stimuli, which influences an approach versus avoidance disposition of exercise behaviours. In particular, self-reported tolerance of the intensity of exercise represents an individual's ability to continue exercising at an imposed level of intensity that is uncomfortable or unpleasant (Ekkekakis et al., 2005a). For example, if during exercise an individual feels their muscles starting to burn and they feel discomfort and displeasure, some individuals may slow down or stop, and others may persevere a little longer. The tolerance subscale attempts to tap into how an individual may cognitively regulate sensory information from the body which may influence how they feel and their perception of the exercise experience.

Self-reported tolerance has not been shown to influence affective (the pleasure-displeasure) responses during exercise at intensities below VT. This is presumably because the exercise stimulus (i.e. the associated interoceptive input from the body) is not of sufficient strength to challenge the individual physiologically. However, evidence has shown that self-reported tolerance does explain the degree of displeasure or discomfort experienced during exercise at intensities above VT (Ekkekakis et al., 2005a, 2007).

As described by the DMM (see 2.1.3), the competition between cognitive and sensory (interoceptive) factors occurs at intensities of exercise around VT. Cognitive factors, within the frontal cortex, such as cognitive control (see 2.3.1) and motivational processes (see 2.3.2) are proposed to regulate affective responses. At intensities of exercise above VT, as sensory (interoceptive) input from the body intensifies, the capacity of the frontal cortex becomes limited due to a deregulation of metabolic resources to other areas of the brain (see 2.3.3).

It is proposed that individuals with high self-reported tolerance may be more capable of cognitively regulating how sensory information from the body is perceived during exercise at physiologically challenging intensities (i.e. above VT), than those with a low self-reported tolerance. Individuals with high self-reported tolerance would report less negative affective responses than those with a low self-reported tolerance. In addition, individuals with high self-reported tolerance may exercise for longer at an imposed level of intensity (i.e. above VT) that is uncomfortable or unpleasant.

The present study investigates if self-reported tolerance of the intensity of exercise influences activation (reflected by changes in cerebral haemodynamics using NIRS) in the dorsolateral region of the frontal cortex (both right and left hemispheres) and affective responses during incremental exercise to volitional exhaustion.

Three hypotheses were examined: 1) Activation of the frontal cortex is different between individuals with high or low self-reported tolerance; 2) Individuals with high self-reported tolerance report less negative affective response at intensities above VT; and 3) Individuals with high self-reported tolerance exercise for longer than those with low self-reported tolerance at intensities above VT.

4.2 Methods

Participants

A university cohort of sports science students (n = 259) completed the PRETIE-Q (Ekkekakis et al., 2005a). Participants (n = 28) with the highest (males: n=7; age, M = 20.1, SD = 1.1 years; height, M = 177.1, SD = 5.0 cm; body mass, M = 71.5, SD = 4.1 kg; BMI, M = 22.8, SD = 1.4; females: n=7; age, M = 20.7, SD = 1.4 years; height, M = 164.8, SD = 3.1 cm; body mass, M = 65.0, SD = 7.1 kg; BMI, M = 23.9, SD = 2.5) and lowest (males. n=7; age, M = 21.3, SD = 2.7 years; height, M = 179, SD = 8.4 cm; body mass, M = 77.6, SD = 9.1 kg; BMI, M = 24.2, SD = 2.2; females: n=7; age, M = 20.8, SD = 3.8 years; height, M = 167.4, SD = 5.5 cm; body mass, M = 57.5, SD = 3.8 kg; BMI, M = 20.6, SD = 1.9) tolerance scores were invited to participate.

Measures

The PRETIE-Q (Ekkekakis et al., 2005a) was used to assess individual self-reported tolerance of the intensity of exercise (see 3.2.2). Alpha coefficients of internal consistency in the present study ranged between 0.76 and 0.81 for the tolerance scores of the high and low groups. Cerebral haemodynamics in the left and right dorsolateral region of the prefrontal cortex were measured using dual-channel NIRS as previously described (see 3.3.3.2). Approximate locations of the regions (1-2; right, left) measured are shown in Figure 4.1. Affective responses (pleasure-displeasure) were recorded using the Feeling Scale (Hardy & Rejeski, 1989; see 3.2.1).

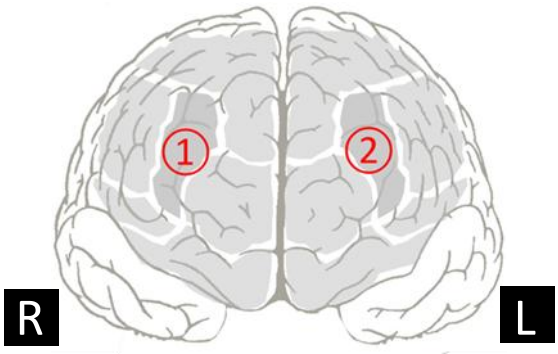


Figure 4.1. Approximate locations of the dorsolateral regions (1-2; right and left hemispheres) of the frontal cortex measured using dual-channel NIRS.

Procedures

Upon arrival at the laboratory, participants were screened (i.e. completed the informed consent form and exercise testing questionnaire) and initial assessments were taken (see 3.3.1). Participants were seated on a recumbent cycle ergometer and the NIRS optodes were carefully positioned (see 3.3.2) and a facemask (to measure metabolic data) was fitted. Participants completed an incremental exercise test to volitional exhaustion (20 W/min) as previously described (see 3.3.3). Cerebral haemodynamics and expired gases were collected continuously and affective responses were recorded pre-exercise, during (every minute) and at the end of exercise.

Data reduction

Following the identification of gas exchange thresholds (VT and RCP) and $\dot{V}O_{2peak}$ (see 3.4.1), primary (ΔO_2Hb and ΔHHb) and secondary (ΔtHb and $\Delta HbDiff$) cerebral haemodynamic variables and affective responses were extracted at time points corresponding to pre-exercise (affective responses only) and intensities of exercise; 80% of VT (below VT), VT, RCP and End (see 3.4.3).

Statistical analyses

The independent variables were Group (high, low) based on self-reported tolerance, Region (1, 2) and Time (corresponding to pre-exercise and intensities below VT, VT, RCP and End). The dependent variables were changes in cerebral haemodynamics (primary [ΔO_2Hb, ΔHHb] and secondary [ΔtHb, $\Delta HbDiff$]), affective responses and exercise duration (seconds).

To examine hypothesis 1, a Group (2; high, low) by Region (2; right, left) by Time (4; below VT, VT, RCP, End) mixed model ANOVA was conducted for each of the primary (ΔO_2Hb, ΔHHb) and secondary (ΔtHb, $\Delta HbDiff$) NIRS variables. To examine hypothesis 2, a Group (2; high, low) by Time (5; pre-exercise, below VT, VT, RCP, End) mixed model analysis of variance (ANOVA) was conducted for affective responses. Finally, to examine hypothesis 3, two independent sample t-tests with a Bonferoni correction (alpha of .05 / 2 [number of tests] = .025) examined the duration of exercise (from VT to RCP, RCP to End) of the high and low tolerance groups.

4.2 Results

4.2.1 Preliminary analyses

To examine that there were no differences in fitness (indicated by $\dot{V}O_2$ $ml \cdot kg^{-1} \cdot min^{-1}$) between males and females in the high and low tolerance groups at time points corresponding to intensities of exercise (below VT, VT, RCP, End), a Group (2) by Gender (2) by Time (4) mixed model ANOVA was conducted. The analysis showed no differences in VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$) between groups (main and interaction effects; $p > .05$).

However, $\dot{V}O_2$ (ml·kg^{-1}·min^{-1}) was (a) higher in males (M = 32.4, SD = 5.1) than females (M = 29.1, SD = 2.7; $F(1, 24) = 4.73$, $p = .040$, $\eta_p^2 = .17$), and (b) significantly different at each intensity: below VT (M = 19.0, SD = 2.7), VT (M = 23.7, SD = 3.2), RCP (M = 36.1, SD = 5.3) and End (M = 43.8, SD = 6.4) of exercise ($F(1, 26) = 1141.48$, $p < .001$, $\eta_p^2 = .98$). A Gender by Time interaction indicated that males had a higher VO_2 (ml·kg^{-1}·min^{-1}) than females at each intensity of exercise ($F(1, 26) = 4.52$, $p = .040$, $\eta_p^2 = .16$; see Table 4.1.).

Table 4.1. Oxygen uptake ($\dot{V}O_2$ ml·kg^{-1}·min^{-1}) for males and females in the high and low tolerance group at each time point. Males indicated a higher $\dot{V}O_2$ than females at each intensity of exercise (*$p < .05$).

	High tolerance group			Low tolerance group		
Intensity	Males* (n =7)	Females (n =7)	Total (n =14)	Males* (n =7)	Females (n =7)	Total (n =14)
below VT	20.9 (2.8)	18.1 (1.7)	19.5 (2.7)	18.8(3.2)	18.0 (1.6)	18.4 (2.5
VT	26.1 (3.5)	22.7 (2.3)	24.4 (3.4)	23.6 (4.0)	22.5 (2.0)	23.0 (3.1)
RCP	40.1 (4.7)	34.8 (3.5)	37.8 (5.1)	36.7 (7.0)	34.2 (3.6)	35.4 (5.5)
End ($\dot{V}O_{2peak}$)	49.4 (5.9)	41.7 (4.2)	45.6 (6.3)	43.0 (8.2)	41.1 (4.0)	42.1 (6.3)

To ensure self-reported tolerance scores, as assessed by the PRETIE-Q, were different between the two groups (high, low) and that there was no difference in overall time to exhaustion, two independent t-tests were conducted. These tests showed that self-reported tolerance scores were significantly different between high (M = 33.1, SD = 2.3, range 30 to 38) and low (M = 21.1, SD = 1.9, range 18 to 24; $t(26) = 15.07$, $p < .001$) tolerance groups. No difference ($p > .05$) in overall time (sec) to exhaustion between high (M = 676, SD = 116) and low (M = 646, SD = 93) tolerance groups was shown.

4.3.2 Cerebral haemodynamics (hypothesis 1)

Cerebral ΔO_2Hb

A significant main effect of Time, $F(1,33) = 99.39$, $p < .001$, $\eta_p^2 = .79$, and significant Group by Region, $F(1,26) = 4.37$, $p = .047$, $\eta_p^2 = .14$, and Group by Region by Time, $F(1,34) = 4.14$, $p = .039$, $\eta_p^2 = .14$, interactions were recorded for ΔO_2Hb (μM). Follow up tests indicated that ΔO_2Hb remained stable from below VT ($M = 2.04$, $SD = 2.39$) to VT ($M = 2.71$, $SD = 2.82$), then increased from VT to RCP ($M = 12.14$, $SD = 5.51$) and RCP to End ($M = 13.49$, $SD = 6.87$) in both groups. In the left region, ΔO_2Hb was greater in the low ($M = 8.33$, $SD = 3.88$) than the high tolerance ($M = 6.80$, $SD = 4.36$) group. In the right region, ΔO_2Hb was similar between the low ($M = 7.46$, $SD = 4.01$) and high tolerance ($M = 7.78$, $SD = 4.27$) groups. The three factor interaction indicated that from VT to RCP, the low tolerance group indicated a significantly greater ΔO_2Hb in the left than the right region at RCP (and when compared to both regions in the high tolerance group). From RCP to End, ΔO_2Hb remained stable in both regions in the low tolerance group, but significantly increased in both regions in the high tolerance group. Therefore, at End, ΔO_2Hb remained greater in the left than the right region in the low, whereas ΔO_2Hb was greater in the right than the left region in the high tolerance group. The three factor interaction (*M, SEM*) for ΔO_2Hb is shown in Figure 4.2.

The means and standard deviations (hypothesis 1) of cerebral haemodynamic variables (μM) in this study are provided in Appendix 3a.

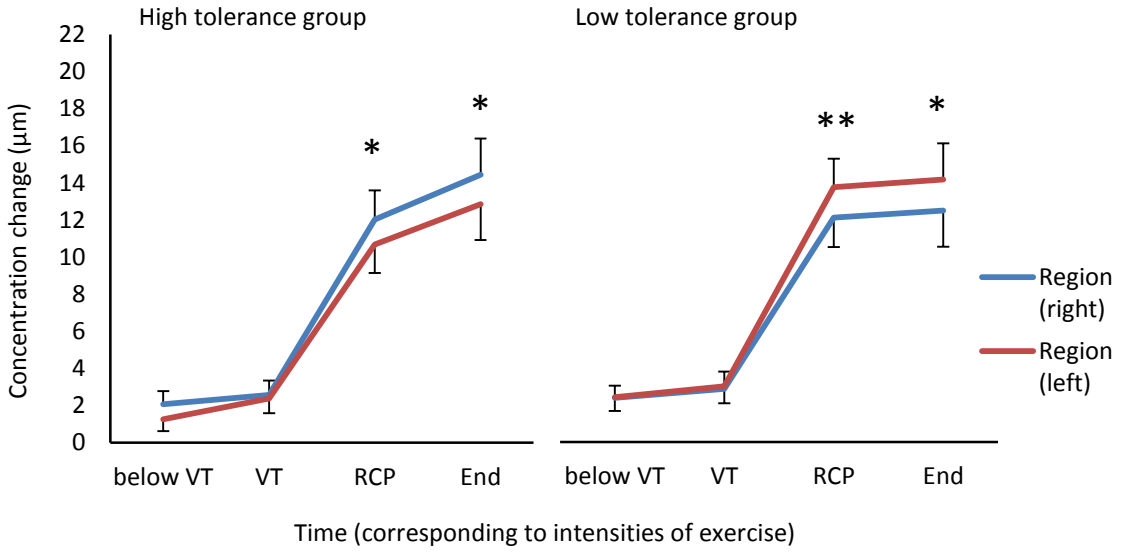


Figure 4.2. Cerebral oxygenation (ΔO_2Hb) in the frontal cortex at each time point for the high and low tolerance groups, *M* and *SEM*. * indicates significantly greater ΔO_2Hb than the other region within group. ** indicates significantly greater ΔO_2Hb than the other region within and between groups.

Cerebral ΔHHb

Significant main effects of Region, $F(1,26) = 5.33$, $p = .029$, $\eta_p^2 = .17$, and Time, $F(1,31) = 50.99$, $p < .001$, $\eta_p^2 = .66$, were recorded for ΔHHb (µM). Follow up tests indicated that ΔHHb was greater in the right ($M = 1.32$, $SD = 1.23$) than the left ($M = 1.01$, $SD = 1.32$) region in both groups. Cerebral ΔHHb remained stable from below VT ($M = .16$, $SD = .53$) to VT ($M = .06$, $SD = .68$), then increased from VT to RCP ($M = 1.03$, $SD = 1.68$) and RCP to End ($M = 3.41$, $SD = 2.53$) in both groups.

Cerebral ΔtHb

A significant main effect of Time, $F(1,32) = 128.66$, $p < .001$, $\eta_p^2 = .83$, and a Group by Region by Time, $F(1,37) = 3.67$, $p = .049$, $\eta_p^2 = .12$, interaction were recorded for ΔtHb (µM). Follow up tests indicated that ΔtHb remained stable from below VT ($M = 2.20$, $SD = 2.33$) to VT ($M = 2.77$, $SD = 2.80$), then increased from VT to RCP ($M = 13.16$, $SD = 6.26$) and RCP to End ($M = 16.89$, $SD = 7.50$) in both groups. The three factor interaction indicated that from VT to RCP, ΔtHb was significantly lower in the left region in the high tolerance than low tolerance group. From RCP to End, ΔtHb was greater in the high tolerance than the low tolerance group. At End, ΔtHb remained similar in the left and right regions in the low, whereas ΔtHb was greater in the right than the left region in the high

tolerance group (and when compared to both regions in the low tolerance group). The three factor interaction (*M, SEM*) for ΔtHb is shown in Figure 4.3.

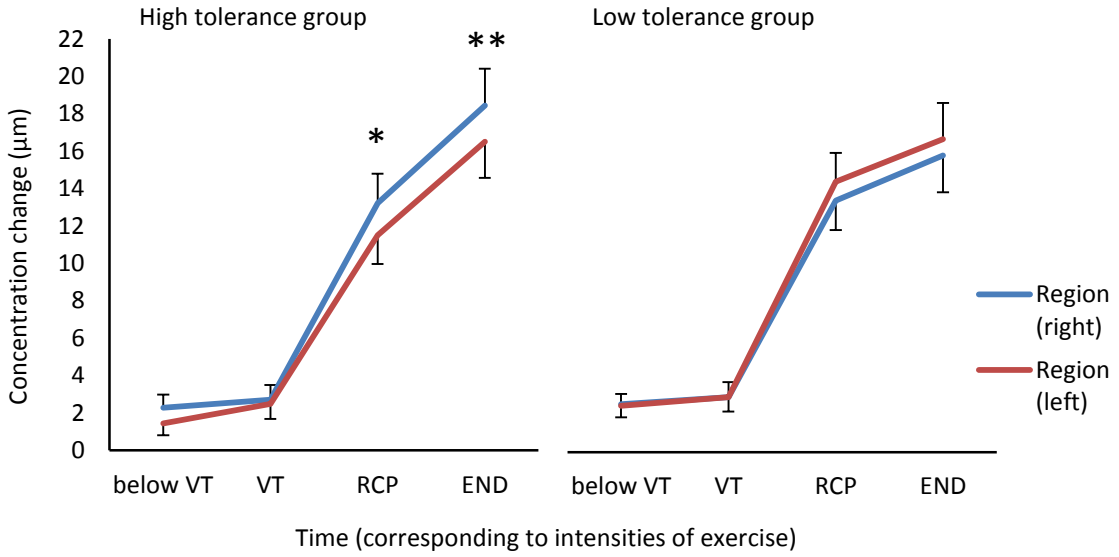


Figure 4.3. Cerebral blood volume (ΔtHb) in the frontal cortex at each time point for the high and low tolerance groups, *M* and *SEM*. * indicates significantly less ΔtHb in the left region than the other group. ** indicates significantly greater ΔtHb than the other region within and between groups.

Cerebral ΔHbDiff

A significant main effect of Time, $F(1,33) = 57.49$, $p < .001$, $\eta_p^2 = .69$., was recorded for ΔHbDiff (µM). Follow up tests indicated that ΔHbDiff remained stable from below VT ($M = 1.88$, $SD = 2.57$) to VT ($M = 2.65$, $SD = 3.00$), then increased from VT to RCP ($M = 11.11$, $SD = 5.22$) and remained stable from RCP to End ($M = 10.08$, $SD = 7.14$) in both groups.

4.3.3 Affective responses (hypothesis 2)

A significant main effect of Time, $F(2, 39) = 38.35$, $p < .001$, $\eta_p^2 = .56$, and a significant Group by Time, $F(4, 104) = 3.57$, $p = .009$, $\eta_p^2 = .05$, interaction were recorded for affective responses. Follow up tests indicated that affective responses declined from pre-exercise ($M = 2.43$, $SD = 1.35$) to below VT ($M = 1.86$, $SD = 1.24$), but remained stable until VT ($M = 1.68$, $SD = 1.44$). Affective responses declined from VT to RCP ($M = -.11$, $SD = 2.04$) and RCP to End ($M = -1.04$, $SD = 2.52$). The interaction indicated that the

decline in affective responses from pre-exercise to below VT was a result of the responses in the low group. Both groups reported similar affective responses from below VT to VT, at which point there was a greater decline in the low than the high tolerance group (see Figure 4.3). Therefore, despite positive affective responses in both groups at VT, affective responses were more negative at RCP and End in the low than the high tolerance group. The two factor interaction (*M, SEM*) for affective responses is shown in Figure 4.4.

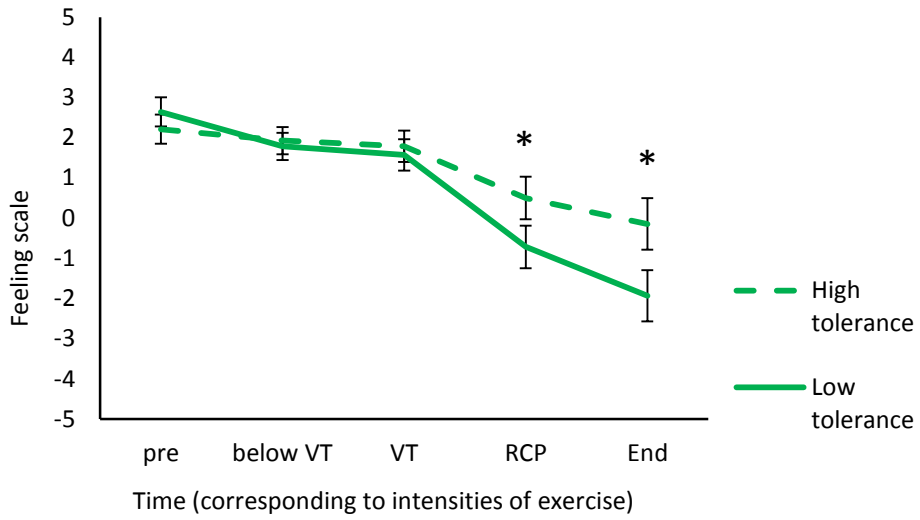


Figure 4.4. Affective responses reported at each time point for the high and low tolerance groups, *M* and *SEM*. * indicates significantly more positive affective responses in the high than the low tolerance group.

4.3.4 Exercise Duration (hypothesis 3)

There was no difference in the duration (sec) of exercise from VT to RCP between the high ($M = 250$, $SD = 44$) and low ($M = 260$, $SD = 53$) tolerance group ($p > .025$). However, the duration of exercise from RCP to End was significantly longer in the high ($M = 145$, $SD = 39$) than the low ($M = 113$, $SD = 24$) tolerance group ($t(26) = 2.62$, $p = .014$). A significant positive correlation was shown between the duration of exercise from RCP to End and self-reported tolerance ($r = .48$, $p < .001$).

4.4 Discussion

Self-reported tolerance of the intensity of exercise is a predisposed trait that influences how an individual feels and their ability to continue exercising at levels of intensity associated with feelings of displeasure and discomfort (Ekkekakis et al., 2005a). It was proposed that activation in the frontal cortex would be different between individuals with high and low self-reported tolerance.

To the author's knowledge, this is the first study to investigate cerebral haemodynamics during exercise and the influence of self-reported tolerance of the intensity of exercise using the PRETIE-Q. The main findings show that individuals with high and low self-reported tolerance demonstrate different asymmetrical patterns of activation within the dorsolateral region of the frontal cortex during exercise at intensities above VT (hypothesis 1). No differences in the haemodynamic response were observed between the high and low tolerance groups at intensities below VT. According to the DMM, intensities of exercise below VT do not induce such intense sensory input from the body; therefore the frontal cortex remains unchallenged and affective responses are predominantly positive. Self-reported tolerance would not be expected to influence activation of the frontal cortex by cognitive control and motivational processes.

During exercise at intensities from VT to RCP, there were no differences in cerebral haemodynamics in the right region between the high and low tolerance groups. However, in the left region, the low tolerance group indicated greater oxygenation (ΔO_2Hb) and blood volume (ΔtHb) than the high tolerance group. Oxygen extraction (ΔHHb) and saturation (ΔHbDiff) increased in both regions in both groups. This implies that activation in the left region in the low tolerance group was greater than that observed in both the right and left regions of the high tolerance group at RCP. It is proposed that at these intensities greater activation in the left region in the low tolerance group is due to an increase in cognitive control and motivational processes as a way of regulating and maintaining frontal cortex function in order to compete with the increased sensory input from the body. This would allow the individual to continue exercise at such challenging intensities. The low tolerance group would be expected to require more cognitive processing than the high tolerance group in order to endure the challenging intensities of exercise.

During exercise at intensities from RCP to End, there were differences in cerebral haemodynamics between high and low tolerance groups in both the left and right regions. In the low tolerance group, oxygenation (ΔO_2Hb) and blood volume (ΔtHb) remained stable in both regions until the end of exercise; however oxygenation (ΔO_2Hb) and blood volume (ΔtHb) were still greatest in the left than the right region. These responses coupled with the rise in deoxygenation (ΔHHb) and stable saturation ($\Delta HbDiff$; observed in both regions and groups at these intensities), indicate reduced activation (i.e. reduced blood flow relative to oxygen extraction) in the frontal cortex. Therefore, the low tolerance group were unable to maintain frontal cortex activation which potentially limited their cognitive processing capacity at these higher intensities.

In the high tolerance group, oxygenation (ΔO_2Hb) and blood volume (ΔtHb) increased in both regions until the end of exercise; however oxygenation (ΔO_2Hb) and blood volume (ΔtHb) were greater in the right than the left region. These responses coupled with the rise in deoxygenation (ΔHHb) and stable saturation ($\Delta HbDiff$: observed in both regions and groups at these intensities) indicate maintained cerebral activation (increased blood flow relative to oxygen extraction) in the frontal cortex. The maintained activation in the left region presumably indicates cognitive attempts to continue exercising, possibly through similar mechanisms to those observed in the low tolerance group from VT to RCP. However, the high tolerance group did not engage their cognitive efforts until they reached higher intensities of exercise i.e. RCP to End (exhaustion). The high tolerance group showed greatest activation in the right region which was not observed in either region in the low tolerance group. This activation is explained by an increase in blood volume due to the rise in oxygenation (ΔO_2Hb and blood flow) coupled with the increase in oxygen extraction (ΔHHb) and stable saturation ($\Delta HbDiff$; the latter two observed between the two regions and groups). Therefore, greater activation in the right region of the high tolerance group indicated a unique maintenance of frontal cortex activation at intensities near exhaustion (i.e. RCP to end of exercise). It is proposed that activation in the right hemispheric region is due to cognitive control processes involved in the regulation of negative affect (Beauregard et al., 2001; Ochsner et al., 2004).

Both groups reported positive affective responses at VT, however individuals with high self-reported tolerance reported less negative affective responses at intensities of exercise above VT than those with low self-reported tolerance. Although not statistically significant

(p = .067), the decline in affective responses above VT and tolerance scores indicated that a high tolerance score was associated with a less negative affective response (hypothesis 2), as previously shown by Ekkekakis et al. (2007).

Despite no differences in fitness ($\dot{V}O_2$ ml·kg^{-1}·min^{-1}) between the two groups, individuals with high self-reported tolerance exercised for longer above their individually determined RCP compared to those with low self-reported tolerance (hypothesis 3). The duration of exercise was positively associated with self-reported tolerance scores at intensities of exercise from RCP to End. This finding indicates that those individuals with high self-reported tolerance can continue to exercise at levels of intensity associated with feelings of displeasure and discomfort (Ekkekakis et al., 2005a). However, it may be that those individuals are able to maintain frontal cortex function more efficiently as their affective responses are not as negative.

Self-reported tolerance of the intensity of exercise is associated with the cerebral haemodynamic response during exercise. It appears that those individuals with low self-reported tolerance utilise cognitive mechanisms in left hemispheric regions during exercise at intensities above the VT and proximal to the RCP. Those individuals with high self-reported tolerance do not appear utilise the same left hemispheric areas of the frontal cortex until higher intensities of exercise (i.e. above the RCP). Additionally, individuals with a high tolerance can induce activation in right hemispheric areas of the frontal cortex during exercise at intensities near exhaustion. Self-reported tolerance potentially influences, or is influenced by, the way an individual perceives and self-regulates somatosensory information during exercise which in turn influences the regulation of affective responses.

These findings provide initial support that maintenance of frontal cortex activation during exercise influences affective responses in line with the DMM. It is proposed that active involvement of the frontal cortex suppressed the aversive stimuli (e.g. negative affect) theorised to be mediated by the amygdala driven by intensified sensory (interoceptive) input from the body. Self-reported tolerance of the intensity of exercise may influence the interplay between the frontal cortex and amygdala. It is suggested by the model that individuals with high self-reported tolerance have the ability to retain cognitive processing (therefore frontal cortex activation) at such challenging intensities of exercise. A less negative affective response would be experienced due to activation in the frontal cortex

which overrides subcortical areas of the brain. It is hypothesised that the intensified sensory (interoceptive) input, particularly to the amygdala, provides a greater challenge to those individuals with low self-reported tolerance to maintain cognitive processing.

In support of the proposal of the DMM, individuals with a low tolerance indicated reduced cortical activation at intensities above RCP. This was shown by stable oxygenation (ΔO_2Hb) in the presence of increases in deoxygenation (ΔHHb; i.e. lack of oxygen supply relative to demand). Individuals with a high tolerance had continued activation at intensities above RCP. This was shown by increases in oxygenation (ΔO_2Hb) and deoxygenation (ΔHHb; i.e. adequate oxygen supply relative to demand) in the frontal cortex. Therefore the low tolerance group indicated deregulation (hypofrontality effects) in the frontal cortex (Dietrich, 2003, 2006; see 2.3.3).

The role of individual differences, which have been proposed to influence affective responses to the intensity of exercise, or more specifically influence self-regulatory approaches in dealing with exercise induced negative affect, are highlighted in this study. However, no measures of the exact cognitive mechanisms have been identified here. A qualitative approach to understand an individual's motivation and the coping strategies employed during exercise or intervention of a cognitive strategy is required. Furthermore, the identification of specific regions (and asymmetry) of the frontal cortex using neuroimaging methods during exercise is necessary.

Previous research has indicated withdrawal-related behaviours are associated with activation of the right hemisphere (Davidson, 1993; see 2.3.2). The increased activation of the right frontal cortex observed in those individuals with high tolerance does not reflect withdrawal-type behaviour due to their perseverance of exercise at intensities above the RCP. It is postulated that right frontal cortex activation is due to active avoidance of withdrawal-type behaviour (as opposed to promoting an 'approach-related behaviour) in the presence of negative affect (Woo, Kim, Kim, Petruzzello & Hatfield, 2009), which allowed individuals with high self-reported tolerance to continue exercising. This would indicate differential cognitive processes used by individuals with low self-reported tolerance.

Alternatively, Craig's theory of forebrain emotional asymmetry (2005; see 2.3.2) may explain the increase in activation within the right hemisphere of the individuals with a high

tolerance. Craig proposes lateralisation of interoceptive input from afferent cues. Parasympathetic afferents activate primarily the left and sympathetic afferents the right insular cortices. Due to the increased sympathetic activity exercise induces, heightened activation of the right insular may have had an impact on the overlying regions of the right frontal cortex measured in this study (Woo et al., 2009). Although there were no physiological differences between the two groups, the perceptual representation of the afferent input may be influenced by the individual's self-reported tolerance of the intensity of exercise. It is plausible that the individuals with high self-reported tolerance had a more acute ability to regulate their somatosensory perception. Nonetheless the frontal cortex remained activated and therefore influenced affective responses (i.e. they were less negative). It is important to highlight that a range of factors such as training status may have contributed to the differences in perceptual and haemodynamic responses between the high and low tolerance groups. In this cross-sectional study, the training status of the participants was not assessed, however as previously indicated, no differences in aerobic power between the two groups were shown. Future research should address the potential influence or relationship of training status and self-reported tolerance of the intensity of exercise on haemodynamic responses.

This study has provided neurophysiological evidence (through the use of NIRS) of differential patterns of activation of the frontal cortex due to individual differences (i.e. self-reported tolerance) measured using the PRETIE-Q. Potential mechanisms to explain activation in the frontal cortex during exercise were theorised (cognitive control and motivational processes) and the capacity of the frontal cortex to maintain activation as the intensity of exercise increased was indicated. At intensities of exercise above VT, activation in the frontal cortex was associated with affective responses. Activation in (eight) sub regions of the frontal cortex and the association with affective responses will be examined further in chapter 5 (study II).

Chapter 5

Study II: Cerebral blood flow in sub regions of the frontal cortex during incremental exercise: An examination of affective responses

The previous chapter (study I), showed that self-reported tolerance influences activation in the frontal cortex during incremental exercise to exhaustion. Differential and asymmetrical activation in the frontal cortex was observed. Activation in the frontal cortex was associated with affective responses to exercise at intensities above VT. Therefore, this chapter (study II) examines (without self-reported tolerance) activation in (eight) sub regions of the frontal cortex to further examine differential and asymmetrical activation during incremental exercise to exhaustion and affective responses.

This study formed the basis of a poster presentation at the Australasian Brain Stimulation Meeting, Melbourne, Australia, July 2013 and an oral presentation at the Asics conference of Science and Medicine in Sport, Phuket, Thailand, October 2013. Tempest, G., Eston. R., Parfitt, G. *Blood flow in the brain during exercise: A multi-channel near infrared spectroscopy study.*

5.1 Background

In the recent meta-analysis (previously described in 2.5.1), Rooks et al. (2010) identified that during exercise at sub-maximal intensities (≥ 60% $\dot{V}O_{2peak}$) individuals indicated an increase in oxygenation, deoxygenation and blood volume. Furthermore, during exercise at and near maximal intensities individuals indicated reduced oxygenation and blood volume, and a continued rise in deoxygenation (as observed in the low tolerance group above RCP [study I]).

Rooks et al. (2010) reported large variability in the cerebral haemodynamic response, particularly cerebral oxygenation (ΔO_2Hb) during exercise at sub-maximal and near maximal intensities, which they attributed to training status. The authors reported that oxygenation decreased earlier during exercise in those individuals with an average $\dot{V}O_{2peak}$ and later in those with an above average $\dot{V}O_{2peak}$ (see 2.5.2). Timinkul et al. (2008) analysed individuals' (n=10) haemodynamic data from an incremental exercise test and reported substantial variations in that the pattern of oxygenation (ΔO_2Hb) between individuals. Ekkekakis (2009) proposes that such variations in the haemodynamic response represent individualised neuronal activation in the frontal cortex.

The findings from study I echo the pattern of the cerebral haemodynamic response across exercise intensities as identified by Rooks et al. (2010). In study I, individuals were classified as having an average $\dot{V}O_{2peak}$ (M = 43.8, SD = 6.4 ml·kg^{-1}·min^{-1}) comparable to those described by Rooks et al. (2010; M = 40.3, SD = 10.4 ml·kg^{-1}·min^{-1}). The low tolerance group showed reduced activation (stable oxygenation [ΔO_2Hb] and increased deoxygenation [ΔHHb]) in the frontal cortex at intensities of exercise above RCP. However, the high tolerance group showed maintained activation (increased oxygenation [ΔO_2Hb] and deoxygenation [ΔHHb]) in the frontal cortex at intensities above RCP. These findings indicate the influence of individual differences and may explain variability in the haemodynamic response, particularly potential hypofrontality effects at higher intensities of exercise.

Variability in the cerebral haemodynamic response may further be attributed to lack of standardised exercise intensities to individual metabolic markers (i.e. gas exchange thresholds). Furthermore, variability reported by Rooks et al. (2010) and the findings in study I may be attributed to different measurement regions in the right and left

hemispheres of the frontal cortex (not considered by Rooks et al., 2010; or examined in study I).

Firstly, Rooks et al. (2010) recognised the need for future research to standardise exercise intensities to metabolic markers as cerebral oxygenation has been shown to decrease at intensities of exercise associated with RCP (Bhambhani et al., 2007) or at least decrease in some individuals (as shown in study I). Secondly, the pattern of cerebral oxygenation in sub regions (in both hemispheres) of the frontal cortex (using multi-channel NIRS) during incremental exercise has not been examined.

The DMM (described in 2.1.3) explains that at intensities of exercise above VT (approximately 60% aerobic power; hard exercise according to Rooks et al., 2010); the frontal cortex has to work harder to compete with sensory (interoceptive) input from the body to promote a more positive affective response. The frontal cortex is theorised to maintain activation through cognitive control (see 2.3.1) and motivational processes (see 2.3.2) in order to promote a more positive affective response. However, changes in the cerebral haemodynamic response, particularly a reduction in oxygenation (i.e. blood flow) in the presence of increased deoxygenation (ΔHHb), would indicate reduced neuronal activation.

The transient hypofrontality hypothesis (Dietrich, 2003, 2006) explains why oxygenation status may decline in the frontal cortex during exercise (see 2.3.3) and provides a mechanism by which the DMM explains the regulation of affective responses during exercise (see 2.3.3). Transient hypofrontality would reduce the capacity of the frontal cortex to maintain cognitive processes during exercise and would impact affective responses. Additionally, in line with the transient hypofrontality hypothesis, different areas of the frontal cortex may become deregulated at different intensities of exercise which may further explain variability in the cerebral haemodynamic response reported previously.

The present study investigates activation in sub regions of the frontal cortex (reflected by changes in cerebral haemodynamics using multi-channel NIRS) and affective responses during incremental exercise to volitional exhaustion.

Three hypotheses were examined: 1) Activation in the frontal cortex is different in sub regions of the frontal cortex at intensities of exercise above VT; 2) Affective responses are more positive at exercise intensities corresponding to below VT and more negative at

exercise intensities corresponding to RCP and end of exercise; and 3) Activation in sub regions of the frontal cortex is associated with affective responses during exercise at intensities above VT.

5.2 Methods

Participants

Participants (n = 25; male = 13; age, M = 25.6, SD = 3.4 years; height, M = 174.8, SD = 6.8 cm; body mass, M = 71.8, SD = 8.3 kg) were recruited at a university campus.

Measures

Cerebral haemodynamics were measured in eight sub regions (1-8) of the frontal cortex using multi-channel NIRS as previously described (see 3.3.3.3). Approximate locations are shown in Figure 5.1. Affective responses (pleasure-displeasure) were recorded using the Feeling Scale (Hardy & Rejeski, 1989; see 3.2.1).



Figure 5.1. Approximate locations of sub regions (1-8) of the frontal cortex measured using multi-channel NIRS. Odd numbers identify superior and even numbers identify inferior regions of the frontal cortex.

Procedures

Upon arrival at the laboratory, participants were screened (i.e. completed the informed consent form and exercise testing questionnaire) and initial assessments were taken (see 3.3.1). Participants were seated on a recumbent cycle ergometer and the NIRS optodes

were carefully positioned (see 3.3.2) and a facemask (to measure metabolic data) was fitted. Participants completed an incremental exercise test to volitional exhaustion (20W/min) as previously described (see 3.3.3). Cerebral haemodynamics and expired gases were collected continuously and affective responses were recorded pre-, during (every minute) and at the end of exercise.

Data reduction

Following the identification of gas exchange thresholds (VT and RCP) and $\dot{V}O_{2peak}$ (see 3.4.1), primary (ΔO_2Hb and ΔHHb) and secondary (ΔtHb and $\Delta HbDiff$) cerebral haemodynamic variables were extracted at time points corresponding to intensities of exercise; 80% of VT (below VT), VT, RCP and End of exercise (see 3.4.3). Affective responses at time points corresponding to each of the intensities of exercise were selected.

Statistical analyses

The independent variables were Region (1-8), Time (corresponding to pre-exercise and intensities below VT, VT, RCP and End). The dependent variables were changes in cerebral haemodynamics (primary [ΔO_2Hb, ΔHHb] and secondary [ΔtHb, $\Delta HbDiff$]) and affective responses.

To examine hypothesis 1, a series of Region (8; 1-8) by Time (4; below VT, VT, RCP, End) analysis of variance (ANOVA) with repeated measures were conducted for each of the primary (ΔO_2Hb, ΔHHb) and secondary (ΔtHb, $\Delta HbDiff$) cerebral haemodynamic variables. To examine hypothesis 2, a one-way ANOVA with repeated measures for Time (5; pre-exercise, and at time points corresponding to below VT, VT, RCP and End) was conducted for affective responses. Finally, to examine hypothesis 3, Pearson's bivariate correlation analyses were conducted to examine the relationships between cerebral haemodynamics (ΔO_2Hb, ΔHHb) and affective responses during exercise.

5.3 Results

Participants' physiological data are reported in Table 5.1.

Table 5.1. Oxygen uptake (ml ·kg^{-1}·min^{-1}) at each time point and duration of exercise to exhaustion, *M* (*SD*).

		(*n* = 25)
$\dot{V}O_2$(ml ·kg^{-1}·min^{-1})	Below VT	22.8 (2.9)
	VT	28.5 (3.7)
	RCP	37.7 (4.6)
	End ($\dot{V}O_{2peak}$)	41.8 (5.2)
Exercise duration (sec)		641 (126)

5.3.1 Cerebral haemodynamics (hypothesis 1)

Cerebral ΔO_2Hb

Significant main effects of Region, $F(4, 105) = 3.67$, $p = .006$, $\eta_p^2 = .13$, and Time, $F(1, 27) = 16.21$, $p < .001$, $\eta_p^2 = .40$, and a Region by Time, $F(5, 123) = 4.54$, $p < .001$, $\eta_p^2 = .16$, interaction were recorded for ΔO_2Hb (μM). Follow up tests indicated that ΔO_2Hb was greater in Regions 2, 4, 6, 8 (inferior) than Regions 1, 3, 5, 7 (superior; see Table 5.2.). Cerebral ΔO_2Hb increased from below VT ($M = .35$, $SD = 1.65$) to VT ($M = 1.33$, $SD = 2.17$) and VT to RCP ($M = 4.60$, $SD = 5.03$), but remained stable RCP to End ($M = 4.90$, $SD = 5.78$).

The means and standard deviations (hypothesis 1) and correlation coefficients (hypothesis 3) of the cerebral haemodynamic variables (μM) in this study are provided in Appendix 3b.

Table 5.2. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex, *M* (*SD*). * Significantly greater ΔO_2Hb in inferior than superior regions.

Right hemisphere		*Left hemisphere*		
①	③	⑤	⑦	*Superior regions*
1.51 (3.86)	2.35 (4.45)	2.22 (3.38)	2.72 (3.09)	
②	④	⑥	⑧	*Inferior regions**
3.21 (3.79)	3.43 (3.65)	3.46 (3.33)	3.45 (3.68)	

The two factor interaction indicated that from RCP to End, ΔO_2Hb increased in Region 4 and declined in Region 1 (see Figure 5.2.).

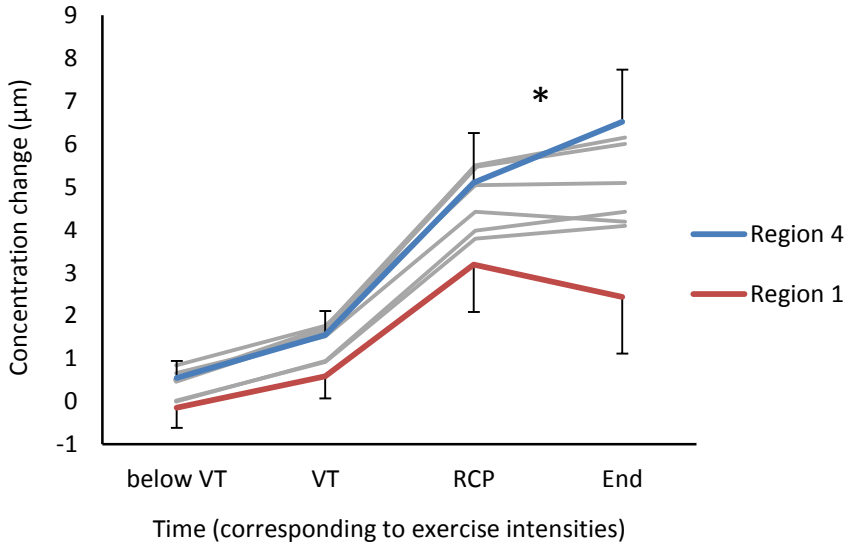


Figure 5.2. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex at each time point, *M* and *SEM*. * Significant increase of ΔO_2Hb in region 4 and decrease in region 3, than other regions (grey lines).

Cerebral ΔHHb

A significant main effect of Time, $F(1, 27) = 77.54$, $p < .001$, $\eta_p^2 = .76$, was recorded for ΔHHb (µM). Follow up tests indicated that ΔHHb remained stable from below VT (*M* =

.70, *SD* = .77) to VT (*M* = .87, *SD* = .76), then increased from VT to RCP (*M* = 2.57, *SD* = 1.47) and RCP to End (*M* = 4.30, *SD* = 2.03).

Cerebral ΔtHb

A significant main effect of Time, $F(1, 27) = 33.71$, $p < .001$, $\eta_p^2 = .58$, and a Region by Time, $F(5, 125) = 2.66$, $p = .024$, $\eta_p^2 = .10$, interaction were recorded for ΔtHb. Follow up tests indicated that ΔtHb increased from below VT (*M* = 1.06, *SD* = 1.70), to VT (*M* = 2.19, *SD* = 2.18), RCP (*M* = 7.17, *SD* = 5.73) and End (*M* = 9.20, *SD* = 6.64). The two factor interaction indicated that from RCP to End, ΔtHb increased in Region 4 and remained stable in Region 1 (see Figure 5.3.).

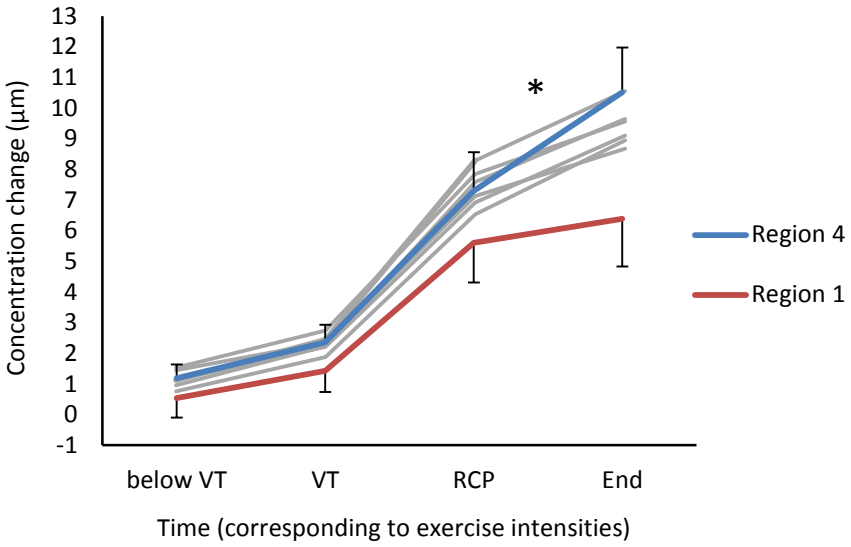


Figure 5.3. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex at each time point A Region by Time interaction for cerebral ΔtHb, *M* and *SEM*. * Significant increase in ΔtHb in Region 4 and a significantly lower ΔtHb in Region 1, than other Regions (grey lines).

Cerebral ΔHbDiff

A significant main effect of Region, $F(4, 91) = 3.28$, $p = .016$, $\eta_p^2 = .12$, and a Region by Time, $F(5, 120) = 5.39$, $p < .001$, $\eta_p^2 = .18$, interaction were recorded for ΔHbDiff (µM). Follow up tests indicated that ΔHbDiff was greater in Regions 2, 4, 6, 8 (inferior) than Regions 1, 3, 5, 7 (superior). *M* (*SD*)s are presented in Table 5.3.

Table 5.3. Cerebral deoxygenation (ΔHHb) in sub regions of the frontal cortex, *M* (*SD*).

* Significantly greater ΔHHb than other regions.

Right hemisphere		*Left hemisphere*		
①	③	⑤	⑦	*Superior regions*
-.46 (4.31)	-.09 (4.83)	-.09 (3.37)	.53 (3.18)	
②	④	⑥	⑧	*Inferior regions**
.99 (4.31)	1.52 (3.93)	1.82 (3.43)	1.29 (3.86)	

The two factor interaction indicated that from VT to RCP and RCP to End, ΔHbDiff increased and was greater in Regions 4 and 6, respectively, compared to Regions 1, 3, 5 and 7 (superior) (see Figure 5.4.).

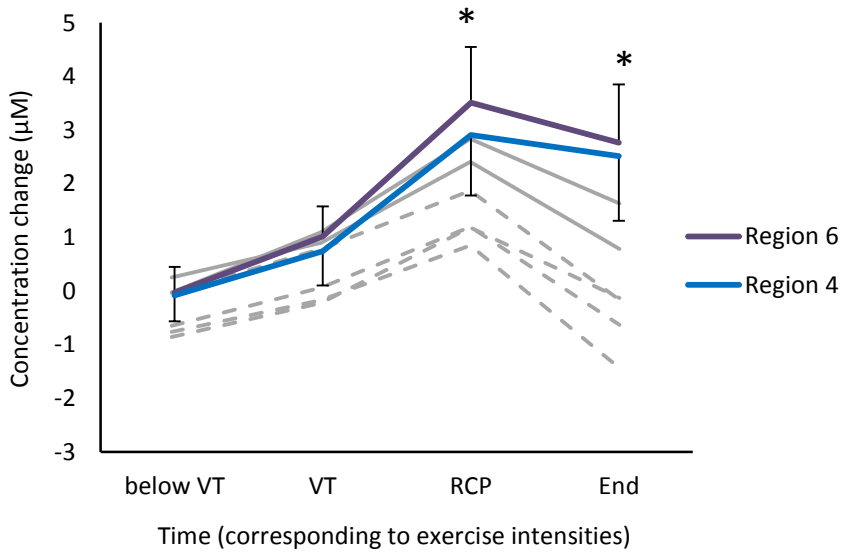


Figure 5.4. Cerebral oxygen saturation (ΔHbDiff) in sub regions of the frontal cortex at each time point, *M* and *SEM*. * Significantly greater ΔHbDiff in regions 4 and/or 6, than regions 1, 3, 5, 7 (dashed grey lines- regions 2 and 8 are indicated by the solid grey lines).

5.3.2 Affective responses (hypothesis 2)

A significant main effect of Time, $F(2, 39) = 68.63$, $p < .001$, $\eta_p^2 = .74$, was recorded for affective responses. Affective responses significantly declined from pre-exercise to below VT, remained stable until VT and significantly declined from VT to RCP and RCP to the end of exercise (see Figure 5.5).

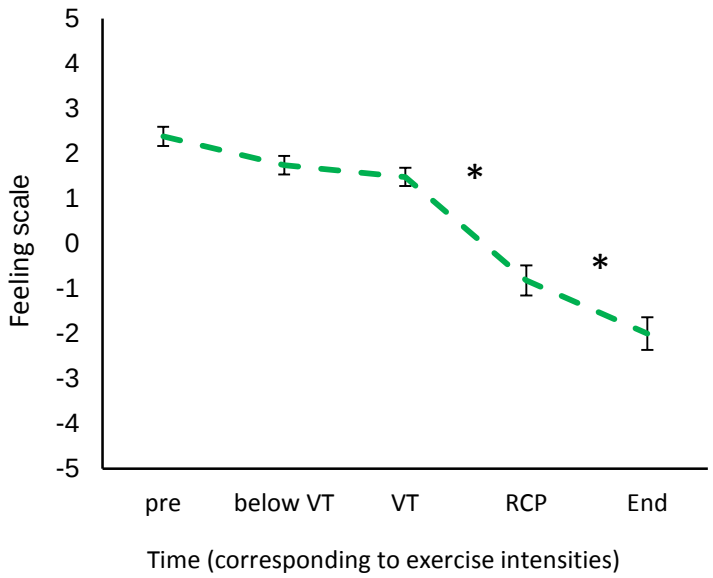


Figure 5.5. Affective responses reported at each time point, *M* and *SEM*.

* Significant decline in affective responses between intensities.

5.3.3 Correlations (hypothesis 3)

Cerebral haemodynamics and affective responses (hypothesis 3)

Exercise below VT and VT

No significant relationships were shown between affective responses and ΔO_2Hb and ΔHHb in any region of the frontal cortex. Correlations ranged between, $r = .30$ and -.37.

Exercise at RCP

Significant inverse relationships were shown between affective responses and ΔO_2Hb in Region 1, $r = -.55$, $p = .004$ and Region 2, $r = -.53$, $p = .007$, in the right hemisphere; and in

all Regions (5-8) in the left hemisphere which ranged between, r = -.50 and -.62, p's < .011. Region 7 indicated the greatest correlation. No significant relationships were recorded for ΔHHb.

Exercise at End (exhaustion)

Significant inverse relationships were shown between affective responses and ΔO_2Hb in Region 4, r = -.46, p = .021, in the right hemisphere; and in all Regions (5-8) in the left hemisphere which ranged between, r = -.45 and -.56, p's < .022. No significant relationships were recorded for ΔHHb.

5.4 Discussion

The present study investigated activation in sub regions of the frontal cortex using multi-channel NIRS during incremental exercise to volitional exhaustion. The pattern of the cerebral haemodynamic response was examined at intensities of exercise standardised to individual gas exchange thresholds (Ekkekakis, 2009; Rooks et al., 2010) and data extracted at time points corresponding to below VT, VT, RCP and end of exercise (exhaustion). The influence of activation of the frontal cortex and affective responses during exercise were explored.

The Rooks et al. (2010) meta-analysis indicated variability in oxygenation (ΔO_2Hb) during exercise at sub-maximal and maximal intensities. The authors attributed some of this variability to the individual's training status ($\dot{V}O_{2peak}$), reported by Rooks et al. (2010) to have an average $\dot{V}O_{2peak}$ (M = 40.3, SD = 10.4 ml ·kg^{-1}·min^{-1}) similar to that reported in the present study (M = 41.8, SD = 5.2 ml ·kg^{-1}·min^{-1}; although much greater variability in Rooks et al.). The findings from study I suggest that variability in the haemodynamic response can also attributed to individual differences (i.e. self-reported tolerance) at intensities above RCP.

This study examined the haemodynamic response from eight sub regions (in the left and right hemispheres) of the frontal cortex. It was proposed that measuring haemodynamics during exercise standardized to individual metabolic processes and from multiple regions of the frontal cortex would help to explain variability in the haemodynamic response (i.e.

oxygenation; ΔO_2Hb) as identified by Rooks et al. (2010). Furthermore, in line with the transient hypofrontality hypothesis (Dietrich, 2003, 2006) it was proposed that a deregulation may occur in different regions of the frontal cortex during exercise at different intensities.

At intensities of exercise below VT, oxygenation (ΔO_2Hb) increased, resulting in an increase in blood volume (ΔtHb) and saturation (ΔHbDiff) in all regions of the frontal cortex. However, oxygen extraction (deoxyhaemoglobin; ΔHHb) remained stable. Therefore the frontal cortex appeared to be adequately supplied with oxygen relative to the demand. According to the DMM, exercise at these intensities does not induce sufficient sensory input from the body to challenge the individual; therefore cognitive processes remain dominant over subcortical processes receiving sensory input from the body. A similar response was observed at these intensities in study one (in both the high and low tolerance groups).

At intensities of exercise at VT, greater oxygenation (ΔO_2Hb) and saturation (ΔHbDiff) in bilateral inferior regions (2, 4, 6, 8) were observed. As the intensity of exercise increased from VT to RCP, oxygenation (ΔO_2Hb) and deoxygenation (ΔHHb) increased in all regions, resulting in an increase in blood volume (ΔtHb) and saturation (ΔHbDiff). However, greater oxygenation (ΔO_2Hb) and saturation (ΔHbDiff) remained in the inferior regions compared to all superior regions. According to the transient hypofrontality hypothesis (Dietrich 2003, 2006), the first stage of deregulation occurs in the dorsolateral region of the frontal cortex (corresponding to the superior regions measured). The haemodynamic response was greater in the inferior regions at these intensities of exercise indicating a reduced activation in the left and right dorsolateral prefrontal regions.

At intensities of exercise from RCP to end of exercise (exhaustion), oxygenation (ΔO_2Hb) continued to increase in inferior regions (greatest in region 4 followed by regions 6 and 8), but remained stable or declined in superior regions. Deoxygenation (ΔHHb) continued to increase in all regions and blood volume (ΔtHb) increased in most regions except the most lateral area in the right dorsolateral region of the frontal cortex (region 1). The increase in blood volume (sum of ΔO_2Hb and ΔHHb) indicates activation in the inferior regions and reduced activation in the superior regions at these intensities. This is because the inferior regions showed an increase in oxygenation (ΔO_2Hb) coupled with an increase in extraction (ΔHHb; therefore adequate oxygen supply for demand) indicating activation. However, the

superior regions showed a decline or plateau in oxygenation (ΔO_2Hb) coupled with an increased extraction (ΔHHb: therefore lack of oxygen supply to demand). This is supported by greater saturation (ΔO_2Hb minus ΔHHb) in inferior regions compared to superior regions at these intensities. Activation in bilateral inferior regions located closest to the midline (regions 4 and 6; the most anterior portion of the frontal cortex) indicated the greatest activation at the end of exercise (exhaustion).

The transient hypofrontality hypothesis describes that the second stage of deregulation occurs in the ventromedial prefrontal cortex. Although this area was not directly measured in this study, it lays beneath the anterior frontal cortex (i.e. regions 4 and 6). It is hypothesized that the increase in activation in the anterior frontal cortex (regions 4 and 6) at exhaustion are due to the shift in metabolic resources from superior regions (regions 1,3, 5 and 7; stage one) towards ventromedial regions (located beneath regions 4 and 6; stage two) as the intensity of exercise increased. Therefore, the cerebral haemodynamic response appeared to shift in a top-down direction from VT to RCP, then outside-in (lateral to midline) at intensities from RCP to end of exercise as proposed by the transient hypofrontality hypothesis.

The different patterns of the cerebral haemodynamic response in sub regions in the frontal cortex as the intensity of exercise increases may explain some of the variability previously reported. Rooks et al. (2010) indicated that there was variability in the degree of activation (oxygenation) at intensities of exercise >60% aerobic power and near exhaustion. As recommended by the authors, the intensities of exercise in this study were standardized to individual metabolic markers. Using multi-channel NIRS different patterns of the cerebral haemodynamic response were observed in the different sub regions measured (i.e. superior regions indicate less activation [reduced ΔO_2Hb] than inferior regions) and between hemispheres (i.e. greater ΔO_2Hb in region 4). These findings indicate that variability in the cerebral haemodynamic response may be due to regions the haemodynamic response is measured using NIRS. Methodological considerations of optode placement between different studies should be acknowledged and the effects of incremental versus constant-load exercise protocols. Study I indicated self-reported tolerance contributed to variability in the haemodynamic response therefore future work should further examine other potential influencing factors of haemodynamic variability i.e. gender, population, exercise protocol and mode etc.

Evidence for the transient hypofrontality hypothesis provides support to the DMM, which indicates that as the intensity of exercise becomes more challenging, the frontal cortex must compete with subcortical areas (i.e the amygdala) of the brain to maintain functional capacity. The ability of the frontal cortex to remain active is proposed by the DMM to influence affective responses through cognitive control (see 2.3.1) and motivational processes (see 2.3.2). The pattern of affective responses recorded is in line with the dose-response relationship and the intensity of exercise (Ekkekakis et al., 2011). Despite a decline in affective responses from pre-exercise to intensities below VT, affective responses remained positive until intensities above VT (hypothesis 2). The frontal cortex appeared to be adequately saturated at these intensities. However, at RCP and the end of exercise (exhaustion) affective responses were uniformly negative.

Study I indicated an inverse association between deoxygenation (ΔHHb) and the decline in affective responses at intensities above VT. In this study, cerebral oxygenation (ΔO_2Hb) was inversely associated with affective responses at intensities above VT (i.e. RCP and End). Greater cerebral oxygenation (ΔO_2Hb) in all regions of the left hemisphere was associated with a more negative affective response at intensities of exercise at the RCP and the end of exercise. Greater activation in the left hemisphere and the presence of a negative affective response may reflect cognitive control processes involved in attempts to maintain and continue exercise (see 2.3.1). The increase in activation in the left hemisphere would indicate approach-type motivation in line with Davidson's valence asymmetry hypothesis (1993). However, approach-type motivation is typically associated with a more positive affect (see 2.3.2). These findings indicate that activation in the left frontal cortex is potentially involved in approach-type motivation in the presence of negative affective responses, presumably through cognitive control processes towards a more positive or less negative affective response (Davidson, Putnam & Larson, 2000). Inverse correlations were also observed between oxygenation (ΔO_2Hb) in some regions (1 and 2) of the right hemisphere and affective responses (as observed in study I).

At intensities of exercise at RCP, a more negative affective response was associated with greater cerebral oxygenation in the most lateral (inferior-superior) regions of the right hemisphere, but not those closest to the midline. At the end of exercise, only the inferior region closest to the midline in the right hemisphere (region 4) showed an inverse relationship between cerebral oxygenation (ΔO_2Hb) and affective responses. Although

some caution should be taken in the actual significance of these moderate linear correlations, the association between oxygenation (ΔO_2Hb) in both hemispheres, but particularly in the right hemisphere at intensities above VT, is interesting.

This study extended findings from study I to determine variability in the haemodynamic response to during exercise which may influence the regulation of affective responses. This study showed that cerebral activation shifts in a top-down manner from superior to inferior regions, with greatest activation in regions closest to the midline at the end of exercise. Consistent associations between oxygenation in the left hemisphere and affective responses were observed at intensities above VT. The association between oxygenation and affective responses in the right hemisphere from the most lateral regions to the midline (inferior regions) was observed between intensities at RCP and end of exercise. These findings provide some support for the DMM and the hypofrontality hypothesis and contribute to an understanding of the neural basis of affective responses to exercise.

Chapter 6

Study III: Cerebral blood flow in the frontal cortex during exercise: An examination of a cognitive strategy (imagery)

The previous chapters (studies I and II) showed differential and asymmetrical activation in regions of the frontal cortex during incremental exercise to exhaustion. The findings showed that these differential activation patterns occurred (between sub regions and hemispheres) at intensities of exercise above VT. Furthermore, affective responses were associated with activation in the frontal cortex at these intensities.

The following two chapters examine the influence of a cognitive strategy (study III) and increased sensory (interoceptive) input (using prior exhaustive activity; study IV) upon frontal cortex activation and affective responses, during constant-load exercise at intensities above VT.

The purpose of these studies was to emphasise the influence of the cognitive (III) and interoceptive (IV) modes of the DMM to understand their impact upon affective responses to exercise.

This study formed the basis of an experimental paper published in the *Journal of Sport and Exercise Psychology:* Tempest, G., Parfitt, G. (2013). Imagery use and affective responses during exercise: An examination of cerebral haemodynamics using Near Infrared Spectroscopy, *35,* 503-513.

6.1. Background

In line with the previous studies and literature (see 2.1.2) affective responses during exercise become less positive or negative at intensities of exercise above VT (Ekkekakis, 2011). According to the DMM (see 2.1.2) cognitive processes in the frontal cortex become challenged by the increased sensory input from the body at intensities above VT which results in a negative affective state. Studies I and II reported for the first time changes in the cerebral haemodynamic response at these intensities which may influence affective responses during incremental exercise to exhaustion. These data suggest that maintenance of frontal cortex activation at intensities above VT could allow a more positive affective response to be maintained (see 2.3). Theoretically, a cognitive or psychological strategy could have the potential to promote a positive affective responses (increase feelings of pleasure) during exercise at intensities where responses would otherwise be negative (Blanchard, Rodgers & Gauvin, 2004; Lind, Welch & Ekkekakis, 2009; Salmon, Hanneman & Harwood, 2010); although the effects of such strategies upon activation in the frontal cortex during exercise has not been examined.

Imagery is a cognitive strategy that involves recreating experiences using information from all the senses i.e. sight, sound, smell, and touch, stored in the memory (Weinberg, 2008). During exercise, individuals might image themselves doing a specific activity (e.g. exercising on a bike) and focus on their senses (e.g. the sound of the wheel spinning, the feeling of their legs pumping) to achieve a specific goal for that activity (e.g. to complete a certain distance). Frequent exercisers have reported using imagery techniques more than less-frequent exercisers for cognitive and motivational purposes i.e. goal attainment and psychological management (Hall, 1995; Hausenblas, Hall, Rodgers & Munroe, 1999).

The use of imagery techniques during and following moderate intensity exercise (50% HRR) has recently been shown to improve affective responses (Stanley & Cumming 2010a; 2010b). Based upon Lang's (1979) bioinformational theory of emotional imagery, Stanley and Cumming (2010a) examined the effectiveness of technique, enjoyment and energy focused imagery scripts. Following exercise, affective responses were significantly more positive in participants who used enjoyment focused scripts and reductions in exhaustion were marginally decreased in participants who used energy focused scripts (Stanley & Cumming, 2010a). These results support the use of imagery as a suitable cognitive strategy

during exercise to help promote a more positive or pleasurable experience. However, the findings from Stanley & Cumming (2010a) are based upon moderate intensity (i.e. below VT) exercise where affective responses are frequently reported to be mostly positive (Ekkekakis et al., 2011) and the exercise intensity (50% HRR) was not standardised to individual metabolic markers i.e. gas exchange thresholds (GET).

A cognitively-driven strategy such as imagery should engage and maintain frontal cortex activation during exercise at intensities which would otherwise induce a negative affective state (i.e. above VT), and result in a more positive (or less negative) affective response through a top-down regulatory process.

Previous research has used NIRS to study parieto-occipital areas of the cortex to investigate the affective perception of imagery (Köchel et al., 2011) and in combination with electroencephalographic (EEG) methods (Herrmann et al., 2008). This research has successfully replicated findings investigating visual imagery and affective perception using fMRI methods (Schienle, Schäfer, & Vaitl, 2008; Schienle, Schäfer, Pignanelli, & Vaitl, 2009). In recent fMRI work, participants used imagery (kinetic and visual) to imagine themselves walking. This resulted in activation in fronto-parietal regions, specifically the right and left dorsolateral prefrontal cortices and the anterior insula (Cremers, Dessoullieres & Garraux, 2012; van der Meulen, Allali, Rieger, Assal & Vuilleumier, 2012). However, no studies have used NIRS to examine areas of the frontal cortex and imagery use and affective responses to exercise.

The present study investigates if imagery use influences activation (reflected by changes in haemodynamics using NIRS) in the dorsolateral region of the frontal cortex (both right and left hemispheres) and affective responses during constant-load exercise at intensities (standardised to GETs) 5% above VT (above VT) and RCP to end of exercise (exhaustion)

Three hypotheses were examined: 1) Activation of the frontal cortex is greater in the imagery than in the control condition; 2) Affective responses are (a) more positive at exercise intensities above VT than at exercise intensities RCP to exhaustion; and (b) more positive in the imagery than in the control condition; and 3) Activation of the frontal cortex is associated with affective responses to exercise and imagery use.

6.2 Methods

Participants

Participants (n = 14; male = 7; age, M = 21.8, SD = 2.2 years; height, M = 173.9, SD = 9.7 cm; body mass, M = 73.2, SD =18.2 kg) were recruited from a university campus.

Measures

Cerebral haemodynamics in the left and right dorsolateral region of the prefrontal cortex were measured using dual-channel NIRS as previously described (see 3.3.3.2). Approximate locations of the regions (1-2; right and left hemispheres) measured are shown in Figure 6.1. Affective responses (pleasure-displeasure) were recorded using the Feeling Scale (Hardy & Rejeski, 1989; see 3.2.1).

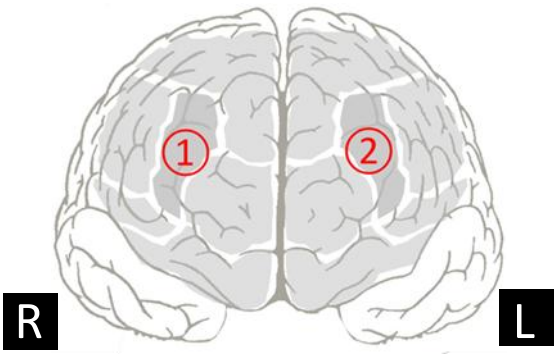


Figure 6.1. Approximate locations of the dorsolateral regions (1-2; right and left hemispheres) of the frontal cortex measured using dual-channel NIRS.

Imagery Script

The imagery script combined enjoyment and energy focused imagery in line with previous work by Stanley and Cumming (2010a, 2010b). The authors showed that enjoyment focused imagery improved individual's affective responses as it provided the necessary response propositions for a more positive feeling state during exercise at moderate intensities (below VT). Energy focused imagery, despite having response propositions to increase energy levels thought to impact motivational and emotional responses, did not increase a more positive feeling state any more than imagery without affective content (i.e. technique imagery). However, energy focused imagery did appear to decrease feelings of

physical exhaustion which would be beneficial for improving feeling states, particularly at higher intensities of exercise (above VT). Therefore, the combination of enjoyment and energy focused imagery was used as it should positively influence affective responses during exercise at intensities above VT.

The imagery script was read to each participant by the experimenter (lasting approximately 5 mins) as they were seated comfortably in a quiet room outside the exercise laboratory. The script required participants to recall a previous exercise situation when they had found exercise enjoyable and when they had felt energised. The use of their own personal experience has been shown to be more effective that using a standardised or generic imagery script (Lang, 1979; Wilson, Smith, Burden & Holmes, 2010). Participants were asked to recreate the feelings and to imagine their previous experience as vividly as possible (e.g. sounds, colours, smells, and people). After recalling how pleasurable and energised they felt, they were instructed to use these memories in the exercise they were about to perform.

The participants were then seated on the recumbent cycle ergometer in the exercise laboratory and the script was read to the participant by the experimenter once again. Excerpts of the imagery scripts were read to the participants every 2-3 minutes to keep the participants focused on feelings of enjoyment and energy during exercise. In the control condition, participants received no instructions or prompts during exercise and sat comfortably in the quiet room before exercise in place of the imagery script. To check participant's perceived ability to use imagery and to evaluate their ease of use and the effect of the imagery script during exercise, participants were asked to complete a post-exercise imagery script evaluation (see Stanley & Cumming, 2010a). The evaluation uses a 7-point Likert scale to provide a rating of participant's visual and kinetic abilities (how well the image of exercise was created and how strongly they felt could feel the action of the imagined exercise, respectively), clarity, emotion, energy and enjoyment evoked from the imagery script. The evaluation scores were used as an indicator of compliance to and effectiveness of the imagery techniques. The relationship between post-exercise imagery scores and cerebral haemodynamics in the imagery condition was examined (hypothesis 3).

Procedures

This study employed a crossover design and required participants to visit the laboratory on three occasions. Each participant completed a control and experimental (i.e. imagery) condition to examine the effects of imagery upon cerebral haemodynamic and affective responses. On the first visit, participants were screened and initial assessments were taken (see 3.3.1). Participants were seated on a recumbent cycle ergometer and a facemask (to measure metabolic data) was fitted. Participants completed an incremental exercise test to volitional exhaustion (20W/min) as previously described (see 3.3.3). Gas exchange thresholds (VT and RCP) and $\dot{V}O_{2peak}$ were identified (see 3.4.1) and workloads corresponding to exercise intensities were determined (see 3.4.2).

The participants returned for a further two sessions; imagery and control (in a randomised, counterbalanced order). Participants were seated on a recumbent cycle ergometer and the NIRS optodes were carefully positioned (see 3.3.2) and a facemask (to measure metabolic data) was fitted. Following a three minute warm up (50 W), both sessions involved exercising for 10 minutes at a workload corresponding to 5% above VT followed by 10 minutes passive recovery, and then exercise from RCP to exhaustion. A 10 minute duration of exercise at the above VT intensity was chosen to ensure all participants would complete the exercise. Participants reported their affective responses pre- and post-exercise, and at two minute intervals during exercise.

Participants workloads and $\dot{V}O_2$ (l/min) as a percentage of $\dot{V}O_{2peak}$ corresponding to intensities above VT were, *M* =108 W (*SD* = 21) and *M* =55.3 % (*SD* = 2.6), respectively, and RCP were, *M* = 177 W (*SD* = 32) and *M* = 84.5 % (*SD* = 2.7), respectively.

Data Reduction

To standardise data points between participants and enable analysis of the Intensity (above VT and RCP to exhaustion) and Time (selected points during exercise above VT and RCP to exhaustion) factors, percentage duration of exercise was used. The primary (ΔO_2Hb and ΔHHb) and secondary (ΔtHb and ΔHbDiff) cerebral haemodynamic variables were time-aligned with the gas data and 20 second averages were extracted from the associated GETs; point of 5% above VT and RCP, followed by 20, 40, 60, 80 and 100% duration of exercise. The duration of exercise at intensities above VT was 10 minutes, and the mean from RCP

to exhaustion was 9.35 min (*SD* = 5.47) in the imagery and 8.43 min (*SD* = 4.02) in the control condition. Affective responses were expressed as a percentage of the duration of exercise to include pre- and post-exercise measures at intensities above VT and RCP to exhaustion; pre-, GET, 20, 60, 100%, post-exercise.

Statistical Analyses

The independent variables were Condition (imagery, control), Region (right, left), Time (corresponding to pre-exercise, GETs [VT and RCP] and 20-100% duration of exercise) and Intensity (above VT, RCP). The dependent variables were changes in primary (ΔO_2Hb, ΔHHb) and secondary (ΔtHb, ΔHbDiff) cerebral haemodynamics, affective responses and scores from the post-exercise imagery evaluation.

To examine hypothesis 1, separate Condition (2; imagery, control) by Region (2; right, left) by Time (6; GET, 20, 40, 60, 80, 100%) analyses of variance (ANOVA) with repeated measures were conducted on each of the primary (ΔO_2Hb, ΔHHb) and secondary (ΔtHb, ΔHbDiff) NIRS variables at each of the exercise intensities; above VT and RCP to exhaustion. To examine hypothesis 2, a Condition (2; imagery, control) by Time (6; pre-, GET, 20, 60, 100%, post-exercise) by Intensity (2; above VT, RCP to exhaustion) ANOVA with repeated measures was conducted on the affective responses. Finally, to examine hypothesis 3, Pearson's bivariate correlation analysis was used to examine the relationships between cerebral haemodynamics (ΔO_2Hb, ΔHHb), affective responses and post-exercise imagery evaluation scores (imagery condition only).

6.3 Results

6.3.1 Preliminary Analyses

To ensure the intensity of exercise (indicated by $\dot{V}O_2$ l/min) was the same between the two exercise sessions (imagery, control), and that these were not different to the initial incremental exercise test, a Session (3) by Intensity (3) ANOVA with repeated measures was conducted. In this analysis, three intensities were compared; 5% above VT, RCP and End (exhaustion). The analysis showed that $\dot{V}O_2$ (l/min) at 5% above VT (*M* = 1.71, *SD* = 0.38), RCP (*M* = 2.49, *SD* = 0.56) and End (*M* = 2.93, *SD* = 0.65) were significantly different, $F(1, 18) = 206.58$, $p < .001$, $\eta_p^2 = .94$. No significant main effect of Session, *F*(2,

26) = .18, p >.05, η_p^2 = .01, or Session by Intensity interaction, $F(4, 52) = .62$, $p > .05$, η_p^2 = .05, were recorded.

The post-exercise imagery evaluation scores indicated participants visual (M = 5.8, SD = 1.1) and kinetic ability (M = 5.1, SD = 1.1), clarity (M = 5.6, SD = 0.8), and emotion (M = 5.5, SD = 0.7), energy (M = 5.2, SD = 1.5) and enjoyment (M = 4.6, SD = 1.4) evoked from the script.

6.3.2 Cerebral haemodynamics (hypothesis 1)

Exercise above VT

Cerebral ΔO_2Hb

A significant main effect of Time, $F(1, 18) = 21.76$, $p < .001$, $\eta_p^2 = .63$, was recorded for ΔO_2Hb (μM). Follow up tests indicated that ΔO_2Hb increased from GET to 100% duration of exercise (see Table 6.1).

Cerebral ΔHHb

A significant main effect of Condition, $F(1, 13) = 14.70$, $p = .002$, $\eta_p^2 = .53$, was recorded for ΔHHb (μM). Follow up tests indicated greater ΔHHb in the imagery (M = 0.02, SD = 0.97) than the control (M = -1.07, SD = 1.41) condition.

Cerebral ΔtHb

A significant main effect of Time, $F(1, 17) = 21.90$, $p < .001$, $\eta_p^2 = .63$, was recorded for ΔtHb (μM). Follow up tests indicated an increase of ΔtHb from GET to 100% duration of exercise (see Table 6.1).

The means and standard deviations (hypothesis 1) and correlation coefficients (hypothesis 3) of the cerebral haemodynamic variables (μM) in this study are provided in Appendix 3c.

Cerebral ΔHbDiff

Significant main effects of Condition, $F(1, 13) = 7.45$, $p = .017$, $\eta_p^2 = .36$, and Time, $F(1, 19) = 19.93$, $p < .001$, $\eta_p^2 = .61$, were recorded for ΔHbDiff (μM). Follow up tests indicated less ΔHbDiff in the imagery ($M = 5.09$, $SD = 3.32$) than the control ($M = 7.48$, $SD = 3.08$) condition. Cerebral ΔHbDiff increased from GET to 100% duration of exercise (see Table 6.1).

Table 6.1. Cerebral haemodynamics (ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff) at each time point during exercise above VT, *M* (*SD*).

* Significant at each time point.

NIRS Measure (μM)	Time (corresponding to GET and 20-100% duration of exercise)					
	GET	20	40	60	80	100
ΔO_2Hb *	3.54 (2.81)	4.55 (3.32)	5.63 (3.25)	6.23 (3.59)	7.07 (3.68)	7.52 (4.01)
ΔtHb *	3.17 (2.77)	4.11 (3.43)	5.09 (3.36)	5.65 (3.64)	6.50 (3.74)	6.85 (4.00)
ΔHbDiff *	3.90 (2.35)	4.99 (2.90)	6.17 (2.85)	6.81 (3.14)	7.64 (3.23)	8.19 (3.43)

Exercise RCP to exhaustion

Cerebral ΔO_2Hb

A significant main effect of Time, $F(1, 17) = 16.29$, $p < .001$, $\eta_p^2 = .56$, was recorded for ΔO_2Hb (μM). Follow up tests indicated that ΔO_2Hb increased from GET to 80%, and remained stable from 80% to 100% duration of exercise (see Table 6.2).

Cerebral ΔHHb

A significant main effect of Time, $F(2, 21) = 16.61$, $p < .001$, $\eta_p^2 = .55$, was recorded for ΔHHb (μM). Follow up tests indicated that ΔHHb increased from GET to 100% duration of exercise (see Table 6.2).

Cerebral ΔtHb

A significant main effect of Time, $F(1, 17) = 28.98$, $p < .001$, $\eta_p^2 = .69$, and a significant Region by Time, $F(3, 33) = 3.74$, $p = .026$, $\eta_p^2 = .22$, interaction were recorded for ΔtHb (μM). Follow up tests indicated that ΔtHb increased from GET to 100% duration of exercise, with a greater increase of ΔtHb in the left compared to the right hemisphere between 40 and 60% duration of exercise (see Table 6.2).

Cerebral ΔHbDiff

A significant main effect of Time, $F(1, 17) = 6.48$, $p = .015$, $\eta_p^2 = .33$, was recorded for ΔHbDiff (μM). Follow up tests indicated ΔHbDiff increased from GET to 100% duration of exercise (see Table 6.2).

Table 6.2. Cerebral haemodynamics (ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff) at each time point during exercise at RCP to exhaustion, *M* (*SD*). R = region (right) and L = region (left). [a] = Main effect of Time; [b] = Region by Time Interaction.

NIRS Measure (μM)	Time (corresponding to GET and 20-100% duration of exercise)					
	GET	20	40	60	80	100
ΔO_2Hb [a]	2.65 (3.87)	4.80 (4.46)	5.96 (4.64)	6.81 (4.91)	7.44 (5.26)	7.69 (5.55)
ΔHHb [a]	1.87 (2.24)	2.36 (2.57)	2.69 (2.67)	2.98 (2.78)	3.22 (2.94)	3.44 (3.15)
ΔtHb [a]	4.52 (3.13)	7.16 (3.65)	8.65 (3.70)	9.79 (3.82)	10.65 (4.04)	11.14 (4.17)
ΔHbDiff [a]	.78 (5.15)	2.45 (5.87)	3.27 (6.09)	2.83 (6.43)	4.22 (6.98)	4.25 (7.35)
ΔtHb-R [b]	4.51 (2.69)	7.12 (3.00)	8.50 (3.01)	9.38 (3.02)	10.39 (3.35)	10.81 (3.13)
ΔtHb-L [b]	4.53 (2.76)	7.20 (3.41)	8.81 (3.42)	10.19 (3.50)	10.92 (3.76)	11.46 (3.95)

6.3.3 Affective responses (hypothesis 2)

Significant main effects of Condition, $F(1, 13) = 29.06$, $p < .001$, $\eta_p^2 = .69$, Time, $F(1, 19) = 54.42$, $p < .001$, $\eta_p^2 = .81$, and Intensity, $F(1, 13) = 61.88$, $p < .001$, $\eta_p^2 = .83$, and significant Condition by Time, $F(2, 32) = 9.10$, $p < .001$, $\eta_p^2 = .41$, and Time by Intensity,

$F(2, 26) = 38.17$, $p < .001$, $\eta_p^2 = .75$, interactions were recorded for affective responses. Follow up tests indicated that affective responses were more positive in the imagery (M = 2.3, SD = 1.4) than the control condition (M = 1.2, SD = 1.8), and declined at each time point (pre-, M = 3.86, SD = .20; GET, M = 1.96, SD = .33; 20%, M = 1.09, SD = .39; 40%, M = .36, SD = .45; 100%, M = -.05, SD = .43; post-exercise, M = 3.09, SD = .19) between conditions and intensities. Affective responses were more positive at intensities above VT (M = 3.2, SD = 1.3) than RCP to exhaustion (M = 0.2, SD = 1.6). The Condition by Time interaction indicated that relative to the imagery condition, there was a greater decline in affective responses in the control from pre- to 100% exercise duration (see Figure 6.2).

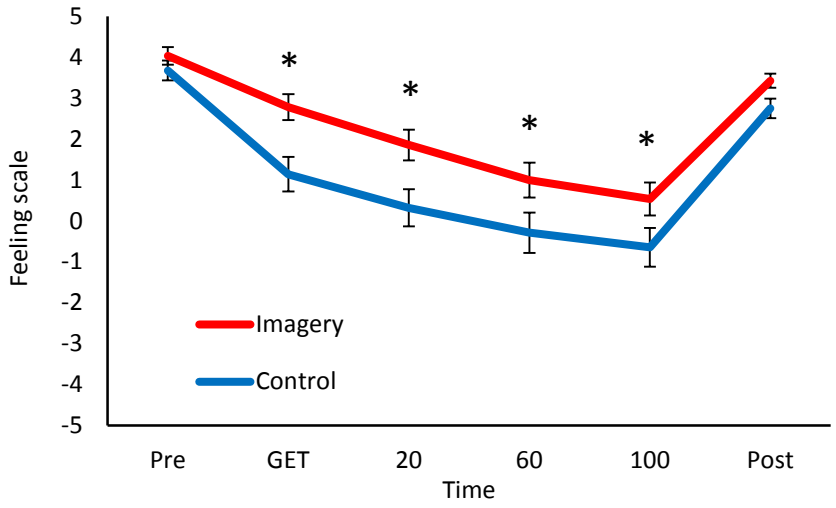


Figure 6.2. Affective responses reported at each time point in the imagery and control conditions, *M* and *SEM*. * Significantly more positive affective responses in the imagery condition.

The Time by Intensity interaction indicated that affective responses were stable pre-, during and post-exercise at intensities above VT, but became more negative from pre-exercise to 100% of the duration at intensities RCP to exhaustion (see Figure 6.3).

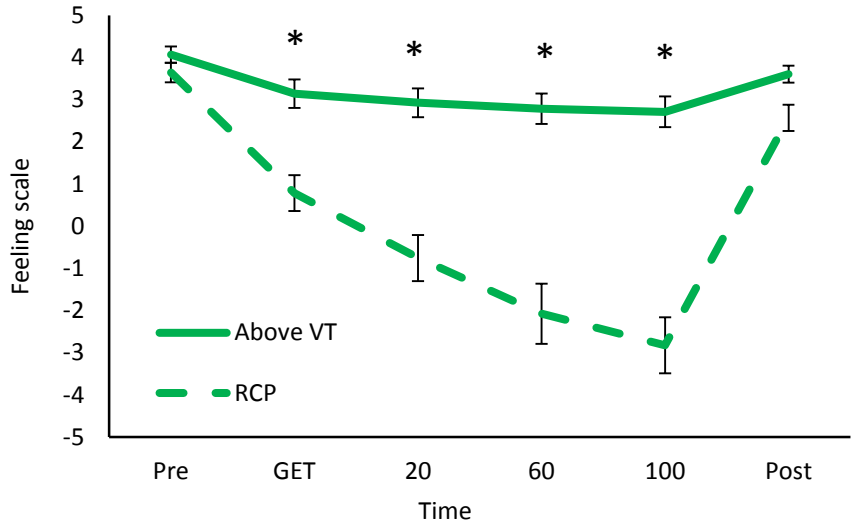


Figure 6.3. Affective responses reported at each time point during exercise at intensities above VT and at RCP to exhaustion, *M* and *SEM*. * Significantly more positive affective responses at intensities above VT.

6.3.4 Correlations (hypothesis 3)

Imagery use, cerebral haemodynamics and affective responses during exercise (hypothesis 3)

Exercise intensities above VT, cerebral ΔHHb in the right region and affective valence at 100% duration of exercise indicated a significant inverse relationship in the imagery, $r = -.55$, $p = .043$, but not the control condition, $r = .06$, $p > .05$.

The post-exercise imagery evaluation scores of visual and kinetic ability and enjoyment, revealed positive relationships with cerebral ΔO_2Hb and ΔHHb in the right and left regions which ranged between, $r = .26$ and .68. Significant relationships were shown between cerebral ΔHHb and visual ability, $r = .56$, $p = .036$, and kinetic ability, $r = .55$, $p = .039$, in the left region. The enjoyment evoked from the imagery script was positively associated with cerebral ΔO_2Hb, $r = .60$, $p = .023$, in the left region. There were no significant correlations between post-exercise imagery evaluation scores and cerebral haemodynamics at intensities of exercise from RCP to exhaustion.

6.4 Discussion

The present study investigated the influence of enjoyment and energy focused imagery on activation of the frontal cortex and affective responses during exercise at standardised intensities; above VT and RCP to exhaustion. Imagery as a cognitive strategy should promote a more pleasurable exercise experience at intensities that may otherwise lead to displeasure by maintaining activation in the frontal cortex.

Participants were able to use imagery as indicated by the post-exercise imagery evaluation scores (with all means over the midpoint of the scale), which were similar to scores reported in previous work (Stanley & Cumming, 2010a).

Functional activation of the frontal cortex (approximately within the dorsolateral region) reflects cognitive processes and provides some support for hypothesis 1. At intensities of exercise above VT, cerebral haemodynamics indicated greater deoxygenation (ΔHHb; i.e. oxygen extraction) and a trend of lower oxygenation (ΔO_2Hb, $p = .094$, $\eta_p^2 = .20$) across the exercise duration in the imagery than in the control condition. Oxygen saturation (ΔHbDiff) was lower due to imagery use. Despite a similar increase in blood volume (ΔtHb) in both of the conditions, the patterning of oxygenation (ΔO_2Hb) and deoxygenation (ΔHHb) indicated increased activation of the frontal cortex in the imagery condition. At exercise intensities from RCP to exhaustion, no differences between the imagery and control conditions were observed. These findings indicate differential activation within the region of the dorsolateral frontal cortex due to imagery use during exercise at a physiologically challenging intensity (above VT). However at higher exercise intensities (RCP to exhaustion), imagery (as a cognitive strategy) had little influence on frontal cortex activity as there were no differences in cerebral haemodynamics similar to those observed at intensities above VT.

In line with the DMM, imagery use may have served to engage and maintain activity of the frontal cortex to oppose the potentially aversive state induced by increases in exercise intensity which influenced the regulation of affective responses. Consistent with evidence towards the dose-response pattern of affective responses and the intensity of exercise (see Ekkekakis et al., 2011), affective responses at exercise intensities above VT were more positive than those from RCP to exhaustion (hypothesis 2a). Affective responses reported in the imagery condition were more positive than in the control condition (hypothesis 2b).

The imagery type used in this study was based on enjoyment and energy response propositions. Enjoyment focused imagery has been shown to improve affective responses during (Stanley & Cumming, 2010b) and post-moderate intensity exercise (Stanley & Cumming, 2010a). This study extends those findings. With exercise intensity standardised to 5% above VT (*M* = 59.5, *SD* = 7.9 % HRR) and RCP (*M* = 84.3, *SD* = 5.0 % HRR), rather than 50% HRR (the intensity set to reflect moderate intensity by Stanley and Cumming, 2010a), the combination of enjoyment and energy focused imagery helped to maintain more positive affective responses during exercise. Although there was a decline in affect, as seen in previous studies examining affective responses at (Ekkekakis et al., 2008; Rose & Parfitt, 2010) and above VT (Ekkekakis et al., 2004, 2005b; Parfitt et al., 2006; Rose & Parfitt, 2007; Sheppard & Parfitt, 2008; Welch et al., 2007) in the imagery condition, the decline was not as steep as that recorded in the control condition. Imagery use (as a cognitive strategy) therefore acted to suppress the decline in affect at exercise intensities above VT.

Relationships between cerebral haemodynamics, affective responses and imagery use were shown at intensities above VT (hypothesis 3). Firstly, more positive affective responses were reported in the imagery condition, which were associated with greater oxygen extraction (deoxygenation; ΔHHb) in the region in the right hemisphere. A similar finding was shown in studies I and II. These associations are in line with research that has shown right lateralised regions of the frontal cortex are involved in the down-regulation of negative affect (Beauregard et al., 2001; Ochsner et al., 2004). Secondly, higher scores of visual and kinetic ability (shown to occur simultaneously: Cumming & Ste-Marie, 2001) indicated linear relationships with greater oxygen extraction (ΔHHb) in the left hemisphere. These moderate correlations provide some support to recent data from fMRI research which has examined dorsolateral prefrontal cortex activation during imagery (Cremers et al., 2012; van der Meulen et al., 2012). Finally, there were no significant correlations between cerebral haemodynamics, affective responses and imagery use at intensities from RCP to exhaustion. The significant correlations recorded provide some objective evidence to confirm that imagery as a cognitive strategy was associated with increased activation in the frontal cortex and affective responses during exercise at intensities above VT.

The exact mechanisms to explain how affective responses were influenced at intensities above VT and RCP to exhaustion due to imagery use cannot be precisely determined. The

trajectory of affective responses in the Condition by Time interaction indicates imagery may have a buffering effect at the initial onset of exercise (from pre-exercise to GET), as beyond GET the decline between the conditions remains constant until the end of exercise (see Figure 6.2). Imagery was selected as a cognitive strategy that should maintain frontal activation and influence affective responses through cognitive processes. The dorsolateral region of the frontal cortex has been previously shown to impact on the reappraisal (Ochsner et al., 2004) and inhibition (Beauregard et al., 2001) of negative affect. Alternatively, it is possible that imagery use may have simply distracted the individual through attentional processes from the aversive stimuli. In actively imaging, participant's focus would be on the imagined feelings and away from the physical discomfort experienced during the actual exercise. These would be appropriate explanations for the intensities above VT. However, the mechanisms to explain the affective responses at exercise intensities RCP to exhaustion are more speculative. The significant Condition by Time interaction for affective responses suggests that imagery had a positive influence irrespective of intensity, however, the dorsolateral area showed no differential activation between the control and imagery conditions. Therefore, imagery was not able to engage the frontal cortex at this intensity. Any influence on affective responses is presumably attributed to other areas of the frontal cortex, such as the anterior cingulate and insula, which are highly involved in physiological and interoceptive awareness (Craig, 2005; see 2.3.2). In combination with ventrolateral and ventromedial frontal cortices (see 2.2.3.2), these areas may have influenced affective responses through a different (lower-order) regulatory mechanism at the higher exercise intensities.

Despite no differences in cerebral haemodynamics between the imagery and control conditions during exercise from RCP to exhaustion, there were changes in cerebral haemodynamics across the exercise duration. Cerebral oxygenation increased until 80% exercise duration and remained stable until exhaustion, whereas cerebral deoxygenation and blood volume increased from RCP to exhaustion. The rise in deoxygenation and the plateau of oxygenation above 80% duration indicated the onset of a hypofrontality response near exhaustion (Dietrich, 2006; see 2.3.3). The hypofrontality or deregulatory response of activation in the frontal activation was less profound during constant-load than incremental exercise shown in studies I and II. The use of multi-channel NIRS may have indicated different activation patterns in sub regions of the frontal cortex to explain variability near exhaustion.

Asymmetrical hemispheric differences were shown at exercise intensities from RCP to exhaustion. Cerebral blood volume increased in the region of the left hemisphere between 40 and 60% duration of exercise. It is speculated that this increase in blood volume indicated final cognitive attempts to continue exercising at the challenging intensities.

According to the DMM (Ekkekakis, 2003; Ekkekakis & Acevedo, 2006), a cognitive strategy would be beneficial at exercise intensities around VT to engage the frontal cortex and uphold cognitive-dominance; therefore a more positive affective response could be maintained. However, the sensory input from the body would be too strong at intensities from RCP to exhaustion. Although this study did not measure amygdala activity (i.e. the 'low road' described in 2.1.3), increased activation in the frontal cortex and the more positive affective responses in the imagery condition suggest that imagery use may have served to down-regulate the potentially aversive state induced by exercise at intensities above VT (i.e. via 'high road' as described in 2.1.3). However, at intensities RCP to exhaustion sensory input to subcortical areas intensifies (i.e. the 'low road') and the frontal cortex cannot maintain activation (or dominance) to down regulate the amygdala. Imagery appears to have some influence on affective responses at these intensities although no change in cerebral haemodynamics between the conditions was shown. The frontal cortex indicated some hypofrontality effects which occurred just before the end of the exercise (80% duration).

This study provides evidence that engaging activation in the frontal cortex during exercise at intensities above VT positively influences affective responses. However, a cognitive strategy did not influence activation in the frontal cortex at intensities above RCP. It is theorised that at these intensities the dominance of frontal cortex input to the amygdala via the 'high road' is challenged as interoceptive input from the body to the amygdala via the 'low road' is intensified.

Chapter 7

Study IV: Cerebral blood flow in the frontal cortex during exercise: The influence of prior exhaustive activity

The previous chapter (study III) showed that a cognitive strategy could engage the frontal cortex during exercise at intensities above VT, but not RCP. It is proposed that at these intensities, the cognitive mode of input to the amygdala is challenged by the intensified sensory input from the body. This chapter (study IV) examines the influence of increased sensory (interoceptive) input from the body (using prior exhaustive activity) upon frontal cortex activation and affective responses, during constant-load exercise at similar intensities (i.e. above VT).

This study formed the basis of an abstract submitted for presentation at the 6th Exercise and Sports Science Australia Conference and Sports Dieticians Australia Update "Research to Practice", Adelaide, Australia, April 2014. Tempest, G., Eston, R., Parfitt, G. *Prior exhaustive activity reduces frontal cortex activation and influences affective responses to subsequent exercise.*

7.1 Background

Physiological arousal (i.e. sensory input from the body) during exercise shapes an individual's perception and experience of the activity i.e. how hard they are working (Hampson, St Clair Gibson, Lambert & Noakes, 2001) and how they feel (Craig, 2005; Ekkekakis & Acevedo, 2006). An individual's experience of exercise, particularly their affective response, is proposed to be influenced by the interplay between subcortical areas of the brain which receive sensory input from the body and higher order (cognitive) processes in the frontal cortex (explained by the DMM; see 2.1.3). The input from sensory (interoceptive) processes and cognitive processes reflect the 'high road' and 'low road', respectively, of input to the amygdala (LeDoux 1996; Ekkekakis, 2003; Ekkekakis & Acevedo, 2006: see 2.2.1 and Figure 2.5).

Ascending sensory pathways from the peripheral nervous system feed information from nociceptors (pain), metaboreceptors (chemical), thermoreceptors (temperature), mechanoreceptors and baroreceptors (touch, pressure, tension) to the central nervous system (see Ament & Verkerke, 2009). These inputs are integrated in subcortical regions of the brain i.e. medulla and pons (Craig 1996; see 2.2.2). Through specialised spinothamalocortical pathways, sensory (interoceptive) awareness is represented in areas of the frontal cortex (Craig, 2002; Damasio, 1999) particularly in the right anterior cingulate and insula cortices (Craig, 2006; Critchley et al., 2004). The representation of interoceptive awareness in the frontal cortex is linked with subjective (positive and negative) feeling states (Critchley, 2005; Pollatos, Gramann & Schandry, 2007) and may be influenced by cognitive control processes (Ochsner & Gross, 2004). However, the effects of different states of physiological arousal through intensified sensory (interoceptive) inputs that might influence activation in sub regions of the frontal cortex involved in the regulation of affect has not been examined.

During exercise at intensities above VT, feelings of discomfort and displeasure are commonly reported (Ekkekakis et al., 2004, 2008; Ekkekakis et al., 2011; see 2.1.3). According to the DMM, the challenge of increasing ascending sensory (interoceptive) input to the amygdala is greatest at intensities above VT (and RCP) to exhaustion (Ekkekakis, 2003). The VT and RCP define markers of increased physiological arousal due to increases in the respiratory response (pulmonary oxygen uptake) required for energy metabolism

during exercise (see Figure 2.3). Studies I and II indicated that during incremental exercise at intensities above VT (and RCP) as physiological arousal increased, deregulation in the frontal cortex occurred. This is presumably due to the reallocation of metabolic resources to subcortical areas of the brain involved in processing sensory input from the body (Dietrich, 2003, 2006; see 2.3.3). These findings indicate limitations of the capacity of the frontal cortex during exercise at these intensities. A reduction or deregulation of activation in the frontal cortex is proposed to influence affective responses by inhibiting cognitive mechanisms, which attempt to maintain a more positive affective response (Ekkekakis, 2003). Study III showed greater activation in the frontal cortex due to the use of a cognitive strategy and that affective responses were more positive at intensities just (5 % aerobic power) above VT. However, the effects of increased physiological arousal (intensified interoceptive input from the body) at these intensities (above VT), and its influence upon the capacity of the frontal cortex has not been investigated.

An acute bout of exercise following prior exhaustive activity may intensify physiological arousal during exercise, particularly at intensities above VT (i.e. intensifying input via the 'low road' to the amygdala of the DMM). Acute exercise following exhaustive activity has been shown to increase an individuals perceived exertion; how hard they felt they were working (Eston, Faulkner, St Clair Gibson, Noakes & Parfitt, 2007). The ability of the frontal cortex to maintain activation in a state of heightened physiological arousal and increased exertion or discomfort presumably would impact an individual's affective response. This in turn may influence how long the individual would persevere or continue with that activity.

Therefore, in the presence of increased sensory input from the body, participants would perceive the exercise as being harder and would exercise for less time (presumably due to increased feelings of discomfort) at intensities above VT. It is proposed that a deregulation of metabolic resources (i.e. blood flow) would occur in the frontal cortex. Theoretically, this would be due to a shift of the resources to subcortical areas of the brain required to manage sensory input from the body (Dietrich, 2003, 2006). Deregulation in regions of the frontal cortex would reduce the influence of cognitive mechanisms which would impact an individual's perception of exercise (i.e. how hard they perceive they are working and how they feel).

The present study investigates if prior exhaustive activity (used to induce a state of increased sensory and physiological arousal) influences activation (reflected by changes in haemodynamics using multi-channel NIRS) in sub regions of the frontal cortex and affective responses during constant-load exercise at intensities (standardised to GETs); VT and RCP to end of exercise (exhaustion). The influence of prior exhaustive activity upon how hard the individuals feel they are working (using the rating of perceived exertion [RPE] scale), affective responses and exercise duration (at intensities corresponding to RCP to exhaustion) will be investigated.

Three hypotheses were examined: 1) Activation in the frontal cortex is reduced during exercise at intensities corresponding to VT and RCP following prior exhaustive activity than in the control condition; 2) RPE is higher and affective responses are more negative during exercise following prior exhaustive activity than in the control condition; and 3) Individuals reach their individually determined VT and RCP sooner and exercise for less time following prior exhaustive activity than in the control condition.

7.2 Methods

Participants

Participants (n = 20; males = 10; *M, SD*, age, M = 25.3, SD = 3.0 years; height, M = 174.9, SD = 6.7 cm; body mass, M = 72.7, SD = 8.0 kg) were recruited at a university campus.

Measures

Cerebral haemodynamics were measured in eight sub regions (1-8) of the frontal cortex using multi-channel NIRS as previously described (see 3.3.3.3). Approximate locations of the sub regions (1-8) measured are shown in Figure 7.1. Affective responses (pleasure-displeasure) were recorded using the Feeling Scale (Hardy & Rejeski, 1989; see 3.2.1).



Figure 7.1. Approximate locations of sub regions (1-8) of the frontal cortex measured using multi-channel NIRS. Odd numbers identify superior and even numbers identify inferior regions of the frontal cortex.

Perceived exertion was recorded using the 6-20 category RPE scale (Borg, 1998; see Appendix 4). Participants reported their overall perceived exertion with numbers and verbal anchors, ranging from no exertion at all (6), somewhat hard (13), to maximal effort (20). Affective responses (pleasure-displeasure) were recorded using the Feeling Scale (Hardy & Rejeski, 1989; see 3.2.1).

Procedures

This study employed a crossover design and required participants to visit the laboratory on three occasions. Each participant completed a control and experimental (i.e. prior exhaustive activity) condition to examine the effects of prior exhaustive activity upon cerebral haemodynamic and perceptual responses and exercise duration.

The study required the participants to visit the laboratory on three occasions. On the first visit, participants were screened (i.e. completed the informed consent form and exercise testing questionnaire) and initial assessments were taken (see 3.3.1). Participants were fitted with a facemask (to measure metabolic data) and completed an incremental exercise test to volitional exhaustion (20 W/min) as previously described (see 3.3.3). Participants VT, RCP and $\dot{V}O_{2peak}$ were identified (see 3.4.1) and workloads corresponding to exercise intensities were determined (see 3.4.2). The participants returned for a further two sessions; control and experimental conditions (in a randomised, counterbalanced order).

In the control condition, participants completed a three minute warm up (50 W), followed by 10 minutes of exercise at a workload corresponding to VT. Participants then continued to exercise at a workload corresponding to 5% below RCP until exhaustion.

In the experimental condition, participants completed an incremental (20 W/min) exercise test to exhaustion on a recumbent cycle ergometer (see 3.3.3). This incremental exercise test was used to induce a state of increased sensory and physiological arousal. Following a 10 minute period to allow recovery, participants completed exercise as described in the control condition (i.e. intensities corresponding to VT [for 10 min] and 5% below RCP to exhaustion).

In the control and experimental conditions, the 10 minute duration of exercise at intensities corresponding to VT was chosen to ensure all participants would complete the exercise before the increase in workloads corresponding to 5 % below RCP. Participants reported their RPE and affective responses pre-exercise (affect only) and every minute during.

Participant's workloads and $\dot{V}O_2$ (l/min) as a percentage of $\dot{V}O_{2peak}$ at intensities corresponding to VT were *M* = 156 W (*SD* = 26) and *M* = 66.9% (*SD* = 2.6), respectively, and RCP were *M* = 209 W (*SD* = 33) and *M* = 88.9% (*SD* = 2.5), respectively.

Data Reduction

To standardise data points between participants and to enable a comparison between conditions (control and experimental), $\dot{V}O_2$ (l/min) at the point of VT and RCP (identified in the initial exercise test) were matched to that achieved in the control and experimental conditions during exercise at the corresponding workloads. Due to the exponential increase in $\dot{V}O_2$ at intensities around the RCP (Gaesser & Poole, 1996), the workload was set at 5% below RCP so that the point of RCP would be automatically achieved and then located. Cerebral haemodynamic variables, RPE and affective responses were then extracted at time points corresponding to the point of VT and RCP, in addition to the end of exercise at VT (VT-end) and RCP (exhaustion). Affective responses included a pre-exercise measure.

Statistical Analyses

The independent variables were Condition (control, experimental), Region (1-8) and Time (corresponding to pre-exercise and intensities VT, VT-end, RCP and End). The dependent variables were changes in primary (ΔO_2Hb, ΔHHb) and secondary (ΔtHb, $\Delta HbDiff$) cerebral haemodynamics, RPE and affective responses.

To examine hypothesis 1, a series of Condition (2; control, experimental) by Region (8; 1-8) by Time (4; VT, VT-end, RCP, End) analysis of variance (ANOVA) with repeated measures were conducted on the primary [ΔO_2Hb, ΔHHb] and secondary [ΔtHb, $\Delta HbDiff$] NIRS variables. To examine hypothesis 2, a Condition (2; control, experimental) by Time (4; VT, VT-end, RCP, End) and a Condition (2; control, experimental) by Time (5; pre-exercise, VT, VT-end, RCP, End) ANOVA with repeated measures was conducted on RPE and affective responses, respectively. Finally, to examine hypothesis 3, a Condition (2; control, experimental) by Time (3; VT, RCP, End) ANOVA with repeated measures was conducted on the duration of exercise (sec).

7.3 Results

7.3.1 Preliminary Analyses

To determine (a) that the point of VT and RCP (indicated by $\dot{V}O_2$ l/min) were the same between the two exercise sessions (control and experimental conditions), and that these were not different to the incremental exercise test (IET), and (b) any differences in VO_2 (l/min) at End (exhaustion) between the sessions, a Session (3; IET, control, experimental) by Time (3; corresponding to VT, RCP and End [exhaustion]) ANOVA with repeated measures was conducted. The analysis indicated no Session by Time interaction, $F(3,53) = 2.74$, $p = .057$, $\eta_p^2 = .13$. However, there were significant main effects of Session, $F(2,38) = 4.45$, $p = .018$, $\eta_p^2 = .19$, and Time, $F(1,25) = 343.17$, $p < .001$, $\eta_p^2 = .95$, for $\dot{V}O_2$. Oxygen uptake (l/min) was greater in the IET ($M = 2.7$, $SD = .4$) than in the control ($M = 2.6$, $SD = .4$) and experimental conditions ($M = 2.6$, $SD = .4$), and significantly different at time points corresponding to VT ($M = 2.1$, $SD = .3$), RCP ($M = 2.8$, $SD = .5$) and End ($M = 3.1$, $SD = .5$).

Heart rate (HR) was compared between the two conditions (experimental and control) at time points corresponding to pre-exercise, VT, VT-end, RCP and End (exhaustion) in a Condition (2) by Time (5) ANOVA with repeated measures. The analysis indicated a significant main effect of Condition, $F(1,19) = 68.34$, $p < .001$, $\eta_p^2 = .78$, and Time, $F(2,47) = 591.98$, $p < .001$, $\eta_p^2 = .97$, and a Condition by Time interaction, $F(2, 35) = 39.70$, $p < .001$, $\eta_p^2 = .68$, for HR. Heart rate (bpm) was greater in the experimental (M = 145.9, SD = 9) than the control (M = 135.2, SD = 7) condition and significantly different at time points corresponding to pre-exercise (M = 81.3, SD = 10.2), VT (M = 134.7, SD = 7.6), VT-end (M = 148.9, SD = 10.2), RCP (M = 164.7, SD = 10.9), and exhaustion (M = 172.3, SD = 10.5). Heart rate was greater in the experimental, pre-exercise (M = 93.5, SD = 14.5), than control condition (M = 69.0, SD = 8.8), but similar at VT (experimental, M = 139.3 , SD = 8.7; control, M = 129.9, SD = 8.6), VT-end (experimental, M = 152.8, SD = 10.0; control, M = 145.2, SD = 11.0), RCP (experimental , M = 166.2, SD = 11.5; control, M = 163.4, SD = 11.0), and End (experimental, M = 172.8, SD = 10.9; control, M = 173.8, SD = 10.6).

7.3.2 Cerebral haemodynamics (hypothesis 1)

Cerebral ΔO_2Hb

Significant main effects of Condition, $F(1,19) = 15.07$, $p < .001$, $\eta_p^2 = .44$, Region, $F(4,80) = 12.54$, $p < .001$, $\eta_p^2 = .40$, and Time, $F(1,27) = 55.0$, $p < .001$, $\eta_p^2 = .74$, and significant Condition by Time, $F(2,38) = 28.86$, $p < .001$, $\eta_p^2 = .60$, and Region by Time, $F(7,125) = 10.88$, $p < .001$, $\eta_p^2 = .36$, interactions were recorded for ΔO_2Hb (μM). Follow up tests indicated that ΔO_2Hb was greater in the control (M = 7.31, SD = 3.38) than the experimental (M = 4.48, SD = 3.05) condition and in Regions 6 and 8 (inferior regions of the left hemisphere). M (SD)s are presented in Table 7.1.

The means and standard deviations (hypothesis 1) of the cerebral haemodynamic variables (μM) in this study are provided in Appendix 3d.

Table 7.1. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex, *M* (*SD*).

* Significantly greater ΔO_2Hb in regions 6 and 8 than other regions.

Right hemisphere		*Left hemisphere*		
①	③	⑤	⑦	*Superior regions*
4.38 (2.79)	5.04 (3.16)	5.36 (3.30)	5.52 (2.81)	
②	④	⑥	⑧	*Inferior regions*
5.61 (3.04)	6.31 (3.24)	7.67* (3.00)	7.28* (3.17)	

Cerebral ΔO_2Hb increased from VT (M = 1.15, SD = 1.21) to VT-end (M = 6.46, SD = 2.76) and RCP (M = 7.40, SD = 3.68) to End (M = 8.58, SD = 4.46). The Condition by Time interaction indicated that ΔO_2Hb was greater at VT-end in the control than the experimental condition. Cerebral ΔO_2Hb continued to increase until End in the control, but remained stable in the experimental condition (see Figure 7.2).

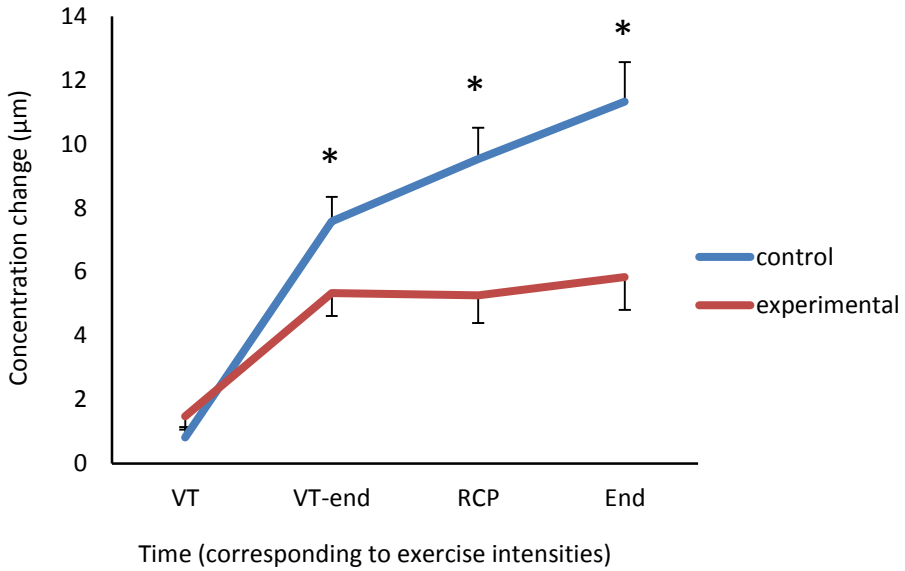


Figure 7.2. Cerebral oxygenation (ΔO_2Hb) in the experimental and control conditions at each time point, *M* and *SEM.* * Significantly greater ΔO_2Hb in the control than the experimental condition.

The Region by Time interaction indicated that ΔO_2Hb was greater at VT-end, RCP and End in Regions 6 and 8 (inferior regions of the left hemisphere) than other regions (see Figure 7.3).

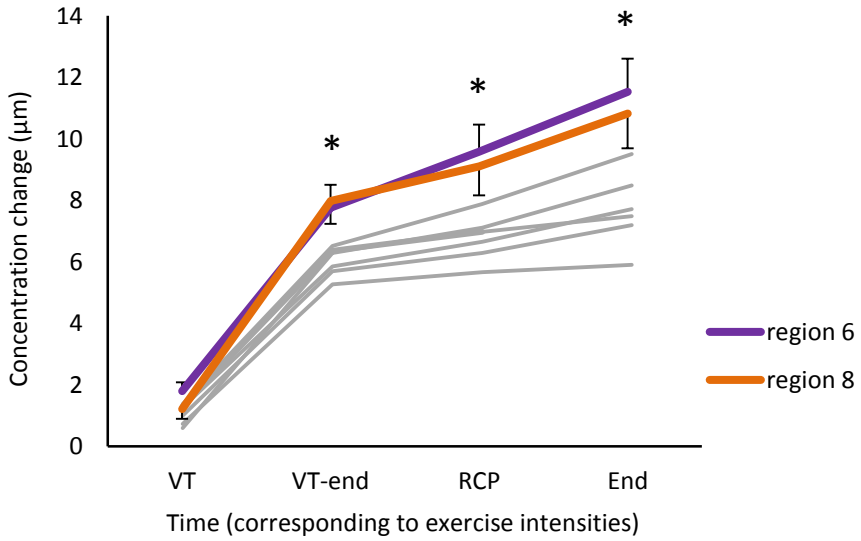


Figure 7.3. Cerebral oxygenation (ΔO_2Hb) in sub regions of the frontal cortex at each time point, *M and SEM.* * Significantly greaterΔO_2Hb in regions 6 and 8 than other regions (grey lines).

Cerebral ΔHHb

Significant main effects of Region, $F(4,68) = 3.20$, $p = .022$, $\eta_p^2 = .14$, and Time, $F(1,28) = 63.19$, $p < .001$, $\eta_p^2 = .77$, and a significant Condition by Region by Time, $F(6, 120) = 2.40$, $p = .029$, $\eta_p^2 = .11$, interaction were recorded for ΔHHb (µM). Follow up tests indicated that ΔHHb was greater in Regions 2 than 7. *M* (*SD*)s are presented in Table 7.2.

Table 7.2. Cerebral deoxygenation (ΔHHb) in sub regions of the frontal cortex, *M* (*SD*).

* Significantly greater ΔHHb in region 2 than 7.

Right hemisphere		*Left hemisphere*		
①	③	⑤	⑦	*Superior regions*
2.25 (1.75)	2.52 (1.56)	2.27 (1.40)	1.64 (1.23)	
②	④	⑥	⑧	*Inferior regions*
2.59* (1.98)	2.01 (1.38)	1.76 (1.09)	1.66 (1.40)	

Cerebral ΔHHb remained stable from VT (*M* = .52, *SD* = .62) to VT-end (*M* = .96, *SD* = .99) and increased from VT-end to RCP (*M* = 2.28, *SD* = 1.41) and End (*M* = 4.6, *SD* = 2.39). The three factor interaction indicated that from VT to VT-end, in the control condition, ΔHHb increased in Region 2, decreased in Region 7 and remained stable in other regions (1, 3, 4, 5, 6 and 8), whereas ΔHHb increased in the experimental condition (see Figure 7.4).

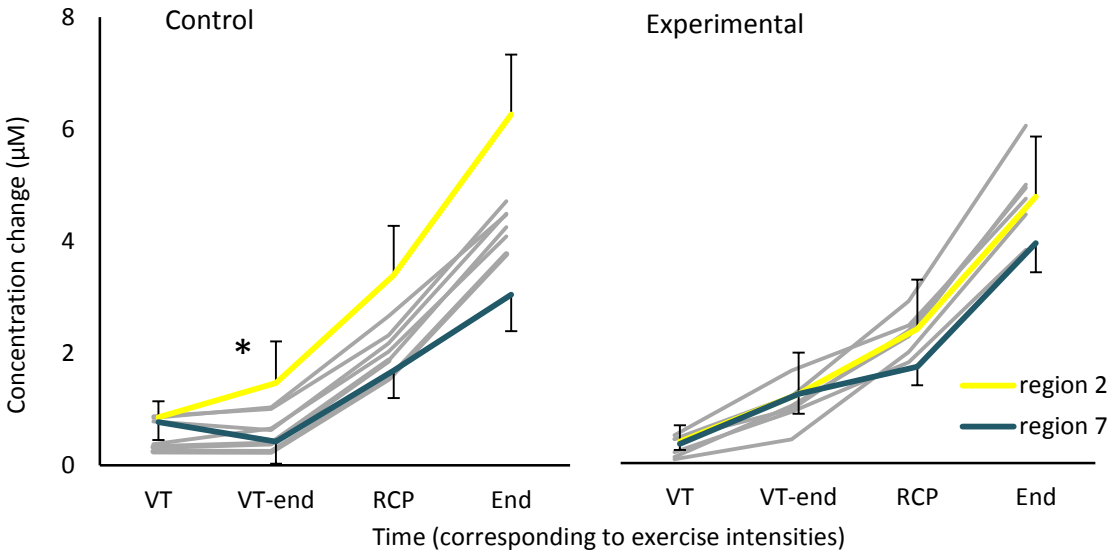


Figure 7.4. Cerebral deoxygenation (ΔHHb) in the experimental and control conditions at each time point, *M* and *SEM*. * Significant increase of ΔHHb in region 2 and decrease in region 7 in the control condition.

Cerebral ΔtHb

Significant main effects of Condition, $F(1,19) = 13.13$, $p = .002$, $\eta_p^2 = .41$, Region, $F(7,133) = 6.42$, $p < .001$, $\eta_p^2 = .25$, and Time, $F(1,25) = 87.10$, $p < .001$, $\eta_p^2 = .82$, and significant, Condition by Time, $F(2,37) = 16.12$, $p < .001$, $\eta_p^2 = .46$, Region by Time, $F(6, 122) = 6.26$, $p < .001$, $\eta_p^2 = .25$, and Condition by Region by Time, $F(6, 109) = 2.25$, $p = .046$, $\eta_p^2 = .11$, interactions were recorded for ΔtHb (μM). Follow up tests indicated that ΔtHb was greater in the control ($M = 9.36$, $SD = 3.90$) than the experimental ($M = 6.61$, $SD = 3.30$) condition and in Regions 6 and 8 (inferior regions of the left hemisphere) than the other regions. *M* (*SD*)s are presented in Table 7.3.

Table 7.3. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex, *M* (*SD*).

* Significantly greater ΔtHb in region 6 and 8 than the other regions.

Right hemisphere		*Left hemisphere*		
①	③	⑤	⑦	*Superior regions*
6.57 (3.06)	7.61 (3.22)	7.66 (4.15)	7.10 (3.36)	
②	④	⑥	⑧	*Inferior regions*
8.19 (3.65)	8.34 (3.55)	9.43* (3.48)	9.00* (3.88)	

Cerebral ΔtHb increased from VT ($M = 1.67$, $SD = 1.09$) to VT-end ($M = 7.42$, $SD = 3.44$) and RCP ($M = 9.68$, $SD = 4.09$) to End ($M = 13.17$, $SD = 5.20$). The Condition by Time interaction indicated that ΔtHb was greater at VT-end in the control than the experimental condition. Cerebral ΔtHb continued to increase until End in the control, but remained stable from VT-End to RCP and increased RCP to End in the experimental condition (see Figure 7.5).

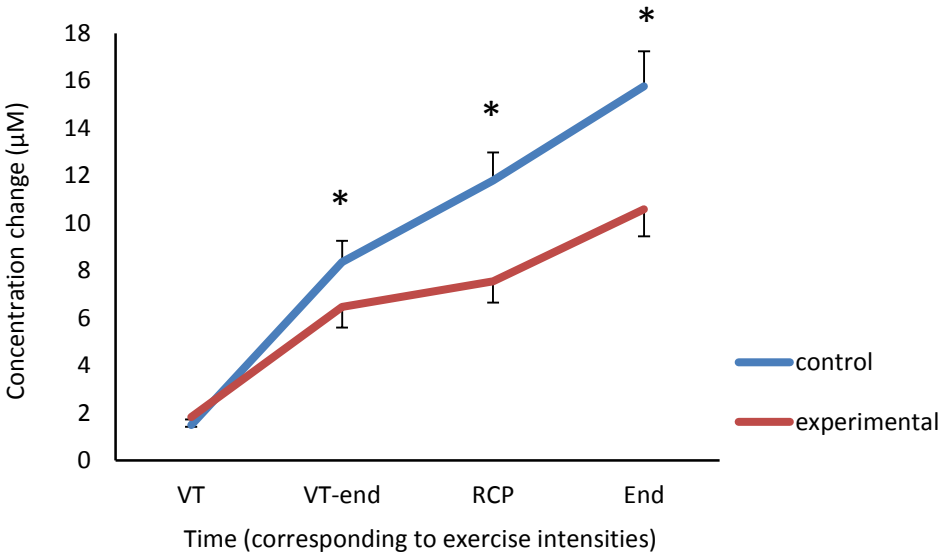


Figure 7.5. Cerebral blood volume (ΔtHb) in the experimental and control conditions at each time point, *M* and *SEM.* * Significantly greater ΔtHb in the control than the experimental condition.

The Region by Time interaction indicated that ΔtHb was greater at VT-end, RCP and End in Regions 6 and 8 (inferior regions of the left hemisphere: see Figure 7.6) than other regions.

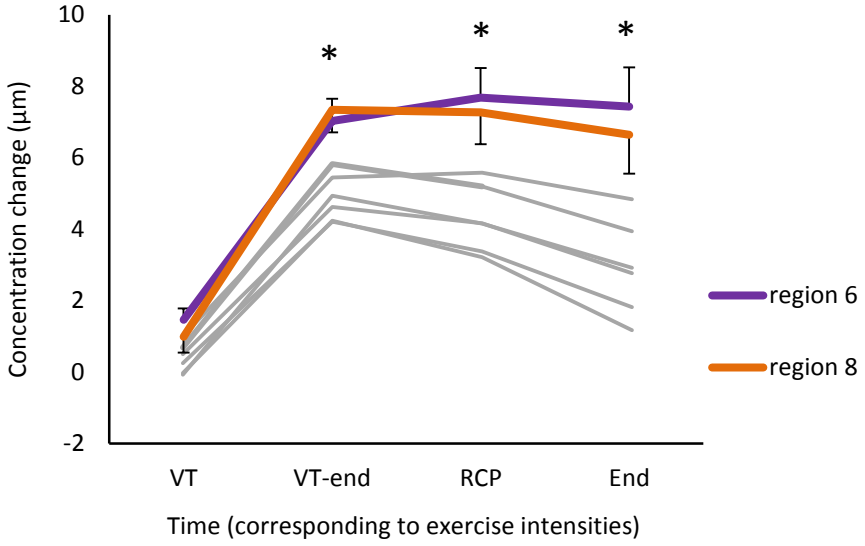


Figure 7.6. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex at each time point, *M* and *SEM*. * Significantly greater ΔtHb in regions 6 and 8 than other regions (grey lines).

The three factor interaction indicated that ΔtHb was greater at End in Regions 2 and 6 in the control than the experimental condition (see Figure 7.7).

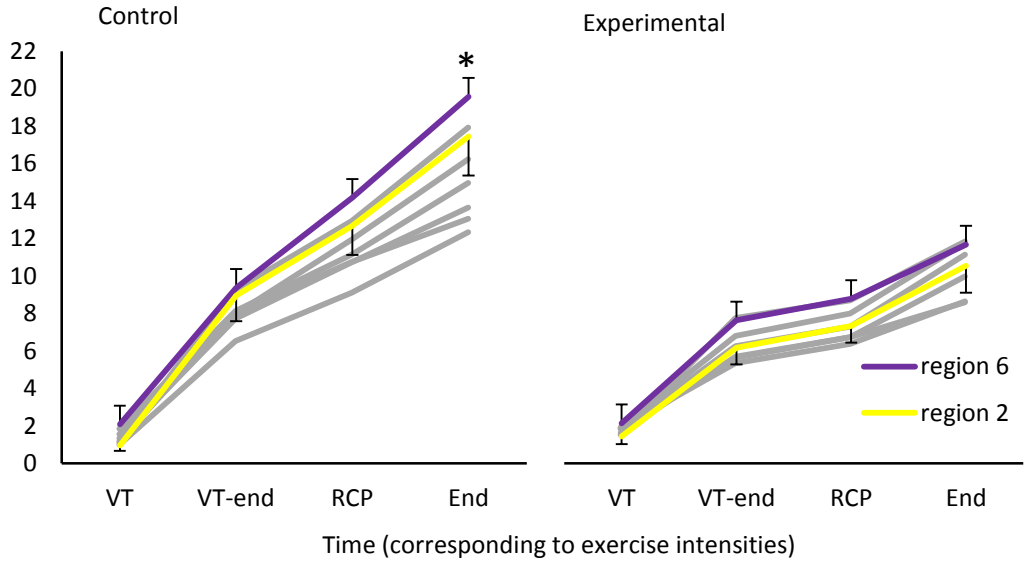


Figure 7.7. Cerebral blood volume (ΔtHb) in sub regions of the frontal cortex in the experimental and control conditions at each time point, *M* and *SEM*. * Significantly greater ΔtHb in Regions 2 and 6 in the control than the experimental condition.

Cerebral ΔHbDiff

Significant main effects of Condition, $F(1,19) = 14.68$, $p < .001$, $\eta_p^2 = .44$, Region, $F(4,73) = 12.41$, $p < .001$, $\eta_p^2 = .40$. and Time, $F(2,32) = 21.40$, $p < .001$, $\eta_p^2 = .53$, and significant Condition by Time, $F(2,43) = 30.63$, $p < .001$, $\eta_p^2 = .62$, and Region by Time, $F(5,90) = 8.94$, $p < .001$, $\eta_p^2 = .32$, interactions were recorded for ΔHbDiff (μM). Follow up tests indicated that ΔHbDiff was greater in the control ($M = 5.27$, $SD = 3.44$) than the experimental ($M = 2.35$, $SD = 3.19$) condition and in Regions 6 and 8 (inferior regions of the left hemisphere) than Regions 1 and 3. *M* (*SD*)s are presented in Table 7.4.

Table 7.4. Cerebral oxygen saturation (ΔHbDiff) in sub regions of the frontal cortex, *M (SD).*

* Significantly greater ΔHbDiff in regions 6 and 8 than 1 and 3.

Right hemisphere		*Left hemisphere*		
①	③	⑤	⑦	*Superior regions*
2.20 (3.54)	2.47 (3.83)	3.07 (2.98)	3.97 (2.74)	
②	④	⑥	⑧	*Inferior regions*
3.04 (3.61)	4.28 (3.49)	5.90* (2.83)	5.56* (3.07)	

Cerebral ΔHbDiff increased from VT (*M* = .63, *SD* = 1.58) to VT-end (*M* = 5.50, *SD* = 2.32), but remained stable from RCP (*M* = 5.13, *SD* = 3.78) to End (*M* = 3.98, *SD* = 4.91). The Condition by Time interaction indicated that ΔHbDiff was greater at VT-end in the control than the experimental condition. From VT-End to RCP and End, ΔHbDiff remained stable in the control, but declined in the experimental condition (see Figure 7.8).

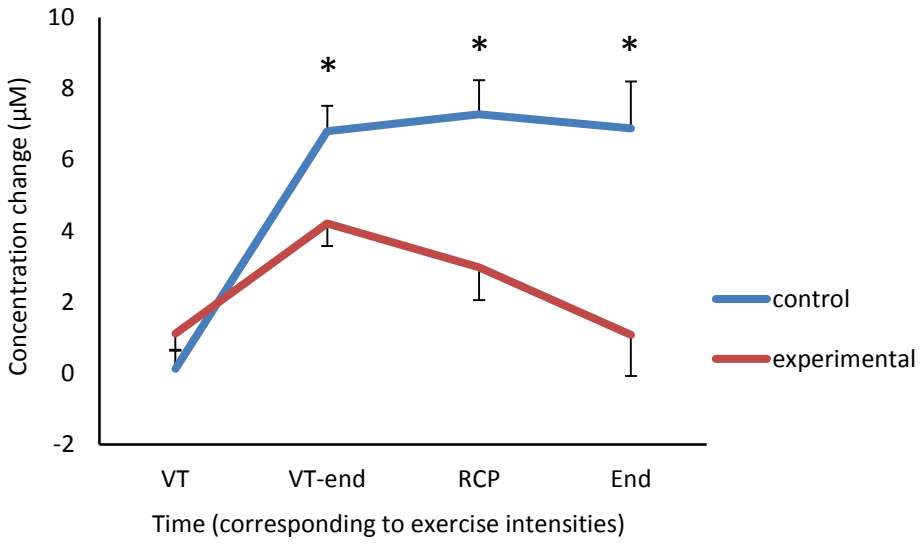


Figure 7.8. Cerebral oxygen saturation (ΔHbDiff) in the experimental and control conditions at each time point, *M* and *SEM*. * Significantly greater ΔHbDiff in the control than the experimental condition.

The Region by Time interaction indicated that ΔHbDiff was greater at VT-end, RCP and End in Regions 6 and 8 (inferior regions of the left hemisphere) than other regions (see Figure 7.9).

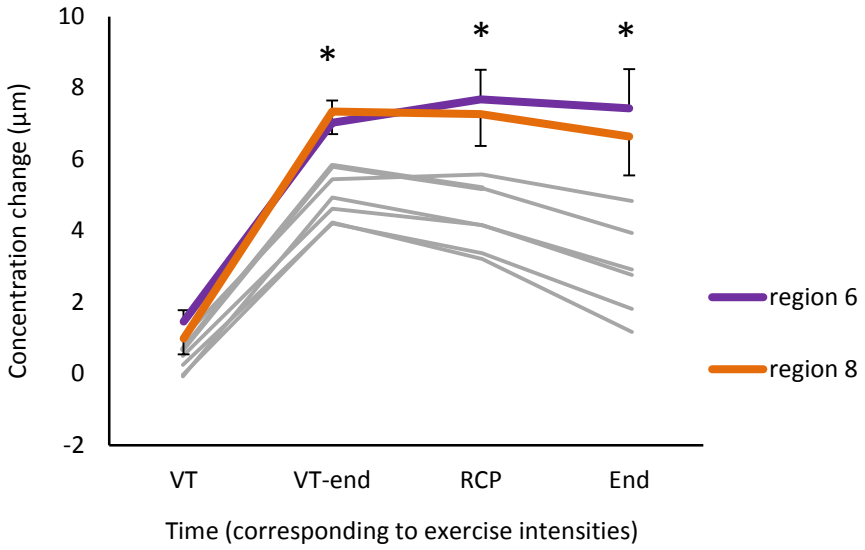


Figure 7.9. Cerebral oxygen saturation (ΔHbDiff) in sub regions of the frontal cortex at each time point, *M* and *SEM*. * Significantly greater ΔHbDiff in regions 6 and 8 than other regions (grey lines).

7.3.3 Rating of perceived exertion and affective responses (hypothesis 2)

Significant main effects of Condition, $F(1,19) = 4.83$, $p = .040$, $\eta_p^2 = .20$, and Time, $F(2,36) = 241.83$, $p < .001$, $\eta_p^2 = .92$, and a significant Condition by Time, $F(3,57) = 229.39$, $p < .001$, $\eta_p^2 = .25$, interaction was recorded for RPE. Follow up tests indicated that RPE was higher in the experimental ($M = 16.7$, $SD = 1.3$) than the control condition ($M = 16.3$, $SD = .9$), and lower during exercise at VT ($M = 14.0$, $SD = 1.4$) than RCP ($M = 17.8$, $SD = 1.1$). The interaction indicated that RPE was higher from VT to VT-end in the experimental condition but increased significantly in the control condition from VT-end to RCP. There were no differences in RPE between conditions at RCP and End (see Figure 7.10).

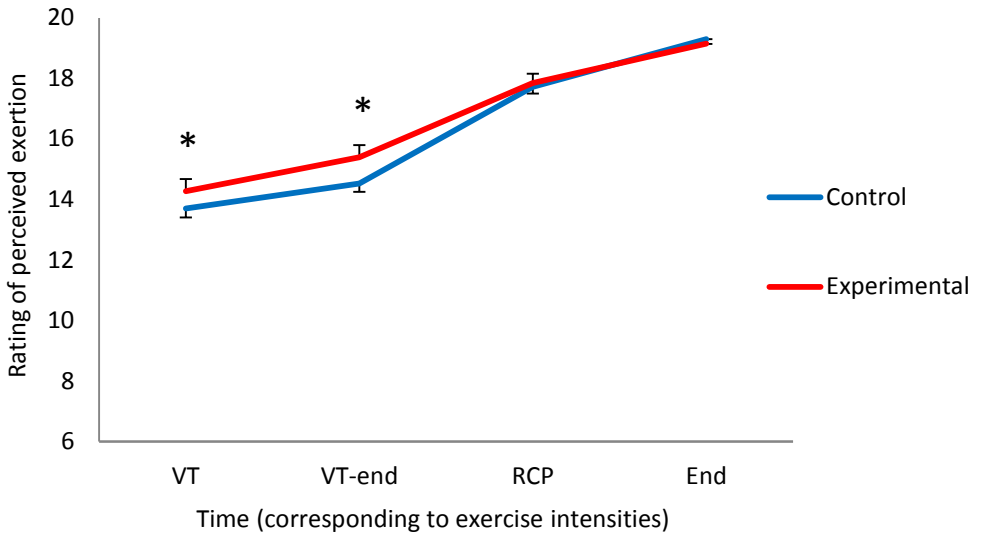


Figure 7.10. Rating of perceived exertion reported in the experimental and control conditions at each time point, *M* and *SEM*. * Significantly higher RPE in the experimental than the control condition.

Significant main effects of Condition, $F(1,19) = 15.87$, $p < .001$, $\eta_p^2 = .46$, and Time, $F(2,37) = 105.75$, $p < .001$, $\eta_p^2 = .85$, and a significant Condition by Time, $F(3,55) = 6.37$, $p < .001$, $\eta_p^2 = .25$, interaction were recorded for affective responses. Follow up tests indicated that affective responses were lower in the experimental ($M = -.69$, $SD = 1.0$) than the control ($M = .18$, $SD = .81$) condition and declined over time (pre-exercise, $M = 2.2$, $SD = .8$; VT, $M = 1.0$, $SD = .9$; VT-end, $M = .4$, $SD = .9$; RCP, $M = -1.7$, $SD = 1.4$; End, $M = -3.2$, $SD = 1.7$). The interaction indicated that affective responses were more negative in the experimental than the control condition pre-exercise. Despite a decline in affective responses in both conditions over time, the decline was greater in the experimental condition from VT to VT-end (see Figure 7.11).

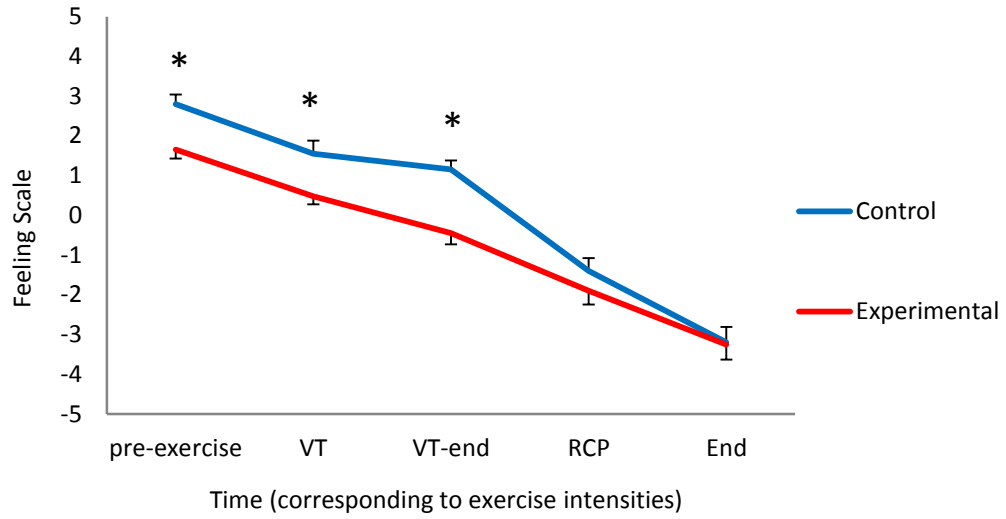


Figure 7.11. Affective responses reported in the experimental and control conditions at each time point, *M* and *SEM*. * Significantly less positive affective response in the experimental than the control condition.

7.3.4 Exercise duration (hypothesis 3)

Significant main effects of Condition, $F(1,19) = 32.17$, $p < .001$, $\eta_p^2 = .63$, and Time, $F(1,22) = 78.67$, $p < .001$, $\eta_p^2 = .81$, and a significant Condition by Time, $F(1,24) = 5.77$, $p = .018$, $\eta_p^2 = .23$, interaction were recorded for exercise duration. Follow up tests indicated that exercise duration (sec) was longer in the control ($M = 319$, $SD = 112$) than the experimental ($M = 203$, $SD = 71$) condition and that exercise duration at VT ($M = 146$, $SD = 32$) and RCP ($M = 154$, $SD = 82$) were different to End ($M = 483$, $SD = 177$). The interaction indicated that from RCP to End, exercise duration was relatively longer in the control ($M = 575$, $SD = 233$) than the experimental ($M = 391$, $SD = 158$) condition.

7.4 Discussion

The present study investigated the influence of prior exhaustive activity (experimental condition) upon activation in sub regions of the frontal cortex (reflected by changes in haemodynamics) during exercise at intensities corresponding to VT and RCP. Prior exhaustive activity was completed to induce a state of increased physiological arousal (i.e. sensory [interoceptive] input from the body), to examine activation in sub regions of the frontal cortex which may impact an individual's perceptions of exercise (i.e. how hard they are working and how they are feeling).

Oxygen uptake at the point at which VT and RCP occurred was used as a marker to standardise the intensities of exercise and compare data between conditions. Heart rate was greater in the experimental condition pre-exercise, which indicated a higher state of physiological arousal prior to subsequent exercise.

The pattern of the cerebral haemodynamic response in sub regions of the frontal cortex during exercise at intensities corresponding to VT and RCP were compared in the experimental (a state of higher physiological arousal) and control conditions. Cerebral haemodynamic variables were standardised prior to exercise at intensities corresponding to VT in the control and experimental conditions. In line with hypothesis 1, reduced activation in the frontal cortex was observed in the experimental condition during exercise. Soon after the start of exercise at intensities corresponding to VT, the haemodynamic response was similar between the two conditions. During the first 10 minutes of exercise (to VT-end), relative to the control, oxygenation (ΔO_2Hb), blood volume (ΔtHb) and saturation (ΔHbDiff) were lower in the experimental condition. Oxygen extraction (deoxygenation; ΔHHb) remained relatively stable apart from an increase in region 2 (right inferior and most lateral region) and a decrease in region 7 (left superior and most lateral region) in the control condition, whereas oxygen extraction appeared to increase in all regions in the experimental condition. These findings indicate that the supply of metabolic resources (blood flow [indicated by ΔO_2Hb] and blood volume [ΔtHb]) were reduced and oxygen demand (oxygen extraction [indicated by ΔHHb]) was greater in the experimental condition at these intensities.

At the RCP, oxygenation (ΔO_2Hb), deoxygenation (ΔHHb), blood volume (ΔtHb) and saturation (ΔHbDiff) were lower in the experimental condition. During exercise from RCP to exhaustion, oxygenation (ΔO_2Hb) remained stable, oxygen extraction (ΔHHb) and blood volume (ΔtHb) increased and saturation (ΔHbDiff) decreased in the experimental condition. This indicates a reduced oxygen supply (ΔO_2Hb) and lower availability (i.e. saturation; ΔHbDiff) relative to demand (ΔHHb). At the same exercise intensities, oxygenation (ΔO_2Hb), deoxygenation (ΔHHb) and blood volume (ΔtHb) increased and saturation (ΔHbDiff) remained stable in the control condition. This indicates a greater oxygen supply (ΔO_2Hb) and availability (i.e. saturation; ΔHbDiff) relative to demand (ΔHHb).

These findings show that relative to the control condition, there was a reduced capacity of the frontal cortex (i.e. metabolic resources available) in the experimental condition during exercise at intensities from VT to exhaustion. The pattern of the cerebral haemodynamic response in the experimental condition is similar to that shown to occur during prolonged fatiguing exercise (Rupp et al., 2013). These results suggest that intensified sensory (interoceptive) input from the body (physiological arousal) from prior exhaustive exercise reduces activation in the frontal cortex.

Participants reported their perceived exertion and affective responses during exercise and provided some support for hypothesis 2. The RPE were higher and affective responses were lower in the experimental condition during exercise at intensities corresponding to VT and RCP. The RPE increased in both conditions and affective responses declined in the experimental condition. However, between VT and VT-end affect was stable in the control condition. Therefore, in the experimental condition participants perceived that they were working harder and felt worse during exercise at intensities at VT, despite a similar heart rate between the two conditions. It is suggested that these responses are due to intensified interoceptive input following the prior exhaustive activity.

At intensities corresponding to RCP to exhaustion, RPE and affective responses were similar in the experimental and control conditions. Participants perceived they were working at the same level of exertion and their affective responses were similarly negative in both conditions. This is despite the reduced cerebral haemodynamic response observed in the experimental condition at these intensities. Therefore, prior exhaustive activity influenced perceived exertion and affective responses during exercise at workloads from VT to RCP, but not from RCP to Exhaustion. Eston et al. (2007) showed that at the onset and end of a constant-load cycling exercise (75% $\dot{V}O_{2peak}$) to exhaustion, RPE was similar in a fatigue and non-fatigued state. However, RPE was different during exercise.

Affective responses were less negative in the control condition at the end of the VT period, even though responses in both conditions were similarly negative at the point of RCP. In line with the DMM, the negative affective responses reported at RCP in both conditions were attributed to the intense interoceptive processes that occur at these intensities. As indicated earlier, previous studies have shown that affective responses are uniformly negative at intensities at RCP (Ekkekakis et al., 2011). An alternative explanation may be

due to the study methods. Oxygen uptake was used as a marker to match the cerebral haemodynamic and perceptual responses to the point of VT and RCP in the two conditions. In the control condition, both the VT and RCP occurred later (by approximately 80 seconds) into the exercise session. The decline in affective responses observed in the control condition at RCP may be due to exercising for a slightly longer duration to reach the point of RCP.

The shorter exercise duration to reach VT and RCP in the experimental condition provides evidence that prior exhaustive activity influenced physiological arousal (indicated by the greater pre-exercise heart rate and the higher overall RPE), which potentially influenced the cerebral haemodynamic response. Furthermore, in support of hypothesis 3, despite no differences in the perceptual (RPE and affective) responses between the experimental and control conditions during exercise at workloads corresponding to RCP to exhaustion, participants exercised for less time in the experimental condition.

The findings of this study provide some support for the DMM (Ekkekakis, 2003; Ekkekakis & Acevedo, 2006; see 2.1.3). The DMM proposes that the competition between cognitive processes and sensory (interoceptive) inputs to the amygdala are influenced by the shift in metabolic resources (i.e. oxygen supply in the blood) from the frontal cortex to subcortical areas of the brain during exercise at challenging intensities (i.e. above VT). This mechanism is proposed by the transient hypofrontality hypothesis (Dietrich 2003, 2006; see 2.3.3). This hypothesis could potentially explain the reduced cerebral haemodynamic responses (i.e. smaller ΔO_2Hb, ΔtHb and $\Delta HbDiff$) in the frontal cortex observed at intensities corresponding to VT and RCP in the experimental condition. It is proposed that metabolic resources (i.e. blood flow [indicated by ΔO_2Hb]) were re-allocated to subcortical areas of the brain involved in sensory and motor processes (Dietrich 2003, 2006; see 2.3.3). Affective responses reported during exercise at intensities corresponding to VT and VT-end may have been influenced by prior exhaustive activity. Potentially, the completion of prior exhaustive activity may have led to the reduced function of the frontal cortex and triggered a more negative affective response during exercise at VT due to increased activity in the amygdala. In line with the DMM, hypoactivation in the frontal cortex would shut down the 'high road' of cognitive inputs to the amygdala and a more negative (aversive) state would be generated (see 2.2.1 and Figure 2.5). Although not measured in this study, it is proposed that by the same mechanism a more negative

affective response would be observed during exercise at intensities below VT following exhaustive activity.

At intensities corresponding to RCP to exhaustion, affective responses were uniformly negative in both conditions and presumed to be regulated primarily by subcortical areas of the brain via the 'low road'. It is speculated that the reduced activation in the sub-regions of the frontal cortex, due to prior exhaustive activity, did not directly influence affective responses at these higher intensities as affective responses are uniformly negative at these intensities anyway. As previously inferred, the sensory (interoceptive) inputs to the amygdala would already be the predominant mode of input to the amygdala, and this would explain the similar negative affective responses reported in both conditions at these intensities. However, these explanations remain speculative as no measure of activity in the amygdala was recorded.

Asymmetrical differences were observed for oxygen extraction (ΔHHb) between the experimental and control conditions at intensities from VT to VT-end. Oxygen extraction (ΔHHb) was greater in the right inferior region (2; most lateral) than the left superior region (7; most lateral) and remained stable in other regions in the control condition. However, oxygen extraction (ΔHbDiff) appeared to increase in all regions in the experimental condition. These responses, coupled with the reduced oxygen supply (ΔO_2Hb) in all regions in the experimental condition, indicate that activation, particularly in these regions (2 and 7), was influenced by exercise in a state of intensified sensory (interoceptive) input from the body.

At the end of exercise, blood volume (ΔtHb) was greater in the right inferior region (2; most lateral) and left inferior region (6; closest to midline) in the control condition. It appears that blood volume in the right inferior region was driven by deoxygenation (ΔHHb) whereas blood volume (ΔtHb) in the left inferior region was driven by greater oxygenation (ΔO_2Hb) in the control condition. These findings indicate less pronounced asymmetrical differences in blood volume (ΔtHb) at the end of exercise due to prior exhaustive activity.

The different patterns of activation of the right and left hemispheres in the control condition may indicate processes involved in cognitive control (Ochsner & Gross et al., 2004; see 2.3.1) and motivational approach mechanisms (Davidson, 1993; see 2.3.2).

However, this pattern of activation was not observed during exercise in the experimental condition. It is proposed that prior exhaustive activity potentially disrupts these processes. During exercise at VT, affective responses were more negative in the experimental condition, which would suggest that differential activation patterns (indicated by ΔHHb) in regions of the frontal cortex could influence the regulation of affective responses at these intensities.

In the left hemisphere, oxygenation (ΔO_2Hb), blood volume (ΔtHb) and oxygen saturation (ΔHbDiff) were greater in inferior regions in the left hemisphere (regions 6 and 8) than other regions of the frontal cortex in both conditions. Increased activation in the left hemisphere may be due to cognitive processes involved in approach-motivation towards the completion of exercise (Davidson, 1993). The pattern of activation (i.e. ΔHHb and ΔtHb) in the right hemisphere in the present study appears to have been influenced by prior exhaustive activity.

In summary, the current findings provide an insight into the effects of prior exhaustive exercise (used to induce a state of increased sensory and physiological arousal) upon activation in sub regions of the frontal cortex during constant-load exercise at intensities at VT and RCP to exhaustion. Activation in the frontal cortex was reduced and individuals reported working harder and feeling worse during exercise at intensities corresponding to VT following prior exhaustive activity. Furthermore, differential activation patterns were shown in sub regions of the frontal cortex during exercise to exhaustion due to prior exhaustive activity.

This study provides evidence that prior exhaustive activity influences activation in the frontal cortex and affective responses to exercise at intensities from VT to exhaustion. It is theorised that at these intensities of exercise the frontal cortex is less capable of maintaining dominant inputs to the amygdala via the 'high road' and that interoceptive input from the body drives activity in the amygdala via the 'low road'. However, this study did not provide a measure of activity in the amygdala to understand the interplay between cognitive and interoceptive modes described by the DMM to regulate affective responses. Chapter 8 (study V) attempts to explore the influence of activity in the amygdala during exercise at different intensities.

Chapter 8

Study V: Cerebral blood flow in the frontal cortex and activity in the amygdala during exercise: The influence of self-reported tolerance

The previous chapters have examined the capacity of the frontal cortex to understand the neural basis of affective responses during exercise at different intensities. However, no measures of activity in the amygdala, theorised to play a role in the regulation of affective responses to exercise intensities above VT, in line with the DMM, have been provided. Therefore, this chapter examines the two neural components (frontal cortex and amygdala) proposed to influence affective responses to exercise at different intensities. Furthermore, the influence of tolerance to the intensity of exercise was examined.

The idea for the use of acoustic startle methodology in this study was conceived by Associate Professor Panteleimon Ekkekakis.

This study formed the basis of an oral presentation at the 23rd Annual Australasian Society for Psychophysiology, Wollongong, Australia, November 2013. Tempest, G., Parfitt, G., Ekkekakis, P. *Insight into the neural basis of why we feel how we feel during exercise.*

8.1 Background

As demonstrated in studies I-IV in this thesis, the use of NIRS during exercise has been able to capture activation in the frontal cortex, reflected through changes in the cerebral haemodynamic response. The findings have so far examined the functional capacity of the frontal cortex during exercise at different intensities. The DMM (see 2.1.3) proposes that affective responses are influence by the interplay between cognitive and sensory (interoceptive) inputs to the amygdala (see Figure 2.5). At intensities below VT, it is proposed that affective responses are predominantly regulated by cognitive processes via the 'high road'. At intensities of exercise above VT (proximal to RCP), affective responses are predominantly regulated by sensory (interoceptive) input via the 'low road'. No evidence of amygdala activity has been shown during exercise in humans thus far.

The methods available to measure activity in the amygdala during exercise are limited due to (a) its position deep within the temporal lobes and (b) lack of neuroimaging techniques available for use during dynamic exercise. However, in affective neuroscience, the startle response has been widely used to provide an indication of central nervous system activity in subcortical lower-order brain structures to investigate appetitive and defensive motivational states (Grillon & Baas, 2003; Lang, Bradley & Cuthbert, 1998). Appetitive and defensive motivational states are similar to approach-withdrawal type motivational behaviours described by Craig (1996, 2006) and Davidson (1993, 2001).

The startle response (or reflex) is an evolutionarily protective defence mechanism triggered when potentially dangerous or threatening stimuli are detected. Using acoustic stimuli, the most widely studied component of the startle response is the acoustic startle eye blink response (ASER). The eye blink response occurs upon hearing a sudden loud burst of sound in which the autonomic system prepares for action. The neural circuitry of the ASER has been extensively studied in animal models (see Davis, Walker & Lee, 1999). The ASER includes two pathways; a primary obligatory circuit, involving the cochlear nucleus, nucleus reticularis pontis cauldalis and the facial motor nucleus; and a secondary modulatory circuit, which includes projections to the reticular site from the central nucleus of the amygdala (Davis et al., 1999; Koch, 1999). Upon hearing a loud burst of sound the strength of the eyelid contraction as it closes indicates a change in the amplitude of the response (i.e. a measurable change in the height of the eye blink trace). The change in

amplitude to affective (positive, neutral or negative) stimuli has been attributed to the modulatory response of the secondary pathway due to the involvement of the amygdala (Davis et al., 1999).

It has been repeatedly shown that when presented with an acoustic startle probe (i.e. a loud burst of noise) in the presence of aversive stimuli, the amplitude of the eye blink response is increased. However, in the presence of appetitive or novel stimuli, the amplitude of the eye bink response is decreased (Dawson, Schell & Bohmelt, 1999). The ASER is more robust when an acoustic probe is paired with aversive stimuli, which has been shown to induce a stronger potentiated response, as opposed to the less robust effects of a reduced response with neutral and positive stimuli (Skolnick & Davidson, 2002).

Some studies have attempted to examine the effects of exercise on mood states by measuring the eye blink response. O'Connor and Petruzzello (1992) investigated the anxiety-reducing effects of exercise upon spontaneous eye blink rate (used to infer central nervous system activity) following an acute bout of exercise. The authors indicated no changes in eye blink rate following 15 or 30 minutes treadmill exercise at 75% peak oxygen power ($\dot{V}O_{2peak}$). In a similar study, Tieman et al. (2001) used acoustic startle stimuli and indicated no changes in eye blink response following 20 minutes cycling exercise at 40% and 75% $\dot{V}O_{2peak}$, concluding that metabolic arousal through physical exertion did not influence the ASER. However, Smith, O'Connor, Crabbe and Dishman (2002) indicated that when paired with affective stimuli (positive, neutral, negative pictures) the ASER was reduced following 25 minutes cycling exercise at 40% and 70% $\dot{V}O_{2peak}$. The nature of the pre-post-test study designs employed by these studies does not reflect the influence exercise may have upon ASER during exercise.

Smith and O'Connor (2003) compared responses to positive, neutral and negative affective stimuli during 20 minutes cycling at 20% and 50% aerobic power. Data showed that positive affective stimuli were associated with decreased ASER during cycling. The authors report that reliable ASER measures could be recorded during exercise, but that the two intensities chosen in their study may not have evoked sufficiently different affective responses to influence a change in motivational state (i.e. a change in amplitude). This study provides support for the potential application of the technique to be used during exercise; however these methods have not been used during exercise at intensities above

50% aerobic power. Smith and O'Connor (2003) raised concerns that motor activity may a) diminish the response of the facial motor nucleus in the primary pathway; or b) interfere with the modulation of the motivational (appetitive or defensive) state in the secondary pathway.

The methods employed to measure the ASER in the studies above (and in most ASER studies) is electromyography (EMG). Electrodes are placed over the orbicularis oculi (the facial muscle which closes the lower eyelid during blinking). Following the administration of the acoustic probe the responsiveness (speed of contraction) provides a measure of the eye blink amplitude. However, exercise-induced changes in muscle tension (Smith & O'Connor, 2003) and sweat beneath the EMG electrodes may be a concern when using EMG to measure ASER during exercise, especially at higher intensities. Therefore a potential solution may be the use of a photo-electric cell which measures eye blinks using infrared reflectance. This technique involves placing a photo-electric cell in front of an individual's eye which measures the change in the light reflected off the eye when the individual closes the eye lid (Blumenthal et al, 2005). The amount of light reflected from the eye is proportional to the degree of lid closure. Amplitude is calculated as the difference from the onset (first inflection at the start of the response) to the peak (highest point) of the response indicating the strength of contraction similar to EMG measures. This technique has been compared with EMG measures during a prepulse inhibition startle design which showed adequate sensitivity for detecting the ASER (Lovelace, Elmore & Filion, 2006). The authors recommended the use of this technique for startle paradigms investigating the modulation of affective states.

The use of the ASER technique incorporating light reflectance would provide a non-contact and non-invasive measure of the modulatory role and involvement of the amygdala upon an individual's motivational and affective state during exercise. Light reflectance, as opposed to EMG methods should reduce interference of a) non-startle related facial movements induced by challenging exercise intensities, and b) potential effects of the muscle tension, heat, sweat etc. which would otherwise interfere with EMG signal.

The combined use of NIRS and the ASER technique allows activation of the frontal cortex and activity in the amygdala to be measured during exercise at different intensities. In line with the DMM, the interplay between the frontal cortex (cognitive processes) and potentially the amygdala (driven by increased sensory inputs from the body) which

determines the pattern of affective responses during exercise can be explored. The addition of the assessment of self-reported tolerance to the intensity of exercise allows the influence of this trait upon the competition between cognitive and sensory processes, which impact affective responses to be examined (see 2.4.1.1).

The present study explores activation (reflected by changes in cerebral haemodynamics using NIRS) in the dorsolateral region of the frontal cortex (both left and right hemispheres) and the amygdala (reflected by amplitude of the ASER) during constant-load exercise (standardised to GETs) 80 % aerobic power below VT (below VT), VT and RCP. The influence of frontal cortex activation and activity in the amygdala upon affective responses will be examined. Further, the influence of tolerance of the intensity of exercise upon activation in the frontal cortex, activity in the amygdala and affective responses will be determined.

Five hypotheses are examined: 1) Activation in the frontal cortex increases from below VT to VT and RCP; 2) Amplitude of the eye blink response is different at each intensity of exercise; 3) Affective responses are positive during exercise at intensities below VT and negative during exercise at intensities at RCP; 4) Activation in the frontal cortex and amplitude of the eye blink response are associated with affective responses during exercise; and 5) Self-reported tolerance is associated with activation in the frontal cortex, amplitude of the eye blink response and affective responses.

8.2 Methods

Participants

Participants (n= 20, females =12; age, M = 24.2, SD = 5.8 years; height, M = 172.0, SD = 7.8 cm; body mass, M = 69.5, SD = 10.4 kg) were recruited from a university campus.

Measures

Cerebral haemodynamics in the left and right dorsolateral region of the prefrontal cortex were measured using dual-channel NIRS as previously described (see 3.3.3.2). Approximate locations of the regions (1-2) measured are shown in Figure 8.1.

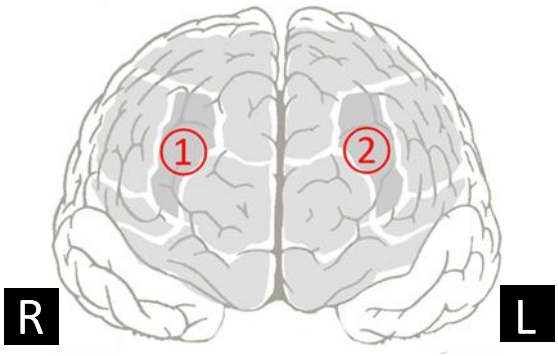


Figure 8.1. Approximate locations of the dorsolateral regions (1-2; right and left hemispheres) of the frontal cortex measured using dual-channel NIRS.

Affective (pleasure-displeasure) responses were recorded using the Feeling Scale (Hardy & Rejeski, 1989). The PRETIE-Q was used to assess individual self-reported tolerance of the intensity of exercise (Ekkekakis et al., 2005a). Participant's self-reported tolerance scores (n = 19; one participant did not complete the PRETIE-Q) in the present study ranged between 19 and 32 (M = 29, SD = 4).

Acoustic startle eye blink setup

The ASER hardware (SRH-LAB, San Diego Instruments, San Diego, CA) comprised of an infrared photo-electric cell which was mounted on an adjustable wire arm and attached to a head strap. The cell was aligned with the sclera of the participant's left eye. The front edge of the cell was just far enough from the eye to avoid the eyelashes touching when the participant blinked. Light reflected off the eye was recorded as the eyelids closed in response to an acoustic stimulus. Participants were required to focus their eyes straight ahead towards a fixed point to minimise interference due to unnecessary eye movement during recording (for infrared methods see Lovelace et al., 2006). The acoustic probe was a 50 millisecond (ms), 110 decibel (db) burst of white noise with a near-instantaneous rise/fall time delivered binaurally through headphones. Continuous broadband white noise at 60 db was delivered during a 5 minute acclimatisation period immediately prior to the first probe administered, which continued throughout recording to control for background noise. A trial is defined as a single probe (110 db burst of white noise) and its associated eye blink response. Each trial was separated with variable inter-trial intervals (between 18-30 sec).

Procedures

The study required participants to visit the laboratory on four occasions. On the first visit, participants were screened (i.e. completed the informed consent form and exercise testing questionnaire), initial assessments were taken (see 3.3.1) and completed the PRETIE-Q. Participants were seated on a recumbent cycle ergometer and their shoulders and torso were secured using velcro straps and they were given an inflatable neck pillow and to minimise movement. The ASER headgear was then applied.

Participants completed a 10 minute ASER paradigm to (a) provide within-session reliability of the ASER for 10 minute duration and (b) familiarise the participants with the ASER protocol. Following a 5 minute acclimatisation period (continuous broadband white noise at 60 db), the participants sat passively and were asked to keep their eyes fixed to a given point while ASER measures were recorded for 10 minutes. These responses were not included in the experimental analysis but provided baseline measures and within-session reliability of the ASER for 10 minute duration. Following a short break, the participants were fitted with a facemask (to measure metabolic data). Participants completed an incremental exercise test to volitional exhaustion (20 W/min) as previously described (see 3.3.3). Gas exchange thresholds (VT and RCP) were identified (see 3.4.1) and workloads corresponding to exercise intensities were determined (see 3.4.2).

The participants returned for a further three (experimental) sessions which involved exercising at a workload corresponding to below VT or VT for 10 minutes, or 5 % below RCP to exhaustion (in a randomised, counterbalanced order to control for potential between-session order effects). The NIRS optodes were carefully positioned (see 3.3.2) and the ASER headgear and facemask were applied. Following a 5 minute warm up (20 W) during the ASER acclimatisation period (continuous broadband white noise at 60 db), participants began exercise at their individually determined workloads as the first ASER trial was administered. Cerebral haemodynamics, expired gases and heart rate were collected continuously. Participants reported their affective responses pre-exercise, and every minute during exercise (between startle probes).

Data Reduction

Eye blink responses were acquired, processed and scored using SRH-LAB software. Eye blink amplitudes were calculated by subtracting 50 ms pre-trial baseline activity from the greatest activity between 20-150 ms following probe onset (peak response). Trials with no perceptible eye blink response, or a pre-trial baseline activity greater than three standard deviations about the mean were excluded from further analysis. Eye blink responses measured in the first session were scored and averaged every minute over the 10 minute duration for within-session reliability analysis. In accordance with Blumenthal et al. (2005) and Skolnick and Davidson (2002), eye blink responses recorded during exercise were z-transformed within-subject and between-sessions (below VT, VT and RCP). Response probabilities (%) from each session (below VT, VT and RCP) were calculated as the number of scored trials divided by the number of administered trials.

At exercise intensities below VT and VT, once a steady metabolic state at the determined intensities had been achieved (approx. 4 minutes; Wasserman, 1994) the mean of each variable (NIRS [ΔO_2Hb, ΔHHb, ΔtHb, ΔHbDiff], ASER [amplitude] and affective responses) were calculated between minutes 5 to 10. At intensities of exercise 5% below RCP, a mean was calculated from the point at which each participant's RCP occurred (indicated by $\dot{V}O_2$l/min) until minute 10 or until exhaustion, whichever occurred first. The group mean and standard deviation of exercise duration (sec) from the point of RCP to minute 10 or exhaustion was calculated.

Statistical analyses

The independent variables were Region (right, left) and Intensity (below VT, VT, RCP). The dependent variables were changes in cerebral haemodynamics (primary [ΔO_2Hb, ΔHHb] and secondary [ΔtHb, ΔHbDiff]), amplitude of the eye blink response, affective responses and exercise duration (seconds).

To examine hypothesis 1, a series of Region (2; right, left) by Intensity (3; below VT, VT, RCP) analysis of variance (ANOVA) with repeated measures were conducted for each primary (ΔO_2Hb, ΔHHb) and secondary (ΔtHb, ΔHbDiff) cerebral haemodynamic variables. To examine hypotheses 2 and 3, one-way Intensity (below VT, VT and RCP) ANOVAs with repeated measures were conducted for amplitude of the eye blink response

and affective responses, respectively. Finally, to examine hypotheses 4 and 5, Pearson's bivariate correlation analyses were conducted to examine the relationships between; cerebral oxygenation, amplitude of the eye blink response and affective responses (hypothesis 4); and self-reported tolerance, cerebral oxygenation, eye blink amplitude and affective responses (hypothesis 5).

8.3 Results

8.3.1 Preliminary analyses

Within-session reliability of the ASER protocol (trials administered over 10 minute duration) was verified using one-way random effects Intra-class Correlations (*R*) and Cronbach's alpha (α). This analysis showed that amplitude of the eye blink response between each minute of the baseline measure indicated high concurrence, $R = .77$ ($\alpha = .97$).

Eye blink response probabilities (%) for each intensity were generally high; below VT ($M = 94$, $SD = 15$), VT ($M = 94$, $SD = 10$) and RCP ($M = 85$, $SD = 20$). Mean exercise duration at intensities of exercise corresponding to RCP (point of RCP to minute 10; $n = 14$, or exhaustion; $n = 6$) was, $M = 395$ ($SD = 74$) seconds.

To ensure the intensity of exercise ($\dot{V}O_2$ l/min) was the same between the initial incremental exercise test (IET; session one) and the experimental sessions at the corresponding intensities; below VT, VT or RCP, a Session (2) by Intensity (3) analysis of variance (ANOVA) with repeated measures was conducted. The analysis showed that $\dot{V}O_2$ (l/min) below VT ($M = 1.22$, $SD = .23$), VT ($M = 1.51$, $SD = .26$) and RCP ($M = 2.42$, $SD = .52$), were significantly different, $F(1,20) = 261.44$, $p < .001$, $\eta_p^2 = .93$. No significant main effect of Session or Session by Intensity interaction was recorded ($p > .05$).

The means and standard deviations (hypothesis 1) and correlation coefficients (hypothesis 4 and 5) of the cerebral haemodynamic variables (μM) in this study are provided in Appendix 3e.

8.3.2 Cerebral haemodynamics (hypothesis 1)

Cerebral ΔO_2Hb

Significant main effects for Region, $F(1,19) = 4.62$, $p = .045$, $\eta_p^2 = .20$, and Intensity, $F(2,38) = 20.70$, $p < .001$, $\eta_p^2 = .52$, and a significant Region by Intensity, $F(2,38) = 3.52$, $p = .040$, $\eta_p^2 = .16$, interaction were recorded for ΔO_2Hb (μM). Follow up tests indicated that ΔO_2Hb was greater in the left ($M = 5.3$, $SD = 2.5$) than the Region in the right ($M = 4.3$, $SD = 2$) hemisphere and increased as the intensity of exercise increased; below VT ($M = 2.20$, $SD = 1.66$), VT ($M = 5.28$, $SD = 2.16$), and RCP ($M = 7.00$, $SD = 4.01$). The interaction indicated greater ΔO_2Hb in the left hemisphere at RCP (see Figure 8.2).

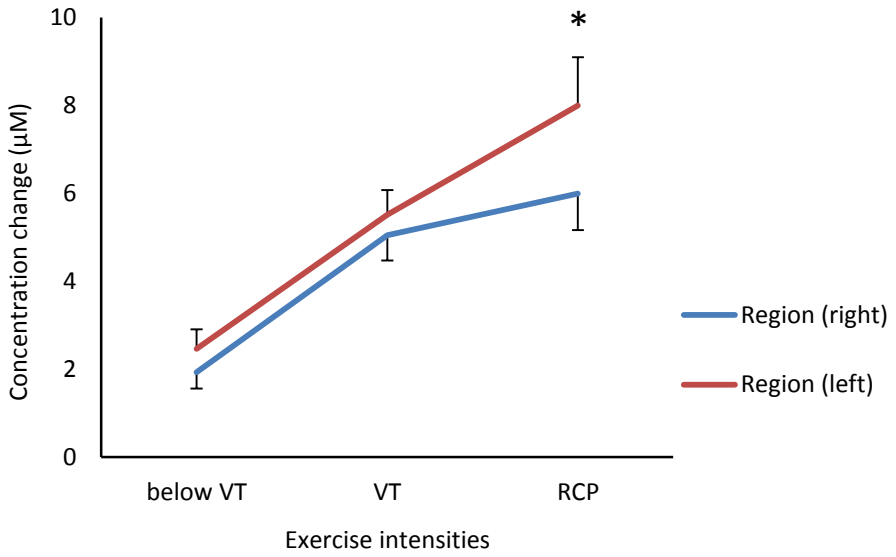


Figure 8.2. Cerebral oxygenation (ΔO_2Hb) in regions of the frontal cortex at each intensity of exercise. *M* and *SEM*. * Significantly greater ΔO_2Hb in the left than right region.

Cerebral ΔHHb

A significant main effect of Intensity, $F(2,38) = 13.54$, $p < .001$, $\eta_p^2 = .42$, was recorded for ΔHHb (μM). Follow up tests indicated that ΔHHb was similar at intensities below VT ($M = -.46$ $SD = 1.27$) and VT ($M = -.53$, $SD = 1.18$), and greater at RCP ($M = 1.48$, $SD = 2.15$).

Cerebral ΔtHb

A significant main effect of Intensity, $F(1,27) = 48.82$, $p < .001$, $\eta_p^2 = .72$, was recorded for ΔtHb (μM). Follow up tests indicated that ΔtHb was greater as the intensity of exercise increased; below VT ($M = 1.74$, $SD = 1.08$), VT ($M = 4.31$, $SD = 1.90$) and RCP ($M = 8.25$, $SD = 3.47$).

Cerebral ΔHbDiff

A significant main effect of Intensity, $F(2,38) = 5.95$, $p = .006$, $\eta_p^2 = .24$, and a significant Region by Intensity, $F(1,19) = 4.95$, $p = .012$, $\eta_p^2 = .21$, interaction was recorded for ΔHbDiff (μM). Cerebral ΔHbDiff was lower at intensities below VT ($M = 2.66$, $SD = 2.66$) and greater at intensities VT ($M = 5.81$, $SD = 2.98$) and RCP ($M = 5.52$, $SD = 5.37$). The interaction indicated greater ΔHbDiff in the region in the left hemisphere at RCP (see Figure 8.3).

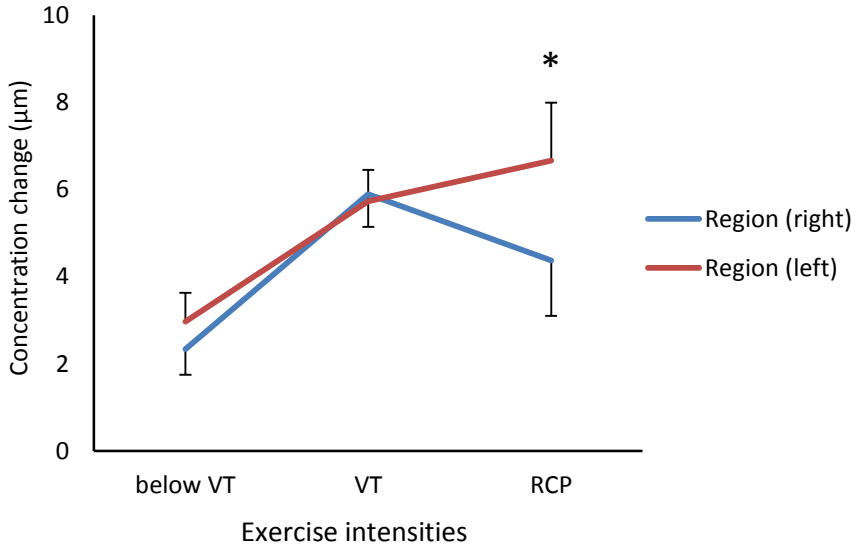


Figure 8.3. Cerebral oxygen saturation (ΔHbDiff) in regions of the frontal cortex at each intensity of exercise, *M* and *SEM.* * Significantly greater ΔHbDiff in the left than right region.

8.3.3 Acoustic startle eye blink response (hypothesis 2)

A significant main effect of Intensity, $F(2,38) = 3.28$, $p < .05$, $\eta_p^2 = .15$) was recorded for amplitude of the eye blink response. Follow up tests indicated that amplitude of the eye blink response reduced as the intensity of exercise increased; below VT ($M = -.01$, $SD = .23$), VT ($M = -.43$ $SD = 1.02$), and RCP ($M = -.66$ $SD = 1.18$).

8.3.4 Affective responses (hypothesis 3)

A significant main effect for Intensity, $F(2,38) = 130.17$, $p < .001$, $\eta_p^2 = .87$, was recorded for affective responses. Follow up tests indicated affective responses declined as the intensity of exercise increased; below VT ($M = 2.8$, $SD = 1.1$), VT ($M = 1.8$, $SD = 1.5$) and RCP ($M = -2.8$, $SD = 1.7$).

8.3.4 Correlations (hypothesis 4 and 5)

Cerebral oxygenation, amplitude of the eye blink response and affective responses below VT, VT and RCP (hypothesis 4)

Cerebral ΔO_2Hb in the right and left hemisphere indicated a significant relationship with amplitude of the eye blink response at VT, $r = .46$, $p = .040$, and $r = .55$, $p = .013$, respectively. Cerebral ΔO_2Hb in the right hemisphere indicated a significant inverse relationship with affective responses at RCP, $r = -.51$, $p = .023$.

The relationships between amplitude of the eye blink response and affective responses were not significant but positive below VT, $r = .38$, $p > .05$, and marginally negative at VT, $r = -.18$, $p > .05$. However, a significant inverse relationship was shown at RCP, $r = -.51$, $p = .022$. The decline in amplitude of the eye blink response and affective responses across intensities (RCP minus below VT) indicated a significant inverse relationship, $r = -.46$, $p = .040$.

Self-reported tolerance, cerebral oxygenation, amplitude of the eye blink response and affective responses (hypothesis 5)

Self-reported tolerance was inversely associated with amplitude of the eye blink response at RCP, $r = -.50$, $p = .031$, and the decline in amplitude of the eye blink response from below

VT to RCP, r = -.47, p = .042. Self-reported tolerance was positively associated with the decline in affective responses from below VT to RCP, r = .50, p = .031.

8.4 Discussion

The present study investigated activation in the frontal cortex (reflected by changes in cerebral haemodynamics using NIRS), activity in the amygdala (reflected by amplitude of the ASER) and affective responses during constant-load exercise at intensities below VT, VT and RCP. The influence of self-reported tolerance of the intensity of exercise upon activation in the frontal cortex, activity in the amygdala and affective responses were also examined. Based on hypotheses generated from the DMM, it was proposed that the interplay between cognitive processes in the frontal cortex and sensory input to the amygdala determine affective responses during exercise. The DMM has not been tested fully due to the lack of methods available to measure activity in the amygdala. This study was designed to provide evidence of activity in the amygdala by measurement of the ASER during exercise at intensities standardised to physiological markers (VT and RCP). This study combined the use of NIRS and measurement of the ASER using infrared reflectance, as opposed to EMG methods during exercise. Within-session reliability of the ASER over a 10 minute duration at baseline revealed a high concurrence of eye blinks minute to minute. The response probabilities of the eye blink responses during exercise at different intensities were generally high (> 85%), but large variability was indicated. These preliminary checks provide initial support for the current study design that a) stable eye blink responses can be achieved during a 10 minute session; and b) the infrared reflectance method can record a high percentage of eye blinks associated with the acoustic probe during exercise at different intensities.

Cerebral haemodynamics were recorded in the right and left dorsolateral regions of the frontal cortex at intensities of exercise below VT, VT and RCP and the findings are in line with hypothesis 1. At intensities of exercise at VT, oxygenation (ΔO_2Hb), blood volume (ΔtHb) and saturation (ΔHbDiff) were greater than, and oxygen extraction (deoxyhaemoglobin; ΔHHb) remained similar to, intensities below VT. The increased cerebral haemodynamic response indicated adequate oxygen supply (ΔO_2Hb) relative to demand (ΔHHb) between the two intensities.

At intensities of exercise at RCP, oxygenation (ΔO_2Hb), oxygen extraction (ΔHHb) and blood volume (ΔtHb) were greater than, and saturation (ΔHbDiff) remained similar to VT. A greater oxygen supply was met with a higher oxygen demand at intensities at RCP. Oxygenation (ΔO_2Hb) and saturation (ΔHbDiff) were greater in the left than the right hemisphere at RCP. The haemodynamic responses indicated an increased oxygen supply and demand in the frontal cortex as the intensity of exercise increased from below VT to RCP. These findings are consistent with the cerebral haemodynamic response observed during incremental exercise in studies I and II. The asymmetrical activation (left greater than right hemisphere) may be the result of cognitive processes involved in approach-type motivational behaviours to continue exercise at higher intensities of exercise (Davidson, 1993). Greater left than right activation at these intensities was shown in studies II and IV.

The previous studies within this thesis have focussed upon activation in the frontal cortex and provided a theoretical explanation of the role of the amygdala in the regulation of affective responses to the intensity of exercise. In line with hypothesis 2, this study employed ASER techniques to provide evidence of amygdala activity during exercise at different intensities to examine the sensory (interoceptive) mode of the DMM (see 2.2.2). Due to inter- and intra-individual variability in the ASER response within and between sessions, the amplitude of the ASER response was standardised across intensities for each individual in line with previous methods (Smith et al., 2002; Skolnick & Davidson 2002). Previous findings have commonly reported that the amplitude of the ASER response decreases in the presence of positive, and increases in the presence of negative affective stimuli. However, the amplitude of the ASER responses in this study declined as the intensity of exercise increased. The decline in ASER is contrary to the expected response. The ASER response should have increased as the intensity of exercise increases and affective responses declined (i.e. greater amplitude = more negative affect the higher the intensity).

To investigate the reduction in ASER amplitude, post-hoc two-step hierarchical regression analyses were conducted to explore variance in the ASER at each intensity. Step one included physiological variables ($\dot{V}O_2$, heart rate and partial pressure end-tidal carbon dioxide) as a composite z-scores to examine the physiological effects of exercise. Step two included cerebral oxygenation and affective responses. No significant models were shown and given the sample size was low (n = 20), these results were not presented. The

physiological variables were expected to explain variance in the ASER and may have accounted for the reduction in the ASER as the intensity of exercise was increased. However, as the ASER data were standardised to account for inter- and intra-individual variability within and between sessions (i.e. across the intensities), the influence of the physiological variables may have been masked.

There are a number of other potential contributions which may explain the reduction in amplitude of the ASER across exercise intensities. Firstly, the application of the ASER technique during exercise, a task which requires great attentional and physiological demand, may leave fewer resources available that can be directed by the startle probe, resulting in a diminution in amplitude (Hackley, 1999) as the exercise intensity increases. Secondly, Smith et al. (2003), hypothesised that a diminished ASER response during exercise may be due to active engagement in an appetitive-behaviour to evade defensive-behaviour in the presence of a threat. This may inadvertently cue a reduction in the ASER in the presence of negative or aversive stimuli. As the ASER is a defensive reflex not influenced by socio-cognitive mechanisms, it may be that associative physiological processes or physiological interference induced by exercise may have influenced motivational engagement. Finally, Tieman et al. (2001) proposed that ASER amplitude would be reduced following exercise (75% $\dot{V}O_{2peak}$) on the basis of pharmacological evidence which indicates that increases in neurotransmitters (norepinephrine, serotonin and dopamine) associated with anxiety-reducing effects during exercise may serve to dampen the ASER. These explanations remain speculative and require further investigation to understand the reduction in the ASER as the intensity increased.

Participants reported their affective responses during exercise at each intensity. Affective responses were positive at intensities below VT and VT whereas affective responses became negative at RCP (hypothesis 3). These findings are consistent with studies I and II on this thesis and in the current literature (Ekkekakis et al., 2011). The DMM indicates that maintained activation of the frontal cortex at these higher intensities of exercise is required to maintain a more positive or less negative affective response especially around VT. However, a uniformly negative affective response is associated with intensities at RCP.

In line with the DMM, a significant relationship was observed between activation in the frontal cortex and the amygdala during exercise at intensities at the VT (hypothesis 4).

Increased oxygenation (ΔO_2Hb) was related to greater amplitude of the ASER (presumably indicating increased physiological arousal, negative affect and/or motivational intensity). This finding supports the proposal of competition between frontal cortex and amygdala due to the increased physiological challenge of exercise at these intensities. Subsequently, greater cerebral oxygenation (ΔO_2Hb) in the right hemisphere was associated with a less negative affective response at RCP.

Cognitive control processes (2.3.1) and Davidson's (1993) asymmetry hypothesis may explain this relationship. Davidson suggests that the right hemisphere is associated with negative affect; however this is associated with withdrawal-type behaviours. Although, the left hemisphere showed no significant relationship, the greater activation in the left hemisphere is consistent with approach-related behaviours. Craig's (2005) theory of forebrain emotional asymmetry would suggest the right hemisphere is associated with sympathetic activity related to arousal and danger (see 2.3.3). Therefore activation in the right hemisphere may be due to homeostatic representation of the autonomic nervous system associated with withdrawal-type motivation, as theorised in study IV. Alternatively, the association of affect and oxygenation (ΔO_2Hb) in the right hemisphere may be explained by variability in the mechanisms used by individuals to counteract the negative affective or aversive stimuli through appraisal processes (Ochsner & Gross, 2004). However, without qualitative data and a specific research design to test such interpretations, these explanations remain speculative (see 9.6).

Despite, the reduction in the ASER amplitude, linear relationships between the ASER and affective responses at each intensity are of interest, but should be interpreted with some caution. At intensities below VT, a low-moderate (although, $p > .05$) correlation indicated that positive affective responses were associated with a greater ASER amplitude. Although, this appears to be in the wrong direction, research suggests that the amygdala may also become activated through attentional displacement or during novel tasks (Hackley, 1999). Affective responses were positive at these intensities. At VT, almost no correlation ($p > .05$) was shown between affective responses and the ASER. The DMM postulates that the interplay between the dual-modes (cognitive processes and sensory inputs) is greatest at these intensities (i.e. around VT) which account for variability in affective responses. It may be that no relationship was shown at these intensities due to this variability with some individuals feeling positive, and others negative at this intensity (Rose & Parfitt, 2007).

However at higher intensities of exercise (RCP) reports of negative affective responses were associated with a greater ASER. In line with previous research, and as predicted by the DMM, a negative affective response was associated with greater activity in the amygdala (i.e. potentiation of the ASER). This is potentially representative of a defensive (or withdrawal-type) motivational state through modulation of the secondary neural pathway of the ASER.

The association between cerebral oxygenation, ASER and affective responses during exercise at intensities at and above the VT provides support for the DMM. Firstly, the interaction between the frontal cortex and amygdala at intensities of exercise at VT has for the first time been indicated during exercise. Secondly, at intensities of exercise at RCP, where affective responses are uniformly negative, the amygdala appears to be somewhat involved in the regulation of affective responses during exercise. Furthermore, the greater the decline towards a negative affective state, the smaller the decline in the ASER from intensities below VT to RCP. This implies that those individuals who reported feeling more negative had less of a decline in the ASER indicating a greater ASER compared to those individuals who felt less negative. Although this finding supports the general notion that greater ASER is associated with negative affective stimuli and a defensive motivational state, the interpretation of linear bivariate correlations is limited. As was predicted by the DMM, an association was shown between activation in the frontal cortex (left and right hemispheres) and activity in the amygdala (i.e. ASER) at intensities above VT.

Self-reported tolerance of the intensity of exercise is proposed to influence activation of the frontal cortex in response to interoceptive input from the body (hypothesis 5). Tolerance describes the ability of how an individual may regulate their exercise experience during exercise which may influence the way they feel. Linear correlations with the self-report measure indicated that a high tolerance score was associated with a reduced ASER at RCP. Therefore, those individuals with high self-reported tolerance had potentially less activity in the amygdala and potentially a reduced (defensive) motivational state at RCP. In fact, those individuals with high self-reported tolerance indicated a greater decline (i.e. lower amplitude) in ASER and a smaller decline (i.e. less negative) in affective responses as the intensities of exercise increased from below VT to RCP. Self-reported tolerance, measured using the PRETIE-Q is proposed to underscore individual differences in the responses to exercise beyond somatometric, physiological and exteroceptive differences

(Ekkekakis et al., 2005a). On this basis, self-reported tolerance could potentially be linked with an individual's motivational state indicated by the ASER.

In summary, the current findings provide a novel insight into the role of the frontal cortex and amygdala during exercise in line with the DMM. Although the use of acoustic startle methods was exploratory and no validity of the response has been reported at high intensities of exercise (> 50% aerobic power), the findings suggest that the amygdala could modulate motivational and affective states during exercise at intensities above VT. For the first time, this study has provided some neurophysiological indication that the amygdala is involved in the regulation of affective responses during exercise at intensities above VT. Cognitive control processes involved in the regulation of affective responses in the frontal cortex and their relationship with the amygdala, particularly during the regulation of negative affect still require further investigation. Self-reported tolerance, assessed using a simple questionnaire, is associated with activation in the frontal cortex and activity in the amygdala during exercise above VT. These findings provide some evidence in support of the DMM and the neural basis to explain the pattern of affective responses to the intensity of exercise.

Chapter 9

Discussion and conclusions

This chapter provides a general discussion of the findings of the five studies of this thesis. The main outcomes are outlined followed by a discussion of the findings with relevance to current theories. Limitations and considerations of the current research are identified and future directions are provided. This chapter concludes with a brief summary of the neural basis of affective responses to exercise at different intensities.

9.1 Review of aims

Evidence so far has established a dose-response pattern of the intensity of exercise and the pleasure-displeasure individuals feel (Ekkekakis et al., 2011). However, the underlying neural mechanisms of why individuals feel how they feel during exercise have not been confirmed. The neural basis and mechanisms of affective responses during exercise at intensities standardised to gas exchange thresholds (i.e. VT and RCP) have previously been proposed (Ekkekakis, 2003; Ekkekakis et al., 1999). The DMM suggests that affective responses during exercise are regulated by the interplay between the frontal cortex and the amygdala (Ekkekakis, 2003; Ekkekakis & Acevedo, 2006). The frontal cortex must maintain activation through cognitive control and motivational processes to prevent deregulation in order to override activity in the amygdala driven by sensory (interoceptive) input from the body. Maintenance of frontal cortex activation would lead to a more positive or less negative affective experience as the amygdala is associated with negative or aversive states. The predisposed trait of tolerance to the intensity of exercise, which may explain why some individuals feel better than others, may influence the ability to maintain frontal cortex activation.

The primary aim of this thesis was to investigate activation in the brain (frontal cortex and amygdala) and affective (pleasure-displeasure) responses during exercise at different intensities. The secondary aim was to examine the influence of the predisposed trait of tolerance of the intensity of exercise upon activation in the brain and affective responses.

Study I examined if self-reported tolerance influenced activation in the dorsolateral region (right and left) of the frontal cortex and study II examined activation in (eight) sub regions of the frontal cortex, during incremental exercise to exhaustion. Differential and asymmetrical patterns were observed in both studies at intensities of exercise above VT. Therefore, studies III and IV examined activation in the frontal cortex during constant-load exercise at intensities corresponding to and above VT. Study III examined the influence of a cognitive strategy to increase frontal cortex activation, and study IV examined the influence of prior exercise to induce a heightened state of physiological awareness. These studies emphasized the influence of the cognitive (III) and interoceptive (IV) modes of the DMM. Finally, to further test the DMM, study V attempted to examine both activation in the frontal cortex and activity in the amygdala during exercise at different

intensities (i.e. below VT, VT and RCP) and the influence of self-reported tolerance. Affective responses to exercise were examined in each study.

In the following section, the main outcomes are outlined followed by a discussion of the findings with relevance to current theories. Limitations and considerations of the current research are identified and future directions are provided. This chapter concludes with a brief summary of the neural basis of affective responses to exercise at different intensities.

9.2 Main findings

9.2.1 Affective responses

Following recommendations by Ekkekakis et al. (1999), a dose-response pattern of affective responses to the intensity of exercise has been well-established (Ekkekakis et al., 2011). The pattern of affective responses in studies I –V indicate that at intensities of exercise below VT, affective responses are positive and above VT (i.e. RCP) affective responses are uniformly negative. The pattern of these responses is in line with the previous literature (Ekkekakis et al., 2011) and has been shown during both incremental (studies I and II) and constant-load (studies III, IV and V) exercise.

9.2.2 Cerebral haemodynamics

The use of NIRS during exercise is becoming increasing popular. However few studies have used NIRS to define the functional capacity of the frontal cortex in terms of neuronal activation, which may impact upon an individual's feeling state (i.e. affective responses). The studies in this thesis were designed to determine activation in the brain in order to understand the potential influence this may have upon the pleasure-displeasure an individual may feel at different intensities of exercise. The standardisation of the intensity of exercise, which has for over a decade been acknowledged in studies examining the pattern of affective responses (Ekkekakis et al., 1999; Ekkekakis et al., 2011), has more recently been encouraged in studies examining the haemodynamic response using NIRS during exercise (Ekkekakis, 2009; Rooks et al., 2010). In this thesis, exercise intensities were standardised to individual metabolic markers (i.e. gas exchange thresholds) corresponding to VT and RCP.

The five studies used dual or multi-channel NIRS to determine functional neuronal activation in the frontal cortex during exercise. The findings indicate that activation in the frontal cortex can be maintained (i.e. increases in oxygenation [ΔO_2Hb] coupled with increases in deoxygenation [ΔHHb]) from the start of exercise to VT and RCP. However, activation in the frontal cortex is reduced (i.e. no increase or a decrease in oxygenation [ΔO_2Hb] coupled with increases in deoxygenation [ΔHHb]) from RCP to exhaustion. These findings are in line with previous research investigating the cerebral haemodynamic response across exercise intensity domains (Rooks et al., 2010). However, Rooks et al. (2010) report large variability in the haemodynamic response across exercise intensity domains. Differences in variability between the data within this thesis and Rooks et al. (2010) may be attributed to a) the homogenous population of participants (i.e. similar age bracket and physically activity status); b) standardising the intensities of exercise to individual gas exchange thresholds rather than % of maximal oxygen uptake; c) the use of incremental or constant-load exercise; and/or d) the placement of the NIRS recording sites (from bilateral or multiple sub regions of the cortex).

This thesis contributes new evidence of the haemodynamic response from bilateral and sub regions of the frontal cortex. Firstly, asymmetrical patterns of activation in the frontal cortex were shown at intensities of exercise above VT (studies I-V). Secondly, the use of multi-channel NIRS indicated that activation in the frontal cortex appeared to shift in sub regions of the frontal cortex as the intensity of exercise increased (study II). Thirdly, these differential and asymmetrical patterns of activation were influenced by (a) self-reported tolerance of the intensity of exercise (study I), a cognitive strategy (study III) and prior exhaustive activity (study IV), during incremental (study I) and during constant-load exercise (study III, IV). Finally, activation in the frontal cortex was associated with activity in the amygdala. It is proposed that these changes in the haemodynamic response influence the regulation of affective responses (see 9.4). These changes are by no means exclusive to an explanation of the neural basis of affective responses. For example, they may explain an individual's perception of exercise (how hard the exercise feels), their decision-making processes (to continue or discontinue their behaviour) and the mechanisms involved in managing sensory input and motor output.

9.2.3 Acoustic startle eye blink response

The novel use of acoustic startle methodology (study V) was to determine activity in the amygdala during different intensities of exercise. Despite the strong theoretical basis of the role of the amygdala in the regulation of affective responses during exercise (Ekkekakis, 2003; Ekkekakis & Acevedo, 2006), little evidence has been provided. The use of acoustic startle methods was explored to provide, for the first time, an indication of amygdala activation during exercise in an attempt to understand the regulation of affective responses. Somewhat promising findings from the study are that (a) increased activation in the frontal cortex, and (b) negative affective responses were associated with activity in the amygdala at intensities of exercise at VT and RCP to volitional exhaustion, respectively. Affective responses have been shown to be uniformly negative at these intensities (Ekkekakis et al., 2004, 2008; Ekkekakis et al., 2011; as shown in studies I-V) and therefore it would be expected that amygdala activity would be more strongly related to affective responses at these intensities (Ekkekakis, 2003; Ekkekakis & Acevedo, 2006). It is unclear if the amygdala was activated through physiological arousal or attentional processes or if the eye blink response was somehow confounded due to exercise (i.e. intense physiological arousal; Hackley, 1999; Smith et al., 2002). Despite this limitation, the results provide some support that the amygdala could be involved in the regulation of affective response to exercise at physiologically challenging intensities.

9.3 The dual-mode model (DMM)

The main tenet of the DMM (Ekkekakis, 2003; Ekekakis & Acevedo, 2006), is that at intensities of exercise above VT, the frontal cortex must maintain activation in order to override the increasing sensory input from the body (i.e. during exercise) to the amygdala. The frontal cortex is involved in promoting a more positive affective state and the amygdala is associated with aversive affective states. Activation in the frontal cortex is presumed to be due to cognitive control (i.e. appraisal and suppression; see 2.3.1) and motivational (see 2.3.2) processes involved in promoting a positive or avoiding a negative affective state. According to the DMM, hypoactivation in the frontal cortex would induce a negative affective response (see 2.3.3)

Findings in this thesis support that activation in the frontal cortex is maintained during exercise up to intensities at RCP, however activation reduces near exhaustion. Linear correlations indicated an inverse relationship between affective responses and activation (oxygenation [ΔO_2Hb]; studies II and V] and oxygen extraction [deoxygenation [ΔHHb]; study I]) in the frontal cortex at intensities above VT. These findings imply that at intensities of exercise which challenge homeostasis (i.e. above VT) greater activation was associated with a more negative affective response. The DMM explains that activation in the frontal cortex, which dominates input to the amygdala via the 'high road', would override activity in the amygdala driven by sensory input from the body via the 'low road'. The frontal cortex had to work harder (increase activation) in the presence of increased somatosensory input to attenuate negative affect. Exploratory use of the acoustic startle methods showed associations between (a) increased activation in the frontal cortex (at VT) and (b) negative affective responses (at RCP), and activity in the amygdala and affective responses during exercise (study V). This suggests involvement of the amygdala during exercise at these higher intensities of exercise. The DMM would explain that around VT the frontal cortex has to work harder to override activity in the amygdala due to the increasing dominant input via the 'low road'. Furthermore, affective responses were inversely associated with activation in the right (studies II and V) and left (study II) hemispheres pertaining to the role of cognitive control processes involved in the inhibition of activity in the amygdala by the frontal cortex (see 9.4.2). The findings in this thesis provide some support for the proposed neural basis (frontal cortex and amygdala) of affective responses to exercise by the DMM.

9.4 Mechanisms of affective responses

As previously highlighted (see 1.1), the exact cognitive control and motivational processes responsible for affective responses were not explicitly recorded, but their potential involvement was theorised to explain activation in the frontal cortex (reflected by haemodynamics). Cognitive control processes and theories of frontal cortex asymmetry to explain affective states and motivational-related behaviours are embedded within the 'cognitive' mode in the framework of the DMM (Ekkekakis, 2003; Ekkekakis & Acevedo, 2006). The findings of this thesis primarily indicate the functional capacity of the frontal

cortex to host cognitive processes that may be involved in the regulation of affective response to exercise.

9.4.1 Cognitive control processes

Cognitive control processes occur in the frontal cortex and provide top-down regulation of subcortical structures (i.e. the amygdala), particularly in the presence of negative stimuli (see 2.3.1). The findings from study I suggest that individuals with high self-reported tolerance may have employed different cognitive control processes during exercise at intensities above VT, than those with low self-reported tolerance. The participants were not required to attend to or inhibit their affective responses during exercise; they were only asked to report these responses. Therefore, it is presumed the activation patterns recorded by NIRS reflected automatic cognitive control processes related to the perception and modulation of sensory input from the body described by the predisposed trait of tolerance of the intensity of exercise (see 2.4.1).

Imagery, as a cognitive strategy, was used during exercise to engage cognitive processes in the frontal cortex (study III). Unlike, the cognitive reappraisal and inhibitory control processes presented (see 2.3.1), evidence of a top-down regulatory mechanism (if any) of imagery and affective states is not possible. However, imagery use has been shown to (a) improve affective responses to moderate intensity exercise, (b) involve areas of the frontal cortex, and (c) be commonly and easily used by exercisers (see 6.1). The findings showed that imagery use increased frontal cortex activation and affective response at intensities of exercise above VT, which are commonly associated with negative affective states. These findings indicate the capacity of the frontal cortex to host cognitive processes and improve affective responses at intensities above VT using a cognitive strategy.

The use of imagery indicated no differences in frontal cortex activation relative to a control condition at higher intensities of exercise (at RCP); even though affective responses were improved (i.e. less negative). These findings indicate a somewhat limited capacity of the frontal cortex, specifically the dorsolateral regions, to host cognitive processes at intensities corresponding to RCP. It is proposed that other areas of the frontal cortex not directly measured (i.e. ventromedial and orbitofrontal regions) were involved in the regulation of affective responses at higher intensities. As described by Dietrich (2006), these regions are

second-order structures of the five-stage hierarchy of consciousness proposed to regulate affective responses and feeling states.

Following exhaustive activity a limited capacity of the frontal cortex was observed during exercise above VT and at RCP, but affective responses were no more negative than the control condition at RCP. Activation in the frontal cortex was increased, due to a cognitive strategy, and reduced, due to prior exhaustive activity (presumably because of increased interoceptive inputs from the body), at intensities of exercise above VT. These findings highlight the interplay between the frontal cortex and sensory processes at these intensities in line with the DMM. However, activation in the frontal cortex was not different, due to the use of a cognitive strategy, but reduced, due to prior exhaustive activity, at intensities above RCP. These findings indicate a dominance of sensory (interoceptive) inputs from the body at these intensities. Therefore, the capacity of the dorsolateral and surrounding sub regions of the frontal cortex appears to be limited during exercise at intensities at the RCP. This proposal is in line with the DMM and transient hypofrontality hypothesis (see 9.4.3).

Cognitive control processes may induce top-down regulation of affective responses during exercise more effectively than imagery. Reappraisal or inhibitory tasks may produce stronger activation and have a more beneficial application during exercise at intensities above VT. Cognitive control processes have indicated lateralised functions in which they influence positive and negative affective states (see 2.3.1). Appraisal of affective stimuli may influence and even strengthen the link between the frontal cortex and amygdala and improve affective responses during exercise. However, such tasks may demand too much cognitive effort and the capacity of the frontal cortex may become further compromised.

9.4.2 Frontal cortex asymmetry

A large body of converging evidence supports the representation of motivation-related affective states and frontal cortex asymmetry (Davidson, 1993, 2004), although some evidence has failed to support this hypothesis (Hagemann et al., 1998) and proposed separate motivation and affective constructs (Harmon-Jones, 2003, 2004; see 2.3.2).

Activation in the right and left dorsolateral regions of the frontal cortex were directly compared in studies I, III and V. Although these studies were not designed to specifically test the asymmetry hypothesis, the direct comparison of hemispheres has been encouraged

(Davidson & Irwin, 1999). Activation was dominant in primarily the left hemisphere during exercise at VT and RCP (study V), however activation was greatest near and at maximal intensities in the right hemisphere (studies I, II). These activation patterns may be representative of lateralised cognitive control processes involved in the regulation of negative affect (see 2.3.1).

A direct comparison of hemispheres was not conducted in studies II and IV, but activation in sub regions of the frontal cortex was examined. These findings showed different activation patterns between sub regions at intensities above VT. Activation appeared to be consistently greater in the left than the right hemisphere. However, when affective responses were uniformly negative, activation appeared to be greatest in the right (primarily inferior regions closet to the midline; the anterior frontal cortex, approximately BA10) than the left hemisphere at exhaustion. The association of greater activation in right relative to the left anterior portion of the frontal cortex and negative stimuli has been shown previously (Davidson, 1993).

Inverse relationships between affective responses and activation (oxygenation [ΔO_2Hb] and deoxygenation [oxygen extraction; ΔHHb]) in (sub) regions (left and right hemispheres) of the frontal cortex were observed at intensities above VT (studies I and II). However, the directions of these associations (i.e. negative) were the same for regions in both hemispheres. According to the valence asymmetry hypothesis (Davidson, 1993) it would (rather simplistically) be expected that in the presence of affective stimuli the relationships between left and right hemispheres would be different (left-positive and right-negative associations). Davidson and colleagues have suggested that areas in the left frontal cortex have a dominant role in both promoting positive affective states and in inhibiting negative states (via down-regulation of subcortical structures), through processes not proposed to be independent of one another (Davidson & Sutton, 1995; Davidson et al., 2000). In this instance, it is not clear which processes may be occurring. Activation in the frontal cortex during exercise may be more reflective of the approach (left hemisphere) and withdrawal (right hemisphere) motivation components described by the hypothesis. Harmon-Jones (2004) exclusively defines frontal asymmetry as approach-withdrawal motivations. This would suitably suggest that activation was due to cognitive processes involved in active avoidance (right hemisphere) and approach motivation (left hemisphere) in the presence of negative affective stimuli. It is presumed that lateralised processes would be similar to those

induced through cognitive control processes of negative affect (Ochsner & Gross, 2005, 2007; Ochsner et al., 2004). This would explain the greater increase in blood volume (ΔtHb) observed in the right hemisphere in individuals in the high tolerance group at intensities above RCP shown in study I.

The findings in this thesis lend support to an approach-withdrawal motivation explanation of frontal cortex asymmetry during exercise. The asymmetrical patterns of activation shown by the studies in this thesis can be interpreted in terms of potential cognitive processes involved in the regulation of affective responses (see 2.3.1). Despite moderate correlations of affective responses (valence) with activation in the frontal cortex, the interpretation of these associations in terms of the valence-asymmetry component of the asymmetry hypothesis is unclear. However, this conclusion is based upon limited information as no attempts or adequate measures to examine the basis of frontal cortex asymmetry (motivation, valence or both) were conducted.

The motivational component of the valence asymmetry hypothesis has parallels with Craig's model of forebrain asymmetry (2002, 2005). At intensities of exercise above VT, activation in regions of the left hemisphere would be associated with approach-type behaviours related to nourishment and safety (compatible with the parasympathetic branch of the autonomic nervous system). However, at intensities of exercise at and near exhaustion, activation in regions of the right hemisphere would be associated with withdrawal-type behaviours related to arousal and danger (compatible with the sympathetic branch of the autonomic nervous system). Craig's model relates to the anterior cingulate and insular cortices, which are situated beneath lateral prefrontal areas of the cortex. Whilst NIRS cannot measure activation in the anterior cingulate and insular cortices using NIRS, the haemodynamic response recorded may be indicative of changes in these regions (via spreading activation).

9.4.3 Frontal cortex deregulation

A fundamental mechanism of the DMM is the transient hypofrontality hypothesis (Dietrich, 2003, 2006; see 2.3.3). Evidence of the hypofrontality response during exercise using NIRS is apparent in the literature, but it is not always explicitly interpreted as a protective mechanism, rather a consequence of physiological parameters. Described as a deregulation of the frontal cortex or shift in cerebral metabolic resources, the hypothesis is

proposed to explain reduced cognitive functions and affective states due to exercise (Dietrich, 2003, 2006).

Rooks et al. (2010) in a meta-analysis of NIRS data recorded during exercise reported that a hypofrontality response, described as a rise in deoxygenation (ΔHHb) over and above the decline in oxygenation (ΔO_2Hb), occurs at maximal intensities. However, the interpretation of the hypofrontality response proposed by Dietrich (2003, 2006) appears to be more of a subtle deregulation of activation in the frontal cortex (a plateau or decrease in ΔO_2Hb with an increase in ΔHHb). The variability in this responses (i.e. increase in ΔHHb above ΔO_2Hb) at near maximal intensities of exercise still remains somewhat undetermined.

Changes in the haemodynamic response recorded in studies within this thesis indicate deregulation or hypoactivation in the frontal cortex during exercise at intensities above VT. The dorsolateral region of the frontal cortex indicated differential patterns of activation influenced by self-reported tolerance (study I). Using multi-channel NIRS, greater activation was observed in superior regions of the frontal cortex (study II). This deregulation or hypofrontality effects were greatest during incremental (studies I and II) compared to constant-load or steady state exercise (studies III - V). One reason for this may be that during incremental exercise, the termination of exercise is due to reaching maximal physiological capacity at an increasing workload. However, during constant-load exercise (at intensities [RCP] to exhaustion), not only would the termination of exercise be due to reaching maximal physiological capacity, but it may provide a greater cognitive challenge which could impact upon the termination of exercise (before a true maximal physiological capacity is achieved). This was observed in study IV. A greater oxygen uptake was achieved at exhaustion during the initial incremental exercise test than exhaustion during the two constant-load exercise sessions. Therefore, constant-load exercise may be more cognitively demanding and more heavily influenced by individual differences (e.g. cognitive control processes, approach-withdrawal motivation and/or self-reported tolerance). These factors could contribute to the variability in the haemodynamic response.

Variability in the cerebral haemodynamic response across exercise intensity domains was reported by Rooks et al. (2010; briefly mentioned above). The authors indicated that this variability may be due to an individual's exercise experience or physiological fitness. However, study II suggests that variability may be explained by the region (or hemisphere) of the frontal cortex measured in each of the individual studies, which was not considered

by Rooks et al. (2010). The findings from studies I and II in this thesis provide support and somewhat advance our understanding of the variability in the haemodynamic response. In study I, despite no differences in physiological variables (i.e. $\dot{V}O_{2peak}$), individuals defined as having a high self-reported tolerance showed differential patterns of activation at intensities of exercise above VT than those with low self-reported tolerance. In study II, the use of multi-channel NIRS indicated differences in the haemodynamic response in sub regions of the frontal cortex at intensities above VT. These findings indicate that not only 'training' status may contribute to variability in the haemodynamic response (as suggested by Rooks et al., 2010), but also individual differences such as an individual's self-reported tolerance of the intensity of exercise (assessed using the PRETIE-Q). Furthermore, study IV indicated that prior exercise (to induce a heightened state of interoceptive awareness) influenced the magnitude of the haemodynamic response (reduced relative to the control condition) in all regions and influenced regional activation. Therefore, a number of additional factors could explain inter-and intra-individual variability of the haemodynamic response in the frontal cortex; the regulation (influenced by predisposed traits) of exercise stimuli (interoceptive cues) and the regions of the brain measured.

9.4.4 Tolerance of the intensity of exercise

Self-reported tolerance of the intensity of exercise describes how an individual may perceive sensory (interoceptive) cues during exercise which influences the way they feel during exercise and potentially whether or not they persist at the activity (see 2.4.1). As highlighted above, self-reported tolerance was associated with differential activation in the frontal cortex (study I) and activity in the amygdala at intensities above VT (at RCP; study V). It is proposed that self-reported tolerance influences the cognitive processes employed by individuals and contributes to the interplay between frontal cortex and sensory inputs to the amygdala as described by the DMM (Ekkekakis et al., 2005a; Ekkekakis et al., 2007). The findings from this thesis support self-reported tolerance as a potential method to identify how an individual may respond to exercise at different intensities. For example, data from study I suggested that, an individual with a low tolerance would report a more negative affective response at a lower intensity (around VT) and employ different cognitive processes (and activation patterns in the frontal cortex) than an individual with high self-reported tolerance. The neurophysiological associations with self-reported tolerance at intensities of exercise above VT support the presumption that it is a dispositional trait to

the intensity of exercise. As suggested by Ekkekakis et al. (2005a) self-reported tolerance (measured using a simple questionnaire) could be used as a screening method for individuals entering exercise programs. Self-reported tolerance would allow the identification of individuals who may under- or over-exert themselves and it would help to tailor an exercise plan that is effective (physiologically and psychologically), safe and specific for a particular individual. However, more research is required to understand the sensitivity of the PRETIE-Q to neurophysiological responses and exercise behaviours and to examine further if self-reported tolerance is a dispositional trait to the intensity of exercise (see 9.7). The preference construct of the PRETIE-Q was not examined on the basis that tolerance is more closely related to interoceptive factors (i.e. increases in the intensity of exercise). This was shown as tolerance (and not preference) has been shown to explain variance in negative affective responses (driven by interoceptive factors) to exercise at intensities above VT (Ekkekakis et al. 2007).

9.5 Limitations

Affective responses during exercise were measured using the Feeling Scale (Hardy & Rejeski, 1989). This scale has received the most attention in measuring affective responses to exercise (as outlined in 2.1). However, this 11-point bipolar scale has received criticism for its simplicity (McAuley & Courneya, 1994; Rudolph & Kim, 1996). This is despite the role that the scale has played in helping to confirm variability (i.e. fluctuations in affective responses) shown by individuals during exercise at different intensities (mainly around VT). This patterning went unnoticed in early research that tended to use pre-post test study designs (see 2.1). The brevity of the scale allows for multiple measures of affect to be taken during exercise and the common utilisation of this scale in the literature allows comparisons to be made between studies.

The feeling scale was used to explore associations between affective responses and physiological (NIRS, ASER) and other self-report (tolerance) variables using bivariate linear correlations. Despite some correlations indicating moderate to strong relationships, caution should be taken in their interpretation. To fully understand the relationships between affective responses and activation in the frontal cortex and amygdala, appropriate and adequately powered research designs and regression analyses should be employed. In

this thesis, the primary use of bivariate linear correlations was to complement the interpretations and explanations from the ANOVA.

The measurement of the haemodynamic response using NIRS is well understood (Ferrari & Quaresima, 2012); however the interpretation in relation to psychological factors and physiological parameters (especially measures in the brain) is complex. The widespread use of NIRS in different populations and under different conditions will help to provide a clearer understanding of variability in the haemodynamic response. Both males and females took part in all of the studies within this thesis, however the influence of gender, specifically the effects of the menstrual cycle, upon the haemodynamic responses during exercise are unknown. It may be desirable for future research to control for the menstrual cycle. Furthermore, it is unclear if profuse sweating during exercise may cause a reduction in blood volume, which could impact upon the haemodynamic responses measured using NIRS.

The spatial resolution of NIRS is relatively poor compared to other neuroimaging techniques (fMRI, PET). However, NIRS provides adequate temporal resolution, is relatively low-cost in comparison to other techniques and most importantly is suitable for use during exercise. Although the spatial resolution of NIRS measures may be improved with the use of multi-channel systems or using NIRS in combination with neuro-navigation techniques or MRI which would improve the scope of application of NIRS. In this thesis, the locations of optodes were calculated using the 10/20 EEG system for electrode placement. This is a standardised system which determines regions of the brain approximate to Brodmann's cortical areas. However, individual differences in brain architecture and slight variations in the placements of optodes are factors which could further contribute to the variability in response.

In an attempt to determine the neural basis of affective responses to exercise, or that of any other psychophysiological sensations, it is important to acknowledge that areas of the brain are integrated and have extensive connections between cortical and subcortical areas. It is difficult to determine specific functions of individual areas of the brain without acknowledging the potential contributions or influence from other (cortical and subcortical) areas. For example, by measuring activity in the dorsolateral prefrontal area, it may be difficult to distinguish between what is considered to be local activity and more regional activity due to contributions from surrounding regions such as the ventromedial

prefrontal cortex or the insula (Fecteau 2007; Ghashghaei & Barbas, 2002; Woo et al., 2009). These areas are also involved in affect regulation (as describe in 2.2), but were not directly measured. In the studies in this thesis using NIRS, it is proposed that relatively superficial (localised) frontal areas were recorded, but the influence of potential activity in underlying areas must be considered. The use of multi-channel NIRS (as shown in studies II and III) showed different activation patterns in sub regions of the cortex indicating more localised rather than regional activation in frontal areas. However, the superficial or underlying areas measured to examine the neural basis of affect regulation are discussed in terms of the capacity of these areas to be involved, rather than their discreet function (for example, if they up-, or down-regulate affective responses; see 2.3.1).

The exercise protocols used in this thesis (incremental and constant-load) are not representative of what an individual may complete as part of their exercise regime. The duration of exercise at the selected intensities (10 minutes above VT and RCP to exhaustion; studies III and IV) would not meet the requirements of current guidelines for physical activity recommendations (30 minutes of moderate to vigorous physical activity on most, preferable all days, per week). However, the studies in this thesis were conducted to examine mechanisms and provide evidence towards a neurobiological basis of the regulation for affective responses during exercise at different intensities. The intensities were chosen to standardise the intensities of exercise to examine if imagery, as a cognitive strategy, could maintain frontal cortex function (study III) and if prior activity would impair the capacity of the frontal cortex (study IV), at intensities of exercise which are predominantly aversive. The data from these two studies suggest that if individuals exercise at intensities above VT (which is feasible in prescribed programs) or exercise when physiologically fatigued, then affective responses are likely to be negative.

Participants in studies I-V were actively engaged in exercise and therefore their affective responses may have been more positive (less negative) than if they were inactive or sedentary. The responses to exercise (haemodynamic, affective, RPE and eye blink) measured in these studies will almost certainly be varied in other populations from those recorded in these studies. For example, in study I, individuals classified as having a low self-reported tolerance were only mid-range (M = 21, SD = 2) on the tolerance scale (range 8 to 40). It is anticipated that individuals with a tolerance score closer to 8 would respond differently (i.e. report a more negative affective response, terminate exercise earlier).

Therefore, although these findings may not be representative of typical responses to exercise in populations that the applications of this research may target (i.e. sedentary/low-active populations), the findings do contribute new evidence of psychological and physiological responses to exercise at different intensities in an active population. In addition, the laboratory environment, presence of the experimenter and wearing a facemask may all be factors that could impact upon an individual's affective response. These responses may be different if the exercise was completed outside of the laboratory environment without an experimenter and without a facemask. Furthermore, the data in this thesis were collected during exercise using a recumbent cycle ergometer. Future research should consider the influence of different types of exercise (i.e. cycling or treadmill) and postural effects (i.e. recumbent versus upright activity) upon an individual's affective and haemodynamic responses.

9.6 Directions for future research

This thesis has highlighted the functional capacity of regions of the frontal cortex during exercise at different intensities. The regulation of affective responses has been explored. To advance this research distinct cognitive control (i.e. appraisal or suppression) and motivational (approach-withdrawal) processes should be experimentally tested. Firstly, paradigms used in fMRI studies could be replicated using NIRS. The sensitivity of the NIRS technique could then be examined and compared with other neuroimaging methods. Secondly, these paradigms could be applied during exercise at different intensities to examine their influence upon affective responses. The differences between appraisal and reappraisal processes (indicating lateralised areas of the frontal cortex; Ochsner et al., 2004) and how they impact affective responses could be explored.

In the case of imagery use, as a cognitive strategy, to improve affective responses, it is necessary for future research to examine the effectiveness of this technique in previously sedentary participants. As the energy and enjoyment imagery employed in study III required participants to recall previous positive exercise experiences, this specific imagery may be difficult or less effective in sedentary individuals. Previously sedentary individuals may not have a 'pleasant' experience of exercise to imagine. Commencing exercise at a low intensity, or at a self-selected pace could provide this pleasant experience. Alternative

cognitive strategies worth examining include the use of association and disassociation, particularly as association strategies may influence the awareness of interoceptive cues (Neumann & Heng, 2011).

The rationale for measuring affective responses using the Feeling Scale (Hardy & Rejeski, 1989) has been provided (see 3.2.1). However, in order to provide a richer explanation of affective responses to the intensity of exercise qualitative data and the use of other validated measures could be incorporated into research study designs. In conjunction with the use of NIRS to measure activation in the frontal cortex during exercise, qualitative descriptors may help to define psychophysiological relationships to fully understand a neural basis of affective responses. It is recommended that affective responses be measured in conjunction with other perceptual responses, such as perceived exertion, which is influenced by interoceptive input from the body during exercise.

The use of NIRS in place of EEG may contribute to the existing literature to further unpack determinants of frontal cortex asymmetry, such as the associated affective and motivational constructs, of which there has been recent debate (Hagemann et al., 1998; Harmon-Jones, 2003, 2004). Studies could use NIRS and EEG techniques in combination with ASER at rest and during exercise.

The use of acoustic startle methods in study V was novel and further methodological and analytical procedures would be of benefit to systematically identify what the components of these methods reflect when measured during exercise (i.e. arousal in subcortical structures, motivational intensity or affective states). However before these questions can be examined, the validity of the acoustic startle response during exercise (specifically at intensities >50% aerobic power) needs to be determined. If the acoustic startle response is shown to be valid, this method could potentially provide a suitable psychophysiological indicator of neural processes involved during exercise that could not otherwise be measured. In combination with NIRS (and/or EEG), studies could employ functional tasks such as, affective picture viewing before, after and even during exercise. This would help to determine affective processing in sub regions of the frontal cortex and help to unpack determinants of frontal cortex asymmetry (affective and motivational constructs) on which there has been debate (Hagemann et al., 1998; Harmon-Jones, 2003, 2004).

Additionally, the neural basis of the regulation for affective responses to exercise proposed by the DMM could be further examined.

The findings of these studies provide an understanding of the underlying mechanisms of affective responses to exercise. However, based upon these mechanisms relevant interventions could be created to help improve an individual's exercise experience (i.e. make people feel good, or at least not bad). An obvious intervention would be to use imagery techniques shown to improve affective responses at moderate intensities (Stanley & Cumming, 2009) and those (5%) above VT (study III) to allow individuals to benefit both physiologically and psychologically from exercise participation.

As previously introduced (9.2.4), the PRETIE-Q has the potential to be used as a screening method or as part of a health assessment for individuals starting an exercise program. Studies are encouraged to incorporate this measure, so that this method can be explored in terms of its sensitivity towards responses to exercise and future behaviours. Furthermore, the genetic basis of the dispositional and trait-like nature of the PRETIE-Q could be examined. Recent research has identified single-nucleotide polymorphisms related to subjective responses to exercise (Karoly et al., 2012). Self-reported tolerance (and also preference) could be explored in a similar way. The methods to examine polymorphisms are relatively inexpensive and the collection of samples from large populations (using saliva swabs) could be easily achieved.

The neural basis of affective responses to exercise at different intensities is far from being fully understood. The interplay between cognitive and sensory processes which may influence the regulation of affective responses in the brain is extremely complex. As previously highlighted, an examination of the exact cognitive control processes involved is one main focus. However, another would be to examine components of the sensory (i.e. interoceptive) inputs induced by exercise.

The studies in this thesis provided few measures of the sensory stimuli (interoceptive cues) evoked during exercise involved in the 'interoceptive mode' described by the DMM. In this thesis indirect measures of respiratory processes were recorded using online gas analysis. Due to the aerobic nature of the exercise performed (i.e. cycling) the sensory stimuli were presumably driven by the respiratory and cardiovascular system. A future avenue would be to examine different forms of exercise (e.g. strength and resistance training) in comparison

to aerobic exercise to activate different interoceptive cues and determine their influence upon affective responses. Another, albeit rather challenging line of inquiry, would be to take peripheral biochemical measures during exercise to determine a profile of what may be happening in the body during exercise (at standardised intensities) that could impact the dual-modes.

To extend and validate the novel findings of this thesis, replication and integration of the study methods and designs is required. Studies III and V examined the interplay between cognitive and sensory processes at intensities above VT. These study designs could be combined to examine the capacity of the frontal cortex (using NIRS) and the use of a cognitive strategy in the presence of intensified interoceptive input (following prior exhaustive activity). In addition, acoustic startle methods could be employed. However the use of acoustic startle methods during exercise is dependent upon (a) the development of a suitable protocol and (b) the determination of what the response reflects (i.e. arousal, motivation and/or valence). The combination of these relatively novel methods to understand affective responses to exercise at different intensities will advance this area of research from a mechanistic perspective.

9.7 Conclusions

The findings in this thesis have provided some confirmation that regions of the frontal cortex and the amygdala provide a neural basis of affective responses to exercise at different intensities. The five studies have shown that deregulation of frontal cortex activation occurs during exercise at intensities above VT to exhaustion. Functional capacity of the frontal cortex is limited which impacts how an individual may feel. The activity in the amygdala is associated with activation in the frontal cortex and affective responses at higher intensities of exercise (at VT and RCP). The predisposed trait of tolerance of the intensity of exercise influences activation in the frontal cortex and is associated with activity in the amygdala. The potential role of cognitive control processes and motivational mechanisms involved in the regulation of affective responses to exercise have been proposed.

In summary, what happens in areas of the brain (i.e. the frontal cortex and amygdala) during exercise contributes to *why* we feel how we feel during exercise. These findings provide neurophysiological evidence of the interplay between cognitive factors and sensory processes which influence an individual's exercise experience, particularly whether they feel good or not. This understanding forms the basis to develop ways to help guide individuals to feel good (or more importantly not feel bad) during exercise to potentially increase exercise participation and adherence.

References

Acevedo, E.O., Gill, D.L., Goldfarb, A.H., & Boyer, B.T. (1996). Affect and perceived exertion during a two-hour run. *International Journal of Sports Psychology, 27*, 286-292.

Alves, N. T., Aznar-Casanova, J. A., & Fukusima, S. S. (2008). Patterns of brain asymmetry in the perception of positive and negative facial expressions. *Laterality: Asymmetries of Body, Brain and Cognition, 14*, 256-272.

Amaral, D.G., & Price, J.L. (1984). Amygdalo-cortical projections in the monkey (*Macaca fascicularis*). *The Journal of Comparative Neurology. 230*, 465-496.

Ament, W., & Verkerke, G.J. (2009). Exercise and fatigue. *Sports Medicine, 39*, 389-422.

American College of Sports Medicine. (2013). ACSM's guidelines for exercise testing and prescription (9th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Armony, J.L. (2013). Current Emotion Research in Behavioral Neuroscience: The Role(s) of the Amygdala. *Emotion Review 5*, 104-115.

Australian Government, Department of Health and Ageing. (2008). *National Health Survey*. [online] Available at: http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-active-evidence.htm [Accessed 20 February 2012].

Australian Government, Department of Health and Ageing. (2005). *National Physical Activity Guidelines for Adults*. [online] Available at: http://www.health.gov.au/internet/main/publishing.nsf/Content/phd-physical-activity-adults-pdf-cnt.htm [Accessed 20 February 2012].

Banks, S.J., Eddy, K.T., Angstadt, M., Nathan, P.J., & Phan, K.L. (2007). Amygdala-frontal connectivity during emotion regulation. *Social, Cognitive, and Affective Neuroscience, 2*, 303-312.

Barrett, L.F., & Wager, T. (2006). The structure of emotion: evidence from the neuroimaging of emotion. *Current Directions in Psychological Science, 15*, 79-85.

Batson, C.D., Shaw, L.L., & Oleson, K.C. (1992). Differentiating affect, mood, and emotion: Toward functionally based conceptual distinctions. In M.S. Clark (Ed.), *Review of personality and social psychology* (Vol 13, pp. 294-326). Newbury Park, CA: Sage.

Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural Correlates of conscious self-regulation of emotion. *Journal of Neuroscience, 21*, RC165.

Beaver, W.L., Wasserman, K., & Whipp, B.J. (1986). A new method for detecting anaerobic threshold by gas exchange. *Journal of Applied Physiology, 8,* 2020-2027.

Beckmann, M., Johansen-Berg, H., Rushworth, M.F.S. (2009). Connectivity-based parcellation of human cingulated cortex and its relation to functional specialization. *Journal of Neuroscience, 29,* 1175-90.

Berkman, E.T., & Lieberman, M.D. (2010). Approaching the bad and avoiding the good: Lateral prefrontal cortical asymmetry distinguishes between action and valence. *Journal of Cognitive Neuroscience, 22,* 1970-1979.

Bernard, J.F., Bester, H., & Besson, J.M. (1996). Involvement in the spino-parabrachio-amygdaloid and -hypothalamic pathways in the autonomic and affective emotional aspects of pain. *Progress in Brain Research, 107*, 243-255.

Bhambhani, Y., Malik, R., & Mookerjee, S. (2007). Cerebral oxygenation declines at exercise intensities above the respiratory compensation threshold. *Respiratory Physiology and Neurobiology, 156,* 196-202.

Bixby, W.R., Spalding, T.W., & Hatfield, B.D. (2001). Temporal dynamics and dimensional specificity of the affective response to exercise of varying intensity: Differing pathways to a common outcome. *Journal of Sport and Exercise Psychology, 23,* 171-190.

Blanchard, C.M., Rodgers, W.M., & Gauvin, L. (2004). The influence of exercise duration and cognitions during running on feeling states on an indoor running track environment. *Psychology of Sport and Exercise, 5*, 119-133.

Blumenthal, T.D., Cuthbert, B.N., Filion, D.L., Hackley, S., Lipp, O.V., & Van Boxtel, A. (2005). Committee report: Guidelines for human startle eyeblink electromyographic studies. *Psychophysiology, 42*, 1-15.

Booth, F.W., Chakravarthy, M.V., Gordon, S.E., & Spangenburg, E.E. (2002). Waging war on physical inactivity: using modern molecular ammunition against an ancient enemy. *Journal of Applied Physiology, 93*, 3-30.

Borg, G. (1998). *Borg's Perceived Exertion and Pain Scales.* Human Kinetics; Champaign, IL.

Botvinick, M.M., Braver, T.S., Barch, D.M., Carter, C.S., & Cohen, J.C. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*, 624-652.

Boutcher, S.H. (1990). Aerobic fitness: Measurement and issues. *Journal of Sport and Exercise Psychology, 12,* 235-247.

Boutcher, S.H., & Landers, D.M. (1988). The effects of vigorous exercise on anxiety, heart rate, and alpha activity of runners and non-runners. *Psychophysiology*, *25,* 696-702.

Bush, G., Luu, P., Posner, M.I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science, 4,* 215-222.

Canadian Society for Exercise physiology. (Revised 2002). *Physical Activity Readiness Questionnaire.* Retrieved from http://www.csep.ca/cmfiles/publications/parq/par-q.pdf.

Canli, T., Desmond, J.E., Zhao, Z., Glover, G., & Gabrieli, J.D. (1998). Hemispheric asymmetry for emotional stimuli detected with fMRI. *NeuroReport, 9,* 3233-3239.

Carmichael, S.T., & Price, J.L. (1995). Limbic connections of the orbital and medial prefrontal cortex in macaque monkeys. *The Journal of Comparative Neurology, 363,* 615-641.

Carver, C.S., & White, T.L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology*, 67, 319-333.

Cecheto, D.R. & Saper, C.B. (1990). Role of the cerebral cortex in autonomic function. In A.D. Loewy & K.M. Spyer (Eds.), Central Regulation of Autonomic Functions (pp. 208-223). Oxford: Oxford University Press.

Clapper, R.L. (1990). Adult and adolescent arousal preferences: The revised Reducer-Augmenter Scale. *Personality and Individual Differences, 11*, 1115-1122.

Coan, J.A., & Allen, J.J. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology, 40,* 106-114.

Cohen, J.D., Dunbar, K., & McClelland, J.L. (1990). On the Control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review, 97*, 332-361.

Craig, A.D. (1996). An ascending general homeostatic afferent pathway originating in lamina I. In G. Holstege, R. Bandler & C.B. Saper (Eds.), *The Emotional Motor System, Progress in Brain Research*, (Vol. 107, pp. 225-242). Amsterdam: Elsevier.

Craig, A.D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Neuroscience, 3,* 655-666.

Craig, A.D. (2005). Forebrain emotional asymmetry: a neuroanatomical basis? *Trends in Cognitive Sciences, 9,* 566-571.

Craig, A.D. (2006). Physical activity and the neurobiology of interoception. In E.O. Acevedo & P. Ekkekakis (Eds.), *Psychobiology of physical activity* (pp. 15-28). Champaign, IL: Human Kinetics.

Cremers, J., Dessoullieres, A., & Garraux, G. (2012). Hemispheric specialization during mental imagery of brisk walking, *Human Brain Mapping, 33*, 873-882.

Critchley, H.D. (2005). Neural mechanisms of autonomic, affective, and cognitive integration. *The Journal of Comparative Neurology,* 493, 154-166.

Critchley, H.D., Corfield, D.R., Chandler, M.P., Mathias, C.J., & Dolan, R.J. (2000). Cerebral correlates of autonomic cardiovascular arousal: a functional neuroimaging investigation in humans. *Journal of Physiology, 523,* 259-70.

Critchley, H.D., Wiens, S., Rotshtein, P., Ohman, A., & Dolan, R.J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience, 7,* 189-195.

Cumming, J., & Ste-Marie, D.M. (2001). The Cognitive and Motivational Effects of Imagery Training: A Matter of Perspective. *The Sport Psychologist, 15*, 276-288.

Damasio, A.R. (1994). *Descartes' error: Emotion, reason and the human brain*, New York: Grosset/Putnam.

Damasio, A.R. (1999). *The Feeling of What Happens: Body and Emotion in the Making of Consciousness*. New York: Harcourt Brace.

Davidson, R.J. (1993). Cerebral asymmetry and emotion: Conceptual and methodological conundrums. *Cognition and Emotion, 7,* 115-138.

Davidson, R.J. (2001). The neural circuitry of emotion and affective style: Prefrontal cortex and amygdala contributions. *Social Science Information, 40*, 11-37.

Davidson, R.J. (2002). Anxiety and affective style: Role of prefrontal cortex and amygdala. *Biological Psychiatry, 51*, 68-80.

Davidson, R.J. (2004). What does the prefrontal cortex "do" in affect: Perspectives in frontal EEG asymmetry research. *Biological Psychology, 67,* 219-234.

Davidson, R.J., Ekman, P., Saron, C.D., Senulis, J.A., & Friesen, W.V. (1990). Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology I. *Journal of Personality and Social Psychology, 58,* 330-341.

Davidson, R.J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Science, 3,* 11-21.

Davidson, R.J., Jackson, D.C., & Kalin, N.H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin, 126*, 890-909.

Davidson, R.J., Schaffer, C.E., & Saron, C. (1985). Effects of lateralized presentations of faces on self-reports of emotion and EEG asymmetry in depressed and non-depressed subjects. *Psychophysiology, 22,* 353-364.

Davidson, R.J., & Sutton, S.K. (1995). Affective neuroscience: The emergence of a discipline. *Current Opinion in Neurobiology, 5*, 217-224.

Davis, M., Walker, D.L., & Lee, Y. (1999). Neurophysiology and neuropharmacology of startle and its affective modulation. In M.E. Dawson, A.M. Schell & A.H. Böhmelt, (Eds.). *Startle modification: Implications for neuroscience, cognitive science, and clinical science* (pp. 95–113). Cambridge, UK: Cambridge University Press.

Del Giorno, J.M., Hall, E.E., O'Leary, K.C., Bixby, W.R., & Miller, P.C. (2010). Cognitive function during acute exercise: A test of Transient Hypofrontality Theory? *Journal of Sport and Exercise Psychology, 32*, 312-323.

Dawson, M. E., Schell, A., M., & Böhmelt, A. H. (Eds.). (1999). *Startle modification: Implications for neuroscience, cognitive science, and clinical science*. Cambridge, UK: Cambridge University Press.

Delp, M.D., Armstrong, R.B., Godfrey, D.A., Laughlin, M.H., Ross, C.D., & Wilkerson, M.K. (2001). Exercise increases blood flow to locomotor, vestibular, cardiorespiratory and visual regions of the brain in miniature swine. *Journal of Physiology, 533*, 849-859.

Dietrich, A. (2003). Functional neuroanatomy of altered states of consciousness: The transient hypofrontality hypothesis. *Consciousness and Cognition 12,* 231-256.

Dietrich, A. (2006). Transient hypofrontality as a mechanism for the psychological effects of exercise. *Psychiatry Research, 145*, 79-83.

Dietrich, A., & Sparling, P.B. (2004). Endurance exercise selectively impairs prefrontal-dependent cognition. *Brain and Cognition, 55,* 516-524.

Dishman, R.K., Berthoud, H.R., Booth, F.W., Cotman, C.W.,Edgerton,V.R., Fleshner, . . . Zigmond, M.J. (2006). Neurobiology of Exercise. *Obesity, 14*, 345-356.

Dolan, R.J., & Vuilleumier, P. (2003). Amygdala Automaticity in Emotional Processing. *Annals of the New York Academy of Sciences, 985,* 348-355.

Duncan, A., Meek, J.H., Clemence, M., Elwell, C.E., Tyszczuk, L., Cope, M., & Delpy, D.T. (1995). Optical pathlength measurements on adult head, calf and forearm and the head of the newborn infant using phase resolved optical spectroscopy. *Physics in Medicine and Biology, 40,* 295-304.

Duncan, J., & Owen, A.M. (2000). Common regions of the human frontal lobe recruited by diverse cognitive demands. *Trends in Neuroscience, 23*, 475-483.

Ekkekakis, P. (2003). Pleasure and displeasure from the body: Perspectives from exercise. *Cognition and Emotion, 17*, 213-239.

Ekkekakis, P. (2009). Illuminating the black box: Investigating prefrontal cortical dynamics during exercise with near-infrared spectroscopy. *Journal of Sport and Exercise Psychology*, *31*, 505-53.

Ekkekakis, P., & Acevedo, E.O. (2006). Affective responses to acute exercise: Toward a psychobiological dose-response model. In E.O. Acevedo & P. Ekkekakis (Eds.), *Psychobiology of physical activity* (pp. 91-109). Champaign, IL: Human Kinetics.

Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2004). Practical markers of the transition from aerobic to anaerobic metabolism during exercise: Rationale and a case for affect-based exercise prescription. *Preventive Medicine, 38*, 149-159.

Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2005a). Some like it vigorous: Individual differences in the preference for and tolerance of exercise intensity. *Journal of Sport and Exercise Psychology, 27*, 350-374.

Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2005b). Variation and homogeneity in affective responses to physical activity of varying intensities: An alternative perspective on dose-response based on evolutionary considerations. *Journal of Sports Sciences, 23*, 477-500.

Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2008).The relationship between exercise intensity and affective responses demystified: To crack the forty-year-old nut, replace the forty-year-old nutcracker! *Annals of Behavioral Medicine, 35*, 136-149.

Ekkekakis, P., Hall, E.E., Van Landuyt, L.M., & Petruzzello, S.J. (2000). Walking in (affective) circles: Can short walks enhance affect? *Journal of Behavioral Medicine, 23*, 245-275.

Ekkekakis, P., Lind, E., Hall, E.E., & Petruzzello, S.J. (2007). Can self-reported tolerance of exercise intensity play a role in exercise testing? *Medicine and Science in Sports and Exercise, 39*, 1193-1199.

Ekkekakis, P., Parfitt, G., & Petruzzello, S.J. (2011). The pleasure and displeasure people feel when they exercise at different intensities: Decennial update and progress towards a tripartite rationale for exercise intensity prescription. *Sports Medicine, 41*, 641-671.

Ekkekakis, P., & Petruzzello, S.J. (1999). Acute aerobic exercise and affect: current status, problems and prospects regarding dose-response. *Sports Medicine, 28,* 337–74.

Ekkekakis P., & Petruzzello, S.J. (2000). Analysis of the affect measurement conundrum in exercise psychology: I. Fundamental issues. *Psychology of Sport and Exercise, 1*, 71-88.

Ekkekakis, P., & Petruzzello, S.J. (2002). Analysis of the affect measurement conundrum in exercise psychology: IV. A conceptual case for the affect circumplex. *Psychology of Sport and Exercise, 3*, 35-63.

Eston, R., Faulkner, J., St Clair Gibson, A., Noakes, T., & Parfitt, G. (2007). The effect of antecedent fatiguing activity on the relationship between perceived exertion and physiological activity during a constant load exercise task. *Psychophysiology, 44*, 779-786.

Exercise and Sports Science Australia. (Version 1, 2011). *Adult Pre-exercise Screening System.* Retrieved from http://www.essa.org.au/wp/wp-content/uploads/Screen-tool-version-v1.1.pdf.

Eysenck, H.J. (1989). Adult Eysenck Personality Questionnaire. In L.J. Eaves, H.J. Eysenck, & N.G. Martin (Eds.), *Genes, culture and personality: An empirical approach* (pp. 420-423). London: Academic.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39,* 175-191.

Fecteau, S., Knoch, D., Fregni, F., Sultani, N., Boggio, P., & Pascual-Leone., A. (2007). Diminishing Risk-Taking Behavior by Modulating Activity in the Prefrontal Cortex: A Direct Current Stimulation Study. *The Journal of Neuroscience*, *27*, 12500-12505.

Ferrari, M., Mottola, L., & Quaresima, V. (2004). Principles, Techniques, and Limitations of Near Infrared Spectroscopy. *Canadian Journal of Applied Physiology, 29,* 463-487.

Ferrari, M., & Quaresima, V. (2012). A brief review on the history of human functional near-infrared spectroscopy (fNIRS) development and fields of application. *Neuroimage, 63,* 921-935.

Flaten, M.A. (1993). A comparison of electromyographic and photoelectric techniques in the study of classical eyeblink conditioning and startle reflex modification. *Journal of Psychophysiology, 3*, 230-237.

Fontes, B., Okano, H., De Guio, F., Schabort, E.J., Min, L.L., Basset, F.A., . . . Noakes, T.D. (2013). Brain activity and perceived exertion during cycling exercise: an fMRI study. *British Journal of Sports Medicine.*

Gaesser, G.A., & Poole, D.C. (1996). The slow component of oxygen uptake kinetics in humans. *Exercise and Sport Science Reviews, 24*, 25-70.

Garber, C.E., Blissmer, B., Deschenes, M.R., Franklin, B.A., Lamonte, M.J., Lee, I., . . . Swain, D.P. (2011). American College of Sports Medicine position stand: Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Medicine and Science in Sports and Exercise, 43,* 1334-1359.

Gaskill, S.E., Ruby, B.C., Walker, A.J., Sanchez, O.A., Serfass, R.C., & Leon, A.S. (2001). Validity and reliability of combining three methods to determine the ventilatory threshold. *Medicine and Science in Sports and Exercise, 33*, 1841-1848.

Ghashghaei, H.T., & Barbas, H. (2002). Pathways for emotion: interactions of prefrontal and anterior temporal pathways in the amygdala of the rhesus monkey. *Neuroscience*, *115,* 1261-1279.

Gauvin, L., & Brawley, L.R. (1993). Alternative Psychological Models and Methodologies for the Study of Exercise and Affect. In P. Seraganian (Ed.), *Exercise Psychology: The Influence of Exercise on Psychological Processes* (pp. 146–171). New York: Wiley.

Goldin, P.R., McRae, K., Ramel, W., & Gross, J.J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry, 63*, 577-586.

Grillon, C., & Baas, J. (2003). A review of the modulation of the startle reflex by affective states and its application in psychiatry. *Clinical Neurophysiology, 114*, 1557–1579.

Grimm, S., Beck, J., Schuepbach, D., Hell, D., Boesiger, P., Bermpohl, F., . . . Northoff, G. (2008). Imbalance between left and right dorsolateral prefrontal cortex in major depression is linked to negative emotional judgment: An fMRI study in severe major depressive disorder. *Biological Psychiatry, 63,* 369-376.

Hackley, S. (1999). Implications of blink reflex research for theories of attention and consciousness. In M.E. Dawson, A.M. Schell & A.H. Böhmelt (Eds.). *Startle modification: Implications for neuroscience, cognitive science and clinical science* (pp. 137-56). New York: Cambridge University Press.

Hagemann, D., Naumann, E., Becker, G., Maier, S., & Bartussek, D. (1998). Frontal brain asymmetry and affective style: A conceptual replication. *Psychophysiology*, *35*, 372-388.

Hall, C. (1995). The motivational function of mental imagery for participation in sport and exercise. In J. Annett, B. Cripps, & H. Steinberg (Eds.). *Exercise addiction: Motivation for participation in sport and exercise*. Leicester, UK: British Psychological Society.

Hall, E.E., Ekkekakis, P., & Petruzzello, S.J. (2002). The affective beneficence of vigorous exercise revisited. *British Journal of Health Psychology, 7*, 47-66.

Hampson, D.B., St Clair Gibson, A., Lambert, M.I., & Noakes, T.D. (2001). The influence of sensory cues on the perception of exertion during exercise and central regulation of exercise performance. *Sports Medicine, 31*, 935-52.

Hardy, C.J., & Rejeski, W.J. (1989). Not what, but how one feels: The measurement of affect during exercise. *Journal of Sport and Exercise Psychology, 11*, 304-317.

Harmon-Jones, E. (2003). Anger and the behavioural approach system. *Personality and Individual differences, 35*, 995-1005.

Harmon-Jones, E. (2004). On the relationship of anterior brain activity and anger: examining the role of attitude toward anger. *Cognition and Emotion 18*, 337–361.

Haskell, W.L., Lee, I.M., Pate, R.R., Powell, K.E., Blair, S.N., Franklin, B.A., . . . Bauman, A. (2007). Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise, 39*, 1423-1434.

Hausenblas, H.A., Hall, C.R., Rodgers, W.M., & Munroe, K. J. (1999). Exercise imagery: its nature and measurement. *Journal of Applied Sport and Exercise Psychology, 11*, 304-317.

Herrmann, M.J., Huter, T., Plichta, M.M., Ehlis, A.C., Alpers, G.W., Muhlberger, A., & Fallgatter, A.J. (2008). Enhancement of activity of the primary visual cortex during processing of emotional stimuli as measured with event-related functional near infrared spectroscopy and event-related potentials. *Human Brain Mapping, 29*, 28–35.

Huppert, T.J., Hoge, R.D., Diamond, S.G., Franceschini, M.A., & Boas, D.A. (2006). A temporal comparison of BOLD, ASL, and NIRS hemodynamic responses to motor stimuli in adult humans. *Neuroimage 29*, 368–382.

Ide, K., & Secher, N.H. (2000). Cerebral blood flow and metabolism during exercise. *Progress in Neurobiology, 61,* 397-414.

Iwamoto, G.A., Wappel, S.M., Fox, G.M., Buetow, K.A., & Waldrop, T.G. (1996). Identification of diencephalic and brainstem cardiorespiratory areas activated during exercise. *Brain Research, 726*, 109-122.

Jackson, D.C., Mueller, C.J., Dolski, I., Dalton, K.M., Nitschke, J.B., Urry, H.L., . . . Davidson, R.J. (2003). Now you feel it, now you don't: Frontal EEG asymmetry and individual differences in emotion regulation. *Psychological Science, 14,* 612-617.

Janse Van Rensburg, K., Taylor, A.H., Hodgson, T., & Benattayallah, A. (2009). Acute exercise modulates cigarette cravings and brain activation in response to smoking-related images: an fMRI study. *Psychopharmacology, 203,* 589-98.

Jöbsis, F. (1977). Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. *Science, 198,* 1264–1267.

Kahneman, D. (1999). Objective happiness. In D. Kahneman, E. Diener & N. Schwarz (Eds.), *Well-being: Foundations of hedonic psychology* (pp. 3-25). New York: Russell Sage Foundation Press.

Karoly, H.H., Stevens, C.J., Magnan, R.E., Harlaar, N., Hutchison, K.E., & Bryan, A.D. (2012). Genetic Influences on Physiological and Subjective Responses to an Aerobic Exercise Session among Sedentary Adults. *Journal of Cancer Epidemiology*, Article ID 540563.

Kim, S.H., & Hamann, S. (2007). Neural correlates of positive and negative emotion regulation. *Journal of Cognitive Neuroscience, 19*, 776-798.

Koechlin, E., Ody, C., & Kouneiher, F. (2003). The architecture of cognitive control in the human prefrontal cortex, *Science, 302*, 1181-1185.

Koch, M. (1999). The neurobiology of startle. *Progress in Neurobiology, 59*, 107-128.

Köchel, A., Plichta, M.M., Schäfer, A., Leutgeb,V., Scharmüller, W., Fallgatter, A.J., & Schienle, A. (2011). Affective perception and imagery: A NIRS study. *International Journal of Psychophysiology, 80*, 192-197.

Kohn, P.M. (1985). Sensation-seeking, augmenting-reducing, and strength of the nervous system. In J.T. Spence & C.E. Izard (Eds.), *Motivation, emotion and personality* (pp. 167-173). Amsterdam: Elsevier.

Kringelbach, M.L. (2005). The human orbitofrontal cortex: linking reward to hedonic experience. *Nature Reviews Neuroscience, 6,* 691-702.

Kringelbach, M.L., O'Doherty, J., Rolls, E.T., & Andrews, C. (2003). Activation of the Human Orbitofrontal Cortex to a Liquid Food Stimulus is Correlated with its Subjective Pleasantness. *Cerebral Cortex, 13,* 1064 -1071.

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology, 16,* 495-512.

Lang, P. J. (1980) Behavioural treatment and bio-behavioural assessment: Computer applications. In J. B. Sodowski, J. H. Johnson, & T. A. Williams (Eds.), *Technology in mental health care delivery systems* (pp. 119–137). Norwood, NJ: Ablex.

Lang P.J., Bradley, M.M., & Cuthbert, B.N. (1998). Emotion, motivation, and anxiety: Brain mechanisms and psychophysiology. *Biological Psychiatry, 44,* 1248-1263.

Larsen, R.J., & Diener, E. (1992). Promises and problems with the circumplex model of emotion. In M.S. Clark (Ed.). *Review of Personality and Social Psychology: Emotion,* (Vol. 13, pp 25-59). Newbury Park, CA: Sage.

Larsen, R.J., & Ketelaar, T. (1989). Extraversion, neuroticism, and susceptibility to positive and negative mood induction procedures. *Personality and Individual Differences, 10*, 1221-1228.

Larsen, R.J., & Ketelaar, T. (1991). Personality and susceptibility to positive and negative emotional states. *Journal of Personality and Social Psychology, 61*, 132-140.

Lazarus, R.S. (1991). *Emotion and adaptation*. Oxford: Oxford University Press.

LeDoux, J.E. (1986). Sensory Systems and Emotion: A Model of Affective Processing. *Integrative Psychiatry*, 4, 237-238.

LeDoux, J.E. (1996). *The Emotional Brain: the Mysterious Underpinning of Emotional Life.* New York: Simon & Schuster.

LeDoux, J.E. (2012). Rethinking the emotional brain. *Neuron, 73*, 653-676.

Lind, E., Joens-Matre, R.R., & Ekkekakis, P. (2005). What intensity of physical activity do formerly sedentary middle-aged women select? Evidence of a coherent pattern from physiological, perceptual, and affective markers. *Preventive Medicine, 40*, 407-419.

Lind, E., Welch, A.S., & Ekkekakis, P. (2009). Do "mind over muscle" strategies work? Examining the effects of attentional association and dissociation on exertional, affective, and physiological responses to exercise. *Sports Medicine, 39*, 743-764.

Lovelace, C.T., Elmore, W.R., Filion, D.L. (2006). Infrared reflectance as an alternative to EMG for measuring prepulse inhibition of startle eyeblink. *Psychophysiology, 43,* 511-515.

McAuley, E. & Courneya, K.S. (1994). The subjective exercise experiences scale (SEES): development and preliminary validation. *Journal of Sport and Exercise Psychology, 16*, 163-177.

Mehta, J.P., Verber, M.D., Wieser, J.A., Schmit, B.D., & Schindler-Ivens, S.M. (2009). A novel technique for examining human brain activity associated with pedaling using fMRI. *Journal of Neuroscience Methods, 179*, 230-239.

Mehta, J.P., Verber, M.D., Wieser, J.A., Schmit, B.D., & Schindler-Ivens, S.M. (2012). The effect of movement rate and complexity on functional magnetic resonance signal change during pedaling. *Motor Control, 16*, 158-75.

Morgan, W.P. (1997). Methodological considerations. In W.P., Morgan (Ed.) *Physical activity and Mental Health* (pp. 3-32). Washington, DC: Taylor & Francis.

Miller, E.K. & Cohen, J.D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167-202.

Myers-Schulz, B., & Koenigs, M. (2012). Functional anatomy of ventromedial prefrontal cortex: Implications for mood and anxiety disorders. *Molecular Psychiatry, 17,* 132-141.

Neumann, D. L., & Heng, S. (2011). The effect of associative and dissociative attentional focus strategies on muscle activity and heart rate during a weight training exercise. *Journal of Psychophysiology, 25,* 1-8.

Nelson, M.E., Rejeski, W.J., Blair, S.N., Duncan, P.W., Judge, J.O., King, A.C., . . . Castaneda-Sceppa, C. (2007). Physical activity and public health in older adults: Recommendation from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise, 39*, 1435-1445.

Nesse, R.M. (1990). Evolutionary explanations of emotions. *Human Nature, 1*, 261–289.

Nesse, R.M. (1998). Emotional disorders in evolutionary perspective. *British Journal of Medical Psychology, 71,* 397–416.

Nybo, L., & Nielsen, B. (2001). Perceived exertion during prolonged exercise with progressive hyperthermia is associated with an altered electrical activity of the brain. *Journal of Applied Physiology, 91*, 2017-2023.

O'Connor, P.J., & Petruzzello, S.J. (1992). Spontaneous eyeblinks and state anxiety following exercise. *International Journal of Neuroscience, 62*, 57-63.

Obrig, H., Hirth, C., Junge-Hulsing, J.G., Doge, C., Wolf, T., Dirnagl, U., & Villringer, A. (1996). Cerebral oxygenation changes in response to motor stimulation. *Journal of Applied Physiology, 81,* 1174-1183.

Ochsner, K.N., Bunge, S.A., Gross, J.J., & Gabrieli, J.D.E. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14,* 1215-1229.

Ochsner, K.N. & Gross, J.J. (2004). Thinking makes it so: A social cognitive neuroscience approach to emotion regulation. In R. Baumeister & K. Vohs (Eds.). *The Handbook of self-regulation* (pp. 221-255). New York: Guilford Press.

Ochsner, K.N., & Gross, J.J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9,* 242-249.

Ochsner, K.N. & Gross, J.J. (2007). The neural architecture of emotion regulation. In J.J. Gross, & R. Buck (Eds.). *The Handbook of Emotion Regulation* (pp. 87-109). New York: Guilford Press.

Ochsner, K.N., & Gross, J.J. (2008). Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Current Directions in Psychological Science, 17*, 153-158.

Ochsner, K.N., Ray, R.D., Robertson, E.R., Cooper, J.C., Chopra, S., Gabrieli, J.D.E., & Gross, J.J. (2004). For better or for worse: Neural Systems Supporting the Cognitive Down- and Up-regulation of Negative Emotion. *Neuroimage, 23,* 483-499.

Ochsner, K.N., Silvers, J.A. & Buhle, J.T. (2012). Functional imaging studies of emotion regulation: A synthetic review and evolving model of the cognitive control of emotion. *Annals of the New York Academy of Sciences, 1251*, E1-E24.

Ogoh, S., Dalsgaard, M.K., Yoshiga, C.C., Dawson, E.A., Keller, D.M., Raven, P.B., & Secher, N.H. (2005). Dynamic cerebral autoregulation during exhaustive exercise in humans. *American Journal of Physiology - Heart and Circulatory Physiology, 288,* 1461-1467.

Ongur, D., & Price, J.L. (2000). The organization of networks within the orbital and medial prefrontal cortex of rats, monkeys and humans. *Cerebral Cortex, 10*, 206.

Parfitt, G., Rose, E.A., & Burgess, W.M. (2006). The psychological and physiological responses of sedentary individuals to prescribed and preferred intensity exercise. *British Journal of Health Psychology, 11*, 39-53.

Penedo, F.J., & Dahn, J.R. (2005). Exercise and well-being: a review of mental and physical health benefits associated with physical activity. *Current Opinion in Psychiatry, 18,* 189-193.

Perrey, S. (2012). NIRS for Measuring Cerebral Hemodynamic Responses During Exercise. In H. Boecker, C.H. Hillman, L. Scheef, & H.K. Strüder (Eds.). *Functional Neuroimaging in Exercise and Sport Sciences*, (pp.335-349). New York: Springer.

Petrides, M. (2005) Lateral prefrontal cortex: architectonic and functional organization. *Philosophical Translations of the Royal Society B: Biological Sciences, 360*, 781–795.

Petruzzello, S.J., Ekkekakis, P., & Hall, E.E. (2006). Physical activity, affect, and electroencephalogram studies. In E.O. Acevedo & P. Ekkekakis (Eds.), *Psychobiology of physical activity* (pp. 111-128). Champaign, IL: Human Kinetics.

Petruzzello, S.J., Hall, E.E., & Ekkekakis, P. (2001). Regional brain activation as a biological marker of affective responsivity to acute exercise: Influence of fitness. *Psychophysiology, 38,* 99-106.

Petruzzello, S.J., & Landers, D.M. (1994). State anxiety reduction and exercise: Does hemispheric activation reflect such changes? *Medicine and Science in Sports and Exercise*, *26*, 1028-1035.

Petruzzello, S.J., & Tate, A.K. (1997). Brain activation, affect and aerobic exercise: An examination of both state-independent and state-dependent relationships. *Psychophysiology, 34,* 527-533.

Phan, K.L., Fitzgerald, D.A., Nathan, P.J., Moore, G.J., Uhde, T.W., & Tancer, M.E. (2005). Neural substrates for voluntary suppression of negative affect: A functional magnetic resonance imaging study. *Biological Psychiatry, 57*, 210-219.

Phan, K.L., Wager, T.D., Taylor, S.F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: A meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage, 16*, 331-348.

Pitman, R.K., Shalev, A.Y., & Orr, S.P. (2000). Post-traumatic stress disorder: Emotion, conditioning, and memory. In M.S. Gazzaniga (Ed.). *The Cognitive Neurosciences* (pp. 1133–1147). Cambridge, MA: MIT Press.

Pollatos O., Gramann, K., Schandry, R. (2007). Neural systems connecting interoceptive awareness and feelings. *Human Brain Mapping, 28*, 9-18.

Pontifex, M.B., & Hillman, C.H. (2007). Neuroelectric and behavioral indices of interference control during acute cycling. *Clinical neurophysiology, 118,* 570-580.

Posner, M.I. (1995). Attention in cognitive neuroscience: an overview. In M.S. Gazzaniga (Ed.). *The Cognitive Neurosciences* (pp. 615–624). Cambridge, MA: MIT Press.

Rempel-Clower, N., & Barbas, H. (1998) Topographic organization of connections between the hypothalamus and prefrontal cortex in the rhesus monkey. *Journal of Computational Neurology, 398*, 393–419.

Reynolds, S.M., & Zahm, D.S. (2005). Specificity in the projections of prefrontal and insular cortex to ventral striatopallidum and the extended amygdala. *Journal of Neuroscience, 25,* 11757–11767.

Rolls, E.T. (1999). *The brain and emotion.* New York: Oxford University Press.

Rolls, E.T. (2004). The functions of the orbitofrontal cortex. *Brain and Cognition, 55*, 11-29.

Rooks, C.R., Thom, N.J., McCully, K.K., & Dishman, R.K. (2010). Effects of incremental exercise on cerebral oxygenation measured by near-infrared spectroscopy: a systematic review. *Progress in Neurobiology, 92*, 134-50.

Rose, E.A., & Parfitt, G. (2007). A quantitative analysis and qualitative explanation of the individual differences in affective responses to prescribed and self-selected exercise intensities. *Journal of Sport and Exercise Psychology, 29*, 281-309.

Rose, E.A., & Parfitt, G. (2010). Exercise experience influences affective and motivational outcomes of prescribed and self-selected intensity exercise. *Scandinavian Journal of Medicine and Science in Sports*, Published online: doi: 10.1111/j.1600-0838.2010.01161.x.

Rudolph, D.L., & Kim, J.G. (1996). Mood responses to recreational sport and exercise in a Korean sample. *Journal of Social Behavior and Personality, 11,* 841-849.

Rupp, T., & Perrey, S. (2008). Prefrontal cortex oxygenation and neuromuscular responses to exhaustive exercise. *European Journal of Applied Physiology, 102,* 153–163.

Russell, J.A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39*, 1161-1178.

Russell, J.A, & Feldman Barrett, L. (1999). Core affect, prototypical emotional episodes, and other things called emotion: Dissecting the elephant. *Journal of Personality and Social Psychology, 76*, 805-819.

Salmon, P., Hanneman, S., & Harwood, B. (2010). Associative/Dissociative cognitive strategies in sustained physical activity: Literature review and proposal for a mindfulness-based conceptual model. *Sport Psychologist, 24*, 127-156.

Saper, C.B. (2002). The central autonomic nervous system: Conscious visceral perception and autonomic pattern generation. Annual Review of Neuroscience, 25, 433-469.

Schaefer, S.M., Jackson, D.C., Davidson, R.J., Aguirre, G.K., Kimberg, D.Y., & Thompson-Schill, S.L. (2002). Modulation of amygdalar activity by the conscious regulation of negative emotion. *Journal of Cognitive Neuroscience, 14*, 913-921.

Schienle, A., Schäfer, A., Pignanelli, R., Vaitl, D. (2009). Worry tendencies predict brain activation during aversive imagery. *Neuroscience Letters, 461*, 289–292.

Schienle, A., Schäfer, A., Vaitl, D. (2008). Individual differences in disgust imagery: a functional magnetic resonance imaging study. *Neuroreport, 19*, 527–530.

Schneider, M., Dunn, A., & Cooper, D. (2009). Affect, exercise and physical activity among healthy adolescents. *Journal of Sport and Exercise Psychology 31*, 706-723.

Sheppard, K.E., & Parfitt, G. (2008). Patterning of physiological and affective responses during a graded exercise test in sedentary men and boys. *Journal of Exercise Science and Fitness, 6*, 121-129.

Shibuya, K., & Tachi, M. (2006). Oxygenation in the motor cortex during exhaustive pinching exercise. *Respiratory, Physiology and Neurobiology, 153,* 261-266.

Shibuya, K., Tanaka, J., Kuboyama, N., Murai, S., & Ogaki, T. (2004). Cerebral cortex activity during supramaximal exhaustive exercise. *Journal of Sports Medicine and Physical Fitness, 44,* 215-219.

Skolnick, A. J., & Davidson, R. J. (2002). Affective modulation of eyeblink startle with reward and threat. *Psychophysiology, 39*, 835-850.

Smith, J.C., & O'Connor, P.J. (2003). Physical activity does not disturb the measurement of startle and corrugator responses during affective picture viewing. *Biological Psychology, 63*, 293-310.

Smith, J.C., O'Connor, P.J., Crabbe, J.B., & Dishman, R.K. (2002). Emotional responsiveness after low- and moderate-intensity exercise and seated rest. *Medicine and Science in Sports and Exercise, 34*, 1158-67.

Stanley, D.M. & Cumming, J. (2010a). Not just how one feels, but what one images? The effect of imagery use on affective responses to moderate exercise. *International Journal of Sport and Exercise Psychology, 8*, 343-359.

Stanley, D. M. & Cumming, J. (2010b) Are we having fun yet? Testing the effects of imagery use on the affective and enjoyment responses to acute moderate exercise. *Psychology of Sport and Exercise, 11*, 582-590.

Subudhi, A.W., Dimmen, A.C., & Roach, R.C. (2007) Effects of acute hypoxia on cerebral and muscle oxygenation during incremental exercise. *Journal of Applied Physiology, 103,* 177-183.

Sutton, S.K., & Davidson, R.J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8,* 204-210.

Svedahl, K., & MacIntosh, B.R..(2003). Anaerobic threshold: The concept and methods of measurement. *Canadian Journal of Applied Physiology, 28,* 299-323.

Takano, N. (2000). Respiratory compensation point during incremental exercise as related to hypoxic ventilatory chemosensitivity and lactate increase in man. *Japanese Journal of Physiology, 50*, 449-455.

Thayer, J.F., & Lane, R.D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders, 61,* 201-216.

Tieman, J.G., Peacock, L.J., Cureton, K.J., & Dishman, R.K. (2001). Acoustic startle eyeblink response after acute exercise. *International Journal of Neuroscience, 106*, 21-33.

Timinkul, A., Kato, M., Omori, T., Deocaris, C.C., Ito, A., Kizuka, T., . . . Soya, H. (2008). Enhancing effect of cerebral blood volume by mild exercise in healthy young men: A near-infrared spectroscopy study. *Neuroscience Research, 61*, 242-248.

Tuscan, L-A., Herbert, J.D., Forman, E.M., Juarascio, A.S., Izzetoglu, M., & Schultheis, M. (2013). Exploring frontal asymmetry using functional near-infrared spectroscopy: a preliminary study of the effects of social anxiety during interaction and performance tasks. *Brain Imaging and Behavior, 7,* 140-153.

Tye, K.M., Prakash, R., Kim, S.Y., Fenno, L.E., Grosenick, L., Zarabi, H., . . . Deisseroth, K. (2011). Amygdala circuitry mediating reversible and bidirectional control of anxiety. *Nature, 471*, 358-362.

van der Meulen, M., Allali, G., Rieger, S. W., Assal, F., & Vuilleumier, P. (2012). The influence of individual motor imagery ability on cerebral recruitment during gait imagery. *Human Brain Mapping*, Published online: doi:10.1002/hbm.22192.

van der Zee, P., Cope, M., Arridge, S.R., Essenpreis, M., Potter, L.A., Edwards, A.D., . . . Delpy, D.T. (1992). Experimentally measured optical pathlengths for the adult head, calf and forearm and the head of the newborn infant as a function of interoptode spacing. *Advances in Experimental Medicine and Biology, 316*, 143–53.

Van Hoesen, G.W., Morecraft, R.J., & Vogt, B.A. (1993). Connections of the monkey cingulate cortex. In: B.A. Vogt & M. Gabriel (Eds.). Neurobiology of cingulate cortex and limbic thalamus: A comprehensive handbook, (pp. 249-283). Boston: Birkhauser.

Van Landuyt, L.M., Ekkekakis. P., Hall, E.E., & Petruzzello, S.J. (2000). Throwing the mountains into the lakes: On the perils of nomothetic conceptions of the exercise-affect relationship. *Journal of Sport and Exercise Psychology, 22*, 208–234.

Wager, T.D., Jonides, J., & Reading, S. (2004). Neuroimaging studies of shifting attention: A meta-analysis. *NeuroImage, 22,* 1679-1693.

Wager, T.D., Phan, K.L., Liberzon, I., & Taylor, S.F. (2003). Valence, gender, and lateralization of functional brain anatomy in emotion: A meta-analysis of findings from neuroimaging. *Neuroimage, 19*, 513–531.

Wang, C.C., Chu, C.H., Chu, I.H., Chan, K.H., & Chang, Y.K. (2013). Executive function during acute exercise: the role of exercise intensity. *Journal of Sport and Exercise Psychology, 35*, 358-67.

Wasserman, K. (1986). The anaerobic threshold: definition, physiological significance and identification. *Advances in Cardiology, 35*, 1-23.

Wasserman, K. (1994). Coupling of external to cellular respiration during exercise: the wisdom of the body revisited. *American Journal of Physiology - Endocrinology and Metabolism, 266,* 519-539.

Weinberg, R. (2008). Does imagery work? Effects on performance and mental skills. *Journal of Imagery Research in Sport and Physical Activity, 3*, Issue 1, Article 1.

Welch, A.S., Hulley, A., Ferguson, C., & Beauchamp, M.R. (2007). Affective responses of inactive women to a maximal incremental exercise test: A test of the Dual-Mode Model. *Psychology of Sport and Exercise, 8*, 401-423.

Wells, J.G., Balke, B., & Van Fossan, D.D. (1957). Lactic acid accumulation during work; a suggested standardization of work classification. *Journal of Applied Physiology, 10*, 51-55.

Whipp, B.J., Davis, J.A., Torres, F., & Wasserman, K. (1981). A test to determine parameters of aerobic function during exercise. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 50,* 217-21.

Williams, D.M., Dunsiger, S., Ciccolo, J.T., Lewis, B.A., Albrecht, A.E., & Marcus, B.H. (2008). Acute affective response to a moderate-intensity exercise stimulus predicts physical activity participation 6 and 12 months later. *Psychology of Sport and Exercise, 9*, 231-245.

Williams, D.M., Dunsiger, S., Jennings, E.G., & Marcus, B.H. (2012). Does affective valence during and immediately following a 10-min walk predict concurrent and future physical activity? *Annals of Behavioral Medicine, 44*, 43-51.

Williamson, J., McColl, R., Mathews, D., Ginsberg, M. & Mitchell, J.H. (1999). Activation of the insular cortex is affected by the intensity of exercise. *Journal of Applied Physiology, 87*, 1213-1219.

Williamson, J., Nobrega, A.C.L., McColl, R., Mathews, D., Winchester, P., Friberg, L., Mitchell, J.H. (1997). Activation of the insular cortex dynamic exercise in humans. *Journal of Physiology, 504*, 764-764.

Wilson, C., Smith, D., Burden, A., & Holmes, P. (2010). Participant-generated imagery scripts produce greater EMG activity and imagery ability. European Journal of Sport *Science, 10*, 417-425.

Woo, M., Kim, S., Kim, J., Petruzzello, S.J., & Hatfield, B.D. (2009). Examining the exercise-affect dose-response relationship: does duration influence frontal EEG asymmetry? *International Journal of Psychophysiology, 72*, 166-172.

World Health Organisation. (2010). *Global recommendations on physical activity for health.* [online]. Available at: http://www.who.int/rpc/guidelines/9789241599979/en/index.html. [Accessed 20 February 2011].

Yeung, R.R. (1996). The acute effects of exercise on mood state. *Journal of Psychosomatic research, 40,* 123-41.

Appendices

Appendix 1

Information sheets and informed consent

1a Study I (chapter 4)

1b Studies II and IV (chapters 5 and 7)

1c Study III (chapter 6)

1d Study V (chapter 8)

Appendix 1a

INFORMATION SHEET FOR PARTICIPANTS

Brain responses to physical activity using near infrared spectroscopy

Thank you for showing an interest in this project. Please read this information sheet carefully before deciding whether or not to participate. It is important that you understand why the research is being undertaken and what it will involve. Ask us if there is anything not clear or if you would like more information. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the project about?
This project is looking at what happens in the brain during physical activity using a neuroimaging technique called Near Infrared Spectroscopy (NIRS). Oxygenation levels during physical activity will be measured to allow us to identify parts of the brain and processes being used. This will contribute to a better understanding of the psychological and biological effects of physical activity.

What type of participants are needed?
We are looking for men and women between 18 and 45 years of age. To take part in this study you should be in good health and a non-smoker.

What will participants be asked to do?
There will be one visit lasting around one hour at St Lukes Campus, University of Exeter. You should not drink alcohol for 24 h, or have caffeine and food 2 h before testing.

Some basic questionnaires will be completed about your general health and physical activity habits. Your height, weight, heart rate and blood pressure will be recorded. You will then be seated on an exercise bike with a reclining back rest.

To measure how much oxygen is being used by the brain (specifically the front part in this study) near infrared light will be used. Measurements of your head will be taken, like the distance from ear to ear, forehead to neck; placement of two rubber two rubber strips (about 4cm by 1cm). They will be placed on either side of your forehead near to the hairline. They contain a light source which shines infrared light onto the skin, and a light detector which measures how much light is reflected back. The amount of light reflected depends on how much oxygen is in the blood underneath the rubber strip. This technique is very safe and you will not experience any unusual sensations from the light. Adhesive gel will be used to hold the strips in place with a bandage over the top.

You will wear a facemask so we can monitor your breathing rate. The facemask will still allow you to see and talk to the researcher so any problems at any time during your participation will be dealt with immediately. You will then begin exercising for a short warm up. As the test begins you will experience a slight increase in the load you pedal at each minute until you feel that you need to stop. While you are completing the exercise session you will be asked at several intervals to rate how you feel and how hard you are working. The exercise will take around 15 minutes.

Can participants change their mind and withdraw from the project?
You may withdraw from the project at any time and without any disadvantage to yourself of any kind. You may also request that any information collected from you to date be destroyed or deleted and not be used either now or in the future.

What data or information will be collected and what use will be made of it?
Name, address and contact details will be collected along with the answers to the health and safety questionnaires. Your height, weight, heart rate and blood pressure will be recorded. You will wear a facemask to monitor your breathing rate to show how hard you are working during exercise. Only the research team will have access to the data. Names and addresses will be removed and replaced with ID codes. All data will be stored in study files on a password protected computer. You are most welcome to request a copy of the results of the study should you wish.

What if participants have any questions?
If you have any questions about our project, either now or in the future, please feel free to contact either:

Research Student:	or	Research Supervisor:
Gavin Tempest		Associate Professor Gaynor Parfitt
(contact number)		*(contact number)*
(email address)		*(contact number)*

What do I do next?
If you have read and understood everything that we want you to do and are happy to take part in the study, please inform the researcher.

This project has been reviewed and approved by the Ethics Committee
of the School of Sport and Health Sciences.

CONSENT FORM FOR PARTICIPANTS

Brain responses to physical activity using Near Infrared Spectroscopy

I have read the information sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage.

I know that:-

1. My participation in the project is entirely voluntary;

2. I am free to withdraw from the project at any time without any disadvantage.

2. My height, weight, heart rate and blood pressure will be noted. My breathing and blood flow (using NIRS) will be monitored during exercise and rest.

3. I may experience some physical discomfort during the exercise sessions. This discomfort will cease at the end of the session.

4. The personal data will be destroyed at the conclusion of the project but any raw data on which the results of the project depend will be retained in secure storage for future academic use.

5. The results of the project may be published but my anonymity will be preserved.

I agree to take part in this project.

..... ……

(Signature of participant) (Date)

This project has been reviewed and approved by the Ethics Committee of the School of Sport and Health Sciences.

Appendix 1b

UniSA Sansom Institute for Health Research

PARTICIPANT INFORMATION STATEMENT

The effects of fatigue on brain blood flow and perceptions of exercise intensity

You are invited to participate in a study that investigates individual perception of how you feel and how hard you perceive you are working during exercise, and how this relates to changes in oxygen being used in the brain. We aim to investigate this during fresh and fatigued conditions. More specifically, we hope to learn how exercise intensity is related to oxygenation in the brain. You are invited to participate because you report that you currently exercise at a moderate intensity for more than 150 minutes a week.

If you agree to participate in this study you will be randomly allocated to one of two groups. Participants in both groups will complete up to two graded exercise tests (GXTs) and two fixed intensity exercise bouts over two or three visits to the laboratory. The GXTs will commence at a low intensity, and will become progressively harder throughout the duration of the test. These bouts of exercise will finish when you are so tired that you have to stop. Following a 10 minute rest after some of the GXTs, you will be asked to complete a separate exercise bout at a fixed intensity until you are so tired that you have to stop. During these sessions we will ask you to rate how hard you are working and how you feel. Visits to the lab that require a GXT will last either 45 mins or 1.5 hours in total.

During each exercise test your heart rate and breathing (using a face mask) will be continuously recorded. To measure how much oxygen is being used by the brain during exercise, near infrared light will be used. Measurements of your head will be taken, such as the distance from ear to ear and forehead to neck. A cap will be placed on your head which holds two sets of sensors. Some of these sensors emit light onto tissue, and others measure the light a short distance away. This method indicates how much oxygen is within the blood in the area underneath the sensors. This technique is very safe and you will not experience any unusual sensations from the near infrared light. Adhesive stickers will be used on your forehead and a bandage will wrap around your head to secure the cap in place. A small accelerometer (which measures movement) will be clipped onto the bandage like a small head torch. A photograph will be taken of the position of the cap and sensors on your forehead as a reference of the location for subsequent sessions. You are welcome to see all the apparatus and techniques to be used prior to the study. You are always free to stop the study at any time.

You will have at least 48 hours in between each visit to the lab. You should not drink alcohol for 24 hours, or have caffeine or food two hours before testing. You may experience some discomfort during the exercise sessions but this discomfort will be transient. During your first visit to the lab we will assess a number of health measures to ensure you are fit to participate in the study (including height, body mass (weight), resting blood pressure and questions about your current health). On your first visit you will complete a questionnaire about your preference for and tolerance of exercise intensity.

Any information that is obtained from this study and that can be identified with you will remain confidential (except as required by law). Data will be stored in a form that is re-identifiable, but we will keep the coding for re-identification password secured and separately from the data file. Data will be kept for 5 years. Data will be stored in a locked cabinet at the Exercise for Health and Human Performance Group, University of South Australia. Results will be reported in scientific publications in a way in which you cannot be identified. If you would like to receive your own personal data and know about the results of this study, please tell the investigator and we will arrange for these to be sent to you at the end of the study.

Your participation in this project is voluntary. Your decision whether or not to participate will not prejudice your future relations with the University of South Australia. If you decide to participate you are free to withdraw your consent and to discontinue your participation at any time without prejudice.

If you have any questions, please ask us. If you have any additional questions later, Gavin (contact email) will be happy to answer them.

This project has been approved by the University of South Australia's Human Research Ethics Committee. If you have any ethical concerns about the project or questions about your rights as a participant please contact the Executive Officer of this Committee, Tel: (contact number) Email: (contact email).

You will be given a copy of this form to keep.

UniSA | Sansom Institute for Health Research

CONSENT FORM

Project title: The effects of fatigue on blood flow in the brain and perceptions of exercise intensity.

- I have read the Participant Information Sheet and the nature and purpose of the research project has been explained to me. I understand and agree to take part.
- I confirm that I am between the ages of 18 and 45 years of age.
- I understand the purpose of the research project and my involvement in it.
- I understand that I may withdraw from the research project at any stage and that this will not affect my status now or in the future.
- I understand that I can withdraw my consent following completion of the study.
- I understand that while information gained during the study may be published, I will not be identified and my personal results will remain confidential.
- I give/do not give the researcher my permission to use the data collected for this study for the subsequent research specified in the participant information sheet for the study.
- I give/do not give the researcher my permission to store my data in a form that is re-identifiable.
- I give/do not give the researcher my permission to take a photograph of the equipment set-up as specified in the information sheet for the study.
- I understand that I may receive a copy of my personal results and a summary of the project findings.

Name of participant…….....

Signed…….**Date**…….

I have provided information about the research to the research participant and believe that he/she understands what is involved.

Researcher's signature and date……...

This project has been approved by the University of South Australia's Human Research Ethics Committee. If you have any ethical concerns about the project or questions about your rights as a participant please contact the Executive Officer of this Committee, Tel: (contact number) Email: (contact email).

Appendix 1c

INFORMATION SHEET FOR PARTICIPANTS

Brain responses to imagery use during exercise using near infrared spectroscopy.

Thank you for showing an interest in this project. Please read this information sheet carefully before deciding whether or not to participate. It is important that you understand why the research is being undertaken and what it will involve. Ask us if there is anything not clear or if you would like more information. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the project about?
This project is looking at what happens in the brain during exercise using a neuroimaging technique called Near Infrared Spectroscopy (NIRS). The measurement of oxygenation lin the brain using NIRS during exercise will allow us to determine function in those parts of the brain which will contribute to a better understanding of the psychological and biological effects of exercise.

What type of participants are needed?
We are looking for men and women between 18 and 45 years of age. To take part in this study you should be in good health and a non-smoker.

What will participants be asked to do?
There will be four visits at St Lukes Campus, University of Exeter. The first visit will last between 45-60 minutes and the subsequent ttwo 45-75 minutes. You will be tested at the same time of day with at least 48 hours in between each visit. You should not drink alcohol for 24 h, or have caffeine and food 3 h before testing.

Upon arrival at the first session, some basic questionnaires will be completed about your general health and physical activity habits. Your height, weight, heart rate and blood pressure will be recorded. You will then be seated on an exercise bike with a reclining back rest.

To measure how much oxygen is being used by the brain (specifically the front part in this study) during exercise, near infrared light will be used. Measurements of your head will be taken, like the distance ear to ear, forehead to neck; for the placement of two rubber strips (about 4cm by 1cm). They will be placed on either side of your forehead near to the hairline. They contain a light source, which shines infrared light onto the skin, and a light detector, which measures how much light is reflected back. The amount of light reflected depends on how much oxygen there is in the blood underneath the rubber strip. This technique is very safe and you will not experience any unusual sensations from the light. Adhesive stickers will be used to hold the strips in place with surgical tape and a bandage over the top. A photograph will be taken of the position of the probes on your forehead as a reference of the location for subsequent sessions.

You will wear a facemask so we can monitor your breathing rate. The facemask will still allow you to see and talk to the researcher so any problems at any time during your participation will be dealt with immediately. You will have a travel pillow to stabilise your head and neck and some Velcro straps (like a seatbelt) to minimise movement while in the bike chair. You will then

begin exercising for a short warm up. As the test begins you will experience a slight increase in the load you pedal at each minute until you feel that you need to stop. While you are completing the exercise session you will be asked at several intervals to rate how hard you are working. The exercise will take around 15 minutes.

In the subsequent two sessions you will complete two short exercise bouts at different set rates (moderate to high intensity). Upon arrival you will be positioned on the exercise bike as before. One session will involve the use of enjoyment and energy imagery techniques, after which you will be asked to complete a post exercise imagery script and imagery ability evaluation. The other session will involve exercise only.

Can participants change their mind and withdraw from the project?
You may withdraw from the project at any time and without any disadvantage to yourself of any kind. You may also request that any information collected from you to date be destroyed or deleted and not be used either now or in the future.

What data or information will be collected and what use will be made of it?
Name, address and contact details will be collected along with the answers to the health and safety questionnaires. Your height, weight, heart rate and blood pressure will be recorded. You will wear a facemask to monitor your breathing rate during exercise as well as probes being placed on your forehead to measure how hard you are working during exercise. A picture will be taken of the probe location for future reference. Only the research team will have access to the data. Names and addresses will be removed and replaced with ID codes. All data will be stored in study files on a password protected computer. You are most welcome to request a copy of the results of the study should you wish.

What if participants have any questions?
If you have any questions about our project, either now or in the future, please feel free to contact either:

Research Student:	or	Research Supervisor:
Gavin Tempest		Associate Professor Gaynor Parfitt
(contact number)		*(contact number)*
(email address)		*(email address)*

What do I do next?
If you have read and understood everything that we want you to do and are happy to take part in the study, please inform the researcher.

This project has been reviewed and approved by the Ethics Committee
of the School of Sport and Health Sciences.

CONSENT FORM FOR PARTICIPANTS

Brain responses to imagery use during exercise using near infrared spectroscopy

I have read the information sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage.

I know that:-

1. My participation in the project is entirely voluntary;

2. I am free to withdraw from the project at any time without any disadvantage.

3. My height, weight, heart rate and blood pressure will be noted. My breathing and blood flow (using NIRS) will be monitored during exercise and rest.

4. A photograph will be taken of the position of the probes on my forehead as a reference of their location.

5. I may experience some physical discomfort during the exercise sessions. This discomfort will cease at the end of the session.

6. The personal data will be destroyed at the conclusion of the project but any raw data on which the results of the project depend will be retained in secure storage for future academic use.

7. The results of the project may be published but my anonymity will be preserved.

I agree to take part in this project.

..... ……

(Signature of participant) (Date)

This project has been reviewed and approved by the Ethics Committee of the School of Sport and Health Sciences.

Appendix 1d

INFORMATION SHEET FOR PARTICIPANTS

Brain responses to physical activity using acoustic startle eye blink methods and near infrared spectroscopy

Thank you for showing an interest in this project. Please read this information sheet carefully before deciding whether or not to participate. It is important that you understand why the research is being undertaken and what it will involve. Ask us if there is anything not clear or if you would like more information. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the project about?
This project is looking at what happens in the brain during physical activity using a reflex called the acoustic startle eye-blink response (ASER) and a neuroimaging technique called Near Infrared Spectroscopy (NIRS). The eye-blink response is very sensitive to changes in how a person feels (pleasure-displeasure). This combined with the measurement of oxygenation levels through NIRS during physical activity will allow us to identify parts of the brain and processes being used which will contribute to a better understanding of the psychological and biological effects of physical activity.

What type of participants are needed?
We are looking for men and women between 18 and 45 years of age. To take part in this study you should be in good health and a non-smoker.

What will participants be asked to do?
There will be four visits at St Lukes Campus, University of Exeter. The first visit will last between 60-90 minutes and the subsequent three 30-45minutes. You will be tested around the same time of day with a minimum of 48 hours between each visit. You should not drink alcohol for 24 h, or have caffeine and food 3 h before testing.

Upon arrival at the first session, some basic questionnaires will be completed about your general health and physical activity habits. Your height, weight, heart rate and blood pressure will be recorded. You will then be seated on an exercise bike with a reclining back rest. You will complete a short hearing task by listening to different tones through a set of headphones and responses recorded.

You will then be given a headband which has a small light detector attached, which will sit around 1cm in front of your eye. This detects the light reflected from your pupil. It will not touch your eye and you will still be able to blink as normal. You will wear a facemask so we can monitor your breathing rate. The facemask will still allow you to see and talk to the researcher so any problems at any time during your participation will be dealt with immediately. You will have a travel pillow to stabilise your head and neck and some Velcro straps (like a seatbelt) to minimise movement while in the bike chair.

You will then complete a 15 minute acoustic startle response task. This involves listening to white noise through a set of headphones; this will sound like a continuous unchanging blur of conversation. You will hear loud but very short (less than a second) bursts of sound (equivalent

to the sound level at a rock concert) throughout the test, which will make you blink. At first it may make you jump but any discomfort will cease following testing. The sound level is very safe and there are no lasting side effects from participating in this task. Once the task is completed you will remove the headband and headphones.

You will then complete a short exercise test using the neuroimaging method; NIRS. To measure how much oxygen is being used by the brain (specifically the front part in this study) near infrared light will be used. Measurements of your head will be taken, like the distance from ear to ear, forehead to neck; to locate the placement of two rubber strips (about 4cm by 1cm). They will be placed on either side of your forehead near to the hairline. They contain a light source which shines infrared light onto the skin, and a light detector which measures how much light is reflected back. The amount of light reflected depends on how much oxygen is in the blood underneath the rubber strip. This technique is very safe and you will not experience any unusual sensations from the light. Adhesive gel will be used to hold the strips in place with a bandage over the top.

You will continue to wear a facemask so we can monitor your breathing rate. You will then begin exercising for a short warm up. As the test begins you will experience a slight increase in the load you pedal at each minute until you feel that you need to stop. While you are completing the exercise session you will be asked at several intervals to rate how you feel and how hard you are working. The exercise will take around 15 minutes.

In the subsequent three sessions you will complete three short exercise tests (one per session) at different set rates, each of 10 minutes duration. Upon arrival you will be positioned on the exercise bike as before. The startle task will be combined with NIRS during the exercise so the rubber strips will be placed on your forehead and you will wear the headband, headphones and facemask. You will hear bursts of sound through the headphones during the exercise, which you should try to ignore.

Can participants change their mind and withdraw from the project?
You may withdraw from the project at any time and without any disadvantage to yourself of any kind. You may also request that any information collected from you to date be destroyed or deleted and not be used either now or in the future.

What data or information will be collected and what use will be made of it?
Name, address and contact details will be collected along with the answers to the health and safety questionnaires. Your height, weight, heart rate and blood pressure will be recorded. You will wear a facemask to monitor your breathing rate to show how hard you are working during exercise. A video camera will record the acoustic startle task to record spontaneous eye blinks. Only the research team will have access to the data. Names and addresses will be removed and replaced with ID codes. All data will be stored in study files on a password protected computer. You are most welcome to request a copy of the results of the study should you wish.

What if participants have any questions?
If you have any questions about our project, either now or in the future, please feel free to contact either:

Research Student:	or	Research Supervisor:
Gavin Tempest		Associate Professor Gaynor Parfitt
(*contact number)*		(contact number)
(email address)		(email address)

What do I do next?
If you have read and understood everything that we want you to do and are happy to take part in the study, please inform the researcher.

This project has been reviewed and approved by the Ethics Committee
of the School of Sport and Health Sciences.

CONSENT FORM FOR PARTICIPANTS

Brain responses to physical activity using acoustic startle eye blink methods and near infrared spectroscopy

I have read the information sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage.

I know that:-

1. My participation in the project is entirely voluntary;

2. I am free to withdraw from the project at any time without any disadvantage.

3. My height, weight, heart rate and blood pressure will be noted. My breathing and blood flow (using NIRS) will be monitored during exercise and rest. Eye movement during the acoustic startle task will be video recorded.

4. I may experience some discomfort due to the acoustic startle stimuli (loud bursts of sound) and some physical discomfort during the exercise sessions. This discomfort will cease at the end of the session.

5. The personal data will be destroyed at the conclusion of the project but any raw data on which the results of the project depend will be retained in secure storage for future academic use.

6. The results of the project may be published but my anonymity will be preserved.

I agree to take part in this project.

.....

……

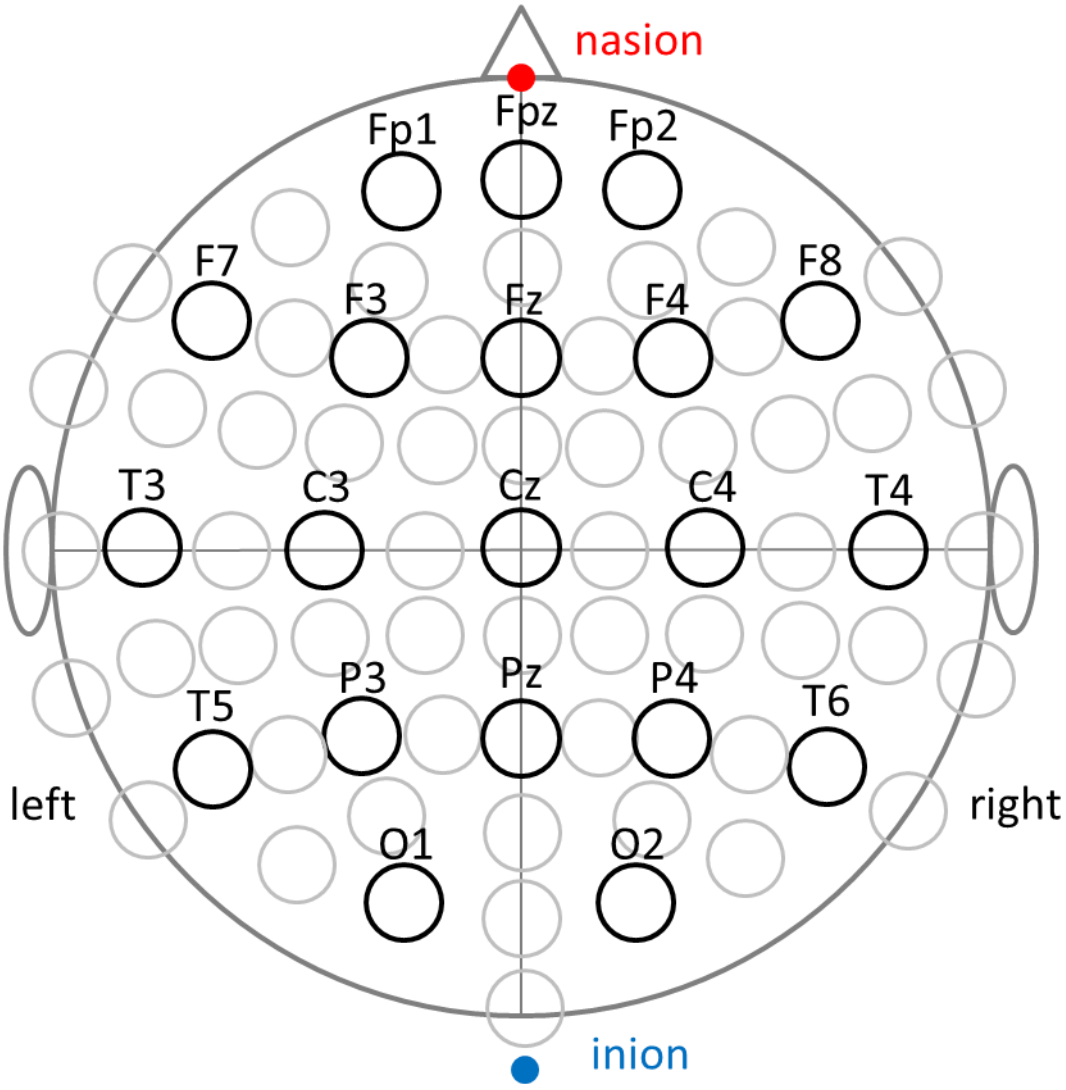
(Signature of participant) (Date)

This project has been reviewed and approved by the Ethics Committee of the School of Sport and Health Sciences.

Appendix 2

The international 10/20 system for electrode placement

Appendix 2



The 10 / 20 EEG electrode placement map is represented by the black and grey circles, respectively. Regions labelled indicate: F = frontal, Fp = frontal polar, C = central' P = parietal, T = temporal, O = occipital and z = zero (midline).

The *nasion* is the dent at the top of the nose and the *inion* is the bony part of the skull above the neck.

The figure is reproduced and modified from Hughes, J.R. (1994). *EEG in Clinical Practice* (2nd Ed.). Newton, MA: Butterworth-Heinemann.

Appendix 3

Additional data

3a Study I (chapter 4)

3b Study II (chapter 5)

3c Study III (chapter 6)

3d Study V (chapter 7)

3e Study IV (chapter 8)

Appendix 3a

Study I (chapter 4)

Means and (*SD*)s from separate Group (high, low tolerance) by Region (right, left) by Time (corresponding to intensities below VT, VT, RCP, End) ANOVAS with repeated measures for cerebral ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff (μM).

Cerebral ΔO_2Hb

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
High tolerance	Right	2.08 (2.85)	2.57 (3.31)	12.03 (5.76)	14.44 (7.72)
	Left	1.26 (2.33)	2.39 (3.53)	10.69 (5.52)	12.86 (7.95)
Low tolerance	Right	2.39 (2.42)	2.87 (2.44)	12.09 (6.03)	12.48 (6.93)
	Left	2.42 (2.34)	3.01 (2.31)	13.73 (5.96)	14.16 (6.53)

Cerebral ΔHHb

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
High tolerance	Right	.21 (.63)	.14 (.79)	1.19 (1.18)	4.02 (2.28)
	Left	.18 (.57)	.09 (.93)	.82 (1.58)	3.39 (2.85)
Low tolerance	Right	.18 (.47)	.08 (.62)	1.36 (2.19)	3.65(2.88)
	Left	.07 (.57)	-.06 (.74)	.74 (2.07)	2.58 (2.50)

Cerebral ΔtHb

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
High tolerance	Right	2.29 (2.86)	2.72 (3.62)	13.22 (5.83)	18.45 (7.53)
	Left	1.44 (2.20)	2.49 (3.28)	11.51 (5.77)	16.51 (8.21)
Low tolerance	Right	2.57 (2.31)	2.95 (2.25)	13.45 (7.06)	15.87 (7.68)
	Left	2.49 (2.35)	2.95 (2.26)	14.47 (7.34)	16.74 (7.94)

Cerebral ΔHbDiff

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
High tolerance	Right	1.87 (2.98)	2.43 (3.17)	10.84 (5.93)	10.42 (8.53)
	Left	1.09 (2.59)	2.30 (3.98)	9.87 (5.72)	9.21 (8.70)
Low tolerance	Right	2.21 (2.60)	2.79 (2.77)	10.73 (5.71)	9.10 (7.29)
	Left	2.35 (2.45)	3.07 (2.58)	13.00 (5.06)	11.58 (5.89)

Appendix 3b

Study II (chapter 5)

Means and (*SD*)s from separate Region (1-8) by Time (below VT, VT, RCP, End) ANOVAS with repeated measures for cerebral ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff (µM).

Cerebral ΔO_2Hb

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
Region	1	-.15 (2.32)	.58 (2.59)	3.18 (5.52)	2.44 (6.59)
	2	.84 (2.45)	1.77 (2.99)	5.09 (5.48)	5.15 (6.39)
	3	-.02 (2.43)	.94 (2.86)	4.02 (6.60)	4.46 (7.60)
	4	.55 (1.95)	1.55 (2.79)	5.10 (5.75)	6.52 (6.08)
	5	.00 (1.56)	.91 (1.89)	3.83 (5.52)	4.13 (6.44)
	6	.45 (1.96)	1.61 (2.39)	5.56 (5.73)	6.23 (5.59)
	7	.65 (1.54)	1.52 (1.99)	4.46 (4.62)	4.23 (5.93)
	8	.49 (2.50)	1.71 (3.14)	5.53 (5.39)	6.07 (6.02)

Cerebral ΔHHb

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
Region	1	.69(1.79)	.84(1.89)	2.42(2.54)	3.95(3.72)
	2	.67(1.22)	.96(1.42)	2.78(2.56)	4.45(3.89)
	3	1.02(1.64)	1.25(1.61)	2.93(2.00)	4.68(2.01)
	4	.63(1.27)	.81(1.19)	2.19(1.72)	4.00(2.82)
	5	.73(.96)	.94(1.04)	2.73(1.52)	4.85(2.46)
	6	.48(1.26)	.60(1.18)	2.05(1.33)	3.46(1.80)
	7	.77(1.05)	.83(1.07)	2.69(2.04)	4.48(2.91)
	8	.61(1.43)	.71(1.38)	2.79(2.11)	4.53(2.41)

Cerebral ΔtHb

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
Region	1	.54(3.17)	1.42(3.45)	5.61(6.49)	6.39(7.79)
	2	1.51(2.62)	2.73(3.41)	7.88(6.71)	9.60(8.20)
	3	1.10(2.92)	2.19(3.29)	6.95(7.13)	9.14(7.77)
	4	1.18(2.24)	2.36(2.86)	7.29(6.33)	10.52(7.31)
	5	.73()(1.68)	1.85(2.14)	6.56(6.15)	8.98(7.72)
	6	.93(2.27)	2.22(2.52)	7.62(5.87)	9.69(6.29)
	7	1.43(1.87)	2.35(2.15)	7.15(5.54)	8.71(7.61)
	8	1.10(3.04)	2.42(3.44)	8.32(6.28)	10.60(6.97)

Cerebral ΔHbDiff

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
Region	1	-.84(2.67)	-.26(2.94)	.76(5.63)	-1.51(7.34)
	2	.16(2.84)	.81(3.20)	2.31(5.32)	.70(6.67)
	3	-.94(2.76)	-.31(3.27)	1.09(6.66)	-.21(7.96)
	4	-.08(2.41)	.74(3.20)	2.91(5.65)	2.52(6.04)
	5	-.73(1.98)	-.03(2.17)	1.11(5.28)	-.71(5.96)
	6	-.03(2.38)	1.02(2.81)	3.51(5.17)	2.76(5.43)
	7	-.12(1.86)	.69(2.38)	1.78(4.51)	-.25(5.41)
	8	-.11(2.70)	1.00(3.42)	2.74(5.25)	1.54(5.96)

Pearson's bivariate correlation coefficients (*r*)

Cerebral ΔO_2Hb and affective responses

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
Region	1	-.173	-.088	-.553**	-.380
	2	-.115	-.082	-.526**	-.325
	3	-.059	.08	-.287	-.296
	4	-.001	.113	-.356	-.460*
	5	-.298	-.173	-.499*	-.517*
	6	-.068	-.088	-.592**	-.560*
	7	-.093	.083	-.615**	-.549*
	8	.042	.105	-.533**	-.457*

*$p < .05$, **$p < .01$.

Cerebral ΔHHb and affective responses

		Time (corresponding to intensities of exercise)			
		below VT	VT	RCP	End
Region	1	.082	.006	-.116	-.148
	2	.060	-.143	-.036	-.032
	3	.119	.155	-.227	-.193
	4	.033	.029	-.183	-.154
	5	.031	.225	-.260	-.314
	6	.191	.371	-.004	-.077
	7	.095	.279	.075	-.034
	8	.044	.300	.115	.066

*$p < .05$, **$p < .01$.

Appendix 3c

Study III (chapter 6)

Means and (*SD*)s from separate Condition(control, imagery) by Region (right, left) by Time (corresponding to intensities GET, 20, 40, 60, 80, 100% exercise duration) ANOVAS with repeated measures for cerebral ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff (μM).

Exercise (5%) above VT

Cerebral ΔO_2Hb

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	3.87(2.11)	4.67(2.77)	6.04(3.02)	6.70(3.37)	7.20(3.62)	7.71(4.07)
	Left	4.47(2.78)	5.16(3.18)	6.69(2.93)	7.54(2.96)	8.25(3.37)	8.59(3.77)
Imagery	Right	2.84(3.21)	4.17(3.54)	4.87(3.35)	5.45(3.82)	6.37(3.91)	6.85(4.15)
	Left	2.97(3.13)	4.20(3.81)	4.90(3.72)	5.23(4.20)	6.45(3.81)	6.93(4.03)

Cerebral ΔHHb

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	-.97(1.37)	-1.04(1.50)	-1.18(-.92)	-1.27(1.61)	-1.23(1.71)	-1.31(1.84)
	Left	-.92(1.10)	-.91(1.10)	-.92(1.11)	-1.02(1.32)	-1.00(1.31)	-1.09(1.45)
Imagery	Right	.21(1.09)	.10(.82)	-.04(.81)	-.04(.86)	-.02(1.12)	-.08(1.56)
	Left	.21(.94)	.08(.81)	-.02(.83)	-.01(.82)	-.03(.88)	-.19(1.07)

Cerebral ΔtHb

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	2.90(2.42)	3.63(2.90)	4.86(2.78)	5.43(2.89)	5.97(3.08)	6.39(7.50)
	Left	3.55(3.01)	4.25(3.32)	5.77(3.16)	6.52(3.22)	7.26(3.55)	7.50(3.92)
Imagery	Right	3.04(2.94)	4.27(3.76)	4.83(3.75)	5.41(4.19)	6.35(4.20)	6.77(4.33)
	Left	3.18(2.71)	4.28(3.74)	4.88(3.74)	5.22(4.26)	6.42(4.11)	6.74(4.25)

Cerebral ΔHbDiff

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	4.84(2.61)	5.71(3.39)	7.22(3.90)	7.97(4.43)	8.44(4.75)	9.02(5.25)
	Left	5.38(2.97)	6.07(3.41)	7.61(3.10)	8.57(3.27)	9.25(3.68)	9.68(4.16)
Imagery	Right	2.63(3.79)	4.06(3.50)	4.91(3.10)	5.48(3.63)	6.39(3.93)	6.93(4.53)
	Left	2.76(3.74)	4.12(4.05)	4.92(3.88)	5.24(4.31)	6.48(3.70)	7.12(4.10)

Exercise RCP to exhaustion

Cerebral ΔO_2Hb

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	3.33(3.98)	5.23(4.33)	6.32(4.84)	6.97(4.99)	7.80(5.37)	7.88(5.61)
	Left	2.93(3.86)	4.84(4.25)	5.92(4.16)	6.94(4.63)	7.61(4.63)	7.60(5.05)
Imagery	Right	2.22(3.65)	4.74(4.39)	5.77(4.43)	6.40(4.50)	6.99(4.99)	7.31(5.08)
	Left	2.11(3.97)	4.40(4.88)	5.82(5.14)	6.93(5.52)	7.35(6.07)	7.98(6.46)

Cerebral ΔHHb

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	1.43(1.99)	1.76(2.50)	2.09(2.72)	2.22(2.68)	2.65(2.93)	2.72(2.91)
	Left	1.76(2.08)	2.33(2.44)	2.63(2.60)	2.76(2.58)	3.02(2.69)	3.21(2.75)
Imagery	Right	2.03(2.33)	2.50(2.61)	2.81(2.67)	3.18(2.88)	3.34(3.04)	3.72(3.43)
	Left	2.25(2.54)	2.83(2.74)	3.24(2.71)	3.76(2.97)	3.85(3.12)	4.14(3.52)

Cerebral ΔtHb

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	4.76(3.65)	6.99(3.92)	8.41(4.46)	9.19(4.26)	10.45(4.90)	10.59(4.79)
	Left	4.68(3.23)	7.17(3.77)	8.55(3.76)	9.70(3.99)	10.64(4.03)	10.80(4.27)
Imagery	Right	4.25(2.50)	7.25(2.89)	8.58(2.58)	9.58(2.60)	10.33(2.80)	11.02(2.88)
	Left	4.37(3.12)	7.23(4.00)	9.07(4.00)	10.68(4.45)	11.19(4.42)	12.12(1.73)

Cerebral ΔHbDiff

		Time (corresponding to intensities of exercise)					
		GET	20	40	60	80	100
Control	Right	1.90(5.12)	3.47(5.88)	4.23(6.47)	4.76(6.78)	5.15(7.12)	5.16(7.54)
	Left	1.17(5.29)	2.51(5.81)	3.29(5.82)	4.18(6.34)	4.59(6.41)	4.39(6.91)
Imagery	Right	.19(5.59)	2.24(6.62)	2.96(6.84)	3.21(7.09)	3.65(7.77)	3.59(8.18)
	Left	-.14(5.90)	1.57(6.83)	2.58(7.17)	3.17(7.66)	3.50(8.58)	3.85(9.26)

Pearson's bivariate correlation coefficients (*r*)

Exercise (5%) above VT

Cerebral ΔO_2Hb, ΔHHb and affective responses (100% exercise duration)

	Control	Imagery
Cerebral ΔO_2Hb - Right	.170	.036
Cerebral ΔO_2Hb - Left	.221	-.164
Cerebral ΔHHb - Right	.060	-.564*
Cerebral ΔHHb - Left	-.040	-.128

*$p < .05$.

Cerebral ΔO_2Hb, ΔHHb and post-imagery evaluation scores (100% exercise duration)

	Visualise	Feel	Clarity	Emotion	Energised	Enjoyment
Cerebral ΔO_2Hb - Right	.386	.355	.417	-.186	.299	.415
CerebralΔ O_2Hb - Left	.445	.255	.58	-.001	.358	.601*
Cerebral ΔHHb - Right	.355	.288	-.021	.058	.428	.486
Cerebral ΔHHb - Left	.564*	.556*	.336	.181	.363	.431

*$p < .05$.

Exercise RCP to Exhaustion

Cerebral ΔO_2Hb, ΔHHb and affective responses (100% exercise duration)

	Control	Imagery
Cerebral O_2Hb - Right	-.215	.015
Cerebral O_2Hb - Left	-.172	-.087
Cerebral ΔHHb - Right	-.183	-.045
Cerebral ΔHHb - Left	-.242	-.119

Cerebral ΔO_2Hb, ΔHHb and post-imagery evaluation scores (100% exercise duration)

	Visualise	Feel	Clarity	Emotion	Energised	Enjoyment
Cerebral O_2Hb - Right	-.363	-.333	-.340	.383	-.107	-.088
Cerebral O_2Hb - Left	-.467	-.298	-.394	.269	-.081	-.043
Cerebral ΔHHb - Right	.327	.104	.424	-.346	-.021	-.033
Cerebral ΔHHb - Left	.243	.087	.337	-.232	-.044	-.057

Appendix 3d

Study IV (chapter 7)

Means and (*SD*)s from separate Condition (control, experimental) by Region (1-8) by Time (VT, VT-end, RCP, RCP-end) ANOVAS with repeated measures for cerebral ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff (µM).

Cerebral ΔO_2Hb

	Region	Time (corresponding to intensities of exercise)			
Condition		VT	VT-end	RCP	RCP-end
Control	1	.29(1.72)	5.99(3.47)	7.02(4.41)	7.92(5.33)
	2	.10(1.99)	7.48(4.66)	9.28(5.25)	11.18(7.03)
	3	.57(1.61)	6.77(3.01)	8.16(4.62)	9.25(6.17)
	4	.89(1.76)	7.41(3.90)	10.00(5.05)	12.24(6.52)
	5	1.06(1.44)	7.21(4.29)	8.91(5.92)	10.34(7.29)
	6	1.69(1.35)	8.91(3.83)	12.25(4.42)	15.25(5.11)
	7	.94(1.56)	7.76(4.01)	9.22(4.74)	10.17(5.29)
	8	.96(1.72)	9.12(4.07)	11.48(5.52)	14.24(6.49)
Experimental	1	1.14(1.81)	4.53(3.59)	4.29(4.57)	3.87(4.81)
	2	1.05(2.05)	5.08(3.09)	4.94(3.81)	5.79(5.06)
	3	1.40(1.82)	4.60(3.70)	4.41(4.83)	5.13(5.87)
	4	1.82(2.37)	5.61(3.62)	5.74(4.16)	6.78(5.67)
	5	1.45(1.53)	4.48(3.26)	4.37(4.43)	5.07(5.28)
	6	1.89(2.23)	6.61(4.10)	6.92(4.42)	7.80(5.51)
	7	1.63(2.10)	4.94(3.32)	4.73(4.25)	4.80(4.10)
	8	1.47(2.28)	6.85(3.56)	6.72(3.99)	7.41(5.00)

Cerebral ΔHHb

	Region	Time (corresponding to intensities of exercise)			
Condition		VT	VT-end	RCP	RCP-end
Control	1	0.85(1.10)	0.69(1.50)	2.25(2.72)	4.57(4.45)
	2	0.86(1.32)	1.47(2.18)	3.40(3.51)	6.27(5.20)
	3	0.92(1.13)	1.10(1.51)	2.73(2.16)	4.55(3.49)
	4	0.44(0.86)	0.73(1.20)	2.10(1.97)	4.16(3.18)
	5	0.94(1.32)	1.07(1.93)	2.39(2.67)	4.79(3.12)
	6	0.39(0.92)	0.46(1.41)	1.94(1.89)	4.32(2.38)
	7	0.77(1.42)	0.42(1.75)	1.69(2.18)	3.05(2.90)
	8	0.31(1.01)	0.31(1.12)	1.61(2.82)	3.84(3.60)
Experimental	1	0.56(.61)	1.72(1.43)	2.53(1.68)	4.80(3.19)
	2	0.38(.60)	1.23(1.25)	2.39(1.74)	4.76(3.42)
	3	0.50(.54)	1.26(1.38)	2.96(1.70)	6.10(3.62)
	4	0.18(.75)	1.09(1.34)	2.33(1.73)	5.05(3.30)
	5	0.50(.66)	1.03(.91)	2.44(1.56)	4.99(2.79)
	6	0.26(.53)	0.98(1.68)	1.87(1.15)	3.88(1.95)
	7	0.34(.58)	1.24(1.60)	1.72(1.47)	3.92(2.31)
	8	0.14(.73)	0.49(1.34)	2.05(1.53)	4.52(2.87)

Cerebral ΔtHb

	Region	Time (corresponding to intensities of exercise)			
Condition		VT	VT-end	RCP	RCP-end
Control	1	1.15(1.24)	6.69(4.14)	9.27(5.34)	12.49(6.94)
	2	.96(1.27)	8.95(6.12)	12.68(6.93)	17.44(9.34)
	3	1.49(1.55)	7.88(3.55)	10.89(5.21)	13.80(7.01)
	4	1.33(1.31)	8.13(4.47)	12.10(5.97)	16.39(7.57)
	5	2.01(1.43)	8.29(5.30)	11.30(7.58)	15.13(9.02)
	6	2.09(1.29)	9.37(4.44)	14.18(5.14)	19.57(5.54)
	7	1.71(1.14)	8.19(4.49)	10.91(5.79)	13.21(6.78)
	8	1.27(1.09)	9.43(4.52)	13.09(7.10)	18.08(8.39)
Experimental	1	1.70(1.76)	5.76(4.00)	6.81(4.34)	8.67(4.97)
	2	1.44(1.78)	6.17(3.96)	7.33(3.95)	10.54(6.42)
	3	1.90(7.85)	6.32(4.17)	7.38(4.64)	11.22(5.64)
	4	2.00(2.15)	6.88(4.06)	8.08(4.08)	11.83(6.11)
	5	1.94(1.87)	5.71(4.12)	6.81(4.85)	10.06(6.37)
	6	2.14(2.34)	7.64(4.61)	8.78(4.77)	11.68(6.06)
	7	1.97(2.07)	5.43(4.19)	6.45(4.65)	8.73(4.88)
	8	1.60(2.15)	7.83(4.89)	8.77(4.80)	11.93(6.74)

Cerebral ΔHbDiff

	Region	Time (corresponding to intensities of exercise)			
Condition		VT	VT-end	RCP	RCP-end
Control	1	-.56(2.61)	5.30(3.39)	4.77(5.02)	3.36(6.95)
	2	-.76(3.12)	6.02(3.92)	5.89(5.65)	4.91(8.11)
	3	-.34(2.32)	5.67(3.19)	5.42(4.98)	4.71(7.18)
	4	.45(2.44)	6.68(3.65)	7.91(4.80)	8.08(6.92)
	5	.12(2.36)	6.14(4.03)	6.52(5.18)	5.55(6.66)
	6	1.30(1.91)	8.46(3.70)	10.31(4.44)	10.93(5.73)
	7	.17(2.76)	7.34(4.26)	7.53(4.56)	7.12(5.18)
	8	.64(2.60)	8.82(3.90)	9.86(5.14)	10.39(6.29)
Experimental	1	.57(2.06)	3.30(3.61)	1.76(5.35)	-.93(6.48)
	2	.67(2.45)	3.99(2.64)	2.54(4.42)	1.03(5.78)
	3	.91(1.96)	2.88(3.76)	1.45(5.56)	-.98(7.95)
	4	1.65(2.78)	4.35(3.68)	3.41(4.89)	1.73(6.98)
	5	.95(1.43)	3.24(3.08)	1.94(4.55)	.08(5.55)
	6	1.63(2.25)	5.59(3.74)	5.05(4.36)	3.93(5.62)
	7	1.29(2.23)	4.45(2.85)	3.01(4.34)	.88(4.52)
	8	1.33(2.61)	5.87(2.67)	4.66(3.68)	2.89(4.58)

Appendix 3e

Study V (chapter 8)

Means and (*SD*)s from separate one-way Intensity (below VT, VT, RCP) ANOVAS with repeated measures for cerebral ΔO_2Hb, ΔHHb, ΔtHb and ΔHbDiff (μM).

Cerebral ΔO_2Hb

		Time (corresponding to intensities of exercise)		
		below VT	VT	RCP
Region	Right	1.93 (1.66)	5.05 (2.55)	6.00 (3.72)
	Left	2.46 (1.99)	5.52 (2.50)	7.99 (4.94)

Cerebral ΔHHb

		Time (corresponding to intensities of exercise)		
		below VT	VT	RCP
Region	Right	-.41 (1.32)	-.85 (1.29)	1.62 (2.48)
	Left	-.51 (1.35)	-.22 (1.35)	1.33 (2.18)

Cerebral ΔtHb

		Time (corresponding to intensities of exercise)		
		below VT	VT	RCP
Region	Right	1.53 (1.45)	4.20 (2.28)	7.61 (2.78)
	Left	1.95 (1.20)	4.43 (2.43)	8.89 (4.69)

Cerebral ΔHbDiff

		Time (corresponding to intensities of exercise)		
		below VT	VT	RCP
Region	Right	2.34 (2.63)	5.89 (3.34)	4.37 (5.67)
	Left	2.97 (2.94)	5.73 (3.21)	6.66 (5.95)

Pearson's bivariate correlation coefficients (*r*)

Exercise below VT

	ASER (Amplitude)	Affect	Tolerance
Cerebral ΔO_2Hb - Right	.385	-.173	-.182
Cerebral ΔO_2Hb - Left	.273	-.137	.289
Cerebral ΔHHb - Right	.056	.195	-.298
Cerebral ΔHHb - Left	.071	-.079	.049
ASER (Amplitude)		.378	-.287
Affect			-.301

Exercise at VT

	ASER (Amplitude)	Affect	Tolerance
Cerebral ΔO_2Hb - Right	.462*	-.307	-.436
Cerebral ΔO_2Hb - Left	.545*	-.194	-.293
Cerebral ΔHHb - Right	-.183	.188	-.023
Cerebral ΔHHb - Left	.041	.062	-.316
ASER (Amplitude)		-.177	-.380
Affect			.071

*$p < .05$.

Exercise at RCP

	ASER (Amplitude)	Affect	Tolerance
Cerebral ΔO_2Hb - Right	.159	-.505*	-.081
Cerebral ΔO_2Hb - Left	.049	-.315	.082
Cerebral ΔHHb - Right	-.273	.385	.222
Cerebral ΔHHb - Left	-.220	.227	.360
ASER (Amplitude)		-.509*	-.496*
Affect			.354

*$p < .05$.

Appendix 4

Modified 6-20 Rating of Perceived Exertion Scale (Borg, 1998)

6	No exertion at all
7	
8	Extremely light
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard (heavy)
16	
17	Very hard
18	
19	Extremely hard
20	Maximal exertion